Periodontal Manifestations of Local and Systemic Diseases

Colour Atlas and Text

For Personal Use Only
Library of School of Dentistry, TUMS

HIGH DENT

PRODUCTS SERVICES TRAINING

همیار دندانسازان و دندانپزشکان در پروتز

مرکز تخصصی پروتزهای دندانی

های دنت

طراحی و ساخت انواع پروتزهای دندانی بویژه ایمپلنت

برگزار کننده دوره های آموزشی تخصصی وجامع دندانسازی و...

با ما همراه باشید...

WWW.HIGHDENT.IR

@highdent

@highdent

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

George Laskaris · Crispian Scully

Periodontal Manifestations of Local and Systemic Diseases

Colour Atlas and Text

With a Contribution by Dimitris N. Tatakis

Foreword by Prof. Jan Lindhe

With 392 Figures in Colour
and Appendix with 8 Tables

For Personal Use Only
Library of School of Dentistry, TUMS

Springer

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

ISBN 978-3-642-62788-0

Library of Congress Cataloging-in-Publication Data

Laskaris, George. Periodontal manifestations of local and systemic diseases/George Laskaris, Crispian Scully; with a chapter on Normal periodontium by Dimitris Tatakis, and a foreword by Lan Lindhe. p.; cm. Includes bibliographical references and index. ISBN 978-3-642-62788-0 1. Oral manifestations of general diseases – Atlas. I. Scully, Crispian. II. Title. [DNLM: 1. Oral Manifestations – Atlas. 2. Periodontium – pathology – Atlas. WU 17 L345p 2003] RC815.L373 2003 617.6'32–dc21

This work is subject to copyright. All rights are reserved, whether the whole or part of the material is concerned, specifically the rights of translation, reprinting, reuse of illustrations, recitation, broadcasting, reproduction on microfilm or in any other way, and storage in data banks. Duplication of this publication or parts thereof is permitted only under the provisions of the German Copyright Law of September 9, 1965, in its current version, and permission for use must always be obtained from Springer-Verlag. Violations are liable for prosecution under the German Copyright Law.

http://www.springer.de

© Springer-Verlag Berlin Heidelberg 2003
Originally published by Springer-Verlag Berlin Heidelberg in 2003

The use of general descriptive names, registered names, trademarks, etc. in this publication does not imply, even in the absence of a specific statement, that such names are exempt from the relevant protective laws and regulations and therefore free for general use.

Product liability: The publishers cannot guarantee the accuracy of any information about dosage and application contained in this book. In every individual case the user must check such information by consulting the relevant literature.

Cover design: eStudio Calamar, Pau, Spain
Product management and layout: B. Wieland, Heidelberg
Reproduction and typesetting: AM-production, Wiesloch

SPIN 10718566 24/3130 – 5 4 3 2 1 0

ISBN 978-3-642-62788-0 ISBN 978-3-642-55596-1 (eBook)
DOI 10.1007/978-3-642-55596-1

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Dedication to Christos Laskaris

Christos Laskaris was the son of Professor George Laskaris and his wife Vivi. Christos was a final year student in the School of Dentistry, University of Belgrade. He was a bright young 24 year old man with an engaging personality, full of energy and vitality, always polite and friendly, always having a good word to say for everybody, always smiling and willing to help. We had all hoped he would succeed his father as an outstanding stomatologist.

It was a tremendous misfortune to loose Christos so unexpectedly, and so unjustly, from a sudden illness.

This book is dedicated to his memory.

Professor Crispian Scully

This book is also dedicated to our families for their constant support of our efforts.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Authors

George Laskaris · MD, DDS, PhD
Head of Oral Medicine Clinic, "A. Sygros" Hospital
Associate Professor of Oral Medicine
Medical School, University of Athens
37 Ipsilandou Street
10676 Athens
Greece

Crispian Scully · CBE, MD, PhD, MDS, MRCS, FDSRCS, FDSRCPS, FFDRCSI, FDSRCSE, FRCPath, FMedSci
Director
Eastman Dental Institute for Oral Health Care Sciences
University College London
256 Gray's Inn Road
London WC1X 8LD
United Kingdom

Contributor

Dimitris N. Tatakis · DDS, PhD
Section of Periodontology
College of Dentistry, Health Sciences Center
The Ohio State University, Columbus, Ohio
USA

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Foreword

I am delighted to see the appearance of the first comprehensive atlas recognizing the important relationship between periodontal and systemic health and disease. The atlas is not intended to cover the new areas of interest in relation to periodontal health and cardiac and other disease, but rather to demonstrate the systemic diseases that manifest to the periodontist and hygienist. The atlas thus covers a vast range of systemic disorders that may present with gingival or periodontal lesions and highlights the importance of the periodontal and oral medicine teams in diagnosing and managing patients with sometimes complex medical problems.

The authors, Professor George Laskaris and Professor Crispian Scully, are both active clinicians, teachers and researchers in oral medicine, with wide experience and special interest in the oral medicine/periodontal interface and both have published widely in their fields.

I welcome this contribution to the advancement of care for the patient with periodontal disease.

Jan Lindhe
Professor Emeritus of Periodontology
University of Gothenburg, Sweden

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Preface

The periodontium is a part of the oral tissues of great interest to dentists and particularly to the periodontal team. The importance has now been recognized to extend beyond local disorders to a wide range of conditions that may affect periodontal health.

The most recent classification proposed by the International Workshop for the Classification of Periodontal Diseases and Conditions in 1999 is testimony to an increasing scientific interest of the international dental community in periodontal pathology, and the wide range of local and systemic disease affecting the periodontium.

This atlas aims to illustrate and evaluate diseases targeting the gingivae and periodontium from the viewpoint of oral medicine and to increase further the awareness and interest of dentists, periodontologists and hygienists regarding the vast spectrum of diseases that can affect the gingivae and periodontium. It is also hoped that it will help open new scientific and professional avenues by demonstrating the important role of these professional groups in the art and science of medicine. The ultimate beneficiaries should be our patients – not least since several of the diseases may be life-threatening and require special expertise in early diagnosis and treatment.

Due to the greater variety of diseases affecting the gingivae and periodontium than was recognized in the above-mentioned workshop, we encountered a number of difficulties in adhering fully to that classification and thus elected to adopt a classification based on three main groups of pathologic entities: local diseases, manifestations of systemic disorders in the gingivae and periodontium, and cysts, tumours and neoplasms.

For each entity we present the clinical manifestations, frequency of involvement of the gingivae and other structures in the mouth and elsewhere, differential diagnosis, laboratory work-up, and principles of treatment. The illustrations are arranged so that for every disease there is one or more illustration demonstrating representative lesions of the gingivae. In addition, for the systemic diseases, we have often included photographs of lesions of other areas of the oral mucosa, extra-oral mucosal surfaces, or the skin, in order to better emphasize the spectrum of clinical manifestations.

We are certain that, with more comprehensive biological and medical education, dentistry will open new horizons in health services within the next few years. This was the motivation for the publication of the present work, and it is our hope that the atlas will help in the realization of this goal.

George Laskaris, Athens, 2002

Crispian Scully, London, 2002

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Acknowledgements We thank our patients and nurses who have taught us so much over the years, and continue to do so, and all those students and colleagues with whom we have worked and interacted, who may have shared the clinical care of some patients, and/or may have knowingly or otherwise contributed ideas or content. In this respect we thank especially Professors Oslei Paes de Almeida (Brazil), Jose-Vicente Sebastian-Bagan (Spain), Johann Beck-Managetta (Austria), Roman Carlos (Guatemala) , Marco Carrozzo (Italy), Roderick Cawson (UK), Pedro Diz Dios (Spain), Drore Eisen (USA), Joel Epstein (Canada), Eleni Gagari (USA), Sergio Gandolfo (Italy), Andreas Katsabas (Greece), Christos Kittas (Greece), Jens Pindborg (Denmark; deceased), Stephen Porter (UK), Peter Reichart (Germany), Pierre-Luigi Sapelli (Italy), Sol (Bud) Silverman (USA), John Stratigos (Greece), Isaac Van der Waal (Netherlands) and Antony Vareltzidis (Greece). We are grateful to Dimitris Tatakis for contributing the chapter on Normal Periodontium, and to colleagues who contributed clinical illustrations, including Drs A. Parashis, N. Papadogeorgakis, S. Ashan, M. Glick, M. Griffiths, S. Porter, T. Hodgson, O. Almeida, M. Spostos, and N. Kumar.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Contents

Part I The Normal Periodontium

1 Introduction . . . 3
Anatomy . . . 3
Clinical Features of the Gingiva . . . 3
Biochemistry and Physiology . . . 9
Microbiology and the Role of Microorganisms in the Pathogenesis of Periodontal Disease . . . 10
Pathology – Immunological Mechanisms . . . 11
Diagnosis of Periodontal Diseases . . . 13
Periodontal Examination and Diagnosis . . . 13

Part II Local Diseases and Lesions

2 Inflammatory Diseases . . . 23
Dental Plaque-Induced Gingivitis . . . 23
Chronic Periodontitis . . . 25
Aggressive Periodontitis . . . 27
Mouthbreathing Gingivitis . . . 30
Gingival Diseases of Specific Origin . . . 31
Granulomatous Gingivitis . . . 31
"Geographic" Gingivitis . . . 33

3 Developmental Disorders . . . 37
Torus Mandibularis . . . 37
Multiple Exostoses of the Maxillary Alveolar Process . . . 38
Fibrous Tuberosity . . . 38

4 Drug-Influenced Gingival Lesions and Diseases . . . 41
Gingival Enlargement Due to Phenytoin . . . 41
Gingival Enlargement Due to Calcium Channel Blockers . . . 42
Gingival Enlargement Due to Ciclosporin . . . 44
Plasma Cell Gingivitis (Idiopathic Circumorificial Plasmacytosis) . . . 47
Allergic Gingivitis . . . 49
Heavy Metal Deposition and Other Causes of Hyperpigmentation . . . 51

5 Abscesses and Fistulas . . . 55
Gingival Abscess . . . 55
Periodontal Abscess . . . 57
Pericoronal Abscess . . . 57
Tooth Abscess . . . 58
Oroantral Fistula . . . 60

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

6 Physical Lesions 63
Self-Inflicted Injury 63

7 Chemical Lesions 67

8 Thermal Lesions 71

9 Lesions Due to Radiation 73
Mucositis 73
Osteoradionecrosis 74

10 Pigmented Disorders 77
Physiological Pigmentation 77
Melanotic Macule 79
Naevus 80
Metal Tattoo 83
Smoker's Melanosis 85

11 Allergic and Foreign Body Reactions 89
Reactions to Dental Materials and Oral Health Care Products 89
Materia Alba 91

12 Bacterial Infections 93
Necrotizing Ulcerative Gingivitis 93
Necrotizing Ulcerative Periodontitis 95
Pericoronitis 96

13 Viral Infections 99
Herpetic Gingivitis 99
Primary 99
Secondary 101
Papillomaviruses 103
Condyloma Acuminatum 103
Verruca Vulgaris 103
Papilloma 105

14 Fungal Infections 109
Acute Pseudomembranous Candidiasis 109
Erythematous Candidiasis 110

15 Aphthous Ulceration 113

Part III Systemic Diseases

16 Genetic Disorders 119
Hereditary Gingival Fibromatosis 119
White Sponge Naevus 120
Laband Syndrome 121
Papillon-Lefèvre Syndrome 123
Hypophosphatasia 125
Glycogen Storage Disease 1 b 126
Hurler's Syndrome 127
Focal Palmoplantar and Oral Mucosa Hyperkeratosis Syndrome . . 129
Familial Acanthosis Nigricans 131
Darier's Disease 133

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Cowden's Disease . . . 134
Tuberous Sclerosis . . . 136
Gardner's Syndrome . . . 138
Sturge-Weber Angiomatosis . . . 138
Klippel-Trenaunay-Weber Syndrome . . . 140
Hereditary Epidermolysis Bullosa . . . 142
Chondroectodermal Dysplasia . . . 145
Focal Dermal Hypoplasia Syndrome . . . 147
Orofaciodigital Syndrome Type I . . . 148
Ehlers-Danlos Syndrome . . . 149
Hereditary Haemorrhagic Telangiectasia . . . 151
Cystic Fibrosis . . . 153
Down's Syndrome . . . 154

17 Bacterial Infections . . . 159
Tuberculosis . . . 159
Syphilis . . . 161
Actinomycosis . . . 163
Gonococcal Infection . . . 164

18 Viral Infections . . . 167
Measles . . . 167
Chickenpox . . . 168
Herpes Zoster . . . 169
Infectious Mononucleosis . . . 171
Hand-Foot-and-Mouth Disease . . . 173

19 HIV Infection . . . 177
Bacterial Infections . . . 178
Necrotizing Ulcerative Gingivitis . . . 178
Necrotizing Ulcerative Periodontitis . . . 178
Bacillary Angiomatosis . . . 180
Other Bacterial Infections . . . 180
Viral Infections . . . 182
Herpes Simplex Virus . . . 182
Herpes Zoster . . . 182
Cytomegalovirus . . . 182
Condyloma Acuminatum . . . 184
Mycoses . . . 184
Linear Gingival Erythema . . . 184
Candidiasis . . . 185
Systemic Mycoses . . . 187
Neoplasms . . . 187
Kaposi's Sarcoma . . . 187
Non-Hodgkin's Lymphomas . . . 189
Drug Reactions . . . 190

20 Systemic Mycoses . . . 193
Histoplasmosis . . . 193
Mucormycosis . . . 194
Paracoccidioidomycosis . . . 195

21 Haematological Disorders and Malignancies . . . 197
Familial Neutropenia . . . 197
Cyclic Neutropenia . . . 198
Agranulocytosis . . . 200

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Aplastic Anaemia . . . 200
Thrombocytopenic Purpura . . . 202
Plasminogen Deficiency . . . 203
Myelodysplastic Syndrome . . . 204
Leukaemias . . . 206
Multiple Myeloma . . . 209
Soft Tissue Plasmacytoma . . . 211
Non-Hodgkin's Lymphoma . . . 212
Burkitt's Lymphoma . . . 213
Cutaneous T-cell Lymphoma . . . 214
Hodgkin's Disease . . . 216

22 Skin Diseases . . . 219
Erythema Multiforme . . . 219
Toxic Epidermal Necrolysis . . . 221
Cicatricial Pemphigoid . . . 223
Bullous Pemphigoid . . . 225
Linear IgA Disease . . . 227
Pemphigoid Gestationis . . . 229
Epidermolysis Bullosa Acquisita . . . 230
Dermatitis Herpetiformis . . . 232
Pemphigus Vulgaris . . . 234
Lichen Planus . . . 236
Chronic Ulcerative Stomatitis . . . 239
Psoriasis . . . 240

23 Autoimmune Diseases . . . 245
Lupus Erythematosus . . . 245
Sjögren's Syndrome . . . 247
Systemic Sclerosis . . . 249
Mixed Connective Tissue Disease . . . 250
Graft-Versus-Host Disease . . . 251
Behçet's Disease . . . 253
Reiter's Disease . . . 255

24 Orofacial Granulomatosis . . . 259
Melkersson-Rosenthal Syndrome . . . 259
Crohn's Disease . . . 261
Sarcoidosis . . . 263
Pyostomatitis Vegetans . . . 264

25 Metabolic Disorders . . . 267
Porphyria . . . 267
Amyloidosis . . . 268
Langerhans Cell Histiocytosis . . . 270

26 Endocrine Disorders . . . 273
Diabetes Mellitus . . . 273
Sex Steroid Hormones . . . 274
Addison's Diseases . . . 276

27 Paraneoplastic Disorders . . . 279
Malignant Acanthosis Nigricans . . . 279
Paraneoplastic Pemphigus . . . 280

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

28 Vitamin Deficiency 283
Scurvy 283

Part IV Cysts, Tumours, Potentially Malignant, Neoplasms and Fibro-Osseous Lesions

29 Cysts 287
Periapical Cyst 287
Eruption Cyst 288
Gingival Cyst of Newborn 289
Gingival Cyst of Adult 290
Palatine Papilla Cyst 291
Dentigerous Cyst 292
Keratocyst 294

30 Tumour-Like Lesions 297
Fibrous Epulis and Denture-Induced Granuloma (Epulis Fissuratum) 297
Pyogenic Granuloma 299
Giant Cell Granuloma (Giant Cell Epulis; Peripheral Giant Cell Granuloma) 301
Central Giant Cell Lesion 303
Haemangioma 303
Lymphangioma 304

31 Potentially Malignant 307
Erythroplasia 307
Keratoses/Leukoplakia 308

32 Benign Neoplasms 313
Fibromas 313
Lipoma 313
Neurofibroma 314
Ossifying Fibroma 316
Chondroma 318
Salivary Adenomas 319
Pleomorphic Adenoma (Mixed Salivary Gland Tumour) 319

33 Malignant Neoplasms 321
Oral Squamous Cell Carcinoma 321
Verrucous Carcinoma 323
Salivary Carcinomas 324
Mucoepidermoid Carcinoma 324
Acinic Cell Carcinoma 324
Adenoid Cystic Carcinoma (Cylindroma) 325
Osteosarcoma 325
Ewing's Sarcoma 326
Leiomyosarcoma 327
Chondrosarcoma 328
Metastatic Neoplasms 329
Classic Kapos'i Sarcoma 331
Melanoma 332

34 Fibro-osseous Lesions 335
Paget's Disease 335

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Appendix 337
Classification of Gingival Lesions by Signs and Symptoms 339

Subject Index. 343

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

PART I

The Normal Periodontium

Dimitris N. Tatakis

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

CHAPTER 1

Introduction

This chapter will review the normal periodontium (anatomy and physiology), the etiology and pathogenesis of gingivitis and periodontitis, and the diagnosis of periodontal diseases.

Anatomy

Periodontal Tissues

The following tissues, in order of encounter from the vestibular or oral aspect, constitute the periodontium and support the teeth: gingiva, alveolar bone, periodontal ligament and cementum. Under ideal healthy conditions, the gingiva is the only clinically observable periodontal tissue and the tissue on which most of the pathological manifestations occur. For this reason, the emphasis in this introductory chapter will be on the gingiva.

Clinical Features of the Gingiva

The gingiva, i. e. the keratinized mucosa that covers the teeth and the alveolar bone, is demarcated from the alveolar mucosa by the mucogingival junction (MGJ) (Figs. 1–5). In health, the gingiva should have a light or salmon pink colour, compared to a much darker or red colour for the alveolar mucosa (Figs. 1, 2). Depending on the extent of skin pigmentation of the individual, the gingiva may also display significant pigmentation (Fig. 4).

The gingiva is divided into free or marginal and attached gingiva (Fig. 5). The free gingiva constitutes the movable, most coronal portion of the tissue. The attached gingiva is the immovable portion that extends to the MGJ. In less than 50 % of gingiva examined, a shallow groove runs parallel to the gingival margin and delineates the free from the attached gingiva (Fig. 5). This gingival groove is located 1–2 mm apical to the margin and its formation appears to depend on the organization of the underlying connective tissue collagen fibres. The attached gingiva often exhibits stippling (orange peel-like appearance) of the surface, most evident on the facial aspect of maxillary anterior teeth (Figs. 2, 4). In periodontally healthy subjects, the buccolingual dimension (thickness) of facial gingiva ranges between 0.80 mm (mandibular canines) and 1.52 mm (mandibular 2nd molars). The apicocoronal dimension (width) of the attached gingiva ranges between 1 and 10 mm and varies by site. It is the narrowest on the lingual of mandibular incisors, the facial of mandibular canines and first bicuspids, and the facial of maxillary first bicuspids. It is widest on the facial of maxillary incisors and the lingual of mandibular molars.

For Personal Use Only
Library of School of Dentistry, TUMS

G. Laskaris et al., *Periodontal Manifestations of Local and Systemic Diseases*
© Springer-Verlag Berlin Heidelberg 2003

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 1

Normal periodontium. The gingiva appears with a typical salmon pink colour

FIGURE 2

Normal periodontium. Note stippling of attached gingiva

FIGURE 3

Normal periodontium. The mucogingival junction is demonstrated by iodine staining of the alveolar mucosa (same case as Fig. 2)

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 4

Normal periodontium. Normal pigmentation in an individual with moderate skin pigmentation. Note stippling of attached gingiva

FIGURE 5

Normal periodontium. The free and attached gingiva and the gingival groove are evident, particularly on the incisors

For Personal Use Only
Library of School of Dentistry, TUMS

Histology of the Gingiva

The gingiva consists of epithelium and connective tissue (Fig. 6). The gingival epithelium, according to location and histological characteristics, can be divided into three types: oral, sulcular and junctional epithelium.

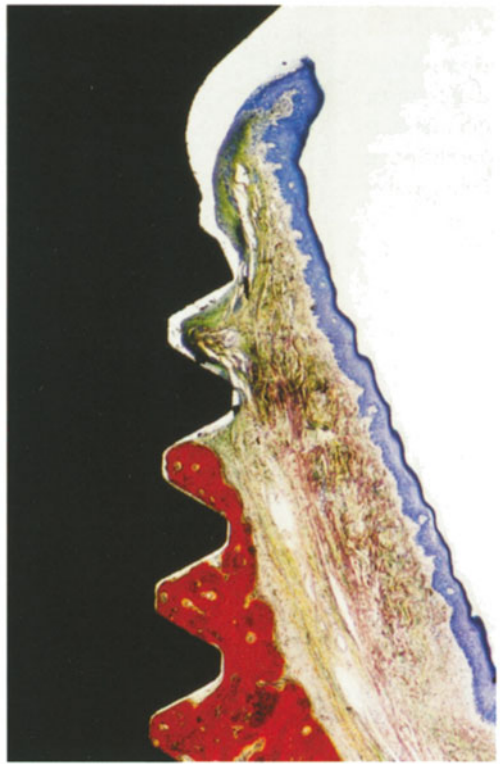
The oral epithelium (OE) lines the surfaces facing the oral cavity. The OE is thick, stratified, orthokeratinized or parakeratinized, with elongated, narrow pegs (projections). The epithelium is separated from and linked to the underlying connective tissue by a basement membrane (basal lamina). The underlying lamina propria is dense and firmly attached to the periosteum without distinct submucosa.

The connective tissue of the gingiva is primarily composed of fibroblasts, collagen and elastin fibres, vasculature and neuronal processes. Other cells normally present include mesenchymal and immune system cells, e. g. monocytes, macrophages, neutrophils, lymphocytes and plasma cells. The collagen fibres of the gingival connective tissue are either randomly scattered in the extracellular ground substance or organized in characteristically oriented bundles. These bundles are named based on

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 6

Animal histology of gingiva around a titanium dental implant. The epithelium is stained *blue*, the connective tissue is *green*, and the alveolar bone is *red*



the location and direction of the fibres. Circular, dentogingival, dentoperiosteal, and transseptal fibres are the main groups described.

The sulcular epithelium is the non-keratinized epithelium with shallow pegs that lines the sulcus and faces the tooth. Its stratified structure resembles that of oral epithelium, except for the lack of complete keratinization. It continues into the junctional epithelium, which is the epithelial tissue attached to the tooth (enamel, cementum or dentine) and located at the bottom of the sulcus.

The junctional epithelium (JE), whose length is typically 1–3 mm, is a non-keratinized, stratified epithelium that is only one to two layers thick at its apical end and a few layers thick at its coronal end, i.e. at the base of the sulcus. The connective tissue underlying the JE is highly vascularized. Beneath the JE, inflammatory cell infiltrates can be observed even in clinically healthy gingiva.

The distance between the depth of the gingival sulcus, i.e. the most coronal limit of the junctional epithelium, and the alveolar crest has been termed biological width. It consists of the sum of the junctional epithelium (epithelial attachment) length and the length of the connective tissue attachment located apical to the junctional epithelium and coronal to the alveolar crest. The biological width reportedly ranges from 0.75 to 4.33 mm, while the epithelial attachment and the connective tissue attachment ranges are 0.32 to 3.27 mm and 0.29 to 1.84 mm, respectively. The mean values reported are 1.14 mm for the epithelial attachment and 0.77 mm for the connective tissue attachment. The biological width may be significantly greater in teeth treated for periodontitis.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 7

Alveolar bone.
The alveolar bone proper (socket wall) and its numerous openings are evident

Alveolar Bone

The alveolar bone, which is clinically visible only in pathological conditions, has a structure similar to bone elsewhere in the body. The alveolar bone proper is the thin compact bone forming the walls of the tooth socket, and radiographically corresponds to the structure called lamina dura (Fig. 7). The alveolar bone proper is the bone that anchors the periodontal ligament fibres (Sharpey's fibres, see below). The wall of the alveolus shows numerous openings that allow passage of vessels and nerve fibres (Fig. 7).

Under ideal conditions, the crest of the interdental bone is located 1–2 mm apical to the cementoenamel junction (CEJ) of the adjoining teeth. The shape and location of the crest depends on the width of the interdental space and the relative position of the CEJs.

The buccal and lingual compact bone plates of the alveolar process surround trabecular bone. The trabeculation pattern of this bone varies widely, and depends on functional conditions. Like all other bone, alveolar bone undergoes constant remodelling, which entails both bone formation and bone resorption. Systemic (metabolic) bone diseases may affect alveolar bone in a manner similar to their effect on long bones.

Periodontal Ligament

The periodontal ligament (PDL) is a specialized loose connective tissue located between the alveolar bone and the tooth. PDL is the tissue that attaches the tooth (cementum) to the alveolar bone. It is a highly vascularized and innervated tissue with considerable adaptive capacity in response to mechanical stimulation. This property can probably be attributed to the very high turnover rate of the PDL connective tissue. The PDL contains precursor cells that give rise to bone-forming (osteoblasts) and cementum-forming (cementoblasts) cells. In addition to the above connective tissue cellular elements, PDL contains epithelial cells, the epithelial rests of Malassez. These remnants of the enamel organ (Hertwig's epithelial root sheath) are thought to contribute to cyst formation. Recent studies suggest that the epithelial rests may have functions related to the development of PDL innervation.

The principal collagen fibres of the PDL are organized in characteristically oriented bundles: the dentoalveolar crest group, the horizontal group, the oblique group, the apical group and the interradicular group.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

The principal PDL fibres that are embedded in the alveolar bone and in cementum are called Sharpey's fibres.

The space between tooth and alveolar bone that is occupied by the PDL (PDL space) can vary, on average, from 0.08 mm for impacted teeth to 0.2 mm for teeth under function. There is wide variation, however, among individuals and teeth. The PDL space and changes in PDL space width, either in response to mechanical forces or as a consequence of diseases that significantly affect collagen turnover (e.g. scleroderma), can be visualized radiographically.

Cementum

Although it is part of the tooth structure, cementum is also considered a tissue of the periodontium, as it helps attach the tooth to the gingiva and the alveolar bone. Cementum (and/or dentine) is clinically exposed when gingival recession is present (Fig. 8).

Cementum is found in two forms: acellular and cellular. Acellular cementum, as the name implies, does not contain embedded cementoblasts (cementocytes). It covers the cervical portion of the root and extends apically to a varying extent. The thickness of acellular cementum varies from a few microns near the cervix up to 200 µm towards the apex. Cellular cementum contains embedded cementocytes and thus resembles bone in structure. Cellular cementum thickness increases with age and may reach several millimetres. Although it shares structural characteristics with bone, unlike bone, cementum lacks vasculature, exhibits continuous apposition throughout life and does not normally undergo remodelling. Pathological resorption may occur under several different conditions, including excessive mechanical stimulation. Such resorption may be followed by repair, i.e. deposition of cellular cementum.

Cementum may also be visualized in radiographs, especially when significantly increased in thickness (hypercementosis).

Vasculature and Innervation

As alluded to previously, several periodontal tissues exhibit a high degree of vascularization. The gingiva receives its blood supply from supraperiosteal vessels as well as vessels emerging from the alveolar bone. The gingival blood vessels are typically arterioles, small veins and capillaries. Underlying the sulcular and junctional epithelium is a rich plexus of arterioles and venules. The PDL is the other highly vascularized periodontal tissue, with vessels originating from the alveolar bone and the dental (periapical) vasculature. The PDL vessels predominantly run parallel to the long axis of the tooth and are located closer to the bone surface than the cementum.

The gingiva is supplied by nerve fibres stemming from the trigeminal nerve (second and third division). These fibres reach the gingiva either from the supraperiosteal area (like the vessels) or through the coronal portion of the PDL.

The PDL is richly innervated by fibres originating either in the trigeminal ganglion or the mesencephalic trigeminal nucleus. These demonstrate differential distribution along the root. Ruffini-like nerve endings provide the PDL with mechanoreceptor function. Besides Ruffini-like endings, lamellated corpuscles and free nerve endings have been described in the PDL.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 8

Clinically evident cementum and/or dentine because of recession in the canine and premolar area

Biochemistry and Physiology

In animal studies, the quantitative composition of the gingiva has been reported to be 39 % epithelium, 5 % infiltrated connective tissue (ICT) and 56 % non-infiltrated connective tissue (NCT). ICT is present even in clinically healthy gingiva; the inflammatory infiltrate is composed of polymorphonuclear leukocytes (neutrophils), monocytes and macrophages, lymphocytes and plasma cells.

Water accounts for 66 % of the gingival tissue, while collagen fibres occupy 70 % of the connective tissue volume of NCT and only 18 % of the volume of ICT, indicative of the significant loss of collagen associated with inflammation of the gingiva. This loss clinically translates to the "transformation" of a very firm and dense tissue (healthy gingiva) to a friable and oedematous one.

Although collagen type I is the most abundant collagen type present in the connective tissue, with collagen type III being the second most prevalent type, other collagen types (e. g. IV, V, VI and XII) have also been found in the periodontium. Collagen types I and III constitute the bulk of the gingival fibres, while type V collagen filaments run parallel to the main fibres. In PDL, type I collagen is the main constituent of the principal fibres, although collagen type III, V, VI and XII are also found. Type I and III collagens are the principal component of cementum and alveolar bone organic matrix.

Besides collagen, glycosaminoglycans (e. g. dermatan sulphate, heparan sulphate, chondroitin sulphate, hyaluronate) and other extracellular matrix proteins (e. g. fibronectin, elastin, tenascin) contribute to the formation of the organic matrix. The organic matrix of the mineralized tissues contains several non-fibrous proteins such as osteopontin and bone sialoprotein-II. The organic matrix of alveolar bone and cementum also contains growth factors (biologically active protein molecules) which may be significant for tissue repair and regeneration. Examples of such polypeptides include platelet-derived growth factors (PDGFs), fibroblast growth factors (FGFs), transforming growth factors (TGFs), and bone morphogenetic proteins (BMPs).

The gingiva has a high turnover rate. It has been estimated that the turnover time for gingival epithelium is 10–12 days, while the reported

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

half-life of mature collagen is approximately 10 days in the lamina propria of the gingiva.

When subclinical inflammation is present in the gingiva, a fluid appears in the gingival crevice. This gingival crevicular fluid (GCF) is an exudate, with a composition that represents roughly a 30% dilution of serum. Besides serum proteins, GCF contains proteins produced locally in the gingiva, e.g. immunoglobulins synthesized and released by locally residing plasma cells.

Microbiology and the Role of Microorganisms in the Pathogenesis of Periodontal Disease

Colonization of the oral cavity with microorganisms occurs during birth or soon thereafter, through contact. Establishment of common plaque species such as *Streptococcus sanguis* is evident after tooth eruption. Studies have shown that pathogenic plaque bacteria, whether cariogenic or periodontopathogenic, are transmitted from parents to children.

The classic experimental gingivitis studies of Löe and coworkers in the 1960s provided proof that dental plaque is the etiological factor for gingivitis, a reversible condition. Subsequently, human epidemiological studies and animal experiments showed that plaque accumulation is a factor that leads to the development of periodontitis, i.e. attachment loss, which is an irreversible condition.

The development of bacterial plaque, as it accumulates and matures, progresses from a supragingival collection of aerobic or facultative, $Gram^+$ cocci and non-motile rods, to a subgingival aggregation of anaerobic, $Gram^-$, motile rods and other morphotypes. The organization of dental plaque in a biofilm, defined as a "matrix-enclosed bacterial populations adherent to each other and/or to surfaces or interfaces", has special implications for the survival and management of the oral flora, especially in relation to the pathogenesis and treatment of periodontitis.

The normal flora, or the flora that is compatible with periodontal health, comprises primarily $Gram^+$ facultative microorganisms. This flora includes *S. sanguis*, *S. mitis*, *Actinomyces naeslundii*, *A. viscosus*, and *Veillonella parvula*, among other species.

The development of gingivitis is associated with an increase in $Gram^-$ species. Species associated with gingivitis include: *Fusobacterium nucleatum*, *Treponema denticola*, *Prevotella intermedia*, *Campylobacter rectus*, *Actinomyces*, and *Veillonella*.

In periodontitis, the subgingival flora is characterized by a significant shift to $Gram^-$ anaerobic species, such as *Porphyromonas gingivalis*, *Bacteroides forsythus*, *Actinobacillus actinomycetemcomitans*, *Campylobacter rectus*, *P. intermedia*, *Eikenella corrodens*, and *Treponema* species. A few of these bacteria have been more strongly implicated in certain forms of periodontitis, e.g. *A. actinomycetemcomitans* in localized aggressive periodontitis (previously classified as localized juvenile periodontitis, see "Diagnosis of Periodontal Diseases"). Certain species exhibit strong associations with each other, such as *P. gingivalis*, *B. forsythus*, and *T. denticola*; these bacteria are often found together and have been strongly associated with severe periodontitis.

Advances in PCR (polymerase chain reaction) cloning of subgingival bacteria have resulted in the identification of both cultivable and non-cultivable bacteria from clinical specimens. PCR results show a greater complexity of subgingival microbiota than previously realized. It seems likely that non-culture-based microbial analysis will identify new periodontal

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

pathogens. Recent studies have also raised the possibility that certain viruses may contribute to the pathogenic process of some forms of periodontitis.

Pathology – Immunological Mechanisms

Pathology and Clinical Correlates

Quantitative analysis of human gingival biopsies from experimental gingivitis and periodontitis studies has led to the description of four histopathological stages in the development of the disease.

The initial lesion develops within 4 days of the first exposure to plaque and is characterized by acute inflammation, vasculitis, migration of polymorphonuclear neutrophils (PMNs), alteration in the coronal aspect of the junctional epithelium (JE) and loss of collagen around blood vessels. The clinical feature of this stage is increased gingival crevicular fluid flow.

The early lesion has, in addition to the features of the initial lesion, the following characteristics: accumulation of lymphocytes, altered (damaged) fibroblasts, further loss of interstitial collagen and proliferative changes in the basal cell layer of JE. The collagen loss in the infiltrated area may be as high as 70 %. The predominance of lymphocytes is the distinguishing feature of this stage, which develops within 7 days of the beginning of plaque accumulation and is clinically evident as gingivitis.

As time progresses, the early lesion evolves into the established lesion, which clinically corresponds to chronic gingivitis. This stage is characterized by continued presence of inflammation, appearance of plasma cells and immunoglobulins in the tissue, connective tissue loss, and severe changes in the junctional epithelium. The duration of the established lesion is indeterminate; chronic gingivitis may persist for a long time as such, without ever progressing to periodontitis.

The advanced lesion corresponds to periodontitis clinically and has, in addition to the features of the established lesion, the following characteristics: alveolar bone loss, pocket formation (attachment loss), distant fibrosis and altered plasma cells.

Increased flow of gingival crevicular fluid (GCF) is the first clinical sign to appear during development of gingivitis. GCF flow correlates well with the degree of histological inflammation. Another clinical sign of gingival inflammation is bleeding on probing. Bleeding sites, when compared to non-bleeding sites, exhibit significantly higher levels of inflammation.

Immunological Mechanisms – Immunopathogenesis

The epithelium of the gingiva forms the first natural barrier of the periodontium against microorganisms and foreign substances. The pathogenic potential of certain bacteria implicated in periodontitis, e. g. *P. gingivalis*, appears to be related to their ability to invade epithelial cells.

Polymorphonuclear leukocytes (PMNs or neutrophils) are an essential component of the innate or non-specific immune response against bacteria. Systemic conditions associated with periodontal involvement, such as localized aggressive periodontitis, diabetes, end cyclic neutropenia among others, are characterized by qualitative or quantitative PMN defects. Hereditary PMN disorders result in severe susceptibility to bacterial infections, in general, and periodontal infections, in particular.

Although PMNs are critical for periodontal health, evidence also suggests that these cells may be responsible, at least in part, for some of the tissue destruction encountered in periodontal diseases; in GCF or diseased gingiva from periodontitis patients, the collagenase present is pre-

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

dominantly of PMN origin. This has led to the pursuit of pharmacological anti-collagenase interventions.

Besides PMNs, macrophages and natural killer (NK) cells are also cellular components of the innate immune responses. Upon their migration from the vessel into the tissues, monocytes differentiate into macrophages, which contribute to pathogen elimination by phagocytosis. Hereditary defects in monocyte function, such as leukocyte adhesion deficiency syndromes, may lead to severe periodontal involvement (e.g. generalized aggressive periodontitis in children).

The adaptive or specific immune response relies on lymphocyte function. The gingiva contains significant numbers of the various lymphocyte classes and subclasses, distributed in the lamina propria and the infiltrated gingival connective tissue. Lymphocytes are categorized according to their function into B lymphocytes (B cells) and T lymphocytes (T cells). B cells, when appropriately stimulated, differentiate into plasma cells. The presence of high numbers of plasma cells in the infiltrated connective tissue is considered characteristic of long-standing periodontal pathology. T cells are classified as cytotoxic T cells and helper T cells. T helper cells have been classified into two major phenotypes, Th1 and Th2 cells, based on their functional properties, including the profile of cytokines produced. Some of the cytokines (see below) lead to pronounced inflammatory reactions, while others suppress inflammatory processes. The overall characteristics of the gingival immune (inflammatory) response to plaque bacteria appear to depend on the balance between Th1 and Th2 responses. Genetic predisposition to the development of responses with an exacerbated inflammatory component may lead to greater tissue destruction, i.e. severe periodontitis.

Severe immunodeficiencies, such as the congenital lack of both T and B cells, lead to lethal infections. Acquired immunodeficiencies, such as the severe depletion of T cells following HIV infection (AIDS), are also characterized by high susceptibility to infection, including the development of necrotizing ulcerative periodontitis.

Cytokines, protein factors produced by cells that affect other cells, have emerged as critical components in the immune response to plaque and the ensuing periodontal tissue destruction. Experimental periodontitis studies indicate that interleukin-1 (IL-1) and tumour necrosis factor (TNF) play a major role in the development of local inflammatory infiltrates and the accompanying alveolar bone loss. Interleukin-8 (IL-8) and monocyte chemoattractant protein-1 (MCP-1) are two cytokines that direct recruitment of PMNs and monocytes, respectively, to specific tissue sites such as the junctional epithelium and the subepithelial connective tissue. It appears that pathogens, such as *P. gingivalis*, may have the ability to inhibit local IL-8 production, which results in a lack of PMN recruitment and persistence of the pathogen. A lack of interleukin-10 (IL-10), a cytokine with significant anti-inflammatory properties, leads to more severe periodontal bone loss, as indicated from animal studies. Therefore, the relative balance between pro-inflammatory (IL-1, TNF) and anti-inflammatory (IL-10) cytokines may determine the severity of inflammation and tissue destruction.

Lipid molecules with pro-inflammatory activities such as the arachidonic acid metabolites (prostaglandins, thromboxanes, and leukotrienes) have been shown to be involved in inflammatory processes that lead to significant tissue remodelling or tissue destruction. The processes in question may be physiological (e.g. orthodontic tooth movement) or pathological (e.g. gingivitis, periodontitis) in nature. Use of non-steroidal anti-inflammatory drugs (NSAIDs), whose mechanism of action includes inhibi-

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

tion of prostaglandin synthesis, prevents the development of experimental gingivitis and halts the tissue damage associated with periodontitis.

Collectively, these findings suggest that control of the various inflammatory processes can prevent/limit the tissue destruction of periodontitis and prevent/reverse the tissue changes observed in gingivitis.

Diagnosis of Periodontal Diseases

Periodontal Examination and Diagnosis

The periodontal examination should include a written medical and dental history, a patient interview and a clinical and radiographic examination. The patient should be given the opportunity to consent to the examination procedures by signing an appropriate informed consent.

Self-Reported Medical and Dental History, Patient Interview

Self-Reported Medical History. This textbook is testament to the fact that numerous systemic conditions have important manifestations in the periodontium, in general, and the gingiva, in particular. Therefore, the significance of a complete medical history cannot be overstressed. Use of a written questionnaire to assess medical/dental history is essential. It should include a review of systems, with an emphasis on diseases known to affect the periodontal tissues and on conditions that may interfere with or complicate treatment. It should also elicit information on pertinent social/environmental factors (e.g. use of tobacco products, occupational exposure to hazardous materials). A list of relevant medications should be included, with a check/circle option.

Self-Reported Dental History. Identification of previous periodontal involvement/treatment, oral hygiene practices, and other relevant dental conditions and treatment (e.g. tooth extractions, orthodontic treatment, removable partial dentures) that may impact periodontal health should be included.

Patient Interview. During the interview process, the patient's chief complaint should be established. Then medical and dental histories are reviewed. Details regarding the timing and reasons behind any significant findings in the medical (e.g. specific drug treatment) or dental (e.g. time and reason for previous tooth extractions) history should be elicited. The interview process should be structured in a manner that helps ascertain the patient's dental/periodontal IQ (intelligence quotient) and treatment expectations. At the end of the interview, the practitioner should have a clear view of the patient's relevant or contributory past medical and dental history, of the need for any further medical clearance or consultation, and possibly of the need for medical or dental specialist referral.

Clinical Examination

The clinical examination should include recording of vital signs (blood pressure, pulse) and examination of extraoral and intraoral tissues. The extraoral exam should include evaluations of the skin and appendages, of the lips, of the temporomandibular joints (TMJ) and assessment of possible lymph node involvement. The intraoral exam should include visual assessment of all structures. Where necessary, tactile assessment (palpation) should also be performed. If indicated by the patient's chief complaint/medical history or by clinical findings, additional vital signs (e.g. temperature and respiration rate) should be recorded.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

The proper examination of the periodontium requires charting of the clinical parameters described below on appropriate form(s). The examination should start with visual assessment of the gingiva, alveolar mucosa and teeth (Figs. 1, 2). Deviations from normal colour and surface anatomy or structure are recorded. In some patients this may require the removal of loose debris, food particles, plaque accumulations or excessive supragingival calculus from certain areas. The presence of such material is recorded, as well as the presence of factors that lead to plaque retention. Such factors may be iatrogenic as in inadequate restorative treatments (e.g. amalgam overhangs, open crown margins), pathological as in carious lesions, and anatomical/developmental in nature such as deep radicular grooves, enamel projections, and enamel hypoplasia. Although visual inspection may provide abundant information, the proper detection of these factors requires detailed examination with a sharp explorer.

After the visual assessment, periodontal probing is performed. Typically, probing should be recorded in six sites per tooth (mid-buccal, distobuccal, distolingual, mid-lingual, mesiolingual and mesiobuccal). However, the entire circumference of the tooth should be probed for proper examination and data collection. Using a periodontal probe with appropriate length marks, the depth of the gingival sulcus is measured to the nearest mm (probing depth: PD). In periodontal health, the probing depth varies between 0.5 and 4.5 mm, depending on the site and the individual (Fig. 9). It should be pointed out that evidence from histological studies indicates that the clinical probing depth does not necessarily correspond to the histological sulcus (or pocket) depth. This is because the tip of the probe may easily penetrate through the sulcular or junctional epithelium, depending on the applied force, the size of the instrument, and the inflammatory status of the tissues.

From a practical standpoint, probing is what is usually performed and recorded. Should one need to have more detailed information, then clinical attachment level (CAL) measurements will be required. Concurrently with PD, the distance from the free gingival margin (GM) to the cementoenamel junction (CEJ) is measured (GM-CEJ). This measure, in conjunction with the probing depth, allows determination of the clinical attachment level (CAL). The formula used is CAL=PD–(GM-CEJ). In the event of marginal tissue recession, the GM-CEJ distance is given a negative value. In this manner, in sites with gingival recession, CAL equals the sum of PD and recession (GM-CEJ). The amount of CAL is used to determine the severity of periodontitis (e.g. slight, moderate and severe for CAL levels of 1–2, 3–4, and 5, respectively). During probing, sites exhibiting bleeding on probing (BOP) or presence of purulent exudate (pus) are also recorded.

Probing should be performed with the probe held parallel to the long axis of the tooth (Fig. 9). In the interproximal areas, the probe needs to be slightly angulated to reach the deepest site under the contact point. When calculus is present, the probe should be gently guided past the deposit to reach the bottom of the pocket. In sites with significant calculus deposits, probing may be difficult or impossible and result in inaccuracies. In addition, coexistent severe gingival inflammation may render probing unfeasible due to patient pain sensitivity. In such cases, debridement and/or anaesthesia may be necessary prior to periodontal probing and charting.

Furcation involvement and tooth mobility should be assessed and recorded. Furcation involvement is evaluated using a curved periodontal probe (Fig. 10). Furcations are usually classified as Class I, II or III, depending on degree of involvement, i.e. loss of attachment in the interradicular area in a horizontal direction. Tooth mobility is classified as 1, 2 or 3, depending on increasing severity. Typically, each tooth is held between two

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 9

Periodontal probing. The probe is held parallel to the long axis of the tooth

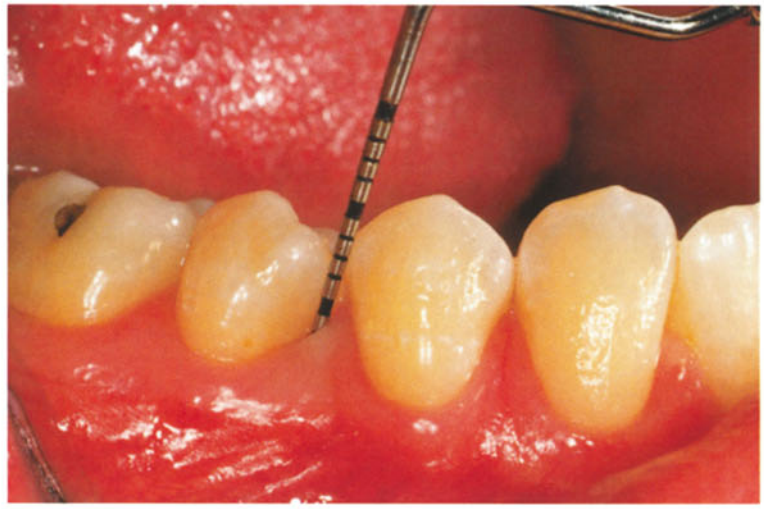


FIGURE 10

Furcation examination. The curved probe is used to detect furcation involvement

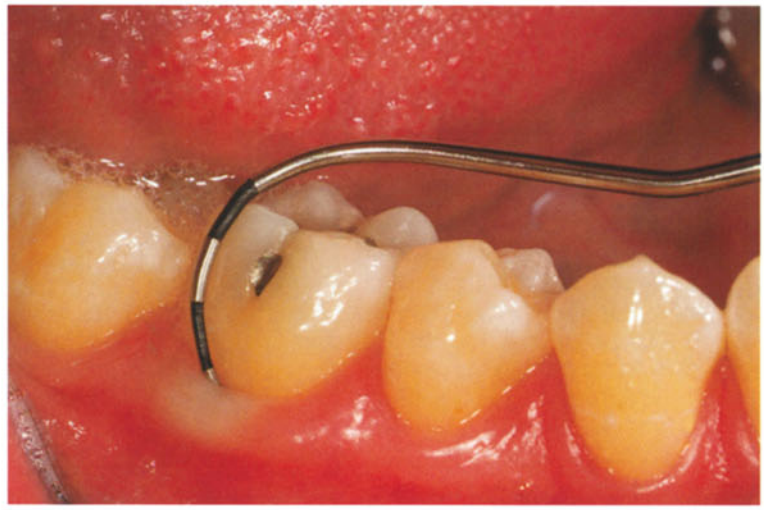
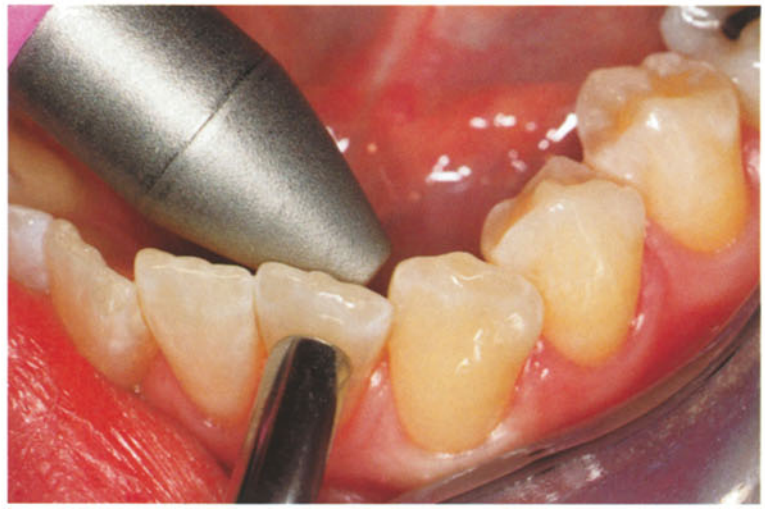


FIGURE 11

Determination of tooth mobility. The tooth is held between the two instruments



For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

hard instruments and pushed in a labiolingual (and apical) direction to determine mobility (Fig. 11).

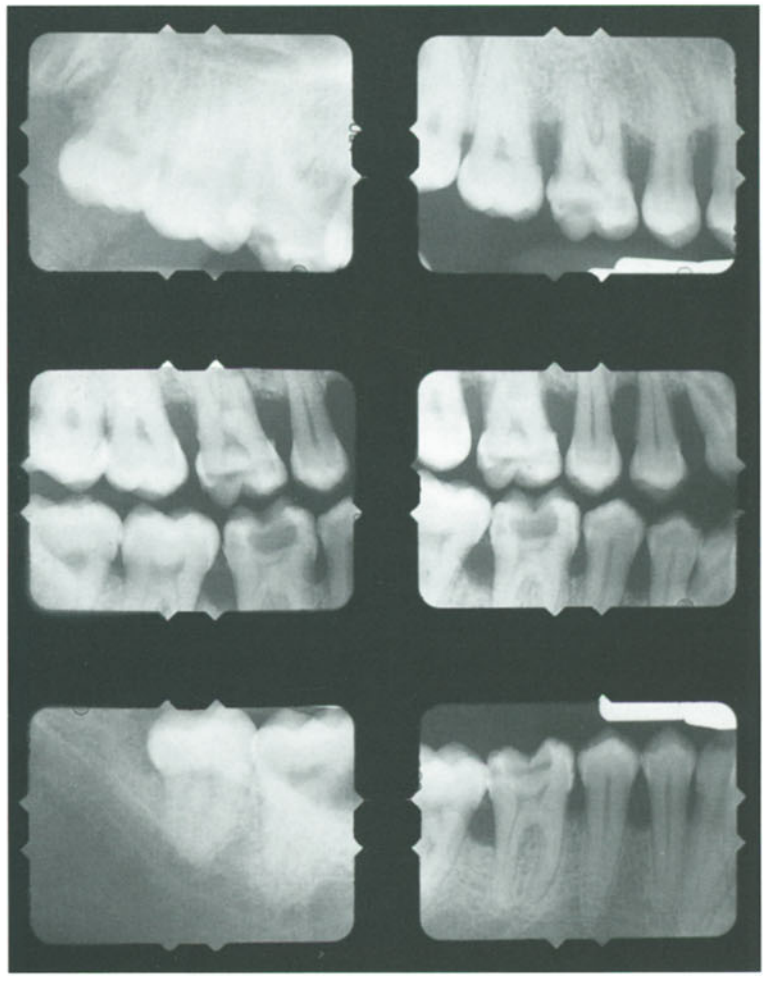
The radiographic examination (see "Radiographic Examination") provides a two-dimensional depiction of the alveolar crest levels around the teeth. However, should the practitioner desire detailed information regarding the osseous anatomy, e.g. prior to surgical treatment planning, then bone sounding may be performed. Bone sounding, which requires anaesthesia, consists of probing through the gingiva (or mucosa) until resistance from the bone is met. Bone sounding allows the health care provider to identify the shape and dimensions of osseous defects clinically (e.g. intrabony defects) without reflecting a flap. It helps corroborate and expand on information provided by the radiographic examination and is useful in planning the surgical treatment of periodontal defects.

Radiographic Examination

The radiographic evaluation is an integral part of the periodontal examination, as radiographic findings complement and corroborate clinical findings. The purpose of the periodontal radiographic examination is to determine the level of the alveolar bone crest and to identify changes in the hard (bone and teeth) and soft (PDL) periodontal tissues that cannot be visualized clinically. Radiographs also help identify calculus deposits, inadequate restorations (overhangs, etc.) and carious lesions on teeth.

FIGURE 12

Radiographic examination. Periapical and bitewing radiographs of right sextant. The patient, a 24-year-old, systemically healthy, non-smoker, female, exhibits moderate to severe bone loss. Calculus deposits are evident on the bitewing radiographs. The patient was diagnosed with generalized aggressive periodontitis



For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

The best determination of alveolar bone levels is made from a full-mouth set of periapical and bitewing radiographs of diagnostic quality (Fig. 12). Ideally, vertical bitewing radiographs should be taken. In periodontal health, the alveolar crest level is located 1–2 mm apical to the CEJ; the interdental crest is also parallel to a line connecting the CEJ of two adjacent teeth. In cases of severe periodontitis, only vertical bitewing radiographs will allow depiction of the interdental alveolar crest levels, which may be located several millimetres apical to the CEJ (Fig. 12). Depending on the presence of specific lesions, additional types of radiographic evaluation, e.g. occlusal radiographs or CT scans, may be necessary.

Diagnosis

At the end of the periodontal examination, the oral health care provider should reach a working (clinical) diagnosis based on the history, symptoms and signs present. He or she should ascertain the need for any laboratory tests or medical and dental specialist referral that may be necessary for definitive diagnosis and management of the case.

In 1999, the American Academy of Periodontology organized the International Workshop for a Classification of Periodontal Diseases and Conditions. At the conclusion of this workshop a new classification system of periodontal diseases was adopted (Table 1). In this new classification scheme, which is more comprehensive than previous ones, there are eight major categories, including gingival diseases, chronic periodontitis, aggressive periodontitis, periodontitis as a manifestation of systemic diseases, necrotizing periodontal diseases, abscesses of the periodontium, periodontitis associated with endodontic lesions, and developmental or acquired deformities and conditions. Of all the conditions listed, gingivitis associated with dental plaque and generalized chronic periodontitis are the two most common periodontal diseases.

For Personal Use Only
Library of School of Dentistry, TUMS

TABLE 1

Classification of periodontal diseases (from Armitage 1999)

I. Gingival Diseases

A. Dental plaque-induced gingival diseases

1. Gingivitis associated with dental plaque only
2. Gingival diseases modified by systemic factors
   - a. Associated with the endocrine system
     - 1) Puberty-associated gingivitis
     - 2) Menstrual cycle-associated gingivitis
     - 3) Pregnancy-associated
     - 4) Diabetes mellitus-associated gingivitis
   - b. Associated with blood dyscrasias
     - 1) Leukaemia-associated gingivitis
     - 2) Other
3. Gingival diseases modified by medications
   - a. Drug-influenced gingival diseases
     - 1) Drug-influenced gingival enlargements
     - 2) Drug-influenced gingivitis
       - a) Oral contraceptive-associated gingivitis
       - b) Other
4. Gingival diseases modified by malnutrition
   - a. Ascorbic acid-deficiency gingivitis
   - b. Other

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

TABLE 1

(continued)

B. Non-plaque-induced gingival lesions

1. Gingival diseases of specific bacterial origin
2. Gingival diseases of viral origin
   a. Herpesvirus infections
      1) Primary herpetic gingivostomatitis
      2) Recurrent oral herpes
      3) Varicella-zoster infections
   b. Other
3. Gingival diseases of fungal origin
   a. Candida species infections
   b. Linear gingival erythema
   c. Histoplasmosis
   d. Other
4. Gingival lesions of genetic origin
   a. Hereditary gingival fibromatosis
   b. Other
5. Gingival manifestations of systemic conditions
   a. Mucocutaneous disorders
      1) Lichen planus
      2) Pemphigoid
      3) Pemphigus vulgaris
      4) Erythema multiforme
      5) Lupus erythematosus
      6) Drug-induced
      7) Other
   b. Allergic reactions
      1) Dental restorative materials
      2) Reactions attributable to
         a) Toothpastes/dentifrices
         b) Mouthrinses/mouthwashes
         c) Chewing gum additives
         d) Foods and additives
      3) Other
6. Traumatic lesions (factitious, iatrogenic, accidental)
   a. Chemical injury
   b. Physical injury
   c. Thermal injury
7. Foreign body reactions
8. Not otherwise specified (NOS)

II. Chronic periodontitis

A. Localized

B. Generalized

III. Aggressive periodontitis

A. Localized

B. Generalized

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

TABLE 1

(continued)

IV. Periodontitis as a manifestation of systemic diseases

A. Associated with haematological disorders

1. Acquired neutropenia
2. Leukaemias
3. Other

B. Associated with genetic disorders

1. Familial and cyclic neutropenia
2. Down syndrome
3. Leukocyte adhesion deficiency syndromes
4. Papillon-Lefèvre syndrome
5. Chediak-Higashi syndrome
6. Histiocytosis syndromes
7. Glycogen storage disease
8. Infantile genetic agranulocytosis
9. Cohen syndrome
10. Ehlers-Danlos syndrome (types IV and VIII)
11. Hypophosphatasia
12. Other

C. Not otherwise specified (NOS)

V. Necrotizing periodontal diseases

A. Necrotizing ulcerative gingivitis (NUG)

B. Necrotizing ulcerative periodontitis (NUP)

VI. Abscesses of the periodontium

A. Gingival abscess

B. Periodontal abscess

C. Pericoronal abscess

VII. Periodontitis associated with endodontic lesions

A. Combined periodontic-endodontic lesions

VIII. Developmental or acquired deformities and conditions

A. Localized tooth-related factors that modify or predispose to plaque-induced gingival diseases/periodontitis

1. Tooth anatomical factors
2. Dental restorations/appliances
3. Root fractures
4. Cervical root resorption and cemental tears

B. Mucogingival deformities and conditions around teeth

1. Gingival/soft tissue recession
2. Lack of keratinized gingiva
3. Decreased vestibular depth
4. Aberrant frenum/muscle position
5. Gingival excess
   a. Pseudopocket
   b. Inconsistent gingival margin
   c. Excessive gingival display
   d. Gingival enlargement (see I.A.3. and I.B.4.)
6. Abnormal colour

C. Mucogingival deformities and conditions on edentulous ridges

D. Occlusal trauma

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Reading List

Armitage GC (1999) Development of a classification system for periodontal diseases and conditions. Ann Periodontol 4:1–6

Byers MR, Dong WK (1989) Comparison of trigeminal receptor location and structure in the periodontal ligament of different types of teeth from the rat, cat, and monkey. J Comp Neurol 279:117–127

Eger T, Muller HP, Helnecke A (1996) Ultrasonic determination of gingival thickness. Subject variation and influence of tooth type and clinical features. J Clin Periodontol 23:839–845

Fowler C, Garrett S, Crigger M, Egelberg J (1982) Histologic probe position in treated and untreated human periodontal tissues. J Clin Periodontol 9:373–385

Graves DT, Delima AJ, Assuma R, Amar S, Oates T, Cochran D (1998) Interleukin-1 and tumor necrosis factor antagonists inhibit the progression of inflammatory cell infiltration toward alveolar bone in experimental periodontitis. J Periodontol 69:1419–1425

Greenstein G, Polson A, Iker H, Meitner S (1981) Associations between crestal lamina dura and periodontal status. J Periodontol 52:362–366

Hassell TM (1993) Tissues and cells of the periodontium. Periodontol 2000 3:9–38

Lamster IB, Smith QT, Celenti RS, Singer RE, Grbic JT (1994) Development of a risk profile for periodontal disease: microbial and host response factors. J Periodontol 65:511–520

Madianos PN, Papapanou PN, Sandros J (1997) *Porphyromonas gingivalis* infection of oral epithelium inhibits neutrophil transepithelial migration. Infect Immun 65:3983–3990

Mariotti A (1993) The extracellular matrix of the periodontium: dynamic and interactive tissues. Periodontol 2000 3:39–63

Page RC, Schroeder HE (1976) Pathogenesis of inflammatory periodontal disease. A summary of current work. Lab Invest 34:235–249

Plecash JM, Bentley JP (1982) Crosslink analysis as an indicator of collagen turnover in periodontal ligament from functioning and non-functioning teeth in the dog. Arch Oral Biol 27:463–468

Saffar JL, Lasfargues JJ, Cherruau M (1997) Alveolar bone and the alveolar process: the socket that is never stable. Periodontol 2000 13:76–90

Schroeder HE, Munzel-Pedrazzoli S, Page R (1973) Correlated morphometric and biochemical analysis of gingival tissue in early chronic gingivitis in man. Arch Oral Biol 18:899–923

Schroeder HE, Listgarten MA (1997) The gingival tissues: the architecture of periodontal protection. Periodontol 2000 13:91–120

Selvig KA (1965) The fine structure of human cementum. Acta Odontol Scand 23:423–441

Smith RG, Çakici S, Newcombe RG (1996) Variations in the clinical sulcus depth of healthy human gingiva: a longitudinal study. J Periodontal Res 31:181–186

Tatakis DN (1993) Interleukin-1 and bone metabolism: a review. J Periodontol 64:416–431

Taubman MA, Kawai T (2001) Involvement of T-lymphocytes in periodontal disease and in direct and indirect induction of bone resorption. Crit Rev Oral Biol Med 12:125–135

Vacek JS, Gher ME, Assad DA, Richardson AC, Giambarresi LI (1994) The dimensions of the human dentogingival junction. Int J Periodontics Restorative Dent 14:154–165

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

PART II Local Diseases and Lesions

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

CHAPTER 2

Inflammatory Diseases

Dental Plaque-Induced Gingivitis

Definition
- Dental plaque-induced gingivitis (chronic gingivitis) is gingivitis caused by the accumulation of dental bacterial plaque.

Etiology
- Accumulation of dental plaque on the tooth close to the gingivae.

Gingival Involvement
- Common; affects over 90% of the population.

Other Sites of Involvements
- None

Main Clinical Features
- Painless
- May be bleeding from the gingival crevice, particularly when brushing
- Gingival margins slightly red and swollen (Figs. 13–16)
- Eventually mild gingival hyperplasia

Diagnosis
- Diagnosis is from history and clinical features.

Differential Diagnosis
- Desquamative gingivitis
- Trauma
- Drugs and plasma cell gingivostomatitis
- Infections, e.g., primary herpetic stomatitis, candidiasis
- Granulomas, e.g., Crohn's disease, orofacial granulomatosis or sarcoidosis
- Erythroplasia
- Kaposi's sarcoma

Treatment
- Plaque control
- Where necessary, calculus removal by scaling and polishing

FIGURE 13

Plaque-induced gingivitis; early

For Personal Use Only
Library of School of Dentistry, TUMS

G. Laskaris et al., *Periodontal Manifestations of Local and Systemic Diseases*
© Springer-Verlag Berlin Heidelberg 2003

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 14

Plaque-induced gingivitis; early

FIGURE 15

Plaque-induced gingivitis; advanced

FIGURE 16

Plaque-induced gingivitis; advanced

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Chronic Periodontitis

Definition

- Chronic periodontitis is inflammation of the gingivae and periodontal membrane (ligament).

Etiology

- Plaque accumulation and calcification to form calculus (tartar) above (supra-) and/or below (sub-gingival) the gum-line
- Anaerobes such as *Porphyromonas gingivalis*, *Bacteroides forsythus* and *Actinobacillus actinomycetemcomitans* are implicated
- The inflammatory reaction initiated by plaque is associated with progressive loss of periodontal ligament and alveolar bone and, eventually, tooth mobility and loss:
  - The gingiva detaches from the tooth.
  - The periodontal membrane and alveolar bone are damaged.
  - An abnormal gap (pocket) develops between the tooth and gingiva.
  - Debris and pus may be expressed from the pockets (pyorrhoea).
- The teeth may slowly loosen and, without treatment, may eventually be lost.
- Subjects are rendered particularly susceptible by genetic and/or environmental factors including:
  - Cigarette smoking
  - Interleukin-1 gene polymorphisms
  - Immune depression
  - Diabetes
  - Osteoporosis

Periodontal Involvement

- Exclusively

Other Sites of Involvements

- Studies are on-going to test possible effects of periodontal disease on systemic health and relationships with:
  - Atherosclerosis
  - Hypertension
  - Coronary heart disease
  - Cerebrovascular disease
  - Low birth weight babies
  - Effects on diabetic control

Main Clinical Features

- Chronic Periodontitis
  - Is typically seen in adults.
  - Is painless but may be associated with bleeding, halitosis (oral malodour) and a foul taste (Figs. 17–21).
  - May be a sequel of chronic gingivitis.

Diagnosis

- Diagnosis is from history and clinical features supplemented by:
  - Periodontal probing
  - Imaging

Differential Diagnosis

- Other causes of tooth mobility including
  - Trauma
  - Neoplasms
  - Immune defects

Treatment

- Improvement in oral hygiene
- Scaling and polishing
- Sometimes curettage
- Surgical removal of the pocket wall and removal of diseased tissue may be needed

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 17

Chronic periodontitis; early

FIGURE 18

Chronic periodontitis; early

FIGURE 19

Chronic periodontitis; moderately advanced

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 20

Chronic periodontitis; moderately advanced

FIGURE 21

Chronic periodontitis; advanced

For Personal Use Only
Library of School of Dentistry, TUMS

Aggressive Periodontitis

Definition

- This is where infection leads to more rapidly progressive periodontal disease.

Etiology

- Genetic variations in genes and gene products of cytokines and other inflammatory/destructive mediators appear to underlie early onset and severe types of periodontal disease,as well as specific genetic syndromes involving early periodontitis.
- The interleukin 1 (IL-1) genotype (IL-1A +4845 and IL-1B +3954) appears in about 30% of most Caucasian and African-American populations studied, and increases the risk of severe inflammatory periodontal disease by about 7 times.
- HLA serotypes may be associated with periodontal disease. The HLA-DRB1*1501-DQB1*0602 genotype has been found with increasing frequency in early onset periodontitis (EOP) and is associated with a strong T lymphocyte response against the *Porphyromonas gingivalis* antigen Ag53p141–161.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

- Several other mechanisms apart from T cell responses may be involved in genetic susceptibility to periodontal disease, but particularly:
  - Inflammatory mediators such as interleukins, prostaglandins, tumor necrosis factor alpha (TNF-α) and matrix metalloproteinases (MMPs)
  - Regulation of IgG subclass antibody responses
  - Polymorphonuclear neutrophilic leukocytes (PMNL) are essential for the phagocytosis and killing of a range of microorganisms, including periodontal pathogens
  - Periodontitis may develop despite good control of plaque and is then typically related to an inherited immune defect such as:
    - *Accelerated* (prepubertal) periodontitis is a rare condition seen in in children.
    - *Localized juvenile* periodontitis is characterised by localized periodontal destruction, classically in the permanent incisor and first molar regions in adolescents or young adults in the absence of poor oral hygiene or gross systemic disease.
    - *Juvenile periodontitis* (periodontosis) is seen especially in females, and may be associated with minor defects of neutrophil function and with microorganisms such as *Actinobacillus actinomycetemcomitans* and capnocytophaga.
    - *Papillon-Lefèvre* syndrome, Down syndrome, type VIII Ehlers-Danlos syndrome, and hypophophatasia.
  - An acquired immune defect such as in:
    - Poorly controlled diabetes mellitus
    - White cell dyscrasias including neutrophil defects and neutropenias
    - HIV/AIDS

Periodontal Involvement

- Exclusively

Other Sites of Involvements

- None unless there is recurrent infection in the immune defects

Main Clinical Features

- The features are those of marginal gingivitis and:
  - Rapid destruction of alveolar bone support
  - Deep pocket formation (Figs. 22–24)
  - Tooth mobility
  - Tooth migration
  - Halitosis

Diagnosis

- Diagnosis is from history and clinical features
- Diagnosis is supplemented by radiographical features

Differential Diagnosis

- A miscellany of immune disorders

Treatment

- Oral hygiene
- Scaling and root planing
- Periodontal surgery
- Antimicrobials

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 22

Aggressive periodontitis

FIGURE 23

Aggressive periodontitis

FIGURE 24

Aggressive periodontitis

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Mouthbreathing Gingivitis

Definition ▶ Hyperplastic gingivitis related to mouthbreathing. The various factors that can cause a mouthbreathing habit include asthma, allergies and enlarged glandular tissue.

Etiology ▶ Mouthbreathing, especially nocturnally. Mouthbreathing, increased lip separation and decreased upper lip coverage at rest are all associated with higher levels of plaque and gingival inflammation.

Gingival Involvement ▶ Common

Other Sites of Involvements ▶ None

Main Clinical Features ▶ Enlarged gingivae, especially labially in the anterior maxilla (Figs. 25, 26)

FIGURE 25

Mouth breathing gingivitis

FIGURE 26

Mouth breathing gingivitis

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Diagnosis
- Diagnosis is from history and clinical features

Differential Diagnosis
- Chronic gingivitis
- Hereditary gingival fibromatosis
- Drug-induced gingivitis
- Pregnancy gingivitis
- Sarcoidosis
- Crohn's disease
- Leukaemia
- Wegener's granulomatosis
- Amyloidosis
- Scurvy
- Mucopolysaccharidoses
- Mucolipidoses

Treatment
- Attention to nasal airway (ENT opinion)
- Occasionally surgery

Gingival Diseases of Specific Origin

Granulomatous Gingivitis

Definition
- Gingival swelling where lymphoedema and non-caseating granulomas may be seen.

Etiology
- It is unclear where in the spectrum of Crohn's disease/sarcoidosis/allergy/infection these lesions (and related conditions such as Melkersson-Rosenthal syndrome and granulomatous cheilitis) lie.
- Some patients develop:
  - Granulomatous reactions (orofacial granulomatosis) because of an adverse reaction to various food additives such as
    - Cinnamic aldehyde
    - Benzoates
    - Butylated hydroxyinosole
    - Dodecyl gallate (in margarine)
    - Menthol (in peppermint oil)
  - Crohn's disease after some time

Gingival Involvement
- Rare; purple granulomatous enlargements may appear on the gingiva.

Other Sites of Involvements
- Lips or face

Main Clinical Features
- Onset is usually in young adult life
- Earliest manifestation is sudden diffuse or occasionally nodular non-tender swellings of the lip (cheilitis granulomatosa) or face
- Granulomatous gingiva enlargements (Figs. 27, 28)
- Lingual, palatal, and buccal involvement also may occur
- Mucosal lesions include thickening and folding to produce:
  - A "cobblestone" type of appearance
  - Mucosal tags.
- Linear ulcers in the vestibule, often with granulomatous masses flanking them, classically involve the buccal sulcus

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 27

Granulomatous gingivitis

FIGURE 28

Granulomatous gingivitis

Diagnosis

- Diagnosis is from history and clinical features supplemented by
- Histopathology
- Investigation to exclude Crohn's disease (endoscopy, imaging and histopathology of the gastrointestinal tract)
- Investigations to exclude sarcoidosis (chest imaging, serum angiotensin converting enzyme, and gallium scan)
- Patch tests to exclude reactions to foodstuffs or additives.

Differential Diagnosis

- Chronic gingivitis
- Desquamative gingivitis
- Trauma
- Drugs and plasma cell gingivostomatitis
- Infections e.g. primary herpetic stomatitis, candidiasis
- Erythroplasia
- Kaposi's sarcoma
- Hyperplastic gingivitis
- Hereditary gingival fibromatosis

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

- Pregnancy
- Leukaemia
- Wegener's granulomatosis
- Amyloidosis
- Scurvy
- Mucopolysaccharidoses
- Mucolipidoses

Treatment

- Possible provoking dietary components should avoided
- Drug management has included:
  - Clofazimine
  - Nonsteroidal antiinflammatory agents
  - Antibiotics
  - Antimalarials
  - Mast cell stabilizers
  - Intralesional corticosteroid injections

"Geographic" Gingivitis

Definition

- Erythema migrans (geographic stomatitis, benign migratory glossitis, stomatitis areata migrans)
  - Typically affects the tongue
  - Is a benign inflammatory condition unrelated to cutaneous erythema migrans.
  - Is common, affecting about 1–2% of the population

Etiology

- The aetiology is unknown
- There is a genetic background
  - A positive family history may be obtainable.
  - HLA findings have been equivocal, with reports of associations with B15 and DR7.
  - Many patients have a fissured tongue (plicated tongue)
- Some patients
  - Have atopic allergies such as hay fever
  - Relate the lesions to a particular food e.g. cheese
  - Relate the lesions to stress.
  - Have psoriasis, mainly the severe pustular variety

Gingival Involvement

- Rare

Other Sites of Involvements

- The tongue is usually, but not invariably, affected

Main Clinical Features

- Rounded, sometimes scalloped, map-like reddish areas with a white margin
- The patterns change from day to day and even over a few hours (Figs. 29–31)
- It may be asymptomatic or sore

Diagnosis

- Diagnosis is from history and clinical features supplemented by
- Blood and urine examination to exclude anaemia and diabetes.

Differential Diagnosis

- Lichen planus
- Candidosis
- Deficiency glossitis
- Generalized pustular psoriasis

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 29

Geographic gingivitis

FIGURE 30

Geographic tongue

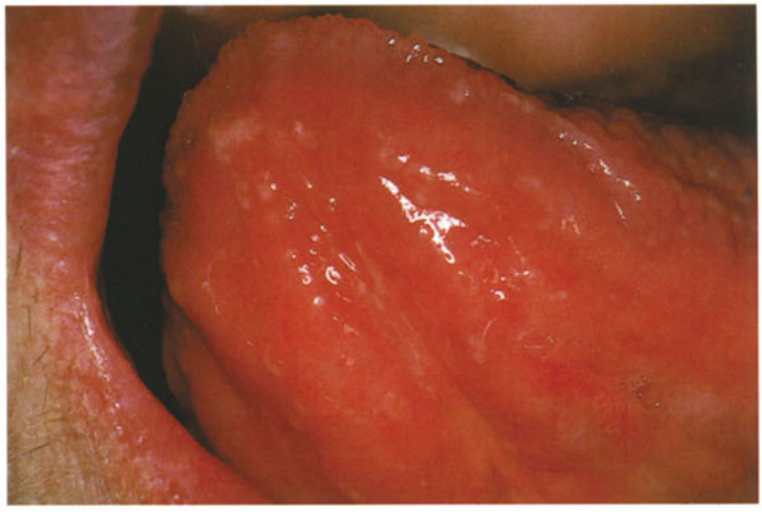


FIGURE 31

Geographic stomatitis

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

- Reiter's syndrome
- Syphilis
- Ulcerative colitis
- Acrodermatitis continua of Hallopeau
- Larva migrans

Treatment

- Reassurance.
- Topical corticosteroids may be employed for severely symptomatic patients.

Reading List

Clerehugh V, Tugnait A. Periodontal diseases in children and adolescents: I. Aetiology and diagnosis. Dent Update. 2001;28(5):222–30, 232

Coventry J, Griffiths G, Scully C, Tonetti M. ABC of Oral Health 3. Periodontal disease. British Medical Journal 2000; 321: 36–39

Espelid M, Bang G, Johannessen AC, Leira JI, Christensen O.Geographic stomatitis: report of 6 cases. J Oral Pathol Med. 1991 Oct;20(9):425–428.

Haffajee AD, Socransky SS. Relationship of cigarette smoking to attachment level profiles. J Clin Periodontol. 2001;28(4):283–295.

Hodge P, Michalowicz B. Genetic predisposition to periodontitis in children and young adults. Periodontol 2000. 2001;26:113–134.

Hoffmann T, Bruhn G, Richter S, Netuschil L, Brecx M. Clinical controlled study on plaque and gingivitis reduction under long-term use of low-dose chlorhexidine solutions in a population exhibiting good oral hygiene. Clin Oral Investig. 2001;5(2):89–95.

Holmstrup P. Non-plaque-induced gingival lesions. Ann Periodontol. 1999 Dec;4(1):20–31.

Jacobson L. Mouthbreathing and gingivitis. 1. Gingival conditions in children with epipharyngeal adenoids. J Periodontal Res. 1973;8(5):269–277.

Kinane DF, Lappin DF. Clinical, pathological and immunological aspects of periodontal disease. Acta Odontol Scand. 2001;59(3):154–160.

Kinane DF, Marshall GJ. Periodontal manifestations of systemic disease. Aust Dent J. 2001;46(1):2–12.

Kinane DF, Podmore M, Ebersole J. Etiopathogenesis of periodontitis in children and adolescents. Periodontol 2000. 2001;26:54–91.

Klouda PT, Porter SR, Scully C, Corbin SA, Bradley BA, Smith R and Davies RM. Association between HLA-A9 and rapidly progressive periodontitis. Tissue Antigens 1986; 28: 146–149.

Mariotti A. Dental plaque-induced gingival diseases. Ann Periodontol. 1999 Dec;4(1):7–19.

Meisel P, Carlsson LE, Sawaf H, Fanghaenel J, Greinacher A, Kocher T. Polymorphisms of Fc gamma-receptors RIIa, RIIIa, and RIIIb in patients with adult periodontal diseases. Genes Immun. 2001;2(5):258–262.

Meyle J, Gonzales JR. Influences of systemic diseases on periodontitis in children and adolescents. Periodontol 2000. 2001;26:92–112.

Mignogna MD, Fedele S, Lo Russo L, Lo Muzio L. Orofacial granulomatosis with gingival onset. J Clin Periodontol. 2001 Jul;28(7):692–696.

Mooney J, Hodge PJ, Kinane DF. Humoral immune response in early-onset periodontitis: influence of smoking. J Periodontal Res. 2001;36(4):227–232.

Morita M, Wang HL. Association between oral malodor and adult periodontitis: a review. J Clin Periodontol. 2001;28(9):813–819.

Scully C, Cochran KM, Russell RI et al. Crohn's disease of the mouth: an indication of intestinal involvement. Gut 1982; 23: 198–201.

Seifert R, Wenzel-Seifert K. Defective Gi-protein coupling in two formyl peptide receptor mutants associated with localized juvenile periodontitis. J Biol Chem. 2001 Sep 14

Shapira L, Stabholz A, Rieckmann P, Kruse N. Genetic polymorphism of the tumor necrosis factor (TNF)-alpha promoter region in families with localized early-onset periodontitis. J Periodontal Res. 2001 Jun;36(3):183–186

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Stokes N, Della Mattia D. A student research review of the mouthbreathing habit: discussing measurement methods, manifestations and treatment of the mouthbreathing habit. Probe. 1996;30(6):212–214.

Thomas DW, Baboolal K, Subramanian N, Newcombe RG. Cyclosporin A-induced gingival overgrowth is unrelated to allograft function in renal transplant recipients. J Clin Periodontol. 2001;28(7):706–709.

Wagaiyu EG, Ashley FP. Mouthbreathing, lip seal and upper lip coverage and their relationship with gingival inflammation in 11–14 year-old schoolchildren. J Clin Periodontol. 1991;18(9):698–702.

Wiesenfeld D, Ferguson MM, Mitchell DN, Macdonald DG, Scully C, Cochran K, Russell RI. Orofacial granulomatosis; clinical and pathological analysis. Quart J Med 1985; 213: 101–113.

Yamazaki K, Tabeta K, Nakajima T, Ohsawa Y, Ueki K, Itoh H, Yoshie H. Interleukin-10 gene promoter polymorphism in Japanese patients with adult and early-onset periodontitis. J Clin Periodontol. 2001 Sep;28(9):828–832.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

CHAPTER 3

Developmental Disorders

Torus Mandibularis

Definition
- A bilateral asymptomatic benign bony lump or lumps, lingual to the mandibular premolars

Etiology
- Developmental
- Seen especially in Asian and Black races
- May be associated with parafunction
- Seen mainly in males, and rarely in Turner's syndrome (4S,X)

Gingival Involvement
- Common

Other Sites of Involvement
- Often associated with torus palatinus; a slow-growing, asymptomatic, benign bony lump in midline of palate

Main Clinical Features
- Bony lumps, which grow slowly, appear in adolescence and usually cease growth in late teens (Fig. 32).
- Bony hard with normal overlying mucosa, asymptomatic, benign and of no consequence.

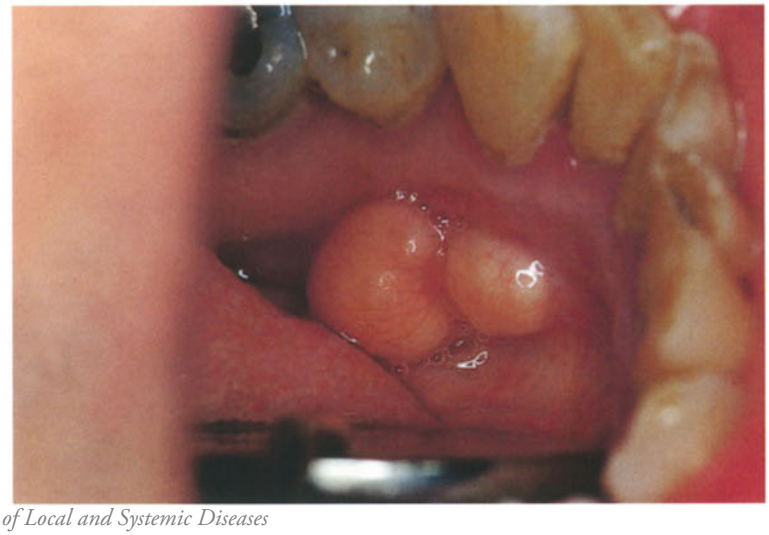
Diagnosis
- Diagnosis is from history and clinical features, supplemented by:
  - Radiographic examination.

Differential Diagnosis
- Torus mandibularis – exclude unerupted teeth
- Torus palatinus – exclude neoplasms

Treatment
- Excise or reduce only if causing severe difficulties with dentures.

FIGURE 32

Torus mandibularis



For Personal Use Only
Library of School of Dentistry, TUMS

G. Laskaris et al., *Periodontal Manifestations of Local and Systemic Diseases*
© Springer-Verlag Berlin Heidelberg 2003

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Multiple Exostoses of the Maxillary Alveolar Process

Definition
- Multiple exostoses of the maxillary alveolar process

Etiology
- Genetic

Gingival Involvement
- Rare

Other Sites of Involvement
- Occasionally arise beneath a bridge pontic (sub-pontic osseous hyperplasia)

Main Clinical Features
- Exostoses typically buccal to maxillary premolars and molars (Figs. 33, 34).
- Appear in adolescence.
- Grow slowly and usually cease growth in late teens.
- Most are asymptomatic, benign and of no consequence.

Diagnosis
- Diagnosis is from history and clinical features, supplemented by:
  - Radiography.

Differential Diagnosis
- Osteoma

Treatment
- Leave alone

Fibrous Tuberosity

Definition
- A fibrous tuberosity

Etiology
- Idiopathic

Gingival Involvement
- Uncommon

Other Sites of Involvement
- None

Main Clinical Features
- Enlarged tuberosities, mainly palatally (Figs. 35–37)

Diagnosis
- Diagnosis is from history and clinical features.

Differential Diagnosis
- Hereditary gingival fibromatosis
- Drugs
- Neoplasms
- Pregnancy gingivitis
- Sarcoidosis
- Crohn's disease
- Leukaemia

Treatment
- Occasionally surgery

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 33

Multiple exostoses

FIGURE 34

Multiple exostoses

FIGURE 35

Fibrous tuberosity

For Personal Use Only
Library of School of Dentistry, TUMS

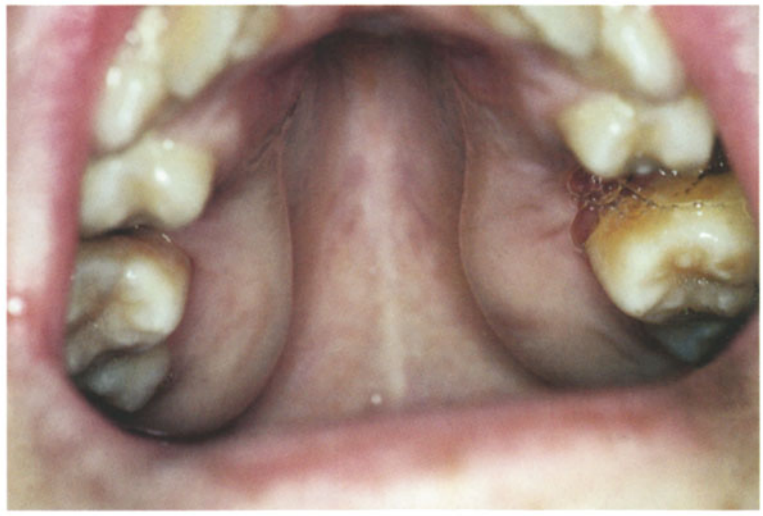
WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 36

Fibrous tuberosity

FIGURE 37

Fibrous tuberosity



For Personal Use Only
Library of School of Dentistry, TUMS

Reading List

Alvesalo L, Mayhall JT, Varrela J (1996) Torus mandibularis in 45,X females (Turner syndrome). Am J Phys Anthropol 101:145–149

Antoniades DZ, Belazi M, Papanayiotou P (1998) Concurrence of torus palatinus with palatal and buccal exostoses: case report and review of the literature. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 85:552–557

Jainkittivong A, Langlais RP (2000) Buccal and palatal exostoses: prevalence and concurrence with tori. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 90:48–53

Seah YH (1995) Torus palatinus and torus mandibularis: a review of the literature. Aust Dent J 40:318–321

Sirirungrojying S, Kerdpon D (1999) Relationship between oral tori and temporomandibular disorders. Int Dent J 49:101–104

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

CHAPTER 4

Drug-Influenced Gingival Lesions and Diseases

Gingival Enlargement Due to Phenytoin

Definition

- Gingival swelling in response to the use of phenytoin

Etiology

- Exact mechanism unknown
- Positive correlation between the severity of gingival overgrowth and:
  - Inflammation
  - Plaque score
  - Calculus accumulation
  - Pocket depths

Gingival Involvement

- Common

Other Sites of Involvement

- This may be associated with hypertrichosis.

Main Clinical Features

- Gingival enlargement (Figs. 38, 39). Characteristically is:
  - Firm, painless, pale, tough with coarse stippling and relatively little tendency to bleed. It is seen:
    - On the papillae mainly.
    - On buccal and labial gingiva more than palatal and lingual gingiva.
    - Not in edentulous sites.
    - First 2–3 months after treatment is started.
    - Peaking at around 1 year of drug use, it is aggravated by poor oral hygiene.

FIGURE 38

Drug-induced gingival swelling due to phenytoin

For Personal Use Only
Library of School of Dentistry, TUMS

G. Laskaris et al., *Periodontal Manifestations of Local and Systemic Diseases*
© Springer-Verlag Berlin Heidelberg 2003

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 39

Drug-induced gingival swelling due to phenytoin

Diagnosis
- Diagnosis is from history and clinical features.

Differential Diagnosis
- Chronic gingivitis
- Hyperplastic gingivitis
- Hereditary gingival fibromatosis
- Drugs
- Pregnancy gingivitis
- Sarcoidosis
- Crohn's disease
- Leukaemia
- Wegener's granulomatosis
- Amyloidosis
- Scurvy
- Mucopolysaccharidoses
- Mucolipidoses

Treatment
- In consultation with physician, change or reduce dose of the drug.
- Improve oral hygiene.
- Gingival surgery may be needed.

Gingival Enlargement Due to Calcium Channel Blockers

Definition
- Gingival swelling in response to use of calcium channel blockers

Etiology
- Calcium channel blockers, mainly the dihydropyridines such as nifedipine used as antihypertensives, can produce gingival hyperplasia similar to that induced by phenytoin.
- Exact mechanism is unknown.

Gingival Involvement
- Typically affects the papillae, which become red and puffy and tend to bleed.

Other Sites of Involvement
- Hypertrichosis

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 40

Drug-induced gingival swelling due to nifedipine

FIGURE 41

Drug-induced gingival swelling due to nifedipine

FIGURE 42

Drug-induced gingival swelling due to nifedipine

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Main Clinical Features
- Changes often develop slowly – over weeks rather than days, usually painless and aggravated by poor oral hygiene.
- Starts interdentally, especially labially.
- Papillae are firm, pale and enlarge to form false vertical clefts (Figs. 40–42).

Diagnosis
- Diagnosis is from history and clinical features.

Differential Diagnosis
- Chronic gingivitis
- Hyperplastic gingivitis
- Hereditary gingival fibromatosis
- Drugs
- Pregnancy gingivitis
- Sarcoidosis
- Crohn's disease
- Leukaemia
- Wegener's granulomatosis
- Amyloidosis
- Scurvy
- Mucopolysaccharidoses
- Mucolipidoses

Treatment
- In consultation with a physician, change or reduce dose of the drug.
- Improve oral hygiene.
- Gingival surgery may be needed.

Gingival Enlargement Due to Ciclosporin

Definition
- Gingival swelling in response to use of ciclosporin

Etiology
- Exact mechanism unknown
- Usually aggravated by poor oral hygiene

Gingival Involvement
- *Ciclosporin* (cyclosporine), an immunosuppressive drug, can cause gingival swelling, but only a third of patients may be affected, more commonly children.

Other Sites of Involvement
- Hypertrichosis

Main Clinical Features
- Ciclosporin hyperplasia closely resembles that induced by phenytoin.
- It is seen mainly anteriorly and labially.
- It is exacerbated by poor oral hygiene and concurrent administration of nifedipine.
- It appears not to be proportional to the drug dose used.
- Papillae are firm, pale and enlarge to form false vertical clefts (Figs. 43–47).
- Changes develop slowly – over weeks rather than days – and are usually painless.

Diagnosis
- Diagnosis is from history and clinical features.

Differential Diagnosis
- Chronic gingivitis
- Hyperplastic gingivitis
- Hereditary gingival fibromatosis
- Drugs

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 43

Drug-induced gingival swelling due to ciclosporin

FIGURE 44

Drug-induced gingival swelling due to ciclosporin

FIGURE 45

Drug-induced gingival swelling due to ciclosporin

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 46

Drug-induced gingival swelling due to ciclosporin

FIGURE 47

Drug-induced gingival swelling due to ciclosporin

- Pregnancy gingivitis
- Sarcoidosis
- Crohn's disease
- Leukaemia
- Wegener's granulomatosis
- Amyloidosis
- Scurvy
- Mucopolysaccharidoses
- Mucolipidoses

Treatment

- In consultation with a physician, change or reduce dose of the drug.
- Improve oral hygiene.
- Gingival surgery may be needed.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Plasma Cell Gingivitis (Idiopathic Circumorificial Plasmacytosis)

Definition

▶ Plasma-cell gingivitis (PCG) is a rare, non-symptomatic erythematous and oedematous reaction to various substances, found in the attached gingiva.

Etiology

▶ Chewing gum additives such as:
- Cinnamonaldehyde
- Cinnamon oil
- Dichlorophene
- Hexylresorcinol
- Mint

Gingival Involvement

▶ Uncommon

Other Sites of Involvement

▶ Rarely, other oral or pharyngeal sites. Lesions with similar clinical appearance are also found in the genital mucosa of patients with a PCG.

FIGURE 48

Plasma-cell gingivitis

FIGURE 49

Plasma-cell gingivitis

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 50

Plasma-cell gingivitis

FIGURE 51

Plasma-cell gingivitis

For Personal Use Only
Library of School of Dentistry, TUMS

Main Clinical Features

- Clinically, PCG is a sharply demarcated red area within the attached gingiva (Figs. 48–51).

Diagnosis

- Diagnosis is from history and clinical features, supplemented by:
  - Histopathology; abundant plasma cells are found in the connective tissue beneath a thin epithelium.

Differential Diagnosis

- Chronic gingivitis
- Hyperplastic gingivitis
- Hereditary gingival fibromatosis
- Drugs
- Pregnancy gingivitis
- Sarcoidosis
- Crohn's disease
- Leukaemia
- Wegener's granulomatosis
- Amyloidosis

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

- Scurvy
- Mucopolysaccharidoses
- Mucolipidoses

Treatment
- Corticosteroids

Allergic Gingivitis

Definition
- An uncommon erythematous and oedematous reaction to various substances

Etiology
- Constituents of perfumes, sprays, creams, mouthwashes, dentifrices and breath fresheners:
  - Furocoumarin
  - Bergamot oil
  - Eugenol
  - Others
- A wide range of other allergens:
  - Food and additives
  - Natural products
  - Chewing gum additives
  - Dental materials
  - Rubber products, such as dental dam or impression materials
  - Silicone-based impression materials
  - Eugenol and other essential oils
  - Mercury
  - Nickel
  - Beryllium
  - Acrylic resin components
  - Epoxy resin components
  - Drugs

Gingival Involvement
- Uncommon

Other Sites of Involvement
- Various

Main Clinical Features
- Swollen red gingivae (Figs. 52–54)

Diagnosis
- Diagnosis is from history and clinical features. A careful history is required, concentrating on the use of chewing gum or oral medications.
- Patch tests on the skin are usually negative.

Differential Diagnosis
- Chronic gingivitis
- Desquamative gingivitis
- Trauma
- Drugs
- Infections, e.g. primary herpetic stomatitis, candidiasis
- Granulomas, e.g. Crohn's disease, orofacial granulomatosis or sarcoidosis
- Erythroplasia
- Kaposi's sarcoma

Treatment
- Withdraw the offending agent.
- Corticosteroids occasionally.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 52

Drug-induced lesion due to hydroxyurea

FIGURE 53

Drug-induced lesion due to sanguinaria

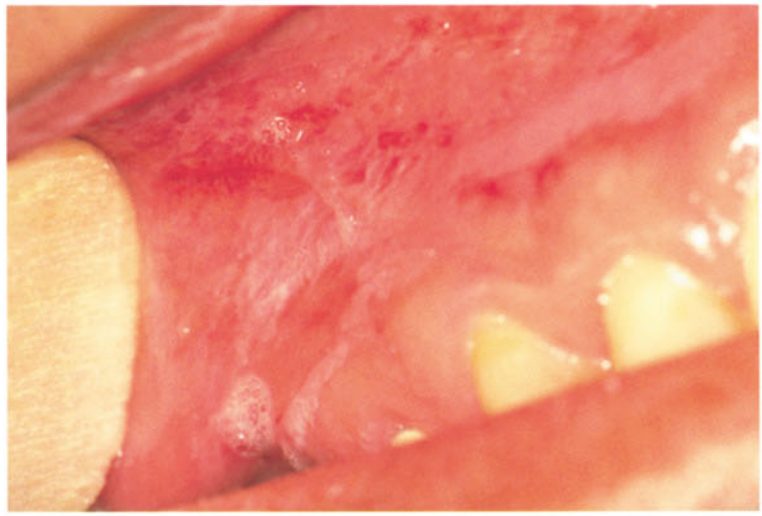


FIGURE 54

Drug-induced lesion due to sanguinaria

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Heavy Metal Deposition and Other Causes of Hyperpigmentation

Definition

- Hyperpigmentation induced by habits, drugs or other chemicals

Etiology

- Betel (Areca) nut.
- Smoking.
- Drugs:
  - Phenytoin.
  - Antimalarials.
  - ACTH.
  - Clofazimine.
  - Contraceptive pill.
  - Zidovudine.
  - Ketoconazole.
  - Busulphan.
  - Minocycline.
  - Phenothiazines.
- Heavy metals, rarely:
  - Amalgam.
  - Arsenic.
  - Bismuth.
  - Copper.
  - Gold.
  - Lead.
  - Mercury.
  - Platinum.
  - Silver.
  - Zinc.
- Staining of gingivae adjacent to crowns or other restorative dental materials appears due mainly to silver, sulphur and selenium.
- Rare cases of toxicity are related to use of platinum in cancer therapy.
- Many heavy metals formerly implicated are not used now therapeutically.
- Cases of heavy metal toxicity related to industrial exposure are still occasionally seen.
- Metal sulphides form due to the activity of plaque microorganisms and produce pigmentation.

Gingival Involvement

- Rare

Other Sites of Involvement

- Any oral location
- Occasionally skin, nails or other sites

Main Clinical Features

- Hyperpigmentation (sometimes called Burton's line): brown or blue or black line, mainly at gingival margin and particularly in inflamed sites (Figs. 55–57).
- Sometimes heavy metal poisoning may result in tooth loss.
- Rarely, cobalt may induce orofacial granulomatosis.
- Patchy mucosal hyperpigmentation with, in smokers or betel users, tooth staining.

Diagnosis

- Diagnosis is from history and clinical features, supplemented by:
  - Histopathology in some instances.
- Imaging may be helpful whenever hyperpigmentation may be caused by amalgam, graphite or a foreign body.
- Photographs may be useful for future comparison.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 55

Drug-induced gingival lesions due to bismuth

FIGURE 56

Drug-induced gingival lesions due to lead

FIGURE 57

Betel nut staining

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Differential Diagnosis

- Racial pigmentation
- Localized irritation, e.g. smoking
- Amalgam tattoo
- Naevus
- Melanotic macule
- Malignant melanoma
- Kaposi's sarcoma
- Drugs, e.g. antimalarials
- Addison's disease

Treatment

- Remove the cause.
- Treatment of any heavy metal poisoning.
- Reassurance.

Reading List

Bjorkner B, Niklasson B (1997) Contact allergy to the UV absorber Tinuvin P in a dental restorative material. Am J Contact Dermat 8:6–7

Ciancio SG (1996) Medications as risk factors for periodontal disease. J Periodontol 67[10 Suppl]:1055–1059

Das SJ, Parkar MH, Olsen I (2001) Upregulation of keratinocyte growth factor in cyclosporin A-induced gingival overgrowth. J Periodontol 72:745–752

Diamond JP, Chandna A, Williams C, Easty DL, Scully C, Eveson J, Richards A (1991) Tranexamic acid-associated ligneous conjunctivitis with gingival and peritoneal lesions. Br J Ophthalmol 75:753–754

Frisken K (1993) The diagnosis and management of oral-facial allergic responses in clinical practice. J N Z Soc Periodontol 75:26–28

Gargiulo AV, Ladone JA, Ladone PA, Toto PD (1995) Case report: plasma cell gingivitis A. CDS Rev 88:22–23

Haberman AL, Pratt M, Storrs FJ (1993) Contact dermatitis from beryllium in dental alloys. Contact Dermatitis 28:157–162

Hedin CA, Karpe B, Larsson A (1994) Plasma-cell gingivitis in children and adults. A clinical and histological description. Swed Dent J 18:117–124

Kanerva L, Alanko K, Estlander T (1999) Allergic contact gingivostomatitis from a temporary crown made of methacrylates and epoxy diacrylates. Allergy 54:1316–1321

Kerr DA, McClatchey KD, Regezi JA (1971) Allergic gingivostomatitis (due to gum chewing). J Periodontol 42:709–712

Lewis FM, Shah M, Gawkrodger DJ (1995) Contact sensitivity to food additives can cause oral and perioral symptoms. Contact Derm 33:429–430

Marshall RI, Bartold PM (1998) Medication induced gingival overgrowth. Oral Dis 4:130–151

Matsson L, Moller C (1990) Gingival inflammatory reactions in children with rhinoconjunctivitis due to birch pollinosis. Scand J Dent Res 98:504–509

Maurice PD, Hopper C, Punnia-Moorthy A, Rycroft RJ (1988) Allergic contact stomatitis and cheilitis from iodoform used in a dental dressing. Contact Dermatitis 18:114–116

Moore MM, Burke FJ, Felix DH (2000) Allergy to a common component of resin-bonding systems: a case report. Dent Update 27:432–434

Morisaki I, Dol S, Ueda K, Amano A, Hayashi M, Mihara J (2001) Amlodipine-induced gingival overgrowth: periodontal responses to stopping and restarting the drug. Spec Care Dentist 21:60–62

Morton RS, Dongari-Bagtzoglou AI (1999) Regulation of gingival fibroblast interleukin-6 secretion by cyclosporine A. J Periodontol 70:1464–1471

Palmer RM, Eveson JW (1981) Plasma-cell gingivitis. Oral Surg Oral Med Oral Pathol 51:187–189

Pemberton M, Yeoman CM, Clark A, Craig GT, Franklin CD, Gawkrodger DJ (1993) Allergy to octyl gallate causing stomatitis. Br Dent J 175:106–108

Reed BE, Barrett AP, Katelaris C, Bilous M (1993) Orofacial sensitivity reactions and the role of dietary components. Case reports. Aust Dent J 38:287–291

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Regezi JA, Taylor CG, Spinelli FR Jr, Lucas RN (1972) Allergic gingivostomatitis with laryngeal manifestations: report of a case. J Oral Surg 30:373–377

Saito K, Mori S, Tanda N, Sakamoto S (2000) Immunolocalizaiton of c-Myc and bcl-2 proto-oncogene products in gingival hyperplasia induced by nifedipine and phenytoin. J Periodontol 71:44–49

Somacarrera ML, Lucas M, Scully C, Barrios C (1997) Effectiveness of periodontal treatments on cyclosporine-induced gingival overgrowth in transplant patients. Br Den J 183:89–94

Yamada H, Nishimura F, Naruishi K, Chou HH, Takashiba S, Albright GM, Nares S, Iacopino AM, Murayama Y (2000) Phenytoin and cyclosporin A suppress the expression of MMP-1, TIMP-1, and cathepsin L, but not cathepsin B in cultured gingival fibroblasts. J Periodontol 71:955–960

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

CHAPTER 5

Abscesses and Fistulas

Gingival Abscess

Definition ▶ An abscess in the gingiva

Etiology ▶ Usually a foreign body such as dental floss or particle of food or dental restorative material

Gingival Involvement ▶ Rare

Main Clinical Features ▶ Pain, swelling and erythema (Fig. 58)

Diagnosis ▶ Diagnosis is from history and clinical features.

Differential Diagnosis ▶ Periodontal abscess
▶ Periapical abscess

Treatment ▶ Drainage

FIGURE 58

Gingival abscess

For Personal Use Only
Library of School of Dentistry, TUMS

G. Laskaris et al., *Periodontal Manifestations of Local and Systemic Diseases*
© Springer-Verlag Berlin Heidelberg 2003

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 59

Periodontal abscess

FIGURE 60

Periodontal abscess

FIGURE 61

Periodontal abscess

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Periodontal Abscess

Definition
- An abscess arising in a periodontal pocket

Etiology
- Seen almost exclusively in patients with chronic periodontitis.
- Debris and pus cannot escape easily from the periodontal pocket and therefore an abscess results.
- May follow impaction of a foreign body.
- Rarely related to a lateral root canal on a non-vital tooth.

Gingival Involvement
- Uncommon

Other Sites of Involvement
- None

Main Clinical Features
- Lateral periodontal abscesses usually present with:
  - Pain
  - Swelling
- Discharge either through the pocket or buccally, but more coronally than a periapical abscess (Figs. 59–61)

Diagnosis
- Diagnosis is from history and clinical features, supplemented by:
  - Imaging.

Differential Diagnosis
- Gingival abscess
- Periapical abscess
- Neoplasm

Treatment
- Establish drainage, usually by curettage

Pericoronal Abscess

Definition
- An abscess in the pericoronal region of an impacted tooth, typically a mandibular third molar

Etiology
- Trauma
- Plaque accumulation
- Necrotizing gingivitis
- A foreign body such as food
- Impaired immunity such as in leukaemia or infectious mononucleosis

Gingival Involvement
- Rare

Other Sites of Involvement
- None

Main Clinical Features
- Pain, swelling (Fig. 62), erythema and trismus

Diagnosis
- Diagnosis is from history and clinical features.

Differential Diagnosis
- Periodontal abscess
- Periapical abscess

Treatment
- Drainage
- Antimicrobials
- Surgical opinion on the partially erupted tooth

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 62

Pericoronal abscess

Tooth Abscess

Definition
- An abscess around the apex of a non-vital tooth

Etiology
- Untreated, dental caries can progress through dentine to the pulp which becomes inflamed (pulpitis) producing severe persistent pain (toothache). The pulp undergoes necrosis when inflammation can spread around the tooth apex (periapical periodontitis), forming an abscess (periapical, or dental, abscess).
- Occasionally a dental abscess arises from:
  - A necrotic pulp in a tooth damaged by trauma.
  - A foreign body.
  - Unusual oral infections such as actinomycosis, nocardiosis or botryomycosis.

Gingival Involvement
- Common

Other Sites of Involvement
- None

Main Clinical Features
- Most abscesses present with:
  - Pain, and swelling (Figs. 63, 64).
  - Sometimes fever and/or cervical lymph node enlargement.
- Most abscesses discharge in the mouth on the buccal gingiva but occasionally they discharge palatally or lingually or on the chin or submental region or elsewhere (Figs. 65–67).

Diagnosis
- Diagnosis is from history and clinical features, supplemented by:
  - Vitality testing.
  - Radiographic imaging.

Differential Diagnosis
- Periodontal abscess
- Rarely, metastasis

Treatment
- Dental abscesses are drained by tooth extraction, incision and drainage, or through the root canal (endodontics). Antimicrobials may be indicated.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 63

Periapical abscess

FIGURE 64

Periapical abscess

FIGURE 65

Fistula

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 66

Fistula

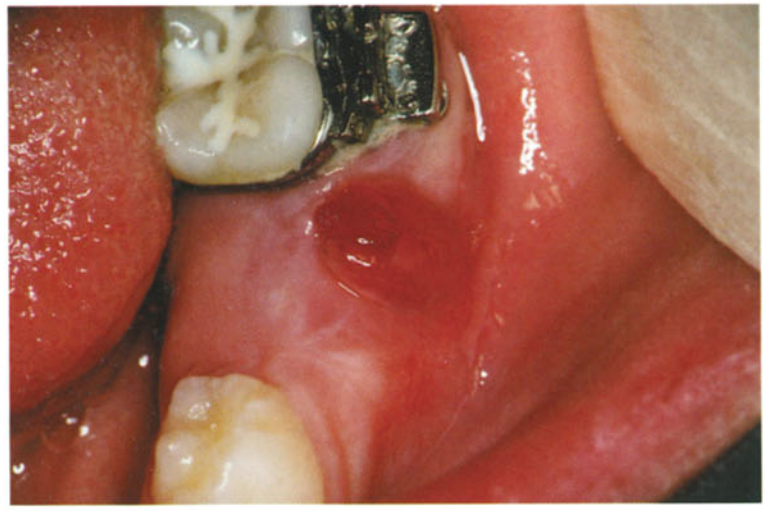
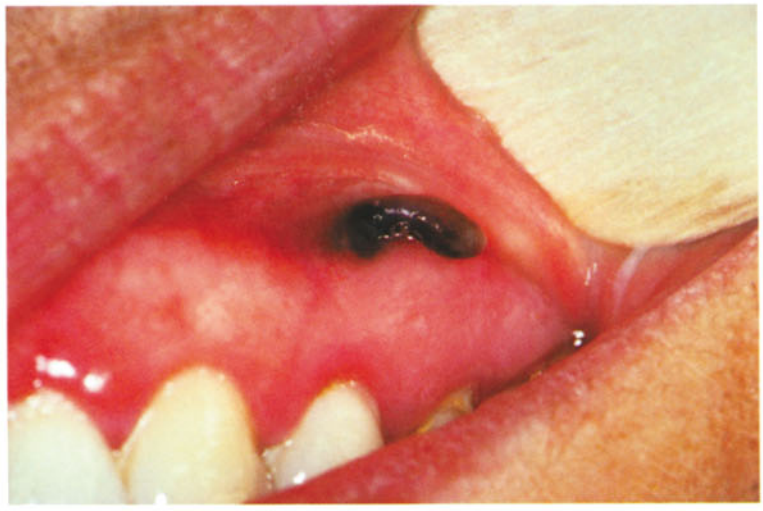


FIGURE 67

Fistula with pigmentation



For Personal Use Only
Library of School of Dentistry, TUMS

Oroantral Fistula

Definition

- A fistula is a non-anatomical epithelial lined tract joining two epithelial surfaces. An oro-antral fistula (OAF) is between the mouth (lined with squamous epithelium) and the maxillary antrum (lined with a ciliated columnar epithelium with mucous glands: respiratory epithelium).

Etiology

- As the sinus enlarges with growth it approaches closer to the teeth and the risk of creating an OAF when removing upper teeth, or of displacing teeth or roots into the antrum, is therefore much greater in adults. OAF is most often caused by the extraction of maxillary teeth, especially the molars or premolars, closely related to the antrum. Major fistulae tend to occur if the maxillary tuberosity is fractured, particularly if several teeth are attached.
- Persistent OAFs tend to occur when a molar tooth root is accidentally displaced into the maxillary sinus or when a molar tooth is extracted.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 68

Oroantral fistula

One situation that is especially prone to fistula formation is the extraction of a single standing maxillary molar or premolar whose roots are surrounded both posteriorly and anteriorly by the maxillary antrum. Roots of any maxillary premolar or molar tooth may be displaced into the antrum but the complete tooth most commonly displaced into the antrum is the unerupted upper third molar. On the other hand, small OAFs occur most frequently with extraction of the first or second molar.

- Rarely, facial trauma, neoplasms or osteomyelitis of the maxilla and surgery to the maxilla may lead to fistula formation.

Gingival Involvement

- Rare

Main Clinical Features

- Signs and symptoms from an OAF may occur immediately after the extraction of a tooth or may take some weeks to develop (Fig. 68).
- In the early post-extraction period, there may be ipsilateral nose bleed (epistaxis).
- The most common symptom is reflux of fluid from the mouth into the nose when the patient drinks. Fluid then dribbles from the nose.
- A little later, as sinusitis develops, an unpleasant ipsilateral nasal discharge may arise.

Diagnosis

- Diagnosis is from history and clinical features. An obvious OAF may be seen in the mouth but often a small hole cannot be seen.
- A way of testing for an established fistula is to place a small cotton wool pledget in the area, and ask the patient to squeeze their nose and then to attempt to blow it. This will force the cotton wool out of the fistula, and bubbles of air into the mouth through the fistula. Failing this, very gentle probing with a blunt probe, e.g. a lacrimal duct dilator, will reveal the fistula but if it is suspected that a fistula has been newly created, it is better to assume the presence of an OAF and conduct a repair without such testing.
- Sometimes a red lump is noticed in the region of an OAF. This is prolapsed antral mucosa or occasionally a prolapsed antral polyp or mucosal cyst. Sometimes a fairly large fistula may be temporarily closed by

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

a prolapse of the antral mucosa. A blunt probe will push back the prolapsed tissue and reveal an obvious fistula.

- Symptoms of sinusitis may develop later but, in approximately one-quarter of patients with a fistula, there are no symptoms. This is usually because the fistula is so small that it is closed off by surrounding mucosa, particularly if it is in the buccal sulcus where the cheek may lie against it.

Differential Diagnosis

- Oronasal fistula

Treatment

- The treatment indicated depends on the rapidity with which the diagnosis of OAF has been established.
- If diagnosis has been made as soon as fistula has been created – assuming that a tooth or tooth root has not been displaced into the antrum – the immediate treatment is to close the tooth socket. For the experienced practitioner, the procedure is to raise a buccal flap and advance it over the fistula. The less experienced practitioner should simply suture the socket. The patient should then be given prophylactic treatment against maxillary sinusitis which would otherwise delay healing of the fistula; that is:
  - The patient should avoid blowing their nose.
  - Antibiotics such as erythromycin or co-trimoxazole, nose drops (ephedrine 0.5% or xylometazoline hydrochloride 0.1%) and inhalations (such as menthol and benzoin inhalation BP or menthol and eucalyptus inhalation BP qds) should be used.
- Up to 48 h after the tooth extraction, an OAF can be repaired by advancement of a buccal flap but prophylactic antibiotics are indicated.

Reading List

Ahl DR, Hilgeman JL, Snyder JD (1986) Periodontal emergencies. Dent Clin North Am 30:459–472

Flynn TR (2000) The swollen face. Severe odontogenic infections. Emerg Med Clin North Am 18:481–519

Herrera D, Roldan S, Gonzalez I, Sanz M (2000) The periodontal abscess (I). Clinical and microbiological findings. J Clin Periodontol 27:387–394

Herrera D, Roldan S, O'Connor A, Sanz M (2000) The periodontal abscess (II). Short-term clinical and microbiological efficacy of 2 systemic antibiotic regimes. J Clin Periodontol 27:395–404

Herrera D, Roldan S, Sanz M (2000) The periodontal abscess: a review. J Clin Periodontol 27:377–386

Kuriyama T, Karasawa T, Nakagawa K, Saiki Y, Yamamoto E, Nakamura S (2000) Bacteriologic features and antimicrobial susceptibility in isolates from orofacial odontogenic infections. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 90:600–608

Meng HX (1999) Periodontal abscess. Ann Periodontol 4:79–83

American Academy of Periodontology (2000) Parameter on acute periodontal diseases. J Periodontol 71[5 Suppl]:863–866

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

CHAPTER 6

Physical Lesions

Self-Inflicted Injury

- Abrasive toothbrushing
- Chewing on sticks
- Self-induced wounds
- Tattooing

Etiology

- Most gingival physical damage is from abrasive toothbrushing, usually over-vigorous horizontal brushing.
- Children may damage their gingiva with a fingernail.
- Gingival recession may be induced by oral jewelry such as tongue studs.
- Gingival damage may be seen in:
  - Learning disability.
  - Lesch-Nyhan syndrome.
  - Syndromes with pain insensitivity:
    - Congenital indifference to pain.
    - Riley Day syndrome.

Gingival Involvement

- Uncommon

Other Sites of Involvement

- Any

Main Clinical Features

- Damage to the gingival margin or other areas accessible to the dominant hand usually (Figs. 69–73).

Diagnosis

- Diagnosis is from history and clinical features.

Differential Diagnosis

- Neoplasms
- Aphthae
- Systemic disease
  - Infections
  - Haematological disease
  - Gastrointestinal disease
  - Mucocutaneous disease
  - Drugs

Treatment

- Psychotherapy
- Protective splints

For Personal Use Only
Library of School of Dentistry, TUMS

G. Laskaris et al., *Periodontal Manifestations of Local and Systemic Diseases*
© Springer-Verlag Berlin Heidelberg 2003

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 69

Gingival trauma: toothbrushing

FIGURE 70

Gingival trauma: toothbrushing

FIGURE 71

Gingival trauma: hyperplasia due to toothbrushing

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 72

Gingival trauma: from a fingernail

FIGURE 73

Gingival trauma: from a fingernail

For Personal Use Only
Library of School of Dentistry, TUMS

Reading List

Baer PN, Archard HO (1974) Factitial disease of the gingiva and buccal mucosa. Report of a case. N Y State Dent J 40:33–36

Beckett H, Buxey-Softley G, Gilmour AG (1995) Self-inflicted gingival injury. Br Dent J 178:246

Boardman R, Smith RA (1997) Dental implications of oral piercing. J Calif Dent Assoc 25:200–207

Braham RL (1984) Oral soft tissue lesions in children and adolescents. Practitioner. 228:319–326

Chen M, Scully C (1992) Tongue piercing: a new fad in body art. Br Den J 172

Er N, Ozkavaf A, Berberoglu A, Yamalik N (2000) An unusual cause of gingival recession: oral piercing. J Periodontol 71:1767–1769

Krejci CB (2000) Self-inflicted gingival injury due to habitual fingernail biting. J Periodontol 71:1029–1031

Kretchmer MC, Moriarty JD (2001) Metal piercing through the tongue and localized loss of attachment: a case report. J Periodontol 72:831–833

Miller AS, Fantasia JE (1988) Localized gingival ulceration. Gen Dent 36:344–350

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Price SS, Lewis M (1997) Body piercing involving oral sites. J Am Dent Assoc 128:1017–1020

Scully C (1981) Orofacial manifestations of the Lesch-Nyhan syndrome. Int J Oral Surg 10:380–383

Tatakis DN, Milledge JT (2000) Severe gingival recession in trisomy 18 primary dentition. A clinicopathologic case report of self-inflicted injury associated with mental retardation. J Periodontol 71:1181–1186

Tsai TP (2001) Extraction as a treatment alternative follows repeated trauma in a severely handicapped patient. Dent Traumatol 17:139–142

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

CHAPTER 7

Chemical Lesions

Definition
- Chemically induced lesions often present as mucosal burns or white lesions.

Etiology
- Drugs
  - Analgesic tablets
  - Cocaine deliberately rubbed into the gingivae or vestibule
  - Pancreatin can cause ulceration
- Mouthwashes
  - Chlorhexidine
  - Others, especially alcohol-containing washes
- Dental procedures
  - Acids (chromic, trichloracetic, phosphoric)
  - Self-curing resins, especially epoxy resins
- Natural products
  - Tobacco products including smokeless tobacco
  - Sanguinaria
  - Areca nut
  - The houseplant Dieffenbachia
  - The enzyme bromelin in pineapple
  - Others

Gingival Involvement
- Rare

Other Sites of Involvement
- Frequent

Main Clinical Features
- Mucosal burns or white lesions (Figs. 74–80)

Diagnosis
- Diagnosis is from history and clinical features.

Differential Diagnosis
- Materia alba
- Keratoses
- Carcinoma
- Candidiasis (thrush: candidal leukoplakia)
- Epstein-Barr virus (hairy leukoplakia)
- Syphilis (syphilitic mucous patches and keratosis)
- Lichen planus
- Lupus erythematosus

Treatment
- Remove the offending agent

For Personal Use Only
Library of School of Dentistry, TUMS

G. Laskaris et al., *Periodontal Manifestations of Local and Systemic Diseases*
© Springer-Verlag Berlin Heidelberg 2003

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 74

Mucosal burn:
chemical – from a mouthwash

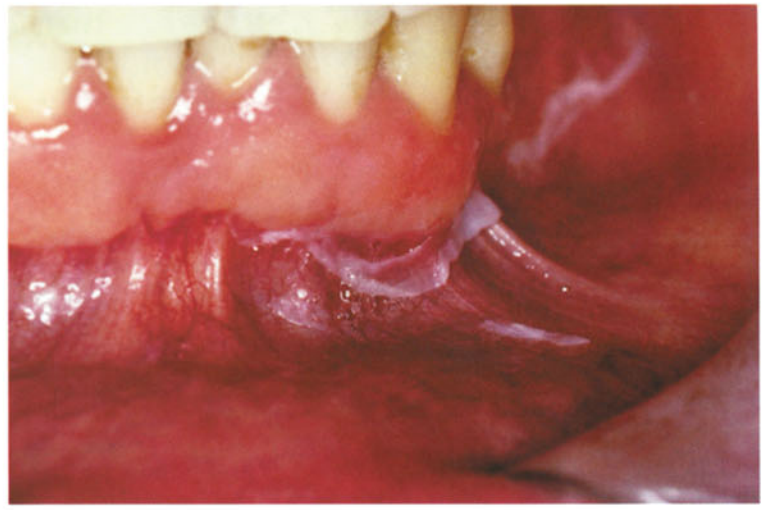


FIGURE 75

Mucosal burn:
chemical – from aspirin

FIGURE 76

Mucosal burn:
chemical – from hydrogen peroxide

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 77

Mucosal burn:
chemical – from trichloracetic acid

FIGURE 78

Mucosal burn:
chemical – from alcohol

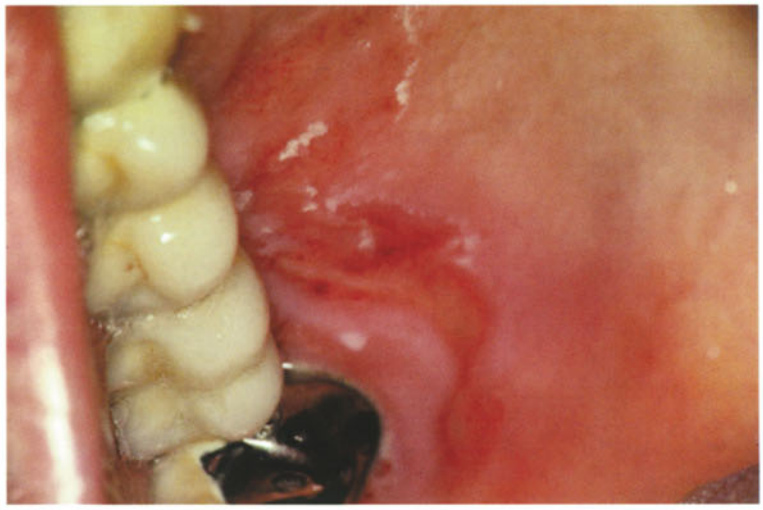
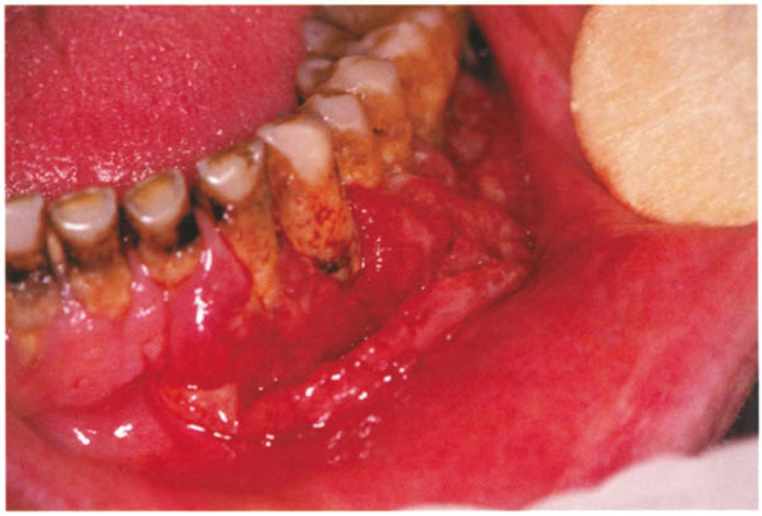


FIGURE 79

Mucosal burn:
chemical – from iodine

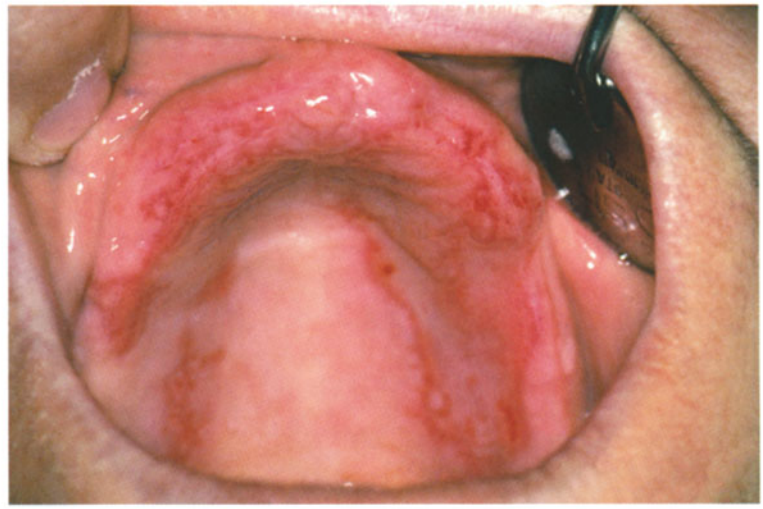


For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 80

Mucosal burn: chemical – from acrylic monomer



Reading List

De Bruyne MA, De Moor RJ, Raes FM (2000) Necrosis of the gingiva caused by calcium hydroxide: a case report. Int Endod J 33:67–71

Parry J, Porter SR, Scully C, Flint S, Parry MG (1996) Mucosal lesions due to oral cocaine use. Br Den J 180:462–464

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

CHAPTER 8

Thermal Lesions

Definition
- Thermally induced lesions often present as mucosal burns or white lesions.

Etiology
- Hot foods
- Hot instruments (dental handpiece or extraction forceps usually)
- Electrical burns
- Cryosurgery

Gingival Involvement
- Rare

Other Sites of Involvement
- Seen especially on the palate or tongue, for example, so-called pizza-palate

Main Clinical Features
- White lesions, blisters or ulcers (Fig. 81)

Diagnosis
- Diagnosis is from history and clinical features.

Differential Diagnosis
- Materia alba
- Burns
- Keratoses
- Carcinoma
- Candidiasis (thrush: candidal leukoplakia)
- Epstein-Barr virus (hairy leukoplakia)
- Syphilis (syphilitic mucous patches and keratosis)
- Lichen planus
- Lupus erythematosus

Treatment
- Remove the offending agent.
- Supportive care.

For Personal Use Only
Library of School of Dentistry, TUMS

G. Laskaris et al., *Periodontal Manifestations of Local and Systemic Diseases*
© Springer-Verlag Berlin Heidelberg 2003

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

CHAPTER 9

Lesions Due to Radiation

Mucositis

Definition
- Radiotherapy involving the mouth invariably produces burns and mucositis and if it involves the salivary glands, often produces xerostomia.

Etiology
- Ionizing radiation

Gingival Involvement
- Uncommon

Other Sites of Involvement
- Irradiation can also produce:
  - Xerostomia, which predisposes to:
    - Dental caries.
    - Candidiasis.
  - Osteoradionecrosis.
  - Scarring is later followed by the appearance of telangiectasia.
  - Taste loss.
  - Subsequent neoplasia: for example, radiotherapy to oropharyngeal neoplasms predisposes to subsequent salivary neoplasms.

Main Clinical Features
- Diffuse erythema and ulceration (Figs. 82–83).
- Gingival parameters such as bleeding index, plaque index and gingival recession are all increased after radiotherapy.

Diagnosis
- Diagnosis is from history and clinical features.

Differential Diagnosis
- Burns
- Lichen planus
- Inflammatory gingivitis
- Desquamative gingivitis
- Herpetic stomatitis, candidiasis
- Granulomas, e.g. Crohn's disease, orofacial granulomatosis or sarcoidosis

Treatment
- There is no specific treatment.
- Sucking ice is as satisfactory as any of the multitude of drugs and other agents available.
- Analgesies

For Personal Use Only
Library of School of Dentistry, TUMS

G. Laskaris et al., *Periodontal Manifestations of Local and Systemic Diseases*
© Springer-Verlag Berlin Heidelberg 2003

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 82

Mucosal burn: radiation

FIGURE 83

Mucosal burn: radiation mucositis

For Personal Use Only
Library of School of Dentistry, TUMS

Osteoradionecrosis

Definition

- Bone necrosis and infection arising in a previously irradiated jaw

Etiology

- Radiotherapy involving the jaws, especially the mandible, may predispose to osteoradionecrosis.
- Endarteritis obliterans decreases the vascular supply to bone, predisposing to osteoradionecrosis.
- The initiating factor is often trauma, such as tooth extraction or infection.
- Osteoradionecrosis used to be a common complication of radiotherapy for head and neck cancers but modern techniques have significantly reduced the risk.

Gingival Involvement

- Rare

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 84

Osteoradionecrosis

Other Sites of Involvement

- Muscle involvement can produce trismus.
- Developing teeth can be involved to cause:
  - Hypoplasia.
  - Stunted root formation.
  - Retarded eruption.
- The mandibular condyle, or other growth areas, can be damaged resulting in facial deformity.

Main Clinical Features

- Presentation is of exposed bone in an irradiated mouth with or without:
  - External sinuses (Fig. 84).
  - Pain.
  - Pathological fracture.
- Once established, the process may spread to involve the whole jaw, usually the mandible.

Diagnosis

- Diagnosis is from history and clinical features.
- Microbiology.
- Imaging.
- Others.

Differential Diagnosis

- Osteomyelitis

Treatment

- Long-term antibiotics, especially tetracycline.
- Local cleansing with aqueous 0.2% chlorhexidine.
- Sequestrectomy and surgical intervention should be minimal.
- Resection only as a last resort.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Reading List

Epstein JB, Gorsky M, Epstein MS, Nantel S (2001) Topical azathioprine in the treatment of immune-mediated chronic oral inflammatory conditions. Oral Surg 91:56–61

Fiske J, Lewis D (2001) Guidelines for the oral management of oncology patients requiring radiotherapy, chemotherapy and bone marrow transplantation. J Disabil Oral Health 2:3–14

Harrison LB, Fass DE (1990) Radiation therapy for oral cavity cancer. Dent Clin North Am 34:205–222

Markitziu A, Zafiropoulos G, Tsalikis L, Cohen L (1992) Gingival health and salivary function in head and neck-irradiated patients. A five-year follow-up. Oral Surg Oral Med Oral Pathol 73:427–433

Millard HD, Mason DK (eds) (2000) Perspectives on 1998 World Workshop on Oral Medicine. University of Michigan, Michigan

Simon M, Bartram CR, Friedrich W, Arnold R, Schmeiser T, Hampl W, Muller-Hermelink HK, Heymer B (1991) Fatal B-cell lymphoproliferative syndrome in allogeneic marrow graft recipients. A clinical, immunobiological and pathological study. Virchows Arch B Cell Pathol Incl Mol Pathol 60:307–319

Singh N, Scully C, Joyston-Bechal S (1996) Oral complications of cancer therapies: prevention and management. Clin Oncol 8:15–24

Stiff P (2001) Mucositis associated with stem cell transplantation: current status and innovative approaches to management. Bone Marrow Transplant 27 [Suppl2]:S3–S11

Swinnen L (2001) Post-transplant lymphoproliferative disorders: implications for acquired immunodeficiency syndrome-associated malignancies. J Natl Cancer Inst Monogr 28:38–43

Woo SB, Lee SJ, Schubert MM (1997) Graft-vs-host disease. Crit Rev Oral Biol Med 8:201–216

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

CHAPTER 10

Pigmented Disorders

Physiological Pigmentation

Definition
- Pigmentation related to race (racial pigmentation)

Etiology
- The most usual cause of brown oral mucosal pigmentation is ethnic, mainly in descendants of Blacks, but may be seen even in some fairly light-skinned people.

Gingival Involvement
- Common, especially anteriorly and labially

Other Sites of Involvement
- Many other sites may be affected but pigmentation is most prominent on the gingivae, buccal mucosa and tongue.

Main Clinical Features
- Brown macular areas, most obvious in anterior labial gingivae (Figs. 85, 86), mainly on attached gingivae

Diagnosis
- For generalized or multiple hyperpigmentation which is not clearly racial:
  - Blood pressure should be taken to exclude the hypotension of Addison's disease.
  - Blood tests may be needed if there is suspicion of Addison's disease; plasma cortisol levels and a Synacthen test may be indicated.
- For localized hyperpigmentation:
  - Histopathologically may be indicated.
  - Imaging may be helpful whenever hyperpigmentation may be caused by amalgam, graphite or a foreign body.
  - Photographs may be useful for future comparison.

Differential Diagnosis
- Amalgam, graphite or other tattoos
- Ephelis (freckle)
- Naevus
- Melanotic macules
- Melanoacanthoma
- Malignant melanoma
- Kaposi's sarcoma
- Epithelioid angiomatosis
- Verruciform xanthoma
- Genetic:
  - Racial
  - Peutz-Jeghers syndrome
  - Laugier-Hunziker syndrome
  - Complex of myxomas, spotty pigmentation and endocrine overactivity
  - Carney syndrome

For Personal Use Only
Library of School of Dentistry, TUMS

G. Laskaris et al., *Periodontal Manifestations of Local and Systemic Diseases*
© Springer-Verlag Berlin Heidelberg 2003

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 85

Gingival pigmentation: racial

FIGURE 86

Gingival pigmentation: racial

- Leopard syndrome
- Lentiginosis profusa

▶ Habits:
- Using betel (brown)
- Using the bark of *Juglans regia* for teeth cleaning (brown orange)
- Chewing the seeds of *Cola nitida* (yellow)
- Chewing the leaves of *Catha edulis* (brown)

▶ Drugs:
- Smoking
- Betel
- Antimalarials
- Amiodarone
- Minocycline
- Chlorpromazine
- ACTH
- Zidovudine
- Clofazimine

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

- Ketoconazole
- Methyldopa
- Busulphan
- Menthol
- Contraceptive pill
- Metals

▶ Endocrine:
- Addison's disease
- Albright's syndrome
- Pregnancy

▶ Post-inflammatory

▶ Others:
- Hemochromatosis
- Albright's syndrome
- Generalized neurofibromatosis
- Incontinentia pigmenti
- Whipple's disease
- Wilson's disease
- Gaucher's disease
- HIV disease
- Thalassaemia
- Jaundice

Treatment ▶ Reassurance

Melanotic Macule

Definition ▶ A melanotic macule is a pale brown macule less than 3 mm in diameter, with a poorly defined margin.

Etiology ▶ There is overproduction of melanin.

Gingival Involvement ▶ Very rare

Other Sites of Involvement ▶ Mainly seen on the lips

Main Clinical Features ▶ A pale brown macule less than 3 mm in diameter, with a poorly defined margin (Fig. 87)

Diagnosis ▶ Diagnosis is from history and clinical features, supplemented by:
- Histopathology in some instances.

▶ Imaging can be added, which may be helpful whenever hyperpigmentation may be caused by amalgam, graphite or a foreign body.

▶ Photographs may also be useful for future comparison.

Differential Diagnosis ▶ Racial pigmentation
▶ Localized irritation, e.g. smoking
▶ Amalgam tattoo
▶ Malignant melanoma
▶ Kaposi's sarcoma
▶ Drugs, e.g. antimalarials
▶ Addison's disease

Treatment ▶ Reassurance

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 87

Gingival pigmentation: melanotic macule

Naevus

Definition

- Naevi are circumscribed developmental anomalies, usually brown, blue or black pigmented lesions.

Etiology

- Melanocytic naevi can appear or increase in size and colour with age, at adolescence and in pregnancy or after the administration of ACTH. There are several types:
  - Lentigo, a brown circular macule due to increased melanocytes. Occasionally these are seen in hereditary syndromes such as NAME, LAMB and LEOPARD syndromes.
  - Intramucosal naevus, consisting of a collection of naevus cells in the lamina propria without involvement of the epithelium, is most common (about 60%).
  - Blue naevus, consisting of spindle cells at any level in the lamina propria, accounts for 25%.
- Others:
  - Junctional naevi, rare in the mouth.
  - Compound naevi, rare in the mouth.
  - Combined naevi, rare in the mouth.
  - Naevus of Ota, rare in the mouth.
  - Congenital naevi, rare in the mouth.
  - Dysplastic naevi have not been reported.

Gingival Involvement

- Rare

Other Sites of Involvement

- Any oral site, particularly on the palate or buccal mucosa

Main Clinical Features

- Usually brown (Figs. 88–92)
- Macular or papular
- Do not change rapidly in size or colour
- Painless

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 88

Gingival pigmentation: ephelis

FIGURE 89

Gingival pigmentation: compound naevus

FIGURE 90

Gingival pigmentation: naevus of Ota

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 91

Gingival pigmentation: naevus of Ota

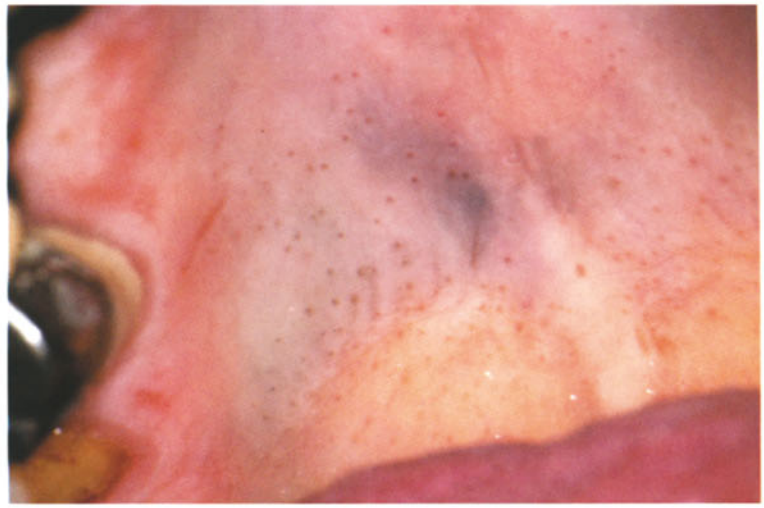


FIGURE 92

Ocular pigmentation: naevus of Ota

Diagnosis

- Diagnosis is from history and clinical features, supplemented by:
  - Histopathology.
- Imaging may be helpful whenever hyperpigmentation may be caused by amalgam, graphite or a foreign body.
- Photographs may be useful for future comparison.

Differential Diagnosis

- Racial pigmentation
- Localized irritation, e.g. smoking
- Amalgam tattoo
- Melanotic macule
- Malignant melanoma
- Kaposi's sarcoma
- Drugs, e.g. antimalarials
- Addison's disease

Treatment

- Excision is recommended both for cosmetic reasons and to exclude malignancy, especially melanoma.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Metal Tattoo

Definition
- A hyperpigmented lesion caused by embedded metal particles or salts

Etiology
- Dental amalgam incorporated into the tissues during conservative dentistry or endodontics is the common cause.

Gingival Involvement
- Common

Other Sites of Involvement
- Typically in the mandibular area, close to the teeth

Main Clinical Features
- Bluish-black macule (Figs. 93–97), especially in the gingiva or vestibule in the premolar–molar region of the mandible

Diagnosis
- Diagnosis is from history and clinical features, supplemented by:
  - Histopathology..
- Imaging may be helpful whenever hyperpigmentation may be caused by amalgam, graphite or a foreign body.
- Photographs may be useful for future comparison.

Differential Diagnosis
- Racial pigmentation
- Localized irritation, e.g. smoking
- Naevus
- Melanotic macule
- Malignant melanoma
- Kaposi's sarcoma
- Drugs, e.g. antimalarials
- Addison's disease

Treatment
- Reassurance or, if in doubt, excise

For Personal Use Only
Library of School of Dentistry, TUMS

FIGURE 93

Gingival pigmentation: amalgam tattoo

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 94

Gingival pigmentation: amalgam tattoo

FIGURE 95

Alveolar mucosa pigmentation: amalgam tattoo

FIGURE 96

Gingival pigmentation: tattoo from palladium crown

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 97

Gingival pigmentation: graphite tattoo

FIGURE 98

Gingival pigmentation: smoker's melanosis

For Personal Use Only
Library of School of Dentistry, TUMS

Smoker's Melanosis

Definition
- Hyperpigmentation induced by tobacco smoking

Etiology
- Tobacco smoking

Gingival Involvement
- Rare

Other Sites of Involvement
- Buccal mucosa

Main Clinical Features
- Brown hyperpigmentation (Fig. 98)

Diagnosis
- Diagnosis is from smoking history and clinical features, supplemented by:
  - Histopathology.
- Imaging may be helpful whenever hyperpigmentation may be caused by amalgam, graphite or a foreign body.
- Photographs may be useful for future comparison.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Differential Diagnosis

- Racial pigmentation
- Amalgam tattoo
- Naevus
- Melanotic macule
- Malignant melanoma
- Kaposi's sarcoma
- Drugs, e.g. antimalarials
- Addison's disease

Treatment

- Reassurance
- Stop habit

Reading List

Anderegg CR Jr, Lyles MB (1992) Graphite tattoo: report of a case and differential diagnosis. Mil Med 157:323–324

Araki S, Murata K, Ushio K, Sakai R (1983) Dose-response relationship between tobacco consumption and melanin pigmentation in the attached gingiva. Arch Environ Health 38:375–837

Ashri N, Gazi M (1990) More unusual pigmentations of the gingiva. Oral Surg Oral Med Oral Pathol 70:445–449

Birt BD, From L, Main JH (1978) The diagnosis of melanotic and other pigmented lesions of the lips and oral mucosa (dark spots in the mouth). J Otolaryngol 7:203–210

Brown FH, Houston GD (1991) Smoker's melanosis. A case report. J Periodontol 62:524–527

Buchner A, Hansen LS (1980) Amalgam pigmentation (amalgam tattoo) of the oral mucosa. A clinicopathologic study of 268 cases. Oral Surg Oral Med Oral Pathol 49:139–147

Buchner A, Hansen LS (1979) Melanotic macule of the oral mucosa. A clinicopathologic study of 105 cases. Oral Surg Oral Med Oral Pathol 48:244–249

Buchner A, Hansen LS (1987) Pigmented nevi of the oral mucosa: a clinicopathologic study of 36 new cases and review of 155 cases from the literature. Part II: analysis of 191 cases. Oral Surg Oral Med Oral Pathol 63:676–682

Cale AE, Freedman PD, Lumerman H (1988) Pigmentation of the jawbones and teeth secondary to minocycline hydrochloride therapy. J Periodontol 59:112–114

Dummett CO (1979) Overview of normal oral pigmentations. Ala J Med Sci 16:262–273

Eisen D, Voorhees JJ (1991) Oral melanoma and other pigmented lesions of the oral cavity. J Am Acad Dermatol 24: 527–537

Fernandez GT (2000) Pigmented lesion of the oral cavity with eight years follow-up. P R Health Sci J 19:165–168

Gorsky M, Buchner A, Moskona D, Aviv I (1984) Physiologic pigmentation of the oral mucosa in Israeli Jews of different ethnic origin. Community Dent Oral Epidemiol 12:188–190

Hansen LS, Buchner A (1981) Changing concepts of the junctional naevus and melanoma. Review of the literature and report of case. J Oral Surg 39:961–965

Lamey P-J, Carmichael F, Scully C (1985) Oral pigmentation, Addison's disease and the results of screening for adrenocortical insufficiency. Br Dentl J 158:297–305

Lockhart PB (1981) Gingival pigmentation as the sole presenting sign of chronic lead poisoning in a mentally retarded adult. Oral Surg Oral Med Oral Pathol 52:143–149

Mani NJ (1985) Gingival tattoo. A hitherto undescribed mucosal pigmentation. Quintessence Int 16:157–159

Manor A, Sperling I, Buchner A (1981) Gingival pigmentation associated with antimalarial drugs. Refuat Hapeh Vehashinayim 28:13–16

Mishiro Y, Ogihara K, Zhang Y, Hu D (1990) Gingival pigmentation in preschool children of Chengdu, West China. J Pedod 14:150–151

Patel K, Cheshire D, Vance A (1998) Oral and systemic effects of prolonged minocycline therapy. Br Dent J 185:560–562

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Porter SR, Glover S, Scully C (1990) Oral hyperpigmentation and adrenocortical hypofunction in a patient with AIDS. Oral Surg Oral Med Oral Path 67:301–302
Powers JM, Capp JA, Koran A (1977) Color of gingival tissues of blacks and whites. J Dent Res 56:112–116
Putter OH, Ouellet D, Putter A, Vilaboa D, Vilaboa B, Fernandez M (1994) A non-traumatic technique for removing melanotic pigmentation lesions from the gingiva: gingiabrasion. Dent Today 13:58, 60
Reichart PA, Phillipsen HP (1998) Betel chewer's mucosa – a review. J Oral Pathol Med 27:239–242
Scully C (1997) Drug-induced oral mucosal hyperpigmentation. Prim Dent Care 4:35–36
Seoane Leston JM, Vazquez Garcia J, Aguado Santos A, Varela-Centelles PI, Romero MA (1998) Dark oral lesions: differential diagnosis with oral melanoma. Cutis 61:279–282
Takeda T, Ishigami K, Shimada A, Ohki K (1996) A study of discoloration of the gingiva by artificial crowns. Int J Prosthodont 9:197–202
Owens BM, Johnson WW, Schuman NJ (1992) Oral amalgam pigmentations (tattoos): a retrospective study. Quintessence Int 23:805–810
Tamizi M, Taheri M (1996) Treatment of severe physiologic gingival pigmentation with free gingival autograft. Quintessence Int 27:555–558
Telang GH, Ditre CM (1994) Blue gingiva, an unusual oral pigmentation resulting from gingival tattoo. J Am Acad Dermatol 30:125–126
Trelles MA, Verkruysse W, Segui JM, Udaeta A (1993) Treatment of melanotic spots in the gingiva by argon laser. J Oral Maxillofac Surg 51:759–761
Unsal E, Paksoy C, Soykan E, Elhan AH, Sahin M (2001) Oral melanin pigmentation related to smoking in a Turkish population. Community Dent Oral Epidemiol 29:272–277
Yeh CJ (1998) Cryosurgical treatment of melanin-pigmented gingiva. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 86:660–663

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

CHAPTER 11

Allergic and Foreign Body Reactions

Reactions to Dental Materials and Oral Health Care Products

Definition

- Reactions to:
  - Materials and foods.
  - Deliberate tattoos.
- Fragments of teeth, dental materials, glass and other materials traumatically introduced into the tissues.
- Shrapnel and other materials can also be accidentally introduced.
- Material close to the gingiva may produce foreign body gingivitis.

Etiology

- Allergic reactions may be to oral health care products or foods or other antigens.
- Foreign bodies are introduced mainly in dental, sports or road traffic accidents, or assaults.
- Gingival inflammation associated with foreign bodies in the connective tissue is termed foreign body gingivitis (FBG). It is not commonly recognized by clinicians. It is more common in females. Most frequently, a red or red-and-white painful, chronic lesion, it has usually been present for less than 1 year and does not resolve with optimization of oral hygiene. It may be clinically confused with lichen planus. There is no gingival site predilection. Microscopically, foreign bodies are associated with the gingival inflammation, and elemental analysis suggests that they are usually derived from abrasives and less commonly from restorative materials.

Gingival Involvement

- Rare

Other Sites of Involvement

- Various

Main Clinical Features

- There may be pigmentation (Fig. 99), inflammation (Figs. 100, 101), whitish areas of necrotic epithelium (Fig. 102) or sometimes, no features.

Diagnosis

- Diagnosis is from history and clinical features. Wounds should always be examined by:
  - Inspection.
  - Probing.
  - Imaging.
  - Ultrasonography (sometimes).

Differential Diagnosis

- Naevus
- Neoplasm

For Personal Use Only
Library of School of Dentistry, TUMS

G. Laskaris et al., *Periodontal Manifestations of Local and Systemic Diseases*
© Springer-Verlag Berlin Heidelberg 2003

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 99

Gingival pigmentation: deliberate tattoo

FIGURE 100

Contact gingivitis from chlorhexidine

FIGURE 101

Contact gingivitis from Hexalen

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 102

Contact gingivitis from cinnamon

Treatment
- Laser or cryosurgery has been used to remove the foreign material.
- Foreign material is best removed at an early stage as it may eventually form:
  - Tattoos.
  - Granulomas.
  - Scars.

Materia Alba

Definition
- Debris consisting of squames, food, microorganisms and other material

Etiology
- Poor oral hygiene

Gingival Involvement
- Common

Other Sites of Involvement
- None

Main Clinical Features
- Whitish plaques that easily can be removed with gauze (Figs. 103, 104)

Diagnosis
- Diagnosis is from history and clinical features.

Differential Diagnosis
- Candidiasis
- Lichen planus
- Keratosis

Treatment
- Oral hygiene

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 103

Materia alba

FIGURE 104

Materia alba

For Personal Use Only
Library of School of Dentistry, TUMS

Reading List

Daley TD, Wysocki GP (1990) Foreign body gingivitis: an iatrogenic disease? Oral Surg Oral Med Oral Pathol 69:708–712

Goodman JR (1984) Fractured composite restoration embedded in the lip. Br Dent J 156:293–294

Gordon S (2000) Foreign body gingivitis associated with a new crown: EDX analysis and review of the literature. Oper Dent 25:344–348

Gordon SC, Daley TD (1997) Foreign body gingivitis: clinical and microscopic features of 61 cases. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 83:562–570

Lombardi T, Kuffer R, Dubrez B (2001) Polishing-paste-induced silica granuloma of the gingiva. Dermatology 203:177–179

Pack RS, Cichon JC (1990) Shrapnel foreign body. Oral Surg Oral Med Oral Pathol 69:266–267

Roth JS, Walczyk J (1994) Occult tooth fragments spontaneously extruded after six months. Cutis 54:253–254

Scott FR, Dhillon AP, Lewin JF, Flavell W, Laws I (1995) Gold granuloma after accidental implantation. J Clin Pathol 48:1070–1071

Taran A, Har-Shai Y, Ullmann Y, Laufer D, Peled IJ (1994) Traumatic self-inflicted bite with embedded tooth fragments in the lower lip. Ann Plast Surg 32:431–433

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

CHAPTER 12

Bacterial Infections

Necrotizing Ulcerative Gingivitis

- Acute necrotizing ulcerative gingivitis (ANUG)
- Vincent's disease
- Trench mouth

Definition

- Non-contagious anaerobic infection associated with overwhelming proliferation of *Borrelia vincentii* and fusiform bacteria

Etiology

- Anaerobic flora implicated includes fusiform bacteria and spirochaete, with proliferation of *Borrelia vincentii*, *Prevotella intermedia*, fusiform bacilli, *Selenomonas* and other anaerobes.
- Predisposing factors include:
  - Smoking.
  - Viral respiratory infections.
  - Poor oral hygiene.
  - Malnutrition.
  - Immune defects, including HIV and other viral infections, and leukaemias.

Gingival Involvement

- Uncommon except in:
  - Lower socioeconomic classes
  - HIV and other immune defects

Other Sites of Involvement

- Occasional spread to contiguous or adjacent tissues

Main Clinical Features

- Typically seen in adolescents and young adults, especially in institutions, armed forces, etc.
- Painful ulceration of the interdental papillae (Figs. 105–108).
- Pronounced tendency to gingival bleeding.
- Halitosis.
- Sialorrhoea.
- ANUG in malnourished, debilitated, or severely immunocompromised patients may extend onto the oral mucosa and skin, resulting in cancrum oris (noma; gangrenous stomatitis). Anaerobes, particularly bacteroides (*Porphyromonas*) species and *Fusobacterium necrophorum*, have been implicated and spreading necrosis eventually penetrates the buccal mucosa, leading to gangrene and ultimately a defect (oro-cutaneous fistula) and scarring.

Diagnosis

- Diagnosis is from history and clinical features, supplemented by:
  - Smear for fusospirochaetal bacteria and leucocytes.
  - Blood picture occasionally.

For Personal Use Only
Library of School of Dentistry, TUMS

G. Laskaris et al., *Periodontal Manifestations of Local and Systemic Diseases*
© Springer-Verlag Berlin Heidelberg 2003

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 105

Necrotizing gingivitis: acute early

FIGURE 106

Necrotizing gingivitis: acute moderate

FIGURE 107

Necrotizing gingivitis: acute advanced

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 108

Necrotizing gingivitis: acute advanced

Differential Diagnosis
- Acute leukaemia
- Herpetic stomatitis

Treatment
- Oral debridement and hygiene instruction
- Peroxide or perborate mouthwashes
- Metronidazole
- Periodontal assessment

Necrotizing Ulcerative Periodontitis

Definition
- Non-contagious anaerobic infection associated with overwhelming proliferation of *Borrelia vincentii* and fusiform bacteria

Etiology
- Predisposing factors include immune defects, particularly where there is also:
  - Poor oral hygiene.
  - Malnutrition.
  - Smoking.
  - Viral respiratory infections.
- Anaerobic flora implicated includes fusiform bacteria and spirochaete, with proliferation of *Borrelia vincentii*, *Prevotella intermedia*, fusiform bacilli, *Selenomonas* and other anaerobes.

Gingival Involvement
- Rare

Other Sites of Involvement
- Occasional spread to contiguous or adjacent tissues (Fig. 109)

Main Clinical Features
- Painful ulceration
- Rapid periodontal destruction (Fig. 110).

Diagnosis
- Diagnosis is from history and clinical features, supplemented by:
  - Smear for fusospirochaetal bacteria and leucocytes.
  - Blood picture occasionally.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 109

Necrotizing gingivitis: acute advanced with early noma

FIGURE 110

Necrotizing periodontitis

Differential Diagnosis
- Acute leukaemia
- Herpetic stomatitis

Treatment
- Oral debridement and hygiene instruction
- Peroxide or perborate mouthwashes
- Metronidazole
- Periodontal assessment

Pericoronitis

Definition
- Inflammation of the operculum over an erupting or impacted tooth

Etiology
- Accumulation of plaque/debris beneath an operculum and irritation by an occluding tooth

Gingival Involvement
- Common in adolescents and teenagers

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 111

Pericoronitis (the white area is caused by materia alba)

Other Sites of Involvement

- None

Main Clinical Features

- The lower third molar is the site most commonly affected, but it is occasionally seen in relation to second molars. Patients complain of:
  - Pain.
  - Trismus.
  - Swelling of operculum (Fig. 111).
  - Halitosis.
- In acute pericoronitis, there may also be:
  - Swollen, red and often ulcerated operculum.
  - Pus from beneath operculum.
  - Some facial swelling.
  - Fever.
  - Regional lymphadenitis.
  - Malaise.

Diagnosis

- Diagnosis is from history and clinical features supplemented by imaging, which is necessary to define the prognosis for the tooth involved.

Differential Diagnosis

- ANUG
- Leukaemia
- Infectious mononucleosis

Treatment

- Remove any irritation from an opposing occluding tooth cusp.
- Irrigate the operculum with saline.
- Possibly apply astringent such as trichloracetic acid to the operculum undersurface.
- Antimicrobials such as metronidazole or penicillin if there are systemic symptoms or signs.
- Consider removing the offending tooth after acute features have subsided.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Reading List

American Academy of Periodontology (2000) Parameter on acute periodontal diseases. J Periodontol 71 [5 Suppl]:863–866

Arendorf TM, Bredekamp B, Cloete CA, Joshipura K (2001) Seasonal variation of acute necrotising ulcerative gingivitis in South Africans. Oral Dis 7:150–154

Batista EL Jr, Novaes AB Jr, Calvano LM, do Prado EA, Goudouris ES, Batista FC (1999) Necrotizing ulcerative periodontitis associated with severe congenital immunodeficiency in a prepubescent subject: clinical findings and response to intravenous immunoglobulin treatment. J Clin Periodontol 26:499–504

Born S, Gaber G, Willgeroth K, Wagner U, Haneke E, Marsch WC (1999) Metastasising malignant lymphoma mimicking necrotising and hyperplastic gingivostomatitis. Eur J Dermatol 9:569–573

Enwonwu CO, Falkler WA Jr, Idigbe EO, Afolabi BM, Ibrahim M, Onwujekwe D, Savage O, Meeks VI (1999) Pathogenesis of cancrum oris (noma): confounding interactions of malnutrition with infection. Am J Trop Med Hyg 60:223–232

Enwonwu CO, Falkler WA Jr, Idigbe EO, Savage KO (1999) Noma (cancrum oris): questions and answers. Oral Dis 5:144–149

Falkler WA Jr, Enwonwu CO, Idigbe EO (1999) Microbiological understandings and mysteries of noma (cancrum oris). Oral Dis 5:150–155

Idigbe EO, Enwonwu CO, Falkler WA, Ibrahim MM, Onwujekwe D, Afolabi BM, Savage KO, Meeks VI (1999) Living conditions of children at risk for noma: Nigerian experience. Oral Dis 5:156–162

Kaimenyi JT (1999) Demography and seasonal variation of acute necrotising gingivitis in Nairobi, Kenya. Int Dent J 49:347–351

Kassirer B (1994) Smoking as a risk factor for gingival problems, periodontal problems and caries. Univ Tor Dent J 7:6–10

Murayama Y, Kurihara H, Nagai A, Dompkowski D, Van Dyke TE (1994) Acute necrotizing ulcerative gingivitis: risk factors involving host defense mechanisms. Periodontol 2000 6:116–124

Myoken Y, Sugata T, Kyo T, Fujihara M, Sugai M (1999) Pseudomonas-induced necrotizing gingivostomatitis. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 88:644–645

Ndiaye FC, Bourgeois D, Leclercq MH, Berthe O (1999) Noma: public health problem in Senegal and epidemiological surveillance. Oral Dis 5:163–166

Novak MJ (1999) Necrotizing ulcerative periodontitis. Ann Periodontol 4:74–78

Peltroche-Llacsahuanga H, Reichhart E, Schmitt W, Lutticken R, Haase G (2000) Investigation of infectious organisms causing pericoronitis of the mandibular third molar. J Oral Maxillofac Surg 58:611–616

Punwutikorn J, Waikakul A, Ochareon P (1999) Symptoms of unerupted mandibular third molars. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 87:305–310

Rowland RW (1999) Necrotizing ulcerative gingivitis. Ann Periodontol 4:65–73

Scully C (2000) Acute necrotizing ulcerative gingivitis; hematological parameters as evidence of nutritional status in non-HIV-infected young adults. Electronic Dental Journal. EJ10226

Wade DN, Kerns DG (1998) Acute necrotizing ulcerative gingivitis-periodontitis: a literature review. Mil Med 163:337–342

Wyss C, Dewhirst FE, Gmur R, Thurnheer T, Xue Y, Schupbach P, Guggenheim B, Paster BJ (2001) *Treponema parvum* sp. nov., a small, glucoronic or galacturonic acid-dependent oral spirochaete from lesions of human periodontitis and acute necrotizing ulcerative gingivitis. Int J Syst Evol Microbiol 51:955–962

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

CHAPTER 13

Viral Infections

Herpetic Gingivitis

Primary

Definition
- Primary infection with herpes simplex virus (HSV)

Etiology
- HSV-type 1 usually, transmitted in saliva
- Rarely HSV-type 2

Gingival Involvement
- Very common; primary herpetic gingivostomatitis is the most common acute viral infection of oral mucosa
- Non-plaque-induced lesions with no attachment loss

Other Involvement
- Tongue, buccal mucosa, palate, lips

Main Clinical Features
- Acute onset.
- Erythema, gingival swelling.
- Vesicles.
- Then erosions (Figs. 112–115) and pain.
- High fever, regional lymphadenopathy.
- General malaise.
- The lesions persist for 1–2 weeks and heal without scarring.

Diagnosis
- Diagnosis is from history and clinical features, supplemented by:
  - HSV immunostaining.
  - Culture.

Differential Diagnosis
- Necrotizing ulcerative gingivitis
- Desquamative gingivitis
- Erythema multiforme
- Pemphigus vulgaris

Treatment
- Symptomatic
- Aciclovir systemically as early as possible

For Personal Use Only
Library of School of Dentistry, TUMS

G. Laskaris et al., *Periodontal Manifestations of Local and Systemic Diseases*
© Springer-Verlag Berlin Heidelberg 2003

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 112

Infective gingivitis: primary herpes simplex stomatitis

FIGURE 113

Infective gingivitis: primary herpes simplex stomatitis

FIGURE 114

Infective gingivitis: primary herpes simplex stomatitis

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 115

Infective gingivitis: primary herpes simplex stomatitis

Secondary

Definition
- Recurrent infection with herpes simplex virus (HSV)

Etiology
- HSV, usually type 1
- Most likely:
  - After trauma, such as a local analgesic injection
  - In immunosuppressed patient, e.g. HIV or leukaemia

Gingival Involvement
- Uncommon

Other Sites of Involvement
- Any oral site

Main Clinical Features
- Multiple vesicles and round scattered ulcers with yellow slough and erythematous halo (Figs. 116–118).
- Ulcers fuse to produce irregular lesions, typically in palate or gingiva.

Diagnosis
- Diagnosis is from history and clinical features, supplemented by:
  - Serology, which is confirmatory but rarely required. A rising titre of antibodies is confirmatory.
- Smear for viral damaged cells, viral culture or electron microscopy is used occasionally.

Differential Diagnosis
- Other causes of mouth ulcers, especially:
  - Hand, foot and mouth disease
  - Herpangina
  - Shingles
  - Erythema multiforme
  - Leukaemia

Treatment
- Soft diet
- Adequate fluid intake
- Antipyretics/analgesics (paracetamol elixir)
- Local antiseptics (0.2% aqueous chlorhexidine mouthwashes)
- Aciclovir orally or parenterally in immunocompromised patients

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 116

Infective gingivitis: recurrent herpes simplex stomatitis

FIGURE 117

Infective gingivitis: recurrent herpes simplex stomatitis

FIGURE 118

Infective gingivitis: recurrent herpes simplex stomatitis

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 119

Infective gingivitis: human papillomavirus; condyloma acuminatum

Papillomaviruses

Condyloma Acuminatum

Definition
- Infection with human papillomavirus (HPV)

Etiology
- HPV-related lesion transmitted by orogenital or oroanal sex

Gingival Involvement
- Rare

Other Sites of Involvement
- Anogenital region

Main Clinical Features
- Warty papules or more smooth-surfaced lesions (Fig. 119)
- Occasionally on tongue or palate

Diagnosis
- Diagnosis is from history and clinical features, supplemented by:
  - Histopathology.

Differential Diagnosis
- Other HPV-related lesions such as papilloma

Treatment
- Medical:
  - Podophyllum
  - Imiquimod
  - Intralesional interferon alpha
- Surgical:
  - Excision
  - Laser
  - Electrosurgery
  - Cryosurgery

Verruca Vulgaris

Definition
- Infection with HPV

Etiology
- Infection with HPV from a skin wart

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 120

Infective gingivitis: human papillomavirus; verruca vulgaris

FIGURE 121

Infective gingivitis: human papillomavirus; verruca vulgaris

FIGURE 122

Infective gingivitis: human papillomavirus; verruca vulgaris

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Gingival Involvement ▶ Rare

Other Sites of Involvement ▶ Mainly skin

Main Clinical Features ▶ Warty papules or more smooth-surfaced lesions (Figs. 120–122)

Diagnosis ▶ Diagnosis is from history and clinical features, supplemented by:
- Histopathology to differentiate from other HPV-related lesions and tumours.

Differential Diagnosis ▶ Other HPV-related lesions such as papilloma

Treatment ▶ Medical:
- Podophyllum
- Imiquimod
- Intralesional interferon alpha

▶ Surgical:
- Excision
- Laser
- Electrosurgery
- Cryosurgery

Papilloma

Definition ▶ Infection with HPV, usually HPV-6 or 11

Etiology ▶ HPV infections

Gingival Involvement ▶ Uncommon
▶ The most common benign soft tissue neoplasm
▶ Found in the 20–50-year age group

Other Sites of Involvement ▶ Mainly lip, sometimes palate, tongue or other sites

Main Clinical Features ▶ Typically asymptomatic
▶ Warty papules or more smooth-surfaced lesion (Figs. 123, 124)
▶ Pedunculated lesion, either pink or white if hyperkeratinized

Diagnosis ▶ Diagnosis is from history and clinical features, supplemented by:
- Histopathology.

Differential Diagnosis ▶ Other HPV-related lesions such as condyloma

Treatment ▶ Medical:
- Podophyllum
- Imiquimod
- Intralesional interferon alpha

▶ Surgical:
- Excision
- Laser
- Electrosurgery
- Cryosurgery

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 123

Infective gingivitis: human papillomavirus; papilloma

FIGURE 124

Infective gingivitis: human papillomavirus; papilloma

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Reading List

Ammatuna P, Campisi G, Giovannelli L, Giambelluca D, Alaimo C, Mancuso S, Margiotta V (2001) Presence of Epstein-Barr virus, cytomegalovirus and human papillomavirus in normal oral mucosa of HIV-infected and renal transplant patients. Oral Dis 7:34–40

Birek C (2000) Herpesvirus-induced diseases: oral manifestations and current treatment options. J Calif Dent Assoc 28:911–921

Di Alberti L, Porter SR, Scully C, Speight P, Zakrzewska J, Artese L, Piattelli A, Ngui SL, Teo CG (1996) Genetic polymorphism of, and new diseases associated with, Kaposi's sarcoma herpes virus (KSHV). J Oral Pathol Med 25:282–266

DiAlberti L, Ngui SL, Porter SR, Speight PM, Scully C, Zakrzewska JM, Williams IG, Artese L, Piattelli A, Teo CG (1997) Presence of human herpesvirus-8 variants in the oral ulcer tissues of Human Immunodeficiency Virus-infected persons. J Infec Dis 1175:703–707

Epstein JB, Scully C (1991) Herpes simplex virus in immunocompromised patients: growing evidence of drug resistance. Oral Surg Oral Med Oral Pathol 72:47–50

Epstein JB, Scully C (1993) Cytomegalovirus: a virus of increasing relevance to oral medicine and pathology. J Oral Pathol Med 22:348–353

Femiano F, Gombos S, Scully C (2001) Recurrent herpes labialis; efficacy of topical therapy with penciclovir compared with acyclovir (aciclovir). Oral Dis 7:31–32

Goldman BD (2000) Herpes serology for dermatologists. Arch Dermatol 136:1158–1161

Kimberlin DW, Lin CY, Jacobs RF, Powell DA, Frenkel LM, Gruber WC, Rathore M, Bradley JS, Diaz PS, Kumar M, Arvin AM, Gutierrez K, Shelton M, Weiner LB, Sleasman JW, de Sierra TM, Soong SJ, Kiell J, Lakeman FD, Whitley RJ (2001) Natural history of neonatal herpes simplex virus infections in the acyclovir era. Pediatrics 108:223–229

Knaup B, Schunemann S, Wolff MH (2000) Subclinical reactivation of herpes simplex virus type 1 in the oral cavity. Oral Microbiol Immunol 15:281–283

Kolokotronis A, Louloudiadis K, Fotiou G, Matiais A (2001) Oral manifestations of infections of infections due to varicella zoster virus in otherwise healthy children. J Clin Pediatr Dent 25:107–112

Leao JC, Porter SR, Scully C (2000) Human herpesvirus 8 and oral health care: an update. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 90):694–704

Porter SR, Scully C, Mutlu S (1995) Viral infections affecting periodontal health. Periodon Clin Invest 15:17–24

Samonis G, Mantadakis E, Maraki S (2000) Orofacial viral infections in the immunocompromised host. Oncol Rep 7:1389–1394

Scully C, Cox M, Prime SS, Maitland NJ (1988) Papillomaviruses: the current status in relation to oral disease. Oral Surg Oral Med Oral Pathol 65:526–532

Scully C, Porter SR, Di Alberti L, Jalal H, Maitland NJ (1988) Detection of Epstein-Barr virus in oral scrapes in HIV infection, in hairy leukoplakia, and in healthy non-HIV-infected persons. J Oral Pathol Med 27:480–482

Scully C (1989) Orofacial herpes simplex virus infections: current concepts on the epidemiology, pathogenesis and treatment and disorders in which the virus may be implicated. Oral Surg Oral Med Oral Pathol 68:701–710

Syrjanen S, Leimola-Virtanen R, Schmidt-Westhausen A, Reichart PA (1999) Oral ulcers in AIDS patients frequently associated with cytomegalovirus (CMV) and Epstein-Barr virus (EBV) infections. J Oral Pathol Med 28:204–209

Triantos D, Boulter AW, Leao JC, Di Alberti L, Porter SR, Scully C, Birnbaum W, Johnson NW, Teo CG (1998) Diversity of naturally occurring Epstein-Barr virus revealed by nucleotide sequence polymorphism in hypervariable domains in the BamH1K and N subgenomic regions. J Gen Virol 79:2809–2817

Triantos D, Leao JC, Porter SR, Scully CM, Teo CG (1998) Tissue distribution of Epstein-Barr virus genotypes in hosts coinfected by HIV. AIDS. 12:2141–2146

Whitley RJ, Roizman B (2001) Herpes simplex virus infections. Lancet 357:1513–1518

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

CHAPTER 14

Fungal Infections

Acute Pseudomembranous Candidiasis

Definition

- Infection with yeasts of the species *Candida*

Etiology

- Predisposing factors to pseudomembranous candidiasis include:
  - *C. albicans.*
  - *C. tropicalis.*
  - *C. glabrata.*
  - *C. parapsilosis.*
  - *C. krusei.*
  - Other *Candida* species.
  - Other genera (*Rhodotorula*, *Saccharomyces*, etc.), which are rare and transient.
- Factors that can increase the liability to candidiasis include:
  - Xerostomia.
  - Smoking.
  - Use of corticosteroids systemically or in the mouth/pharynx.
  - Use of broad-spectrum antimicrobials.
  - Cytotoxic chemotherapy.
  - Irradiation involving the mouth/salivary glands.
  - Malnutrition.
  - Appliances.
  - Immune defects. Candidiasis may be seen in healthy neonates (who have immature immune systems and no acquired immunity). Any T cell defect, especially HIV infection, immunosuppressive and cytotoxic treatment, leukaemias and lymphomas, and cancer can predispose to candidiasis and other fungal infections. Candidiasis can also be seen where there are neutrophil defects such as in diabetes.

Gingival Involvement

- Common

Other Sites of Involvement

- Any oral or mucocutaneous site

Main Clinical Features

- White or creamy plaques that can be wiped off to leave a red base (Fig. 125)

Diagnosis

- Diagnosis is from history and clinical features, supplemented by:
  - Gram-stained smear (hyphae) or oral rinse.

Differential Diagnosis

- Koplik's spots
- Fordyce's spots
- Lichen planus

For Personal Use Only
Library of School of Dentistry, TUMS

G. Laskaris et al., *Periodontal Manifestations of Local and Systemic Diseases*
© Springer-Verlag Berlin Heidelberg 2003

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 125

Infective gingivitis: candidiasis; pseudomembranous

Treatment

- Avoid or reduce smoking.
- Treat any local predisposing cause such as xerostomia.
- Improve oral hygiene.
- Antifungals such as nystatin oral suspension *or* pastilles *or* amphotericin lozenges *or* miconazole gel *or* tablets *or* fluconazole tablets *or* suspension. Itraconazole in the immunocompromised.
- Chlorhexidine and triclosan have some anti-candidal activity.

Erythematous Candidiasis

Definition

- Red lesions due to *Candida*

Etiology

- Predisposing factors to erythematous candidiasis include:
  - *C. albicans.*
  - *C. tropicalis.*
  - *C. glabrata.*
  - *C. parapsilosis.*
  - *C. krusei.*
  - Other *Candida* species.
  - Other genera (*Rhodotorula*, *Saccharomyces*, etc.), which are rare and transient.
- Red lesions may be seen in denture-induced stomatitis, antibiotic-induced stomatitis, sometimes in median rhomboid glossitis, and in HIV infection may develop de novo, may precede pseudomembranous candidiasis, or may arise as a consequence of persistent acute pseudomembranous candidiasis when the pseudomembranes are shed. Factors that can increase the liability to candidiasis include:
  - Xerostomia.
  - Smoking.
  - Use of corticosteroids systemically or in the mouth/pharynx.
  - Use of broad-spectrum antimicrobials.
  - Cytotoxic chemotherapy.
  - Irradiation involving the mouth/salivary glands.
  - Malnutrition.
  - Appliances.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 126

Infective gingivitis: candidiasis; erythematous

FIGURE 127

Infective gingivitis: candidiasis; erythematous, denture-induced

For Personal Use Only
Library of School of Dentistry, TUMS

- Immune defects. Candidiasis may be seen in healthy neonates (who have immature immune systems and no acquired immunity). Any T cell defect, especially HIV infection, immunosuppressive and cytotoxic treatment, leukaemias and lymphomas, and cancer can predispose to candidiasis and other fungal infections. Candidiasis can also be seen where there are neutrophil defects such as in diabetes.

Gingival Involvement

▶ Rare

Other Sites of Involvement

▶ Red lesions that involve the gingivae (Figs. 126, 127) may be seen in denture-induced stomatitis and antibiotic-induced stomatitis. Denture-induced stomatitis is usually chronic atrophic candidiasis and consists of mild inflammation of the mucosa beneath a denture – usually a complete upper denture. In HIV infection, erythematous candidiasis may develop de novo, may precede pseudomembranous candidiasis, or may arise as a consequence of persistent acute pseudomembranous candidiasis when the pseudomembranes are shed.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Main Clinical Features
- Erythema and soreness

Diagnosis
- This is a clinical diagnosis but if the etiology is not absolutely clear, a blood picture, and smears for fungal hyphae and culture may be helpful to management.

Differential Diagnosis
- Inflammatory gingivitis
- Desquamative gingivitis
- Trauma
- Drugs and plasma cell gingivostomatitis
- Infections, e.g. primary herpetic stomatitis, candidiasis
- Granulomas, e.g. Crohn's disease, orofacial granulomatosis or sarcoidosis
- Kaposi's sarcoma

Treatment
- Avoid or reduce smoking.
- Treat any local predisposing cause such as xerostomia.
- Improve oral hygiene.
- Antifungals such as nystatin oral suspension *or* pastilles *or* amphotericin lozenges *or* miconazole gel *or* tablets *or* fluconazole tablets *or* suspension.
- Chlorhexidine and triclosan have some anti-candidal activity.

Reading List

Cannon RD, Chaffin WL (1999) Oral colonization by Candida albicans. Crit Rev Oral Biol Med 10:359–383

Chow CK, Matear DW, Lawrence HP (1999) Efficacy of antifungal agents in tissue conditioners in treating candidiasis. Gerodontology 16:110–118

Clarkson JE, Worthington HV, Eden OB (2000) Prevention of oral mucositis or oral candidiasis for patients with cancer receiving chemotherapy (excluding head and neck cancer). Cochrane Database Syst Rev 2:CD000978

Guggenheimer J, Moore PA, Rossie K, Myers D, Mongelluzzo MB, Block HM, Weyant R, Orchard T (2000) Insulin-dependent diabetes mellitus and oral soft tissue pathologies: II. Prevalence and characteristics of Candida and Candidal lesions. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 89:570–576

Schorling SR, Kortinga HC, Froschb M, Muhlschlegel FA (2000) The role of Candida dubliniensis in oral candidiasis in human immunodeficiency virus-infected individuals. Crit Rev Microbiol 26:59–68

Scully C, El-Kabir M, Samaranayake LP (1994) Candida and oral candidosis. Critical Rev Oral Biol Med 5:124–158

Taillandier J, Esnault Y, Alemanni M (2000) A comparison of fluconazole oral suspension and amphotericin B oral suspension in older patients with oropharyngeal candidosis. Multicentre Study Group. Age Ageing 29:117–123

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

CHAPTER 15

Aphthous Ulceration

Definition

- Recurrent idiopathic mouth ulcers with onset usually in childhood or adolescence

Etiology

- Unclear: immunological changes are detectable but there is no reliable evidence of autoimmune disease or any classic immunological reactions. It may be due to:
  - Changes in cell-mediated immune responses.
  - Cross-reactivity with *Streptococcus sanguis* or heat shock protein.
- Underlying predisposing factors seen in a minority include:
  - Haematinic (iron, folate or vitamin B12) deficiency in 10%–20%.
  - Relationship with luteal phase of menstruation.
  - Stress (and stopping smoking).
  - Food allergies.
  - HIV disease.

Gingival Involvement

- Uncommon

Other Sites of Involvement

- None

Main Clinical Features

- Affects about 25% of population, mostly non-smokers.
- Onset is usually in childhood or adolescence.
- Later onset may signify haematinic deficiency or HIV disease.
- Recurrent round or ovoid ulcers (Figs. 128–132) usually lasting 1 week or 1 month.
- There are three distinct clinical patterns of aphthae:
  - Minor – small ulcers (< 4 mm) on mobile mucosae, healing within 14 days, no scarring (Figs. 128-130).
  - Major – large ulcers (may be > 1 cm), any site including dorsum of tongue and hard palate, healing within 1–3 months, with scarring (Figs. 131, 132).
  - Herpetiform ulcers – multiple minute ulcers that coalesce to produce ragged ulcers.

Diagnosis

- Diagnosis is from history and clinical features, supplemented by:
  - A blood picture, which is useful to reveal possible deficiencies.

Differential Diagnosis

- Local causes of ulcers
- Neoplasms
- PFAPA syndrome (periodic fever, aphthous stomatitis, pharyngitis, and adenopathy)
- Behçet's syndrome
- Systemic disease
  - Infections:
  - Haematological disease

For Personal Use Only
Library of School of Dentistry, TUMS

G. Laskaris et al., *Periodontal Manifestations of Local and Systemic Diseases*
© Springer-Verlag Berlin Heidelberg 2003

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

- Gastrointestinal disease
- Mucocutaneous disease
- Drugs
- Irradiation

Treatment

- ▶ Treat any underlying predisposing factors.
- ▶ Treat aphthae with:
  - Chlorhexidine 0.2% aqueous mouthwash or tetracycline rinses.
  - Topical corticosteroids (hydrocortisone hemisuccinate 2.5 mg pellets or 0.1% triamcinolone acetonide in Orabase).
  - Sometimes more potent topical steroids (e.g. betamethasone, beclomethasone, fluocinonide or fluticasone).
  - Rarely, other agents such as levamisole or thalidomide.

FIGURE 128

Aphthous stomatitis: minor aphthae

FIGURE 129

Aphthous stomatitis: minor aphthae

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 130

Aphthous stomatitis: minor aphthae

FIGURE 131

Aphthous stomatitis: major aphthae

FIGURE 132

Aphthous stomatitis: major aphthae

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Reading List

Brice SL, Cook D, Leahy M, Huff JC, Weston WL (2000) Examination of the oral mucosa and peripheral blood cells of patients with recurrent aphthous ulceration for human herpesvirus DNA. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 89:193–198

Fresko I, Yurdakul S, Hamuryudan V, Ozyazgan Y, Mat C, Tanverdi MM, Yazici H (1999) The management of Behçet's syndrome. Ann Med Interne (Paris) 150: 576–581

Ghate JV, Jorizzo JL (1999) Behçet's disease and complex aphthosis. J Am Acad Dermatol 40:1–18

McBride DR (2000) Management of aphthous ulcers. Am Fam Physician 62:149–154

Natah SS, Hayrinen-Immonen R, Hietanen J, Malmstrom M, Konttinen YT (2000) Immunolocalization of tumor necrosis factor-alpha expressing cells in recurrent aphthous ulcer lesions (RAU). J Oral Pathol Med 29:19–25

Ramirez-Amador VA, Esquivel-Pedraza L, Ponce-de-Leon S, Reyes-Teran G, Gonzalez-Guevara M, Ponce-de-Leon S, Sierra-Madero JG (1999) Thalidomide as therapy for human immunodeficiency virus-related oral ulcers: a double-blind placebo-controlled clinical trial. Clin Infect Dis 28:892–894

Riggio MP, Lennon A, Ghodratnama F, Wray D (2000) Lack of association between Streptococcus oralis and recurrent aphthous stomatitis. J Oral Pathol Med 29:26–32

Padeh S, Brezniak N, Zemer D, Pras E, Livneh A, Langevitz P, Migdal A, Pras M, Passwell JH (1999) Periodic fever, aphthous stomatitis, pharyngitis, and adenopathy syndrome: clinical characteristics and outcome. J Pediatr 135:98–101

Porter SR, Scully C, Pedersen A (1998). Recurrent aphthous stomatitis. Crit Rev Oral Biol Med 9:306–321

Teixeira F, Mosqueda-Taylor A, Montano S, Dominguez-Soto L (1999) Treatment of recurrent oral ulcers with mometasone furoate lotion. Postgrad Med J 75:574

Verpilleux MP, Bastuji-Garin S, Revuz J (1999) Comparative analysis of severe aphthosis and Behçet's disease: 104 cases. Dermatology 198:247–251

Zain RB (2000) Oral recurrent aphthous ulcers/stomatitis: prevalence in Malaysia and an epidemiological update. J Oral Sci 42:15–19

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

PART III Systemic Diseases

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

CHAPTER 16

Genetic Disorders

Hereditary Gingival Fibromatosis

Definition
- Hereditary gingival fibromatosis is a rare genetic disorder that may exist as an isolated disease entity or as part of a syndrome.

Etiology
- Genetic. As a single disease entity it may be sporadic or may be transmitted as an autosomal dominant or rarely autosomal recessive trait.

Gingival Involvement
- Always present
- Non-plaque-induced gingival lesions

Other Sites of Involvement
- May vary according to the syndrome it is associated with. No other involvement in isolated cases.

Main Clinical Features
- Severe generalized gingival overgrowth covering the teeth partially or completely is the characteristic feature (Fig. 133, 134).
- Gingiva are smooth, of a firm consistency and usually normal in colour.
- Appear during childhood and occasionally delay eruption of teeth.

Diagnosis
- Diagnosis is made from history and clinical features, supplemented by:
  - Biopsy and histopathological examination.

Differential Diagnosis
- Several syndromes associated with gingival fibromatosis (Laband, Cross, Ramon, Rutherford)
- Drug-induced gingival overgrowth (phenytoin, ciclosporin, calcium channel blockers)
- Familial acanthosis nigricans
- Crohn's disease
- Amyloidosis
- Leukaemia

Treatment
- Gingivectomy
- Dental plaque control

For Personal Use Only
Library of School of Dentistry, TUMS

G. Laskaris et al., *Periodontal Manifestations of Local and Systemic Diseases*
© Springer-Verlag Berlin Heidelberg 2003

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 133

Congenital gingival fibromatosis. Generalized gingival overgrowth covering the teeth

FIGURE 134

Congenital gingival fibromatosis. Localized early gingival overgrowth

For Personal Use Only
Library of School of Dentistry, TUMS

White Sponge Naevus

Definition
- White sponge naevus, or Cannon's disease, is a relatively uncommon genetic disorder that primarily affects the oral mucosa.

Etiology
- Genetic. It is inherited as an autosomal dominant trait.

Gingival Involvement
- Rare

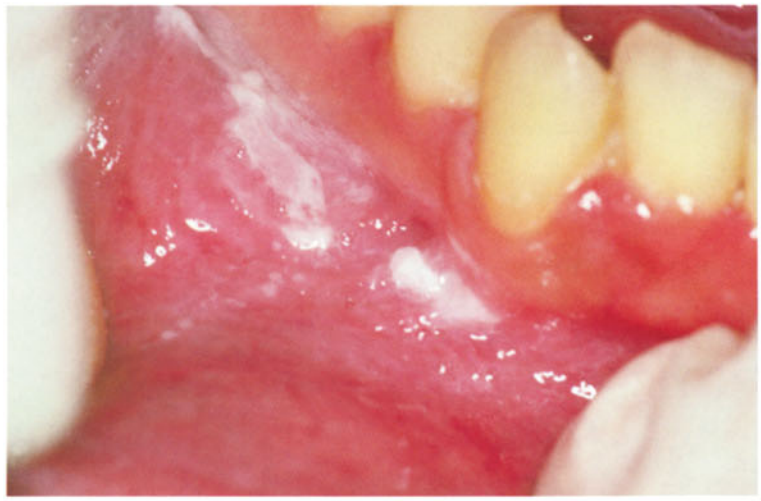
Other Sites of Involvement
- Oral mucosa (buccal, tongue, floor of the mouth, labial), always present
- Other mucosae (vaginal, rectal, oesophageal, nasal), rare

Main Clinical Features
- The gingival lesions present as painless white or grey-white, gingival and alveolar mucosa thickenings with multiple furrows, diffuse plaques with a spongy texture (Fig. 135).
- Similar lesions appear more often in other sites of the oral mucosa.
- Oral lesions become fully fledged by the second decade of life and remain relatively stable thereafter.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 135

White sponge naevus. White thickening of the alveolar mucosa extending to the gingiva



Diagnosis
- The diagnosis is based on clinical and laboratory criteria.
- Biopsy and histopathological examination.
- Cytology is useful.

Differential Diagnosis
- Leukoplakia
- Focal palmoplantar and oral mucosa hyperkeratosis syndrome

Treatment
- No treatment available
- Surgical excision of gingival lesions only for aesthetic purposes

Laband Syndrome

Definition
- Laband syndrome is a rare disorder characterized by a broad spectrum of lesions.

Etiology
- Genetic. It is inherited as an autosomal dominant trait.

Gingival Involvement
- Always present
- Non-dental plaque-induced gingival lesions with no attachment loss

Other Sites of Involvement
- Oral mucosa, rare
- Skin, facies, skeletal
- Liver, spleen

Main Clinical Features
- Gingival fibromatosis is a constant feature and it is manifested at birth or within the first 2 months of life. The gingiva are characteristically enlarged and cover the crowns of the teeth partly or completely (Fig. 136). The enlarged gingiva are usually lobulated and slightly firm on palpation.
- Macroglossia and enlargement of the lips are less common manifestations.
- Large nose and pinnae, hypoplastic or absent thumb nails, rudimentary or missing terminal phalanges, pes cavus, joint hypermobility and hirsutism, are common (Fig. 137).
- Hepatomegaly, splenomegaly and learning disability have been noted.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 136

Laband syndrome. Gingival overgrowth covering the crowns of the teeth

FIGURE 137

Laband syndrome. Hypoplastic thumb nails

Diagnosis

- The diagnosis is based on clinical criteria.

Differential Diagnosis

- Hereditary gingival fibromatosis
- Drug-influenced gingival enlargement (phenytoin, ciclosporin, calcium channel blockers)
- Several syndromes associated with gingival fibromatosis (Rutherford syndrome, Murray-Puretic-Drescher syndrome, Cross and Ramon syndromes)
- Alpha-mannosidosis

Treatment

- Good oral hygiene
- Gingivectomy

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Papillon-Lefèvre Syndrome

Definition

- Papillon-Lefèvre is a rare genetic disorder characterized by periodontal and skin manifestations.

Etiology

- Genetic. It is inherited as an autosomal recessive trait.
- Periodontal lesions are due to multiple leukocyte dysfunctions, and a specific bacterial infection of dental plaque origin (*Actinobacillus actinomycetemcomitans*).

Periodontal Involvement

- Always present
- Periodontitis associated with genetic disorder

Other Sites of Involvement

- Skin, always present (palms, soles, elbows, knees, dorsum of the fingers and toes)

Main Clinical Features

- Clinically, the gingiva are red, swollen and bleed easily. Severe generalized periodontitis with deep periodontal pockets and bone loss are constant features (Fig. 138). Mobility, migration and early loss both of deciduous and permanent teeth following the order of eruption is the rule.
- The skin lesions are characterized by hyperkeratosis of palms and soles (Figs. 139, 140). Hyperkeratosis and psoriasiform scaly red patches may occur in other skin areas.

Diagnosis

- The diagnosis is based on clinical criteria.
- Panoramic radiography.
- Skin biopsy and histopathological examination.

Differential Diagnosis

- Generalized aggressive periodontitis
- Hypophosphatasia, acatalasia
- Chediak-Higashi syndrome
- Langerhans cell histiocytosis
- Glycogen storage disease 1b
- Congenital neutropenia, cyclic neutropenia

Treatment

- Dental plaque control
- Good oral hygiene
- Aromatic retinoids for skin lesions

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 138

Papillon-Lefèvre syndrome. Severe generalized periodontitis in a 13-year-old boy

FIGURE 139

Papillon-Lefèvre syndrome. Hyperkeratosis of the palms

FIGURE 140

Papillon-Lefèvre syndrome. Hyperkeratosis of the soles

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Hypophosphatasia

Definition
- Hypophosphatasia is a rare inherited enzyme deficiency disorder, characterized by decreased serum and tissue alkaline phosphate production and activity.

Etiology
- Genetic. It is usually inherited as an autosomal recessive trait and rarely as an autosomal dominant trait.

Periodontal Involvement
- Very common, particularly in the childhood form of the disease
- Periodontitis associated with genetic disorder

Other Sites of Involvement
- Skeleton, skull, eyes

Main Clinical Features
- The periodontal manifestations include severe loss of alveolar bone and premature loss of deciduous and permanent teeth in the absence of an inflammatory response (Fig. 141). Early exfoliation particularly affects the anterior teeth and may be the only clinical sign of the disease.
- Skeletal abnormalities, growth retardation, craniosynostosis, intracranial hypertension may occur.

Diagnosis
- Diagnosis is made from history and clinical features, supplemented by:
  - Panoramic radiography.
  - Radiographic examination of the skeleton and skull.
  - Measurement of serum alkaline phosphatase and blood and urine phosphoethanolamine.

Differential Diagnosis
- Localized aggressive periodontitis
- Acatalasia
- Papillon-Lefèvre syndrome
- Chediak-Higashi syndrome
- Glycogen storage disease 1b

Treatment
- Good oral hygiene.
- Vitamin D, phosphorus and parathormone have been used with poor results.

FIGURE 141

Hypophosphatasia. Premature loss of deciduous tooth and absence of inflammatory response

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 142

Glycogen storage disease, type 1b. Localized early onset gingivitis

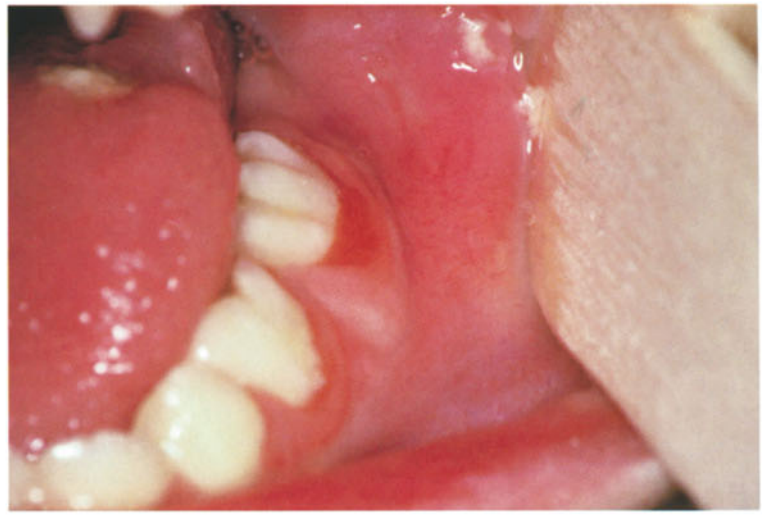


FIGURE 143

Glycogen storage disease, type 1b. Generalized severe periodontitis

For Personal Use Only
Library of School of Dentistry, TUMS

Glycogen Storage Disease 1b

Definition

- Glycogen storage disease 1b is a rare metabolic disorder with severe prognosis.

Etiology

- Genetic. It is inherited as an autosomal recessive trait.
- This is a defect of the microsomal glucose-6-phosphate translocase. The disorder is associated with neutropenia and neutrophil dysfunction.

Periodontal Involvement

- Relatively common
- Periodontitis associated with genetic disorder

Other Sites of Involvement

- Oral mucosa, frequent
- Liver, kidneys, skeleton and many other organs

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 144

Glycogen storage disease, type 1b. Characteristic "doll's" face

Main Clinical Features

- Early onset chronic gingivitis soon leading to severe periodontal disease with alveolar bone loss and teeth migration (Figs. 142, 143).
- The oral mucosal lesions present as discrete, deep, irregular, recurrent ulcers that are usually covered by a whitish pseudomembrane. Vaulted palate and recurrent oral infection may occur.
- Characteristic face (doll's face), liver and kidney enlargement, hypoglycaemia, hyperlipidaemia, growth retardation and recurrent bacterial infection are common (Fig. 144).

Diagnosis

- The clinical diagnosis should be confirmed by laboratory tests.
- Haematological and biochemical examination.
- Liver biopsy and histopathological examination.

Differential Diagnosis

- Congenital neutropenia
- Cyclic neutropenia
- Chediak-Higashi syndrome
- Hypophosphatasia and acatalasia

Treatment

- Dental plaque control.
- Topical oral antiseptics.
- Systemic treatment by the paediatrician.

Hurler's Syndrome

Definition

- Hurler's syndrome is a rare, and the most common and severe form, in the group of mucopolysaccharide metabolic disorders.

Etiology

- Genetic. It is inherited as an autosomal recessive trait.
- The basic defect is the deficiency of the enzyme alpha-L-iduronidase resulting in the accumulation and deposition of heparan sulphate and dermatan sulphate within the tissues and their excessive excretion in the urine.

Gingival Involvement

- Common

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 145

Hurler's syndrome. Severe gingival overgrowth completely covering the crowns of the teeth

Other Sites of Involvement

- Oral mucosa and teeth
- Skeletal
- Cornea
- Internal organ (liver, spleen, heart, lungs and others)

Main Clinical Feature

- Gingival overgrowth that may partially or fully cover the crowns of teeth may be present, particularly in the anterior area of the maxilla (Fig. 145). The overgrowth is probably due to mouth breathing in association with microbial dental plaque and the deposition of heparan and dermatan sulphate within the gingiva.
- Other oral manifestations include macrocheilia, macroglossia, open mouth with protruding tongue, numerous impacted teeth, large interdental spaces and teeth dislocation.
- Growth retardation, craniofacial malformations, characteristic facies, scoliosis, stiff joints and chondrodystrophy are common.
- Corneal clouding that frequently leads to blindness, learning disability, cardiac failure, hypertension, lung infection may also occur.
- The disease presents during infancy and often leads to death usually before the patient reaches 10 years of age.

Diagnosis

- The diagnosis is based on clinical and laboratory criteria:
  - Histochemical evaluation of alpha-L-iduronidase.
  - Detection of heparan sulphate and dermatan sulphate in the urine.
  - Radiographic examination.

Differential Diagnosis

- Hereditary gingival fibromatosis
- Mouth-breathing gingivitis
- Other forms of mucopolysaccharidoses
- Alfa-mannosidosis

Treatment

- Good oral hygiene and gingivectomy
- Management of dental problems
- Supportive for the other manifestations

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Focal Palmoplantar and Oral Mucosa Hyperkeratosis Syndrome

Definition
- Focal palmoplantar and oral mucosa hyperkeratosis syndrome, or hyperkeratosis palmoplantaris and attached gingival hyperkeratosis is an unusual genetic disorder characterized by oral mucosal and dermal hyperkeratosis.

Etiology
- Genetic. It is inherited as an autosomal dominant trait.

Gingival Involvement
- Always present, mainly attached gingiva

Other Sites of Involvement
- Oral mucosa (sites subject to mechanical pressure or friction such as hard palate, retromolar pad mucosa, alveolar mucosa, lateral of the tongue)
- Skin (mainly over the weight-bearing and pressure-related areas, such as the soles and palms)
- Nails

Main Clinical Features
- Marked white hyperkeratosis of the attached gingiva, presenting clinically as leukoplakia, is a consistent finding (Fig. 146). There is no inflammation or attachment loss.
- White hyperkeratotic lesions in other mucosal sites are less frequently observed.
- White hyperkeratotic lesions at the weight-bearing and pressure-related areas of the palms and soles are always present (Figs. 147, 148).
- Nail dystrophy and hyperhidrosis may also occur.
- The hyperkeratosis develops early in childhood and the severity increases with age.

Diagnosis
- The diagnosis is mainly based on clinical criteria.
- Biopsy and histopathological examination are also used.

Differential Diagnosis
- Leukoplakia
- White sponge naevus
- Pachyonychia congenita
- Dyskeratosis congenita

Treatment
- Local and systemic aromatic retinoids with partial success

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 146

Focal palmoplantar and oral mucosa hyperkeratosis syndrome. Generalized white hyperkeratosis of the gingiva

FIGURE 147

Focal palmoplantar and oral mucosa hyperkeratosis syndrome. Hyperkeratosis of the palms

FIGURE 148

Focal palmoplantar and oral mucosa hyperkeratosis syndrome. Hyperkeratosis of the sole

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Familial Acanthosis Nigricans

Definition
- Familial or juvenile acanthosis nigricans is an uncommon benign mucocutaneous disorder, characterized by papillary lesions and occasional skin discolouration.

Etiology
- Genetic. It is inherited by an irregular autosomal dominant trait.

Gingival Involvement
- Relatively common
- Non-dental plaque-related lesions

Other Sites of Involvement
- Oral mucosa (10%–25%), mainly on the tongue, palate, lips
- Skin, always (axillae, groins, neck, umbilicus, genitalia, perianal region)

Main Clinical Features
- Gingival enlargement and in particular the interdental papillae may be present. The enlargement is occasionally so severe that it covers the crowns of teeth. Enlarged gingiva often end up looking papillary in appearance (Figs. 149, 150).
- The oral mucosal lesions appear as multiple, small, painless papillomatous growths. The gingival and oral mucosal lesions exhibit normal colour.
- The skin lesions present as painless dark, velvety, multiple small papillary growths (skin tags) (Fig. 151).
- The disorder usually develops during childhood or at puberty.

Diagnosis
- The diagnosis is usually based on clinical criteria.
- Biopsy and histopathological examination of oral and skin lesions are supportive.

Differential Diagnosis
- Hereditary gingival fibromatosis
- Drug-induced gingival hyperplasia
- Malignant acanthosis nigricans
- Endocrine-related acanthosis nigricans
- Cowden's disease
- Darier's disease

Treatment
- Gingivectomy and good oral hygiene for the gingival lesions
- Surgical reconstruction of skin lesion in cases of severe aesthetic problems

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 149

Familial acanthosis nigricans. Localized gingival enlargement with a papillary pattern

FIGURE 150

Familial acanthosis nigricans. Generalized papillary overgrowth of the alveolar mucosa

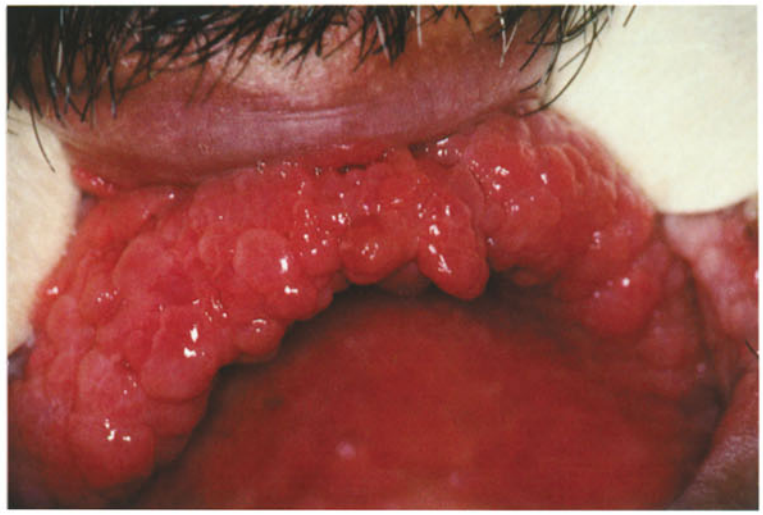


FIGURE 151

Familial acanthosis nigricans. Multiple small papillary growths of the skin (skin tags)

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Darier's Disease

Definition
- Darier's disease or dyskeratosis follicularis is a relatively rare mucocutaneous disorder.

Etiology
- Genetic. It is inherited as an autosomal dominant trait.

Gingival Involvement
- Relatively common
- Non-dental plaque-induced gingival lesions

Other Sites of Involvement
- Oral mucosa (20%–40%)
- Other mucosae (genitalia, rectum, pharynx)
- Skin (always), nails

Main Clinical Features
- The gingival lesions appear as small multiple, whitish confluent papules which may become hypertrophic and are arranged in a reticular pattern or are assuming a cobblestone appearance (Fig. 152).
- Similar lesions appear on the other oral mucosa area, particularly on the palate, tongue and buccal mucosa. The oral lesions usually appear after the cutaneous ones.
- The skin lesions appear as multiple brownish-red, painless, papules that usually coalesce into plaques (Fig. 153). The forehead, ears, scalp, chest and back are more often affected.
- The nails exhibit subungual keratosis and longitudinal ridges and lines.

Diagnosis
- The clinical diagnosis should be confirmed by laboratory tests.
- Biopsy and histopathological examination of oral and skin lesions.

Differential Diagnosis
- Acanthosis nigricans
- Cowden's disease
- Wegener's granulomatosis
- Lichen planus

Treatment
- Good oral hygiene
- Systemic aromatic retinoids

FIGURE 152

Darier's disease. Multiple, small whitish papules on the lower gingiva

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 153

Darier's disease. Multiple papules that coalesce into plaques on the skin

Cowden's Disease

Definition
- Cowden's disease or multiple hamartoma syndrome is an uncommon disorder characterized by a wide spectrum of mucocutaneous lesions usually associated with breast, thyroid, and other organ disorders.

Etiology
- Genetic. It is inherited as an autosomal dominant trait.

Gingival Involvement
- Relatively common
- Non-dental plaque-induced gingival lesions

Other Sites of Involvement
- Oral mucosa, common (lips, tongue, buccal mucosa)
- Skin, common (neck, nasolabial folds, dorsum of the hands and forearms)
- Breast, thyroid gland, common
- Other organs (genitourinary system, gastrointestinal tract, skeletal, eye)

Main Clinical Features
- The gingival lesions appear as multiple, small, smooth, pale or whitish, painless papules or nodules that may be isolated or coalesce in a cobblestone pattern on the free and attached gingiva or the edentulous alveolar mucosa (Figs. 154, 155).
- The oral lesions are similar to those seen on the gingiva.
- The skin lesions appear as multiple papillomatous papules and small nodules that are hair follicle hamartomas (trichilemmomas). Dermal fibromas are also common.
- Several breast and thyroid glands abnormalities including benign and malignant tumours are common.

Diagnosis
- The diagnosis is based on clinical and laboratory criteria.
- Biopsy and histopathological examination is mainly of cutaneous lesions.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 154

Cowden's disease. Multiple, small nodules of the gingiva

FIGURE 155

Cowden's disease. Multiple nodules that coalesce in a cobblestone pattern on the gingiva and alveolar mucosa

Differential Diagnosis

- Focal epithelial hyperplasia
- Acanthosis nigricans
- Tuberous sclerosis
- Multiple endocrine neoplasia syndrome
- Darier's disease
- Lipoid proteinosis

Treatment

- There is no treatment
- Surgical excision of oral and cutaneous lesions only for aesthetic purposes

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Tuberous Sclerosis

Definition
- Tuberous sclerosis of Bourneville-Pringle syndrome, or epiloia, is an uncommon disorder characterized by mucocutaneous angiofibromas, learning disability and seizures.

Etiology
- Genetic. It is inherited as an autosomal dominant trait, with variable expressivity.

Gingival Involvement
- Relatively common (10 %–15 %)
- Non-dental plaque-induced gingival lesions

Other Sites of Involvement
- Oral mucosa, rare
- Skin, always
- Central nervous system
- Others (retinal, heart, kidneys, lungs)

Main Clinical Features
- The gingival lesions are the most characteristic oral lesions and present as multiple, painless, fibrous nodules with smooth surface and normal or whitish colour (Figs. 156, 157). The anterior gingiva is more frequently affected.
- The oral mucosal lesions, although less often, are similar to those seen on the gingiva. Cleft palate and lip, vaulted palate, macroglossia and enamel pits may occur.
- The skin lesions are common and present as multiple small, red to brown nodules (angiofibromas), mainly in the nasolabial folds, and elsewhere in the face (Fig. 158). Periungual fibromas may also seen.
- Learning disability, seizures, and neural hamartomas are common.
- Cardiac rhabdomyoma, multiple retinal hamartomata, lung and renal abnormalities may also occur.

Diagnosis
- The diagnosis is based on clinical and laboratory criteria.
- Biopsy and histopathological examination of cutaneous and oral lesions.
- Brain CT scan.
- Electroencephalography.

Differential Diagnosis
- Cowden's disease
- Neurofibromatosis
- Lipoid proteinosis
- Acanthosis nigricans
- Focal dermal hypoplasia syndrome

Treatment
- Symptomatic
- Surgical excision of gingival lesions only for aesthetic purposes

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 156

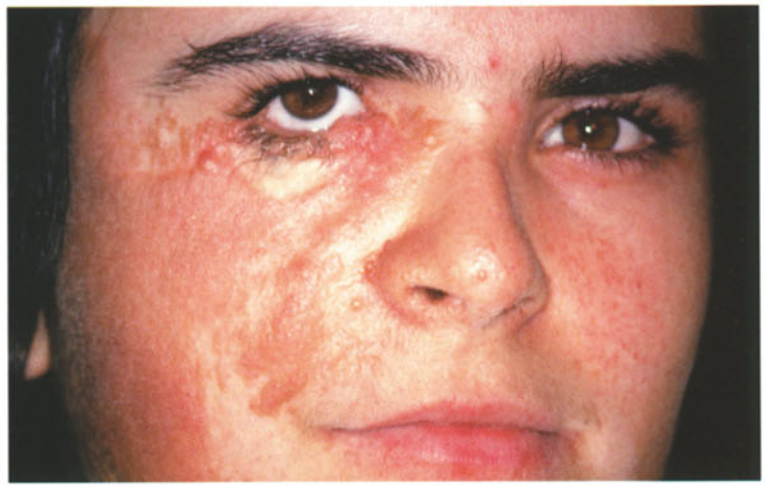
Tuberous sclerosis.
Solitary nodule on the gingiva

FIGURE 157

Tuberous sclerosis.
Multiple gingival nodules

FIGURE 158

Tuberous sclerosis.
Characteristic angiofibromas
on the skin of the face



For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Gardner's Syndrome

Definition
- Gardner's syndrome is a rare disorder in the group of familial colorectal polyposis syndromes.

Etiology
- Genetic. It is inherited as an autosomal dominant trait.

Alveolar Bone Involvement
- Relatively common.
- The gingiva are normal.

Other Sites of Involvement
- Bones (skull, calvaria)
- Skin
- Gastrointestinal tract

Main Clinical Features
- The alveolar bone lesions are characterized by multiple osteomas that present as painless, hard masses covered by normal attached gingiva and alveolar mucosa (Fig. 159). Central osteomas in other sites of the jaws, multiple odontomas, multiple impacted supernumerary teeth and rarely benign oral soft tissue tumours may occur. It is important for dentists to diagnose the oral lesions of the syndrome as early as possible and to refer the patient to a gastroenterologist.
- Multiple osteomas of the skull, paranasal sinuses and calvaria may also develop.
- Gastrointestinal manifestations are common and appear as multiple polyps of the colon and rectum that are characterized by a high rate of malignant transformation into adenocarcinoma.
- Skin lesions are epidermoid cysts, multiple fibromas and hyperpigmentation.

Diagnosis
- The clinical diagnosis should be confirmed by laboratory tests.
- Radiographic examination of the jaws, skull and intestinal tract.
- Biopsy and histopathological examination of tumours and polyps.

Differential Diagnosis
- Isolated osteomas and exostoses (tori)
- Odontogenic tumours
- Peutz-Jeghers syndrome
- Cowden's syndrome

Treatment
- Osteomas do not require excision unless they cause symptoms or cosmetic problems.
- The intestinal polyps must be under observation by the specialist.

Sturge-Weber Angiomatosis

Definition
- Sturge-Weber angiomatosis or encephalotrigeminal angiomatosis is a rare sporadic congenital hamartomatous vascular disorder typically involving areas innervated by the trigeminal nerve.

Etiology
- Obscure; developmental abnormality

Gingival Involvement
- Relatively common

Other Sites of Involvement
- Oral mucosa
- Skin
- Eyes, central nervous system

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 159

Gardner's syndrome. Multiple osteomas of the alveolar bone

FIGURE 160

Sturge-Weber angiomatosis. Unilateral gingival haemangiomas

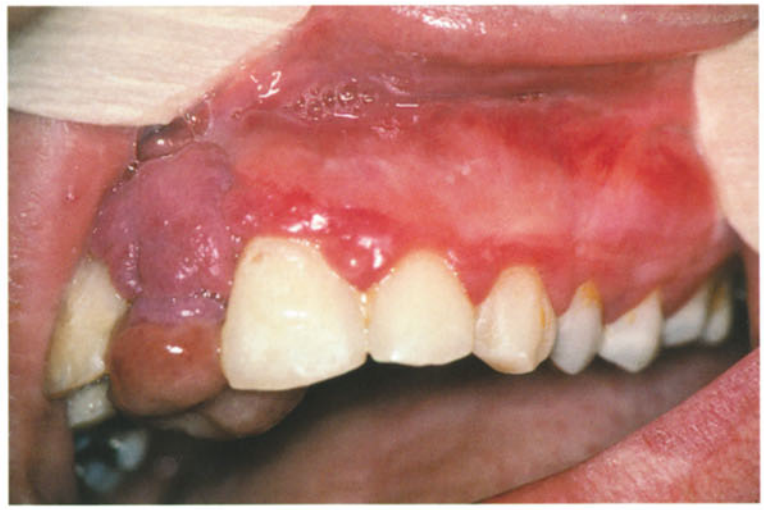


FIGURE 161

Sturge-Weber angiomatosis. Unilateral gingival haemangiomas

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 162

Sturge-Weber angiomatosis. Unilateral facial haemangiomas

Main Clinical Features

- Typically unilateral haemangiomas, usually capillary, on the attached gingiva and the alveolar mucosa may be seen occasionally leading to gingival hyperplasia and premature or rarely delayed eruption (Figs. 160, 161). The lesions have a bright red or purple colour and are usually flat. Dentists must be careful during tooth extraction and periodontal surgery so as to avoid bleeding complications.
- Unilateral oral haemangiomas on the buccal mucosa, tongue, and lips may occur.
- Unilateral facial haemangioma is the most characteristic and consistent feature, it is evident at birth and characteristically it is confined to the area supplied by the trigeminal nerve (Fig. 162)
- Haemangiomas of the leptomeninges, brain calcification, epilepsy, ocular disorders may also be present.

Diagnosis

- The diagnosis is usually based on clinical criteria.
- Histopathological examinations
- CT scan.

Differential Diagnosis

- Solitary haemangioma
- Klippel-Trenaunay-Weber syndrome

Treatment

- Laser therapy is recommended.

Klippel-Trenaunay-Weber Syndrome

Definition

- Klippel-Trenaunay-Weber syndrome is an uncommon dysplastic vascular disorder.

Etiology

- Dysplastic malformation

Gingival Involvement

- Common

Other Sites of Involvement

- Oral mucosa
- Skin
- Eyes
- Bone and soft tissues of extremities

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 163

Klippel-Trenaunay-Weber syndrome. Gingival haemangiomas leading to gingival overgrowth

FIGURE 164

Klippel-Trenaunay-Weber syndrome. Scleral pigmentation

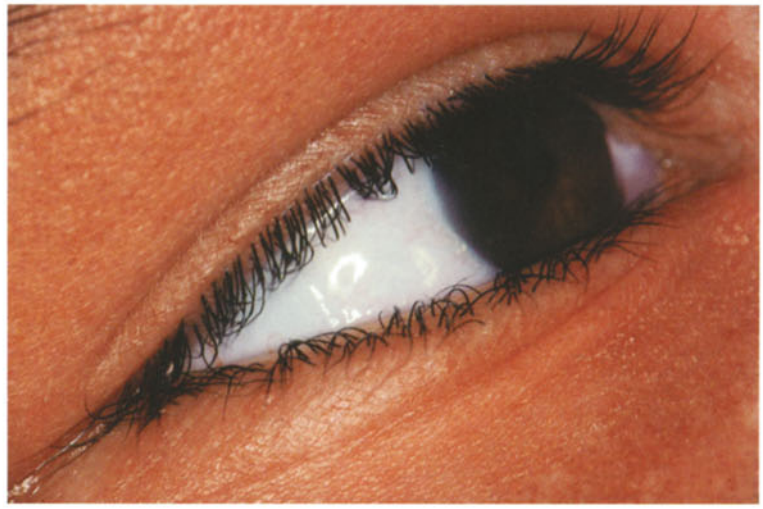


FIGURE 165

Klippel-Trenaunay-Weber syndrome. Haemangiomas of the leg

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Main Clinical Features

- The gingival lesions appear as capillary or cavernous haemangiomas occasionally leading to gingival overgrowth that may cover the crown of the teeth (Fig. 163). Premature tooth eruption and alveolar bone overgrowth may occur.
- The oral mucosal lesions are also haemangiomas, particularly on the hard and soft palate.
- Multiple facial haemangiomas, ocular disorders (scleral pigmentation, glaucoma, cataract, iris heterochromia), vascular masses that involve soft tissue and bone leading to asymmetrical enlargement of the extremities, haemangiomas in internal organs are common (Figs. 164, 165).

Diagnosis

- The diagnosis is based on clinical criteria.

Differential Diagnosis

- Isolated haemangiomas
- Sturge-Weber syndrome
- Maffucci's syndrome

Treatment

- Palliative.
- Laser may improve skin lesions.

Hereditary Epidermolysis Bullosa

Definition

- Hereditary epidermolysis bullosa is a rare chronic, recurrent, mechanobullous group of genetic mucocutaneous disorders.

Etiology

- Genetic. The exact etiology and pathogenesis are obscure.

Classification

- Three major forms and numerous subtypes are recognized:
  - The intraepidermal, type I (characterized by a non-scarring pattern and inherited as an autosomal and X-linked trait.
  - The junctional, type II (characterized by skin atrophy and inherited as an autosomal recessive trait).
  - The dermal, type III (characterized by skin atrophy and scarring and inherited as an autosomal dominant or recessive trait).

Gingival Involvement

- Common, particularly of types II and III
- Non-dental plaque-induced gingival lesions, occasionally associated with attachment loss

Other Sites of Involvement

- Oral mucosa (tongue, buccal mucosa, palate, lips)
- Other mucosae (eyes, larynx, oesophagus, genitals)
- Skin, always (hands, feet, fingers, toes, knee, elbows, scale, nails), mainly in areas of mechanical friction

Main Clinical Features

- The gingival manifestations usually follow the skin lesions and may appear in the form of desquamative gingivitis or as localized or widespread bullae formation that rupture soon leaving painful erosions (Figs. 166, 167). Gingival recession and periodontal destruction may also occur in severe cases.
- The oral mucosal lesions appear as bullae formation that soon rapture, leaving painful erosions or ulcerations that may occasionally lead to scarring. Microstomia, epithelial atrophy, elimination of tongue movement, elimination of vestibular and buccal sulci, lobulated hyperplastic nodules, pitted enamel and teeth caries may occur (Fig. 168).

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 166

Hereditary epidermolysis bullosa, type I. Solitary bulla formation on the alveolar mucosa

FIGURE 167

Hereditary epidermolysis bullosa, type II. Gingival erosions and enlargement

FIGURE 168

Hereditary epidermolysis bullosa, type III. Pitted enamel and tooth caries

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 169

Hereditary epidermolysis bullosa, type II. Bulla formation and scarring of the skin

FIGURE 170

Hereditary epidermolysis bullosa, type III. Scarring and deformities of the hand

FIGURE 171

Hereditary epidermolysis bullosa, type II. Nail dystrophy

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

- The skin manifestations are characterized by bulla formation, ulcerations, scarring, deformities, milia, nail dystrophy and several others (Figs. 169–171).
- The clinical manifestations of the disorder present early in life (infants and children) and the severity of the skin and mucosal lesions depend on the form of the disease.

Diagnosis

- The diagnosis is based on clinical and laboratory criteria.
- Biopsy and histopathological examination.
- Immunofluorescence test.
- Electron microscopic examination.

Differential Diagnosis

- Cicatricial pemphigoid
- Bullous pemphigoid
- Pemphigus vulgaris
- Linear IgA disease

Treatment

- Avoid pressure on the gingiva and oral mucosa.
- Good oral hygiene.
- Topical corticosteroid application.
- Systemic corticosteroid in severe lesions.

Chondroectodermal Dysplasia

Definition

- Chondroectodermal dysplasia or Ellis-van Creveld syndrome is an uncommon disorder of the osteochondrodysplasia group of defects.

Etiology

- Genetic. It is inherited as an autosomal recessive trait.

Gingival Involvement

- Common

Other Sites of Involvement

- Teeth, skeleton, eyes, heart, central nervous system
- Hair and nails

Main Clinical Features

- Fusion of the middle portion of the lower or upper lip to the gingiva, leading to the disappearance of the mucolabial fold and multiple frenulal is the most characteristic oral manifestation (Figs. 172, 173). Oligodontia, and small teeth with conical crowns may occur.
- Chondrodysplasia of the long bones, bilateral polydactyly, shortened extremities, thin and sparse hair, dystrophic nails, heart defects and genital abnormalities are common (Fig. 174).

Diagnosis

- The diagnosis is mainly based on clinical criteria.
- Radiographic examination.

Differential Diagnosis

- Orofaciodigital syndrome
- Other chondrodysplasia syndromes
- Hereditary epidermolysis bullosa, type III

Treatment

- Supportive.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 172

Chondroectodermal dysplasia. Multiple frenulum and disappearance of the mucolabial fold

FIGURE 173

Chondrolectodermal dysplasia. Multiple gingivolabial frenulum

FIGURE 174

Chondroectodermal dysplasia. Polydactyly and nail dystrophy

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Focal Dermal Hypoplasia Syndrome

Definition
- Focal dermal hypoplasia syndrome or Goltz-Gorlin syndrome is an uncommon disorder characterized by multiple abnormalities.

Etiology
- Genetic. It is inherited as an X-linked dominant trait. However, most of the reported patients are sporadic cases.

Gingival Involvement
- Rare

Other Sites of Involvement
- Oral mucosa (tongue, palate, buccal mucosa, lips) and teeth
- Skin, hair and nails
- Musculoskeletal and central nervous systems, eyes

Main Clinical Features
- The gingival lesions are papillomas (Fig. 175).
- Multiple papillomas on the oral mucosa are present in about 50 % of the cases. Oligodontia, hypodontia, enamel dysplasia and malocclusion, oral clefts are common.
- Skin manifestations include atrophic linear lesions, atrophic and hyperpigmented plaques, telangiectasias and soft yellowish nodules (Fig. 176). Sparse hair and dystrophic nails may be observed.
- Syndactyly, brachydactyly, oligodactyly or polydactyly, clinodactyly extremity asymmetry, scoliosis, and microcephaly may occur.
- Eye anomalies, learning disability are less common findings.

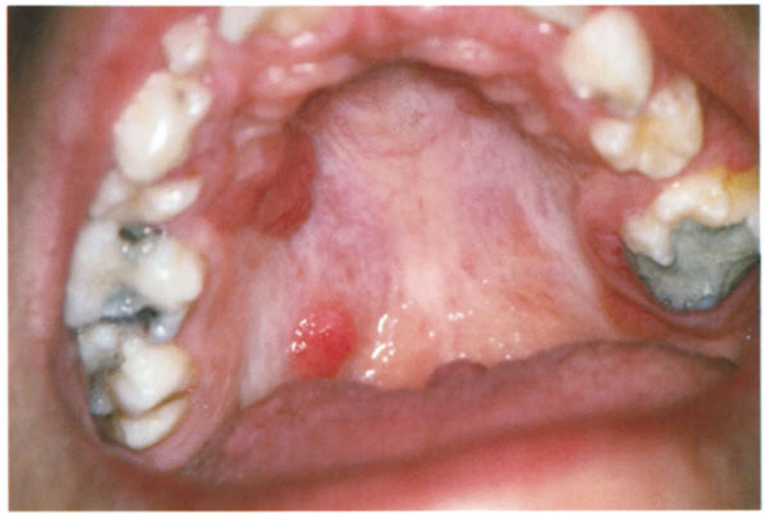
Diagnosis
- The diagnosis is mainly based on clinical criteria.
- Biopsy and histopathological examination of skin and oral lesions.

Differential Diagnosis
- Multiple oral papillomas
- Multiple oral condylomas acuminata
- Focal epithelial hyperplasia
- Incontinentia pigmenti

Treatment
- Surgical excision of oral papillomas
- Supportive for the skin and other lesions

FIGURE 175

Focal dermal hypoplasia syndrome. Multiple papillomas on the gingiva and the palate



For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 176

Focal dermal hypoplasia syndrome. Skin atrophy

Orofaciodigital Syndrome Type I

Definition
- Orofaciodigital syndrome, type I is an uncommon disorder in the group of orofaciodigital syndromes.

Etiology
- Genetic. It is inherited as an X-linked dominant trait, limited to females and fatal in males.

Gingival Involvement
- Common

Other Sites of Involvement
- Oral mucosa, teeth
- Facies, digits
- Skin

Main Clinical Features
- The gingival involvement characterized by multiple hyperplastic fibrous bands traversing the gingivolabial and gingivobuccal fold, hypertrophy and shortening of the frenulum of the lips and tongue (Figs. 177, 178). These may result in hyperplastic gingivitis and gingival recession.
- The oral mucosal lesions include multilobed or bifid tongue, with multiple hamartomas, lips and palate clefts, supernumerary teeth, teeth malposition and missing teeth, hypoplastic mandible.
- Frontal bossing, ocular hypertelorism, short upper lip, broad nasal root, asymmetry of the nostrils, polydactyly, brachydactyly, clinodactyly and syndactyly are common.
- The skin manifestations include xeroderma, milia, alopecia and sparse hair.
- Learning disability and polycystic kidneys may occasionally occur.

Diagnosis
- The diagnosis is based on clinical criteria.

Differential Diagnosis
- Orofaciodigital syndrome type II
- Chondroectodermal dysplasia
- Oculodentodigital syndrome

Treatment
- Surgical reconstruction of oral frenulum and gingivolabial and gingivobuccal folds
- Symptomatic for the rest of lesions

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 177

Orofaciodigital syndrome, type I. Multiple gingivolabial frenulum

FIGURE 178

Orofaciodigital syndrome, type I. Gingivitis and gingival hyperplasia

For Personal Use Only
Library of School of Dentistry, TUMS

Ehlers-Danlos Syndrome

Definition
- Ehlers-Danlos syndrome includes a group of connective tissue disorders that are characterized by defects in collagen synthesis.

Etiology
- Genetic. It is mainly inherited as an autosomal dominant trait.

Classification
- On the basis of clinical features and the mode of inheritance, ten types of the disorders are recognized.

Periodontal Involvement
- Relatively common. Mainly types III, IV and VIII have an increased susceptibility to periodontitis.
- Possibly multigene disorder influenced by dental plaque level.
- Periodontitis associated with genetic disorder.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 179

Ehlers-Danlos syndrome. Generalized periodontitis

Other Sites of Involvement

- Oral mucosa
- Skin
- Joints
- Heart

Main Clinical Features

- Periodontal lesions usually appear in the form of severe generalized aggressive periodontitis with clinical attachment loss and bone destruction, leading to premature loss of permanent teeth (Fig. 179). Gingival bleeding is also common.
- The oral mucosa manifestations include thin and fragile mucosa, prolonged bleeding on minor trauma and decreased wound healing. About 50 % of patients have the ability to touch their nose with the tip of the tongue, compared with 10 % of normal people. Temporomandibular joint hypermobility, multiple pulp stones, enamel, dentin and cementum defects may occur.
- The skin manifestations include thin and fragile skin, hypermobility, haematomas, scarring formation, especially over the knees, elbows and chin.
- Joint hypermobility, cardiovascular and ocular abnormalities may occur, especially mitral valve prolapse.

Diagnosis

- The diagnosis is based on clinical criteria.

Differential Diagnosis

- Aggressive periodontitis associated with other genetic disorders
- Marfan's syndrome
- Cutis laxa

Treatment

- Dental plaque control
- Symptomatic

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Hereditary Haemorrhagic Telangiectasia

Definition
- Hereditary haemorrhagic telangiectasia, or Osler-Rendu-Weber disease, is a relatively rare disorder characterized by permanent small dilations of vessels.

Etiology
- Genetic. It is inherited as an autosomal dominant trait.

Gingival Involvement
- Rare

Other Sites of Involvement
- Oral mucosa (tongue, lips, buccal mucosa, palate)
- Other mucosae (nasal, oesophagus, genital)
- Skin
- Internal organ (liver, spleen, gastrointestinal tract, lungs)

Main Clinical Features
- The gingival lesions appear as small (usually 1–3 mm in size), localized or multiple, bright red macules or nodules that characteristically disappear when pressure is applied (Figs. 180, 181). Gingival recurrent bleeding, usually during or after tooth brushing, is frequent.
- The oral mucosal manifestations are characterized by multiple telangiectasias and rarely bleeding (Fig. 182).
- Persistent recurrent nose bleeding is usually the first and common clinical sign of the disease.
- Multiple telangiectasias on the skin and gastrointestinal tract are common.
- Liver and spleen vascular abnormalities may be less frequent.
- The disease usually manifests during the second or third decade of life.

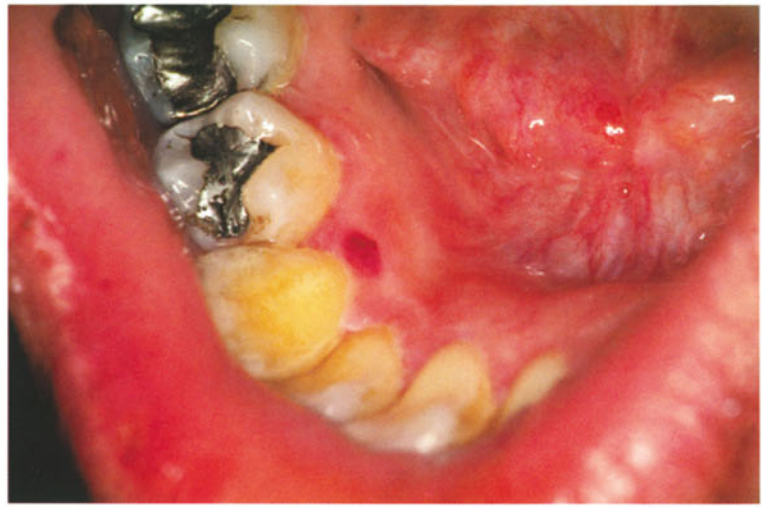
Diagnosis
- The diagnosis is mainly based on clinical criteria.
- Biopsy and histopathological examination.

Differential Diagnosis
- CREST syndrome
- Fabry's disease
- Maffucci's syndrome

Treatment
- Usually no treatment is required for oral lesions.
- If the lesions often bleed, surgical excision or laser therapy is suggested.

FIGURE 180

Hereditary haemorrhagic telangiectasia. Solitary red lesion on the gingiva



For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 181

Hereditary haemorrhagic telangiectasia. Solitary red lesion on the alveolar mucosa

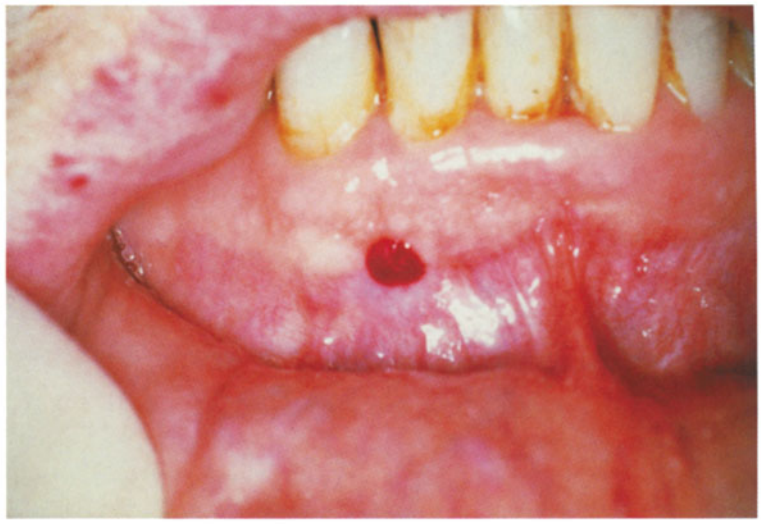


FIGURE 182

Hereditary haemorrhagic telangiectasia. Multiple red lesions on the tongue

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Cystic Fibrosis

Definition

- Cystic fibrosis is a relatively common, multisystemic, life-threatening disorder characterized by a dysfunction of the exocrine glands.

Etiology

- Genetic. It is inherited as an autosomal recessive trait. The pathological gene known as the *CFTR* gene, is located on the long arm of chromosome 7, and causes a disturbance in chloride ion transport across the cell membrane.

Gingival Involvement

- Very rare
- Dental plaque-induced gingivitis associated with xerostomia

Other Sites of Involvement

- Oral mucosa (lips), relatively common
- Pulmonary and gastrointestinal tracts
- Liver, pancreas
- Sweat glands

Main Clinical Features

- The gingival lesions are uncommon and present as a non-specific diffuse erythema on both free and attached gingiva (Fig. 183). The gingival lesions may be due to oral dryness and the dental plaque.
- The oral manifestations include lip enlargement, erythema of the oral mucosa, and occasionally mild xerostomia.
- Chronic coughing, pulmonary infections, shortness of breath, bronchitis, haemoptysis are common.
- Malabsorption, bulky, greasy stools, and failure to gain weight even with adequate food intake are common.
- Liver and pancreatic insufficiency, sweating with salty taste are also common.
- The disease starts early in infancy.

Diagnosis

- The clinical diagnosis should be confirmed by laboratory tests.
- Biopsy and histopathological examination of the lip.
- Elevated chloride, sodium and potassium levels in sweat.
- Absence of pancreatic enzymes in the intestinal fluid.

FIGURE 183

Cystic fibrosis. Gingivitis

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Differential Diagnosis
- Gingivitis associated with dental plaque only
- Lipoid proteinosis
- Cheilitis glandularis and granulomatosa

Treatment
- Dental plaque control for the gingival lesions.
- Palliative high-calorie diet indicated for the systemic manifestations.

Down's Syndrome

Definition
- Down's syndrome, or trisomy 21, is a common disorder with variable clinical manifestations. The incidence in the general population is 1 in 600–800 live births.

Etiology
- Chromosomal disorder of chromosome 21.
- Most patients with Down's syndrome exhibit full trisomy 21. Few cases are mosaic and fewer are the result of translocation.

Periodontal Involvement
- Common
- Dental plaque-induced gingival lesions and frequently periodontitis associated with genetic disorders

Other Sites of Involvement
- Oral cavity, common
- Craniofacies, thorax, abdomen, pelvis, hands and feet

Main Clinical Features
- Clinically the gingiva are red, swollen and bleed easily. (Fig. 184). Gingival hyperplasia may occur due to chronic mouth breathing, poor oral hygiene and other local irritating factors. The gingivitis frequently progresses to generalized early periodontitis, which commences in the deciduous dentition and continues into the permanent dentition. Bone loss, mobility and migration of the teeth are common. All these predispose to the premature loss of teeth.
- Macroglossia, fissured and geographic tongue, high-arched palate, cleft palate, delayed tooth eruption and hypoplastic teeth are the most frequent oral lesions.
- Follicular hyperkeratosis, facial flushing, alopecia areata, and dry skin are common.
- Polydactyly, syndactyly, clinodactyly, hyperextensibility of joints, other skeletal abnormalities, congenital heart anomalies, hormonal disturbances, learning disability, mongoloid slanting of the eyes, short ears, and flat face are common findings.

Diagnosis
- The diagnosis is usually based on clinical criteria.
- Chromosomal analysis.

Differential Diagnosis
- Cyclic neutropenia
- Agranulocytosis
- Leukaemias
- Glycogen storage disease type 1b

Treatment
- Good oral hygiene and plaque control reduce the gingival and periodontal lesions.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 184

Down's syndrome. Severe gingivitis

Reading List

Aldred MI, Bartold PM (2000) Genetic disorders of the gingivae and periodontium. Periodontol 2000 18:7–20

Apaydin A (1995) Ehlers-Danlos syndrome (type VIII). J Nihon Univ Sch Dent 37:214–217

Austin GB, Quart AM, Novak B (1981) Hereditary hemorrhagic telangiectasia with oral manifestations. Oral Surg Oral Med Oral Pathol 51:245–251

Baab DA, Page RC, Ebersole JL, et al (1986) Laboratory studies of a family manifesting premature exfoliation of deciduous teeth. J Clin Periodontol 13:677–683

Bakaeen G, Scully C (1991) Hereditary gingival fibromatosis in a family with the Zimmermann-Laband syndrome. J Oral Pathol Med 20:457–459

Barabas GM (1969) Ehlers-Danlos syndrome. Abnormalities of the enamel, dentine, cementum and the dental pulp: a histologic examination of 24 teeth from 6 patients. Br Dent J 126:509–515

Barrett AP, Buckley DJ, Katelaris CH (1990) Oral complications in type 1b glycogen storage disease. Oral Surg Oral Med Oral Pathol 69:174–176

Bazopoulou-Kyrkanidou E, Papagianoulis L, Papanicolaou S, Mavrou A (1990) Laband syndrome: a case report. J Oral Pathol Med 19:385–387

Bazopoulou E, Laskaris G, Katsabas A, Papanikolaou S (1991) Familial benign acanthosis nigricans with predominant early oral manifestations. Clin Genet 40:160

Beumer J, Trowbridge HO, Silverman S Jr, Eisenberg E (1973) Childhood hypophosphatasia and the premature loss of teeth: a clinical and laboratory study of seven cases. Oral Surg Oral Med Oral Pathol 35:631–640

Bozzo L, Almeida O, Scully C, Aldred MJ (1994) Hereditary gingival fibromatosis. Oral Surg Oral Med Oral Pathol 78:452–454

Breton P, Cambazard M, Rougier M, et al (1988) Manifestations maxillofaciales de la maladie de Cowden. A propos de deux observations. Rev Stomatol Chir Maxillofac 89:87–91

Camisa C, Bikowski JB, McDonald SG (1984) Cowden's disease: association with squamous cell carcinoma of the tongue and perianal basal cell carcinoma. Arch Dermatol 120:677–678

Charon JA, Mergenhagen SE, Gallin JI (1985) Gingivitis and oral ulceration in patients with neutrophil dysfunction. J Oral Pathol 14:150–155

Ellis RWB, Andrew JD (1962) Chondroectodermal dysplasia. J Bone Surg 44B:626–636

Everett FG, Hahn CR (1976) Hereditary hemorrhagic telangiectasia with gingival lesions: review and case reports. J Periodontol 47:295–298

Fenerly A, Laskaris G (1984) Oral manifestation of Darier's disease. Hell Stomatol Rev 28:40–46

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Ferris T, Lamey PJ, Rennie JS (1990) Darier's disease: oral features and genetic aspects. Br Dent J 168:71–73
Flint SR, Keith O, Scully C (1988) Hereditary hemorrhagic telangiectasia: family study and review. Oral Surg Oral Med Oral Pathol 66:440–444
Gardner DG (1971) The oral manifestations of Hurler's syndrome. Oral Surg Oral Med Oral Pathol 32:46–57
Goltz EW, Henderson RR, Hitch JM, Ott JE (1970) Focal dermal hypoplasia syndrome: a review of the literature review. Arch Dermatol 101:1–11
Gomez MR (1988) Tuberous sclerosis. Raven Press, New York
Gorlin RT, Cohen MM, Levin LS (1990) Syndromes of the head and neck. Oxford University Press, New York
Hall EH, Terezhalmy GT (1983) Focal dermal hypoplasia syndrome: case report and literature review. J Am Acad Dermatol 9:443–451
Hamilton RE, Giamsanti JS (1974) Chédiak-Higashi syndrome: report of a case and review of the literature. Oral Surg Oral Med Oral Pathol 37:754–761
Hanake E (1979) The Papillon-Lefèvre syndrome: keratosis palmoplantaris with periodontopathy. Report of a case and review of the cases in the literature. Hum Genetics 51:1–35
Hara T, Mizumo Y, Okubo K, Veda K (1987) Glycogenesis type 1b and periodontitis. J Pediatr 111:952
Harned RK, Buck JL, Olmsted WW et al (1991) Extracolonic manifestations of the familial adenomatous polyposis syndromes. Am J Roentgenol 156:481–485
Hartsfield JK, Kousseff BG (1990) Phenotype overlap of Ehlers-Danlos syndrome types IV and VIII. Am J Med Genet 37:465–470
Hochberg MS, Vazquez-Santiago IA, Sher M (1993) Epidermolysis bullosa. Oral Surg Oral Med Oral Pathol 75:54–57
Hopwood JJ, Morris CP (1990) The mucopolysaccharidoses: diagnosis, molecular genetics and treatment. Mol Biol Med 7:381–404
Kinane D (1999) Blood and lymphoreticular disorders. Periodontol 2000 21:84–93
Kinane D (1999) Periodontitis modified by systemic factors. Ann Periodontol 4:54–63
Krajewska IA, Moore L, Brown JH (1992) White sponge nevus presenting in the esophagus: case report and literature review. Pathology 24:112–115
Laskaris G, Vareltzidis A, Avgerinou G (1980) Focal palmoplantar and oral mucosa hyperkeratosis syndrome: a report concerning five members of a family. Oral Surg Oral Med Oral Pathol 50:250–253
Laskaris G (2000) Color atlas of oral diseases in children and adolescents. Georg Thieme Verlag, Stuttgart
Lipp MJ, Lubit EC (1990) The oral-facial-digital syndrome: case report of a mother and daughter. Cleft Palate 27:311–316
Lygidakis NA, Lindnebaum RH (1989) Oral fibromas in tuberous sclerosis. Oral Surg Oral Med Oral Pathol 68:725–728
Mackler SB, Shoulars HW, Burker EJ (1972) Tuberous sclerosis with gingival lesions. Oral Surg Oral Med Oral Pathol 34:619–624
Orner G (1976) Periodontal disease among children with Down's syndrome and their siblings. J Dent Res 55:778–782
Page RC, Beck JD (1997) Risk assessment for periodontal diseases. Int Dent J 47:61–87
Pindborg JJ, Gorlin RJ (1962) Oral changes in acanthosis nigricans: juvenile type. Acta Derm Venereol (Stockh) 42:63–71
Salapata J, Laskaris G, Drogari E et al (1995) Oral manifestations in glycogen storage disease type 1b. J Oral Pathol Med 24:136–139
Salinas CF, Pai GS, Vera CL et al (1991) Variability of expression of the orofacial digital syndrome type I in black females: six cases. Am J Med Genet 38:574–582
Saxén L, Aula S (1982) Periodontal bone loss in patients with Down's syndrome: a follow-up study. J Periodontol 53:158–162
Sciubba JJ, Brown AM (1977) Oral-facial manifestations of Klippel-Trenaunay-Weber syndrome. Oral Surg Oral Med Oral Pathol 43:227–232
Smith DB, Strand JA, Carter PL (1988) Gardner's syndrome: current concepts and use of a new genetic marker. Mil Med 153:289–293
Steiner M, Could AR, Graves SM, Kuerschner TW (1987) Klippel-Trenaunay-Weber syndrome. Oral Surg Oral Med Oral Pathol 63:208–215

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Stewart RE, Hollister DW, Rimoin DL (A new variant of Ehlers-Danlos syndrome: an autosomal dominant disorder of fragile skin, abnormal scarring and generalized periodontitis. Birth Defects 13:85–93

Swart JG, Lekkas C, Allard RHB (1985) Oral manifestations in Cowden's syndrome. Oral Surg Oral Med Oral Pathol 59:264–268

Tasjian D, Jarrat M (1984) Familial acanthosis nigricans. Arch Dermatol 120:1351–1354

Temple TR, Kimball HR, Kakehashi S, Amen CR (1972) Host factors in periodontal disease: periodontal manifestations of Chédiak-Higashi syndrome. J Periodont Res 10 [Suppl]:26–27

Tumber-Saini SK, Habbick BF, Oles AM et al (1992) The role of saliva in aggregation and adherence of *Pseudomonas aeruginosa* in patients with cystic fibrosis. J Oral Pathol Med 21:299–304

Uitto J, Christiano AM (1993) Inherited epidermolysis bullosa: clinical features, molecular genetics, and pathoetiologic mechanisms. Dermatol Clin 11:549–563

Vasilopoulou A, Laskaris G (1989) Papillon-Lefèvre syndrome: report of two brothers. J Dent Child, September–October :388–391

Watanabe H, Umeda M, Seki T, Ishikawa I (1993) Clinical and laboratory studies of severe periodontal disease in an adolescent associated with hypophosphatasia: a case report. J Periodontol 64:174–180

Winter GB, Geddes M (1967) Oral manifestations of chondroectodermal dysplasia (Ellis-van Creveld syndrome). Br Dent J 122:103–107

Wright JT (1991) Oral soft tissues in hereditary epidermolysis bullosa. Oral Surg Oral Med Oral Pathol 71:440–446

Zachariades N (1987) Gardner's syndrome: report of a family. J Oral Maxillofac Surg 45:438–440

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

CHAPTER 17

Bacterial Infections

Tuberculosis

Definition
- Tuberculosis is a chronic granulomatous infectious disease.

Etiology
- *Mycobacterium tuberculosis*
- Non-dental plaque-induced gingival lesions

Gingival Involvement
- Rare

Other Involvement
- Oral mucosa (tongue, palate, uvula, buccal, lips), rare
- Lungs, almost always

Main Clinical Features
- The gingival lesions may present as chronic, irregular, usually painless, ulcers with vegetating red surface or covered by a grey-yellowish exudate (Figs. 185, 186). Vegetating diffuse gingival enlargement may also occur.
- The oral mucosal lesions manifest as an irregular, vegetating ulcer with a thin border. The surrounding tissue is slightly indurated with inflammation. Regional lymphadenopathy accompanies the oral lesions.
- Tuberculous osteomyelitis of the jaws may rarely occur.
- Gingival and oral manifestations usually represent secondary infection from the initial pulmonary infection and the oral prevalence varies from 0.5% to 1.5%.
- Rarely, oral tuberculosis may be the only manifestation of otherwise silent tuberculosis.
- Low-grade fever, anorexia, malaise, cough, night sweats are early and common symptoms. Chest pain and haemoptysis are also common.

Diagnosis
- The clinical diagnosis should be confirmed by laboratory tests.
- Biopsy and histopathological examination of oral lesions.
- Chest radiographs.
- Mycobacterial examination or culture of infected sputum and tissues.
- Mantoux tuberculin skin test.

Differential Diagnosis
- Traumatic ulcer
- Squamous cell carcinoma
- Primary and secondary syphilis
- Major aphthous ulcer
- Non-Hodgkin's lymphoma
- Wegener's granulomatosis
- Pyostomatitis vegetans

Treatment
- Antituberculous multiple drug regiments that must be left to the specialist

For Personal Use Only
Library of School of Dentistry, TUMS

G. Laskaris et al., *Periodontal Manifestations of Local and Systemic Diseases*
© Springer-Verlag Berlin Heidelberg 2003

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 185

Tuberculosis.
Small ulcer on the upper gingiva

FIGURE 186

Tuberculosis.
Vegetating, irregular ulcer
on the lower gingiva

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Syphilis

Definition
- Syphilis is a sexually transmitted disease involving several organs and systems.

Etiology
- *Treponema pallidum*

Classification
- Congenital.
- Acquired. Acquired syphilis develops in three stages:
  - Primary.
  - Secondary.
  - Tertiary.

Gingival Involvement
- Rare
- Non-dental plaque-induced gingival lesions

Other Involvement
- Oral mucosa, common. Approximately 5%–10% of the primary and over of 40% of secondary syphilis appears on the oral cavity.
- Genitalia, very common.
- Skin, common (secondary and tertiary stages).

Main Clinical Features
- Primary syphilis is characterized by the development of chancre at the site of inoculation of *treponema pallidum*. Chancre begins as an inflammatory papule that ulcerates to form a painless round ulcer with a raised border and indurated base. Chancre on the gingiva is extremely rare (**Figs. 187, 188**). The lips, tongue, soft palate, and tonsils are the most common oral sites affected. Regional lymph node enlargement is a constant finding. Direct orogenital contact is necessary for the development of an oral chancre.
- Secondary syphilis begins 6–8 weeks after the appearance of the chancre. Oral lesions are common in this stage and are mainly characterized by the appearance of mucous patches, which are painless, slightly raised, oval or round erosions or superficial ulcers covered by a greyish-white membrane. Gingival involvement is relatively rare (**Fig. 189**). The tongue, buccal mucosa, palate, tonsils and lips are most frequently affected. The classic signs and symptoms of secondary syphilis are headache, malaise, fever, generalized painless lymph node enlargement and maculopapular skin eruption.
- Tertiary syphilis (late syphilis) develops in about 30% of untreated patients, and is characterized by multisystem involvement and broad spectrum of manifestations. The gumma is the most characteristic oral lesion of this stage, and the hard palate is the most common site of involvement, followed by tongue and lips. Gingiva is not involved.

Diagnosis
- The clinical diagnosis of syphilis should be confirmed by laboratory tests.
- Serological tests.
- Darkfield microscopy.

Differential Diagnosis
- Herpetic oral lesions
- Traumatic oral lesions
- Aphthous ulcer
- Candidiasis
- Erythema multiforme
- Infectious mononucleosis

Treatment
- Penicillin or erythromycin, but it must be left to the physician

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 187

Primary syphilis.
Small chancre on the border of attached gingiva and alveolar mucosa

FIGURE 188

Primary syphilis.
Large chancre on the edentulous alveolar mucosa

FIGURE 189

Secondary syphilis.
Mucous patches on the lower gingiva

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Actinomycosis

Definition
- Actinomycosis is a chronic granulomatous infectious disease.

Etiology
- Mainly *Actinomyces israelii* and rarely other species

Gingival Involvement
- Rare
- Non-dental plaque-induced gingival lesions

Other Involvement
- Jaws
- Oral mucosa
- Cervical lymph nodes

Main Clinical Features
- Oral manifestations are part of the cervicofacial actinomycosis.
- Gingival lesions are extremely rare and may present as localized atypical gingivitis or more commonly as painless alveolar bone swelling covered by slightly erythematous gingiva, usually with draining sinuses (Fig. 190).
- Involvement of the jaws is common but soft tissues (tongue, palate, buccal) may be less frequently involved. Painless, slow-growing, hard-swelling is the classic feature of the disease, usually followed by multiple abscesses that drain to the surface by sinus tracts (Fig. 191). The exudation from the sinus tracts typically contains the characteristic sulphur granules that are colonies of organisms.

Diagnosis
- The clinical diagnosis should be confirmed by laboratory tests.
- Direct bacteriological examination and culture.
- Indirect immunofluorescence.
- Biopsy and histopathological examination.

Differential Diagnosis
- Periodontal and dental abscess
- Tuberculosis
- Systemic mycoses

Treatment
- Penicillin, erythromycin or tetracyclines.
- Surgery is necessary in most cases.

FIGURE 190

Actinomycosis.
Draining sinus on the gingiva

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 191

Actinomycosis.
Multiple sinus tracts on the skin

Gonococcal Infection

Definition
- Gonococcal infection is a sexually transmitted disease that primarily involves the urinary tract and less often the respiratory tract.

Etiology
- *Neisseria gonorrhoeae*

Gingival Involvement
- Rare
- Non-dental plaque-induced gingival lesions

Other Involvement
- Oral mucosa, relatively rare. It is more common among homosexuals and heterosexuals practising oral sex.
- Urethra, anus, cervix.

Main Clinical Features
- Gingival involvement may present as non-specific erythema and slight oedema with occasionally painful erosions (Fig. 192).
- The oral mucosal manifestations present as fiery erythema with or without superficial ulceration, usually covered with a greyish or yellowish exudate. The tonsil and soft palate are the most commonly affected sites followed by the tongue and buccal mucosa.

Diagnosis
- The clinical diagnosis of oral gonococcal infection should always be confirmed by laboratory tests.
- Culture and microscopic identification of microorganism.
- Fluorescent antibody test.

Differential Diagnosis
- Dental plaque-induced gingivitis
- Desquamative gingivitis
- Herpetic gingivitis and stomatitis
- Granulomatous gingivitis
- Streptococcal gingivitis and stomatitis

Treatment
- Frequently oral lesions are self-limited.
- Systemic penicillin or tetracycline.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 192

Gonococcal gingivitis presenting as localized erythema and slight oedema

Reading List

Benoliel R, Asquith J (1985) Actinomycosis of the jaws. Int J Oral Surg 14:195–199
Dimitrakopoulos J, Zouloumis L, Lazaridis N et al (1991) Primary tuberculosis of the oral cavity. Oral Surg Oral Med Oral Pathol 72:712–714
Eng HL, Lu SY, Yang CH, Chen WJ (1994) Oral tuberculosis. Oral Surg Oral Med Oral Pathol 81:415–420
Feder HM (1990) Actinomycosis manifesting as an acute painless lump of the jaw. Pediatrics 85:858–863
Ficarra G, DiLollo S, Pierieoni F, Panzoni E (1993) Actinomycosis of the tongue: a diagnostic challenge. Head Neck 15:53–55
Fiumara NJ (1976) Venereal disease of the oral cavity. J Oral Med 31:36–42
Hutt DM, Judson FN (1986) Epidemiology and treatment of oropharyngeal gonorrhea. Ann Intern Med 104:655–658
Laskaris G (1996) Oral manifestations of infectious diseases. Dent Clin North Am 40:395–423
Manton SL, Egglestone SI, Alexander I, Scully C (1986) Oral presentation of secondary syphilis. Br Dent J 160:237–238
Rivera-Hidalgo F, Stanford TW (1999) Oral mucosal lesions caused by infective microorganisms. I. Viruses and bacteria. Periodontology 2000 21:106–124
Scully C, Monteil R, Sposto MR (1998) Infectious and tropical diseases affecting the human mouth. Periodontology 2000 18:47–79

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

CHAPTER 18

Viral Infections

Measles

Definition
- Measles is a highly communicable infectious disease of childhood.

Etiology
- RNA paramyxovirus belonging to the *Morbillivirus* genus

Gingival Involvement
- Rare

Other Involvement
- Oral mucosa, relatively rare
- Skin, always

Main Clinical Features
- The gingival lesions may present as usually isolated, small round ulcers on the attached gingiva or the alveolar mucosa and occasionally as gingival erythema (Figs. 193, 194). The gingival lesions are not diagnostic.
- The oral mucosal lesions present as Koplik's spots (on the buccal mucosa), redness, petechiae and small round ulcers.
- The skin manifestations present as maculopapular dull red eruptions.
- Constitutional symptoms such as fever, chills, cough, photophobia, conjunctivitis and myalgias begin 3–4 days prior to skin eruption.

Diagnosis
- The diagnosis is based on clinical criteria.

Differential Diagnosis
- Varicella
- Herpetic gingivostomatitis
- Herpetiform ulcers
- Infectious mononucleosis

Treatment
- No treatment is needed for the gingival and oral lesions.
- The general management of patients must be left to the paediatrician.

For Personal Use Only
Library of School of Dentistry, TUMS

G. Laskaris et al., *Periodontal Manifestations of Local and Systemic Diseases*
© Springer-Verlag Berlin Heidelberg 2003

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 193

Measles.
Small round ulcer on the attached gingiva

FIGURE 194

Measles.
Gingival erythema

For Personal Use Only
Library of School of Dentistry, TUMS

Chickenpox

Definition
- Chickenpox or varicella is a highly contagious, self-limited primary infection, mainly in children.

Etiology
- Varicella-zoster virus or HHV-3

Gingival Involvement
- Rare

Other Involvement
- Oral mucosa (palate, buccal, lip), relatively rare
- Skin (face, head, neck), always

Main Clinical Features
- The gingival lesion present usually as diffuse erythema and isolated small vesicles that rupture quickly leaving round shallow ulcerations on an erythematous area (Fig. 195).
- The oral mucosal lesions also present as small vesicles, which rupture quickly leaving small ulcerations.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 195

Chickenpox.
Diffuse gingival erythema

- The skin manifestations include a pruritic red maculopapular rash, vesicles and pustules that rupture, crust and later exfoliate.
- The prodromal symptoms are malaise, fever, anorexia and irritability.

Diagnosis

- The diagnosis is mainly based on clinical criteria and rarely on laboratory tests.
- Tzanck smear examination.
- Polymerase chain reaction (PCR) detection of varicella-zoster virus DNA may be useful.

Differential Diagnosis

- Herpes simplex oral infection
- Minor aphthous ulcers
- Measles

Treatment

- No treatment is needed for oral lesions.
- Supportive treatment should be left to the paediatrician.

Herpes Zoster

Definition

- Herpes zoster is an acute viral disease caused by reactivation of a latent varicella-zoster virus that is transported via sensory nerve axons to the mucosae and/or the skin.

Etiology

- Reactivation of varicella-zoster virus, particular in immunocompromised patients

Gingival Involvement

- Relatively common. Gingival and oral mucosal lesions may occur when second or third divisions of the trigeminal nerve are involved.

Other Involvement

- Oral mucosa (palate, buccal, tongue, lips)
- Skin (face)

Main Clinical Features

- The gingival lesions present as unilateral clusters of vesicles which in 2–3 days rupture leaving painful ulcerations with irregular borders (Figs. 196, 197). Necrosis of the periodontium and alveolar bone may be seen. The ulcers usually heal without scarring in 2–3 weeks.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 196

Herpes zoster.
Ulceration of the lower attached gingiva in mandibular zoster

FIGURE 197

Herpes zoster.
Diffuse erythema and clusters of vesicles on the gingiva and the palatal mucosa in maxillary zoster

FIGURE 198

Herpes zoster.
Typical unilateral lesions on the face in maxillary zoster

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

- Similar unilateral lesions may appear on other oral sites, depending on trigeminal division involvement.
- An itching sensation and pain, which may simulate pulpitis, may precede oral lesions.
- The skin manifestations (trigeminal involvement) appear on the face and are characterized by unilateral clusters of maculopapules on an erythematous base, which soon form vesicles and in 2–3 days evolve into pustules. The pustules form within 5–8 days' crust and persist for 10–20 days (Fig. 198). New lesions continue to appear for several days. Tenderness and pain is always present.
- Constitutional symptoms such as fever, malaise and headache are common.
- Post-herpetic trigeminal neuralgia is the most common complication.

Diagnosis

- The diagnosis is based on clinical criteria and laboratory tests may be necessary only in atypical cases:
  - Viral culture.
  - Cytology smear.
  - Serological tests.
  - Viral DNA studies.

Differential Diagnosis

- Oral herpes simplex infection
- Necrotizing ulcerative gingivitis and periodontitis
- Erythema multiforme
- Leukaemia
- Agranulocytosis

Treatment

- Antiviral drugs (valacyclovir, famciclovir, aciclovir)
- Analgesics and sedatives

Infectious Mononucleosis

Definition

- Infectious mononucleosis is an acute self-limited infectious disease that is transmitted by saliva or blood.

Etiology

- Epstein-Barr virus (or HHV-4)

Gingival Involvement

- Rare

Other Involvement

- Oral mucosa (soft palate, uvula, buccal), relatively common
- Skin (rash)
- Lymph nodes

Main Clinical Features

- The gingival lesions may present as localized or diffuse erythema and less frequently as small ulcerations, particularly at the interdental papillae, mimicking necrotizing ulcerative gingivitis (Figs. 199, 200).
- The oral mucosal manifestations include palatal petechiae, faucial oedema, tonsillar exudate, pericoronitis and occasionally ulcerations.
- The skin lesions present as a maculopapular eruption.
- Generalized lymphadenopathy, profound malaise, sore throat, fever, anorexia and headache are common signs and symptoms.

Diagnosis

- The clinical diagnosis should be confirmed by laboratory tests:
  - Monospot test.
  - Paul-Bunnell test.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 199

Infectious mononucleosis. Diffuse erythema on the upper gingiva and lip

FIGURE 200

Infectious mononucleosis. Localized erythema on the lower gingiva and necrotizing gingivitis

Differential Diagnosis

- Herpetic gingivostomatitis
- Streptococcal gingivitis
- Necrotizing ulcerative gingivitis
- Leukaemia
- Aplastic anaemia

Treatment

- Supportive.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Hand-Foot-and-Mouth Disease

Definition
- Hand-foot-and-mouth disease is an acute virus infection that usually affects children and young adults.

Etiology
- Coxsackie virus A16 and occasionally other types (A_4, A_5, A_9, A_{10}) are responsible.

Gingival Involvement
- Rare

Other Involvement
- Oral mucosa (buccal, labial, tongue, soft palate, uvula), common
- Skin (hand, foot, back, buttocks), common

Main Clinical Features
- The gingival lesions are characterized by small vesicles that soon rupture, leaving slightly painful, shallow ulcers surrounded by a red halo (Fig. 201).
- Similar lesions appear on the oral mucosa (Fig. 202).
- The oral lesions are usually multiple (3–20), 2–6 mm in diameter.
- The skin lesions present as multiple small vesicles (Fig. 203).
- Low-grade fever, malaise, headache precedes the oral and skin lesions.
- The disease lasts 5–8 days.

Diagnosis
- The diagnosis is based on clinical criteria.
- Viral cultures and serological investigation needed only in doubtful cases.

Differential Diagnosis
- Aphthous ulcers
- Herpetic stomatitis
- Herpangina
- Erythema multiforme

Treatment
- Symptomatic

FIGURE 201

Hand-foot-and-mouth disease. Small shallow ulcers on the gingiva

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 202

Hand-foot-and-mouth disease. Multiple ulcers on the tongue

FIGURE 203

Hand-foot-and mouth-disease. Multiple small vesicles on the skin

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Reading List

Bagg J (1996) Common infectious diseases. Dent Clin North Am 40:385–393
Contreras A, Falkler WA, Enwonwu CO et al (1997) Human *Herpesviridae* in acute necrotizing ulcerative gingivitis in children in Nigeria. Oral Microbiol Immunol 12:259–265
Greenberg MS (1996) Herpesvirus infectious. Dent Clin North Am 40:359–368
Katsabas A, Laskaris G, Capetanakis J (1978) Herpes zoster: a clinical study of 227 cases. Odontostomatol Prog 32:68–73
Laskaris G (1996) Oral manifestations of infectious diseases. Dent Clin North Am 40:395–423
Laskaris G (2000) Color atlas of oral diseases in children and adolescents. Georg Thieme Verlag, Stuttgart
Millar EP, Traulis MJ (1994) Herpes zoster of the trigeminal nerve: the dentists role in diagnosis and management. J Can Dent Assoc 60:450–453
Miller CS (1996) Viral infections in the immunocompetent patient. Dermatol Clin 14:225–241
Rivera-Hidalgo F, Stanford TW (1999) Oral mucosal lesions caused by infective microorganisms. I. Viruses and bacteria. Periodontology 2000 21:106–124
Scully C, Monteil R, Sposto MR (1998) Infectious and tropical diseases affecting the human mouth. Periodontology 2000 18:47–70
Scully C, Samaranayake LP (1992) Clinical virology in dentistry and oral medicine. Cambridge Univ Press, Cambridge
Smith S, Ross JR, Scully C (1984) An unusual oral complication of herpes zoster infection. Oral Surg Oral Med Oral Pathol 57:388–389

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

CHAPTER 19

HIV Infection

The spectrum of oral clinical signs and symptoms in HIV-infected patients is wide and its study is of great importance. Information concerning the nature, clinical and laboratory significance, and treatment of the oral lesions in HIV-infected patients has accumulated during the last 2 decades. Indeed, oral lesions in this group of patients have been used as indicators and early clinical signs not only of HIV disease but also of the disease progression and as criteria for entry into clinical trials.

It should be stressed that the oral lesions in HIV infection are indicative of and not truly specific for HIV disease, as several of them can also be found in non-HIV-infected patients. They should always be regarded as markers of HIV disease in correlation with the medical history and with other clinical and laboratory studies, particularly in cases where HIV status is unknown.

The oral lesions are mainly a result of cellular immunodeficiency due to HIV infection and are etiologically divided into five major groups. These include:

1. Infections (bacterial, viral, fungal)
2. Neoplasms
3. Drug reactions
4. Neurological manifestations
5. Lesions of unknown cause

These lesions may represent early or late manifestations of HIV disease, they may be common or rare, and the oral clinician should be familiar with all of them. The oral lesions are classified according to their relationship to HIV-infection into three groups:

- Group I, lesions that are strongly associated with HIV infection.
- Group II, lesions less commonly associated with HIV infection.
- Group III lesions are sometimes seen in HIV infection.

It should be emphasized that a dramatic reduction of oral lesions, including periodontal manifestations, has been observed the last 5 years in HIV-infected patients due to successful therapeutic regimens with the new anti-HIV drugs, resulting in improvement in the immunological status of patients.

It is of great importance for the periodontist to be familiar with periodontal manifestations of HIV infection, as the periodontium is a crucial anatomical region in dental practice.

The most common gingival lesions follow.

For Personal Use Only
Library of School of Dentistry, TUMS

G. Laskaris et al., *Periodontal Manifestations of Local and Systemic Diseases*
© Springer-Verlag Berlin Heidelberg 2003

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 204

HIV infection.
Necrotizing ulcerative gingivitis

Bacterial Infections

Necrotizing Ulcerative Gingivitis (see also p. 93)

- Necrotizing ulcerative gingivitis (NUG) is a common early manifestation in HIV-infected patients, with a prevalence rate of 4%–16%. A high possibility for HIV infection exists, particularly if high-risk behaviour is associated with necrotizing ulcerative gingivitis.

Main Clinical Features

- Clinically, the onset of the disease is either acute or subacute and it presents with fiery red and swollen gingiva and a characteristic destruction of one or more interdental papillae. In the acute stage, ulceration, necrosis and sloughing of both the margin of the gingiva and the tip of the interdental papillae are common findings (Fig. 204).
- Pain, bleeding and halitosis are also common symptoms. The anterior gingiva are most frequently affected.
- Necrotizing ulcerative gingivitis should be limited to lesions only involving gingival tissue with no loss of periodontal attachment.

Diagnosis

- Diagnosis is made on clinical criteria. Necrotizing ulcerative gingivitis may rarely progress to necrotizing stomatitis in HIV-infected patients. The latter is characterized by localized or generalized acute, painful, ulcerative and necrotic lesions of the oral mucosa spread beyond the gingiva.

Treatment

- In the acute phase, treatment of NUG should include metronidazole, for 6–8 days and topical mouthwashes releasing oxygen.
- Conventional periodontal treatment (plaque-control measures, root planing, scaling) should follow.

Necrotizing Ulcerative Periodontitis

- Necrotizing ulcerative periodontitis (NUP) is the most severe form of periodontal disease associated with HIV infection and it develops in patients with marked immunosuppression. The prevalence of NUP is

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 205

HIV infection.
Necrotizing ulcerative periodontitis

FIGURE 206

HIV infection.
Localized necrotizing ulcerative periodontitis

FIGURE 207

HIV infection.
Severe periodontitis presenting with severe oedema, multiple abscesses and bleeding

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

5% or less. This low prevalence may be related to the improved recent medical management of HIV infection.

Main Clinical Features

- NUP is characterized by gingival ulceration and necrosis and a rapid progressive destruction of the periodontal attachment and bone with loosening of the teeth (Figs. 205, 206).
- Spontaneous bleeding, deep-seated pain, halitosis, erythema and oedema are prominent features (Fig. 207).
- The lesions may be localized or less often generalized and are usually not associated with deep pocket formation, because extensive gingival necrosis often coincides with loss of crestal alveolar bone.

Treatment

- Treatment of NUP includes systemic administration of metronidazole or tetracycline in association with topical antibacterial mouthwashes.
- Conventional periodontal treatment maintenance and prevention should follow. Relapses are common.

Bacillary Angiomatosis

- Bacillary angiomatosis is a recently described bacterial infection due to *Bartonella henselae* and *B. quintana*. It is characterized by vascular proliferation and occurs more frequently in HIV-infected patients and rarely in other immunosuppressed individuals or patients with malignancies. Skin, visceral and rarely gingival and oral lesions may occur. Oral bacillary angiomatosis should be regarded as an important prediction marker for HIV infection.

Main Clinical Features

- The oral lesions present as an asymptomatic, erythematous and slightly elevated soft tissue, plaque, or nodule (Fig. 208). The gingiva, palate and the tongue are the most commonly affected sites.
- Clinical diagnosis is difficult because the lesion resembles Kaposi's sarcoma, pyogenic granuloma, pregnancy granuloma, peripheral giant cell granuloma, haemangioma, leiomyoma, brown giant cell tumour, amyloidosis and other vascular lesions.
- The definitive diagnosis is based on histological and EM criteria.

Treatment

- Treatment consists of systemic erythromycin administration.
- Tetracycline or doxycycline may also be used.

Other Bacterial Infections

- Oral syphilis is a relatively common infection occurring in HIV-infected patients. Both primary and secondary syphilis can occur in the oral cavity and rarely, on the gingiva. In patients with HIV disease, the lesions may have a malignant and protracted course. The clinical spectrum of oral manifestations, particularly in secondary syphilis, is very broad, and the differential diagnosis should include aphthous ulcers, candidiasis, lichen planus, lupus erythematosus, non-specific erythema, erythroleukoplakia, squamous cell carcinoma and others. Oral syphilis must be included in the differential diagnosis of oral lesions in HIV-infected patients who should be under routine serological testing.
- Oral tuberculosis should also be regarded in the differential diagnosis of oral lesions in HIV-infected patients, particularly after the recent

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 208

HIV infection. Bacillary angiomatosis presenting as erythema and nodular swelling on the lower gingiva

FIGURE 209

HIV infection. Diffuse necrotizing ulceration on the upper gingiva due to *Pseudomonas aeruginosa*

dramatic increase of tuberculosis in this group of patients. Gingival involvement is rare and present as atypical, irregular ulceration.

- Some other oral infections caused by *Mycobacterium avium-intracellulare*, *Klebsiella pneumoniae*, *Enterobacter cloacae*, *Escherichia coli*, *Actinomyces israelii* and *Pseudomonas aeruginosa* have also been reported predominantly during the later stages of HIV disease. The lesions of all of these may develop on the gingiva (Fig. 209).

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Viral Infections

▶ The most frequent viral oral lesions in HIV-infected patients are caused by members of the herpesvirus group. Less frequent lesions may be caused by various types of human papilloma virus.

Herpes Simplex Virus (see also p. 150)

▶ Primary and recurrent intraoral and perioral herpes simplex is a relatively frequent viral infection, with a prevalence of 5%–10%. The clinical features of oral herpesvirus infection are usually similar to those seen in non-HIV-infected patients; however, in HIV-infected patients with severe immune suppression (CD_4 cell counts usually below 100 cells/mm^3), widespread, severe, and prolonged lesions resemble primary herpetic gingivostomatitis (Fig. 210). In addition, recurrences are also common. Oral aciclovir (800–1200 mg/day) is usually the treatment of choice.

Herpes Zoster (see also p. 169)

▶ Herpes zoster is one of the earliest clinical signs of HIV infection, particularly in individuals under 40 years of age with severe lesions, but without a history of immunosuppression. It would be more accurate to classify oral herpes zoster in young persons as a lesion „strongly associated with HIV infection". In addition, HIV-infected individuals with herpes zoster develop AIDS more frequently that do matched controls, but the prevalence of oral herpes zoster is low (1%–2%). Gingival lesions may occur during second and third phase of trigeminal involvement (Fig. 211). The diagnosis of oral herpes zoster is based on clinical signs and symptoms similar to those seen in non-HIV-infected patients, with the exception of the severity of the lesions. Aciclovir, valaciclovir, or famciclovir is the treatment proposed.

Cytomegalovirus

▶ Oral ulcerations associated with cytomegalovirus (CMV) infection have rarely been reported in HIV-infected patients. However, it is thought that oral lesions due to CMV may be more common, and thus the final diagnosis should be based on histological verification and molecular biology laboratory tests. These oral lesions are usually early signs of disseminated CMV infection and usually involved the gingiva (Fig. 212). The clinical features of CMV ulcers are similar to aphthous ulcers and other viral-associated ulcers as well as to non-specific ulcerations and drug-associated oral ulcerations usually seen in HIV-infected patients. Oral CMV infection is associated with severe immune suppression. Intravenous (IV) ganciclovir and aciclovir in high doses can be used for treatment.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 210

HIV infection.
Herpetic gingivitis

FIGURE 211

HIV infection.
Herpes zoster on the upper gingiva and palate

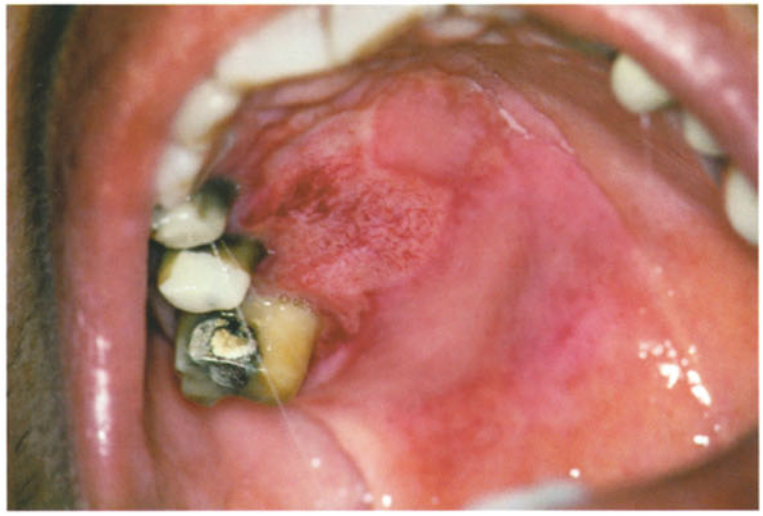


FIGURE 212

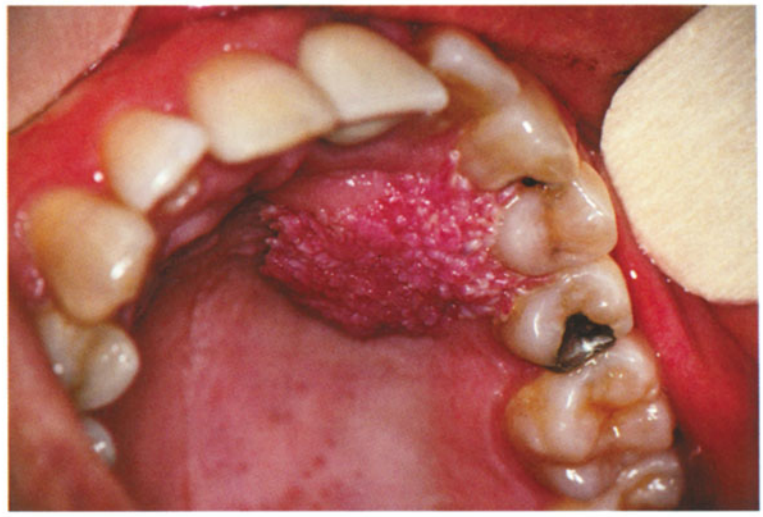
HIV infection.
Gingival ulcer due to cytomegalovirus

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 213

HIV infection. Typical condyloma acuminatum on the upper gingiva



Condyloma Acuminatum (see also p. 103)

- Condyloma acuminatum is relatively common in HIV-infected patients and the gingiva is the most common affected area (Fig. 213).

Mycoses

Linear Gingival Erythema

- Linear gingival erythema is a relatively uncommon gingival lesion presumably indicative of an underlying HIV infection. The lesion has received little attention in the literature and its exact prevalence rate in HIV-infected patients is not yet clear. Although the exact etiology remains unclear, a close association between oral candidiasis and linear gingival erythema exists.

Main Clinical Features

- Clinically, linear gingival erythema is characterized by a fiery red band along the margin of the gingiva and a punctate or diffuse erythema of the attached gingiva (Figs. 214, 215).
- There is no ulceration and evidence of pocket formation or attachment loss.
- Gingival bleeding may occur spontaneously or on probing. However, an important clinical sign of linear gingival erythema is now considered to be the absence of bleeding on probing.
- The lesion does not respond well to plaque-control measures or root planing and scaling. Plaque-control and a high level of oral hygiene are the treatments suggested. Persistent lesions may be related to candida infection and should be managed with antifungals.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 214

HIV infection.
Linear gingival erythema

FIGURE 215

HIV infection.
Linear gingival erythema

For Personal Use Only
Library of School of Dentistry, TUMS

Candidiasis (see also p. 109)

- Oral candidiasis is the most common oral manifestation among patients with HIV, predominantly caused by *Candida albicans* and rarely by other *Candida* species. It may be the initial sign of HIV infection in patients who appear otherwise healthy. In addition, oral candidiasis is usually associated with an increased risk for progression to HIV disease. A significant interaction between oral candidiasis and CD4-cell-decline rate has also been detected. In one study of 700 HIV-infected patients, 70% with oral candidosis had CD4+ cell counts below 200 cells/mm^3. The prevalence of the lesions ranges between 11% and 95%, depending on the study design and the patient group. Except for HIV infection, other predisposing, systemic and local factors can also influence the development of oral candidosis.
- In HIV-infected patients, candidal infection can occur in two major forms: pseudomembranous and erythematous. In addition, different forms of oral candidiasis can coexist in the same patient.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 216

HIV infection. Pseudomembranous candidiasis on the upper gingiva

FIGURE 217

HIV infection. Severe pseudomembranous candidiasis on the upper gingiva

FIGURE 218

HIV infection. Erythematous candidiasis on the upper gingiva

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

- Pseudomembranous candidiasis presents as white or yellowish spots or plaques that can be wiped off, revealing an erythematous surface. The gingival lesions are not common (Fig. 216, 217). The soft palate, buccal mucosa, lateral borders of the tongue, commissures of lips are more frequently affected.
- Erythematous candidiasis presents as a red area usually on the palate and the dorsum of the tongue; less often gingival involvement may occur (Fig. 218). White spots and plaques may be seen, but these are not usually conspicuous.
- The treatment of oral candidiasis includes azole (ketoconazole, fluconazole, itraconazole) and polyene drugs (nystatin and amphotericin B). Resistance of oral candidosis to those drugs in HIV-infected patients is a common finding and recurrences are frequently seen.

Systemic Mycoses (see also p. 193)

- Systemic or deep mycoses have, traditionally, been seen mainly in endemic areas and in developing countries, but they are now increasing in immunocompromised individuals, including HIV-infected patients. Gingival and oral lesions caused by systemic mycoses may occasionally be the primary or even the only manifestation, but they are usually associated with systemic signs and symptoms. HIV-infected patients are at particular risk from disseminating systemic mycoses.
- Of the systemic mycoses with oral manifestations, histoplasmosis and cryptococcosis have been most frequently encountered in HIV-infected patients.
- Sporadic cases of other systemic mycoses with oral manifestations have also been described in HIV-infected patients.
- Most of the systemic mycoses are diagnosed on the basis of a history of endemic areas, a recent trip abroad or an immunocompromised state. The laboratory diagnostic tests include smears, biopsy (staining with PAS, Gomori methenamine silver, Giemsa) and cultures from the affected tissues. In addition, physical examination, chest radiography and occasionally serological tests are necessary for the final diagnosis. In systemic mycoses, the oral lesions clinically present as ulcers, plaques or nodules and treatment includes systemic administration of azoles and amphotericin-B.

Neoplasms

Kaposi's Sarcoma (see also p. 330)

- Kaposi's sarcoma (KS) is the most common HIV-associated neoplasm, occurring after new therapeutic regimens in approximately 5%–10% of the patients with acquired immunodeficiency syndrome (AIDS). It is a potentially multicentric neoplasm that may manifest only with regional involvement in the oropharynx or with multiple-site involvement, including viscera. Up to 60% of AIDS patients with Kaposi's sarcoma have oral lesions and usually the oral mucosa is the first site of involvement, with subsequent progress to multiple sites. The prevalence of KS increases significantly with the progression of the disease. It has been suggested that oral lesions of KS are usually associated with lower CD4 counts than those detected with cutaneous lesions alone. Intraoral KS has a higher predilection for homosexual or bisexual men compared to

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 219

AIDS.
Early lesions of Kaposi's sarcoma presenting as red patch on the alveolar mucosa

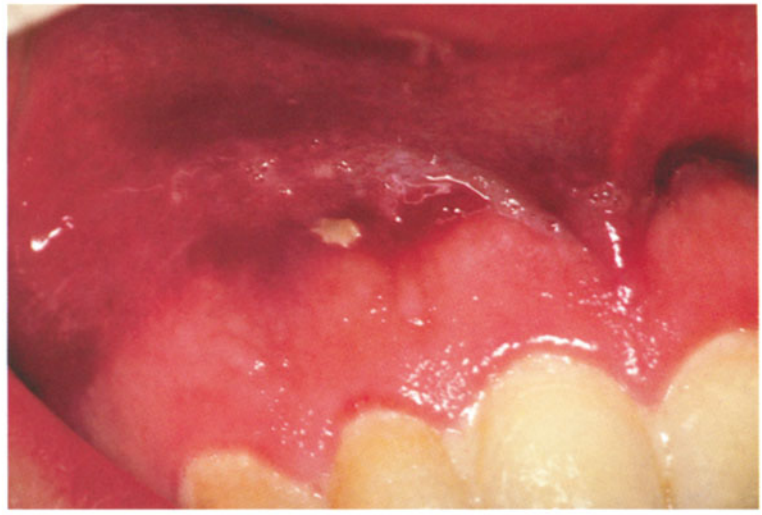


FIGURE 220

AIDS.
Large Kaposi's sarcoma on the upper gingiva

other risk groups for HIV infection. Both the etiology and the pathogenesis of KS are still not well understood; however, human herpes virus 8 (HHV 8) plays an important role in the pathogenesis, presumably in association with other co-factors.

Main Clinical Features

▶ Clinically, in the early phase, the oral lesions appear as a red, purple, or pigmented macule, papule or patch. Later, solitary or multiple elevated and exophytic tumours with or without ulceration may be the most prominent clinical feature (Figs. 219, 220). The palate is the most common site of involvement, followed by the gingiva, buccal mucosa and tongue.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

- A proposal has been suggested for clinical staging of oral KS depending on the extent (T) and the location (L) of tumour involvement and the clinical presentation or symptoms associated with the tumour (S). This staging might provide guidance for treatment and comparison of different treatment approaches among studies.
- A biopsy followed by histopathological examination of oral KS is necessary to confirm the clinical diagnosis.

Differential Diagnosis

- Bacillary angiomatosis
- Pyogenic granuloma
- Peripheral giant cell granuloma
- Leiomyoma
- Angiosarcoma
- Naevi
- Malignant melanoma
- Oral lesions of amyloidosis

Treatment

- Local and systemic treatment depending on the clinical stage. The local treatment can be surgical or laser excision, intralesional injection with vinblastine, sodium tetradecyl sulphate, or interferon and radiotherapy. Systemic treatment is single or combined chemotherapy (vincristine, vinblastine, bleomycin, Adriamycin).

Non-Hodgkin's Lymphomas

- Non-Hodgkin's lymphoma is the second most common malignancy in HIV infection and it develops in patients with CD4 cell counts below 100 cells/mm^3. Up to 3% of the patients would develop a non-Hodgkin lymphoma and the oral cavity accounts for 3% of all malignant lymphomas in patients with HIV infection. Not uncommonly, the mouth is the only site involved at the time of diagnosis. The posterior gingiva and fancer are frequently affected. All groups at risk for HIV infection are susceptible to the development of lymphoma, although some differences do exist in the clinical and histopathological features of individual groups. The majority of lymphomas are of B-cell origin, including the Burkitt's type. Rarely, Hodgkin's disease may manifest intra-orally and locate on the gingiva. Epstein-Barr virus is implicated as a cause for up to 50% of AIDS-associated lymphomas.

Main Clinical Features

- Clinically, oral lymphoma usually presents as a slowly or more often rapidly growing red or purplish, painless or painful mass that may be ulcerated (**Fig. 221**).
- Tooth loosening and paraesthesia are often the presenting symptoms.
- The posterior gingiva, the soft palate and fauces are the most commonly affected sites, while jaw involvement may also occur.
- The final diagnosis should be based on histopathological and histochemical findings.

Differential Diagnosis

- Kaposi's sarcoma
- Soft tissue and dental infection
- Systemic mycoses
- Salivary gland tumours

Treatment

- Treatment of oral lymphomas include multichemotherapy and radiation.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 221

AIDS.
Non-Hodgkin's lymphoma presenting as small ulcer on the upper gingiva

FIGURE 222

AIDS.
Irregular ulcer on the gingival due to drug reaction (DDC)

Drug Reactions

▶ As a result of treatment regimens, various drug-induced gingival and oral lesions commonly occur in HIV-infected patients (Fig. 222). Several of these reactions are associated with ulcerations, although the prevalence rate is generally unknown. This group of lesions includes stomatitis medicamentosa, erythema multiforme, Stevens-Johnson syndrome, toxic epidermal necrolysis, lichenoid reactions, oral pigmentation, oral ulcerations caused by DDC and foscarnet, oral lesions of agranulocytosis and face myositis.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Reading List

Dodd CL, Winkler JR, Heinic GS et al (1993) Cytomegalovirus infection presenting as acute periodontal infection in a patient infected with the human immunodeficiency virus. J Clin Periodontol 20:282–285

EC Clearinghouse on oral problems related to HIV infection and WHO collaborating centre on oral manifestations of the immunodeficiency virus. (1993) Classification and diagnostic criteria for oral lesions in HIV infection. J Oral Pathol Med 22:289–291

Epstein JB, Silverman S Jr (1992) Head and neck malignancies associated with HIV infection. Oral Surg Oral Med Oral Pathol 73:193–200

Ficcara G, Berson AM, Silverman S Jr et al (1988) Kaposi's sarcoma of the oral cavity: a study of 134 patients with a review of the pathogenesis, epidemiology, clinical aspects and treatment. Oral Surg Oral Med Oral Pathol 66:543–550

Glick M, Cleveland DB (1993) Oral mucosal bacillary (epithelial) angiomatosis in a patient with AIDS associated with rapid alveolar bone loss: report of a case. J Oral Pathol Med 22:235–239

Glick M, Muzyka BC, Lurie D et al (1994) Oral manifestations associated with HIV disease as markers for immune suppression and AIDS. Oral Surg Oral Med Oral Pathol 77:344–349

Glick M, Muzyka BC, Salkin LM, Lurie D (1994) Necrotizing ulcerative periodontitis: a marker for immune deterioration and a predictor of the diagnosis of AIDS. J Periodontol 65:393–397

Greenspan JS (1997) Sentinels and signposts: the epidemiology and significance of the oral manifestations of HIV disease. Oral Dis 3 [Suppl I]:S13–1S17

Lamster IB, Erbic JT, Bucklam RS et al (1997) Epidemiology and diagnosis of HIV-associated periodontal diseases. Oral Dis 3 [Suppl I]:S141–S148

Laskaris G (1996) Oral manifestations of infectious diseases. Dent Clin North Am 40:395–423

Laskaris G (2000) Oral manifestations of HIV disease. Clin Dermatol 18:447–455

Laskaris G, Hadjivassiliou M, Stratigos J (1992) Oral signs and symptoms in 160 Greek HIV-infected patients. Oral Surg Oral Med Oral Pathol 21:120–123

Laskaris G, Potouridou I, Laskari M, Stratigos I (1992) Gingival lesions of HIV infection in 178 Greek patients. Oral Surg Oral Med Oral Pathol 74:168–171

Laskaris G, Stergiou G, Kittas C et al (1999) Hodgkin's disease involving the gingiva in AIDS. Oral Oncol Eur J Cancer 28B:39–41

Novak MJ (1999) Necrotizing ulcerative periodontitis. Ann Periodontol 4:74–77

Riley C, London JP, Burmeister JA (1992) Periodontal health in 200 HIV-positive patients. J Oral Pathol Med 21:124–127

Robinson PG, Winkler JR, Palmer G et al (1994) The diagnosis of periodontal conditions associated with HIV infection. J Periodontol 65:236–243

Rowland RW (1999) Necrotizing ulcerative gingivitis. Ann Periodontol 4:65–73

Scully C, de Almeida OP, Sposto MR (1997) The deep mycoses in HIV infection. Oral Dis 13 [Suppl I]:S200–S207

Scully C, Epstein JB, Porter S, Luker J (1990) Recognition of oral lesions of HIV infection. 3. Gingival and periodontal disease and less common lesions. Br Dent J 169:370–372

Scully C, Laskaris G, Pindborg JJ et al (1991) Oral manifestations of HIV infections and their management. I. More common lesions. Oral Surg Oral Med Oral Pathol 71: 158–166

Scully C, Laskaris G, Pindborg J et al (1991) Oral manifestations of HIV infection and their management. II. Less common lesions. Oral Surg Oral Med Oral Pathol 71:167–171

Stratigos J, Laskaris G (1996) Color Atlas of AIDS. Skin and oral manifestations. Zita, Athens

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

CHAPTER 20

Systemic Mycoses

Histoplasmosis

Definition

- Histoplasmosis is a granulomatous systemic mycosis that is mainly endemic in the Ohio and Mississippi valleys in USA, in Latin America, India, east Asia and in Australia. However, sporadic cases may occur in other parts of the word, particularly in HIV infected patients.

Etiology

- *Histoplasma capsulatum* usually

Classification

- Three main forms are recognized:
  - Primary acute pulmonary.
  - Chronic pulmonary.
  - Disseminated form.
- Oral lesions occur mainly in patients with disseminated disease and HIV disease, less frequently with pulmonary histoplasmosis.

Gingival Involvement

- Relatively common

Other Involvement

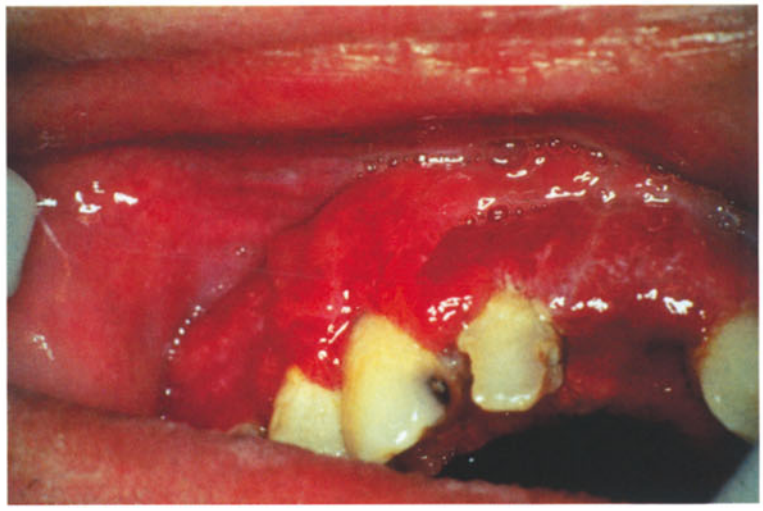
- Oral mucosa, relatively common
- Skin
- Lung and other organs

Main Clinical Features

- The gingival lesions present as a chronic vegetating painful ulcer with a tendency to increase in size and less frequently as nodules (Fig. 223).

FIGURE 223

Histoplasmosis. Vegetating ulceration on the upper gingiva



For Personal Use Only
Library of School of Dentistry, TUMS

G. Laskaris et al., *Periodontal Manifestations of Local and Systemic Diseases*
© Springer-Verlag Berlin Heidelberg 2003

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

- The oral mucosal lesions present also as painful ulcers or nodules usually on the tongue, palate and buccal mucosa.
- Oral lesions of histoplasmosis may be found in a patient without evidence of systemic histoplasmosis.
- Systemic manifestations include cough, fever, weight loss, chest pain, weakness, fatigue and involvements of lymph nodes, spleen, liver, kidneys, gastrointestinal tract and central nervous system.

Diagnosis

- The clinical diagnosis should be confirmed by laboratory tests.
- Biopsy and histopathological examination.
- Culture.
- Serological tests.

Differential Diagnosis

- Oral tuberculosis
- Squamous cell carcinoma
- Other systemic mycoses
- Eosinophilic ulcer
- Wegener's granulomatosis
- Non-Hodgkin's lymphoma

Treatment

- Fluconazole, itraconazole, amphotericin B

Mucormycosis

Definition

- Mucormycosis or zygomycosis or phycomycosis is a relatively rare systemic mycosis that mainly affects immunocompromised patients (patients with leukaemia, non-Hodgkin's disease, severe, poorly controlled diabetes mellitus, AIDS, immunosuppressed patients).

Etiology

- Mainly *Mucor* and *Rhizopus* and rarely other species

Gingival Involvement

- Relatively common

Other Involvement

- Oral mucosa (palate)
- Nasal cavity and paranasal sinuses
- Lung, gastrointestinal tract
- Cardiovascular and central nervous system

Main Clinical Features

- The oral manifestations occur in the rhinocerebral mucormycosis, which is the most common form of the disease.
- The gingival lesions present as erythematous and oedematous areas, which soon progress in black necrotic ulcerations with severe alveolar bone destruction (Fig. 224).
- The oral mucosal lesions usually appear as palatal, black necrotic ulcers, which quickly spread, creating extensive bone destruction.
- The disease usually commences in the nasal cavity or paranasal sinuses with pain, nasal discharge and fever. Facial swelling, headache are also common.
- Without proper treatment, mucormycosis may spread into the orbit and the brain, finally leading to death.

Diagnosis

- The clinical diagnosis should be confirmed by laboratory tests and must be rendered early as soon as possible.
- Biopsy and histopathological examination.
- Smear examination.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 224

Mucormycosis. Necrotic ulcerations and severe alveolar bone and palate destruction

Differential Diagnosis
- Severe necrotizing ulcerative gingivitis and stomatitis
- Malignant granuloma
- Wegener's granulomatosis
- Tuberculosis
- Non-Hodgkin's lymphoma
- Other systemic mycoses

Treatment
- Surgical debridement and systemic amphotericin B

Paracoccidioidomycosis

Definition
- Paracoccidioidomycosis, or South American blastomycosis, is a chronic granulomatous systemic mycosis which is endemic in Brazil and less common in Venezuela, Uruguay, Argentina and Colombia

Etiology
- *Paracoccidioides brasiliensis*

Gingival Involvement
- Relatively common

Other Involvement
- Oral mucosa, relatively common
- Lungs, always

Main Clinical Features
- The gingival lesions present as painful, chronic, granular vegetating ulcer with severe erythema, which may be localized or generalized (Fig. 225).
- The oral mucosal lesions also present as painful exophytic ulcerations with granular surface, usually occurring on the palate, tongue and lips.
- In several cases, the oral lesions follow tooth extraction.
- Chronic pulmonary disease with cough, dyspnea, hemoptysis, fever and weight loss are the presenting feature of the disease.

Diagnosis
- The clinical diagnosis should be confirmed by laboratory tests.
- Biopsy and histopathological examination.
- Smear examination or culture.
- Serological tests.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 225

Paracoccidioidomycosis. Vegetating ulceration on the lower gingiva

Differential Diagnosis

- Pyostomatitis vegetans
- Wegener's granulomatosis
- Tuberculosis
- Other systemic mycoses

Treatment

- Fluconazole, itraconazole
- Amphotericin B
- Sulfamethoxypyridazine

Reading List

De Almeida OPD, Jorge J, Scully C, Bozzo L (1991) Oral manifestations of paracoccidioidomycosis (South American blastomycosis). Oral Surg Oral Med Oral Pathol 72:430–435

Economopoulou P, Laskaris G, Ferekidis E, Kanelis N (1995) Rhinocerebral mucormycosis with severe oral lesions: a case report. J Oral Maxillofac Surg 53:215–217

Ng KH, Siar CH (1996) Review of oral histoplasmosis in Malaysians. Oral Surg Oral Med Oral Pathol 81:303–307

Salisbury PL, Caloss R, Cruz JM et al (1997) Mucormycosis of the mandible after dental extractions in a patient with acute myelogenous leukemia. Oral Surg Oral Med Oral Pathol 83:340–334

Scully C, Almeida OPD (1992) Orofacial manifestations of the systemic mycoses. J Oral Pathol Med 21:289–294

Scully C, Almeida OPD, Sposto MR (1997) Deep mycoses in HIV infection. Oral Dis 3 [Suppl 1]:200–207

Scully C, Monteil R, Sposto MR (1998) Infectious and tropical diseases affecting the human mouth. Periodontology 2000 18:47–70

Sposto MR, Scully C, Almeida OPD et al (1993) Oral paracoccidioidomycosis: a study of 36 South American patients. Oral Surg Oral Med Oral Pathol 75:461–465

Stanford TW, Rivera-Hidalgo F (1999) Oral mucosal lesions caused by infective microorganisms. II. Fungi and parasites. Periodontology 2000 21:125–144

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

CHAPTER 21

Haematological Disorders and Malignancies

Familial Neutropenia

Definition

- Familial neutropenia is a rare haematological disorder characterized by quantitative decrease of neutrophils in the peripheral blood due to improper release of them from the bone marrow. An increased susceptibility of the patients to bacterial infection occurs.

Etiology

- It is inherited as an autosomal dominant and recessive trait.
- Microbial dental plaque modified by quantitative leukocyte disorders are responsible for the periodontal lesions.

Periodontal Involvement

- Common
- Periodontitis associated with haematological disorders

Other Sites of Involvement

- Oral mucosa
- Skin, ear
- Respiratory tract, urinary system

Main Clinical Features

- The main clinical manifestations are recurrent infections and are usually present at birth. Periodontal manifestations include fiery red oedematous gingivitis, severe periodontal lesions with deep periodontal pockets and extensive generalized bone loss leading to tooth mobility and loss (Figs. 226, 227).
- Recurrent oral ulceration, which may lead to scar formation is common.

FIGURE 226

Familial neutropenia. Diffuse erythematous gingivitis on the upper gingiva

For Personal Use Only
Library of School of Dentistry, TUMS

G. Laskaris et al., *Periodontal Manifestations of Local and Systemic Diseases*
© Springer-Verlag Berlin Heidelberg 2003

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 227

Familial neutropenia.
Fiery red oedematous gingivitis

- Recurrent skin infections, otitis media, pneumonia, are constant manifestations.
- The severity of infections depend on quantitative neutrophil decrease.
- Affected children tend to improve with age and some undergo total remission in late childhood.

Diagnosis

- The clinical diagnosis should be confirmed by laboratory tests.
- Complete blood count.
- Radiographic examination.

Differential Diagnosis

- Aggressive periodontitis
- Cyclic neutropenia
- Agranulocytosis
- Leukaemia
- Chediak-Higashi syndrome
- Glycogen storage disease 1b
- Infantile genetic agranulocytosis
- Hypophosphatasia

Treatment

- Good oral hygiene.
- Periodontal treatment (scaling, surgery, antimicrobial agents).
- The systemic treatment must be left to the specialist haematologist.

Cyclic Neutropenia

Definition

- Cyclic neutropenia is a rare haematological disorder, characterized by regular period depletion of neutrophil population, usually in 3-week cycles. The episode of neutropenia is usually short, persist for 5–10 days after which the neutrophil counts gradually return to normal.

Etiology

- The etiology remains unknown and the disorder is thought to be due to a defect of haematopoietic control. An autosomal dominant trait has been recorded in some cases.
- Microbial dental plaque modified by quantitative leukocyte disorders are responsible for the periodontal lesions.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 228

Cyclic neutropenia. Diffuse gingival erythema and oedema

Periodontal Involvement

- Relatively common
- Periodontitis associated with haematological disorders

Other Sites of Involvement

- Oral mucosa
- Skin, ear

Main Clinical Features

- Periodontal manifestations include diffuse gingival inflammation and ulcerations, periodontal pockets and generalized bone loss (Fig. 228).
- Painful, recurrent, oval oral ulcerations covered by a whitish membrane on an erythematous base usually occur.
- Skin infections and otitis media may also occur during an episode of profound neutropenia.
- Low-grade fever, malaise, headache, arthralgia, and cervical adenitis may occur.
- The disease usually begins after 10 years of age.

Diagnosis

- The clinical diagnosis should be confirmed by laboratory tests.
- Repeated complete blood count.
- Bone marrow examination during periods of neutropenia shows decreased numbers of neutrophil precursors.

Differential Diagnosis

- Aggressive periodontitis
- Familial neutropenia
- Agranulocytosis
- Chediak-Higashi syndrome
- Glycogen' storage disease 1b
- Infantile genetic agranulocytosis
- Leukaemia
- Aphthous ulcers
- Herpetic stomatitis

Treatment

- Good oral hygiene.
- Periodontal treatment (scaling, surgery, antimicrobial agents).
- The systemic treatment must be left to the specialist haematologist.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Agranulocytosis

Definition
- Agranulocytosis is an acute haematological disorder characterized by severe depletion of neutrophils or all granulocytes in the blood.

Etiology
- It is often due to adverse drug reactions.
- Idiopathic cases or cases induced by infections may also occur.
- Microbial dental plaque modified by quantitative leukocyte disorders are responsible for the periodontal lesions.

Periodontal Involvement
- Common as generalized periodontal destruction
- Periodontitis associated with haematological disorders

Other Sites of Involvement
- Oral mucosa (buccal mucosa, tongue, palate, tonsils)
- Other mucosae (pharynx, larynx, oesophagus, gastrointestinal tract)
- Respiratory tract

Main Clinical Features
- The periodontal lesions are early manifestations and are characterized by ulceration and necrosis of the marginal and attached gingiva associated with haemorrhage (Fig. 229). Severe generalized bone loss may also occur.
- Necrotic ulcers covered by grey-white or dark, friable pseudomembranes are common findings on the oral mucosa. The ulcers do not have a red halo around them.
- Halitosis, sialorrhoea, dysphagia, difficulty in swallowing, fever, malaise and headache are common.
- Pulmonary and gastrointestinal infections are common.

Diagnosis
- The clinical diagnosis should be confirmed by laboratory test.
- Complete blood count and bone marrow aspiration.

Differential Diagnosis
- Necrotizing ulcerative gingivitis and stomatitis
- Familial neutropenia
- Cyclic neutropenia
- Acute leukaemia
- Other types of neutropenia
- Infectious mononucleosis
- Aplastic anaemia

Treatment
- Good oral hygiene.
- Periodontal treatment should follow the systemic treatment.
- Systemic treatment must be left to the specialist haematologist.

Aplastic Anaemia

Definition
- Aplastic anaemia is a severe haematological disorder characterized by acellular or markedly hypocellular bone marrow resulting in pancytopenia.

Etiology
- Drugs, chemicals, radiation, infections are usually associated with the disease. Idiopathic cases may also occur.

Gingival Involvement
- Common
- Gingival lesions associated with blood dyscrasias

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 229

Agranulocytosis. Severe erythema and necrotic ulcerations of the gingiva

FIGURE 230

Aplastic anaemia. Early necrotic ulceration on the upper gingiva

FIGURE 231

Aplastic anaemia. Ecchymoses on the palate and tongue

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Other Sites of Involvement
- Oral mucosa
- Other mucosae, skin

Main Clinical Feature
- Gingival bleeding is a common early sign. Localized necrotic ulcerations on the marginal gingiva and the interdental papillae, mimic necrotizing ulcerative gingivitis (Fig. 230).
- The oral mucosal lesions appear as necrotic ulcers, petechiae, ecchymoses and pallor of the oral mucosa (Fig. 231).
- Pallor, petechiae, or ecchymoses and purpuric spots of the skin and other mucous membranes are also early signs.
- The onset of the disease is usually insidious and the most common symptoms are fever, weakness, fatigue, fever and mild haemorrhage.
- The course of the disease depends on the severity of aplasia.

Diagnosis
- The clinical diagnosis should be confirmed by laboratory tests.
- Complete blood count and bone marrow aspiration and biopsy.

Differential Diagnosis
- Necrotizing ulcerative gingivitis and stomatitis
- Vitamin C deficiency
- Agranulocytosis
- Neutropenia
- Leukaemia
- Thrombocytopenic purpura
- Infectious mononucleosis

Treatment
- Good oral hygiene.
- Topical oral antiseptics.
- Systemic treatment must be left to the specialist haematologist.

Thrombocytopenic Purpura

Definition
- Thrombocytopenic purpura is a haematological disorder characterized by a profound decrease in platelets in the peripheral blood.

Etiology
- It may be idiopathic due to a primary failure of the bone marrow to generate platelets or secondary due to a myelotoxic drug, virus and HIV infections, leukaemias and other autoimmune diseases, malignancies, radiation, etc.

Gingival Involvement
- Common

Other Sites of Involvement
- Other mucosae (conjunctiva, nose, gastrointestinal, urinary)
- Skin

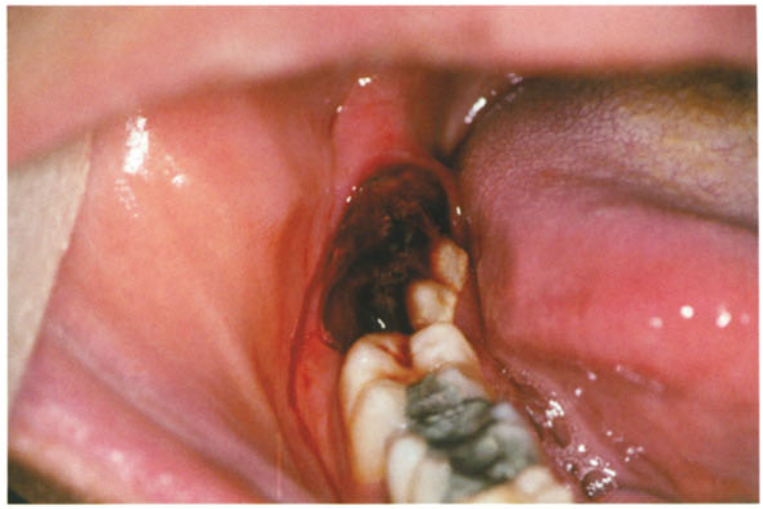
Main Clinical Features
- Gingival bleeding, spontaneous and/ or prolonged is a constant early feature (Fig. 232). There is no evidence that thrombocytopenic purpura increases susceptibility to periodontal disease.
- On the oral mucosa' petechiae, ecchymoses and haematomas may occur.
- Bleeding episodes from gastrointestinal and urinary tracts and epistaxis are common.
- Purpuric skin rash and occasionally bleeding occur.
- The breadth and the severity of the manifestations are related to the extent of platelet reduction.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 232

Thrombocytopenic purpura. Gingival bleeding



Diagnosis
- The clinical diagnosis should be confirmed by laboratory tests.
- Complete blood count and bone marrow aspiration.

Differential Diagnosis
- Leukaemia
- Aplastic anaemia
- Agranulocytosis

Treatment
- Good oral hygiene.
- Systemic and etiological treatment must be left to the specialist haematologist.

Plasminogen Deficiency

Definition
- Plasminogen deficiency, or hypoplasminogenaemia, is a deficiency in plasminogen. In health, body fluid fibrinolytic activity clears fibrin deposits, but if plasminogen is deficient this mechanism fails, with fibrin deposition occurring. Perhaps surprisingly, there is rarely a tendency to thrombosis.

Etiology
- An autosomal recessive disorder

Gingival Involvement
- Common

Other Sites of Involvement
- Corneal involvement and blindness
- Congenital occlusive hydrocephalus

Main Clinical Features
- Generalized painful ulcerated gingival swelling since late childhood (Fig. 233).
- Gingival enlargement is more severe in interdental papillae (Fig. 234).

Diagnosis
- The clinical diagnosis should always be confirmed by laboratory tests.
- Histopathological examination.
- Plasminogen concentration.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 233

Plasminogen deficiency. Gingival ulcerations

FIGURE 234

Plasminogen deficiency. Severe gingival swelling and minor ulcerations

For Personal Use Only
Library of School of Dentistry, TUMS

Differential Diagnosis

- Hereditary gingival fibromatosis and related disorders
- Lipoid proteinosis
- Mucocutaneous amyloidosis
- Mucopolysaccharidosis I-H

Treatment

- Topical heparin or intravenous purified plasminogen concentrate

Myelodysplastic Syndrome

Definition

- Myelodysplastic syndromes form a heterogenous group of refractory anaemias often associated with thrombocytopenia, neutropenia, and/or monocytosis. Older individuals are more often affected.

Etiology

- The exact etiology remains unknown. It may develop secondary to radiotherapy and chemotherapy.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 235

Myelodysplastic syndrome. Gingival swelling and early ulcerations

FIGURE 236

Myelodysplastic syndrome. Gingival ulcerations

Gingival Involvement

- Rare

Other Sites of Involvement

- Oral mucosa
- Other mucosae
- Skin and other systems

Main Clinical Features

- The gingival lesions may appear as chronic localized swelling and/or ulcerations, associated with bleeding (Figs. 235, 236).
- The oral mucosal lesions appear as persistent and recurrent painful ulcerations and bleedings. Less often oral candidiasis may develop.
- Multiple bacterial infections and haemorrhage are the main features of the disease due to neutropenia and thrombocytopenia.
- Malaise, fatigue, fever are common symptoms.

Diagnosis

- The clinical diagnosis should be confirmed by laboratory tests.
- Complete blood count and bone marrow aspiration and biopsy.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Differential Diagnosis
- Leukaemias
- Agranulocytosis
- Neutropenia
- Aplastic anaemia
- Thrombocytopenia
- Non-Hodgkin lymphoma
- Multiple myeloma
- Langerhans cell histiocytosis

Treatment
- Good oral hygiene.
- Topical oral antiseptics.
- Systemic treatment must be left to the specialist haematologist.

Leukaemias

Definition
- Leukaemias are an heterogenous group of serious malignant disorders characterized by defects of leukocyte cell maturation and proliferation

Etiology
- The etiology remains obscure, although several factors are implicated such as genetic, viral infections, chemicals, radiation, immunodeficiency.

Classification
- Depending on the cell type primarily affected, leukaemias are classified into:
  - Lymphocytic
    - Acute
    - Chronic
  - Non-lymphocytic
    - Acute
    - Chronic

Gingival Involvement
- Common. Approximately 30% of patients with acute and 10% with chronic leukaemias present with gingival lesions.
- Pronounced inflammatory reaction of gingiva in relation to the dental plaque present. However, dental plaque is not a prerequisite for gingival lesions.

Other Sites of Involvement
- Oral mucosa
- Skin
- Lymph nodes, spleen, liver

Main Clinical Features
- Generalized gingival swelling of variable severity may appear usually in acute and rarely in chronic leukaemia (Figs. 237–239). Severe gingival enlargement is usually a feature of acute monocytic leukaemia (Figs. 240–243). The gingival enlargement initially starts at the interdental papilla and is followed by the marginal and attached gingiva. The enlargement may be due to both plaque-related chronic inflammation and to infiltration by leukaemic cells.
- Gingival bleeding may be one of the initial oral signs in the:
  - Acute form
  - Chronic form
- Rarely, rapid alveolar bone loss may occur, particularly in the patient with pre-existing periodontitis. Not infrequently, gingival lesions are the initial manifestation of leukaemia.
- Non-specific ulcerations of the oral mucosa and bleeding may occur.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 237

Acute myeloblastic leukaemia. Early gingival swelling

FIGURE 238

Acute myeloblastic leukaemia. Severe gingival enlargement

FIGURE 239

Acute myeloblastic leukaemia. Severe gingival enlargement and bleeding

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 240

Acute myelomonocytic leukaemia. Early red gingival swelling

FIGURE 241

Acute myelomonocytic leukaemia. Severe gingival enlargement

FIGURE 242

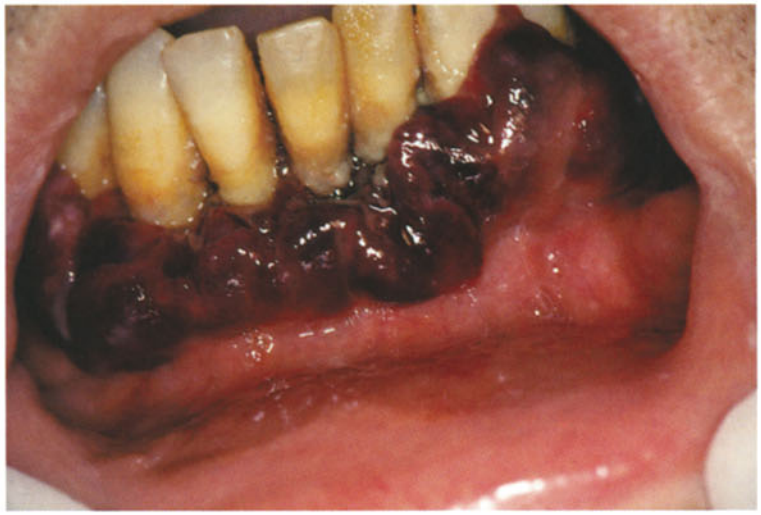
Acute myelomonocytic leukaemia. Severe gingival enlargement

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 243

Acute monocytic leukaemia. Severe red gingival swelling as an initial sign of the leukaemia



- Early symptoms are pallor, anorexia, irritability, fatiguability, low fever, bleeding and lymph node enlargement.
- During chemotherapy, oral viral, bacterial and candida infections are common.

Diagnosis

- The clinical diagnosis should be confirmed by laboratory tests.
- Complete blood count and bone marrow biopsy.
- Biopsy of extramedullary tissue involved.

Differential Diagnosis

- Necrotizing ulcerative gingivitis
- Aplastic anaemia
- Agranulocytosis
- Neutropenia
- Multiple myeloma
- Langerhans cell histiocytosis
- Myelodysplastic syndrome

Treatment

- Good oral hygiene.
- Topical oral antiseptics.
- Systemic treatment must be left to the specialist haematologist.

Multiple Myeloma

Definition

- Multiple myeloma is a relatively rare malignant plasma cell disorder due to overproduction of specific immunoglobulins, not directed against any recognized epitope.

Etiology

- Unknown

Gingival Involvement

- Relatively rare

Other Sites of Involvement

- Oral mucosa (about 14%)
- Skin
- Skull, skeleton, including jaws
- Kidneys

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 244

Multiple myeloma.
Localized gingival swelling

FIGURE 245

Multiple myeloma.
Localized severe gingival swelling

For Personal Use Only
Library of School of Dentistry, TUMS

Main Clinical Features

- Gingival bleeding, localized or generalized gingival swelling and gingival necrotic ulceration usually associated with alveolar bone destruction are the hallmarks of the neoplastic process (Figs. 244, 245). Gingival paraesthesia or anaesthesia may also occur.
- Alveolar bone pain, swelling and tooth mobility are also common.
- Bone pain is usually the presenting symptom. Fatigue, fever, petechiae of the skin, amyloid deposits in various soft tissues may occur.
- The disease usually involves individuals over 50 years of age.

Diagnosis

- The clinical diagnosis should be confirmed by laboratory test.
- Biopsy and histopathological examination of the lesional tissue.
- Radiographic examination.
- Serum and urine protein electrophoresis.

Differential Diagnosis

- Necrotizing ulcerative gingivitis
- Leukaemia
- Aplastic anaemia

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

- Lymphomas
- Neutropenia
- Agranulocytosis
- Langerhans cell histiocytosis

Treatment

- Good oral hygiene.
- Topical oral antiseptics.
- Periodontal treatment.
- Systemic treatment must be left to the specialist.

Soft Tissue Plasmacytoma

Definition

- Soft tissue plasmacytoma, or solitary extramedullary plasmacytoma, is an unusual monoclonal neoplasm that consists of plasma cells indistinguishable from those seen in multiple myeloma.

Etiology

- Unknown

Gingival Involvement

- Rare

Other Sites of Involvement

- Oral mucosa (palate, tongue, floor of the mouth)
- Nasopharynx, nasal cavity, paranasal sinuses, tonsils

Main Clinical Features

- Localized non-tender, well-circumscribed gingival swelling is the most common clinical feature. The gingival surface is usually normal or slightly red and rarely may be ulcerated (Fig. 246). Periodontal destruction may rarely occur. The size of the swelling varies from one several centimetres in diameter.
- Similar clinical features present at the other sites of involvement.
- A number of patients with primary soft tissue plasmacytoma may ultimately develop multiple myeloma.
- The disease affects more often males over 50 years of age.

Diagnosis

- The clinical diagnosis should always be confirmed by laboratory tests.
- Biopsy and histopathological examination.
- Immunohistochemical examination for H and L chain.

FIGURE 246

Soft tissue plasmacytoma. Localized gingival swelling

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Differential Diagnosis
- Non-Hodgkin's lymphoma
- Multiple myeloma
- Dental and periodontal abscesses
- Gingival cyst of adults
- Pleomorphic adenoma

Treatment
- Surgical excision usually in combination with radiotherapy, chemotherapy

Non-Hodgkin's Lymphoma

Definition
- Non-Hodgkin's lymphoma is an heterogenous group of neoplastic disorders that encompass neoplastic proliferation of lymphocytic cell lines (B- and T-cell origin)

Etiology
- Transforming genes and viruses may be involved in the pathogenesis of some forms of lymphomas.

Gingival Involvement
- Rare. The posterior gingiva and retromolar area are more often involved.

Other Sites of Involvement
- Oral mucosa (soft palate, base of the tongue, buccal mucosa) is rarely involved.
- Abdominal, head and neck and mediastinal lymph nodes and Waldeyer's ring are the most common sites of involvement.
- Extranodal involvement is rare.

Main Clinical Features
- Localized, painless, soft gingival swelling, occasionally associated with ulceration are the hallmarks of the gingival involvement (Figs. 247, 248). Periodontal destruction and bone loss are unusual.
- The oral mucosa lesions appear as a soft, painless, reddish or purplish diffuse swellings. The lesion may be ulcerated.
- In the head and neck area the disease presents as a painless mass of lymph nodes that increase slowly. As the disease progresses, the enlarged nodes become more numerous and fixed to surrounding tissues.
- Fever, night sweats, weight loss may occur.

Diagnosis
- The clinical diagnosis should always be confirmed by laboratory tests.
- Biopsy and histopathological examination.
- Immunohistochemical examination.
- Radiography and CT.

Differential Diagnosis
- Soft tissue plasmacytoma
- Hodgkin's disease
- Leukaemias
- Multiple myeloma
- Myelodysplastic syndrome
- Dental and periodontal abscesses

Treatment
- Chemotherapy
- Radiotherapy

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 247

Non-Hodgkin's lymphoma. Localized gingival swelling with ulceration

FIGURE 248

Non-Hodgkin's lymphoma. Gingival and palatal swelling (the ulcer is due to the biopsy)

For Personal Use Only
Library of School of Dentistry, TUMS

Burkitt's Lymphoma

Definition

- Burkitt's lymphoma is a malignant B-cell lymphoma arising within germinal cells of the lymph nodes. The disease is rare in Western countries and common in Africa, usually presenting between 10 and 12 years of age.

Etiology

- Epstein-Barr virus.
- Immunodeficiencies, AIDS, autoimmune disease may predispose to the development of the lymphoma.

Gingival Involvement

- Very rare

Other Sites of Involvement

- Oral mucosa, rare.
- Jaws, very often (60 %–70 %). The maxilla is twice as often affected as the mandible.
- Bone marrow, abdominal region and central nervous system may be involved.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 249

Burkitt's lymphoma.
Localized soft tumour on the gingiva

Main Clinical Features

- The gingival lesions appear as localized gingival swelling or exophytic soft mass tumour that may be ulcerated (Fig. 249). The colour of the mass is reddish. Tumour growth is usually rapid and may produce periodontal destruction and tooth mobility.
- Rapidly proliferative swelling of the jaws, which may produce marked destruction of the facial bone and facial nerve paresis, are the main clinical features of the disease. Bone pain, tenderness and paraesthesia may occur.
- The prognosis depends on the stage of the disease.

Diagnosis

- The clinical diagnosis should always be confirmed by laboratory tests.
- Biopsy and histopathological examination.
- Immunohistochemical examination.
- Panoramic radiographic examination, CT.

Differential Diagnosis

- Kaposi's sarcoma
- Peripheral giant cell granuloma
- Odontogenic tumours
- Ewing's sarcoma
- Non-Hodgkin's lymphomas (other types)
- Amyloidosis
- Fibrous dysplasia
- Cherubism

Treatment

- Radiotherapy and chemotherapy
- Surgical excision

Cutaneous T-cell Lymphoma

Definition

- Cutaneous T-cell lymphoma, or mycosis fungoides, is a rare, slowly progressive non-Hodgkin's lymphoma primarily involving the skin.

Etiology

- It remains obscure.

Gingival Involvement

- Rare

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Other Sites of Involvement

- Oral mucosa, rare
- Skin, always
- Visceral

Main Clinical Features

- Three clinical stages are recognized:
  - Erythematous.
  - Plaque.
  - Tumour.
- The gingival involvement usually occurs during the plaque and tumour stage and always after the skin lesions. Localized painful ulcers and, less often, red plaques or soft masses are the most common gingival lesions (Fig. 250).
- The oral mucosa lesions present as non-specific erythematous plaques or superficial ulcerations or ulcerated tumours.
- The skin lesions begin as an intensely pruritic eruption followed after a long time by the indurated plaque stage and finally by the tumour stage (Fig. 251).
- Males are more frequently affected, usually between 55 and 66 years of age.

FIGURE 250

Cutaneous T-cell lymphoma. Irregular ulceration on the upper gingiva

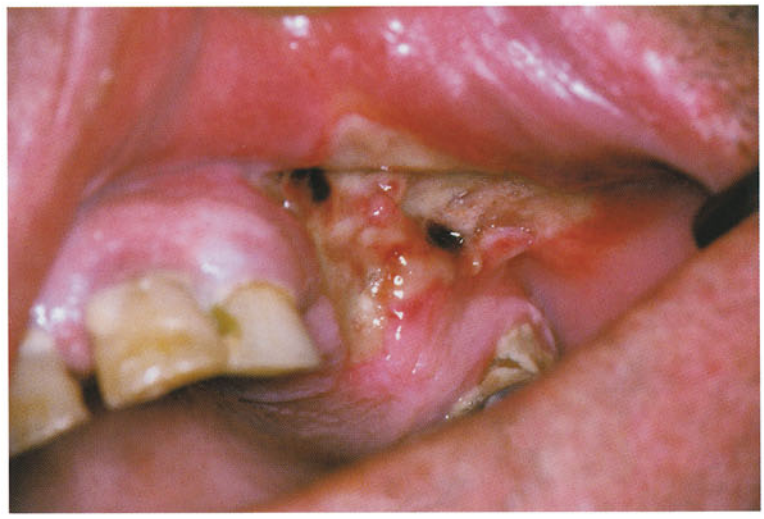


FIGURE 251

Cutaneous T-cell lymphoma. Perioral skin lesions

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Diagnosis
- The clinical diagnosis should be confirmed by laboratory tests.
- Biopsy and histopathological examination.
- Immunohistochemical examination.

Differential Diagnosis
- Non-Hodgkin's lymphoma
- Leukaemia
- Lupus erythematosus

Treatment
- Good oral hygiene
- Photochemotherapy, chemotherapy and radiation for the skin lesions

Hodgkin's Disease

Definition
- Hodgkin's disease is a malignant lymphoproliferative disorder.

Etiology
- Genetic factors, viruses and environmental factors and AIDS, may be involved in the pathogenesis.

Gingival Involvement
- Very rare

Other Sites of Involvement
- Oral mucosa, very rare.
- Lymph nodes, always.
- Extranodular involvement may rarely occur.

Main Clinical Features
- Localized gingival soft swelling or non-specific ulceration are the presenting signs on the gingiva (Fig. 252)
- The most frequent presenting feature is cervical lymph node (single or multiple) enlargement. The involved lymph nodes are firm, mobile and usually non-tender. Low-grade fever, night sweats, weight loss, anorexia, fatigue, malaise, weakness are common symptoms.

Diagnosis
- The clinical diagnosis should be confirmed by laboratory tests.
- Biopsy and histopathological examination.
- Immunohistochemical examination.
- Radiography and CT.

Differential Diagnosis
- Non-Hodgkin's lymphoma
- Leukaemias
- Plasmacytoma

Treatment
- Good oral hygiene
- Radiotherapy, chemotherapy
- Bone marrow transplantation

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 252

Hodgkin's disease. Localized ulceration on the gingiva in a patient with AIDS

Reading List

Anil S, Samaranayake L, Nair R, Beena VT (1996) Gingival enlargement as a diagnostic indicator in leukemia: case report. Aust Dent J 41:235–237

Barrett PA (1984) Gingival lesions in leukaemia: a classification. J Periodontol 55:585–588

Bergmann OJ (1992) Oral infections in haematological patients. Dan Med Bull 39:15–29

Bruce DW, Royer BQ (1995) Multiple myeloma occurring in the jaws: a study of 17 cases. Oral Surg Oral Med Oral Pathol 80:729–744

Colmenero C, Ganallo G, Pintado V et al (1991) AIDS-related lymphoma of the oral cavity. Int J Oral Maxillofac Surg 32:159–168

Crane RM, Heydt S (1979) Gingival involvement in mycosis fungoides. J Oral Surg 37: 585–587

Deasy M, Vogel R, Madedo-Sobrinho B et al (1980) Familial benign chronic neutropenia associated with periodontal disease. J Periodontol 51:206–210

Declerck D, Vinckier F (1988) Oral complications of leukemia. Quintessence Int 8:575–583

Dreizen S, McCredie KB, Keating MJ (1984) Chemotherapy-associated oral hemorrhages in adults with acute leukemia. Oral Surg Oral Med Oral Pathol 57:494–498

Epstein JB, Voss NJS, Stevenson-Moore P (1984) Maxillofacial manifestations of multiple myeloma. Oral Surg Oral Med Oral Pathol 57:267–271

Kinane D (1999) Blood and lymphoreticular disorders. Periodontol 2000 21:84–93

Kinane D (1999) Periodontitis modified by systemic factors. Ann Periodontol 4:54–63

Kirstila V, Sewon L, Laine J (1993) Periodontal disease in three siblings with familial neutropenia. J Periodontol 64:566–570

Laskaris G (2000) Color atlas of oral diseases in children and adolescents. Georg Thieme Verlag, Stuttgart

Laskaris G, Nicolis G, Capetanakis J (1978) Mycosis fungoides with oral manifestations. Oral Surg Oral Med Oral Pathol 46:40–42

Laskaris G, Potouridou I, Laskari M, Stratigos J (1992) Gingival lesions of HIV infection in 178 Greek patients. Oral Surg Oral Med Oral Pathol 74:168–171

Laskaris G, Stergiou G, Kittas C, Scully C (1992) Hodgkin's disease involving the gingiva in AIDS. Eur J Cancer 28B:39–41

Long LM, Jacowy JR, Bawden JW (1983) Cyclic neutropenia: case report of two siblings. Pediatr Dent 5:142–144

Luker J, Scully C, Oakhill A (1991) Gingival swelling as a manifestation of aplastic anemia. Oral Surg Oral Med Oral Pathol 71:55–56

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Lynch MA, Ship II (1967) Initial oral manifestations of leukemia. J Am Den Assoc 75:932–940

Ohishi M, Ooby K, Miyanoshita Y, Yamaguchi K (1988) Acute gingival necrosis caused by drug-induced agranulocytosis. Oral Surg Oral Med Oral Pathol 66:194–196

Page RC, Beck JD (1997) Risk assessment for periodontal diseases. Int Dent J 47:61–87

Pernu HE, Pajari UH, Lanning M (1996) The importance of regular dental treatment in patients with cyclic neutropenia. J Periodontol 67:454–459

Petit JC, Ripamonti U (1990) Multiple myeloma of the periodontium: a case report. J Periodontol 61:132–137

Porter SR (1998) Gingival and periodontal aspects of diseases of the blood and blood-forming organs and malignancy. Periodontol 2000 18:102–110

Porter SR, Luker J, Scully C, Oakhill A (1994) Oral features of a family with benign familial neutropenia. J Am Acad Dermatol 30:877–880

Porter SR, Matthews RW, Scully C (1994) Chronic lymphocytic leukemia with gingival and palatal deposits. J Clin Periodontol 21:559–561

Porter SR, Scully C (1994) Gingival and oral mucosal ulcerations as the initial features of myelodysplasia. Oral Oncology Eur J Cancer 30B:346–350

Rubin MM, Gatta CA, Cozzi GM (1989) Non-Hodgkin's lymphoma of the buccal gingiva as the initial manifestation of AIDS. J Oral Maxillofac Surg 47:1311–1313

Scully C, MacFadyen E, Campbell A (1982) Oral manifestations in cyclic neutropenia. Br J Oral Surg 20:96–101

Scully C, Gokbuget AY, Allen C et al. (2001) Oral manifestations indicative of plasminogen deficiency (hypoplasminogenemia). Oral Surg Oral Med Oral Pathol Oral Radiol Oral Endoc 91:344–347

Shrestha R, McKinley C, Bilir BM, Everson GT (1995) Possible idiopathic thrombocytopenic purpura associated with natural alfa interferon therapy for chronic hepatitic C infection. Am J Gastroenterol 90:1146–1146

Sirois DA, Miller AS, Harwick RD, Vonderheid EC (1993) Oral manifestations of cutaneous T-cell lymphoma. Oral Surg Oral Med Oral Pathol 75:700–705

Spencer P, Fleming JE (1985) Cyclic neutropenia: a literature and report a case. J Dent Child 52:108–113

Sydney SB, Serio F (1981) Acute monocytic leukemia diagnosed in a patient referred because of gingival pain. J Am Den Assoc 1981;103:886–887

Tamir R, Rick AI, Calderon S (1992) Plasmatocytoma of the mandible: a primary presentation of multiple myeloma. J Oral Maxillofac Surg 50:408–413

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

CHAPTER 22

Skin Diseases

Erythema Multiforme

Definition

- Erythema multiforme is an acute mucocutaneous disease that occurs most frequently in young males.

Etiology

- Several factors have been implicated in the etiology of erythema multiforme, including drugs (sulphonamides, penicillin, barbiturates, non-steroidal anti-inflammatory drugs, phenytoin and others), herpes simplex virus infection, *Mycoplasma pneumoniae* infection. However, in more than 50 % of the cases, a causative factor cannot be found.
- Two main forms of the disease are recognized:
  - Erythema multiforme minor features distinct skin lesions with or without mucosal involvement.
  - Erythema multiforme major, or Stevens-Johnson syndrome, which almost invariably involves the oral and other mucosae and the skin as well.

Gingival Involvement

- Relatively uncommon
- Non-plaque-induced lesions with no attachment loss

Other Sites of Involvement

- Oral mucosa (lips, buccal mucosa, tongue, palate), about 20 %–30 % in the minor form and almost invariably there in the major form of the disease.
- Other mucosae (conjunctiva, genitalia).
- Skin is very often affected in a symmetrical pattern (palms, soles, backs of the hands and feet, neck, trunk).

Main Clinical Features

- The gingival lesions may appear as diffuse erythema mimic desquamative gingivitis or localized or diffuse painful erosions covered by a whitish pseudomembrane (Figs. 253, 254).
- The oral mucosa lesions appear as small vesicles that rupture, creating extensive painful erosions covered by pseudomembranes and haemorrhagic crusts (Fig. 255).
- Photophobia, conjunctivitis, keratitis, uveitis and panophthalmitis may also occur.
- The skin lesions present as erythematous maculopapules, usually creating the characteristic target or iris lesions (Fig. 256). Less often purpuric plaques and bullous formation may appear, usually in association with target lesions.
- The disease recurs in 20 %–30 % of the cases.

For Personal Use Only
Library of School of Dentistry, TUMS

G. Laskaris et al., *Periodontal Manifestations of Local and Systemic Diseases*
© Springer-Verlag Berlin Heidelberg 2003

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 253

Erythema multiforme. Early lesions on the gingiva presenting as desquamative gingivitis

FIGURE 254

Erythema multiforme. Erythema and erosions of the gingiva mimic desquamative gingivitis

Diagnosis

- The diagnosis is usually based on clinical criteria. However, occasionally laboratory test should be done.
- Biopsy and histopathological examination.
- Direct immunofluorescence.

Differential Diagnosis

- Primary herpetic gingivostomatitis
- Herpetiform ulcers
- Cicatricial pemphigoid
- Bullous pemphigoid
- Linear IgA disease
- Pemphigoid gestations
- Pemphigus

Treatment

- Systemic corticosteroids
- Aciclovir or other new antivirals may be helpful in the management of patients with recurrent erythema multiforme

For Personal Use Only
Library of School of Dentistry, TUMS

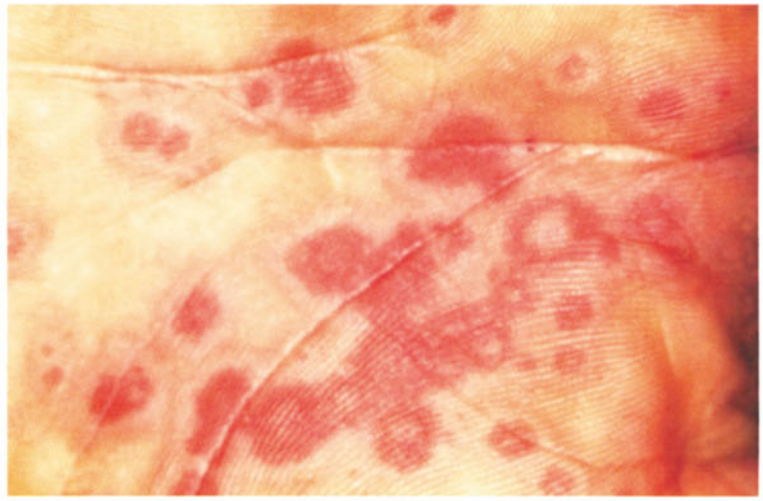
WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 255

Erythema multiforme. Extensive erosions on the tongue and lower lip

FIGURE 256

Erythema multiforme. Characteristic target-like lesions on the palm



For Personal Use Only
Library of School of Dentistry, TUMS

Toxic Epidermal Necrolysis

Definition
- Toxic epidermal necrolysis or Lyell's disease is a rare exfoliative mucocutaneous disease with high mortality.

Etiology
- Drug-induced reaction (sulphonamides, antibiotics, non-steroidal anti-inflammatory agents, anticonvulsants)

Gingival Involvement
- Common
- Non-plaque-induced lesions with no attachment loss

Other Sites of Involvement
- Oral mucosa (lips, buccal mucosa, tongue, palate)
- Other mucosae (conjunctiva, oropharynx, genitalia, anus)
- Skin (entire skin surface may be involved)

Main Clinical Features
- The disease usually commences with a cough, sore throat, burning eyes, low fever, malaise, followed after 1– 2 days by skin and mucous membrane lesions.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 257

Toxic epidermal necrolysis. Early lesions presenting as erythema and erosions on the gingiva mimic necrotizing ulcerative gingivitis

FIGURE 258

Toxic epidermal necrolysis. Sheet-like loss of the epidermis

For Personal Use Only
Library of School of Dentistry, TUMS

- The gingival lesions appear as dense inflammation, with blister formation leading to painful widespread erosions (Fig. 257).
- The oral lesions are characterized by diffuse erythema, blistering and painful, widespread erosions.
- The skin lesions appear as a sheet-like loss of epidermis (Fig. 258). The epidermis is raised by flaccid blisters, which spread on pressure. Nikolsky's sign is positive.

Diagnosis

- The diagnosis is usually made on clinical level.
- Biopsy and histopathological examination.

Differential Diagnosis

- Erythema multiforme major
- Pemphigus
- Staphylococcal scalded skin syndrome

Treatment

- Patients must be admitted as soon as possible to an intensive care unit for management.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Cicatricial Pemphigoid

Definition
- Cicatricial pemphigoid or benign mucous membrane pemphigoid is a chronic bullous disease that primarily affects mucous membranes and rarely the skin.
- The disease affects more frequently females than males (1.5:1), usually during the 5th to 6th decade of life.

Etiology
- Autoimmune

Gingival Involvement
- Very common, approximately 60 %
- Non-plaque-induced lesions with no attachment loss

Other Sites of Involvement
- Oral mucosa (soft palate, buccal mucosa, tongue, lips), 100 %
- Other mucosae (ocular, nose, genitalia, pharynx, larynx, anus)
- Skin, very rare (5 %)

Main Clinical Features
- Frequently the disease affects exclusively the gingiva in the form of desquamative gingivitis (erythema, oedema, desquamation of the epithelium or bullous formation after pressure of the gingiva) (Figs. 259–261).
- The oral mucosa lesions present as erythema and bullae formation that rupture quickly, leaving painful erosions (Fig. 262).
- The lesions usually recur and persist for a long time, leading occasionally to atrophy or scarring (except gingiva).
- The ocular lesions consist of conjunctivitis, symblepharon, trichiasis, dryness and opacity of the cornea, occasionally leading to complete blindness (Fig. 263).

Diagnosis
- The clinical diagnosis should be confirmed by laboratory tests.
- Biopsy and histopathological examination.
- Direct immunofluorescence.
- Indirect immunofluorescence.

Differential Diagnosis
- Lichen planus
- Bullous pemphigoid
- Pemphigus
- Linear IgA disease
- Epidermolysis bullosa acquisita
- Chronic ulcerative stomatitis
- Plasma cell gingivitis
- Herpetic gingivostomatitis

Treatment
- Corticosteroid topical or systemic depending on disease activity
- Immunosuppressive treatment (azathioprine, ciclosporin) only in severe and persistent cases, always as adjuvant to corticosteroid therapy

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 259

Cicatricial pemphigoid. Early lesion on the gingiva presenting as small haemorrhagic bulla formation

FIGURE 260

Cicatricial pemphigoid. Typical desquamative gingivitis

FIGURE 261

Cicatricial pemphigoid. Localized lesions on the gingiva in the form of desquamative gingivitis

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 262

Cicatricial pemphigoid. Extensive erosions on the palate

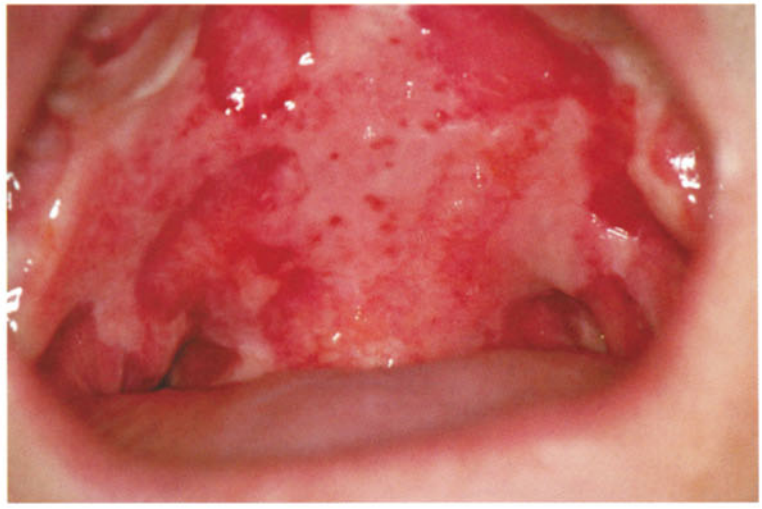


FIGURE 263

Cicatricial pemphigoid. Conjunctivitis and symblepharon

For Personal Use Only
Library of School of Dentistry, TUMS

Bullous Pemphigoid

Definition

- Bullous pemphigoid is a chronic bullous disease that primarily affects the skin and less often the oral mucosa (30%–40%).
- The disease affects more frequently females than males (1.7:1), usually during the 5th to 6th decade of life.

Etiology

- Autoimmune

Gingival Involvement

- Relatively common, about 10%–15%
- Non-plaque-induced lesions with no attachment loss

Other Sites of Involvement

- Oral mucosa (palate, buccal mucosa, tongue, lips), about 30%–40%
- Other mucosae (conjunctiva, oesophagus, vagina, anus)
- Skin, always

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 264

Bullous pemphigoid. Slight erythema of the gingiva and small haemorrhagic bulla formation

FIGURE 265

Bullous pemphigoid. Multiple small haemorrhagic bullae on the gingiva

FIGURE 266

Bullous pemphigoid. Small bullae on the skin

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Main Clinical Features

- Gingival involvement usually appears in the form of desquamative gingivitis (erythema, oedema, desquamation of the epithelium) or as a localized bullous formation that ruptures quickly, leaving painful erosions (Figs. 264, 265).
- Localized or widespread lesions may also appear in other oral mucosa sites.
- The skin lesions usually begin as a non-specific generalized rash and ultimately tense bullae develop isolated or in clusters that rupture, leaving erosions without a tendency to extend peripherally (Fig. 266).
- Nikolsky's sign is negative.
- The disease has a chronic course with exacerbations and remissions and ultimately good prognosis.

Diagnosis

- The clinical diagnosis should be confirmed by laboratory tests.
- Biopsy and histopathological examination.
- Direct immunofluorescence.

Differential Diagnosis

- Cicatricial pemphigoid
- Linear IgA disease
- Pemphigoid gestationis
- Epidermolysis bullosa acquisita
- Pemphigus
- Plasma cell gingivitis

Treatment

- Corticosteroid topical and/or systemic
- Immunosuppressives as adjuvant therapy

Linear IgA Disease

Definition

- Linear IgA disease is a rare vesiculobullous mucocutaneous disorder characterized by linear homogeneous deposition of IgA along the dermoepidermal junction on direct immunofluorescence testing.
- The disease is more common in females than males, with an average onset between 40 and 50 years of age. Children may also be affected.

Etiology

- Autoimmune

Gingival Involvement

- Rare, approximately 5 %–10 %
- Non-plaque-induced lesions with no attachment loss

Other Sites of Involvement

- Oral mucosa (about 20 %–25 %)
- Other mucosae (conjunctiva, nasal, larynx) rarely
- Skin, common, in a symmetrical and cluster form distribution (perioral, genital, buttocks, arms)

Main Clinical Features

- Gingival involvement may appear either in the form of desquamative gingivitis or as a localized bullous formations that rupture, leaving quickly non-specific painful erosions (Figs. 267, 268).
- Localized or widespread lesions may also develop in other oral mucosa sites.
- Skin lesions are characterized by pruritus and vesicles distributed in a symmetrical pattern (Fig. 269).
- The disease has a chronic course and the clinical manifestations are indistinguishable from those seen in cicatricial pemphigoid.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 267

Linear IgA disease. Gingival lesions presenting as desquamative gingivitis

FIGURE 268

Linear IgA disease. Erythema and bullous formation in the form of desquamative gingivitis

FIGURE 269

Linear IgA disease. Small vesicles on the skin

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Diagnosis
- The clinical diagnosis should be confirmed by laboratory tests.
- Biopsy and histopathological examination.
- Direct immunofluorescence.
- Indirect immunofluorescence.

Differential Diagnosis
- Cicatricial pemphigoid
- Bullous pemphigoid
- Epidermolysis bullosa acquisita
- Pemphigoid gestationis
- Pemphigus

Treatment
- Local and systemic corticosteroids
- Dapsone and sulphapyridine

Pemphigoid Gestationis

Definition
- Pemphigoid gestationis or herpes gestationis is a distinct, rare and recurrent bullous dermatosis of pregnancy and the immediate postpartum period.

Etiology
- Unknown. It is currently thought to be immunologically mediated (autoimmune).

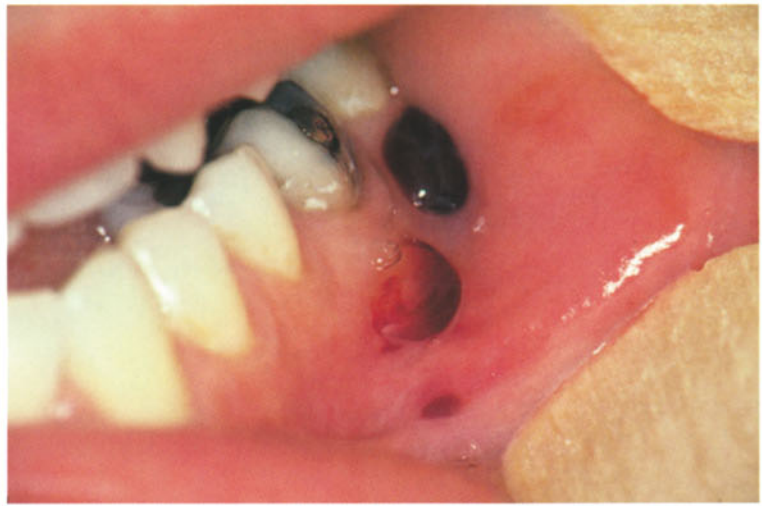
Gingival Involvement
- Very uncommon
- Non-plaque-induced lesions with no attachment loss

Other Sites of Involvement
- Oral mucosa (buccal mucosa, tongue, palate), very rare
- Skin (abdomen, chest, back, face, extremities, palms, soles)

Main Clinical Features
- Gingival lesions may appear as localized haemorrhagic bullae that very soon rupture leaving painful erosions (Fig. 270).
- Multiple bullae that quickly rupture, leaving non-diagnostic painful erosions, may appear on the oral mucosa. The oral lesions always follow skin lesions and quickly retreat.

FIGURE 270

Pemphigoid gestationis. Haemorrhagic bullae on the gingiva



For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 271

Pemphigoid gestationis. Numerous vesicles on the skin

- The skin manifestations are characterized by small papulovesicular lesions that are often grouped and commonly occur on a lesion of erythema and/or urticaria. Numerous widespread vesicles and crusting are common (Fig. 271). Characteristically, all these lesions are extremely pruritic.
- The eruptions usually appear during the second and the third trimester of pregnancy or immediately postpartum and resolve within 3–4 months after delivery.
- Rarely the disease may arise in association with trophoblastic malignancy.

Diagnosis

- The clinical diagnosis should be confirmed by laboratory tests.
- Biopsy and histopathological examination.
- Direct immunofluorescence.
- Indirect immunofluorescence.

Differential Diagnosis

- Bullous pemphigoid
- Linear IgA disease
- Dermatitis herpetiformis
- Pemphigus

Treatment

- Corticosteroid systematically. Azathioprine may also be used as adjuvant therapy.

Epidermolysis Bullosa Acquisita

Definition

- Epidermolysis bullosa acquisita is a rare chronic mechanobullous mucocutaneous disease.

Etiology

- Autoimmune

Gingival Involvement

- Rare
- Non-plaque-induced lesions with no attachment loss

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 272

Epidermolysis bullosa acquisita. Erythema and small vesicle on the gingiva in the form of desquamative gingivitis

FIGURE 273

Epidermolysis bullosa acquisita. Erythema and scarring on the skin

Other Sites of Involvement

- Oral mucosa (buccal mucosa, palate, tongue, lips).
- Skin, over the joints, extended.
- Other mucosae (conjunctival, nasal, oesophageal) are rarely involved.
- Surfaces of the knees, feet, elbows, ankles and hands, nails.

Main Clinical Features

- Gingival involvement may appear either in the form of desquamative gingivitis or as localized bullous formations that rupture, leaving quickly non-diagnostic painful erosions (Fig. 272).
- Localized and, rarely, widespread bullae and erosions may develop, particularly in friction-associated areas of the oral mucosa.
- The skin lesions are characterized by haemorrhagic bullae spontaneously or after mild trauma. The bullae rupture, leaving ulcers that may heal with scarring (Fig. 273). Milia, atrophic areas, hyperpigmentation and nail dystrophy may occur.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Diagnosis
- The clinical diagnosis should be confirmed by laboratory tests.
- Biopsy and histopathological examination.
- Direct immunofluorescence.
- Electron microscopy.

Differential Diagnosis
- Cicatricial pemphigoid
- Bullous pemphigoid
- Linear IgA disease
- Pemphigus

Treatment
- Local and systemic corticosteroid
- Dapsone, azathioprine
- Symptomatic and avoid trauma
- Local and/or systemic antibiotics and antiseptics

Dermatitis Herpetiformis

Definition
- Dermatitis herpetiformis, or Duhring-Brocq disease, is a rare chronic, recurrent pruritic vesicular skin disease.
- The disease is more common between 20 and 50 years of age and males are more frequently affected than females.

Etiology
- Unknown. However, it is thought that immunological mechanisms may be involved in the pathogenesis.
- A strong association with HLA-B_8, HLAA_1, HLA-DR_3, HLA-DW_3, HLA-DW_2 has been found.
- Gluten hypersensitivity plays an important role in the pathogenesis.

Gingival Involvement
- Rare
- Non-plaque-induced gingival lesions with no attachment loss

Other Sites of Involvement
- Oral mucosa (palate, tongue, buccal mucosa, lips) approximately 5%–10%
- Skin (in a symmetrical distribution of the extensor surfaces of the elbows, knees, shoulders, buttocks, posterior neck, scale), always

Main Clinical Features
- Gingival and other oral mucosal lesions appear as localized whitish maculopapular lesions, erythema and formation of bullae that soon rapture, leaving non-specific painful erosions (Figs. 274, 275).
- The oral lesions usually follow the skin eruption.
- The skin lesions appear as erythematous papules or plaques accompanied by severe burning and pruritus as well as small vesicles coalescing in groups in a herpes-like pattern (Fig. 276).
- The disease runs a very prolonged course with remissions and exacerbations.

Diagnosis
- The clinical diagnosis should always be confirmed by laboratory tests.
- Biopsy and histopathological examination.
- Direct immunofluorescence.
- Indirect immunofluorescence.

Differential Diagnosis
- Bullous pemphigoid
- Linear IgA disease
- Pemphigoid gestations
- Pemphigus

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 274

Dermatitis herpetiformis. Erythema and erosions on the gingiva and small bulla on the labial mucosa

FIGURE 275

Dermatitis herpetiformis. Whitish maculopapular lesions on the gingiva and the alveolar mucosa

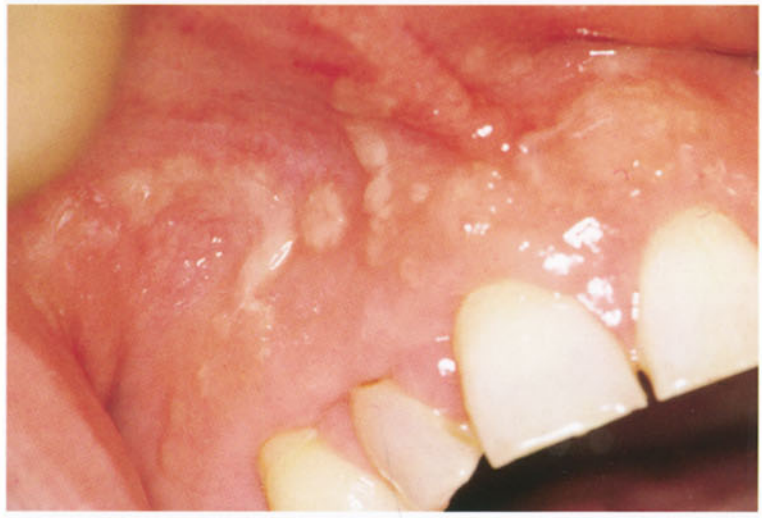
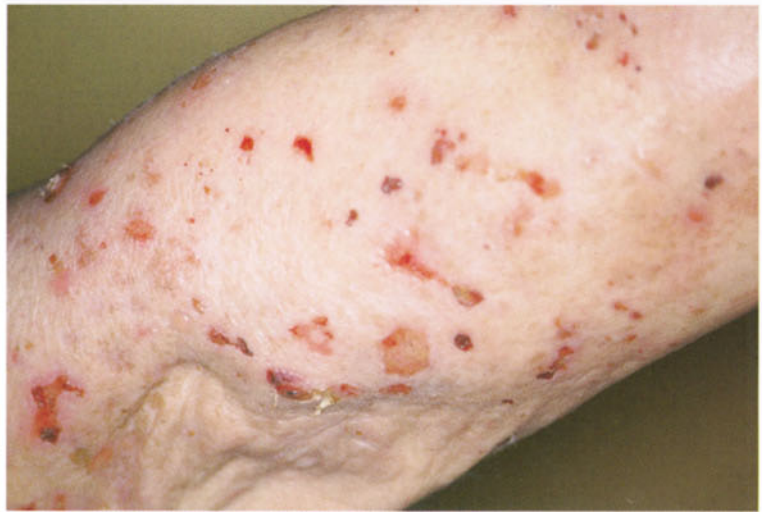


FIGURE 276

Dermatitis herpetiformis. Group of vesicles on the skin in a herpes-like pattern



For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

- Erythema multiforme
- Aphthous ulcers
- Herpetic gingivostomatitis

Treatment

- Gluten-free diet.
- Sulphones and sulphapyridines.
- Corticosteroid in persistent cases.
- Tetracyclines and nicotinamide have also been used.

Pemphigus Vulgaris

Definition

- Pemphigus vulgaris is a chronic mucocutaneous bullous disease.

Etiology

- Autoimmune

Gingival Involvement

- Common, approximately 20 %–30 %
- Non-plaque-induced lesions with no attachment loss

Other Sites of Involvement

- Oral mucosa (soft palate, buccal mucosa, tongue, floor of the mouth, lips), very common 75 %–90 %
- Other mucosae (conjunctiva, nose, genital, rectal, larynx, pharynx)
- Skin

Main Clinical Features

- Gingival lesions may appear either in the form of desquamative gingivitis usually localized or as painful erosions on an erythematous base. Very rarely, gingival lesions may be the only site of involvement (Figs. 277–279).
- The oral mucosa lesions preset as erythema, bullous formation, painful erosions (Fig. 280).
- The skin lesions present as bullous formation that soon rapture, leaving painful erosions.
- Nikolsky's sign positive.

Diagnosis

- The clinical diagnosis should always be confirmed histologically and immunologically.
- Biopsy and histopathological examination.
- Direct immunofluorescence.
- Indirect immunofluorescence.
- Tzank smear test.

Differential Diagnosis

- Cicatricial pemphigoid
- Bullous pemphigoid
- Pemphigoid gestations
- Linear IgA disease
- Dermatitis herpetiformis
- Erythema multiforme
- Epidermolysis bullosa acquisita
- Herpetic gingivostomatitis
- Aphthous ulcers

Treatment

- High dose of corticosteroids
- Azathioprine, ciclosporin, mycophenolate mofetil as adjuvant treatment
- Plasmapheresis

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 277

Pemphigus vulgaris. Early lesions on the gingiva presenting as erythema

FIGURE 278

Pemphigus vulgaris. Erythema and erosions on the gingiva in the form of desquamative gingivitis

FIGURE 279

Pemphigus vulgaris. Erosions on the gingiva and the alveolar mucosa

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 280

Pemphigus vulgaris. Extensive chronic erosions on the tongue and lower lip

Lichen Planus

Definition

- Lichen planus is a common, chronic inflammatory mucocutaneous disease, affecting approximately 0.5%–2.5% of the population.
- Females are affected more often than males, usually between 40 and 60 years of age.

Etiology

- Unknown. Current evidence suggests that there is a local cell-mediated immune response with a T lymphocyte infiltrate.

Gingival Involvement

- Common, approximately 25%–40%
- Non-plaque-induced lesions with no attachment loss

Other Sites of Involvement

- Oral mucosa (buccal mucosa, tongue, lips, palate), approximately 50%–70% of the cases
- Other mucosae (vulva, vagina, penis)
- Skin (usually on the flexor surfaces of the forearms and wrists, the back, the sacral area, nails), commonly in a symmetrical pattern

Main Clinical Features

- Seven clinical forms of oral lichen planus have been described:
  - Papular.
  - Reticular.
  - Erosive.
  - Atrophic.
  - Hypertrophic.
  - Bullous.
  - Pigmented.
- Most patients with oral lichen planus present with a combination of the various clinical forms. Polygonal white papules and lines are the basic clinical disorder of the disease.
- Frequently the disease affects the gingiva and occasionally can be the initial or the only sign of oral involvement. Clinically, gingival lesions may appear in the form of desquamative gingivitis (erythema, oedema, desquamation of the epithelium or vesicle formation after pressure, and sensitive) (Figs. 281–283). White papules and lines, erosions or atrophy may also be found (Fig. 284).

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 281

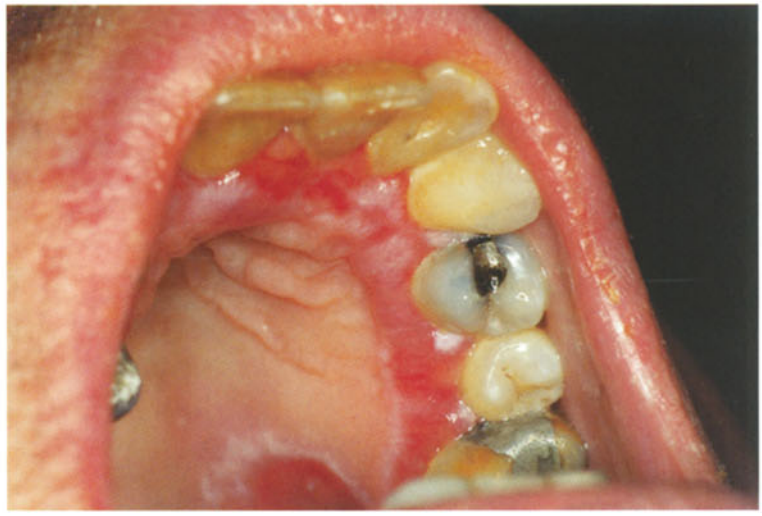
Lichen planus. Erythema and white lines on the gingiva in the form of desquamative gingivitis

FIGURE 282

Lichen planus. Typical lesions of desquamative gingivitis

FIGURE 283

Lichen planus. Diffuse erythema on the palatal gingiva in the form of desquamative gingivitis



For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 284

Lichen planus. Characteristic papules and lines on the gingiva

FIGURE 285

Lichen planus. Typical Wickham's striae on the buccal mucosa

For Personal Use Only
Library of School of Dentistry, TUMS

- On the oral mucosa, small white papules that usually coalesce and form lines (Wickham's striae) and network of lines (Fig. 285). Annular distribution of the papules, painful erosions, atrophic or hypertrophic areas, bullous or pigmented lines may also be seen, depending on the clinical form.
- The skin lesions appear as small, red or violaceous flat, polygonal, shiny and pruritic papules.

Diagnosis

- The diagnosis is usually based on clinical criteria, but tests that may be supportive include:
- Biopsy and histopathological examination.
- Direct immunofluorescence.

Differential Diagnosis

- Cicatricial pemphigoid
- Lupus erythematosus
- Cinnamon contact stomatitis
- Lichen planus reaction due to drugs

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

- Lichenoid reaction to dental restorative material
- Graft-versus-host disease, lichen planus reactions
- Plasma cell gingivitis

Treatment

- Good oral hygiene and avoid pressure on the gingiva.
- Corticosteroid topically in an adhesive base.
- Corticosteroid systemically in severely painful lesions.
- Dapsone, ciclosporin, tacrolimus and azathioprine may also be used in severe cases.

Chronic Ulcerative Stomatitis

Definition

- Chronic ulcerative stomatitis is a rare autoimmune disease that resembles, clinically and histopathologically, lichen planus and lupus erythematosus but possesses a unique immunofluorescent pattern.

Etiology

- Autoimmune

Gingival Involvement

- Common
- Non-plaque-induced gingival lesions with no attachment loss

Other Sites of Involvement

- Oral mucosa, commonly the buccal mucosa and the tongue

Main Clinical Features

- The gingival lesions usually appear in the form of desquamative gingivitis or as localized painful erythema and erosions (Fig. 286).
- The oral mucosa lesions appear as painful erosions usually associated with white reticular lesions identical to those seen in oral lichen planus and discoid lupus erythematosus (Fig. 287).

Diagnosis

- The clinical diagnosis should be confirmed by laboratory tests.
- Biopsy and histopathological examination.
- Direct immunofluorescence.
- Indirect immunofluorescence.
- Both in vivo binding to the oral mucosa and skin of a stratified epithelium-specific antinuclear antibody and high titres of these antibodies in serum are characteristic markers of this disease.

FIGURE 286

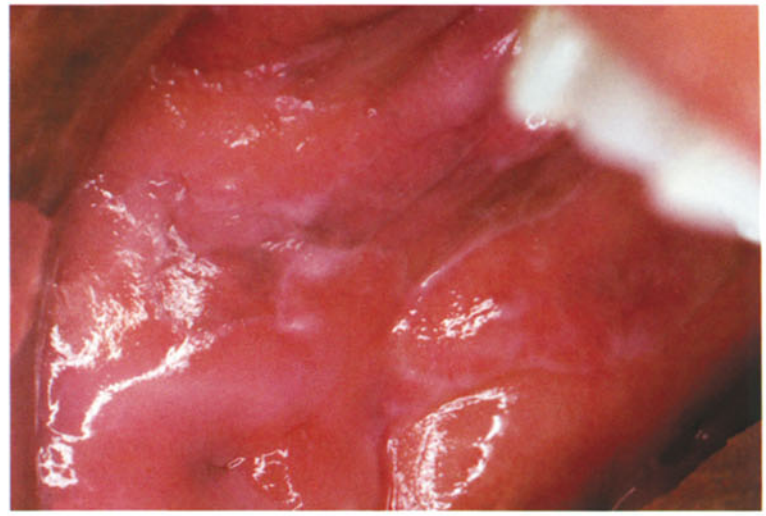
Chronic ulcerative stomatitis. Erythema and erosions on the gingiva in the form of desquamative gingivitis

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 287

Chronic ulcerative stomatitis. White lesions on the buccal mucosa mimic lichen planus



Differential Diagnosis
- Lichen planus
- Discoid lupus erythematosus
- Cicatricial pemphigoid
- Linear IgA disease
- Bullous pemphigoid
- Epidermolysis bullosa
- Pemphigus

Treatment
- Systemic or local corticosteroid
- Hydroxychloroquine sulphate (Plaquenil) and other antimalarial agents

Psoriasis

Definition
- Psoriasis is a common, chronic and recurring inflammatory skin disease.

Etiology
- Unknown

Gingival Involvement
- Very rare
- Non-plaque-induced lesions with no attachment loss

Other Sites of Involvement
- Oral mucosa (tongue, buccal mucosa, lips), rare (approximately 2%–4%)
- Skin (elbows, knees, scalp, lumbar area, nails)

Main Clinical Features
- The gingival lesions may resemble desquamative gingivitis; however, desquamation of the epithelium after pressure does not occur (Fig. 288). This pattern is an exception phenomenon.
- Oral lesions may appear as atypical erythema, white or greyish plaques and more often as circinate lesions mimicking geographic tongue. The oral lesions that usually develop after skin involvement are not diagnostic and a histopathological examination is needed to confirm diagnosis.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 288

Psoriasis.
Unusual involvement of the gingiva in the form of desquamative gingivitis

- Skin lesions are characterized by well-demarcated erythematous plaques with silvery scales on the surface. The lesions are usually asymptomatic.

Diagnosis

- The diagnosis is predominantly clinical but biopsy and histopathological examination are supportive.

Differential Diagnosis

- Geographic tongue and stomatitis
- Cinnamon contact stomatitis
- Lichen planus
- Cicatricial pemphigoid

Treatment

- The gingival lesions should be treated with good oral hygiene and topical corticosteroids.
- The treatment of the skin lesions must be left to the dermatologists.

Reading List

Barrett AW, Scully CM, Eveson JW (1993) Erythema multiforme involving gingiva. J Periodontol 64:910–913

Beutner EH, Chorzelski TP, Parodi A et al (1991) Ten cases of chronic ulcerative stomatitis with stratified epithelium-specific antinuclear antibody. J Am Acad Dermatol 24:781–782

Bystryn JC (1996) Erythema multiforme with mucous membrane involvement and Stevens-Johnson syndrome are clinically different disorders. Arch Dermatol 132:711–712

Chen SH, Chopra K, Evans TY et al (1999) Herpes gestations in a mother and child. J Am Acad Dermatol 40:847–849

Chiossi M, Costa RS, Roselino AF (2000) Titration of herpes gestations factor fixing to C_3 in pemphigoid herpes gestationis associated with choriocarcinoma. Arch Dermatol 136:129–130

Economopoulou P, Laskaris G (1986) Dermatitis herpetiformis: oral lesions as an early manifestations. Oral Surg Oral Med Oral Pathol 62:77–80

Holmstrup P (1999) Non-plaque-induced gingival lesions. Ann Periodontol 4:20–29

Jadinski JJ, Shklar G (1976) Lichen planus of the gingiva. J Periodontol 47:723–733

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Jarmsko WM, Beutner EH, Kumar V et al (1990) Chronic ulcerative stomatitis associated with a specific immunologic marker. J Am Acad Dermatol 22:215–220
Jones LE, Dolby AE (1972) Desquamative gingivitis associated with psoriasis. J Periodontol 43:35–37
Laskaris G (1981) Oral pemphigus vulgaris: an immunofluorescent study of fifty-eight cases. Oral Surg Oral Med Oral Pathol 57:626–631
Laskaris G, Angelopoulos A (1981) Cicatricial pemphigoid: direct and indirect immunofluorescent studies. Oral Surg Oral Med Oral Pathol 51:48–54
Laskaris G, Demetriou N, Angelopoulos A (1981) Immunofluorescent studies in desquamative gingivitis. J Oral Pathol 10:398–407
Laskaris G, Nicolis G (1980) Immunopathology of oral mucosa in bullous pemphigoid. Oral Surg Oral Med Oral Pathol 50:340–345
Laskaris G, Papanicolaou S, Angelopoulos A (1981) Immunofluorescent study of cytologic smears in oral pemphigus: a simple diagnostic technique. Oral Surg Oral Med Oral Pathol 51:531–534
Laskaris G, Satriano RA (1993) Drug-induced blistering oral lesions. Clin Dermatol 11: 545–550
Laskaris G, Sklavounou A, Angelopoulos A (1982) Direct immunofluorescent studies in oral lichen planus. Oral Surg Oral Med Oral Pathol 54:483–487
Laskaris G, Sklavounou A, Stratigos J (1988) Bullous pemphigoid, cicatricial pemphigoid and pemphigus vulgaris: a comparative clinical study of the 178 cases. Oral Surg Oral Med Oral Pathol 54:483–487
Laskaris G, Triantafyllou A, Economopoulou P (1988) Gingival manifestations of childhood cicatricial pemphigoid. Oral Surg Oral Med Oral Pathol 66:349–352
Lewis JE, Beutner EH, Rostami R, Chrzelsky TP (1996) Chronic ulcerative stomatitis with stratified epithelium-specific antinuclear antibodies. Int J Dermatol 35:272–275
Lozada-Nur F, Gorsky M, Silverman S (1989) Oral erythema multiforme: clinical observations and treatment of 95 patients. Oral Surg Oral Med Oral Pathol 67:36–40
Markitziu A, Pisanty S (1983) Gingival pemphigus vulgaris: report of a case. Oral Surg Oral Med Oral Pathol 55:250–252
Matsumara Y (1993) Epidermolysis bullosa acquisita with non-classical distribution of eruptions. J Dermatol 20:159–163
Nausari HC, Anhalt GJ (1995) Bullous skin diseases. Curr Opinion Immunol 7:844–852
Nisengard RJ, Chorzelski T, Mariejowska E, Kryst L (1982) Dermatitis herpetiformis: IgA deposits in gingiva, buccal mucosa and skin. Oral Surg Oral Med Oral Pathol 54:22–25
Nisengard RJ, Levine RA (1995) Diagnosis and management of desquamative gingivitis. Periodontol Insights 2:4–10
Plemons JM, Gonzales TS, Burkhart NW (1999) Vericulobullous diseases of the oral cavity. Periodontology 2000 121:158–175
Porter SR, Bain SE, Scully CM (1991) Linear IgA disease manifesting as recalcitrant desquamative gingivitis. Oral Surg Oral Med Oral Pathol Oral Radiol Oral Endoc 74: 179–182
Rasmussen JE (1995) Erythema multiforme, Stevens-Johnson syndrome, and toxic epidermal necrolysis. Dermatol Nursing 7:37–43
Satoh S, Seishima M, Sawada Y et al (1999) The time course of the change in antibody titres in herpes gestations. Br J Dermatol 140:119–123
Sciubba JJ (1996) Autoimmune aspects of pemphigus vulgaris and mucosal pemphigoid. Adv Dent Res 10:52–56
Scully C, Beyli M, Ferreiro MC et al (1998) Update on oral lichen planus: etiopathogenesis and management. Crit Rev Oral Biol Med 9:86–122
Scully C, Laskaris G (1998) Periodontal manifestations of mucocutaneous disorders. Periodontology 2000 18:81–94
Scully C, Porter SR (1997) The clinical spectrum of desquamative gingivitis. Semin Cutan Med Surg 16:308–313
Scully C, Eisen D, Carrozzo M (2000) Management of oral lichen planus. Am J Clin Dermatol 1:287–306
Scully C, Carrozzo M, Gandolfo S et al (1999) Update on mucous membrane pemphigoid (an immune mediated sub-epithelial blistering disease): a heterogeneous entity. Oral Surg Oral Med Oral Pathol 88:56–68

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Sklavounou A, Laskaris G (1983) Frequency of desquamative gingivitis in skin diseases. Oral Surg Oral Med Oral Pathol 56:141–144
Sklavounou A, Laskaris G (1990) Oral psoriasis: report of a case and review of the literature. Dermatologica 180:157–159
Sklavounou A, Laskaris G (1990) Childhood cicatricial pemphigoid with exclusive gingival involvement. J Oral Maxillofac Surg 19:197–199
Triffet MK, Gibson LE, Leiferman KM (1999) Severe subepidermal blistering disorder with features of bullous pemphigoid and herpes gestations. J Am Acad Dermatol 40:797–801
Vaillant L, Arbeille B, Goga D et al (1990) Cicatricial pemphigoid disclosed by superficial desquamative gingivitis. Clinical and immuno-electron microscopic study of a case. Ann Dermatol Venereol 117:613–620

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

CHAPTER 23

Autoimmune Diseases

Lupus Erythematosus

Definition

- Lupus erythematosus is a chronic inflammatory autoimmune disease with a variable spectrum of clinical forms, in which mucocutaneous clinical manifestations may occur in association with or without multiple organ systems involvement.

Etiology

- It is thought that a genetically susceptible autoimmune mechanism triggered by environmental factors is responsible for the pathogenesis of the disease.

Classification

- The disease is classified into two major forms:
  - Discoid.
  - Systemic.

Gingival Involvement

- Rare
- Non-dental plaque-induced gingival lesions

Other Involvement

- Oral mucosa (15%–25% in discoid form and 30%–45% in systemic form)
- Other mucosae (conjunctiva, nose, genital)
- Skin
- Other organ systems (kidney, cardiovascular system, neurological system, joints)

Main Clinical Features

- The gingival lesions may appear either as localized erythema, usually associated with erosions that may occasionally mimic desquamative gingivitis (Figs. 289, 290). Less frequently lupus erythematosus may present as a non-specific painful ulceration surrounded by whitish spots or lines (Fig. 291).
- The oral mucosa lesions appear as well-defined atrophic red plaques usually surrounded by a sharp elevated border of radiating whitish striae. Erosions, atrophy, telangiectasias, white plaques and xerostomia may also occur.
- The skin lesions are characterized by flat or slightly elevated erythematous papules and plaques with scaling and follicular hyperkeratosis. Many patients in both forms develop the classic „butterfly" rash on the face.
- Systemic lupus erythematosus is characterized by a broad spectrum of clinical and laboratory diagnostic criteria. Constitutional symptoms include fatigue, malaise, fever and weight loss associated with arthralgia and myalgia.

For Personal Use Only
Library of School of Dentistry, TUMS

G. Laskaris et al., *Periodontal Manifestations of Local and Systemic Diseases*
© Springer-Verlag Berlin Heidelberg 2003

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 289

Lupus erythematosus. Irregular erythema and erosions as early lesions on the gingiva

FIGURE 290

Lupus erythematosus. Erosions on the gingiva

FIGURE 291

Lupus erythematosus. Localized erosions on the gingiva

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Diagnosis
- The clinical diagnosis should be confirmed by laboratory tests.
- Biopsy and histopathological examination of oral and skin lesions.
- Direct and indirect immunofluorescence.

Differential Diagnosis
- Lichen planus
- Cicatricial pemphigoid
- Bullous pemphigoid
- Linear IgA disease
- Pemphigus
- Mixed connective tissue disease

Treatment
- Good dental plaque control.
- Topical corticosteroids.
- Antimalarials.
- Systemic corticosteroids.
- The treatment of systemic lupus erythematosus should be left to the specialist.

Sjögren's Syndrome

Definition
- Sjögren's syndrome is a relatively common, chronic autoimmune disorder. The disease is more common in women between 40 and 60 years of age.

Etiology
- Obscure. However, genetic factors, viral infection and autoimmunity may be involved in the pathogenesis.

Classification
- Two forms are recognized:
  - Primary (sicca syndrome) with mainly oral and eye disorders.
  - Secondary, if it coexists with other autoimmune disease, usually rheumatoid arthritis.

Gingival Involvement
- Very rare
- Dental plaque-induced gingival lesions modified by systemic factors

Other Involvement
- Salivary glands
- Lacrimal glands
- Other exocrine glands
- Other organs and systems

Main Clinical Features
- Chronic gingivitis, localized or generalized due to xerostomia and/or candida infection may occur (Fig. 292). Rarely increased alveolar bone loss may also be seen.
- Xerostomia is the hallmark of the disease. The oral mucosa is reddish, dry, smooth, and shiny (Fig. 293). Oral soreness, discomfort and dysphagia are common symptoms. Candidiasis, angular cheilitis, and dental caries are relatively common. Recurrent swelling of parotid and other major salivary glands may occur.
- The ocular manifestations characterized by dryness of the eyes, keratoconjunctivitis sicca and more severe lesions as the disease progress
- In the secondary form of the disease, the clinical manifestations depend on the associated autoimmune diseases.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 292

Sjögren's syndrome. Generalized chronic gingivitis

FIGURE 293

Sjögren's syndrome. Dry, reddish and shiny dorsum of the tongue

Diagnosis

- The clinical diagnosis should be confirmed by laboratory tests.
- Biopsy and histopathological examination of the minor salivary glands.
- Serological tests:
  - Antinuclear antibodies.
  - Anti-SSA (Ro).
  - Anti-SSB (La) antibodies.
  - Rheumatoid factor.

Differential Diagnosis

- Graft-versus-host disease
- Xerostomia due to drugs
- Systemic sclerosis
- Mixed connective tissue disease

Treatment

- Good dental plaque control
- Saliva and tear substitutes
- Antimalarial drugs, corticosteroid, immunosuppressants

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Systemic Sclerosis

Definition

- Systemic sclerosis is a chronic multisystemic inflammatory connective tissue disorder with a broad spectrum of manifestations.

Etiology

- Obscure. However, an autoimmune pathogenesis may be involved. In vitro studies suggest that the products of the autoimmune reaction may influence the behaviour of fibroblasts and endothelial cells leading to the overproduction of connective tissue by fibroblasts.

Gingival Involvement

- Rare
- Non-dental plaque-induced gingival lesions

Other Involvement

- Oral mucosa (relatively common)
- Skin (always)
- Viscera (esophagus, intestinal tract, kidney, lungs, heart, skeletal muscles)

Main Clinical Features

- The gingival lesions are minor and characterized by pallor of free and attached gingiva, gingival regression and very rarely greater evidence of periodontitis (Fig. 294). However, other studies revealed no significant differences in periodontal status between systemic sclerosis patients and healthy controls.
- The oral mucosal lesions include:
  - Xerostomia.
  - Thin and pale oral mucosa.
  - Short and hard lingual frenulum.
  - Atrophy of the tongue papillae.
  - Microglossia.
  - Microstomia.
  - Limited mouth opening.
  - Perioral radial folds.
- On dental radiographs, a diffuse widening of the periodontal ligament space may be seen approximately in 10 %–20 % of patients. Difficulty in swallowing, dysphagia are also common.
- The skin lesions are characterized initially by oedema but as the disease progresses the skin becomes thin, hard, inelastic and pale. Facial

FIGURE 294

Systemic sclerosis. Pallor and thin gingivae

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

skin involvement results in the characteristic mask-like facies. Skin ulcers, telangiectasias, Raynaud's phenomenon are also common.
- CREST syndrome (Calcinosis cutis, Raynaud's phenomenon, esophageal dysmotility, sclerodactyly, telangiectasia) is a clinical limited variant of systemic sclerosis.
- Visceral involvement usually leads to organ failure.

Diagnosis
- The clinical diagnosis should be confirmed by laboratory tests.
- Skin biopsy and histopathological examination.
- Immunological examinations.

Differential Diagnosis
- Epidermolysis bullosa
- Cicatricial pemphigoid
- Lipoid proteinosis

Treatment
- Good oral hygiene
- Systemic treatment includes:
  - Antimalarials
  - Corticosteroids
  - Immunosuppressive drugs
  - D-penicillamine
  - Colchicine
  - Others

Mixed Connective Tissue Disease

Definition
- Mixed connective tissue disease is a multisystemic disorder characterized by the simultaneous clinical manifestations mainly seen in lupus erythematosus, systemic sclerosis and polymyositis.

Etiology
- The cause and pathogenesis remain obscure.

Gingival Involvement
- Rare
- Non-dental plaque-induced gingival lesions

Other Involvement
- Oral mucosa, rare
- Skin
- Skeletal muscles, lymph nodes and several other organs

Main Clinical Features
- The gingival lesions may appear as localized or diffuse erythema and/or non-specific irregular erosions identical to those seen in lupus erythematosus patients (Fig. 295). Periodontal destruction is impossible.
- The oral mucosal lesions are characterized by a non-specific erythema, oedema, erosions and xerostomia.
- The skin and other manifestations are those seen in systemic lupus erythematosus, systemic scleroderma and polymyositis.

Diagnosis
- The clinical diagnosis should always be confirmed by laboratory tests.
- High titres of anti-ribonucleoprotein antibodies (anti-RNP) is the most diagnostic laboratory finding.
- Other serological tests specific for the group of diseases included.

Differential Diagnosis
- Lupus erythematosus
- Systemic scleroderma

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 295

Mixed connective tissue disease. Irregular erosions on the gingiva

- Polymyositis
- Other connective tissue diseases

Treatment

- Good oral hygiene
- Topical corticosteroids
- Systemic treatment includes:
  - Corticosteroids
  - Immunosuppressive drugs
  - Antimalarials

Graft-Versus-Host Disease

Definition

- Graft-versus-host disease (GVHD) is a multisystemic phenomenon due to immune reaction between the transplanted T lymphocytes and the patient's normal tissues.

Etiology

- The disease occurs in recipients of allogeneic bone marrow transplants usually to treat life-threatening diseases of the blood or bone marrow.

Classification

- Two forms of the disorder are recognized:
  - Acute, that is typically observed within 100 days of transplantation
  - Chronic, that develops later than 100 days after transplantation

Gingival Involvement

- Rare
- Non-dental plaque-induced gingival lesions

Other Involvement

- Oral mucosa (40%–80%)
- Skin, common
- Gastrointestinal tract, liver, lungs, eyes

Main Clinical Features

- The gingival lesions are not specific and are characterized by diffuse or localized erythema usually associated with a fine, reticular network of white striae and/or erosions similar to those seen in lichen planus (Fig. 296). The condition occasionally mimics desquamative gingivitis.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 296

Graft-versus-host disease. Diffuse erythema on the gingiva mimic desquamative gingivitis

FIGURE 297

Graft-versus-host disease. Lichenoid lesions on the buccal mucosa

For Personal Use Only
Library of School of Dentistry, TUMS

- The oral mucosal lesions appear as diffuse erythema, lichenoid pattern, and painful ulcerations (Fig. 297). Atrophy of the oral mucosa, a burning sensation and xerostomia are common. The oral mucosal and gingival lesions are more common in the chronic form of the disorder.
- The skin lesions range from mild rash with hyperpigmentation to lesions that may resemble to systemic sclerosis and lichen planus.
- Other signs and symptoms are nausea, vomiting, abdominal pain, diarrhea, liver dysfunction.

Diagnosis

- The diagnosis is usually based on the clinical criteria and less on laboratory tests.
- Biopsy and histopathological examination (oral mucosa and the minor salivary glands, skin).
- Increased salivary sodium concentration.

Differential Diagnosis

- Lichen planus
- Lupus erythematosus

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

- Systemic sclerosis
- Sjögren's syndrome
- Drug reactions
- Leukaemia

Treatment

- Good dental plaque control
- Topical corticosteroids
- Systemic corticosteroids, immunosuppressive drugs, thalidomide

Behçet's Disease

Definition

- Behçet's disease is a chronic multisystemic disorder that affects more commonly certain ethnic groups (Japanese, Turkish, Greek and other Mediterranean origins).

Etiology

- The exact etiology remains obscure. However, genetic, immunological factors, and viral infection may be involved in the pathogenesis of the disease.

Gingival Involvement

- Very rare
- Non-dental plaque-induced gingival lesions

Other Involvement

- Oral mucosa, very common
- Other mucosae (eyes, genitalia, intestinal)
- Skin
- Joints, cardiovascular systems, lungs, central nervous system

Main Clinical Features

- The main oral manifestations of Behçet's disease are recurrent aphthous ulcerations (minor, major, herpetiform, or atypical). Painful (1–2 in number) aphthous ulcers, usually minor or atypical, may rarely develop, usually on the attached gingiva (Figs. 298, 299).
- Conjunctivitis, iritis, iridocyclitis with hypopyon, uveitis are the most frequent eye manifestations.
- The skin lesions present as genital round ulcerations on the scrotum or on the penis or vulva (Fig. 300). Folliculitis, papules, pustules, nodular erythema and less often necrotic lesions are common cutaneous lesions.

FIGURE 298

Behçet's disease.
Aphthous ulcers on the upper gingiva

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 299

Behçet's disease.
Aphthous ulcers on the upper gingiva

FIGURE 300

Behçet's disease.
Typical round ulcers on the scrotum.

- Vascular, intestinal, joint and neurological involvement may occur.
- Pathergy test is positive in about 60 %–70 % of cases.

Diagnosis

- The diagnosis is made on clinical criteria.
- Biopsy and histopathological examinations may be supportive.

Differential Diagnosis

- Aphthous ulcers
- Pemphigus
- Pemphigoid
- Erythema multiforme
- Necrotizing ulcerative gingivitis
- Neutropenia
- Leukaemia
- Langerhans cell histiocytosis

Treatment

- Corticosteroids topically
- Systemic corticosteroids

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

- Immunosuppressive drugs
- Thalidomide
- Colchicine

Reiter's Disease

Definition

- Reiter's disease is an uncommon multisystemic disorder that may be triggered by an infectious agent in a genetically susceptible individual.

Etiology

- The exact etiology remains unknown, although the pathogenesis is mediated by an immunological mechanism.

Gingival Involvement

- Rare
- Non-dental plaque-induced gingival lesions

Other Involvement

- Oral mucosa (20%–40%)
- Genital, eyes, skin, joints, skeletal, cardiovascular system

Main Clinical Features

- The gingival lesions appear as localized or generalized non-specific erythema, usually on the attached gingiva, occasionally associated with superficial, slightly sensitive erosions (Figs. 301, 302).
- The oral mucosal lesions are characterized by diffuse erythematous areas intermixed with thin whitish dots or lines, and superficial erosions. Localized loss of the tongue filiform papillae in the form of geographic tongue may occur (Fig. 303)
- The skin manifestations appear as macular, vesicular or pustular, psoriasiform lesions and keratoderma.
- Other manifestations include conjunctivitis, cyclic balanitis, nongonococcal urethritis, prostatitis, cervicitis and nail disturbances.
- The most characteristic feature of the disease is the symmetrical arthritis of six to seven joints.

Diagnosis

- The clinical diagnosis should be confirmed by laboratory tests.
- Skin biopsy and histopathological examination.
- Blood and serological tests, HLA antigens, synovial fluid analysis.

FIGURE 301

Reiter's disease. Diffuse deep erythema and erosions on the upper gingiva

For Personal Use Only
Library of School of Dentistry, TUMS

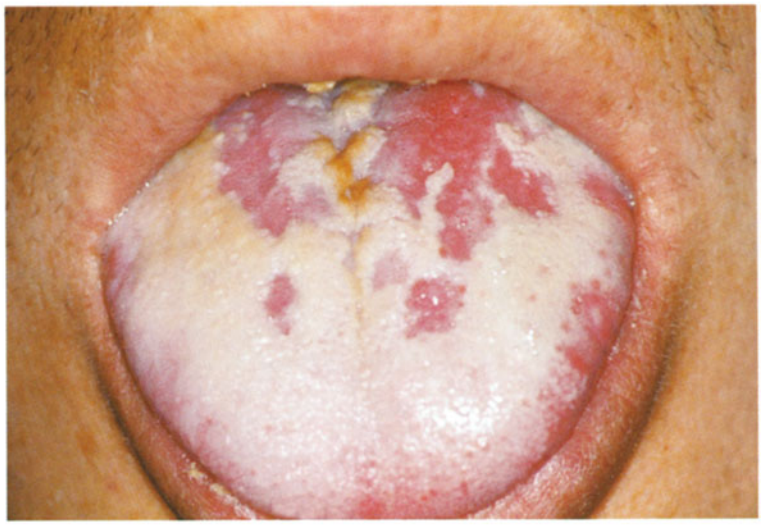
WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 302

Reiter's disease.
Slight erythema and oedema on the upper gingiva

FIGURE 303

Reiter's disease.
Localized loss of the filiform papillae of the dorsum of the tongue



Differential Diagnosis

- Behçet's disease
- Erythema multiforme
- Geographic stomatitis
- Drug eruption

Treatment

- Non-steroidal anti-inflammatory drugs, salicylates
- Corticosteroids

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Reading List

Atkinson K (1990) Chronic graft-versus-host disease. Bone Marrow Transpl 5:69–82

Cervera R, Khamashta MA, Font JH et al (1993) Systemic lupus erythematosus: clinical, immunologic patterns of disease in a cohort of 1,000 patients. Medicine 72:113–124

Eversole LR, Jacobsen PL, Stone CE (1984) Oral and gingival changes in systemic sclerosis (scleroderma). J Periodontol 5:175–178

Gonzales TS, Coleman GC (1999) Periodontal manifestations of collagen vascular disorders. Periodontology 2000 21:94–105

Hiroki A, Nakamura S, Shinohara M, Oka M (1994) Significance of oral examination in chronic graft-versus-host disease. J Oral Pathol Med 23:209–215

International Study Group for Behçet's Disease (1990) Criteria for diagnosis of Behçet's disease. Lancet 335:1078–1080

Laskaris G (2000) Oral diseases in children and adolescence. Georg Thieme Verlag, Stuttgart

Mutlu S, Porter SR, Richards A et al (1991) Periodontal disease in Sjögren's syndrome. Clin Exp Rheumatol 9:317

Nagy G, Kovacs J, Zeher M, Czirjak L (1994) Analysis of the oral manifestations of systemic sclerosis. Oral Surg Oral Med Oral Pathol 77:141–146

Najera MP, Al-Hashimi I, Plemons JM et al (1997) Prevalence of periodontal disease in patients with Sjögren's syndrome. Oral Surg Oral Med Oral Pathol 83:453–457

Oetgen WJ, Boice JA, Lawless OJ (1981) Mixed connective tissue disease in children and adolescents. Pediatrics 67:333–337

Pedersen AM, Reibel J, Nordgarden H et al (1999) Primary Sjögren's syndrome: salivary gland function and clinical oral findings. Oral Diseases 5:128–138

Rhodus NL, Johnson DK (1990) The prevalence of oral manifestations of systemic lupus erythematosus. Quintessence Int 21:461–465

Rothe MJ, Kerdel FA (1991) Reiter syndrome. Int J Dermatol 30:173–180

Schiodt M (1984) Oral manifestations of lupus erythematosus. Int J Oral Surg 13:101–147

Scully C, Porter S (1987) The periodontium and systemic disease. Dental Update 14:247–254

Stratigos A, Laskaris G, Stratigos J (1992) Behçet's disease. Semin Neurol 12:346–357

Vitali C, Bombarderi M, Moutsopoulos HM et al (1997) The European classification criteria for Sjögren's syndrome: proposal for a modification of the rules for classification suggested by the analysis of the receiver operating characteristic (ROC) curve of the criteria performance. J Rheumatol 24 [Suppl 50]:38

Wood RE, Lee P (1988) Analysis of the oral manifestations of systemic sclerosis (scleroderma). Oral Surg Oral Med Oral Pathol 65:172–178

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

CHAPTER 24

Orofacial Granulomatosis

Melkersson-Rosenthal Syndrome

Definition
- Melkersson-Rosenthal syndrome is a rare disorder. It consists of the triad of recurrent facial swelling, facial paralysis and fissured tongue.

Etiology
- Unknown

Gingival Involvement
- Rare
- Non-dental plaque-induced gingival lesions

Other Involvement
- Oral mucosa
- Facial

Main Clinical Features
- Gingival lesions present as irregular, oedematous swelling, slightly erythematous, affecting mainly the anterior interdental papillae and free and attached gingiva (Figs. 304–306).
- Granulomatous cheilitis presenting as diffuse recurrent swelling of the lips is the cardinal feature of the syndrome. Swelling of the lips is usually the first sign. After a long period of attacks, the swelling persists and may become permanent.
- Buccal, palatal and lingual swelling may also occur (Fig. 307).
- Facial nerve palsy, similar to Bell's palsy is present in 20 %–30 % of the cases and is frequently associated with facial oedema. Facial nerve palsy may be the first sign and is usually unilateral.
- Fissured tongue is present in about half of the patients.
- The disease affects equally both sexes and usually young individuals.

Diagnosis
- The diagnosis is based on clinical and laboratory criteria.
- Allergy tests.
- Biopsy and histopathological examination of oral lesions.

Differential Diagnosis
- Angioedema
- Cheilitis glandularis
- Crohn's disease
- Sarcoidosis
- Amyloidosis

Treatment
- Dental plaque control and surgical excision of the gingival lesions.
- Corticosteroid, topical or systemic.
- If facial nerve palsy is present, neurological consultation should be undertaken.

For Personal Use Only
Library of School of Dentistry, TUMS

G. Laskaris et al., *Periodontal Manifestations of Local and Systemic Diseases*
© Springer-Verlag Berlin Heidelberg 2003

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 304

Melkersson-Rosenthal syndrome. Early gingival lesions mimic chronic gingivitis

FIGURE 305

Melkersson-Rosenthal syndrome. Localized gingival swelling

FIGURE 306

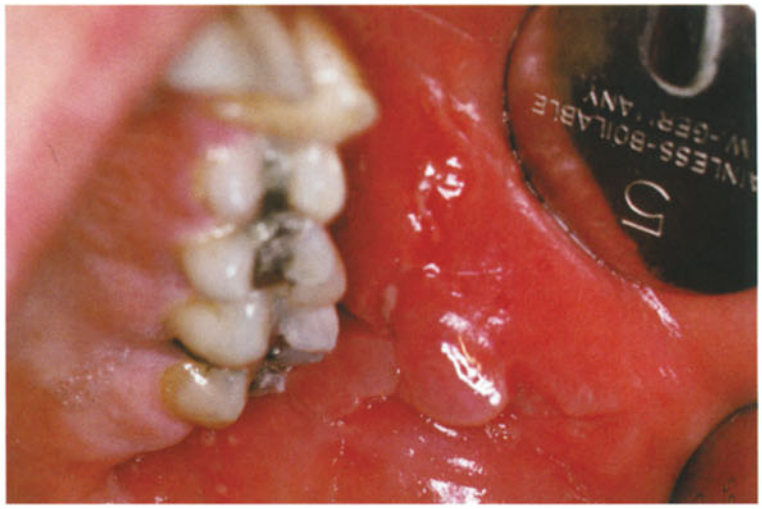
Melkersson-Rosenthal syndrome. Diffuse gingival and labial swelling

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 307

Melkersson-Rosenthal syndrome. Multiple oedematous folds on the buccal mucosa



Crohn's Disease

Definition

- Crohn's disease is a chronic granulomatous inflammatory disease that may affect any part of the gastrointestinal tract from the oral cavity to the anus.

Etiology

- Unknown. A hereditary susceptibility is possible and an immunologically mediated mechanism may be involved in the pathogenesis.

Gingival Involvement

- Relative common
- Non-dental plaque-induced gingival lesions

Other Involvements

- Oral mucosa, common
- Gastrointestinal tract, particularly the terminal ilium, always
- Skin, eyes, joints (rare)

Main Clinical Features

- The gingival lesions present as erythematous and oedematous gingival enlargement, which may be localized or generalized, usually affecting the anterior gingiva (Figs. 308–310). Rarely gingival involvement may be the presenting feature.
- The oral mucosal lesions may manifest as diffuse granulomatous lip swelling, nodular or diffuse swelling of the tongue, soft palate and buccal mucosa, resulting in a cobblestone pattern. Mucosal folds and tags, lip fissures, aphthous or aphthous-like ulcerations, soft tissue granulomas, metallic dysgeusia, and persistent lymph node enlargement may occur.
- The gastrointestinal signs and symptoms include abdominal discomfort, diarrhoea, vomiting, anorexia, weight loss, low-grade fever and rectal bleeding. These signs and symptoms may persist for many years with periods of exacerbation and remission.
- Extra-abdominal manifestations include arthritis, spondylitis and uveitis.

Diagnosis

- The clinical diagnosis should be confirmed by laboratory tests.
- Biopsy and histopathological examination of oral and gut lesions.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 308

Crohn's disease. Localized gingival swelling

FIGURE 309

Crohn's disease. Generalized swelling on the upper gingiva

FIGURE 310

Crohn's disease. Oedematous swelling of the lower gingiva and lips

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Differential Diagnosis
- Melkersson-Rosenthal syndrome
- Sarcoidosis
- Ulcerative colitis
- Pyostomatitis vegetans
- Contact hypersensitivity reactions

Treatment
- Good oral hygiene
- Topical corticosteroid for oral lesions
- Systemic corticosteroids, sulphasalazine, azathioprine, ciclosporin, metronidazole for gut lesions

Sarcoidosis

Definition
- Sarcoidosis is a chronic multisystemic granulomatous disease affecting one or multiple organs or tissues.

Etiology
- Unknown. However, the disease is usually associated with depression of cell-mediated immunity and an overactivity of B cells.

Gingival Involvement
- Rare
- Non-dental plaque-induced gingival lesions

Other Involvement
- Oral mucosa, rare
- Lymph nodes, salivary glands, skin, eyes, bone, nerves
- Lungs, liver, spleen

Main Clinical Features
- The gingival lesions usually present as diffuse erythema extending to alveolar mucosa (Fig. 311). The affected gingiva is slightly oedematous and sensitive, and occasionally may be the initial sign of sarcoidosis (Fig. 312).
- The oral lesions present as painless, solitary or multiple, red nodules that may rarely ulcerate. Lip and less often buccal and tongue swelling may occur. Salivary gland swelling (minor and major), temporomandibular dysfunction, facial nerve palsy may also occur.
- The skin lesions include painless, purple-brown macules, papules or nodules that may be scattered or confluent, lupus pernio, erythema nodosum, scars and plaques. The skin manifestations appear in 25%–30% of cases and may remain the only lesions for long time.
- Lymphadenopathy, liver and spleen enlargement, lung disturbances are common.

Diagnosis
- The clinical diagnosis should be confirmed by laboratory tests.
- Biopsy and histopathological examination.
- Serum levels of angiotensin-converting enzyme (SACE).
- Chest radiography.
- Gallium scan.

Differential Diagnosis
- Crohn's disease
- Tuberculosis
- Amyloidosis
- Melkersson-Rosenthal syndrome

Treatment
- Good oral hygiene and topical corticosteroid for gingival lesions
- Systemic corticosteroids, allopurinol for systemic manifestations

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 311

Sarcoidosis. Diffuse erythema and slight oedema on the gingiva

FIGURE 312

Sarcoidosis. Severe oedema and swelling of the upper gingiva

For Personal Use Only
Library of School of Dentistry, TUMS

Pyostomatitis Vegetans

Definition
- Pyostomatitis vegetans is a relatively rare, pustular disorder of the mouth associated with inflammatory bowel diseases, particularly ulcerative colitis and Crohn's disease.

Etiology
- Unknown

Gingival Involvement
- Common (labial and buccal gingiva)
- Non-dental plaque-induced gingival lesions

Other Involvement
- Oral mucosa (buccal, labial, mucolabial and mucobuccal folds, soft palate, ventral tongue)
- Skin (pyodermatitis vegetans is the equivalent of oral lesions)

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 313

Pyostomatitis vegetans. Early lesions on the gingiva presenting with erythema and oedema

FIGURE 314

Pyostomatitis vegetans. Erythema and multiple pustular microabscesses on the upper gingiva

For Personal Use Only
Library of School of Dentistry, TUMS

Main Clinical Features

- The gingival lesions present as multiple, painless, yellow-white, vegetative mucosal folds that consist of pustular microabscesses that finally may be ulcerated (Figs. 313, 314).
- Similar lesions may be present in other mucosal sites.
- Rarely similar lesions may be present on the skin.

Diagnosis

- The clinical diagnosis should be confirmed by laboratory tests.
- Biopsy and histopathological examination.
- Direct immunofluorescence to rule out chronic bullous oral diseases.

Differential Diagnosis

- Pemphigus vegetans
- Dermatitis herpetiformis
- Wegener's granulomatosis

Treatment

- Systemic corticosteroids, sulphasalazine.
- Spontaneous remission of oral lesions may occur when bowel lesions subside.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Reading List

Chan SWY, Scully C, Prime SS, Eveson J (1991) Pyostomatitis vegetans: oral manifestation of ulcerative colitis. Oral Surg Oral Med Oral Pathol 72:689–692

Gargiulo AV, Ladone JA, Toto PD, Logiudice J (1989) Crohn's disease: early detection by gingival biopsy. Periodont Case Rep 11:20–22

Flemming TF, Shanahan F, Miyasaki KT (1991) Prevalence and severity of periodontal disease in patients with inflammatory bowel disease. J Clin Periodontol 18:690–697

Hayter JP, Robertson JM (1988) Sarcoidosis presenting as gingivitis. Br Med J 296:1504

Hildebrand J, Plezia RA, Rao SB (1990) Sarcoidosis: report of two cases with oral involvement. Oral Surg Oral Med Oral Pathol 69:217–222

Lygidakis C, Tsakanikas C, Ilias A, Vassilopoulos D (1979) Melkersson-Rosenthal syndrome in four generations. Clin Genet 15:189–192

Meisel-Stosiek M, Hornstein OP, Stosiek N (1990) Family study on Melkersson-Rosenthal syndrome. Some hereditary aspects of the disease and review of the literature. Acta Derm Venereol (Stockh) 70:221–226

Misra S, Ament ME. Orofacial lesions in Crohn's disease. Am J Gastroenterol 1996; 91:1651–1653

Plouth M, Jenss H, Meyle J (1991) Oral manifestations of Crohn's disease: an analysis of 79 cases. J Clin Gastroenterol 13:29–37

Rees TD (1999) Orofacial granulomatosis and related conditions. Periodontology 2000 21:145–157

Sloan PJM, O'Neil TCA, Smith CJ, Holdsworth CD (1983) Multisystem sarcoid presenting with gingival hyperplasia. Br J Oral Surg 21:31–35

Storwick GS, Prihoda MB, Fulton RJ, Wood WS (1994) Pyodermatitis-pyostomatitis vegetans: a specific marker for inflammatory bowel disease. J Am Acad Dermatol 31:336–341

Thornhill MH, Zakrzewska JM, Gilkes JJH (1992) Pyostomatitis vegetans: report of three cases and review of the literature. J Oral Pathol Med 21:128–133

Worsaae N, Pindborg JJ (1980) Granulomatous gingival manifestations of Melkersson-Rosenthal syndrome. Oral Surg Oral Med Oral Pathol 49:131–138

Wray D (1984) Pyostomatitis vegetans. Br Dent J 157:316–318

Wray D (1998) Gastrointestinal and granulomatous diseases. Periodontology 2000 18:95–101

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

CHAPTER 25

Metabolic Disorders

Porphyria

Definition

- Porphyria comprises a group of disorders resulting from abnormal porphyrin metabolism.

Etiology

- Inborn enzymatic defects in the halm biosynthetic pathway. It is inherited as an autosomal dominant and recessive trait.

Classification

- Three major groups, each with several types are recognized:
  - Erythropoietic porphyria.
  - Hepatic porphyria .
  - Erythropoietic-hepatic porphyria.
- Gingival lesions occurs only in erythropoietic protoporphyria and porphyria cutanea tarda.

Gingival Involvement

- Uncommon
- Non-dental plaque-induced gingival lesions

Other Involvement

- Oral mucosa, rare
- Skin, common
- Internal organ

Main Clinical Features

- The gingival lesions present as diffuse erythema of free and attached vestibular gingiva, occasionally mimicking desquamative gingivitis (Fig. 315).

FIGURE 315

Porphyria cutanea tarda. Diffuse erythema on the lower gingiva mimics desquamative gingivitis

For Personal Use Only
Library of School of Dentistry, TUMS

G. Laskaris et al., *Periodontal Manifestations of Local and Systemic Diseases*
© Springer-Verlag Berlin Heidelberg 2003

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

- The oral mucosal lesions present as diffuse erythema, atrophy, vesicles and ulcerations, especially on the lips.
- The skin lesions appear mainly on the light-exposed areas and are characterized by photosensitivity, erythema, fragility, vesicles, ulcerations and scar formation, hypertrichosis.

Diagnosis

- The clinical diagnosis should be confirmed by laboratory tests.
- Biopsy and histopathological examination of oral and skin lesions.
- Biochemical tests (erythrocyte, urine, faecal).
- Photobiological tests and direct immunofluorescence.

Differential Diagnosis

- Desquamative gingivitis due to epidermolysis bullosa and cicatricial pemphigoid
- Pellagra

Treatment

- Dental plaque control.
- Systemic therapy must be left to the specialist.

Amyloidosis

Definition

- Amyloidosis is a relatively rare metabolic disorder characterized by the extracellular deposition of an amorphous proteinaceous substance known as amyloid.

Etiology

- Unknown

Classification

- Four major forms are recognized:
  - Primary.
  - Secondary.
  - Familial.
  - Senile.
- Oral lesions present mainly in primary systemic form.

Gingival Involvement

- Relatively rare
- Non-dental plaque-induced gingival lesions

Other Involvement

- Oral mucosa (tongue, lips, buccal), common, particularly in primary form
- Skin
- Gastrointestinal tract, joints, skeletal muscles
- Heart, kidneys and rarely other organs

Main Clinical Features

- The gingival lesions usually present as localized painless nodules or diffuse swelling with characteristic reddish colour (Figs. 316, 317).
- The most common oral mucosal manifestations are petechiae, ecchymoses, nodules, haemorrhagic bullae, ulcers, macroglossia, minor and major salivary gland enlargements, xerostomia, and regional lymph node swelling (Fig. 318). The oral lesions may be the early presenting signs of the disease.
- The most common skin lesions are purpura, petechiae, papules, nodules, bullae, ulcers, and alopecia.
- The most common presenting symptoms are weakness, fatigue, weight loss, oedema, dyspnea, hoarseness, bleeding, pain, carpal tunnel syndrome.
- About 20%–30% of the cases of primary amyloidosis are associated with multiple myeloma. The prognosis is unfavourable.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 316

Amyloidosis.
Localized red swelling on the gingiva

FIGURE 317

Amyloidosis.
Gingival swelling on the upper gingiva

FIGURE 318

Amyloidosis.
Macroglossia in a patient with primary systemic amyloidosis

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Diagnosis
- The clinical diagnosis should be confirmed by laboratory tests.
- Biopsy and histopathological examination with special stains, e. g. Congo red, Sirius red, methyl violet, thioflavine-T.
- Serum and urine electrophoresis.

Differential Diagnosis
- Soft tissue, gingival plasmacytoma
- Sarcoidosis and Crohn's disease
- Benign and malignant neoplasms of the gingiva, e. g. Kaposi's sarcoma, peripheral giant cell granuloma

Treatment
- Symptomatic treatment of gingival and oral lesions.
- Systemic treatment must be left to the specialist.

Langerhans Cell Histiocytosis

Definition
- Langerhans cell histiocytosis is a clinicopathological heterogeneous disorder characterized by proliferation of Langerhans cells.

Etiology
- The cause remains obscure.

Classification
- Three major forms are recognized:
  - Eosinophilic granuloma, that is a localized benign form of the disorder, usually characterized by solitary or multiple bone lesions and soft tissue lesions as well,.
  - Hand-Schüller-Christian disease, that is a chronic disseminated form involving primarily bone, soft tissue and viscera.
  - Lettere-Siwe disease, that is an acute disseminated form involving skin, viscera, bone marrow.

Periodontal Involvement
- Relatively common, particularly in eosinophilic granuloma and Hand-Schüller-Christian disease
- Periodontitis associated with systemic diseases

Other Involvement
- Oral mucosa, jaws
- Bones, bone marrow, skin, visceral

Main Clinical Features
- The gingival lesions present as localized or multiple atypical ulcerations, usually associated with bone destruction and teeth loosening (Figs. 319–322). The gingival lesions may be the only manifestation for a long time, particularly in eosinophilic granuloma.
- The oral mucosa lesions are less common and are characterized by atypical deep ulcerations, ecchymoses, oedema, halitosis, taste disturbances and are more common in Hand-Schüller-Christian and Letterer-Siwe forms.
- Jaw bone destruction, either solitary or multiple, is common, in all three forms of the disorder.
- Other bone involvement, exophthalmos, diabetes insipidus, skin rash, otitis media, hepatomegaly, splenomegaly, lymphadenopathy are common in the two severe forms of the disorder.
- More than 50 % of all cases of the Langerhans cell histiocytosis are seen in patients under 10 years of age.

Diagnosis
- The clinical diagnosis should be confirmed by laboratory tests.
- Biopsy and histopathological examination.
- Immunohistochemical examinations.
- Radiographic examination.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 319

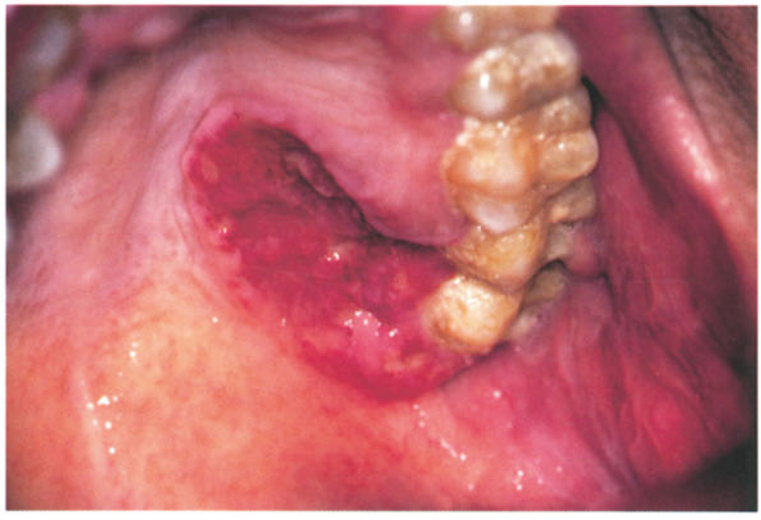
Langerhans cell histiocytosis. Localized swelling on the upper gingiva in a patient with eosinophilic granuloma

FIGURE 320

Langerhans cell histiocytosis. Localized ulceration on the lower gingiva in a patient with eosinophilic granuloma

FIGURE 321

Langerhans cell histiocytosis. Localized gingival ulceration and bone destruction in a patient with eosinophilic granuloma

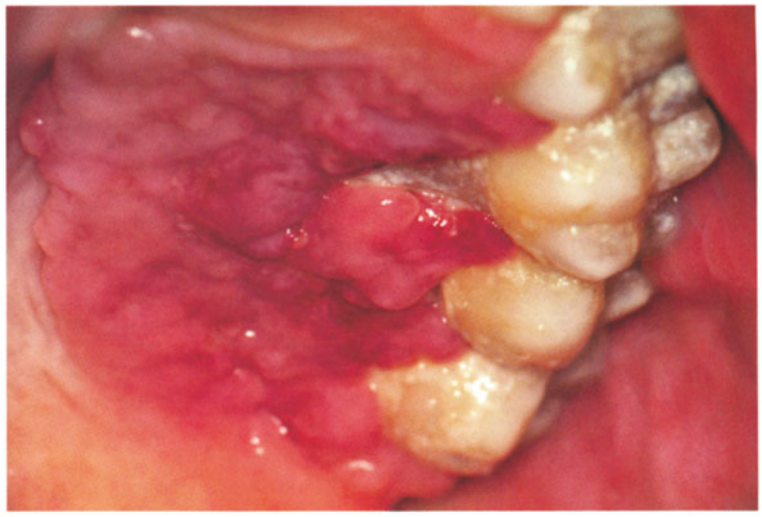


For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 322

Langerhans cell histiocytosis. Extensive gingival andpalatal ulcerations and bone destruction in a patient with Hand-Schüller-Christian disease



Differential Diagnosis

- Necrotizing ulcerative gingivitis and periodontitis
- Aggressive periodontitis
- Traumatic lesions on the gingiva
- Hypophosphatasia, acatalasia
- Haematological disorders
- Eosinophilic ulcer

Treatment

- Dental plaque control and periodontal curettage
- Radiation of local bone lesions
- Corticosteroid and chemotherapy for the systemic manifestations, which should be left to the specialists

Reading List

Al-Hasshimi I, Drinnan AJ, Uthman AA et al (1987) Oral amyloidosis: two unusual case presentations. Oral Surg Oral Med Oral Pathol 63:586–591

Bottomley WK, Gabriel SA, Corio RL et al (1987) Histiocytosis X: report of an oral tissue lesion without bony involvement. Oral Surg Oral Med Oral Pathol 63:228–231

Brun A, Sandberg S (1991) Mechanisms of photosensitivity in porphyric patients with special emphasis on erythropoietic protoporphyria. J Photochem Photobiol 810:285–302

Flick WG, Lawrence FR (1980) Oral amyloidosis as initial symptom of multiple myeloma: a case report. Oral Surg Oral Med Oral Pathol 49:18–20

Gilhuus-Moe O, Koppang HS (1972) Oral manifestations of porphyria. Oral Surg Oral Med Oral Pathol 33:926–933

Hartman KS (1980) Histiocytosis X: a review of 114 cases with oral involvement. Oral Surg Oral Med Oral Pathol 49:38–54

Husby G (1992) Nomenclature and classification of amyloid and amyloidoses. J Int Med 232:511–512

Osband ME, Pochedly C (1987) Histiocytosis X: an overview. Hematol Oncol Clin North Am 1:1–7

Reinish EI, Ravin M, Srolovitz H, Gornisky M (1994) Tongue, primary amyloidosis and multiple myeloma. Oral Surg Oral Med Oral Pathol 77:121–125

Sigala JL, Silverman S Jr, Brody HA, Kuslmer JH (1972) Dental involvement in histiocytosis. Oral Surg Oral Med Oral Pathol 33:42–48

Wray D (1998) Gastrointestinal and granulomatous diseases. Periodontol 2000 18:95–101

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

CHAPTER 26

Endocrine Disorders

Diabetes Mellitus

Definition

- Diabetes mellitus is a chronic, systemic, heterogeneous group of disorders of the metabolism of carbohydrate, protein and fat, resulting in microvascular and macrovascular complications.

Etiology

- It is due either to decreased production of insulin, tissue resistance to the effect of insulin, or a combination of both.

Classification

- Two main forms of diabetes mellitus are recognized:
  - Type 1, insulin dependent (children onset).
  - Type 2, non-insulin dependent (adult onset).

Gingival Involvement

- Common, particularly in type 1
- Dental plaque-induced gingivitis and periodontitis, modified by diabetes mellitus

Other Involvement

- Oral mucosa, relatively rare
- Many organs and systems

Main Clinical Features

- The clinical features of gingivitis associated with diabetes mellitus are characterized by the presence of dental plaque at gingival margin, erythema, oedema, bleeding upon probing, increased gingival exudate and the absence of attachment and alveolar bone loss (Figs. 323, 324). The gingival inflammation is usually reversible after the control of diabetic state. In type 2 diabetes mellitus patients, periodontitis with attachment loss and alveolar bone loss may develop.
- The oral mucosa manifestations are not specific and include xerostomia, superficial erosions, retardation of wound healing, burning mouth, taste disturbances, candidiasis.
- Salivary gland swelling (sialosis).

Diagnosis

- The clinical diagnosis should be confirmed by laboratory tests.
- Blood examination for sugar.

Differential Diagnosis

- Dental plaque-induced gingivitis
- Sex hormone-associated gingivitis

Treatment

- Dental plaque control
- Diabetic control from the specialist

For Personal Use Only
Library of School of Dentistry, TUMS

G. Laskaris et al., *Periodontal Manifestations of Local and Systemic Diseases*
© Springer-Verlag Berlin Heidelberg 2003

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 323

Diabetes mellitus.
Early periodontitis

FIGURE 324

Diabetes mellitus.
Severe periodontitis

For Personal Use Only
Library of School of Dentistry, TUMS

Sex Steroid Hormones

Definition

- Sex steroid hormones (androgens, estrogens and progestins) may influence the periodontal tissue, and particularly the gingiva, to increase the inflammation response present.

Etiology

- Dental plaque gingivitis modified by sex steroid hormones

Classification

- Four forms of gingival disorders may be developed:
  - Puberty-associated gingivitis.
  - Menstrual cycle-associated gingivitis.
  - Pregnancy-associated gingivitis.
  - Oral contraceptive-associated gingivitis.

Gingival Involvement

- Common
- Dental plaque-induced modified by sex steroid hormones

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 325

Early pregnancy-associated gingivitis

FIGURE 326

Severe pregnancy-associated gingivitis and granulomas

FIGURE 327

Severe pregnancy-associated gingivitis and granulomas

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Main Clinical Features
- All four forms of gingivitis display a similar clinical pattern.
- The main clinical features are dental plaque present at gingival margin, redness and oedema of the gingiva (Figs. 325–327), bleeding upon probing, loss of attachment and alveolar bone loss. The lesions can resolve following puberty, ovulation, at parturition, and discontinuation of oral contraceptives.

Diagnosis
- The diagnosis is based on the clinical criteria and laboratory tests.
- Sex hormone measurement.

Differential Diagnosis
- Dental plaque-induced gingivitis
- Diabetes mellitus-associated gingivitis

Treatment
- Dental plaque control
- Balance of sex steroid hormones by the specialist

Addison's Diseases

Definition
- Addison's disease is a rare acquired deficiency in adrenal gland function characterized by insufficient secretion of glucocorticoids and mineralocorticoids.

Etiology
- Destruction of the adrenal cortex due to infection, autoimmunity, infiltration by a tumour, haemorrhage, or infarction. The levels of ACTH and melanocyte-stimulating hormone (MSH) are elevated, causing increased hyperpigmentation of the skin and oral mucosa.

Gingival Involvement
- Relatively common
- Non-dental plaque-induced gingival lesions

Other Involvement
- Oral mucosa (buccal, palate, lips), common
- Skin, common

Main Clinical Features
- The gingival lesions present as multiple melanotic macules or irregular plaques (Figs. 328, 329).
- Similar lesions appear on the other areas of the oral mucosa. The oral manifestations are usually early and valuable diagnostic sign.
- The skin lesions present as diffuse bronze to grey hyperpigmentation, particularly on sun-exposed skin.
- Malaise, weakness, nausea, vomiting and weight loss are common.

Diagnosis
- The clinical diagnosis should be confirmed by laboratory tests.
- ACTH measurement, ACTH stimulation, and assay of plasma cortisol.

Differential Diagnosis
- Normal gingival melanosis
- Smoking melanosis
- Pigmentation due to drugs
- Amalgam tattoo
- Melanotic naevi
- Malignant melanoma

Treatment
- No treatment is needed for gingival and oral lesions.
- Systemic treatment must be left to the specialist.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 328

Addison's disease. Early melanotic macule on the interdental papilla

FIGURE 329

Addison's disease. Generalized pigmentation on the gingiva and the alveolar mucosa

For Personal Use Only
Library of School of Dentistry, TUMS

Reading List

American Academy of Periodontology (2000) Diabetes and periodontal diseases. J Periodontol 71:664–678
Ashri N, Gazi M (1990) More unusual pigmentations of the gingiva. Oral Surg Oral Med Oral Pathol 70:445–449
Löe H (1965) Periodontal changes in pregnancy. J Periodontol 36:209–216
Mariotti A (1994) Sex steroid hormones and cell dynamics in the periodontium. Crit Rev Oral Biol Med 5:27–53
Mariotti A (1999) Dental plaque-induced gingival diseases. Am Periodontol 4:7–17
Miyazaki H, Yamashita, Shirahama R et al (1991) Periodontal condition of pregnant women assessed by CPITN. J Clin Periodontol 18:751–754
Norderyd OM, Machtei EE, Grossi SG et al (1993) Periodontal status of women taking postmenopausal estrogen supplementation. J Periodontol 64:957–962
Novaes AB, Pereira ALA, Moraes ND (1991) Manifestations of insulin-dependent diabetes mellitus in the periodontium of young Brazilian patients. J Periodontol 62:116–122
Sperber GH (1969) Oral contraceptive hypertrophic gingivitis. J Dent Assoc Africa 24:37–40
Stamm JW (1986) Epidemiology of gingivitis. J Clin Periodontol 13:360–366

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

CHAPTER 27

Paraneoplastic Disorders

Malignant Acanthosis Nigricans

Definition
- Malignant acanthosis nigricans is a form of acanthosis nigricans that develops in association with an internal malignancy, usually adenocarcinoma of the stomach, and affects exclusively adults.

Etiology
- It is obscure, although a cytokine-like peptide produced by the malignant tumour may affect the epidermal cells.

Gingival Involvement
- Relatively rare
- Non-dental plaque-induced gingival lesions

Other Involvement
- Oral mucosa, relatively common (about 30%–40%)
- Other mucosae (conjunctiva, anus, vagina, pharynx, oesophagus), rare
- Skin, always

Main Clinical Features
- The gingival lesions present as a diffuse papillomatous overgrowth, of normal colour, that may cover the crowns of the teeth (Fig. 330).
- The oral mucosal lesions also present as diffuse velvety papillary lesions, of normal colour, particularly on the lips, commissures, tongue, and palate.
- Similar lesions may develop in other mucosae.
- The skin lesions appear as brownish, asymptomatic, papillary and rough growth, usually on the axillae, the genitofemoral region and the neck (Fig. 331).
- The mucocutaneous lesions are benign, but are important as they represent a marker for internal malignancy.

Diagnosis
- The diagnosis is usually based on clinical criteria.
- Biopsy and histopathological examination.

Differential Diagnosis
- Familial acanthosis nigricans
- Darier's disease
- Cowden's syndrome
- Multiple papillomas
- Lipoid proteinosis

Treatment
- The mucocutaneous lesions may resolve when the malignancy is treated.
- Dental plaque control and occasionally gingivectomy for the gingival lesions.

For Personal Use Only
Library of School of Dentistry, TUMS

G. Laskaris et al., *Periodontal Manifestations of Local and Systemic Diseases*
© Springer-Verlag Berlin Heidelberg 2003

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 330

Malignant acanthosis nigricans. Generalized papillomatous overgrowths of the gingiva

FIGURE 331

Malignant acanthosis nigricans. Brownish papillary overgrowths on the skin

Paraneoplastic Pemphigus

Definition
- Paraneoplastic pemphigus is a unique, rare variant in the pemphigus group usually associated with lymphoreticular malignancy.

Etiology
- Autoimmune. Autoantibodies again desmoplakins I and II are a specific marker for paraneoplastic pemphigus.

Gingival Involvement
- Relatively common
- Non-dental plaque-induced gingival lesions

Other Involvement
- Oral mucosa, always
- Other mucosae (conjunctiva, pharynx, oesophagus, penis, vagina), relatively common
- Skin, always

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 332

Paraneoplastic pemphigus. Erythema and erosions on the gingiva in a patient with pemphigus associated with chronic lymphocytic leukaemia

Main Clinical Features

- The gingival lesions present as painful, generalized and persistent erosions identical to those seen in pemphigus vulgaris (Fig. 332).
- The oral lesions present as multiple, severe and treatment-resistant erosions on the lips, buccal mucosa, tongue, palate, usually extending to the oropharynx and oesophagus, causing severe sore throat. Nikolsky's sign is positive.
- Persistent bilateral conjunctival erosions and oedema are common. In some cases, ocular lesions result in shrinkage and scarring.
- The skin lesions are polymorphous and include bullae, erythematous and/or verrucous papules or plaques mimicking the "target-like" lesions seen in erythema multiforme.

Diagnosis

- The clinical diagnosis should be confirmed by laboratory tests.
- Biopsy and histopathological examination.
- Direct immunofluorescence.
- Indirect immunofluorescence.
- Immunoprecipitation study.

Differential Diagnosis

- Pemphigus vulgaris
- Bullous pemphigoid
- Cicatricial pemphigoid
- Linear IgA disease
- Erythema multiforme
- Stevens-Johnson syndrome

Treatment

- Systemic corticosteroid alone or in combination with immunosuppressive drugs.
- Treatment of the underlying malignancy is necessary.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Reading List

Anhalt GJ, Kim SC, Stanley JR et al (1990) Paraneopastic pemphigus: an autoimmune mucocutaneous disease associated with neoplasia. N Engl J Med 323:1729–1735

Brown J, Winkelmann RK (1968) Acanthosis nigricans: a study of 90 cases. Medicine 47:33–39

Curth HO (1968) The necessity of distinguishing four types of acanthosis nigricans. Proc XIII Intern Congress of Dermatology I:577

Helm TN, Camisa C, Valenguela R, Allen CM (1993) Paraneoplastic pemphigus: a distinct autoimmune vesiculobullous disorder associated with neoplasia. Oral Med Oral Surg Oral Pathol 75:209–213

Laskaris G, Papavasiliou S, Bovopoulou O, Nicolis G (1980) Association of oral pemphigus with chronic lymphocytic leukemia. Oral Surg Oral Med Oral Pathol 50:244–249

Mostofi RS, Hayden NP, Soltani K (1983) Oral malignant acanthosis nigricans. Oral Surg Oral Med Oral Pathol 56:372–374

Schwartz RA (1994) Acanthosis nigricans. J Am Acad Dermatol 31:1–19

Sklavounou A, Laskaris G (1998) Paraneoplastic pemphigus: a review. Oral Oncol 35:437–440

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

CHAPTER 28

Vitamin Deficiency

Scurvy

Definition

- Scurvy is a rare vitamin C deficiency that is usually limited to individuals (infants and elderly edentulous) whose diets are poor in fresh fruits and vegetables.

Etiology

- Vitamin C (ascorbic acid) deficiency leading to impairment of peptidyl hydroxylation of procollagen and a reduction in collagen formation and secretion by connective tissue. This deficiency leads to capillary fragility that correlates the haemorrhagic features and poor healing of wounds.

Gingival Involvement

- Common. Dental plaque-induced gingival lesions modified by vitamin C deficiency.

Other Involvement

- Oral mucosa
- Skin, hair, nails
- Muscles, joints

Main Clinical Features

- The gingival lesions, which present early, are common and cause generalized swelling and redness of the interdental and marginal gingiva (Fig. 333). Spontaneous gingival haemorrhage and ulcers are also common. Periodontal bone loss and tooth mobility may also occur.
- Oral mucosal petechiae, haemorrhages, ecchymoses and delayed wound healing are commonly seen, as well as enamel hypoplasia of developing teeth.
- Haemorrhage, ecchymoses of the skin, nail beds, muscles and joints are common.
- Severe to moderate anaemia is common. Melena, haematuria and orbital haemorrhages may be found.
- Retrobulbar, subarachnoid, and intracerebral haemorrhages may also occur if treatment is delayed.

Diagnosis

- The diagnosis is mainly based on clinical criteria.
- Platelet and plasma ascorbic acid levels are reduced.

Differential Diagnosis

- Necrotizing ulcerative gingivitis
- Herpetic gingivostomatitis
- Agranulocytosis
- Leukaemia
- Thrombocytopenic purpura

Treatment

- Ascorbic acid therapy
- Plaque control

For Personal Use Only
Library of School of Dentistry, TUMS

G. Laskaris et al., *Periodontal Manifestations of Local and Systemic Diseases*
© Springer-Verlag Berlin Heidelberg 2003

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 333

Scurvy.
Generalized gingival swelling associated with haemorrhages

Reading List

Charbeneau TP, Hurt WC (1983) Gingival findings in spontaneous scurvy: a case report. J Periontol 54:694–697

Oeffinger KC (1993) Scurvy: more than historical relevance. Am Fam Physician 48:609–613

Woolfe SN, Hume WR, Kenney EB (1980) Ascorbic acid and periodontal disease: a review of the literature. J West Soc Periodontol 28:44–56

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

PART IV

Cysts, Tumours, Potentially Malignant, Neoplasms and Fibro-Osseous Lesions

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

CHAPTER 29

Cysts

Periapical Cyst

Definition
- A cyst arising from cell rests of Malassez as a consequence of inflammatory changes

Etiology
- Cell rests proliferate in response to bacterial infection of a necrotic pulp.

Gingival Involvement
- Common

Main Clinical Features
- Most are small, asymptomatic and present as a periapical radiolucency.
- Larger cysts may present clinically with:
  - Swelling (Fig. 334).
  - Pain if infected.
  - Non-vital associated tooth.
  - Rarely, pathological fracture.

Diagnosis
- Diagnosis is from history and clinical features of a non-vital tooth, plus imaging.

Differential Diagnosis
- Other odontogenic cysts

Treatment
- Enucleation plus tooth extraction or endodontics. If the cyst remains after extraction, it is termed a residual cyst.
  - Residual cysts, where one of the inflammatory cysts (usually a radicular cyst) remains after removal of the non-vital tooth from which it arose (Fig. 335)

For Personal Use Only
Library of School of Dentistry, TUMS

G. Laskaris et al., *Periodontal Manifestations of Local and Systemic Diseases*
© Springer-Verlag Berlin Heidelberg 2003

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 334

Odontogenic cyst. Radicular cyst

FIGURE 335

Odontogenic cyst. Residual cyst

For Personal Use Only
Library of School of Dentistry, TUMS

Eruption Cyst

Definition ▶ A cyst arising from the follicle of an erupting tooth

Etiology ▶ Eruption cysts are considered as minor soft tissue forms of follicular cysts. They often burst spontaneously before eruption of the associated tooth and only rarely require excision if impeding normal eruption.

Gingival Involvement ▶ Uncommon

Other Sites of Involvement ▶ None

Main Clinical Features ▶ A bluish fluctuant swelling over an erupting tooth (Figs. 336, 337)

Diagnosis ▶ Diagnosis is from history and clinical features, supplemented by:
- Radiography.
- Others.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 336

Odontogenic cyst. Eruption cyst

FIGURE 337

Odontogenic cyst. Eruption cyst

For Personal Use Only
Library of School of Dentistry, TUMS

Differential Diagnosis ▶ Other odontogenic cysts

Treatment ▶ Usually none

Gingival Cyst of Newborn

Definition ▶ Cyst on the alveolus of neonates

Etiology ▶ Probably developmental, arising from remnants of the dental lamina

Gingival Involvement ▶ Common

Other Sites of Involvement ▶ None

Main Clinical Features ▶ These can be small, multiple, white nodules on the alveolar ridge of newborns and young infants (Fig. 338).

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 338

Odontogenic cyst.
Gingival cyst of newborn

Diagnosis ▶ The diagnosis of childhood gingival cysts is usually based upon clinical features alone.

Differential Diagnosis ▶ Other odontogenic cysts

Treatment ▶ Gingival cysts in children do not require therapy.

Gingival Cyst of Adult

Definition ▶ Cyst on gingiva of adults

Etiology ▶ Gingival cysts of adults arise from epithelial rests in the gingivae.

Gingival Involvement ▶ Common

Other Sites of Involvement ▶ None

Main Clinical Features ▶ Gingival cysts in adulthood usually arise in middle to late life as painless swellings of the interdental or attached gingivae, often in the anterior mandible (Figs. 339, 340).

Diagnosis ▶ Diagnosis is from history and clinical features, supplemented by:
- Histopathological examination.

▶ Radiographic assessment is essential in adults to exclude any lesion likely to be causing bony damage, or any intraosseous pathology spreading to the gingivae.

Differential Diagnosis ▶ Other odontogenic cysts

Treatment ▶ Gingival cysts in adults should be removed to confirm diagnosis. There are no long-term complications.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 339

Odontogenic cyst.
Gingival cyst of adults

FIGURE 340

Odontogenic cyst.
Gingival cyst of adults

For Personal Use Only
Library of School of Dentistry, TUMS

Palatine Papilla Cyst

Definition ▶ A cyst in the palatine papilla

Etiology ▶ These cysts are derived from the vestigial oro-nasal ducts.

Gingival Involvement ▶ Rare

Other Sites of Involvement ▶ None

Main Clinical Features ▶ They may occur either within the nasopalatine canal or in the soft tissues of the palate at the opening of the canal, grow slowly, and may discharge into the mouth giving a salty taste (Fig. 341).

Diagnosis ▶ Diagnosis is from history and clinical features, supplemented by:

- Radiography. This cyst is often discovered during routine radiographic examination. The defect appears on radiographs as a round

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 341

Odontogenic cyst.
Cyst of palatine papilla

or teardrop-shaped, well circumscribed radiolucency. Due to its location, it can easily be misdiagnosed as a lesion of endodontic origin. Radiological examination shows a well-defined rounded ovoid or occasionally heart-shaped defect in the anterior maxilla. Nasopalatine cysts must be distinguished from a normal large anterior palatine fossa, which may be up to 7 mm in diameter, and from radicular cysts associated with the maxillary incisors.

- Histopathology may help.

Differential Diagnosis

- Other odontogenic cysts
- Endodontic lesions

Treatment

- These cysts should be enucleated and seldom recur.

Dentigerous Cyst

Definition

- An odontogenic cyst (follicular cyst) that surrounds the crown of an unerupted tooth

Etiology

- Follicular cysts arise by separation of the dental follicle from around the anatomical crown of an unerupted tooth. Fluid accumulates between the crown and reduced enamel epithelium of an impacted tooth from degeneration of the stellate reticulum, or an accumulation of fluid between the layers of the reduced enamel epithelium.

Gingival Involvement

- Rare

Other Sites of Involvement

- Any tooth-bearing location in the jaws

Main Clinical Features

- They are most common in association with mandibular third molars and maxillary canines.
- Most are small, asymptomatic and found as a radiolucency surrounding the crown of an impacted tooth.
- These cysts may grow to a large size and displace the tooth with which they are associated. Larger cysts may present clinically with swelling

For Personal Use Only
Library of School of Dentistry, TUMS

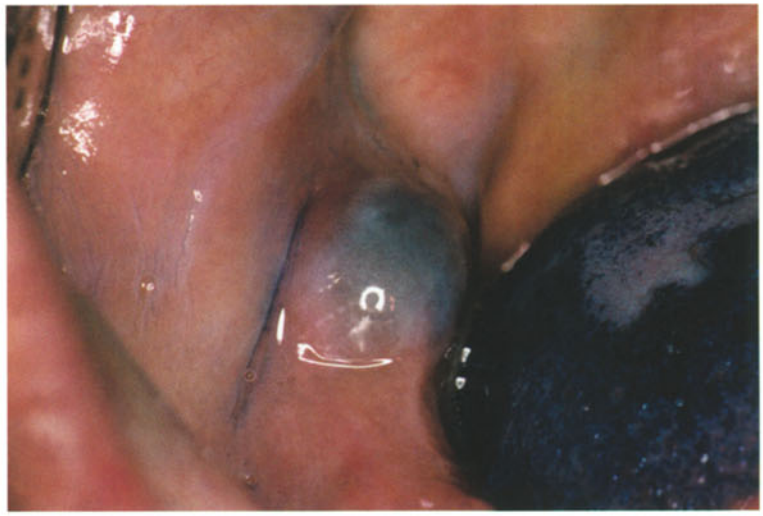
WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 342

Odontogenic cyst. Dentigerous cyst

FIGURE 343

Odontogenic cyst. Dentigerous cyst



For Personal Use Only
Library of School of Dentistry, TUMS

(Figs. 342, 343), pain if infected, resorption of adjacent teeth or tooth missing from arch.

- Rarely, there is a pathological fracture.

Diagnosis

- Diagnosis is from history and clinical features, supplemented by:
  - Radiography and histopathology; the lining of follicular cysts typically consists of flattened stratified epithelium.
- Radiographic assessment; when the follicular space exceeds 5 mm from the crown, it is likely that a cyst is present. However, odontogenic keratocysts and ameloblastomas may occasionally mimic the radiological appearance of follicular cysts.
- Aspiration may assist the diagnosis since the demonstration of keratin squames in a cyst aspirate is virtually diagnostic of a keratocyst. A protein level of less than 4.0 g/100 ml suggests a keratocyst, whereas a value over 5 g/100 ml suggests a radicular or follicular cyst, or rarely a cystic ameloblastoma.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Differential Diagnosis
- Other odontogenic cysts

Treatment
- Enucleation or marsupialization

Keratocyst

Definition
- These cysts (primordial cyst) have generally been thought to arise from remnants of the dental lamina (cell rests of Serres) or its derivatives, but there is some suggestion that they may also arise from basal cells of the oral mucosa.

Etiology
- Idiopathic. Some cases are genetic and seen in Gorlin's syndrome.

Gingival Involvement
- Rare

Other Sites of Involvement
- None

Main Clinical Features
- Many are seen in the mandible, especially posteriorly, and present as a uni- or multi-locular radiolucent swelling with a well-demarcated margin.
- They involve the mandibular angle and may expand to occupy the major part of the mandibular ramus.
- They are painless, unless they become secondarily infected.
- Grow to a large size before giving rise to symptoms (Fig. 344).
- They may resorb the cortical plates and expand into the soft tissues, may envelop unerupted teeth, giving a dentigerous appearance, or may displace teeth.

Diagnosis
- Diagnosis is from history and clinical features.
- Histopathology. The cyst lining usually has a characteristic appearance of a regular keratinized stratified squamous epithelium, commonly five to eight cells thick. Desquamated keratin is often present within the cyst lumen and the fibrous wall is usually thin.
- Biochemistry. Aspiration may assist the diagnosis since the demonstration of keratin squames in a cyst aspirate is virtually diagnostic of a keratocyst. A protein level of less than 4.0 g/100 ml suggests a keratocyst, whereas a value over 5 g/100 ml suggests a radicular or follicular cyst, or rarely a cystic ameloblastoma.

Differential Diagnosis
- Other odontogenic cysts
- Nasopalatine cyst
- Nasolabial cyst
- Solitary bone cyst
- Aneurysmal bone cyst
- Stafne bone cavity

Treatment
- Small unilocular lesions should be thoroughly enucleated.
- When the cysts are large or multiloculated, excision with a margin of surrounding bone is more appropriate.
- Keratocysts tend to recur following removal.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 344

Odontogenic cyst. Keratocyst

Reading List

Bell RC, Chauvin PJ, Tyler MT (1997) Gingival cyst of the adult: a review and a report of eight cases. J Can Dent Assoc. 63:533–535

Binnie WH. Periodontal cysts and epulides. Periodontol 2000. 1999;21:16–32

Blanas N, Freund B, Schwartz M, Furst IM (2000) Systematic review of the treatment and prognosis of the odontogenic keratocyst. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 90:553–558

Ezsias A (2001) Longitudinal in vivo observations on odontogenic keratocyst over a period of 4 years. Int J Oral Maxillofac Surg 30:80–82

Kerezoudis NP, Donta-Bakoyianni C, Siskos G (2000) The lateral periodontal cyst: aetiology, clinical significance and diagnosis. Endod Dent Traumatol 16:144–150

Lombardi T, Kuffer R, Di Felice R, Samson J (1999) Epithelial odontogenic ghost cell tumour of the mandibular gingiva. Oral Oncol 35:439–442

Myoung H, Hong SP, Hong SD, Lee JI, Lim CY, Choung PH, Lee JH, Choi JY, Seo BM, Kim MJ (2001) Odontogenic keratocyst: review of 256 cases for recurrence and clinicopathologic parameters. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 91:328–333

Orsini G, Favia G, Piattelli A (2000) Peripheral clear cell calcifying epithelial odontogenic tumor. Report of a case. J Periodontol 71:1177–1180.

Shear M (1994) Developmental odontogenic cysts. An update. J Oral Pathol Med 23:1–11

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

CHAPTER 30

Tumour-Like Lesions

The term "epulis" is applied to any lump arising from the gingiva.

Epulides are localized gingival swellings and rarely true neoplasms, metastatic carcinomas or other tumours.

Fibrous Epulis and Denture-Induced Granuloma (Epulis Fissuratum)

Definition
- The fibrous epulis resembles a fibroepithelial polyp, but also usually has an inflammatory component.

Etiology
- Probably chronic irritation

Gingival Involvement
- Fibrous epulides are common.
- Epulis fissuratum may be induced by trauma from a denture flange.

Other Sites of Involvement
- None

Main Clinical Features
- The variable inflammatory changes account for the different clinical presentations from red, shiny, and soft lumps to those which are pale, stippled and firm. Commonly, epulides are swellings which are typically firm, round, painless, pedunculated pale swellings (Figs. 345–349) less than 2 cm in diameter.
- Arise from an anterior interdental papilla the marginal or papillary gingiva and sometimes adjacent to sites of irritation (e.g. a carious cavity).

Diagnosis
- The diagnosis is clinical, but most epulides should be removed and examined histologically.

Differential Diagnosis
- Abscesses
- Exostoses
- Hyperplastic gingivitis
- Fibrous epulis
- Cysts
- Giant cell tumour
- Pyogenic granuloma
- Neoplasms

Treatment
- Fibrous epulides should be removed down to the periosteum, which should be curetted thoroughly.

For Personal Use Only
Library of School of Dentistry, TUMS

G. Laskaris et al., *Periodontal Manifestations of Local and Systemic Diseases*
© Springer-Verlag Berlin Heidelberg 2003

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 345

Gingival epulis fissuratum

FIGURE 346

Gingival epulis fissuratum

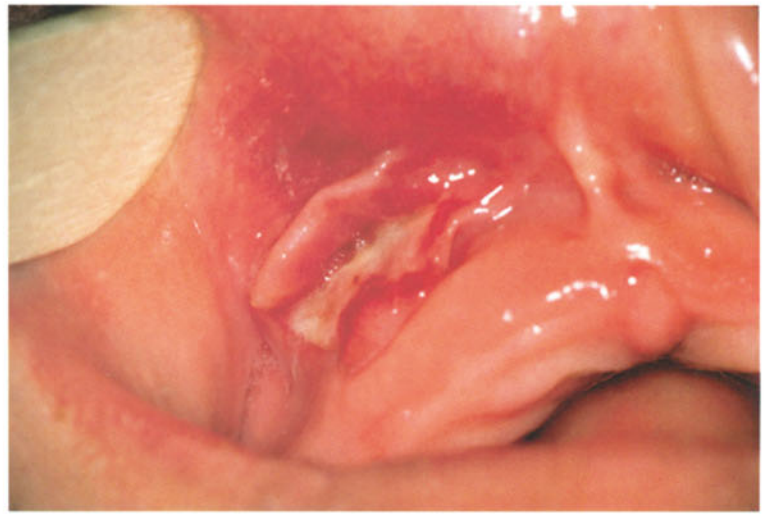


FIGURE 347

Gingival fibrous lump

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 348

Gingival fibrous lump

FIGURE 349

Gingival fibrous lump

For Personal Use Only
Library of School of Dentistry, TUMS

Pyogenic Granuloma

Definition ▶ Pyogenic granuloma is a soft proliferative lesion that readily bleeds.

Etiology ▶ Possibly a reactive vascular lesion

Gingival Involvement ▶ Uncommon except as pregnancy epulides (lesions themselves not histologically distinguishable)

Other Sites of Involvement ▶ The tongue, or rarely the lip

Main Clinical Features ▶ Typically it is a mass that is seen on the gingival margin (Figs. 350–352) and is small (< 3 cm), red, painless, bleeds easily, ulcerates and grows rapidly.

Diagnosis ▶ Diagnosis is from history and clinical features supplemented by histopathology.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 350

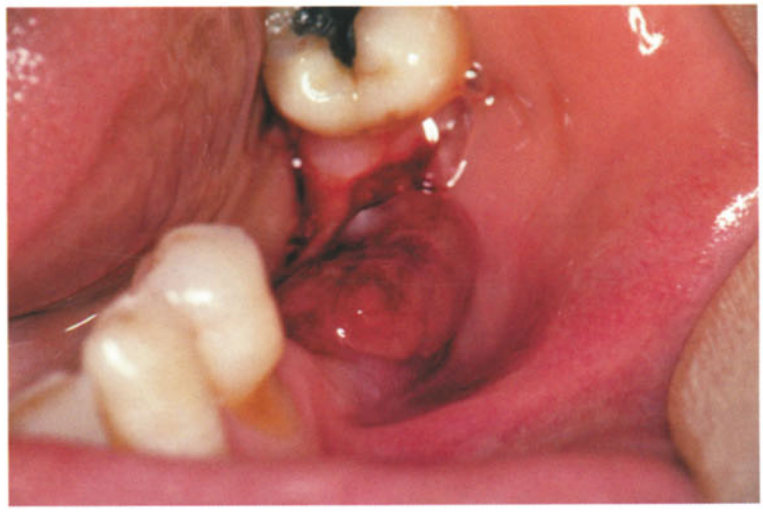
Gingival pyogenic granuloma

FIGURE 351

Gingival pyogenic granuloma

FIGURE 352

Gingival post-extraction granuloma



For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Differential Diagnosis
- Abscesses
- Exostoses
- Hyperplastic gingivitis
- Fibrous epulis
- Cysts
- Giant cell tumour
- Pyogenic granuloma
- Neoplasms
- Kaposi's sarcoma
- Angiomatous proliferations
- Chancre

Treatment
- Surgical excision

Giant Cell Granuloma (Giant Cell Epulis, Peripheral Giant Cell Granuloma)

Definition
- This is a non-neoplastic swelling of proliferating fibroblasts in a highly vascular stroma containing many multinucleate giant cells.

Etiology
- May result from proliferation of giant cells persisting after resorption of deciduous teeth. The lesion probably arises because chronic irritation triggers a reactionary hyperplasia of mucoperiosteum and excessive production of granulation tissue.

Gingival Involvement
- Common

Clinical Features
- Classically, the most notable features (Figs. 353–355) are that it:
  - Has a deep red colour, although older lesions tend to be paler.
  - Arises interdentally.
  - Is seen adjacent to permanent teeth which have had predecessors, so is seen anterior to the permanent molars.
  - Is a benign lesion.

Diagnosis
- Diagnosis is from history and clinical features, supplemented by:
  - Histology, which shows many multinucleated cells distributed widely throughout the lesion or gathered into clumps.
- Imaging.
- Giant cell granulomas unless they are extraosseous, may also be a feature of hyperparathyroidism, and thus levels of plasma calcium, phosphate, and alkaline phosphatase should be assayed.

Differential Diagnosis
- Abscesses
- Exostoses
- Hyperplastic gingivitis
- Fibrous epulis
- Cysts
- Giant cell tumour
- Pyogenic granuloma
- Neoplasms

Treatment
- Treatment depends on cause. If hyperparathyroidism, that condition must be treated. Treatment otherwise is surgical excision.
- Histopathologically, remove local irritants (calculus). The lesion may recur.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 353

Gingival peripheral giant cell granuloma

FIGURE 354

Gingival peripheral giant cell granuloma

FIGURE 355

Gingival peripheral giant cell granuloma

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Central Giant Cell Lesion

Definition
- This is an intra-bony destructive giant cell lesion.

Etiology
- The true nature of these lesions is unknown but, as they are invariably destructive, the term "reparative" giant cell granuloma would appear to be inappropriate.

Clinical Features
- Bony swelling
  - Mostly in the mandible
  - Typically anteriorly
  - Only in the tooth-bearing regions of the jaws
  - Sometimes displaces or resorbs tooth roots
  - Occasionally erodes through the cortical bone where it presents as a domed, purplish submucosal swelling
  - May be symptomless or simulate a malignant neoplasm clinically and imaging

Diagnosis
- Diagnosis is from history and clinical features, supplemented by:
  - Imaging: an ill-defined area of radiolucency.
  - Histology: multinucleated giant cells irregularly distributed in a cellular stroma of plump, spindle-shaped cells, which is often highly vascular.

Differential Diagnosis
- Abscesses
- Exostoses
- Hyperplastic gingivitis
- Fibrous epulis
- Cysts
- Giant cell tumour
- Pyogenic granuloma
- Neoplasms

Treatment
- Curettage
- Central giant cell granulomas may recur but virtually never metastasize.

Haemangioma

Definition
- Numerous thin-walled vessels separated by sparse bands of collagenous tissue.

Etiology
- Haemangioma is a lesion of blood vessels generally regarded as a developmental lesion (hamartoma) and usually present from birth.

Gingival Involvement
- Rare

Other Sites of Involvement
- Haemangiomas are:
  - Usually isolated.
  - Occasionally a component of Sturge-Weber, Klippel-Trenaunay-Weber, Maffucci, Blue Rubber-Bleb naevus or other syndromes.

Main Clinical Features
- A haemangioma usually:
  - Is deep-red (even blue-purple) in colour (Fig. 356).
  - Blanches on pressure.
  - Is fluctuant to palpation.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 356

Gingival haemangioma

- It may have a lobulated or raised surface. Occasionally haemangiomas develop phlebolithiasis.

Diagnosis

- Diagnosis is from history and clinical features.
- Aspiration may aid diagnosis.
- Imaging is useful to show osseous involvement.
- Arteriography may be indicated.

Differential Diagnosis

- Lymphangioma

Treatment

- Haemangiomas are at risk from trauma and prone to excessive bleeding if damaged (e.g. during tooth extraction).
- Haemangiomas are left alone unless causing symptoms, when they are best treated with cryosurgery, or by laser, injection of sclerosant, ligation or embolization of feeding vessels.

Lymphangioma

Definition

- Lymphangioma is of similar structure to a haemangioma, but contains lymph rather than blood.

Etiology

- Congenital

Gingival Involvement

- Rare

Other Sites of Involvement

- Lymphangiomas usually:
  - Are solitary
  - Affect the tongue
  - And are sometimes associated with cystic hygroma

Main Clinical Features

- It often has a "frogspawn" appearance (Figs. 357, 358).

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 357

Gingival lymphangioma

FIGURE 358

Gingival lymphangioma

Diagnosis

- Diagnosis is from history and clinical features.
- Aspiration may aid diagnosis.
- Imaging is useful to show osseous involvement.
- Lymphangiography may be indicated.

Differential Diagnosis

- Haemangioma

Treatment

- Small lymphangiomas need no treatment but cryotherapy can be useful. Larger lesions may require excision.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Reading List

Blum MR, Williams B, Speight PM (1994) An unusual vascular hamartoma on the gingiva of an 11-year-old boy. Int J Paediatr Dent 4:41–45

Dahlkemper P, Wolcott JF, Pringle GA, Hicks ML (2000) Periapical central giant cell granuloma: a potential endodontic misdiagnosis. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 90:739–745

Flaitz CM (2000) Peripheral giant cell granuloma: a potentially aggressive lesion in children. Pediatr Dent 22:232–233

Kaneko R, Tohnai I, Ueda M, Negoro M, Yoshida J, Yamada Y (2001) Curative treatment of central hemangioma in the mandible by direct puncture and embolisation with n-butyl-cyanoacrylate (NBCA). Oral Oncol 37:605–608

Lawoyin JO, Arotiba JT, Dosumu OO (1997) Oral pyogenic granuloma: a review of 38 cases from Ibadan, Nigeria. Br J Oral Maxillofac Surg 35:185–189

Maaita JK (2000) Oral tumors in children: a review. J Clin Pediatr Dent 24:133–135

Munoz M, Monje F, Alonso del Hoyo JR, Martin-Granizo R (1998) Oral angiosarcoma misdiagnosed as a pyogenic granuloma. J Oral Maxillofac Surg 56:488–491

Ong MA, Chai WL, Ngeow WC (1998) Recurrent gigantic pyogenic granuloma disturbing speech and mastication: a case report and literature review. Ann Acad Med Singapore 27:258–261

Orsini G, Fioroni M, Rubini C, Piattelli A (2001). Epithelioid hemangioendothelioma of the oral cavity: report of case. J Oral Maxillofac Surg 59:334–337

Silva-Sousa YT, Coelho CM, Brentegani LG, Vieira ML, de Oliveira ML (2000) Clinical and histological evaluation of granuloma gravidarum: case report. Braz Dent J. 11:135–139

Silverstein LH, Burton CH Jr, Singh BB (1995) Oral pyogenic granuloma in pregnancy. Int J Gynaecol Obstet 49:331–332

Willies-Jacobo LJ, Isaacs H Jr, Stein MT (2000) Pyogenic granuloma presenting as a congenital epulis. Arch Pediatr Adolesc Med 154:603–605

Zain RB, Khoo SP, Yeo JF (1995) Oral pyogenic granuloma (excluding pregnancy tumour) – a clinical analysis of 304 cases. Singapore Dent J 20:8–10

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

CHAPTER 31

Potentially Malignant

Erythroplasia

Definition
- Erythroplasia (erythroplakia) is an isolated red lesion.

Etiology
- Unclear

Gingival Involvement
- Rare

Other Sites of Involvement
- Any oral site, but erythroplasia usually involves the floor of the mouth, the ventrum of the tongue, or the soft palate.

Main Clinical Features
- Erythroplasia is a red velvety lesion level with, or depressed below, the surrounding mucosa (Figs. 359, 360).
- Diagnosis is from history and clinical features, supplemented by:
  - Histopathology; virtually all the lesions show dysplasia, carcinoma in situ, or invasive carcinoma.

Differential Diagnosis
- Other causes of red lesions, especially inflammation and candidiasis

Treatment
- Surgical excision

FIGURE 359

Gingival erythroplakia

For Personal Use Only
Library of School of Dentistry, TUMS

G. Laskaris et al., *Periodontal Manifestations of Local and Systemic Diseases*
© Springer-Verlag Berlin Heidelberg 2003

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 360

Gingival erythroplakia

Keratoses/Leukoplakia

Definition

- Leukoplakia is the term used for hyperkeratotic white mucosal lesions of unknown cause. Without specific histopathological connotations.

Etiology

- Leukoplakia and other keratoses include the following:
  - Idiopathic: most cases.
  - Friction.
  - Tobacco: pipe smoking can cause "nicotinic stomatitis" of palate especially. Smokeless tobacco or betel-chewing can cause keratosis, usually of buccal sulcus.
- Microorganisms.
  - *Candida albicans*. Candidal leukoplakias frequently appear speckled, often affect commissures and have fairly high premalignant potential.
  - Syphilis. Syphilitic leukoplakia especially affects dorsum of tongue and has high premalignant potential.

Gingival Involvement

- Common in frictional keratosis

Other Sites of Involvement

- Any oral site

Main Clinical Features

- Most keratoses (Figs. 361–365) are:
  - Benign.
  - Seen in the buccal mucosa.
  - Smooth plaques (homogeneous leukoplakias).
- Some are:
  - Warty (verrucous leukoplakia) (Fig. 364).
  - Mixed white and red lesions (speckled leukoplakias) (Fig. 365).
- Overall, 1%–3% are premalignant. Premalignant potential is higher in verrucous leukoplakias and is highest in speckled leukoplakias. Some studies show malignant transformation in over 20% of specked leukoplakias and 30% of verrucous leukoplakias over 10 years. Thus keratoses in particular sites or of particular appearance tend to have higher premalignant potential, but epithelial dysplasia, microscopically, is a more reliable guide as may be DNA ploidy.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 361

Gingival leukoplakia. Early homogeneous

FIGURE 362

Gingival leukoplakia. Homogeneous

FIGURE 363

Gingival leukoplakia. Homogeneous

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 364

Gingival leukoplakia. Verrucous

FIGURE 365

Gingival leukoplakia. Speckled

- Frictional keratosis is usually seen at sites of trauma from teeth, also along buccal occlusal line and occasionally on the gingiva beside an outstanding tooth, or on an edentulous ridge. It is homogeneous, benign and resolves when irritation is removed.
- Smoker's keratosis (stomatitis nicotina) is usually caused by pipe or heavy cigarette smoking. The palate, particularly the soft palate, is affected. Red orifices of swollen minor salivary glands of palate within widespread white lesion give striking appearance of red spots on white background. This lesion is benign in itself, but carcinoma may develop nearby.
- Other tobacco-related habits. Tobacco-chewing, snuff dipping or chewing of betel quids or ghutka may lead to keratoses that can be premalignant. Snuff dipping is associated predominantly with verrucous keratoses, which can progress to verrucous carcinoma. Tobacco-related keratoses typically resolve on stopping the habit.
- Candidal leukoplakia is seen particularly in smokers and is especially likely to form speckled leukoplakias at commissures. It may be dysplas-

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

tic and have higher premalignant potential than some other keratoses. Candidal leukoplakias may respond to antifungals and stopping smoking.

- Syphilitic leukoplakia is rare, but seen especially on the dorsum of tongue, and is a feature of tertiary syphilis. Premalignant potential is high.
- Proliferative verrucous leukoplakia (PVL) is a unique type of clinical oral leukoplakia, which appears related to papillomavirus and behaves in a far more aggressive fashion than other forms of leukoplakia. Its aggressiveness relates not only to a high recurrence rate, but more to a very high level of keratosis and relentless progression from a localized simple keratosis to extensive oral disease and squamous carcinomas of verrucous or conventional squamous cell type.

Diagnosis

- Diagnosis is from history and clinical features, supplemented by:
  - Biopsy for possible dysplasia or early malignant change.

Differential Diagnosis

- Materia alba
- Burns
- Carcinoma
- Candidiasis (thrush: candidal leukoplakia)
- Epstein-Barr virus (hairy leukoplakia)
- Syphilis (syphilitic mucous patches and keratosis)
- Lichen planus
- Lupus erythematosus

Treatment

- Treat predisposing factors.
- Surgically remove discrete lesions.
- Consider use of retinoids.

Reading List

Alexander RE, Wright JM, Thiebaud S (2001) Evaluating, documenting and following up oral pathological conditions. A suggested protocol. J Am Dent Assoc 132:329–335

Axell T, Holmstrup P, Kramer IRH, Pindborg JJ, Shear M (1984) International seminar on oral leukoplakia and associated lesions related to tobacco habits. Comm Dent Oral Epidemiol 12:145–154

Barrellier P, Babin E, Louis MY et al (1993) The use of toluidine blue in the diagnosis of neoplastic lesions of the oral cavity. Fr J Rev Stomatol Chir Maxillo-Faciale 944:51–54

Batsakis JG, Suarez P, el-Naggar AK (1999) Proliferative verrucous leukoplakia and its related lesions. Oral Oncol 35:354–359

Chapman M (1988) Tobacco-induced changes: palatal gingiva. Gen Dent 36:54–60

Craig RM Jr, Vickers VA, Correll RW (1989) Erythroplastic lesion on the mandibular marginal gingiva. J Am Dent Assoc 119:543–544

Dufresne RG, Curlin MU (1997) Actinic cheilitis; a treatment review. Dermatol Surg 23:15–21

Epstein JB, Scully C, Spinelli J (1992) Toluidine blue and Lugol's iodine application in the assessment of oral malignant disease and lesions at risk of malignancy. J Oral Pathol Med 21:160–163

Eversole LR, Eversole GM, Kopcik J (2000) Sanguinaria-associated oral leukoplakia: comparison with other benign and dysplastic leukoplakic lesions. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 89:455–464

Gooris PJ, Roodenburg JL, Vermey A, Nauta JM (1999) Carbon dioxide laser evaporation of leukoplakia of the lower lip: a retrospective evaluation. Oral Oncol 35:490–495

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Grady D, Greene J, Daniels TE et al (1990) Oral mucosal lesions found in smokeless tobacco users. J Am Dent Assoc 121:117–123
Hellden L, Jonsson R (1987). A papillary-verrucous lesion in the gingiva, developing into squamous cell carcinoma. J Clin Periodontol 14:56–61
Hogewind WFC, van der Kwast WAM, van der Waal I (1989) Oral leukoplakia with emphasis on malignant transformation: a follow-up study of 46 patients. J Craniomaxillofac Surg 17:128–133
Kabata K, Nakamura S, Ikemura K, Terashima H (1987) A case of erythroplakia of the palate, tongue and gingiva. J UOEH 9:341–345
Kim J, Shin DM, El-Naggar A et al (2001) Chromosome polysomy and histological characteristics in oral premalignant lesions. Cancer Epidemiol 10:319–325
Lee JJ, Hong WK, Hittelman WN, Mao L, Lotan R, Shin DM, Benner SE, Xu XC, Lee JS, Papadimitrakopoulou VM, Geyer C, Perez C, Martin JW, El-Naggar AK, Lippman SM (2000) Predicting cancer development in oral leukoplakia: ten years of translational research. Clin Cancer Res 6:1702–1710
Lippman SM, Hong WK (2001) Molecular markers of the risk of oral cancer. N Engl J Med 344:1323–1326
Marley JJ, Linden GJ, Cowan CG, Lamey PJ, Johnson NW, Warnakulasuriya KAAS, Scully C (1998) A comparison of the management of potentially malignant oral mucosal lesions by oral medicine specialists and oral and maxillofacial surgeons in the UK. J Oral Pathol Med 27:483–488
Schoelch ML, Sekandari N, Regezi JA, Silverman S Jr (1999) Laser management of oral leukoplakias: a follow-up study of 70 patients. Laryngoscope 109:949–953
Sciubba J (1999) Improving detection of precancerous and cancerous oral lesions: computer-assisted analysis of the oral brush biopsy. U.S. Collaborative Oral CDx Study Group. J Am Dent Assoc 130:1445–1457
Scully C, Cawson RA (1996) Potentially malignant oral lesions. J Epidemiol Biostatistics 1:3–12
Scully C (1993) Clinical diagnostic methods for the detection of premalignant and early malignant oral lesions. Comm Dent Health 1 [Suppl 1]:43–52
Scully C (1995) Oral precancer: preventive and medical approaches to management. Oral Oncol Eur J Cancer Part B 131:16–26
Shafer WG, Waldron CA (1975) Erythroplakia of the oral cavity. Cancer 36:1021–1028
Silverman S Jr, Gorsky M, Lozada F (1984) Oral leukoplakia and malignant transformation: a follow-up study of 257 patients. Cancer 53:563–568
Sudbo J, Kildal W, Risberg B et al (2001) DNA content as a prognostic marker in patients with oral leukoplakia. N Engl J Med 344:1270–1278
Tradati N, Grigolat R, Calabrese L, Costa L, Giugliano G, Morelli F, Scully C, Boyle P, Chiesa F (1997) Oral leukoplakias: to treat or not? Oral Oncol 33:317–322
Van der Waal I, Schepman KP, van der Meij EH (2000) A modified classification and staging system for oral leukoplakia. Oral Oncol 36:264–266
World Health Organisation (1978) Collaborating centre for oral precancerous lesions. Definition of leukoplakia and related lesions: an aid to studies in oral precancer. Oral Surg 46:518–539
Wright JM (1994) Oral precancerous lesions and conditions. Semin Dermatol 13:125–131

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

CHAPTER 32

Benign Neoplasms

Fibromas

▶ A benign neoplasm of fibroblastic origin

Lipoma

Definition ▶ A benign lesion of fatty tissue

Etiology ▶ Unclear

Gingival Involvement ▶ Rare

Other Sites of Involvement ▶ There are rare syndromes such as:
- Dercum's disease (multiple lipomatosis)
- Bannayan-Riley-Ruvalcaba syndrome (macrocephaly, multiple lipomas, intestinal hamartomatous polyps, vascular malformations and pigmented macules of the penis)

Main Clinical Features ▶ A painless semi-fluctuant yellowish swelling (Figs. 366, 367).

Diagnosis ▶ Diagnosis is from history and clinical features.
▶ Histopathology.

FIGURE 366

Lipoma

For Personal Use Only
Library of School of Dentistry, TUMS

G. Laskaris et al., *Periodontal Manifestations of Local and Systemic Diseases*
© Springer-Verlag Berlin Heidelberg 2003

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 367

Lipoma

Differential Diagnosis
- Abscesses
- Exostoses
- Hyperplastic gingivitis
- Fibrous epulis
- Cysts
- Giant cell tumour
- Pyogenic granuloma
- Neoplasms

Treatment
- Surgical excision

Neurofibroma

Definition
- Neurofibroma represents a benign overgrowth of all elements of a peripheral nerve (axon cylinder, Schwann cells, and fibrous connective tissue), arranged in a variety of patterns.

Etiology
- Congenital

Gingival Involvement
- Rare

Other Sites of Involvement
- Neurofibroma is a rare lesion which typically affects the tongue. Neurofibromas may occur multiply as a feature of neurofibromatosis and may rarely undergo sarcomatous change.

Main Clinical Features
- Neurofibromas usually present as a swelling (Figs. 368, 369) and may be part of:
  - The multiple endocrine adenoma (MEA) syndrome
  - Neurofibromatosis (NF), which is now recognized as consisting of distinct variants
    - Type I (NF-I), often referred to as von Recklinghausen's disease (Fig. 370), which represents 90 % of all forms of neurofibromatosis. It is a disease with an incidence of 1:3000 neonates with a dominant autosomic form of transmission, but 50 % of all cases are sporadic owing to new mutations. Neurofibromas are seen mainly in NF-I.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 368

Schwannoma

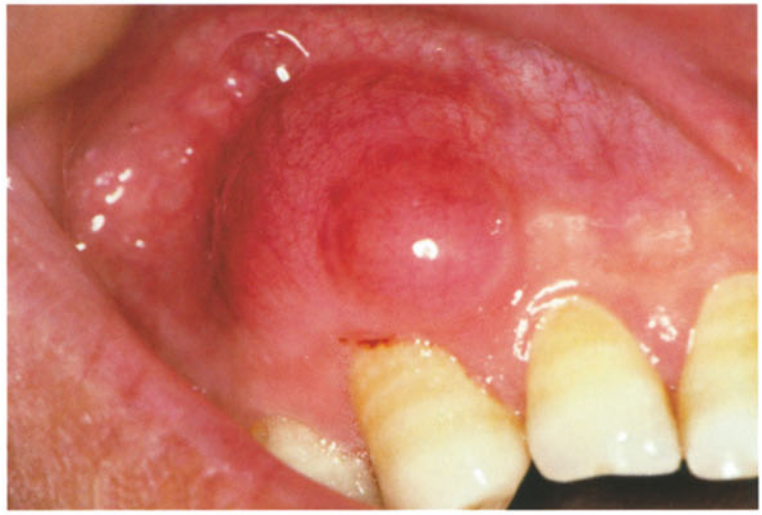


FIGURE 369

Neurofibroma

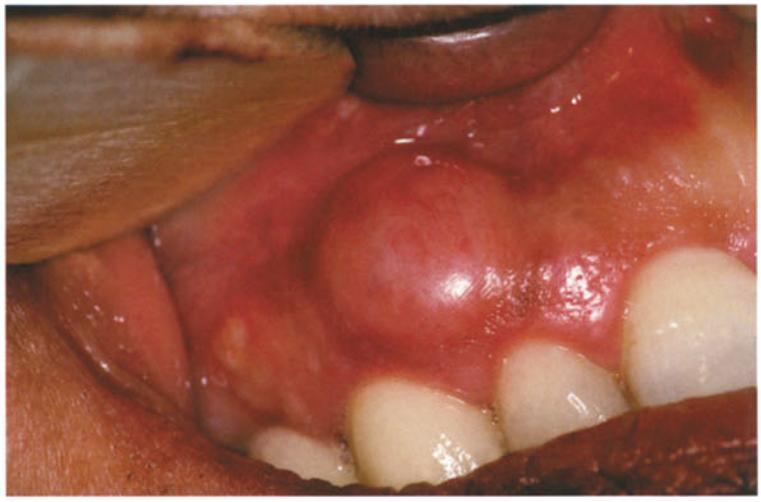


FIGURE 370

Von Recklinghausen's disease. Skin neurofibromas

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

- Type II (NF-II), a much less common disorder, which largely afflicts the central nervous system, often with bilateral acoustic schwannomas and has a more gradual onset. Neurofibromas may be seen in NF-II, but neurilemomas and acoustic neuromas are the predominant neural tumours. Cosmetic lesions include pigmentary changes (cafe-au-lait spots).

Diagnosis
- Diagnosis is from history and clinical features.
- Histopathology.
- Radiography.

Differential Diagnosis
- Abscesses
- Exostoses
- Hyperplastic gingivitis
- Fibrous epulis
- Cysts
- Giant cell tumour
- Pyogenic granuloma
- Neoplasms

Treatment
- Surgical excision

Ossifying Fibroma

Definition
- The nature of the ossifying fibroma is obscure because it has features both of a developmental anomaly and a neoplasm.

Etiology
- Idiopathic

Gingival Involvement
- Uncommon

Other Sites of Involvement
- Usually presents as a lesion arising in the jaw, sometimes only revealed coincidentally by imaging, at other times causing a swelling.

Main Clinical Features
- It presents as a painless, localized slow-growing, hard swelling of the jaw (Figs. 371–374).

Diagnosis
- Diagnosis is from history and clinical features.
- Imaging: a well-defined radiolucency area with a thin sclerotic margin containing irregular opaque masses.
- Histology: little or no distinction between ossifying fibroma and fibrous dysplasia.

Differential Diagnosis
- Abscesses
- Exostoses
- Hyperplastic gingivitis
- Fibrous epulis
- Cysts
- Giant cell tumour
- Pyogenic granuloma
- Neoplasms

Treatment
- Surgical enucleation

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 371

Peripheral ossifying fibroma

FIGURE 372

Peripheral ossifying fibroma

FIGURE 373

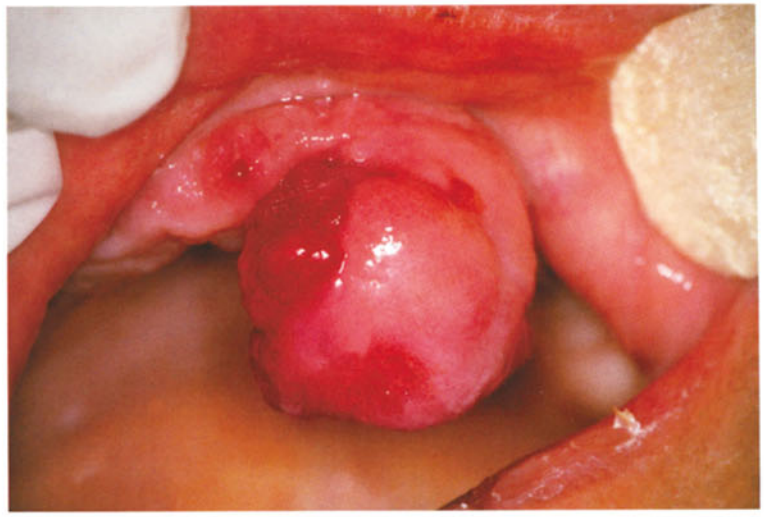
Peripheral ossifying fibroma

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 374

Peripheral ossifying fibroma



Chondroma

Definition
- Benign neoplasm of cartilage

Etiology
- Unclear

Gingival Involvement
- The lesion is uncommon but may present as a swelling involving the gingiva.

Other Sites of Involvement
- Usually presents as a lesion arising in the jaw, sometimes only revealed coincidentally by imaging, at other times causing a swelling.

Main Clinical Features
- This tumour is rare in the jaws. Chondromas are typically found in the elderly in the anterior maxilla, mandibular symphysis and the coronoid and condylar processes. They form slow-growing, painless masses (Fig. 375).
- Multiple enchondromas are seen in the rare Ollier's syndrome but oral tumours are a rare feature of this condition.

Diagnosis
- Diagnosis is from history and clinical features.
- Radiography.
- Histopathology.

Differential Diagnosis
- Abscesses
- Exostoses
- Hyperplastic gingivitis
- Fibrous epulis
- Cysts
- Giant cell tumour
- Pyogenic granuloma
- Neoplasms

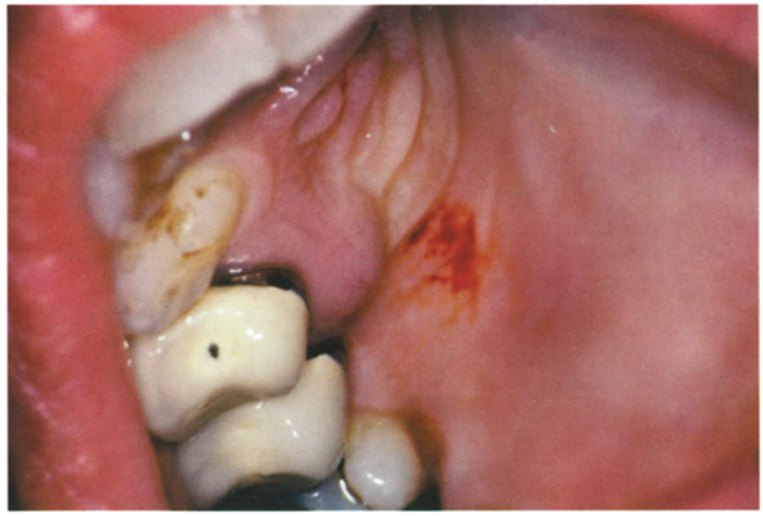
Treatment
- If surgical excision is complete recurrence is unlikely.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 375

Chondroma



Salivary Adenomas

Pleomorphic Adenoma (Mixed Salivary Gland Tumour)

Definition

Pleomorphic adenoma appears to originate from ductal epithelium that proliferates to contribute many duct-like spaces, sheets of epithelial cells and sometimes areas of squamous metaplasia. There are also areas reminiscent of connective tissue such as cartilage. The admixture of epithelial elements with what resembles fibrous, myxoid or cartilage tissue, leads to the name "mixed tumour".

Etiology

- Unknown

Gingival Involvement

- Rare

Other Sites of Involvement

- Intraorally pleomorphic salivary adenomas (PSAs) are seen mainly in the palate

Main Clinical Features

- The PSA is the most common salivary gland neoplasm, usually seen in the parotid, slow-growing and benign mass. Most pleomorphic adenomas are lobulated rubbery swellings with normal overlying mucosa but a bluish appearance (Fig. 376). They are not fixed to deeper tissues.
- Malignant change is uncommon but is suggested clinically by rapid growth, pain or fixation to deep tissues.

Diagnosis

- Diagnosis is from history and clinical features.
- Imaging.
- Histopathology.

Differential Diagnosis

- Other salivary tumours

Treatment

- Excision. The tumour is poorly encapsulated.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 376

Pleomorphic adenoma

Reading List

Allen CM, Miloro M (1997) Gingival lesion of recent onset in a patient with neurofibromatosis. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 84:595–597

Bekisz O, Darimont F, Rompen EH (2000) Diffuse but unilateral gingival enlargement associated with von Recklinghausen neurofibromatosis: a case report. J Clin Periodontol 27:361–365

Berke SL, Alt RM, Boguslaw BI, Sherman PM (1986) Unusual intraoral lipoma associated with the mental nerve. Ann Dent 45:31–32

Brannon RB, Houston GD, Meader CL (1985) Adenomatoid hyperplasia of mucous salivary glands: a case involving the retromolar area. Oral Surg Oral Med Oral Pathol 60:188–190

Giunta JL, Kaplan MA (1990) Peripheral, soft tissue odontomas. Two case reports. Oral Surg Oral Med Oral Pathol 69:406–411

Graham GS, Brannon RB, Houston GD (1988) Fibrolipoma of the gingiva. A case report. J Periodontol 59:118–120

Kimura K, Yamamoto H (1993) Neurofibroma of the gingiva in a child: report of case. ASDC J Dent Child 60:67–70

Kornbrot A, Tatoian JA Jr (1983) Benign soft tissue tumors of the oral cavity. Int J Dermatol 22:207–214

Tahsinoglu M, Timocin N, Cologlu AS, Kuralay T (1972) Gingival lipoma. Case report. Istanbul Univ Dishekim Fak Derg 3:287–290

Tosios K, Laskaris G, Eveson J, Scully C (1993) Benign cartilaginous tumor of the gingiva. A case report. Int J Oral Maxillofac Surg 22:231–233

Wilson DF, MacEntee MI (1974) Papillary cystadenoma of ectopic minor salivary gland origin. Oral Surg Oral Med Oral Pathol 37:915–918

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

CHAPTER 33

Malignant Neoplasms

Oral Squamous Cell Carcinoma

Definition

- Most mouth cancer is oral squamous cell carcinoma (OSCC). Most oral cancer is cancer of the lip (International Classification of Diseases (ICD 140), tongue (ICD 141), gum (ICD 143), floor of the mouth (ICD 144) and unspecified parts of the mouth (ICD 145).

Etiology

- OSCC is seen especially in:
  - Tobacco users.
  - Alcohol users.
  - Betel users.
  - Lower socioeconomic groups.
  - Ethnic minority groups.

Gingival Involvement

- Rare except in some groups, such as Japanese

Other Sites of Involvement

- Some 25% of OSCC affects the tongue – the common intra-oral site. The majority of OSCC involves the lateral border of the tongue and/or the floor of the mouth.

Main Clinical Features

- Features which suggest malignancy (Figs. 377–380) include:
  - Erythroplasia.
  - A granular appearance.
  - An ulcer with fissuring or raised exophytic margins.
  - Abnormal blood vessels supplying a lump.
  - Induration beneath a lesion, i.e. a firm infiltration beneath the mucosa.
  - Cervical lymph node enlargement.
- The whole mucosa should be examined as there may be widespread dysplastic mucosa (field change) or even a second primary neoplasm in the upper aerodigestive tract seen in those with OSCC over 3 years in up to 25%.

Diagnosis

- Diagnosis is from history and clinical features, supplemented by:
  - Histopathology.
- Imaging is indicated to exclude local spread and second primary neoplasms.

Differential Diagnosis

- Other neoplasms
- Major aphthae
- Behçet's syndrome
- Systemic disease:
  - Infections
  - Haematological disease

For Personal Use Only
Library of School of Dentistry, TUMS

G. Laskaris et al., *Periodontal Manifestations of Local and Systemic Diseases*
© Springer-Verlag Berlin Heidelberg 2003

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 377

Gingival squamous cell carcinoma

FIGURE 378

Gingival squamous cell carcinoma

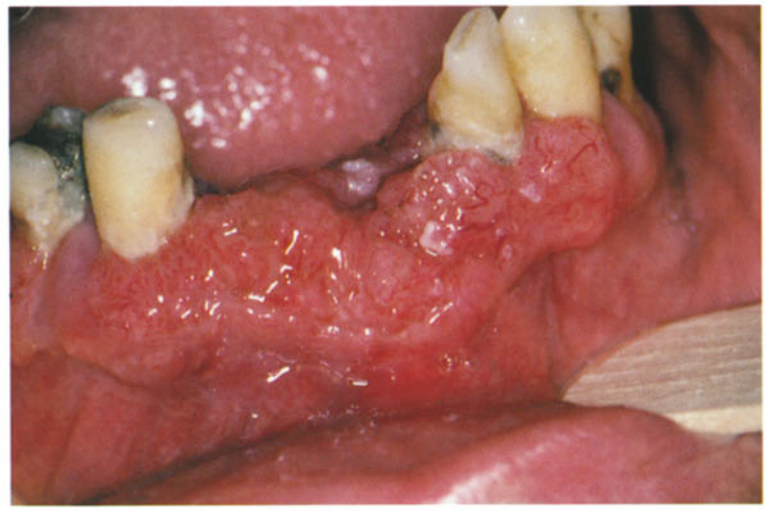
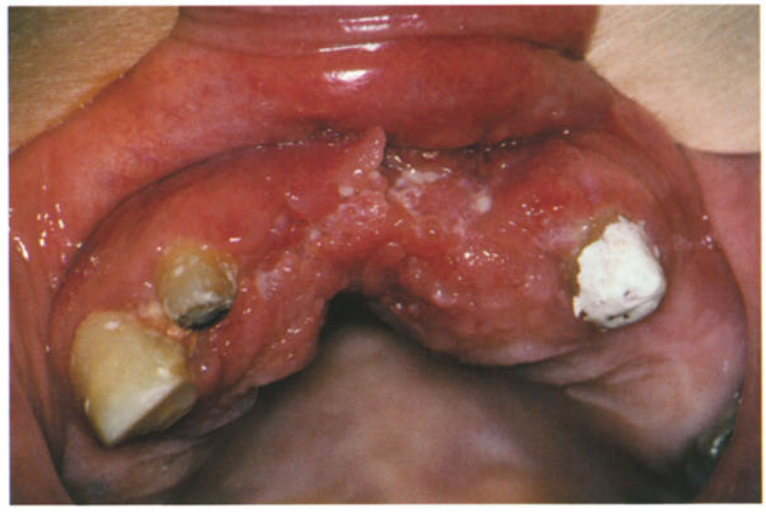


FIGURE 379

Gingival squamous cell carcinoma

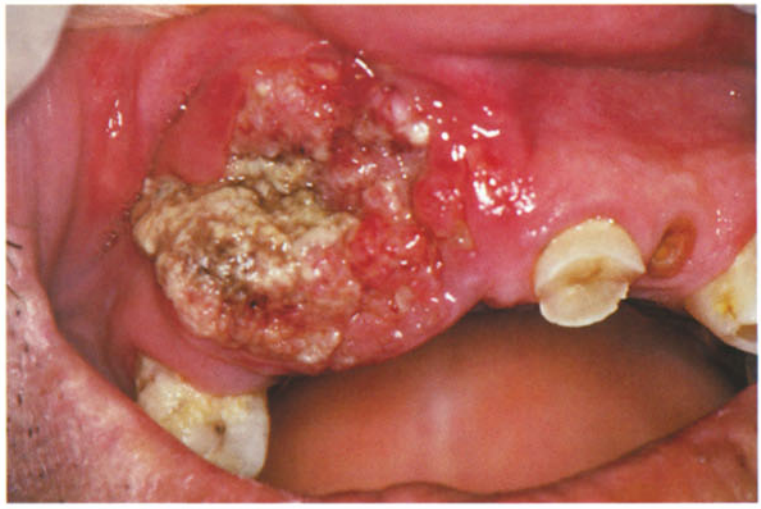


For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 380

Gingival squamous cell carcinoma



- Gastrointestinal disease
- Mucocutaneous disease
- Drugs
- Irradiation

Treatment ▶ Surgery and/or radiotherapy

Verrucous Carcinoma

Definition ▶ A vegetating carcinoma that arises exophytically

Etiology ▶ Human papillomavirus

Gingival Involvement ▶ Common

Other Sites of Involvement ▶ Other mucosal sites (palate, buccal mucosa, tongue)

Main Clinical Features ▶ Nodular or warty white lesion (Figs. 381, 382).

Diagnosis ▶ Diagnosis is from history and clinical features, supplemented by:
- Biopsy.

Differential Diagnosis
- ▶ Abscesses
- ▶ Exostoses
- ▶ Hyperplastic gingivitis
- ▶ Fibrous epulis
- ▶ Cysts
- ▶ Giant cell tumour
- ▶ Pyogenic granuloma
- ▶ Neoplasms

Treatment ▶ Surgery

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 381

Gingival verrucous carcinoma

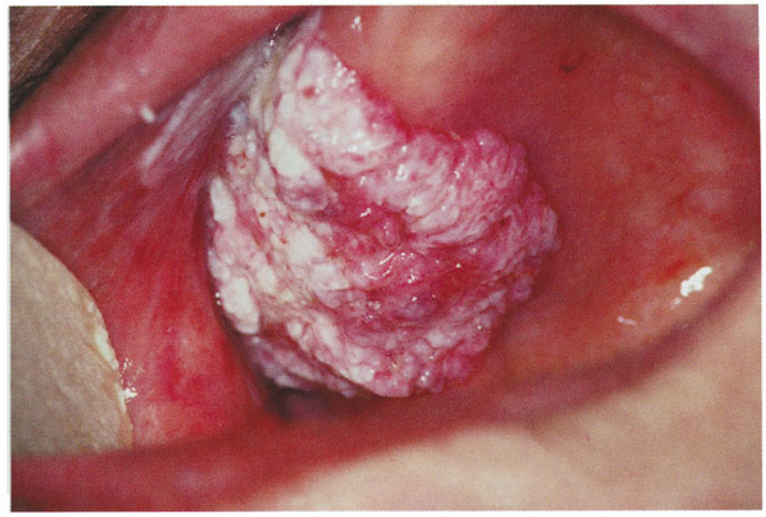


FIGURE 382

Gingival verrucous carcinoma

Salivary Carcinomas

Mucoepidermoid Carcinoma

- Mucoepidermoid carcinoma accounts for up to 10% of salivary gland tumours and is a slow-growing tumour of low-grade malignancy or is benign (Fig. 383).

Acinic Cell Carcinoma

- Acinic cell tumours are very rare tumours that are usually benign, although all grades of malignancy have been reported.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 383

Mucoepidermoid carcinoma

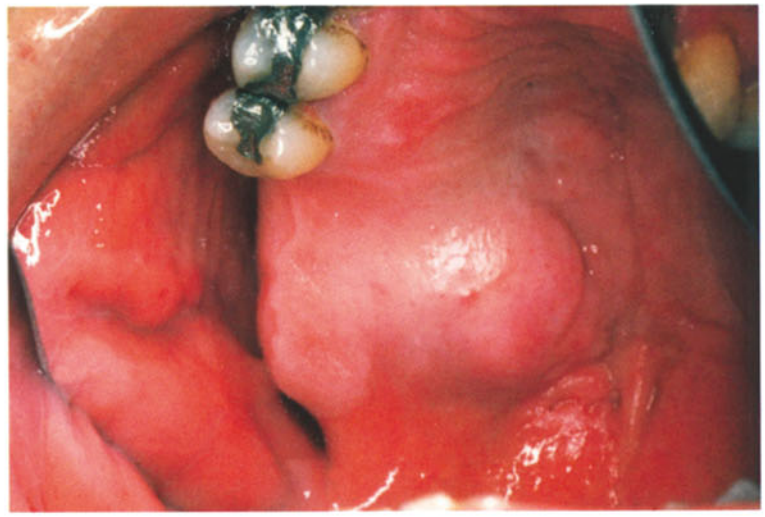
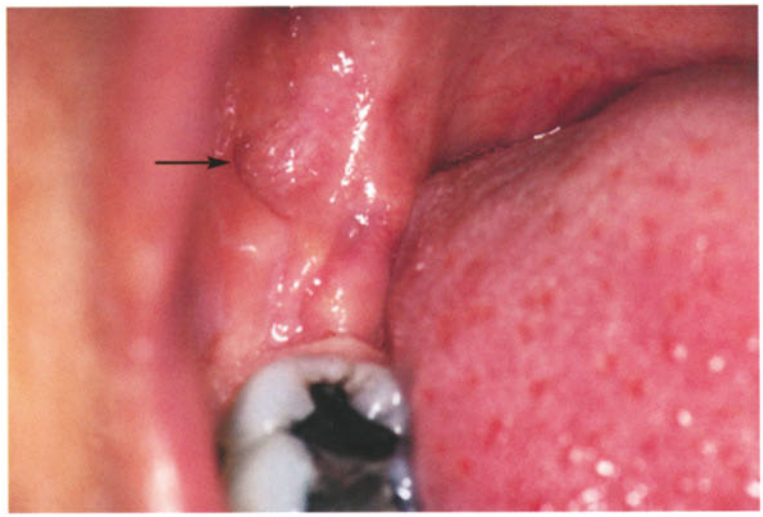


FIGURE 384

Adenocystic carcinoma



For Personal Use Only
Library of School of Dentistry, TUMS

Adenoid Cystic Carcinoma (Cylindroma)

▶ This is a slow-growing malignant tumour which has a tendency to infiltrate, spread perineurally, and metastasize (Fig. 384).

Osteosarcoma

Definition

▶ Malignant neoplasm arising from osteoblasts

Etiology

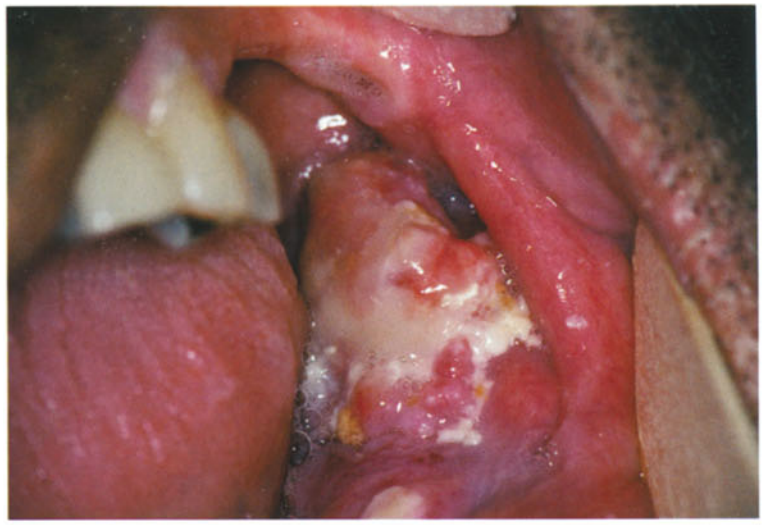
▶ Idiopathic, although there is an increased incidence in patients who:

- Have retinoblastoma.
- Have a history of irradiation for other lesions (about 3% of patients with osteosarcomas).
- Are over 50, which probably represents the small but significant malignant change in patients with pre-existing Paget's disease.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 385

Osteosarcoma



Gingival Involvement ▶ Uncommon but may present as a swelling involving the gingiva

Other Sites of Involvement ▶ Usually presents as a lesion arising in the jaw, sometimes only revealed coincidentally by imaging, at other times causing a swelling

Main Clinical Features ▶ Rapidly-growing bony swelling (Fig. 385).

Diagnosis ▶ Diagnosis is from history and clinical features, supplemented by:
- Imaging.
- Histopathology.

Differential Diagnosis
- ▶ Abscesses
- ▶ Exostoses
- ▶ Hyperplastic gingivitis
- ▶ Fibrous epulis
- ▶ Cysts
- ▶ Giant cell tumour
- ▶ Pyogenic granuloma
- ▶ Neoplasms

Treatment ▶ Resection and chemotherapy

Ewing's Sarcoma

Definition ▶ A primary malignant neoplasm of undifferentiated mesenchymal cells of bone of uncertain origin

Etiology ▶ Unclear

Gingival Involvement ▶ Uncommon but may present as a swelling involving the gingiva

Other Sites of Involvement ▶ Usually presents as a lesion arising in the jaw, sometimes only revealed coincidentally by imaging, at other times causing a swelling

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 386

Ewing's sarcoma on the lower gingiva

Main Clinical Features
- Affects mainly children and young adults, particularly males. Rare in the jaws.
- Tumour is extremely aggressive, expands rapidly (Fig. 386) and invades the soft tissues, and metastasizes early via both lymphatics and blood stream.

Diagnosis
- Diagnosis is from history and clinical features, supplemented by:
  - Imaging: it has either a sunray appearance or an onion-peel appearance due to deposition of new bone in layers.
  - Histopathology.

Differential Diagnosis
- Abscesses
- Exostoses
- Hyperplastic gingivitis
- Fibrous epulis
- Cysts
- Giant cell tumour
- Pyogenic granuloma
- Neoplasms

Treatment
- Combination of chemotherapy and radiotherapy, has led to a 5-year survival rate of over 50%.

Leiomyosarcoma

Etiology
- Idiopathic

Gingival Involvement
- Rare

Other Sites of Involvement
- Oral mucosa
- Other sites

Main Clinical Features
- Non-specific swelling (Fig. 387).

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 387

Leiomyosarcoma on the gingiva

Diagnosis
- Diagnosis is from history and clinical features.
- Biopsy

Differential Diagnosis
- Abscesses
- Exostoses
- Hyperplastic gingivitis
- Fibrous epulis
- Cysts
- Giant cell tumour
- Pyogenic granuloma
- Neoplasms

Treatment
- Surgery

Chondrosarcoma

Definition
- Malignant neoplasm of cartilage

Etiology
- Although isolated cases arise from chondromas, most appear de novo.

Gingival Involvement
- Uncommon but may present as a swelling involving the gingiva

Other Sites of Involvement
- Usually presents as a lesion arising in the jaw, sometimes only revealed coincidentally by imaging, at other times causing a swelling.

Main Clinical Features
- Many grow rapidly and produce extensive local destruction (Fig. 388).

Diagnosis
- Diagnosis is from history and clinical features, supplemented by:
  - Histopathology.
  - Radiography.

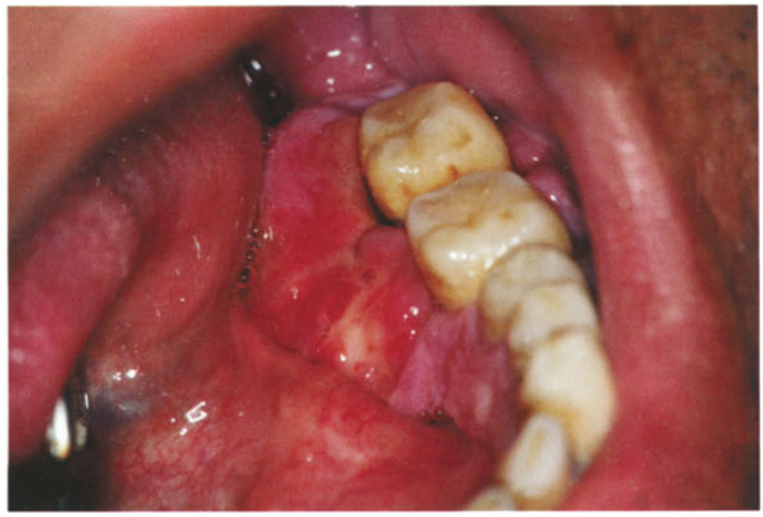
Differential Diagnosis
- Abscesses
- Exostoses
- Hyperplastic gingivitis
- Fibrous epulis

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 388

Chondrosarcoma



- Cysts
- Giant cell tumour
- Pyogenic granuloma
- Neoplasms

Treatment

- Radical surgery

Metastatic Neoplasms

Definition

- Tumour deposits arising from lymphatic or haematogenous spread

Etiology

- Usually arise from carcinomas in the head and neck or from lymphoproliferative lesions, but can also arise from carcinomas especially in (decreasing order of frequency):
  - Breast.
  - Lungs.
  - Kidney.
  - Thyroid.
  - Stomach.
  - Colon.
  - Prostate.
- Metastases can also arise from sarcomas (e.g. angiosarcoma, leiomyosarcoma, osteosarcoma).

Gingival Involvement

- Uncommon but may present as a swelling involving the gingiva or periodontal destruction

Other Sites of Involvement

- Usually presents as a lesion arising in the jaw, sometimes only revealed coincidentally by imaging, at other times causing symptoms. In up to one-third of patients, the jaw lesions are the first manifestation of the tumour.

Main Clinical Features

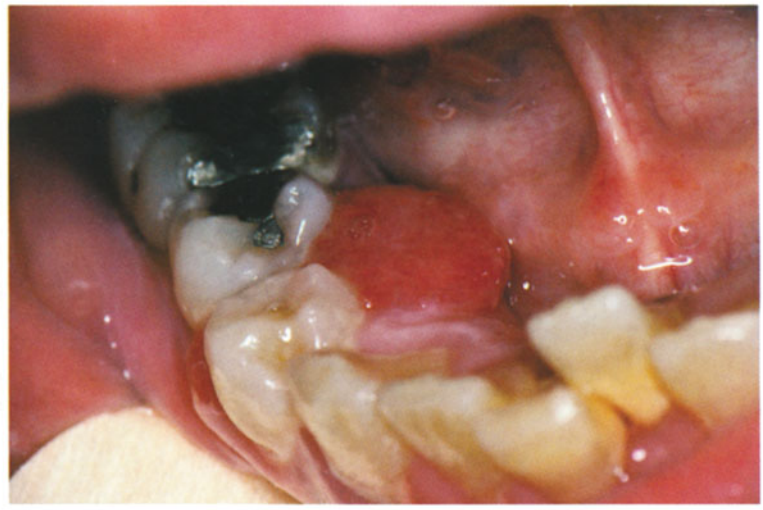
- Metastases to the jaws mainly affect the premolar/molar region of the mandible (Fig. 389). Non-Hodgkin's lymphomas are frequently gingi-

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 389

Gingival metastasis from breast cancer



val or faucial in location. Many metastases are asymptomatic but others manifest with:

- Pain
- Paraesthesia or hypoaesthesia
- Swelling
- Tooth mobility
- Non-healing extraction sockets
- Pathological fracture
- Radiolucency or radio-opacity

Diagnosis

- Diagnosis is from history and clinical features, supplemented by:
  - Radiography.
  - Histopathology.

Differential Diagnosis

- Infections and other causes of radiolucencies.
- Metastases from the prostate tend to be osteoplastic and may be confused imaging with chronic forms of osteomyelitis, Paget's disease and cemental lesions.
- Abscesses.
- Exostoses.
- Fibrous epulis.
- Cysts.
- Giant cell tumour.
- Pyogenic granuloma.
- Other neoplasms.

Treatment

- Treatment of the primary lesion
- Radiotherapy, surgery or chemotherapy

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 390

Classic Kaposi's sarcoma

Classic Kaposi's Sarcoma

Definition
- Kaposi's sarcoma (KS) is an endothelial cell multicentric malignant neoplasm characterized by increased capillary growth and prominent spindle-shaped cells with surprisingly few mitoses, extravazation of erythrocytes, and the appearance of hemosiderin.

Etiology
- Found mainly in elderly Jewish patients and Africans

Gingival Involvement
- Common

Other Sites of Involvement
- The palate, gingiva, tongue, or other oral sites (Fig. 390)
- Multifocal, widespread lesions involving skin, lymph nodes and visceral organs such as the gastrointestinal tract, lung, liver and spleen

Main Clinical Features
- Early lesions of oral KS typically present as a pigmented macule.
- Later lesions present as red, bluish, or purple nodules, sometimes ulcerated.
- Non-discoloured oral KS is also seen.
- KS may also present as cervical lymph node or salivary gland swellings.

Diagnosis
- Diagnosis is from history and clinical features, supplemented by:
  - Histology.

Differential Diagnosis
- Racial pigmentation
- Localized irritation, e.g. smoking
- Amalgam tattoo
- Naevus
- Melanotic macule
- Malignant melanoma
- Epithelioid angiomatosis
- Drugs, e.g. antimalarials
- Addison's disease

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Treatment
- Single and multiple chemotherapy agents. Laser excision, radiotherapy, cryotherapy or intralesional vinblastine or interferon-alpha may produce regression of oral KS.

Melanoma

Definition
- Malignant neoplasm of melanocytes

Etiology
- Sunlight exposure is causal in skin melanomas, which have increased in almost epidemic fashion over the past decades, especially in older fair-skinned peoples.

Gingival Involvement
- Rare

Other Sites of Involvement
- Skin
- Any oral location, particularly the palate

Main Clinical Features
- Early:
  - Brown or black.
  - Initially a macule.
  - Solitary.
  - Small.
  - Asymptomatic.
  - Grow rapidly, initially spreading radially and superficially.
- Later:
  - Increasingly pigmented (Fig. 391).
  - Nodular.
  - Deeply invasive.
  - Satellite lesions.
- Occasionally melanomas are deep-spreading nodular ab initio or are non-pigmented, multiple or large.

Diagnosis
- Diagnosis is from history and clinical features, supplemented by:
  - Histopathology.
- Imaging may be helpful whenever hyperpigmentation may be caused by amalgam, graphite or a foreign body.
- Photographs may be useful for future comparison.

Differential Diagnosis
- Racial pigmentation
- Localized irritation, e.g. smoking
- Amalgam tattoo
- Naevus
- Melanotic macule
- Malignant melanoma
- Kaposi's sarcoma
- Drugs, e.g. antimalarials
- Addison's disease

Treatment
- Melanomas less than about 0.75 mm thick are superficial, rarely metastasize, and can be excised in their entirety with a good prognosis.
- Others have a poor prognosis but can be excised and some centres use chemo- and immunotherapy.

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

FIGURE 391

Malignant melanoma

Reading List

Abdullah BH, Yahya HI, Talabani NA, Alash NI, Mirza KB (2000) Gingival and cutaneous angiosarcoma. J Oral Pathol Med 29:410–412

Alandez J, Llanes F, Herrera JI, Carasol M, Bascones A (1995) Metastatic lung carcinoma involving the periodontium. Report of a case. J Periodontol 66:896–898

Ardekian L, Rosen DJ, Peled M, Rachmiel A, Machtei EE, el Naaj IA, Laufer D (2000) Primary gingival malignant melanoma. Report of 3 cases. J Periodontol 71:117–120

Barker BF, Carpenter WM, Daniels TE, Kahn MA, Leider AS, Lozada-Nur F, Lynch DP, Melrose R, Merrell P, Morton T, Peters E, Regezi JA, Richards SD, Rick GM, Rohrer MD, Slater L, Stewart JC, Tomich CE, Vickers RA, Wood NK, Young SK (1997) Oral mucosal melanomas: the WESTOP Banff workshop proceedings. Western Society of Teachers of Oral Pathology. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 83:672–679

Bschorer R, Lingenfelser T, Kaiserling E, Schwenzer N (1993) Malignant lymphoma of the mucosa-associated lymphoid tissue (MALT) – consecutive unusual manifestation in the rectum and gingiva. J Oral Pathol Med 22:190–192

Cengiz M, Ozyar E, Ersu B, Akyol FH, Atahan IL (1999) High-dose-rate mold brachytherapy of early gingival carcinoma: a clinical report. J Prosthet Dent 82:512–514

Dry SM, Jorgensen JL, Fletcher CD (2000) Leiomyosarcomas of the oral cavity: an unusual topographic subset easily mistaken for nonmesenchymal tumours. Histopathology 36:210–220

Economopoulos T, Fountzilas G, Kostourou A, Daniilidis J, Pavlidis N, Andreopoulou H, Nicolaou A, Papageorgiou E, Mellou S, Dervenoulas J, Stathakis N (1998) Primary extranodal non Hodgkin's lymphoma of the head and neck in adults: a clinicopathological comparison between tonsillar and nontonsillar lymphomas. (Hellenic co-Operative Oncology Group). Anticancer Res 18:4655–4660

Ellis GL, Jensen JL, Reingold IM, Barr RJ (1977) Malignant neoplasms metastatic to gingivae. Oral Surg Oral Med Oral Pathol 44:238–245

Favia G, Maiorano E, Orsini G, Piattelli A (2001) Myxoid liposarcoma of the oral cavity with involvement of the periodontal tissues. J Clin Periodontol 28:109–112

Fuchihata H, Furukawa S, Murakami S, Kubo K, Shimizutani K, Ikeda H, Inoue T, Masaki N (1994) Results of combined external irradiation and chemotherapy of bleomycin or peplomycin for squamous cell carcinomas of the lower gingiva. Int J Radiat Oncol Biol Phys 29:705–709

Goldschmidt PR, Goldschmidt JD, Lieblich SE, Eisenberg E (1999) Leiomyosarcoma presenting as a mandibular gingival swelling: a case report. J Periodontol 70:84–89

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Gomez D, Faucher A, Picot V, Siberchicot F, Renaud-Salis JL, Bussieres E, Pinsolle J (2000) Outcome of squamous cell carcinoma of the gingiva: a follow-up study of 83 cases. J Craniomaxillofac Surg 28:331–335

Horie Y, Suou T, Hirayama C, Urabe N, Yamamoto T, Ikoma H, Hashimoto K, Gomyoda M (1985) Hepatocellular carcinoma metastatic to the oral cavity including the maxilla and the mandible: report of two cases and review of the literature. Gastroenterol Jpn 20:604–610

Koch BB, Trask DK, Hoffman HT, Karnell LH, Robinson RA, Zhen W, Menck HR (2001) National survey of head and neck verrucous carcinoma: patterns of presentation, care, and outcome. Cancer 92:110–120

Landa SJ, Zarem HA (1973) Cancer of the floor of the mouth and the gingiva. Surg Clin North Am 53:135–145

Margiotta V, Franco V, Rizzo A, Porter S, Scully C, Di Alberti L (1999) Gastric and gingival localization of mucosa-associated lymphoid tissue (MALT) lymphoma. An immunohistochemical, virological and clinical case report. J Periodontol 70:914–918

Marta U, Zsuzsanna S, Jozsef B, Zsolt N, Bela S, Gyorgy S (2001) Rare incidence of three consecutive primary tumors in the maxillofacial region: retinoblastoma, leiomyosarcoma, and choriocarcinoma: case report. J Craniofac Surg 12:464–468

Medina BR, Barba EM, Torres AV, Trujillo SM (2001) Gingival metastases as first sign of a primary uterine angiosarcoma. J Oral Maxillofac Surg 59:467–471

Park YW (1998) Non-Hodgkin's lymphoma of the anterior maxillary gingiva. Otolaryngol Head Neck Surg 119:146

Patton LL, Brahim JS, Baker AR (1994) Metastatic malignant melanoma of the oral cavity. A retrospective study. Oral Surg Oral Med Oral Pathol 78:151–156

Piattelli A, Fioroni M, Rubini C (2000) Gingival metastasis from a medullary thyroid carcinoma: case report. J Periodontol 71:112–116

Raut A, Huryn J, Pollack A, Zlotolow I (2000) Unusual gingival presentation of post-transplantation lymphoproliferative disorder: a case report and review of the literature. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 90:436–441

Rogers RS III, Gibson LE (1997) Mucosal, genital and unusual clinical variants of melanoma. Mayo Clin Proc 72:362–366

Sawada Y, Nomura K, Hashimoto I (1986) Lentigo maligna melanoma with deep invasion along the mental nerve. J Maxillofac Surg 14:177–179

Scipio JE, Murti PR, Al-Bayaty HF, Matthews R, Scully C (2001) Metastasis of breast carcinoma to mandibular gingiva. Oral Oncology 37:393–396

Scully C, Eveson JW, Witherow H et al (1993) Oral presentation of lymphoma: case report of T-cell lymphoma masquerading as oral Crohn's disease, and review of the literature. Oral Oncol 29B:225–230

Scully C, Porter SR, Speight P, Eveson JW, Gale D (1999) Adenosquamous carcinoma of the mouth: a rare variant of oral squamous cell carcinoma. Int J Oral Maxillofac Surg 28:125–128

Shedd DP, Von Essen CF, Connelly RR, Eisenberg H (1968) Cancer of the buccal mucosa, palate and gingiva in Connecticut, 1935–1959. Cancer 21:440–446

Suzuki K, Yoshida H, Onizawa K, Onobori M (1999) Metastatic osteosarcoma to the mandibular gingiva: a case report. J Oral Maxillofac Surg 57:864–868

Tanaka N, Amagasa T, Iwaki H, Shioda S, Takeda M, Ohashi K, Reck SF (1994)Oral malignant melanoma in Japan. Oral Surg Oral Med Oral Pathol 78:181–190

Tong AC, Lam KY (2000) Granulocytic sarcoma presenting as an ulcerative mucogingival lesion: report of a case and review of the literature. J Oral Maxillofac Surg 58:1055–1058

Van den Bergh JP, Van der Waal I (1988) Ewing's sarcoma of the mandible: report of a case. J Oral Maxillofac Surg 46:798–800

Wiernik PH, De Bellis R, Muxi P, Dutcher JP (1996) Extramedullary acute promyelocytic leukemia. Cancer 78:2510–2514

Yalcin S, Turoglu HT, Ozdamar S, Sadikoglu Y, Gurbuzer B, Yenici O (1993) Ewing's tumor of the mandible. Oral Surg Oral Med Oral Pathol 76:362–367

Yokobayashi Y, Nakajima T, Fikushima M, Ishiki T (1981) Non-Hodgkin's lymphoma of the gingiva. A case report. Int J Oral Surg 10:359–362

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

CHAPTER 34

Fibro-osseous Lesions

Paget's Disease

Definition

- A disorder in which there is uncoordinated resorption and deposition of bone, producing larger but weaker and very vascular bones (osteitis deformans). Seen mainly in Anglo-Saxons and said to be a common condition affecting up to 5% of those over 55 years of age in some areas.

Etiology

- Unknown: virus has been implicated, possibly measles, canine distemper or respiratory syncytial virus.

Gingival Involvement

- The lesion is uncommon but may rarely present as a swelling involving the gingiva.

Other Sites of Involvement

- Monostotic and polyostotic forms are recognized.

Main Clinical Features

- Jaw swelling (Fig. 392) which is:
  - Typically progressive
  - Sometimes with pain
  - Sometimes with hypercementosis
- Complications may include:
  - Difficult tooth extractions
  - Postoperative bleeding
  - Postoperative infection
  - Constriction of cranial foramina leading to cranial neuropathies
  - Malignant change
  - Arteriovenous shunting, leading to cardiac failure

FIGURE 392

Paget's disease

For Personal Use Only
Library of School of Dentistry, TUMS

G. Laskaris et al., *Periodontal Manifestations of Local and Systemic Diseases*
© Springer-Verlag Berlin Heidelberg 2003

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Diagnosis

- Diagnosis is from history and clinical features.
- Histopathology (osteolytic stage).
- Imaging.
- Scintigraphy useful for revealing early and polyostotic lesions.
- Serum alkaline phosphatase: raised.
- Urinary hydroxyproline: raised.

Differential Diagnosis

- Bone neoplasms
- Other fibro-osseous lesions such as fibrous dysplasia
- Other conditions with a raised alkaline phosphatase, e.g. osteomalacia, hyperparathyroidism and osteoblastic metastatic deposits (e.g. carcinoma of the prostate)

Treatment

- Bisphosphonates or calcitonin slow the pathological process.

Reading List

Bennett J (1988) Paget's disease of bone: an update and dental aspects. Dent Update 15:299–302

Otis LL, Terezhalmy GT, Glass BJ (1986) Paget's disease of bone: etiological theories and report of a case. J Oral Med 41:214–219, 273

Wheeler TT, Alberts MA, Dolan TA, McGorray SP (1995) Dental, visual, auditory and olfactory complications in Paget's disease of bone. J Am Geriatr Soc 43:1384–1391

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Appendix

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Classification of Gingival Lesions by Signs and Symptoms

TABLE 1

	Keratinizing	Non-keratinizing
Gingival white lesions	Frictional keratosis Smoker's keratosis Leukoplakia Leukoplakia due to sanguinaria Hairy leukoplakia Candidal leukoplakia Chronic mucocutaneous candidiasis White sponge naevus Focal palmoplantar and oral mucosa hyperkeratosis syndrome Darier's disease Lichen planus Discoid lupus erythematosus Psoriasis Squamous cell carcinoma Verrucous carcinoma Others	Materia alba Chemical burns Pseudomembranous candidiasis Syphilitic mucous patches Necrotizing ulcerative gingivitis Others

TABLE 2

Gingival red lesions	Dental plaque-induced gingivitis Non-plaque-induced gingival lesions Chronic periodontitis Traumatic lesions Allergic reactions Drug-influenced gingivitis Plasma cell gingivitis Desquamative gingivitis Erythematous candidiasis Linear gingival erythema Herpetic gingivitis Granulomatous gingivitis Melkersson-Rosenthal syndrome Sarcoidosis Crohn's disease Glycogen storage disease type Ib Gonococcal infection Scurvy Gingivitis due to radiation	Erythroplakia Squamous cell carcinoma Leukaemia Cyclic neutropenia Thrombocytopenic purpura Soft tissue plasmatocytoma Capillary haemangioma Sturge-Weber syndrome Klippel-Trénaunay-Weber syndrome Cystic fibrosis Hereditary haemorrhagic telangiectasia Sjögren's syndrome Porphyria Reiter's disease Others

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

TABLE 3

Gingival ulcers and erosions

- Traumatic lesions
- Thermal burns
- Necrotizing ulcerative gingivitis
- Necrotizing ulcerative periodontitis
- Desquamative gingivitis
- Drugs (gold and others)
- Streptococcal gingivitis
- Herpetic gingivitis
- Aphthous ulcer
- Behçet's disease
- Tuberculosis
- Syphilis
- Measles
- Chickenpox
- Herpes zoster
- Gonococcal infection
- Cytomegalovirus infection
- Histoplasmosis
- Mucormycosis
- Paracoccidioidomycosis
- Langerhans cell histiocytosis
- Squamous cell carcinoma
- Other neoplasms
  - Pyostomatitis vegetans
  - Others
- Familial neutropenia
- Cyclic neutropenia
- Agranulocytosis
- Aplastic anaemia
- Leukaemias
- Multiple myeloma
- Non-Hodgkin's lymphoma
- Erythema multiforme
- Toxic epidermal necrolysis
- Pemphigal vulgaris
- Cicatricial pemphigoid
- Bullous pemphigoid
- Linear IgA disease
- Pemphigoid gestations
- Epidermolysis bullosa acquisita
- Hereditary epidermolysis bullosa
- Dermatitis herpetiformis
- Chronic ulcerative stomatitis
- Lichen planus
- Lupus erythematosus
- Mixed connective tissue disease
- Graft-versus-host disease
- Crohn's disease

TABLE 4

Gingival pigmented lesions

- Physiologic pigmentation
- Bismuth deposition
- Lead deposition
- Metal tattoo
- Other tattoo
- Smoker's melanosis
- Pencil peel
- Eruption cyst
- Lentigo
- Freckles
- Naevi
- Malignant melanona
- Addison disease
- Others

TABLE 5

Gingival bullous lesions

- Herpetic gingivitis
- Desquamative gingivitis
- Herpes zoster
- Chickenpox
- Erythema multiforme
- Cicatricial pemphigoid
- Bullous pemphigoid
- Bullous pemphigoid
- Linear IgA disease
- Epidermolysis bullosa acquisita
- Pemphigoid gestationis
- Dermatitis herpetiformis
- Pemphigus vulgaris
- Paraneoplastic pemphigus
- Lichen planus

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

TABLE 6

Gingival lumps and swellings, localized

- Gingival abscess
- Periodontal abscess
- Pericoronal abscess
- Periodontal fistula
- Exostoses
- Eruption cyst
- Gingival cyst of newborn
- Gingival cyst of adult
- Palatine papilla cyst
- Odontogenic cysts
- Condyloma acuminatum
- Verruca vulgaris
- Focal epithelial hyperplasia
- Pyogenic granuloma
- Pregnancy granuloma
- Peripheral giant cell granuloma
- Peripheral ossifying fibroma
- Traumatic fibroma
- Squamous cell carcinoma
- Verrucous carcinoma
- Bacillary angiomatosis
- Kaposi's sarcoma
- Non-Hodgkin's lymphoma
- Burkitt's lymphoma
- Cutaneous T-cell lymphoma
- Other neoplasms
- Granulomatous gingivitis
- Tuberous sclerosis
- Cowden's disease
- Gardner's syndrome
- Focal dermal hypoplasia syndrome
- Actinomycosis
- Crohn's disease
- Others

For Personal Use Only
Library of School of Dentistry, TUMS

TABLE 7

Gingival lumps and swellings, generalized

- Multiple exostoses
- Gingival enlargement due to phenytoin
- Gingival enlargement due to calcium channel blockers
- Gingival enlargement due to ciclosporin
- Hereditary gingival fibromatosis
- Laband's syndrome
- Hurler's syndrome
- Sturge-Weber syndrome
- Klippel-Trénaunay-Weber syndrome
- Pregnancy gingivitis
- Soft tissue plasmacytoma
- Melkersson-Rosenthal syndrome
- Crohn's disease
- Sarcoidosis
- Pyostomatis vegetans
- Amyloidosis
- Malignant acanthosis nigricans
- Familial acanthosis nigricans
- Leukaemia
- Wegener's granulomatosis
- Others

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

TABLE 8

	Local	Systemic
Causes of gingival bleeding	Dental plaque-induced gingivitis Non-plaque-induced gingivitis Chronic periodontitis Aggressive periodontitis Necrotizing ulcerative gingivitis Necrotizing ulcerative periodontitis Pregnancy gingivitis Pregnancy granuloma Pyogenic granuloma – Peripheral giant cell granuloma – Scurvy	Cyclic neutropenia Agranulocytosis Thrombocytogenic purpura Aplastic anaemia Leukaemia Clotting defects Drugs/anticoagulants Sturge-Weber syndrome Klippel-Trénaunay-Weber syndrome Chédiak-Higashi syndrome Desquamative gingivitis Primary herpetic gingivostomatitis Others

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Subject Index

A

abscess
– gingival 55, 56
– periapical 59
– pericoronal 57
– periodontal 56, 57
– tooth 58
acanthosis nigricans
– familial 131, 132
– malignant 279, 280
acrylic monomer, mucosal burn 70
ACTH 276
Actinobacillus actinomycetemcomitans 10, 25
Actinomyces (A.)
– *A. israelii* 181
– *A. naeslundii* 10
– *A. viscosus* 10
actinomycosis 163, 164
Addison's disease 276, 277
adenoma, pleomorphic 319, 320
agranulocytosis 200, 201
AIDS 187
alcohol, mucosal burn 69
allergy/allergic reaction 89–92
– allergic gingivitis 49
amalgam tattoo 83, 84
American Academy of Periodontology 17
amyloidosis 268–270
anaemia, aplastic 200–202
anatomy 3–8
angiofibroma 137
angiomatosis
– bacillary 180, 181
– *Sturge-Weber* 138–140, 303
ANUG (necrotizing ulcerative gingivitis) 93–95
aphthous ulceration 113–115
aspirin, mucosal burn 68
atrophy, skin 148
attachment level, clinical 14
autoimmune diseases 245–256

B

Bacteroides forsythus 10, 25
Bartonella henselae/quintana 180
B-cell 12
Behçet's disease 253–255
betel nut staining 52
biochemistry, gingiva 9, 10
biological width 6
bismuth pigmentation 52
bleeding, gingival 203
BMP (bone morphogenetic protein) 9
bone, alveolar 7
BOP (bleeding on probe) 14
Borrelia vincentii 95
Bourneville-Pringle syndrome 136
bullae, haemorrhagic 224, 225, 228, 229, 233
Burkitt's lymphoma 213, 214
Burton's line 51
butterfly rash 245

C

calcium channel blockers, gingival enlargement 42–44
Campylobacter rectus 10
candidiasis
– acute pseudomembranous 109, 110
– erythematous 110–112
– HIV 185–187
– leukoplakia 308
carcinoma
– acinic cell 324
– adenoid cystic 324, 325
– mucoepidermoid 324, 325
– oral squamous cell (OSCC) 321–324
– verrucous 323, 324
cementoenamel junction 7
cementum 7, 8
chancre 162
chemical lesions 67–70
chickenpox 168, 169
chlorhexidine, contact gingivitis 90
chondroectodermal dysplasia 145, 146
chondroma 318, 319
chondrosarcoma 328
ciclosporin, gingival enlargement 44–46
cinnamon, contact gingivitis 91
classification 339–342
– periodontal diseases, *table* 17–19
– tooth mobility 14–16
collagen 9
– fibres 7
condyloma acuminatum 103, 184
conjunctivitis 225
connective tissue 9
– mixed disease 250, 251
Cowden's disease 134, 135
coxsackie virus 173
CREST syndrome 250
Crohn's disease 31, 261–263
cutaneous T-cell lymphoma 214–216
cylindroma 324, 325
cyst
– dentigerous 292–294
– eruption 288, 289
– follicular 292
– gingival
– – adult 290, 291
– – newborn 289, 290
– keratocyst 294, 295
– palatine papilla 291, 292
– peripical 287, 288
– primordial 294
– radicular 288
cystic fibrosis 153, 154
cytokine 12
cytomegalovirus (CMV) 182, 183

D

Darier's disease 133, 134
dermatitis herpetiformis 232–234
developmental disorders 37–40
diabetes mellitus 273, 274
diagnosis 17
diseases (*see* syndromes)
doll's face 127
Down's syndrome 28, 154, 155
drug reaction 190
drug-influenced gingival lesions and diseases 41–53
Duhring-Brocq disease 232
dysplasia, chondroectodermal 145, 146

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

E

ecchymoses 201
Ehlers-Danlos syndrome 28, 149, 150
Eikenella corrodens 10
Ellis-van Creveld syndrome 145
endocrine disorders 273–277
Enterobacter cloacae 181
ephelis 81
epidermal necrolysis, toxic 221, 222
epidermis, sheet-like loss 222
epidermolysis bullosa
– acquisita 230–232
– hereditary 142–145
– – classification 142
epistaxis 61
epithelium
– junctional 5
– oral 5
Epstein-Barr virus 171
epulis, fibrous 297–299
erosion
– gingival 143, 235, 246, 251, 281
– lip 221
– tongue 221, 236
erythema (E.)
– gingival 168, 172, 180, 181, 201, 222, 228, 233, 235, 237, 239, 265, 281
– – diffuse 169, 170, 199, 237, 252, 255, 264, 267
– – irregular 246
– – linear 184, 185
– – localized 165, 172
– – migrans 33
– – multiforme 218–221
– – slight 226, 256
erythroleukoplakia 180
erythroplasia/erythroplakia 307, 308
Escherichia coli 181
Ewing's sarcoma 326, 327
examination, periodontal 13–17
– clinical examination 13–16
– patient history 13
– radiographic examiation 16, 17
– self-reported medical history 13
exostoses, multiple of the maxillary alveolar process 38, 39
eye, dryness 247

F

facial paralysis 259
FGF (fibroblast growth factor) 9
fibroma 313
– ossifying 316–318
fibromatosis, hereditary gingival 119, 120
fingernail, gingival trauma 65
fistula 59
– oroantral 60–62
– oronasal 62
– with pigmentation 60
focal dermal hypoplasia syndrome 147, 148
foreign body reaction 89–92
frenulum, multiple 146, 149
furcation involvment, examination 14, 15
Fusobacterium nucleatum 10

G

Gardner's syndrome 138, 139
genetic disorders 119–155
giant cell
– central lesion 303
– granuloma 301, 302
gingiva
– biochemistry 9, 10
– clinical features 3
– histology 5, 6
– innervation 8
– physiology 9
– vasculature 8
gingival crevicular fluid (GCF) 10
– increased flow 11
gingival enlargement
– calcium channel blockers 42–44
– ciclosporin 44–46
– leukaemia 207–209
– phenytoin 41, 42
gingivitis 155
– allergic 49, 90, 91
– candidiasis 110, 111
– chronic 260
– cystic fibrosis 153
– dental plaque induced 23, 24
– desquamative 220, 224, 228, 231, 235, 237, 239, 241, 252, 267
– geographic 33–35
– gonococcal 165
– granulomatous 31–33
– herpetic 183
– – primary 99–101
– – secondary 101, 102
– localized 126
– *Melkersson-Rosenthal's* syndrome 260
– mouthbreathing 30, 31
– necrotizing ulcerative (NUG) 93–95
– – acute (ANUG) 93–95
– – HIV 178
– neutropenia, familial 198
– papilloma 106, 147
– plasma cell (idiopathic circumficial plasmocytosis) 47–49
– pregnancy-associated 275
– *Sjögren's* syndrome 248
glycogen storage disease 1b 126, 127
glycosaminoglycans 9
gonococcal infection 164, 165
graft-versus-host disease 251–253
granuloma
– denture-induced 297–299
– eosinophilic 271
– giant cell 301, 302
– post-extraction 300
– pregnancy-associated 275
– pyogenic 299–301
granulomatosis, orofacial 259–265
graphite tattoo 85

H

haemangioma
– facial, unilateral 140
– gingival 141, 303, 304
– – unilateral 139
– leg 141
hand-foot-and-mouth disease 173, 174
Hand-Schüller-Christian disease 270, 272
heavy metal deposition 51–53
herpes zoster 169–171, 182, 183
Hexalen, contact gingivitis 90
HHV-3 168
HHV-4 171
histiocytosis, *Langerhans* cell 270–272
histoplasmosis 193, 194
HIV 177–190
– major groups 177
Hodgkin's disease 216, 217
hot liquid, mucosal burn 72
HPV (human papilloma virus) 103–106, 324
Hurler's syndrome 127, 128
hydrogen peroxide, mucosal burn 68
hydroxyurea 50
hypercementosis 8
hyperkeratosis syndrome, focal palmoplantar and oral mucosa 129, 130
hyperkeratosis
– gingiva 130
– palms / soles 124, 130
hyperpigmentation 51–53
hyperplasia, gingival 149
hypophosphatasia 125
hypoplasia syndrome, focal dermal 147, 148

I

immunology/immunological mechanism 11–13
infection
– bacterial
– – general 159–165
– – HIV 178–181
– – local 93–97
– fungal
– – HIV 184–187
– – local 109–112
– – systemic 187, 193–196
– viral
– – HIV 177–190
– – local 99–106
– – systemic 167–174

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

inflammation, gingival 274
inflammatory diseases 23–35
injury, self-inflicted 63–65
innervation, gingiva 8
interleukin
– 1 (IL-1) 12, 27
– 8 (IL-8) 12
– 10 (IL-10) 12
iodine, mucosal burn 69

J

jaw bone destruction 270, 271

K

Kaposi's sarcoma 187–189
– classic 330, 331
keratocyst 294, 295
keratoses 308–311
– frictional 310
– smoker's 310
Klebsiella pneumoniae 181
Klippel-Trenaunay-Weber syndrome 140–142

L

Laband syndrome 121, 122
Langerhans cell histiocytosis 270–272
lead 52
leiomyosarcoma 327
lesion
– advanced 11
– early 11
– established 11
– initial 11
Letterer-Siwe disease 270
leukaemia 206–209, 281
leukocytes, polymorphonuclear 11, 12
leukoplakia 308–311
– homogenous 309
– proliferative verrucous (PVL) 311
– speckled 310
– verrucous 310
lichen planus 236–239
lichenoid lesion, buccal mucosa 252
linear IgA disease 227–229
lip, erosion 221, 236
lipoma 313, 314
lump, gingival fibrous 298, 299
lupus erythematosus 245–247
Lyell's disease 221
lymphangioma, gingival 304, 305
lymphoma
– *Burkitt's* 213, 214
– cutaneous T-cell 214–216
– *non-Hodgkin's* 189, 190, 212, 213

M

macroglossia 269
macrophages 12
macule, melanotic 79, 80, 277
maculopapular lesion, gingiva 233
materia alba 91, 92
matrix proteins, extracellular 9
MEA syndrome 314
measles 167, 168
melanoma 331, 332
melanosis, smokers 85, 86
melanotic macule 79, 80, 277
Melkersson-Rosenthal syndrome 31, 259–261
metal tattoo 83–85
metastasis 329, 330
microbiology, periodontal disease 10, 11
monocyte chemoattractant protein-1 (MCP-1) 12
mononucleosis, infectious 171, 172
mouthwash, mucosal burn 68
mucolabial fold, disappearance 146
mucormycosis 194, 195
mucosal burn 68–70, 74
mucositis 73, 74
multiple myeloma 209–211
Mycobacterium (M.)
– *M. avium-intracellulare* 181
– *M. tuberculosis* 159
Mycoplasma pneumoniae 219
mycoses
– HIV 184–187
– local 109–112
– systemic 187, 193–196
myelodysplastic syndrome 204–206

N

naevus 80–82
– compound naevus 81
– naevus of *Ota* 81, 82
– white sponge 120, 121
nail dystrophy 144, 146
natural killer cells 12
Neisseria gonorhoeae 164, 165
neurofibroma 314–316
neurofibromatosis 314, 316
neutropenia
– cyclic 198, 199
– familial 197, 198
nifedipine, gingival swelling 43
Nikolsky's sign 227
nodules
– alveolar mucosa 152
– gingiva 135, 137
– tongue 152
non-Hodgkin's lymphoma 189, 190, 212, 213
NUG (necrotizing ulcerative gingivitis) 178
NUP (necrotizing ulcerative periodontitis) 95, 96, 178–180

O

oedema 165, 199, 256, 261, 262, 264, 265
orofaciodigital syndrome type I 148, 149
Osler-Rendu-Weber disease 151
osteoma, alveolar bone 138, 139
osteoradionecrosis 74, 75
osteosarcoma 325, 326
Ota, naevus 81, 82
overgrowth
– gingival 120, 122, 128, 141, 280
– papillary 132

P

Paget's disease 335, 336
palladium tattoo 84
palms
– hyperkeratosis 124
– target-like lesion 221
papilloma 105, 106, 147
papilloma virus, human (HPV) 103–106, 324
Papillon-Lefevre syndrome 28, 123, 124
papules 238
– gingival 133
– skin 134
paracoccidioidomycosis 195, 196
pathology 11–13
PCR (polymerase chain reaction) 10
PDGF (platelet-derived growth factor) 9
pemphigoid
– bullous 225–227
– cicatricial 223–225
– gestationis 229, 230
pemphigus
– paraneoplastic 280, 281
– vulgaris 234–236
pericoronitis 96, 97
periodontal ligament 7, 8
periodontitis
– accelerated (prepubertal) 28
– aggressive 27–29
– chronic 25–27
– early 274
– generalized 124, 126, 150
– localized juvenile 28
– necrotizing ulcerative (NUP) 95, 96, 178–180
periodontium, normal 4
PFAPA syndrome 113
phenytoin, gingival enlargement 41, 42
physical lesions 63–65
physiology, gingiva 9
pigmentation
– gingiva 277
– ocular 82
– physiological 77–79
– racial 78

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

pigmented disorders 77–86
pitted enamel 143
plaque 23, 25
plasminogen deficiency 203, 204
plasmocytoma, soft tissue 211, 212
plasmocytosis, idiopatic circumficial 47–49
polydactyly 146
porphyria 267, 268
Porphyromonas gingivalis 10, 12, 25, 27
Prevotella intermedia 10
probing, periodontal 14, 15
Pseudomonas aeruginosa 181
psoriasis 240, 241
pyostomatitis vegetans 265

R

radiation 74
– lesions 73–75
radiographic examiation 16, 17
recession, examination 14
Reiter's disease 255, 256

S

salivary gland tumour, mixed 319, 320
sanguinaria 50
sarcoidosis 31, 263, 264
sarcoma
– chondrosarcoma 328
– *Ewing's* sarcoma 326, 327
– *Kaposi's* sarcoma 187–189
– – classic 330, 331
– leiomyosarcoma 327
– osteosarcoma 325, 326
scarring, skin 144
schwannoma 315
scleral pigmentation 141
scrotum, round ulcer 254
scurvy 283, 284
sex steroid hormons 274–276
Sjögren's syndrome 247, 248
skin
– perioral lesion 215
– tags 132
smoker, melanosis 85, 86
soles, hyperkeratosis 124
stomatitis
– aphthous 114, 115
– chronic ulcerative 239, 240
– geographic 34
– herpes simplex 100–102
– nicotinic 308
Streptococcus (S.)
– *S. mitis* 10
– *S. sanguis* 10
Sturge-Weber angiomatosis 138–140, 303
swelling
– facial 259
– gingival
– – diffuse 260
– – generalized 262
– – leukaemia 207–209
– – localized 260, 262, 271
– – multiple myeloma 210
– – myelodysplastic syndrome 205
– – *non-Hodgkin's* lymphoma 213
– – plasmocytoma 211
– – red 269
– – scurvy 284
– labial 260
symblepharon 225
syndromes/diseases (names only)
– *Addison's* disease 276, 277
– *BehVet's* disease 253–255
– *Bourneville-Pringle* syndrome 136
– *Cowden's* disease 134, 135
– *Crohn's* disease 31, 261–263
– *Darier's* disease 133, 134
– *Down's* syndrome 28, 154, 155
– *Duhring-Brocq* disease 232
– *Ehlers-Danlos* syndrome 28, 149, 150
– *Ellis-van Creveld* syndrome 145
– *Gardner's* syndrome 138, 139
– *Hand-Schüller-Christian* disease 270, 272
– *Hodgkin's* disease 216, 217
– *Hurler's* syndrome 127, 128
– *Klippel-Trenaunay-Weber* syndrome 140–142
– *Laband* syndrome 121, 122
– *Letterer-Siwe* disease 270
– *Lyell's* disease 221
– *Melkersson-Rosenthal* syndrome 31, 259–261
– *Osler-Rendu-Weber* disease 151
– *Paget's* disease 335, 336
– *Papillon-Lefevre* syndrome 28, 123, 124
– *Reiter's* disease 255, 256
– *Sjögren's* syndrome 247, 248
– *Sturge-Weber* angiomatosis 138–140, 303
– *Turner's* syndrome 37
– *Vincent's* disease 93–95
– *von Recklinghausen's* disease 314
syphilis 161, 162, 180, 308
systemic sclerosis 249, 250

T

tattoo, metal 83–85
– amalgam 83, 84
– deliberate 90
– graphite 85
– palladium 84
T-cells 12
telangiectasia, hereditary hemorrhagic 151, 152
temporomandibular joint, examination 13
TGF (transforming growth factor) 9
thermal lesions 71, 72
thrombocytopenic purpura 202, 203
thumb nails, hypoplastic 122
tongue
– erosion 221, 236
– fissured 259
– macroglossia 269
– *Reiter's* disease 256
– *Sjögren's* syndrome 248
tooth
– caries 143
– mobility, classification 14–16
toothbrushing, traumatic 63, 64
torus mandibularis 37
trauma, gingival 63–65
trenchmouth 93–95
Treponema (T.)
– *T. denticola* 10
– *T. pallidum* 161
trichloracetic acid, mucosal burn 69
tuberculosis 159, 160, 180, 181
tuberosity, fibrous 38–40
tuberous sclerosis 136, 137
tumor, gingiva 214
Turner's syndrome 37

U

ulceration
– ANUG (necrotizing ulcerative gingivitis) 93–95
– aphtous 113–115, 253, 254
– aplastic anaemia 201
– *BehVet's* disease 254
– cutaneous T-cell lymphoma 215
– hand-foot-and-mouth disease 173, 174
– herpes zoster 170, 183
– histoplasmosis 193
– *Hodgkin's* disease 216, 217
– *Langerhans* cell histiocytosis 271, 272
– lupus erythematosus 246
– measles 168
– mucormycosis 195
– myelodysplastic syndrome 205
– *non-Hodgkin's* lymphoma 190, 213
– NUG (necrotizing ulcerative gingivitis) 178
– NUP (necrotizing ulcerative periodontitis) 95, 96, 178–180
– paracoccidioidomycosis 196
– plasminogen deficiency 204
– round, scrotum 254
– tuberculosis 160

For Personal Use Only
Library of School of Dentistry, TUMS

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

V

Varicella zoster virus 168, 169
vasculature, gingiva 8
Veillonella parvula 10
verruca vulgaris 103–105
vesicles
– gingiva 170, 231
– skin 174, 228, 230, 233
Vincent's disease 93–95
virus
– coxsackie virus 173
– cytomegalovirus (CMV) 182, 183
– Epstein-Barr virus 171
– herpes simplex (HSV) 99–102, 182
– HHV-3 168
– HHV-4 171
– human papilloma virus (HPV) 103–106, 324
– varicella zoster virus 168, 169
vitamin deficiency 283, 284
von Recklinghausen's disease 314

W

Whickham's striae 238
white lesion 240
white lines, gingiva 237

X

xerostomia 73, 247, 249

For Personal Use Only
Library of School of Dentistry, TUMS