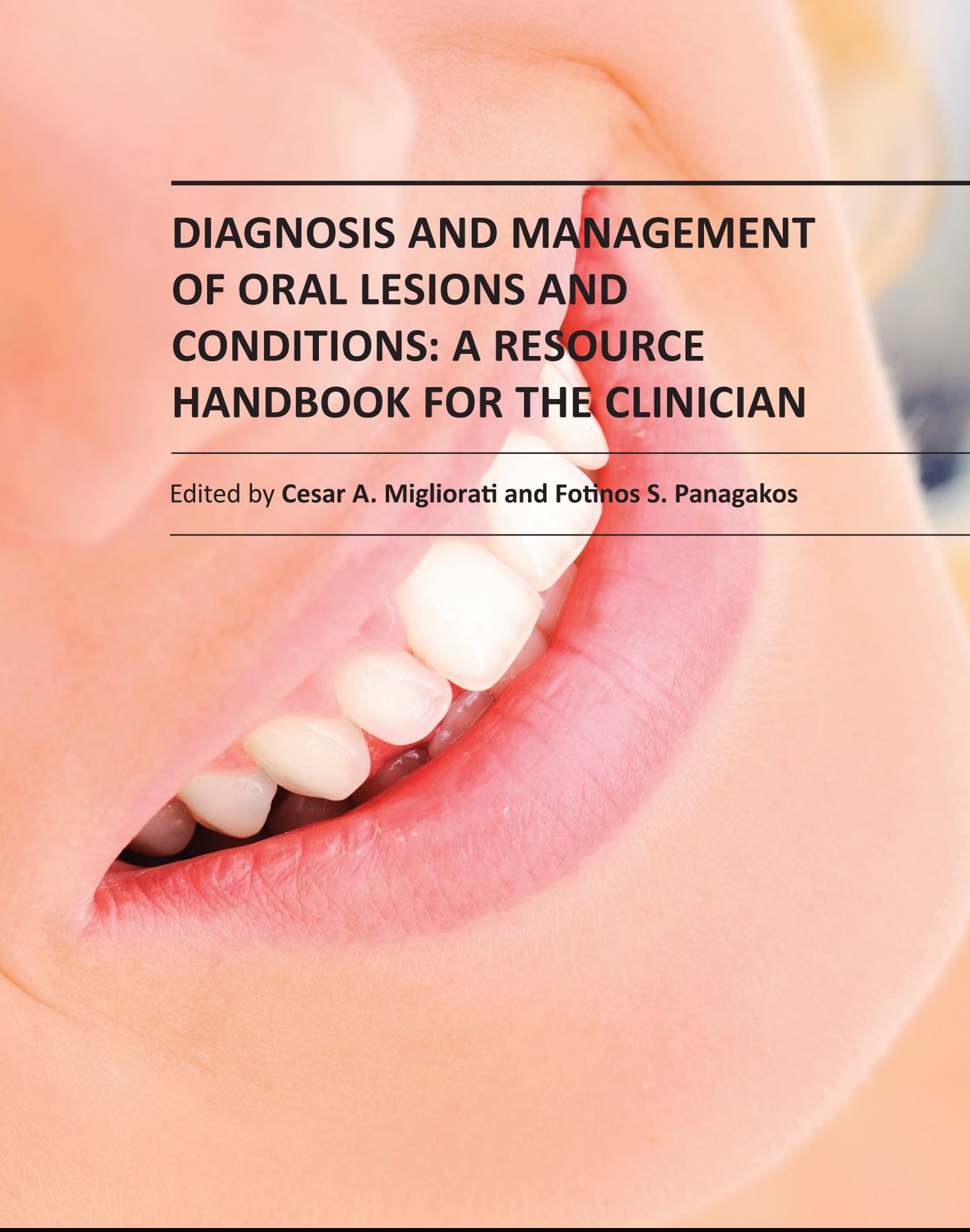

DIAGNOSIS AND MANAGEMENT OF ORAL LESIONS AND CONDITIONS: A RESOURCE HANDBOOK FOR THE CLINICIAN

Edited by **Cesar A. Migliorati and Fotinos S. Panagakos**

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Contents

The Goal

This handbook has the goal of providing a short and objective approach to the diagnosis and management of common oral lesions and conditions likely to be encountered in the daily practice of dentistry by the general practitioner. Each of the lesions/conditions will be grouped based on their nature, inflammatory or infectious, benign or malignant, variants of normal, bony lesions, etc. The individual lesion/condition will be described based on common clinical signs and symptoms, differential diagnosis, best approach for diagnostic confirmation, and brief management strategy. One of the chapters is dedicated to oral hygiene and oral health maintenance recommendations.

Dear Reader:

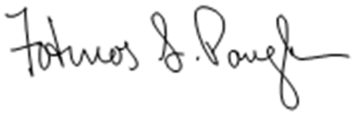
It is with great pride that we present the textbook *Diagnosis and Management of Oral Lesions and Conditions: A Resource Handbook for the Clinician.*

This book has been developed to serve as a resource for dental students, dental hygiene students, medical students, faculty members of dental schools, dental hygiene programs, and medical schools, and for practicing dental and medical professionals. The book is designed to provide the reader with an easy to use reference source regarding oral lesions and how to manage them.

I would like to express our deep appreciation to Dr. Cesar Migliorati and his faculty for their knowledge of this vitally important subject, and their commitment to the project that we are able to bring you this significant work.

Since the launch of its first toothpaste in 1873, the Colgate-Palmolive Company has been a world leader in oral care, both through cutting-edge therapeutics, as well as important educational services to the dental professions. *Diagnosis and Management of Oral Lesions and Conditions: A Resource Handbook for the Clinician* which has been produced and distributed through an educational grant from the company (by which the company provided funding to the publisher), is one shining example of our continuing commitment to ensuring the dental professions' education.

Sincerely,



Fotinos S. Panagakos, DMD, PhD
Global Director, Scientific Affairs

The views expressed in this textbook are those of the authors, not necessarily those of the Colgate-Palmolive Company.

Chapter 1

Normal Oral Cavity Findings and Variants of Normal

Foliate papillae/lingual tonsil

Lymphoid aggregates

Varicosities

Fordyce granules

Leukoedema

Exostosis

Torus Palatinus

Torus Mandibularis

Hairy Tongue

Fissure Tongue

Physiologic Pigmentation

1. Foliate papillae/lingual tonsil

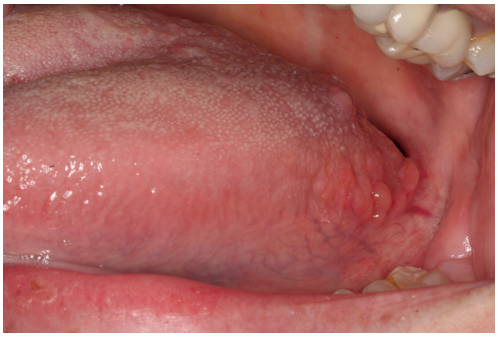
Description: The foliate papillae are vertical ridges of lingual papillae located on the posterior lateral surfaces of the tongue. At the base of the tongue are the lingual tonsils, which consist of normal lymphoid tissue. Similar to other lymphoid aggregates, the lingual tonsils may become hyperplastic or tender secondary to local inflammation or infection.

Etiology: The foliate papillae and lingual tonsils are normal anatomic structures

Treatment: No treatment is indicated

Prognosis: Occasionally, inclusion cysts known as oral lymphoepithelial cysts develop in association with foliate papillae and lingual tonsils. These, however, are benign and treated by simple surgical excision.

Differential Diagnosis: Squamous cell carcinoma.



2. Lymphoid aggregates

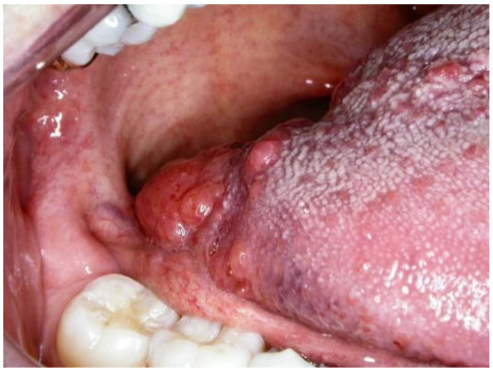
Description: Lymphoid aggregates are collections of normal or focally hyperplastic lymphoid tissue that may occur anywhere within the oral cavity, but most commonly involve the regions of Waldeyer's ring, which includes the oropharynx, lateral tongue, soft palate and floor of mouth.

Etiology: Oral lymphoid aggregates are relatively common and normal.

Treatment: No treatment is indicated, although biopsy is sometimes necessary to rule out other soft tissue lesions.

Prognosis: As with the lingual tonsils and other lymphoid tissue, oral lymphoid aggregates may become inflamed and tender upon local antigenic challenge. These are typically self-limiting or resolve after management of infection or inflammation.

Differential Diagnosis: Other benign lesions



3. Varicosities

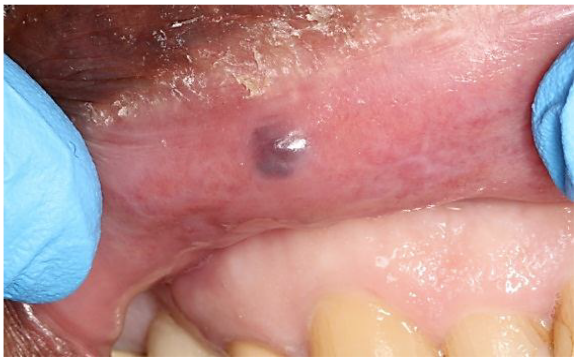
Description: Varicosities are abnormally dilated veins, which are commonly seen in the elderly. They may be seen in any location, but often involve the lips, buccal and labial mucosa and ventral tongue.

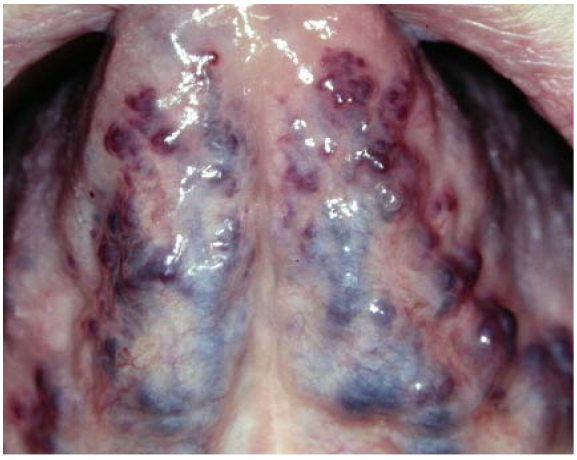
Etiology: Unknown, but may be associated with weakening of the vessel wall consequent to aging.

Treatment: No treatment is necessary except for esthetic reasons.

Prognosis: Occasional lesions may become thrombosed. Otherwise, the prognosis is good

Differential Diagnosis: Varicosities at lips may sometimes resemble mucoceles





4. Fordyce granules

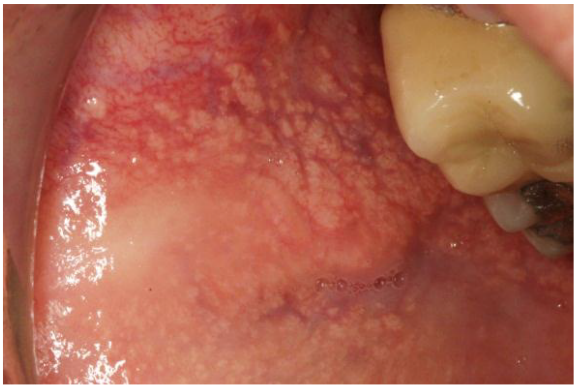
Description: Fordyce granules are one of the more common oral abnormalities. In fact, they may best be considered as a variation of normal. They represent ectopic lobules of sebaceous glands.

Etiology: Unknown

Treatment: No treatment is indicated, although biopsy is sometimes performed to rule out other pathology or for patient reassurance.

Prognosis: Excellent

Differential Diagnosis: None



5. Leukoedema

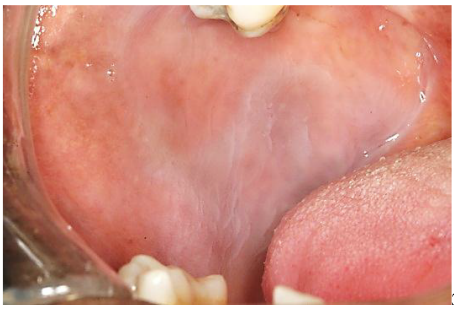
Description: Leukoedema is a benign mucosal abnormality characterized by thickened, edematous epithelium. The condition is recognized most often in African-Americans, although this may be a consequence of background pigmentation. While the condition most commonly involves the buccal mucosa, other sites such as the floor of mouth, lateral and ventral tongue and soft palate may be affected.

Etiology: Unknown

Treatment: The condition is benign and requires no treatment.

Prognosis: Excellent

Differential Diagnosis: While other mucosal alterations may be considered, eversion of the mucosa will result in disappearance of leukoedema and serve as a helpful diagnostic clue.



6. Exostosis

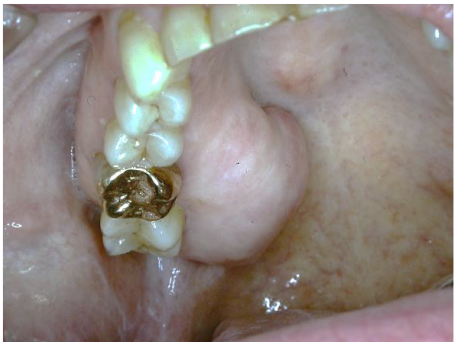
Description: Exostoses are benign projections of bone that arise from the cortex (most often buccal) of the maxillary or mandibular alveolus (While torus palatinus and torus mandibularis are considered to be a variant of exostoses, each is unique and will be presented separately). They are dynamic structures which may slowly change in size in response to stimuli, such as occlusal forces. Exostoses are frequently bilateral.

Etiology: Unknown, although genetic and local factors are believed to play a role in the development of exostoses.

Treatment: Treatment is often unnecessary, although may be indicated in preparation of removable prosthesis or when traumatized and inflamed. If treatment is indicated, surgical removal is curative.

Prognosis: Occasionally, the mucosa overlying exostoses becomes ulcerated and may lead to the development of osteomyelitis. Otherwise, the prognosis is excellent.

Differential Diagnosis: Osteomas, although these are neoplastic processes and show progressive enlargement.



7. Torus palatinus (palatal toru)

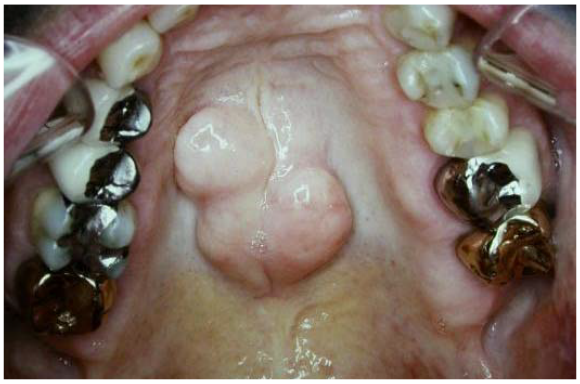
Description: The torus palatinus is a variant of the exostosis which develops from the cortical bone of the palatal vault. Like exostoses, they are dynamic structures that may slowly respond to external stimuli and may be quite variable in size and morphology. While some palatal tori present as single bony nodules others may appear nodular or lobular.

Etiology: Similar to exostoses

Treatment: Unnecessary unless characterized by chronic ulceration, osteomyelitis or in preparation for maxillary prosthesis.

Prognosis: Palatal tori are subjected to significant trauma, which may lead to chronic ulceration of the overlying mucosa. Subsequently, osteomyelitis is not uncommon. Otherwise, the prognosis is excellent.

Differential Diagnosis: Typically, the torus palatinus is unique enough to preclude a differential diagnosis.



8. Torus mandibularis (mandibular torus)

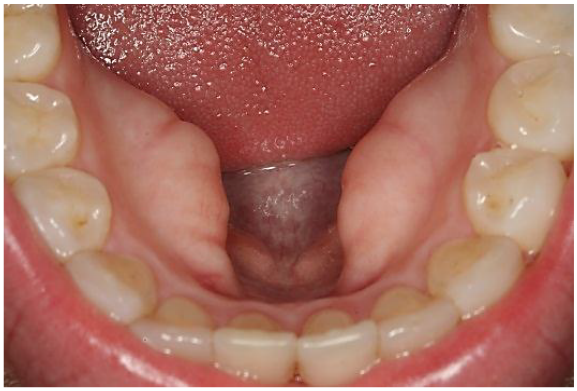
Description: Mandibular tori are variants of exostoses that occur, often bilaterally, along the lingual surface of the mandible and arise from the cortex. Like their palatal counterpart and exostoses, they are dynamic structures.

Etiology: Similar to exostoses

Treatment: Unnecessary unless characterized by chronic ulceration, osteomyelitis or in preparation for mandibular prosthesis.

Prognosis: Like palatal tori, the torus mandibularis is subjected to significant trauma, which may lead to chronic ulceration of the overlying mucosa and subsequent osteomyelitis. Otherwise, the prognosis is excellent.

Differential Diagnosis: Osteoma, although a bilateral presentation and lack of progressive enlargement is typically diagnostic for mandibular tori.



9. Hairy tongue

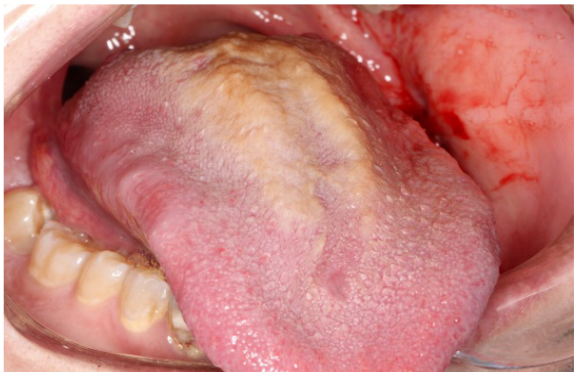
Description: Hairy tongue is a clinical term describing significant elongation of the filiform papillae. The elongation is due to an accumulation of keratin. The condition is often discolored from exogenous sources like coffee or tobacco, or from bacterial pigments. The condition is benign.

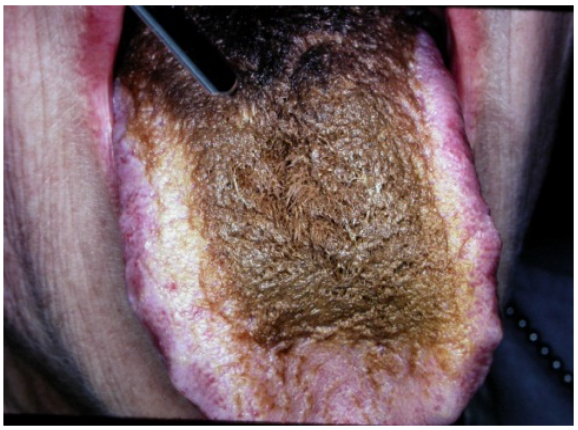
Etiology: Hairy tongue often arises in patients with poor oral hygeine in combination with the use of irritants such as hot beverages or smoking. A predominantly soft diet may also be a contributing factor.

Treatment: The condition will often improve by brushing the tongue or with the use of commercial "tongue scrapers." An inverted spoon may also be used to gently remove the superficial keratin.

Prognosis: The prognosis of hairy tongue is excellent, although it may be associated with a bad taste or halitosis.

Differential Diagnosis: There is no significant differential diagnosis to hairy tongue.





10. Fissured tongue

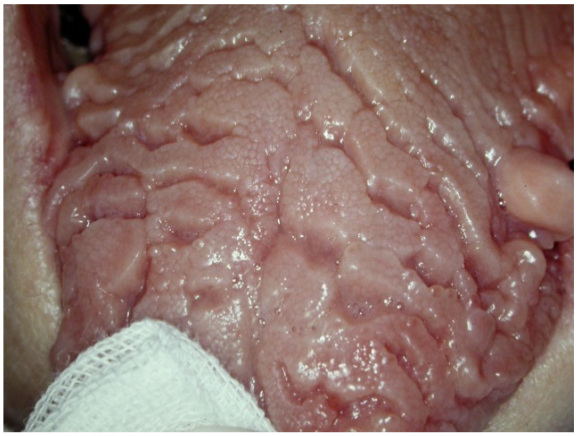
Description: Fissured tongue is a benign condition of the tongue, characterized by the presence of fissures and grooves along the dorsal surface. These may be variable in number, depth and orientation. The condition is seen more commonly in adults than in children. Interestingly, fissured tongue is often seen in combination with erythema migrans (geographic tongue), to be discussed in section 5.

Etiology: Unknown, but heredity seems to play a role in its development.

Treatment: No treatment is necessary for fissured tongue, although optimal hygeine should be encouraged to eliminate food or debris that may become trapped in the deeper grooves.

Prognosis: Excellent, although occasional patients may experience mild burning or irritation.

Differential Diagnosis: Fissured tongue is rather distinct in its clinical presentation and would not likely be mistaken for other entities.



11. Physiologic (racial/ethnic) pigmentation

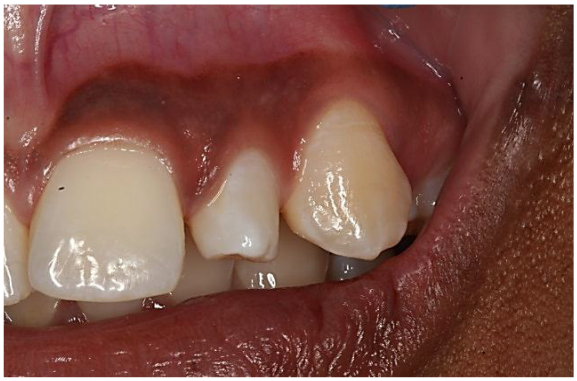
Description: Physiologic pigmentation is a benign melanosis of the oral mucosa seen in individuals of primarily African or African-American descent. The condition may occasionally be noted in Hispanic and other ethnic populations. The pigmentation is typically generalized and symmetrical, and does not show any abrupt change in size or coloration.

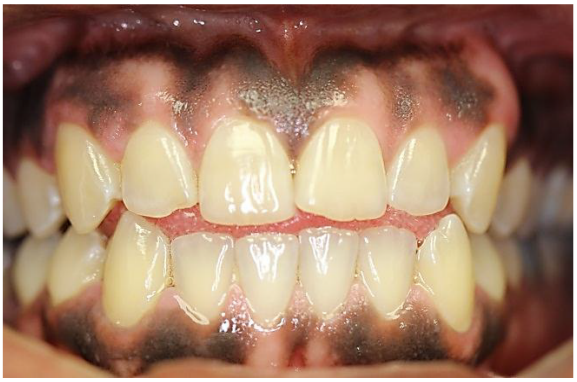
Etiology: The pigmentation is a normal physiologic process.

Treatment: None necessary, although ruling out any of a number of other sources of pigmentation may be necessary.

Prognosis: Excellent

Differential Diagnosis: Addison's disease, smoker's melanosis, drug-related pigmentation, intentional tattooing.





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Chapter 2

Common Benign Dental and Periodontal Lesions

Rampant caries

Periapical granuloma (chronic apical periodontitis)/periapical cyst (radicular cyst, apical periodontal cyst)

Pericoronitis

Localized aggressive periodontitis

Drug-related gingival hyperplasia

Parulis

Fluorosis

Methamphetamine abuse and its effect on oral health

1. Rampant caries

Description: Rampant caries is a particularly aggressive form of caries which presents as caries lesions on many, if not all teeth in a dentition, rapidly progressing lesions, and a relentless course seemingly resistant to traditional preventive methods. A clinical case with an alarming amount of amount of carious destruction, which occurred rather rapidly, or decay which is exceeding the pace of restorative treatment would be referred to as rampant. Different etiological factors present different disease patterns, but they typically affect tooth surfaces which would otherwise rarely be involved, such as facial surfaces of anterior teeth and interproximal surfaces of lower incisors. Rampant caries in preschool children involves pits and fissures, smooth surfaces, and anterior teeth to varying degrees, however the destruction is widespread and rapid.

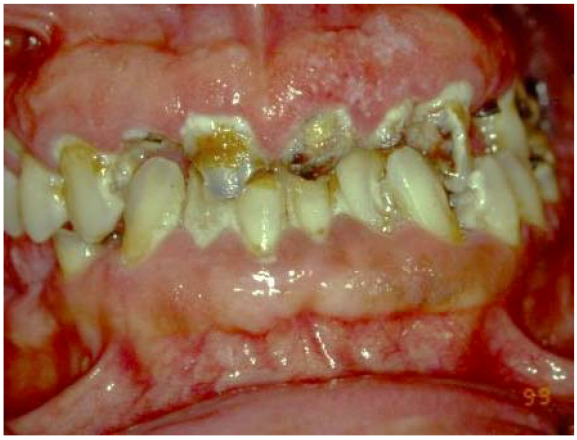
Etiology: Aggressive dental caries is multifactorial, involving inadequate salivary output, frequent consumption of carbohydrates, suboptimal fluoride exposure, previous history of caries activity, and infrequent or ineffective dental care. Inadequate saliva output can arise from systemic disease, disease treatments such as head and neck radiation, and as side effects of many medications, including over the counter, prescribed, and illicit drugs, as well as frequent consumption of caffeinated beverages. Medications with xerostomic adverse effect are additive when taken with others which also produce xerostomia.

Treatment: The treatment of rampant caries needs to be aggressive, employing both preventive and therapeutic strategies. Therapeutic treatment should be high intensity, such as caries

control, short term, and to a defined endpoint. Once the carious processes are under control, more definitive treatment can begin. The approach needs to be multi-pronged as described below. Diet modification is necessary to control rampant caries. Medical illnesses such as diabetes mellitus, depression, and bulimia nervosa must also be relatively well controlled. Providing a disease control program with particular attention to frequent reassessment, using fluoride releasing materials and promoting preventive and therapeutic control strategies not just of the dental disease but the systemic diseases and their treatments, and including diet modification. Patients with xerostomia should have specific dental care protocols and products.

Prognosis: Using a multi-pronged approach on a motivated patient can produce a very good outcome, if it includes many of the following: dietary modification, smoking cessation, low-intensity daily fluoride treatment, anti-bacterial salivary stimulants, periodic high intensity fluoride treatment, adjunctive agents such as chlorhexidine mouth rinse, fluoride containing restorative materials, dentifrices with calcium phosphate, frequent maintenance, and oral hygiene education and practice.

Differential diagnosis: Chronic caries of drug abuse, radiation caries



2. Periapical granuloma (chronic apical periodontitis)

Description: A periapical granuloma is a mass of inflamed granulation tissue at the apex of a nonvital tooth. The condition can be chronic or subacute and may be asymptomatic. Early in the disease there are no radiographic findings and the disease is termed acute apical periodontitis. As the disease process progresses, inflammatory cells activate resorption of surrounding bone causing a radiolucency around the tooth apex which may be large and well defined or ill-defined. Some granulomas may be associated with significant root resorption. Chronic lesions are often asymptomatic. Periapical granulomas may arise subsequent to a periapical abscess, showing discontinuous spurts of progression with periodic acute exacer-

bations. Symptoms include constant dull, throbbing pain. Vitality testing of the tooth reveals a negative response or a delayed positive response. There may be pain to biting or percussion but limited mobility. Symptoms may diminish as the disease becomes chronic.

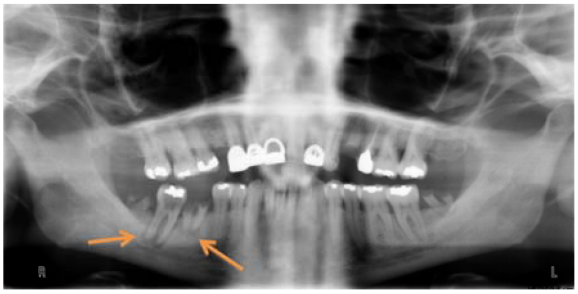
Periapical granulomas should be suspected in about 50% of teeth which have failed to respond to traditional root canal therapy. Most lesions are found during routine radiographic examinations. Granulomas can transform into cysts or abscesses and vice versa without significant radiographic change.

Etiology: These lesions result from the presence of bacteria or their toxic byproducts in the pulpal canal, the periapical tissues or both.

Treatment: Reduction of amount and control of the offending microorganisms is necessary for successful treatment. Nonrestorable teeth should be extracted and apical soft tissues curetted. Antibiotics are not necessary unless there are systemic symptoms or localized signs of swelling and infection.Treatment failure should be followed by endodontic retreatment or periapical surgery.

Prognosis: Lesions may fail to heal if there is persistent infected pulpal tissue, tooth fracture, leaking restorations, cyst formation, extraradicular infection, debris in the periapical area, untreated periodontal disease, apical scar, or fistula into the maxillary sinus.

Differential diagnosis: Periapical fibrous scar, periapical abscess, periapical cyst



3. Periapical cyst (radicular cyst, apical periodontal cyst)

Description: A periapical cyst is a true epithelium lined cyst at the apex of a nonvital tooth caused by inflammation of the periapical epithelium. The prevalence is about 15% of periapical lesions. The lumen of the cyst contains fluid and cellular debris. Most periapical cysts tend to grow slowly.

Cysts are usually asymptomatic unless there is an acute inflammatory exacerbation. The cysts may increase in size and cause swelling and sensitivity and mobility of adjacent teeth. The tooth involved with the cyst is usually nonvital. A loss of lamina dura around the root is seen radiographically with a rounded radiolucency encircling the root apex. Root resorption is

common. These cysts can expand to fill an entire quadrant. Periapical cysts frequently involve primary teeth.

Etiology

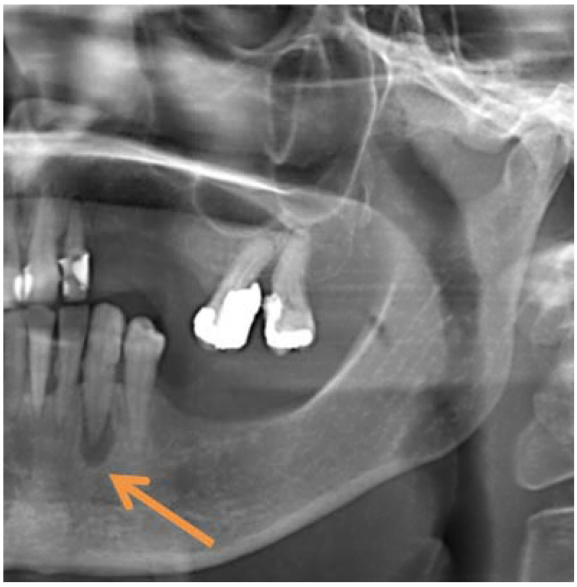
Lateral radicular cysts appear along the lateral aspect of the root and may be a result of inflammatory periodontal disease or pulpal necrosis, spread through accessory canals.Lateral radicular cysts are noted to present as discrete radiolucencies along the lateral tooth root. Loss of lamina dura may or may not be detected.

Vitality testing: Vitality of the tooth in question and adjacent teeth should be performed prior to surgical exploration. If the lesion is determined to be a periapical inflammatory lesion, extraction or conservative root canal therapy is performed. If the periapical radiolucency fails to resolve, then nonsurgical endodontic retreatment or periapical surgery can be performed. If no therapies have worked, biopsy is indicated.

Prognosis: Periapical inflammatory tissue which persists after extraction of the involved tooth may give rise to a residual periapical cyst. Spontaneous resolution can occur. Because myriad odontogenic and nonodontogenic cysts and tumors can appear similar to a residual periapical cyst, it is advised that these cysts should be excised surgically.

Inflammatory cysts do not recur after appropriate management. Fibrous scar formation is possible. Treatment is required for all persistent intrabony pathoses of unknown histopathology.

Differential diagnosis: Periapical granuloma, periapical abscess



4. Pericoronitis

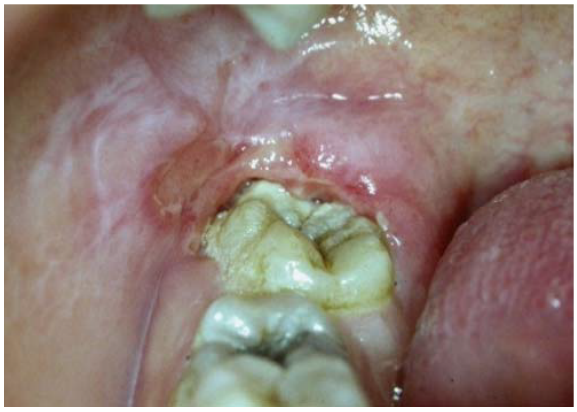
Description: Inflammation of a gingival flap of tissue surrounding the crown of a partially erupted tooth, most often seen with mandibular third molars. The tissue is often erythematous and edematous. Associated symptoms and signs include extreme pain, foul taste, inability to close the jaws due to swelling, fever, lymphadenopathy, malaise, and leukocytosis.

Etiology: Food debris and bacteria entrapped beneath the gingival flap covering a partially erupted tooth. Stress, upper respiratory infections, especially tonsillitis or pharyngitis, may predispose to the development of abscess.

Treatment: Acute symptoms may be managed with antiseptic lavage under the gingival flap followed by warm saltwater rinses at home. If systemic symptoms are present, systemic antibiotics can be used. Removal of the involved tooth or surgical removal of the gingival flap followed by elimination of food debris and bacteria will remove the source of the infection and inflammation. If the patient desires to keep the tooth, long-term maintenance with improved hygiene is indicated.

Prognosis: Good with removal of the involved tooth and appropriate debridement and hygiene.

Differential diagnosis: Periodontal abscess, necrotizing ulcerative periodontitis.



5. Localized aggressive periodontitis (localized juvenile periodontitis)

Description: Aggressive periodontitis appears to be associated with deficiencies in the immune response. The majority of patients have been shown to have demonstrable neutrophil

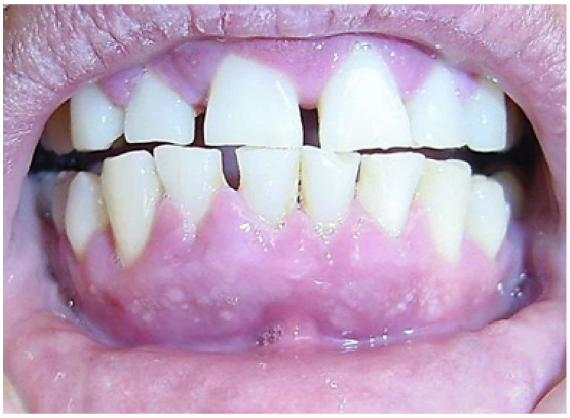
dysfunction without systemic effects. In the localized aggressive disease, there has been found a selective immune dysfunction and specific defect of bactericidal activity toward *A. actinomycetemcomitans*. There is some familial predisposition. The condition begins around the ages of 11 to 13 or circumpubertal. Attachment loss is primarily localized to first molars and incisors. Minimal supragingival plaque or calculus has been documented, however a strong serum antibody response to infecting agents has been found. Bone destruction and loss occurs three to five times faster than in chronic periodontitis. Bone loss patterns are often bilaterally symmetrical. Crestal bone surrounding the affected teeth is arc shaped. The affected teeth are commonly mobile and will migrate. If left untreated, the disease will progress to more generalized disease.

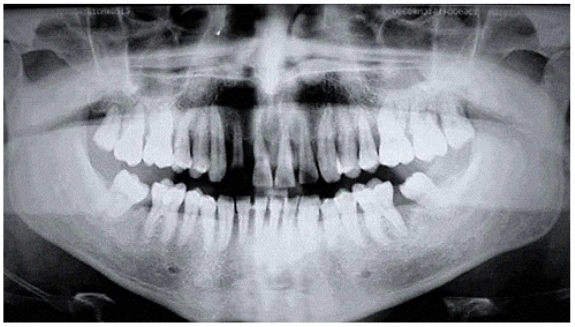
Etiology: There is no association with a systemic disease process; patients are generally otherwise healthy. Organisims commonly found at sites of aggressive periodontal disease include *Actinobacillus actinomycetemcomitans, Prevotella intermedia, Porphyromonas gingivalis* and others.

Treatment: Scaling and root planing alone will not control the aggressive disease. The use of antibiotics in combination with mechanical removal of subgingival plaque and inflamed periodontal tissues is necessary. Antibiotics such as tetracycline, augmentin, minocycline, and erythromycin have been used. Amoxicillin combined with metronidazole in high doses three times daily has been shown to be most effective. All sites should be cleaned at the same visit so that reinfection of previously cleaned areas is avoided. Periodontal surgery may be necessary for correction of residual pockets.

Prognosis: Long term follow up is mandatory, initially at one month, then at three month intervals. Refractory disease should receive additional courses of appropriate antiobiotics. Patients who smoke or present with advanced disease demonstrate worse prognosis.

Differential diagnosis: Generalized aggressive periodontitis, chronic periodontitis





6. Drug-related gingival hyperplasia (drug-related gingival overgrowth)

Description: Drug-related gingival hyperplasia is an increase in gingival size or overgrowth of the gingival tissues in response to particular medications or classes of medication. The gingival enlargement begins in the interdental papillae and spreads across tooth surfaces. Anterior teeth and buccal surfaces are most frequently involved. In advanced cases, the lesions can extend lingually and occlusally and interfere with eating and speech. Edentulous areas are infrequently affected; however hyperplasia can occur under unclean dentures and around implants. Children who use cyclosporine are at greater risk for gingival hyperplasia.

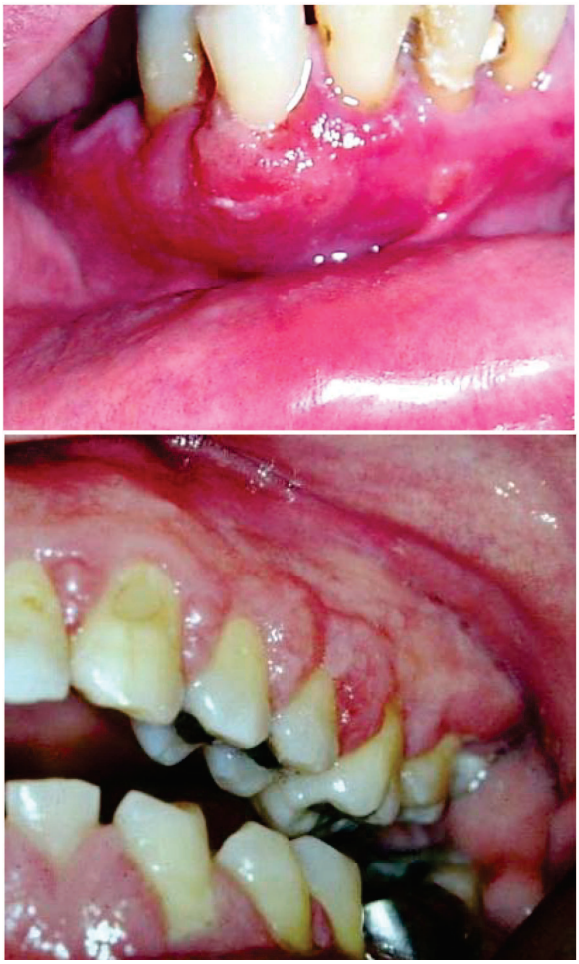
Tissues are generally normal in color and firm, and may be smooth, stippled, or granular in texture. Inflamed tissues may become erythematous and edematous and will bleed easily and become ulcerated and friable. Pyogenic granuloma-like enlargements may be seen in areas of greater inflammation.

Etiology: Hyperplasia is caused by an increase in extracellular matrix or collagen in the tissues due to interference by the drug with normal intracellular collagen degradation and remodeling. Cyclosporine, phenytoin, and nifedipine have a strong association with the condition. Other drugs have weak associations. There is an additive effect increasing the severity of the associated hyperplasia when cyclosporine and nifedipine are used concurrently. There may be an association with certain histocompatibility antigen types. The patient's level of oral hygiene and individual susceptibility are significantly related to the degree of enlargement as is the patient's smoking habit. Smokers have markedly more drug-related gingival hyperplasia.

Treatment: Rigorous oral hygiene results in noticeable clinical improvement. If the medication can be discontinued or another medication substituted, the overgrowth may cease and some regression occurs. Surgical therapy is recommended where aesthetics and function are compromised, followed by professional cleaning and frequent reevaluations and improved home care. Chemosurgical techniques, electrosurgery, or use of a carbon dioxide laser have also achieved satisfactory results. Chlorhexidine rinses can be beneficial in combination with improved home care. Systemic or topical folic acid or short courses of metronidazole or azithromycin have also shown benefit.

Prognosis: The gingival overgrowth leads to increased probing depths and some attachment loss. Recurrence after surgical management is not uncommon and can occur in as little as three months, particularly in cases of poor dental hygiene.

Differential diagnosis: Gingival fibromatosis, pyogenic granuloma.



Drug-related gingival overgrowth – Calcium channel blocker

7. Parulis

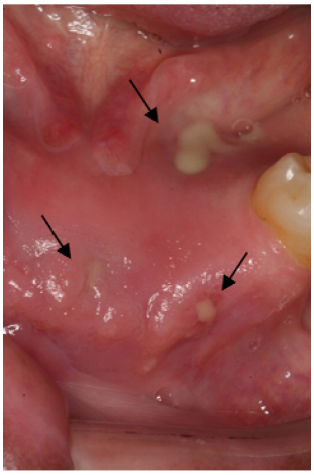
Description: A small erythematous mass of subacutely inflamed granulation tissue, which appears at the intraoral opening of a sinus tract from a periapical abscess. The lesion may be asymptomatic if the infection is chronic and a path of drainage is achieved.

Etiology: This lesion is caused by a dental-related abscess, which has perforated alveolar bone and formed a path of drainage.

Treatment: Elimination of the focus of infection through incisional drainage, root canal therapy, or tooth extraction. Pain may be controlled with NSAIDs. Medically compromised patients may warrant antibiotic therapy if systemic signs or symptoms are present. Persistent lesions may require surgical removal with curettage of the infected sinus tract.

Prognosis: Lesions typically resolve with appropriate treatment of the infection.

Differential diagnosis: Fibroma



8. Fluorosis

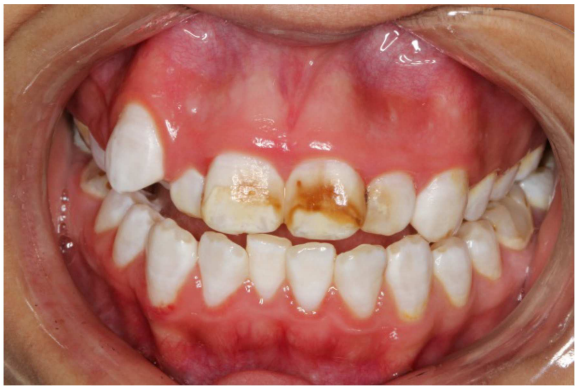
Description: Fluorosis is a condition of hypomineralized enamel, caused by ingestion of excess amounts of fluoride as teeth are developing. Permanent hypomaturation occurs in the developing enamel resulting in increased surface and subsurface porosity and a permanent staining of the teeth. Enamel may appear white and chalky, lusterless, opaque, with zones of yellow to dark brown discoloration. Uncommonly, deep, irregular, brownish pits may represent enamel hypoplasia.The condition appears in a bilaterally symmetrical distribution. The teeth are generally caries resistant.

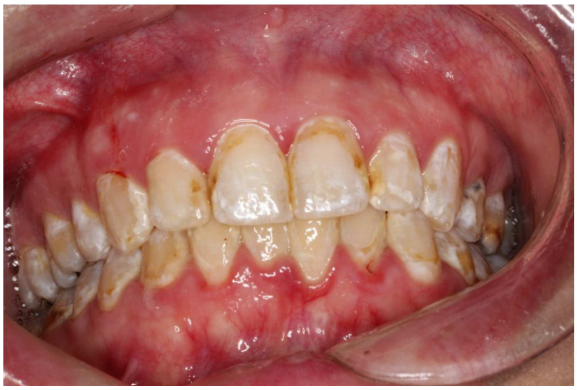
Etiology: Higher than optimal intakes of fluoride during critical periods of tooth development.

Treatment: Mild cases may be treated with surface microabrasion. More severe cases may be treated with composite resin restorations, porcelain veneers, or full coverage porcelain crowns.

Prognosis: Only occurs as tooth enamel is developing. Responds well to cosmetic dentistry procedures.

Differential diagnosis: Molar and incisor hypomineralization due to prolonged antibiotic administration in infancy; syphilitic hypoplasia





9. Methamphetamine abuse and its effect on oral health

Description: Originally developed in the United States to treat narcolepsy and ADHD, it became increasingly used for non-medical indications, such as increased alertness, weight control, and to combat depression.

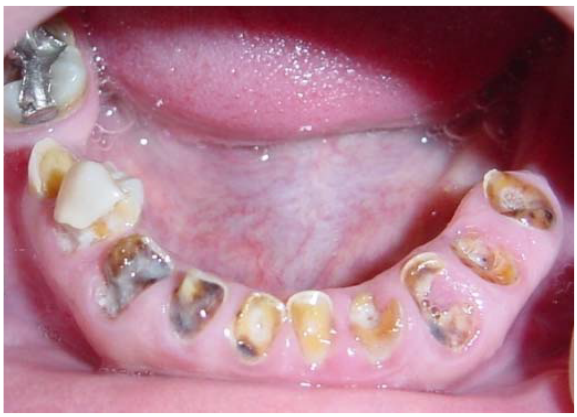
Etiology: Meth users abuse the drug because they feel it gives them greater energy, increased physical ability, and a state of euphoria. Methamphetamine can be smoked, snorted, injected, or taken orally. The majority of users are men between the ages of 19 and 40. The effects of the drug can last up to 12 hours. Drug effects include insomnia, aggressiveness, agitation, hyperactivity, decreased appetite, tachycardia, tachypnea, hypertension, hyperthermia, vomiting, tremors, xerostomia, psychological addiction, violent behavior, anxiety and confusion, depression, paranoia, hallucinations, mood changes, skin lesions, and effects on most organ systems. Delusions of parasites in the skin cause the patient to pick at the skin causing traumatic injury. Rampant dental decay is a common manifestation, which begins on the facial and interproximal smooth surfaces and progress to involvement of all tooth surfaces. Eventually all tooth crowns are destroyed. Poor oral hygiene and extreme drug related xerostomia, resulting in excess consumption of acidic sugar filled soft drinks and refined carbohydrates exacerbate the condition.

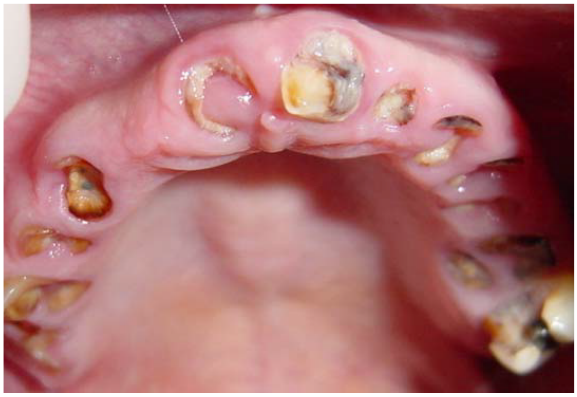
Treatment: Thorough medical and dental history taking is imperitive. Warning signs would include a patient who is emaciated, agitated, and nervous, who exhibits tachycardia, tachypnea, hypertension, hyperthermia, and rampant smooth surface caries. Using local anesthetics containing epinephrine or levonordephrine on these patients can lead to hypertensive crisis, stroke, or myocardial infarction in patients who have recently used meth.Patients should be urged to discontinue acidic, carbonated, caffeinated drinks, tobacco and alcohol.

Medical consultation or referral to a substance abuse center is advised. Dental breakdown will progress rapidly if the abuse continues, sometimes necessitating total odontectomy and denture fabrication. If some teeth are still salvageable, topical fluoride and frequent dental maintenance will benefit if the patient is motivated to stop the drug and improve home care.

Prognosis: Prognosis remains poor while the drug is continued. Many patients will lose most or all of their teeth at young ages.

Differential diagnosis: Rampant caries, xerostomia, diabetes related caries.





Additional reading

Rampant caries

Hildebrandt, G, Larson, TD. Management of Rampant Caries. Journal of the Minnesota Dental Association: Northwest Dentistry. Jan-Feb 2009; 88(1):35-45.51.

Periapical granuloma (chronic apical periodontitis)

Neville, BW, Damm, DD, Allen, CM, Bouguot, JE. Oral and Macillofacial Pathology, third ed. W.B. Saunders Co., Philadelphia. (2009). Pp. 127-130.

Periapical cyst (radicular cyst, apical periodontal cyst)

Neville, BW, Damm, DD, Allen, CM, Bouguot, JE. Oral and Macillofacial Pathology, third ed. W.B. Saunders Co., Philadelphia. (2009). Pp. 130-135.

Pericoronitis

Neville, BW, Damm, DD, Allen, CM, Bouguot, JE. Oral and Macillofacial Pathology, third ed. W.B. Saunders Co., Philadelphia. (2009). Pp. 171-173.

Localized aggressive periodontitis

Neville, BW, Damm, DD, Allen, CM, Bouguot, JE. Oral and Macillofacial Pathology, third ed. W.B. Saunders Co., Philadelphia. (2009). Pp. 173-176.

Drug-related gingival hyperplasia (Drug-related gingival overgrowth)

Neville, BW, Damm, DD, Allen, CM, Bouguot, JE. Oral and Macillofacial Pathology, third ed. W.B. Saunders Co., Philadelphia. (2009). pp. 163-166.

Parulis

Neville, BW, Damm, DD, Allen, CM, Bouguot, JE. Oral and Macillofacial Pathology, third ed. W.B. Saunders Co., Philadelphia. (2009). Pp. 136-137.

Fluorosis

Neville, BW, Damm, DD, Allen, CM, Bouguot, JE. Oral and Macillofacial Pathology, third ed. W.B. Saunders Co., Philadelphia. (2009). pp. 58-60.

Methamphetamine abuse and its effect on oral health

Neville, BW, Damm, DD, Allen, CM, Bouguot, JE. Oral and Macillofacial Pathology, third ed. W.B. Saunders Co., Philadelphia. (2009). Pp. 303.

Chapter 3

Bony Lesions

Idiopathic osteosclerosis
Condensing Osteitis
Nasopalatine duct cyst
Lateral periodontal cyst
Dentigerous cyst
Odontoma (complex/compound)
Stafne defect
Cemento-osseous dysplasia (Focal/florid)
Odontogenic keratocyst (keratocystic odontogenic tumor)
Ameloblastoma

1. Idiopathic osteosclerosis (bone island, bone scar, focal periapical osteopetrosis, enostosis)

Description: Idiopathic osteosclerosis (IO) is an intraosseous growth of non-inflammatory trabecular bone.

Etiology: Unknown origin, but may be considered as a normal developmental anatomic bone variation.

Prevalence: Greater prevalence among Chinese and Blacks than Caucasians. No difference in frequency between males and females.

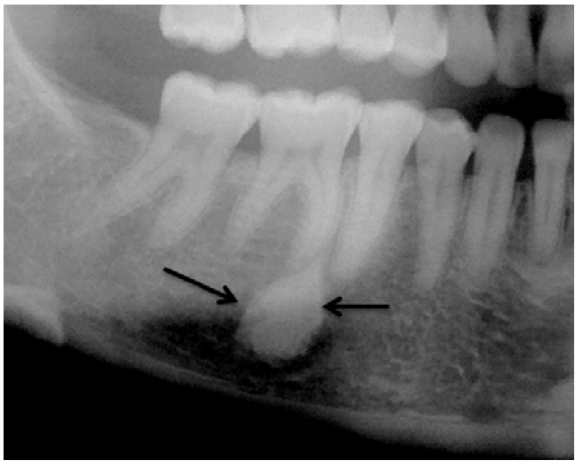
Location: More often in the posterior mandible rather than the maxilla. Most of the lesions are associated with root apices.

Signs and symptoms: Asymptomatic and diagnosed as an incidental finding on a radiograph.

Radiographic findings: In general, IO appears as a round, elliptical or irregular area of increased bone density. Usually appears as a well-defined homogeneous radiopacity that blends in with the surrounding bone.

Treatment: Treatment is neither indicated nor necessary.

Differential diagnosis: Condensing osteitis.



Idiopathic osteosclerosis in close proximity to the mesial root of mandibular first molar.

2. Condensing osteitis (chronic focal sclerosing osteomyelitis, sclerosing osteitis, bone eburnation)

Description: Localized pathologic growth of maxillomandibular bones with mild clinical symptoms.

Etiology: Bone-appositioning inflammatory processes from infection of periapical tissues by organisms of low virulence.

Prevalence: Greater prevalence among children and young adults. No difference in frequency between males and females.

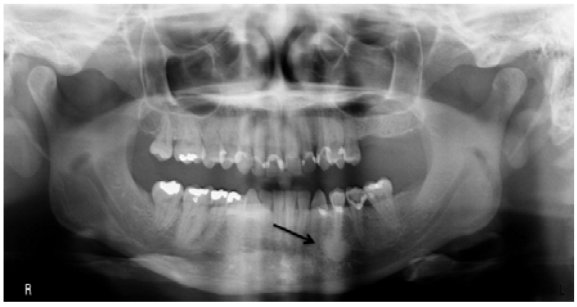
Location: More often in the posterior mandible. Most of lesions surrounds the apices of teeth with pulpitis or pulpal necrosis.

Signs and symptoms: Usually asymptomatic, but the associated low-grade chronic pulpal infection may produce mild symptoms.

Radiographic findings: Uniform dense periapical radiopacity with ill-defined sclerotic margins and large transition to the surrounding bone. The lesion is usually non-expansile and is associated with a carious tooth, which exhibits loss of lamina dura and widening of the periodontal ligament space. The size of CO may vary from 1 mm to 22 mm with mean width and height of 5 mm, and as to shape, it may vary from round to irregular.

Treatment: In symptomatic cases, endodontic therapy or extraction are the choices depending on the particular tooth condition. Asymptomatic cases without obvious caries should be followed with periodic x-ray examination.

Differential diagnosis: Idiopathic osteosclerosis, cementoblastoma.



Condensing osteitis associated with left mandibular premolar with large cervical restoration.

3. Nasopalatine duct cyst (median anterior maxillary cyst, incisive canal cyst)

Description: Developmental non-odontogenic cyst with intraosseous and extraosseous variants.

Etiology: Unknown origin, but this lesion develops from epithelial remnants of the nasopalatine duct contents.

Prevalence: May occur in any age, but is rare in the first decade of life. More frequent in males.

Location: Midline of the anterior palate.

Signs and symptoms: Palatal swelling, anterior tooth displacement, sublabial swelling, low-grade pain, transient salty taste. Adjacent teeth are vital.

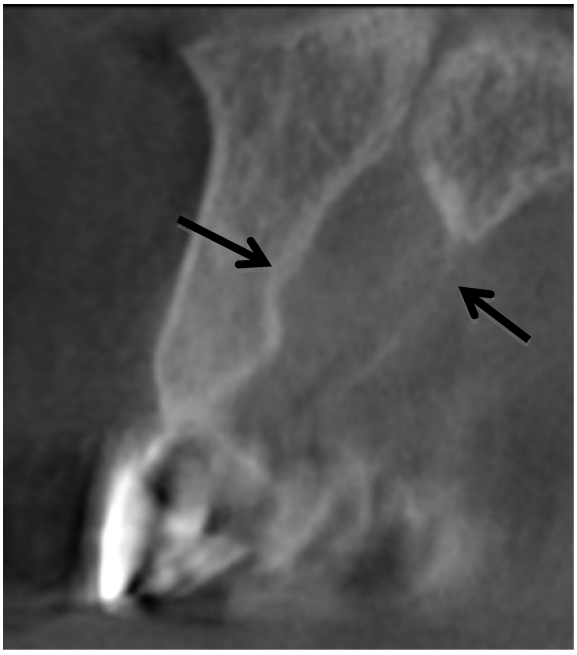
Radiographic findings: Usually intraosseous lesions present as round or ovoid well-defined radiolucency with unique appearance of a "heart shape" caused by the superimposition of the anterior nasal spine over the superior portion of the lesion. Central incisors may be displaced, but radicular resorption is rare. In large lesions, expansion and thinning of the palatal and buccal cortices may be present.

Extraosseus lesions do not present abnormal radiographic findings, because they are entirely within soft tissue.

Prognosis: Good. This cyst rarely recurs.

Treatment: Surgical enucleation for small lesions. Marsupilaization for larger cysts.

Differential diagnosis: Large nasopalatine duct, radicular cyst or granuloma, central giant cell granuloma.



Nasopalatine duct cyts. Note expansion of the incisive canal with soft tissue extension.

4. Lateral inflammatory periodontal cyst (inflammatory periodontal cyst)

Description: Intrabony cystic lesion.

Etiology: This lesion arises from epithelial rests in the lateral periodontium of the root.

Prevalence: Usually identified from second to the ninth decades of life. No gender predilection.

Location: Higher incidence in the mandibular lateral incisor to second premolar and between maxillary lateral incisor and canine.

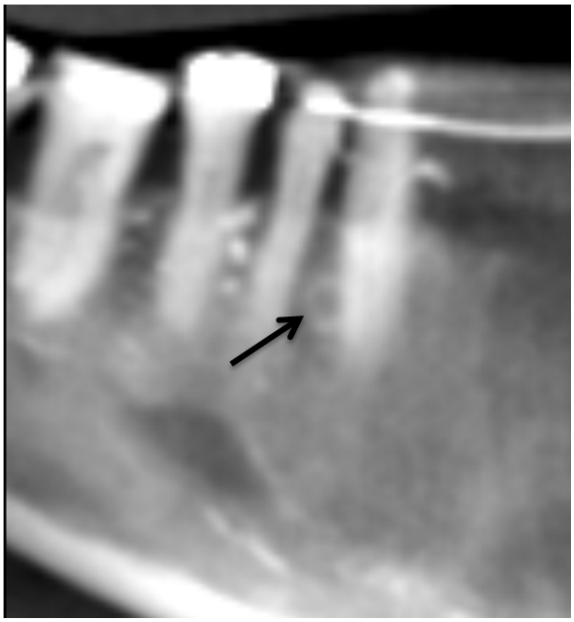
Signs and symptoms: Asymptomatic unless secondary infected.

Radiographic findings: Well-defined round or oval shape radiolucency with sclerotic borders. Usually unilocular with less than 1 cm in diameter. Larger and multilocular lesions have been reported (botryoid odontogenic cysts). In small lesions, associated lamina dura may be absent. In large lesions, cortical expansion and dental displacement may be present.

Treatment: Excisional biopsy or enucleation.

Prognosis: Good. These cysts do not tend to recur.

Differential diagnosis: In small lesions, keratocystic odontogenic tumor, neurofibroma, radicular cyst or mental foramen.



Lateral inflammatory periodontal cyst associated with the distal aspect of mandibular canine. (Image courtesy of Dr. Marcel Noujeim, UTHSCSA).

5. Dentigerous cyst (follicular cyst, eruption cyst)

Description: Pericoronal cystic lesion associated with unerupted dentition. It is classified in eruption, circumferential or lateral dentigerous cysts according to its relationship with an unerupted or supernumerary tooth.

Etiology: Originates from hemodynamic polling of fluid beneath the enamel epithelium or between the epithelium and the crown of an unerupted tooth.

Prevalence: After radicular cysts, dentigerous cysts are the second most common type of cyst in the jaws. Usually, these cysts are identified in patients younger than 20 years of age.

Location: Above the crown, attached to the cemento-enamel junction. Higher occurrence associated with maxillary and mandibular third molars and maxillary canines.

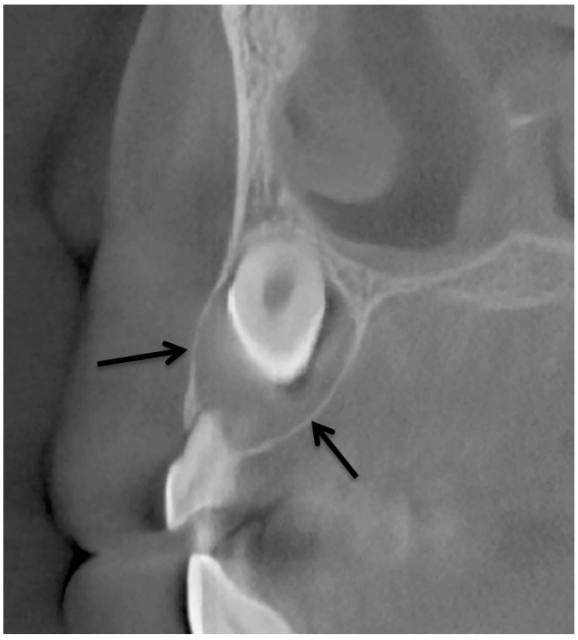
Signs and symptoms: Typically no pain or discomfort. Missing tooth or teeth with hard swelling and facial asymmetry.

Radiographic findings: Typically well-defined unilocular radiolucency with sclerotic outline unless secondarily infected. Radicular resorption and cortical expansion may be present.

Treatment: Enucleation with extraction of associated tooth.

Prognosis: Excellent.

Differential diagnosis: Keratocystic odontogenic tumor, ameloblastoma.



Dentigerous cyst associated with impacted maxillary canine. Note buccal and lingual cortical expansion, relationship with the CEJ of the teeth of origin and radicular resorption of the primary tooth. (Image courtesy of Dr. Marcel Noujeim, UTHSCSA).

6. Odontoma (complex/compound)

Description: Hamartomatous or benign mixed odontogenic tumor. Two forms classified as complex (gross mixture of dental tissues) or compound (multiple tooth-like structures).

Etiology: Unknown.

Prevalence: Usually identified in young patients in the first or second decades of life.

Location: Within the alveolar region of the jaws. Complex form most commonly noted in the mandibular posterior area. Compound form most commonly noted in the maxillary anterior area.

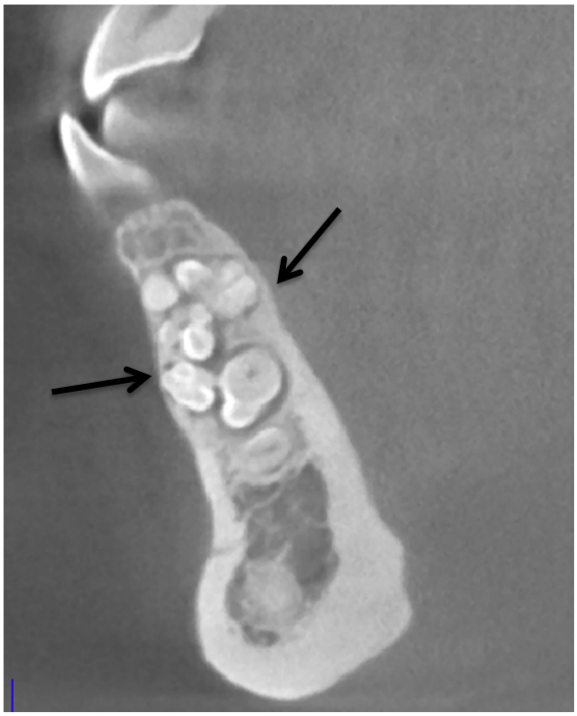
Signs and symptoms: Usually asymptomatic, but pain and swelling have been reported.

Radiographic findings: Well-defined, mixed radiolucent and radiopaque lesion. Jaw expansion may be present with large lesions.

Treatment: Conservative excision/curettage.

Prognosis: Excellent.

Differential diagnosis: Focal sclerosing osteitis, osteoma, ameloblastic fibro-odontoma, adenomatoid odontogenic tumor, calcifying odontogenic tumor.



Odontoma (compound) in the anterior mandible. Note multiple tooth-like structures and slight cortical expansion characteristic of this lesion.

7. Stafne defect (salivary gland depression, lingual bone defect, static bone cyst)

Description: Bone concavity in the lingual surface of the mandible with intact buccal cortex filled with salivary gland or adipose tissue

Etiology: Unknown origin, but it is considered a developmental defect.

Prevalence: Generally detected in patients in the 5th and 6th decade of life.

Location: Usually in the posterior mandible within the submandibular fossa, between the mandibular canal and inferior border of the mandible. Anterior variant is commonly found near the apex of the bicuspids.

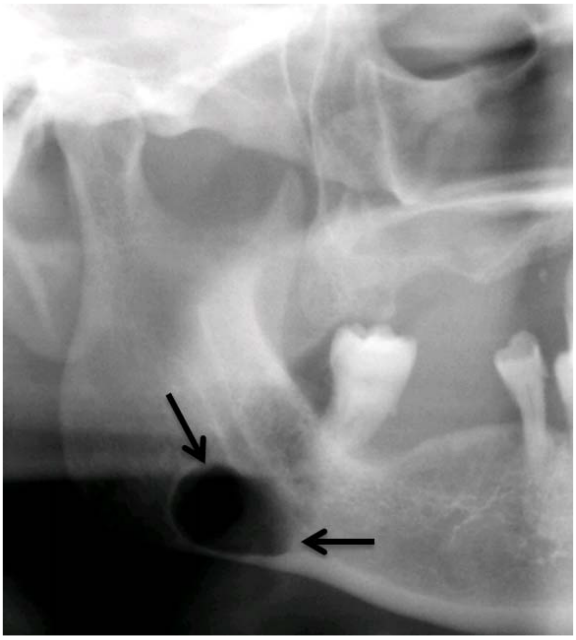
Signs and symptoms: Asymptomatic. Usually discovered incidentally.

Radiographic findings: Usually well-defined round or ovoid radiolucency of variable width with hypercorticated margins.

Treatment: Treatment is neither indicated nor necessary.

Prognosis: Excellent.

Differential diagnosis: Variants in anterior mandible may present similar radiographic features of odontogenic lesions, such as cysts.



Stafne defect in the right posterior mandible. Note location below the mandibular canal and image features similar to a odontogenic cyst.

8. Cemento-osseous dysplasia (periapical/florid)

Description: Gradual replacement of normal cancellous bone by fibrous tissue, abnormal bone and cementum. Periapical COD (PCOD) classifies alterations located in the anterior region. Florid COD (FCOD) classifies to more extensive conditions involving more than 1 quadrant.

Etiology: Unknown origin, but it is considered as a reactive or dysplastic process.

Prevalence: Higher in middle-aged women. More often in Blacks than Whites. Frequently seen in Asians.

Location: Usually confined to the tooth-bearing areas of the jaws or to edentulous alveolar processes. Usually in the apex of mandibular dentition.

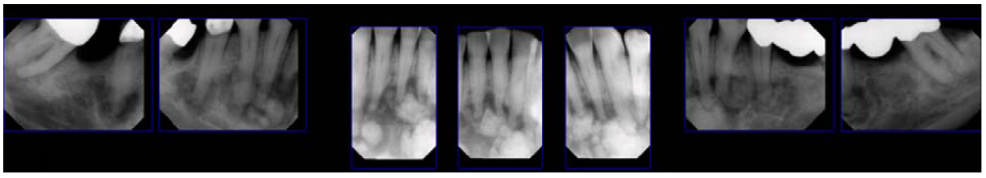
Signs and symptoms: Typically asymptomatic but may be associated with pain, swelling, dental sensitivity and mobility.

Radiographic findings: Radiolucent in early stages, mixed (radiolucent-radiopaque) and radiopaque in late stages. Usually radiolucencies show radiopaque sclerotic bone margins with variable width. Cortical expansion may be present in large lesions.

Treatment: Usually, no treatment is necessary unless patient states symptoms (e.g. pain or discomfort) associated with atrophy of alveolar ridge and exposure of cementum. Infection should be consider.

Prognosis: Good, however osteomyelitis may occur if secondarily infected.

Differential diagnosis: Vary with the stage of development of the lesion. Radiolucent stage: radicular granuloma or cyst. Mixed stage: chronic sclerosing osteomyelitis. Radiopaque stage: odontoma, cementoblastoma.



Cemento-osseous dysplasia (florid form).

9. Keratocystic odontogenic tumor (odontogenic keratocyst)

Description: Odontogenic benign and aggressive cystic neoplasm. Nevoid basal cell carcinoma syndrome should be considered in cases of multiple lesions.

Etiology: Developmental tumor.

Prevalence: Predilection for White males. Peaks in the second and third decades of life.

Location: Most common in the posterior body and ramus of the mandible.

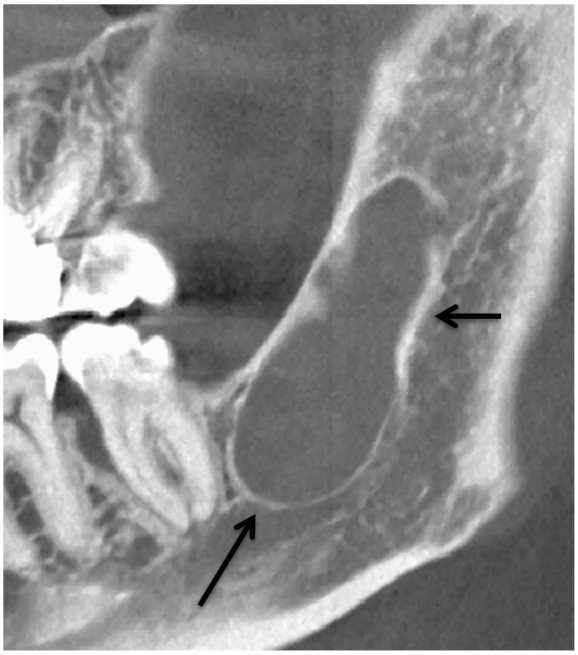
Signs and symptoms: Usually asymptomatic. Pain, swelling and drainage due to secondary infection.

Radiographic findings: Usually scalloped multilocular radiolucency with sclerotic borders. Minimal cortical expansion in the body of the mandible. Considerable expansion in the maxilla or ramus and coronoid process of the mandible.

Treatment: Managed in a case-by-case basis. Excision with bony curettage. No recurrence is reported after resection; however the invasive nature of the procedure should be taken into consideration.

Prognosis: Variable. 1 to 56% of recurrence rate depending on the treatment. Long-term follow-up is recommended.

Differential diagnosis: Dentigerous cyst, ameloblastoma, simple bone cyst, odontogenic myxoma, central giant cell granuloma, central mucoepidermoid carcinoma.



Kerotocystic odontogenic tumor in the posterior mandible extending into the ramus. Note sclerotic borders and displacement of the mandibular canal.

10. Simple bone cyst (traumatic bone cyst, solitary bone cyst)

Description: Cavity in the jaws without epithelial lining. It may be empty or filled with small amount of fluid. May be associated with cemento-osseous dysplasia.

Etiology: Unknown in most cases. Questionable trauma relationship.

Prevalence: Male predilection. Most lesions occur in the first and second decades of life. When associated with cemento-osseous dysplasia, simple bone cysts tend to occur in the fourth decade of life and in female patients.

Location: Mostly found in the ramus or posterior mandible.

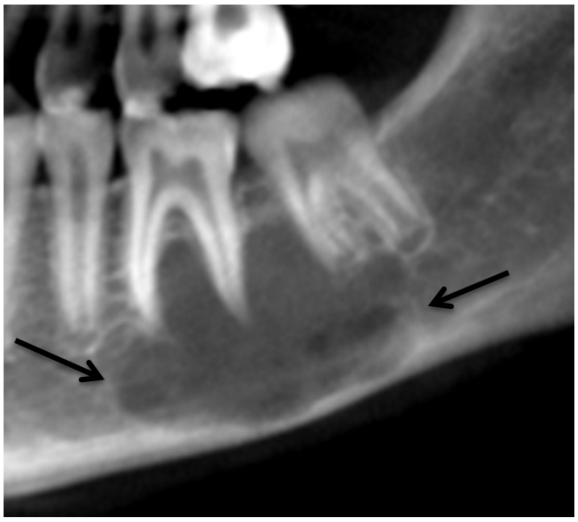
Signs and symptoms: Usually asymptomatic. Empty bony space. Pain and tenderness associated with secondarily infected lesions. Swelling may be observed.

Radiographic findings: Well-defined and scalloped radiolucency extending between the dental roots.

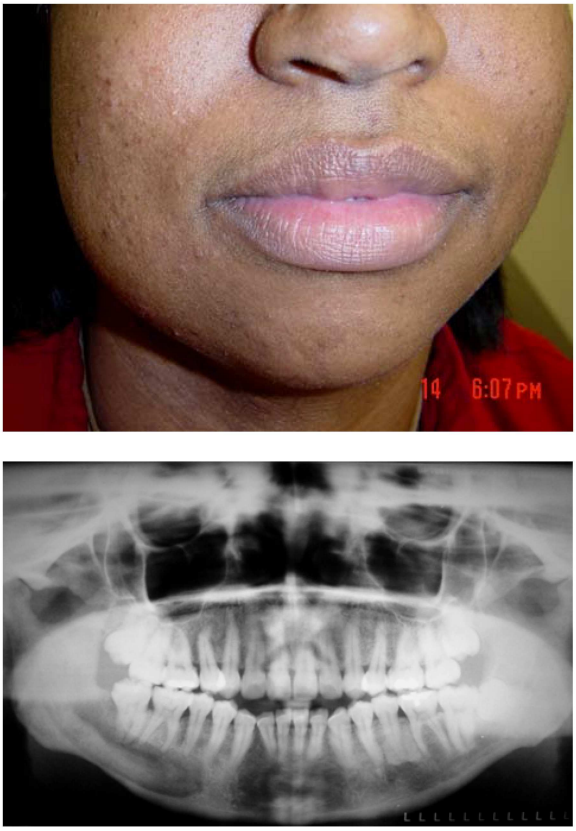
Treatment: Surgical exploration and observation for resolution.

Prognosis: Excellent. Small risk of recurrence.

Differential diagnosis: Hemangioma, early stage of fibrous-osseous lesions, central giant cell granuloma.



Simple bone cyst in the posterior mandible. Note scalloped margins extending between the roots of the molars.



Simple bone cyst

11. Ameloblastoma (adamantinoma)

Description: Benign and aggressive tumor. Malignant variants described.

Etiology: Odontogenic epithelial origin.

Prevalence: Most common odontogenic tumor after odontoma. Most lesions occur in the third to fifth decades of life. Slight predilection for males and Blacks.

Location: Most often in the posterior mandible, mainly in the molar and ramus region and associated with unerupted tooth.

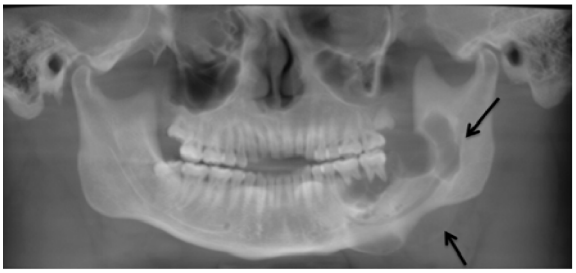
Signs and symptoms: Slow growth. Deformity with facial asymmetry. Minimal symptoms. Usually discovered as an incidental finding.

Radiographic findings: Unilocular or multilocular radiolucency with sclerotic margins. Cortical expansion with or without perforation.

Treatment: Less aggressive and unicystic forms may be treated with local excision. Multicystic form requires local excision or resection.

Prognosis: Good. Recurrence may be higher with conservative treatment. Long-term follow-up is recommended.

Differential diagnosis: Dentigerous cyst, keratocystic odontogenic tumor, odontogenic myxoma, central giant cell granuloma.



Ameloblastoma in the left mandible. Note multilocular and expansile lesion with "knife-edge" radicular resorption of the molars.

12. Odontogenic myxoma (myxofibroma, fibromyxoma)

Description: Benign intra-osseous infiltrative tumor. Rarely aggressive, develops only in the facial bones.

Etiology: Odontogenic ectomesenchymal origin.

Prevalence: Relatively rare. Wide age range, but usually identified in the first to third decade of life. Slight female predilection.

Location: Commonly affect the posterior maxilla or mandible.

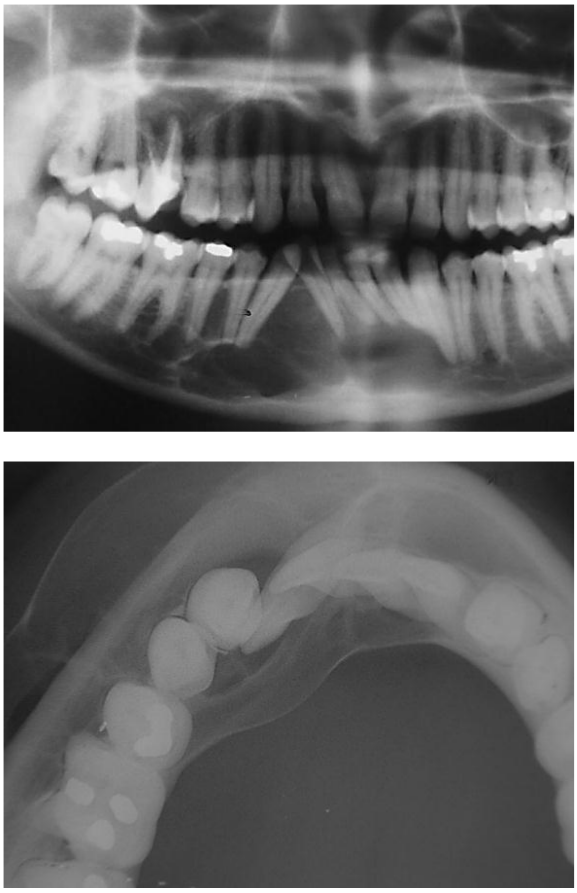
Signs and symptoms: Slow growth. Usually asymptomatic.

Radiographic findings: Most often multilocular well-defined radiolucency with cortical thinning and expansion. Uncommon cortical perforation. Maxillary sinus and zygomatic process may be involved.

Treatment: Excision with bony curettage. Bloc resection is indicated in large lesions.

Prognosis: Good, however high recurrence rate is reported. Long-term follow-up is recommended.

Differential diagnosis: Ameloblastoma, keratocystic odontogenic tumor, central giant cell granuloma, central hemangioma.



Courtesy of Dr. Celso Lemos - Department of Oral Medicine, School of Dentistry, Sao Paulo University

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Chapter 4

Skin Lesions

Seborrheic keratosis (dermatosis papulosa nigra)

Basal cell carcinoma

Melanotic macule

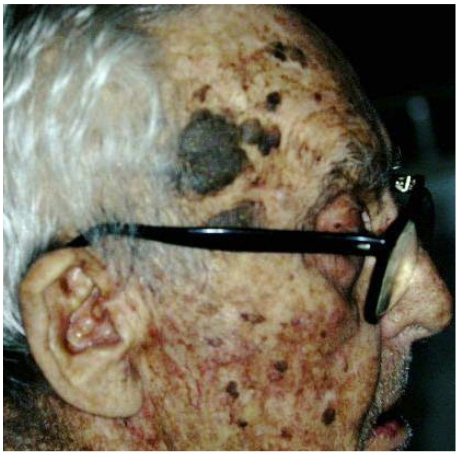
Melanoma

Actinic lentigo

Perioral dermatitis

Latex contact dermatitis

1. Seborrheic keratosis (dermatosis papulosa nigra)



Description: The most common benign epithelial tumors, seborrheic keratoses begin as small "warty" papules with or without pigmentation, which may evolve to plaques with a "stuck on" appearance. They may present as isolated or generalized lesions on the face, trunk, and upper extremities. They tend to increase in numbers over the individual's lifetime. Physical examination reveals a greasy feel and a flat nodule, which may be brown, gray, black, or skin colored, round, oval or irregular, and have a fine stippled texture.

In African Americans, Black Africans, and deeply pigmented South East Asians the lesions appear as myriad tiny to enlarging raised black lesions.

Epidemiology and Etiology: The lesions are hereditary, occurring more commonly and more extensively in males, and they do not appear until after age 30.

Treatment: Light electrocautery can be used to remove the lesions and to prevent recurrence; but this method precludes histopathological verification. Cryosurgery works only on flat lesions and recurrences are possible. Cryosurgery followed by curettage permits histopathologic examination. Solid black lesions should be excised with a punch biopsy to rule out malignant melanoma. Hypopigmentation can occur in darker skinned individuals where keratoses have been removed.

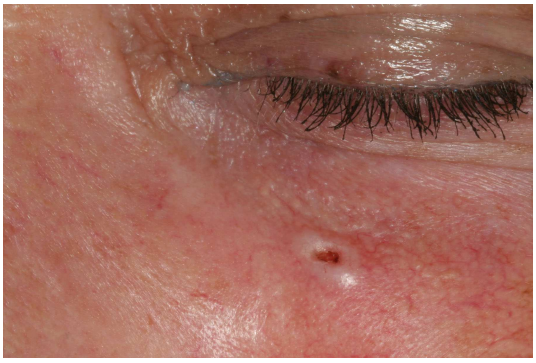
Prognosis: The lesions are seen with increasing age and are benign. They do not become malignant.

Differential diagnosis: The differential diagnosis includes solar lentigo, actinic keratosis, lentigo maligna, lentigo maligna melanoma, and basal cell carcinoma.

2. Basal cell carcinoma

Description: Basal cell carcinoma is the most common type of skin cancer, which can be locally invasive, aggressive, and destructive, but rarely metastasizes. It usually occurs on skin which has the capacity to develop hair follicles. It is rarely seen on the vermilion border of the lips.

The lesions may appear nodular, ulcerating, sclerosing, superficial and multicentric, and pigmented. They may begin as a papule or nodule with a translucent, pearly appearance and be skin colored with superficial telangiectasia. The nodules are usually well defined and firm and may present with focal ulcers with rolled borders (termed rodent ulcers). More than 90 percent of lesions occur on the face and most are isolated single lesions, although multiple lesions can occur. The “danger sites” for development are at the medial and lateral canthi of the eyes, the nasolabial folds, and behind the ears.



2.1. Epidemiology and Etiology

Fair skinned individuals and albinos and persons with a history of extensive sun exposure at young ages are more predisposed to develop basal cell carcinoma later in life. Age of onset is usually over age 40. BCC is seen more frequently in males, and is rare in brown or black skinned individuals. Previous radiotherapy for facial acne greatly increases the risk.

2.2. Treatment

Treatment options include excision with primary closure, cryosurgery or electrosurgery for small lesions, and radiation therapy. Microscopically controlled surgery (Mohs surgery) is the favored approach for excision of lesions in the "danger sites." Cryosurgery and electrocautery can leave scars. Radiation therapy is preferred when there is potential for disfigurement or in old age. Topical treatment with 5-fluorouracil ointment and imiquimod cream is an effective option which will not produce scars, but lengthy treatment is necessary.

2.3. Prognosis

Basal cell carcinoma does not metastasize and lesion sites respond well to surgical treatments. In the "danger sites," the lesions may invade deeper tissues and cause extensive destruction of muscle and bone. Death may result from invasion into the dura mater, hemorrhage of eroded large vessels, or infection.

2.4. Differential diagnosis

Diagnosis is usually made clinically and confirmed histologically. Lesions to consider in the differential diagnosis include dermatofibroma, superficial spreading and nodular melanoma, squamous cell carcinoma, syphilitic chancre, and nevomelanocytic nevi.

3. Melanoma

Description: Cutaneous melanoma is the most malignant tumor of skin structures, and its incidence is on the rise. It is responsible for 80% of deaths from skin cancer in the United States, with an estimated 8,650 deaths annually. The lifetime risk of developing melanoma in the United States in 2010 was 1 in 50. Melanoma represents 5% of newly diagnosed cancers in men and 6% of newly diagnosed cancers in women annually. Melanoma is among the most common cancer types in younger aged individuals. Deaths from melanoma also occur at younger ages than most other cancers. The most common type of melanoma is superficial spreading melanoma. Other types include nodular melanoma, lentigo maligna melanoma, and acral lentiginous melanoma. The TNM classification system is used to stage cutaneous melanomas.

Melanoma recognition is categorized into six signs:

Asymmetry in shape

A **border** which has irregular edges, which can be scalloped, notched, and clearly defined

Color which is not uniform but rather displaying mixed colors of brown, black, gray, blue, red, and white

Diameter which is greater than 6 mm

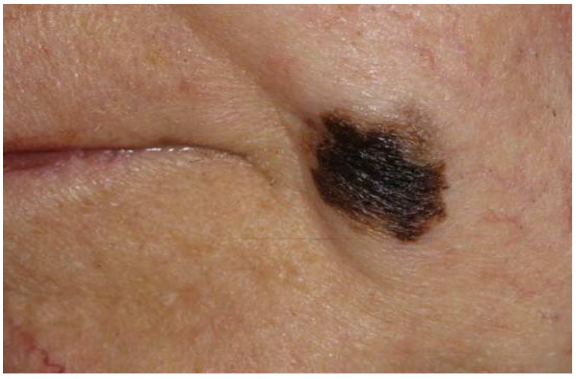
Elevation or surface distortion which can be assessed by side lighting, and

Evolving or increasing in size

Epidemiology and Etiology: The etiology of melanoma is unknown. However genetic predisposition and sun exposure are believed to be factors. Light skinned individuals and those who experienced sunburns during childhood or intermittent burns throughout their youth have a higher incidence. The presence of dysplastic melanocytic nevi, congenital nevomelanocytic nevus, and a family history of melanoma also increase the risk.

There is equal prevalence in males and females and the median age of diagnosis is 65. Rarely seen in brown or black skinned individuals, the highest incidence is in fair or light skinned white individuals, especially those with outdoor occupations and recreational habits. Oral melanoma accounts for less than 1% of all melanomas.

Treatment: Complete surgical excision with a 1 cm margin for lesions which are less than 2mm in thickness is the treatment of choice. Wide surgical excision is indicated for larger, more deeply invasive tumors. Clinically evident regional metastasis in the absence of distant metastasis warrants lymph node dissection. Hypofractionation and neutron beam radiation therapy may be used as adjunctive therapy in some patients. Chemotherapy and immunotherapy also show some promise for treatment.



Prognosis: Early detection and treatment of cutaneous melanoma before metastasis has developed is associated with a high 5-year survival rate. Thicker melanomas at discovery or those with regional lymph node metastasis have a poorer prognosis; and patients with

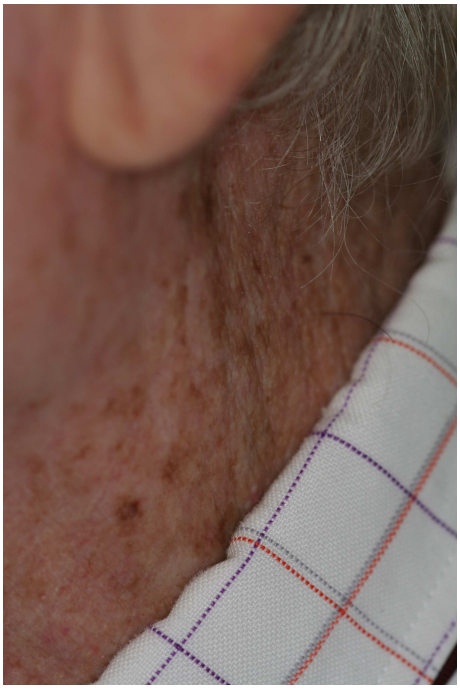
disseminated disease at time of diagnosis have a bleak prognosis. Cutaneous melanomas appearing between the scapulae on the back, the posterior upper arms, posterior and lateral neck, and scalp have worse prognoses. Prognosis is better for women and for patients younger than age 50. Patients with oral mucosal melanomas which are deeper than 0.5 mm and patients with nonpigmented lesions have a poorer prognosis. Continuous follow-up and monitoring are necessary.

Differential diagnosis: Seborrheic keratoses, solar lentigo, melanocytic nevus.

4. Actinic lentigo (lentigo solaris; solar lentigo; age spot; liver spot; senile lentigo)

Description: Actinic lentigo are benign, flat, brown to tan, evenly pigmented macules with well-demarcated irregular borders, commonly found on the dorsa of hands, on the face and arms of elderly Caucasian individuals. They are rarely seen before age 40 and are very common in individuals older than age 70.

Etiology: Chronic exposure to and damage caused by ultraviolet light



Treatment: No treatment is required, however the color intensity of the lesions can be reduced with the use of topical retinoic acid. Additionally, the lesion can be eliminated completely with a Q-switched ruby laser. Cryotherapy, topical hydroquinone, tazarotene, adapalene, and a combination of mequinol and tretinoin can also be used. Preventive therapy includes use of sunscreen.

Prognosis: Actinic lentigo do not undergo malignant transformation. New lesions can develop at other sites or adjacent to the original site. After removal, they do not reoccur in the same site.

Differential diagnosis: Ephelis (freckle), lentigo simplex, meiasma, seborrheic keratosis.

5. Perioral dermatitis

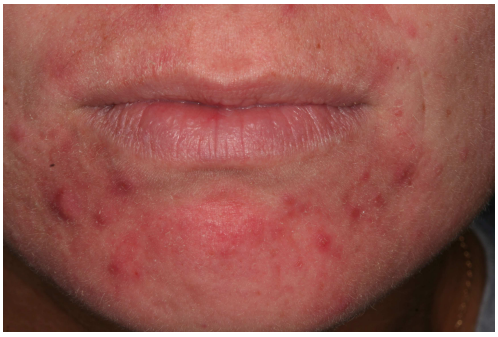
Description: Perioral dermatitis characteristically presents as multiple tiny erythematous papules, microvesicles, and papulopustules which are symmetrically grouped periorally. The nasolabial folds are often involved and a rim of spared skin is seen around the vermilion border of the lips. The lesions often coalesce and confluent plaques may appear eczematous with tiny scales.

Epidemiology and Etiology: Unknown. Seen predominantly in females between ages 16-45. May be aggravated by topical fluorinated glucocorticoids.

Treatment: Oral minocycline, doxycycline, or tetracycline. Topical metronidazole or erythromycin gel. Do not use topical fluorinated glucocorticoids. Caution patients about sun exposure.

Prognosis: The lesions typically show dramatic improvement over several weeks and resolve without recurrence in a few months.

Differential diagnosis: Allergic contact dermatitis, atopic dermatitis, seborrheic dermatitis, rosacea, acne vulgaris, steroid acne, sarcoidosis.



6. Latex (allergic) contact dermatitis

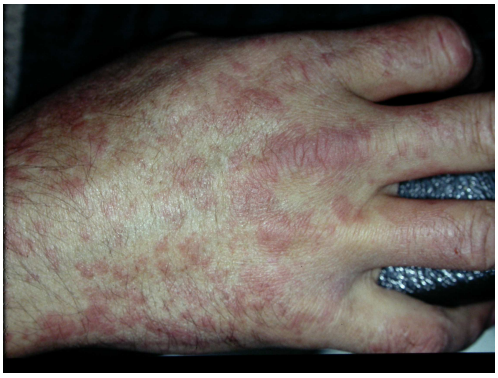
Description: Allergic contact dermatitis is a delayed cell-mediated hypersensitivity reaction which can occur at any age and accounts for 10-50 percent of occupational related illnesses in the United States. Sensitization can occur over weeks to years depending on the strength of the sensitizer. After exposure, the sensitized individual will develop skin eruptions confined to the site of exposure hours to days following the exposure. Repeated exposures will cause the eruptions to worsen. Symptoms include intense itching, stinging and pain, and in severe allergic contact dermatitis fever. Acute skin lesions present as well demarcated erythema and edema. Small papules and vesicles, bullae, and confluent erosions may also develop. Other, non-exposed sites may develop similar signs after several weeks. Chronic skin lesions may show thickening of the epidermis with plaques and scales, lichenification, papules, excoriations, and pigmentation. Chronic contact dermatitis exhibits spreading margins.

Etiology: Allergic contact dermatitis is caused by an allergen that elicits a cell-mediated delayed hypersensitivity reaction. It is an immunologic response, which only occurs in sensitized individuals.

Treatment: Management of the dermatitis involves removing the allergen. Topical glucocorticoids are indicated for mild to moderate cases. Systemic administration of glucocorticoids may be necessary for severe cases. Immunosuppression with oral cyclosporine may be necessary if the allergen cannot be completely avoided.

Prognosis: The dermatitis will reoccur upon re-exposure to the allergen and the eruptions will worsen with repeated exposures.

Differential diagnosis: Irritant contact dermatitis, atopic dermatitis, seborrheic dermatitis, psoriasis, epidermal dermatophytosis, fixed drug eruption, erysipelas phytophotodermatitis.



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Chapter 5

Benign Inflammatory Lesions/Conditions of Oral Mucous Membranes

Aphthous stomatitis
Traumatic ulcer
Geographic tongue (benign migratory glossitis)/erythema migrans
Lichen planus
Inflammatory papillary hyperplasia
Epulis fissuratum (inflammatory fibrous hyperplasia)
Contact stomatitis from cinnamon/medication burn
Dentifrice related sloughing

1. Aphthous stomatitis (canker sores)

Description: Recurrent aphthous stomatitis (RAS) is one of the most common and painful conditions in the oral cavity. Although variable, the lesions typically begin in childhood or adolescence, occur more frequently in females and tend to be seen less often in adults over 40. Although no single causative agent has been identified, the accompanying mucosal damage appears to be a T cell-mediated immunologic reaction.

Aphthous ulcers present in one of three forms: major, minor and herpetiform. Differences in minor and major aphthous ulcers are generally dependent on size and healing. Minor aphthous ulcerations range from 3-10 mm and generally heal within 1-2 weeks. The major form measures more than 1 cm, may take up to 6 weeks to heal, and may scar. They are both found on areas of unattached mucosa, such as the buccal and labial mucosa, as opposed to recurrent herpetic lesions, which are limited to attached mucosa such as gingiva. One to multiple ulcerations may present as shallow, round to oval, yellow-white ulcerations with a red border. Herpetiform aphthae are less common, and while not associated with the herpes virus, are named because of their clinical appearance. Herpetiform aphthae are the least common form of RAS and are often found in a more localized area, usually not on the lips, and consist of small individual lesions which can combine to form larger ulcerations. Although the lesions usually heal within 7-10 days, the outbreaks are often more frequent.

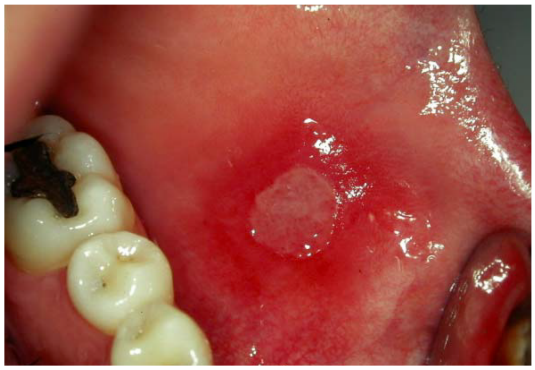
Etiology: The cause is not known, but is most likely multifactorial, and the following have been reported as some of the possible causative factors. Food allergies, stress, trauma,

hormonal influences, smoking cessation, immunologic factors, GI disease such as Crohn's or Celiac Disease and nutritional deficiencies such as B12 may have possible causal factors associated with RAS. Certain HLA types have been associated with aphthae.

Treatment: Review the patient's medical history to rule out the need for medical referral for detection of systemic disease. Aphthous ulcers are an immunologic condition and treatment should be directed toward suppressing the immunologic reaction responsible for the lesion. Many patients with mild or intermittent lesions may not require any treatment or may use over-the-counter anesthetic or protective bioadhesive products. RAS is not associated with herpes or any other viral infection and cannot be treated with anti-viral medications. The chemical cautery agent, silver nitrate, can cause significant soft tissue damage and should not be used in treatment.

Most patients who seek more aggressive treatment respond well to local high potency topical corticosteroids, which carry a lower risk of adverse effects than systemic treatment and should be considered the first line of treatment. Initiation of healing is usually noted within 24-48 hours with 0.05% betamethasone or clobetasol gel. These gels should be applied 4-5 times daily and are more effective intraorally than ointments or creams.

For patients with multiple lesions, or RAS in difficult to reach locations such as the soft palate or tonsillar pillars, syrups or elixirs may be more practical. For example, prednisolone is available in syrup form and can be used in a swish and spit regimen. Another product for use in the fauces is beclomethasone dipropionate aerosol spray. In cases resistant to these medications, systemic steroids in tablet form may be considered or else a swish and swallow steroid syrup can be used for both topical and systemic effects.



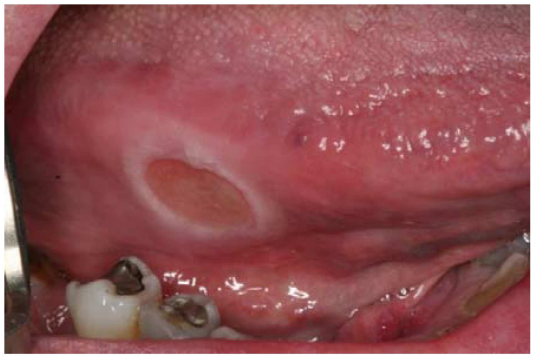
Prognosis: Good, if the patient can gain relief from pain and sustain adequate nutrition and hydration.

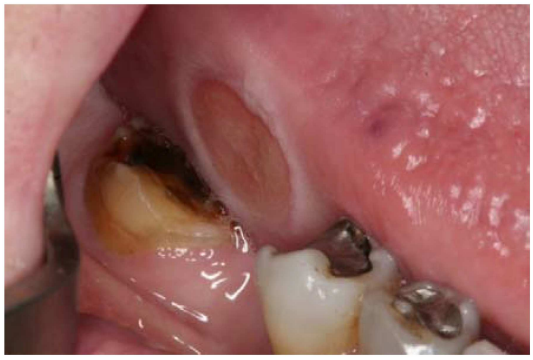
Differential Diagnosis: They are commonly confused with herpetic ulcerations. Possible differential diagnoses include: trauma, soft tissue lesions due to inflammatory bowel disease

or celiac disease, Behcet's syndrome, Sweet's syndrome, MAGIC syndrome, PFAPA syndrome, cyclic neutropenia, vitamin deficiencies and others.

2. Traumatic ulcer

Description: An ulcer is a localized area of discontinuity in surface epithelium. Traumatic ulcers are frequently observed in the oral cavity and can be of such varying size and shape that they are difficult to characterize. Simple traumatic ulcers are most often found on the buccal mucosa, tongue, and lips, but may also occur anywhere in the mouth. They appear as areas of erythema covered by a yellowish membrane of variable thickness. A rolled hyperkeratotic border may develop adjacent to the ulcer.





Etiology: In many cases of traumatic ulceration, there is a corresponding source of irritation. They may be due to mechanical damage from food, self-inflicted injury (such as biting), occur due to mastication, a result of toothbrushing injury, malocclusion, broken down or sharp teeth,

placement of restorations, prosthesis irritation or injury during sleep. Ulcerations can also be caused by thermal, chemical or electrical burns.

A histologically specific type of ulcer with elevated and indurated margins called traumatic ulcerative granuloma with stromal eosinophilia (TUGSE) resembles squamous cell carcinoma. It most often affects the tongue and exhibits deep inflammation that resolves more slowly and typically requires biopsy. An unusual occurrence is that incisional biopsy often appears to initiate healing.

Treatment: Relieving an obvious source of irritation or toxic agent should result in resolution of an ulcer. Symptomatic relief can be provided by over-the-counter bioadhesive preparations. In some cases clinicians advocate application of corticosteroids to speed healing, while others claim this will delay healing.

Prognosis: When the source of the trauma is removed, the ulcer should resolve. If healing does not occur in 2-3 weeks, a biopsy must be performed.

Differential diagnosis: Squamous cell carcinoma, Riga-Fede disease, TUGSE, ulcerative mucosal disease such as lichen planus.

3. Geographic tongue (benign migratory glossitis) / erythema migrans

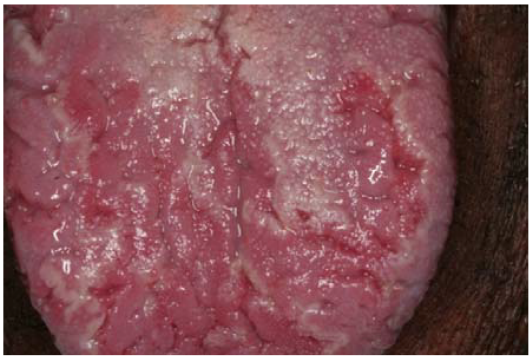
Description: Benign migratory glossitis (BMG) is a common benign lesion that most often affects the tongue and sometimes other oral mucosal surfaces (erythema migrans). It is an inflammatory disorder characterized by multiple erythematous areas representing loss of filiform papillae surrounded by a yellow-white irregular border. The map-like areas of red and white, (hence the name geographic tongue), usually resolve within a few days, but may quickly develop in another area. Patients with fissured tongue often have geographic tongue as an accompanying condition. When the patient is asymptomatic, BMG is often noted on a routine dental exam or a patient/parent may seek consultation when they notice the appearance of the tongue or experience symptoms.

Etiology: Widely considered to be unknown, although some authors propose an association with psoriasis.

Treatment: In the great majority of cases, no treatment is required other than reassuring the patient that BMG is a benign entity. If a patient complains of burning or sensitivity that affects daily life, topical corticosteroids such as betamethasone gel applied in a thin layer may provide symptomatic relief. In a recent study of long standing, symptomatic BMG, cyclosporine rinses or topical 0.1% tacrolimus ointment have been used with success.

Prognosis: Typically, BMG is a chronic condition with periods of exacerbation and remission. According to a recent study, taste is not affected by BMG.

Differential diagnosis: The lesions of geographic tongue are characteristic of the condition, however, it may be misdiagnosed as candidiasis.



4. Lichen planus

Description: Lichen planus is a relatively common chronic, inflammatory, mucocutaneous disease seen most often in middle-aged females. Cutaneous lesions appear as multiple pruritic, purplish, polygonal papules. On close examination, the skin lesions, which occur mostly on extensor surfaces of the extremities, will show a fine lace-like pattern of lines known as Wickham's striae. Oral lichen planus (OLP) occurs most often in the absence of skin lesions. There are basically two main forms of OLP, reticular and erosive. The reticular form is more common and usually appears as multiple lesions with a bilateral symmetrical pattern. It begins as small white lesions which join to form an annular or plaque-like pattern. The plaque-like form may be difficult to distinguish from leukoplakia. A typical appearance shows slender white-gray lines radiating from the oral papules (Wickham's striae). Instead of the reticular pattern, the dorsal tongue may exhibit more of a keratotic plaque-like lesion.

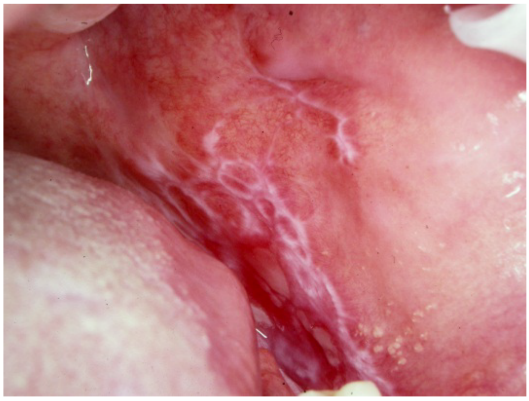
Erosive lichen planus lesions are usually symptomatic to patients and therefore the patient is more likely to seek professional advice. Atrophic, erythematous lesions with central ulceration and fine white striae on the periphery of the erosions are seen clinically. If confined to the gingival mucosa in a pattern scalloping the teeth, this is termed desquamative gingivitis.

Etiology: The cause is unknown, but appears to be multifactorial and is characterized by a T-cell medicated chronic immune response and abnormal epithelial keratinization.

Treatment: The reticular and plaque-like lesions are usually asymptomatic and treatment is not necessary. For symptomatic OLP, topical steroids, such as triamcinolone mouthwash or mixed with orabase, clobetasol or fluocinonide are used first in treatment. If the OLP is severe or affects large areas of the oral mucosa, systemic corticosteroids should be prescribed. Patients should be advised that the lesions will most likely recur and the possibility of candidiasis associated with corticosteroid use may occur. No therapy currently cures OLP; the goal of treatment for symptomatic lesions is palliation.

Prognosis: There is still controversy whether OLP is associated with an increased risk of malignancy. Excessive tobacco and alcohol use should be discouraged and it is recommended

that lesions are observed at least yearly. Isolated erosive lichenoid lesions should also be biopsied to rule out premalignant or malignant lesions.



Differential diagnosis: The clinical features may be diagnostic of the typical reticular form of OLP. However, biopsy is necessary if the form is atypical or if clinical features suggest the possibility of dysplasia or malignancy. There are many oral lichen planus-like or "lichenoid" lesions which can confuse the diagnosis of OLP. Drug reactions, graft-versus-host disease, and oral contact stomatitis related to dental materials, most often amalgam, may be indistinguishable from OLP. In diagnosing erosive lichen planus, other ulcerative or erosive diseases such as lupus erythematosis and chronic ulcerative stomatitis should be ruled out by biopsy and immunofluorescent studies. Gingival lesions of erosive lichen planus may also resemble pemphigoid or pemphigus vulgaris with biopsy and immunofluorescence required.

5. Inflammatory papillary hyperplasia

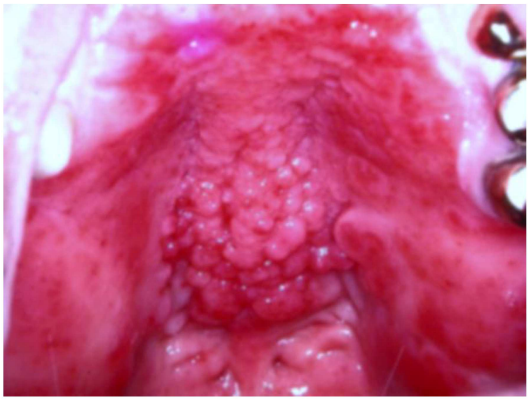
Description: Inflammatory papillary hyperplasia (IPH) is a reactive tissue response that is usually found in the hard palate underneath an ill-fitting dental prosthesis, or even one who exhibits parafunctional habits. It may also occur infrequently in a patient who does not wear prosthesis, such as a mouth breather. Less frequently, this lesion may occur on the mandibular edentulous ridge or the surface of an epulis fissuratum. It is usually asymptomatic and the mucosa is erythematous, with a pebbly appearance. Some believe this lesion is part of a spectrum that involves denture stomatitis as the earliest form.

Etiology: Poorly fitting prostheses, poor prosthesis hygiene and wearing a prosthesis constantly without removal, appear to be the major reasons for occurrence of this lesion. Candida has also been suggested to have a causal relationship.

Treatment: In cases of early IPH, denture removal for extended periods of time may allow the tissue to resume a more normal appearance. Often antifungal therapy involving the mucosa

and denture must accompany daily removal of the prosthesis to provide complete resolution. Meticulous hygiene of the prosthesis and oral cavity should be practiced. In more advanced cases, the excess tissue should be excised prior to fabrication of a new prosthesis. Techniques used may include surgical blade incision, laser surgery and electrosurgery.

Prognosis: Since the condition is benign, the prognosis is good. Once the lesions are resolved, if the patient continues to practice good oral hygiene and allow the tissue to rest daily, IPH should not recur.



Differential diagnosis: Although the appearance of the lesions is characteristic, keratosis follicularis (Darier's disease) may be considered in unusual cases.

6. Epulis fissuratum (inflammatory fibrous hyperplasia)

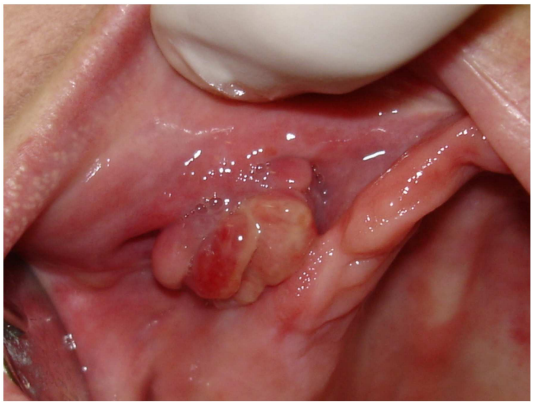
Description: This lesion consists of folds of hyperplastic tissue into which the flange of a complete or partial denture rest, most often in the maxillary anterior vestibule, although sometimes it can be seen lingual to the mandibular ridge. The excess tissue is usually firm and fibrous, but can be inflamed and ulcerated, similar to that of a pyogenic granuloma. The size of an epulis varies greatly, from less than 1 cm to the length of the vestibule. This lesion is seen more often in older individuals, as expected with a denture-related conditions and is more frequently observed in females.

Etiology: Hyperplasia of the oral mucosa in reaction to an ill-fitting complete or partial denture is the usual causative factor.

Treatment: Surgical removal and microscopic examination of the tissue is the definitive treatment for epulis fissuratum. This should be accompanied by some form of correction to the prosthesis or remake of the prosthesis in order to prevent recurrence.

Prognosis: Good with definitive treatment and proper prosthesis fit.

Differential diagnosis: The characteristic appearance of epulis fissuratum is generally diagnostic.



7. Contact stomatitis from cinnamon/medication burn

Description: Products flavored with artificial cinnamon are fairly common. Some of the most frequently found cinnamon flavored products are foods such as ice cream and candy, gum, mints, toothpaste, mouthwashes and floss. In susceptible individuals, the use of toothpaste results more in diffuse gingival involvement with enlargement, edema and erythema. Sloughing of superficial epithelium is common.

The reaction to gum and candy is more localized, with most lesions occurring on the buccal mucosa and lateral tongue. The lesions have an erythematous base, but are often white due to a covering of hyperkeratosis of the surface epithelium and can progress to the dorsum of the tongue.

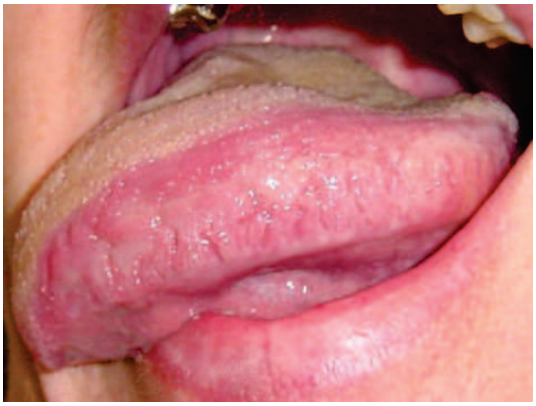
Etiology: Reactions to cinnamon flavoring are found most commonly in products that are associated with prolonged or frequent contact with the oral mucosa, for example, candy, gum, and toothpaste. Tartar control toothpastes contain bitter pyrophosphates which require extensive flavoring to mask and therefore may cause oral lesions. Although less common, there are reports that the spice form of cinnamon may also cause mucosal reactions. Cinnamon contact stomatitis is believed to be a form of intra-oral contact allergy.

Treatment: Discontinuation of the cinnamon-flavoring containing product usually results in resolution of the lesions within a week. If the lesions last longer, a topical corticosteroid may be used for a short time.

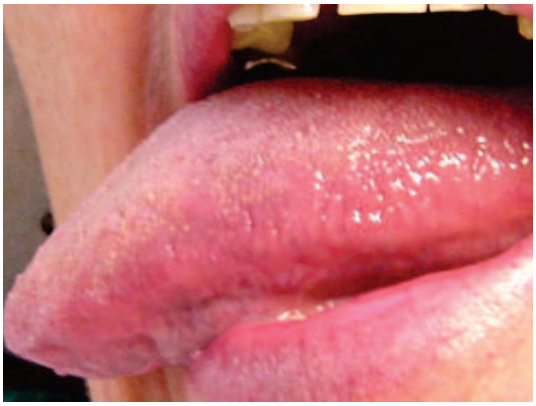
Prognosis: Good, with discontinuation of the offending product

Differential diagnosis: History of the use of cinnamon flavored products, clinical appearance and resolution of the lesions upon cessation of product use are adequate to diagnose contact

stomatitis from cinnamon. Leukoplakia, hairy leukoplakia, and lichenoid reaction may also be considered.



Tongue change from chewing cinnamon gum

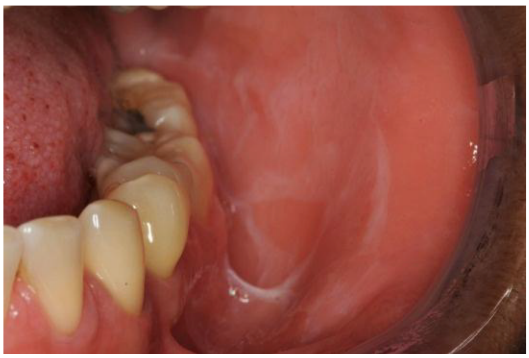


Improvement after discontinuation of cinnamon gum

8. Dentifrice Related Sloughing

Description: Dentifrice related sloughing of the oral mucosa is an increasingly common finding and may be caused by a variety of additives found in many dentifrices. While typically asymptomatic, erythema or a burning sensation is sometimes seen. It is characterized by areas of white, "stringy" sloughing of the superficial keratin layer. Among the associated additives, flavoring agents, abrasives, detergents (i.e. sodium lauryl sulphate), "tartar con-

trol" agents (i.e.tetrasodium and/or tetrapotassium pyrophosphate), and fluorides may result in reactive changes of the oral mucosa. Manufacturers are aware of the problem of dentifrice related sloughing and have sometimes removed a product from availability, adjusted a product's formulation or recommended alternate products for susceptible persons.



Etiology: Hypersensitivity to dentifrice detergent and tartar control agents may result in dentifrice related sloughing. Higher concentrations of detergents are required to solubilize pyrophosphates in a tartar control dentifrice and can lead to reactions to the detergent. Pyrophosphates have also been shown to increase alkalinity which can irritate oral mucosa. A person's adverse reaction to a tartar control dentifrice may also be exacerbated by dry mouth, which is a common finding in the United States' growing elderly population and concomitant increased use of medications.

Treatment: Treatment consists of discontinuation of the offending product.

Prognosis: Prognosis is good, with discontinuation of the offending product.

Differential Diagnosis: Intraoral reaction to other chemical agents

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Chapter 6

Benign Infectious Lesions/Conditions of the Oral Mucous Membrane

Herpes simplex virus infection

Herpes zoster

Candidiasis

Angular cheilitis

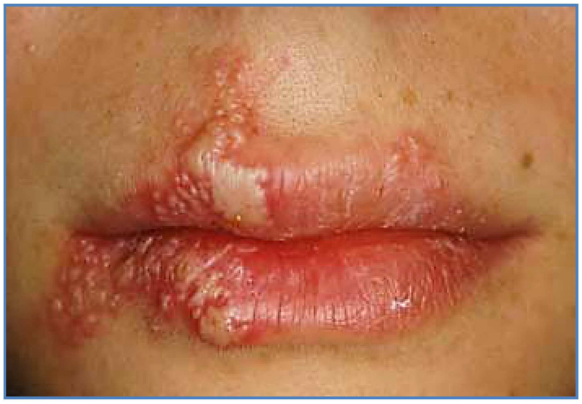
Atrophic glossitis

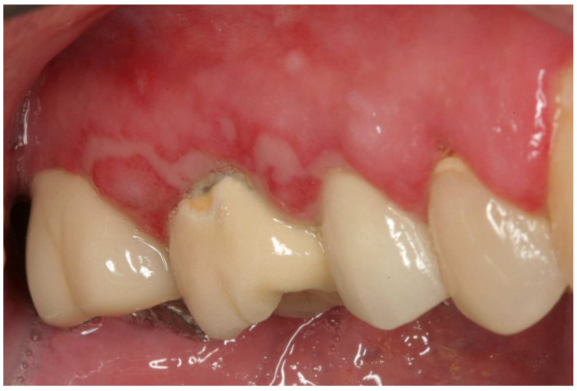
1. Herpes simplex virus infection

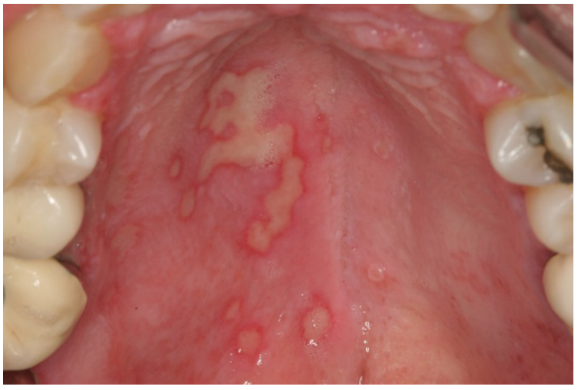
Description: Herpes simplex infection is a common disease that, when clinical signs are present, show striking and distinct oral features. The infection shows two clinical presentations: primary and secondary, or recurrent. Primary herpes simplex: Most primary infections are asymptomatic and occur during childhood. When symptoms occur, clinical features include fever, malaise and tender lymphadenopathy. Oral manifestations are characterized by painful ulcerations that involve both keratinized and nonkeratinized mucosal surfaces (gingivostomatitis). A "punched out" gingival margin is often characteristic. Occasionally, the infection occurs later in life and in an adult may manifest as a pharyngotonsillitis. Patients are typically symptomatic from 1-2 weeks.

Secondary or recurrent herpes simplex: Recurrent herpes affects a small number of patients infected with the virus. Most recurrent lesions affect the vermillion zone of the lip (recurrent herpes labialis). Intraoral lesions are typically limited to the keratinized mucosa and are typically only mildly symptomatic, in contrast to those of the primary outbreak. Recurrent herpes labialis may occur spontaneously or be triggered by such stimuli as sunlight, trauma or systemic infection. It often begins with a prodromal period of a few hours and is characterized by a "tingling" or "itching" sensation. The outbreak progresses to the development of numerous vesicles, which quickly rupture, leaving an amber or honey-colored crust. In the immune competent, lesions heal within a week to 10 days. The frequency of outbreaks is highly variable from patient to patient, with some experiencing recurrences quite regularly.

Etiology: Oral infection is caused by members of the human herpesvirus (HHV) family, herpes simplex type 1 (HSV-1, HHV-1) or, to a lesser extent, herpes simplex type 2 (HSV-2, HHV-2).







Treatment: The primary infection may be treated effectively by NSAIDS and antiviral medications if administered within the initial 72 hours. Topical anesthetics may also be effective in reducing the discomfort. It is necessary for patients to maintain adequate hydration.

Recurrent herpes infection may also be treated effectively, if therapy is initiated at the earliest point of the outbreak. A regimen of high-dose, short duration valacyclovir has shown some efficacy, even aborting the outbreak in some patients when initiated during the prodromal period. Topical antiviral medications may also shorten the duration of the outbreak.

Prognosis: The prognosis is good, with most outbreaks self-limiting. For the immunocompromised patient, however, the infection may be characterized by substantial morbidity, necessitating early therapeutic intervention.

Differential diagnosis: Herpes simplex is often distinct in its presentation and readily diagnosed. For milder cases, or those without significant oral ulceration, infectious mononucleosis may be considered, since tender lymphadenopathy is often a component of both diseases. Other considerations might include hand, foot and mouth disease, herpangina or erythema multiforme.

2. Herpes zoster

Description: Herpes zoster is the recurrent outbreak associated with the virus that causes chicken pox. The virus is a member of the human herpesvirus family and, like other herpes viruses, primary infection ends in latency. Often after many years or decades, the recurrent outbreak is manifested as herpes zoster. Unlike HSV-1, the recurrence is typically limited to a single outbreak. After resolution of the primary varicella-zoster infection, the virus resides in the dorsal spinal ganglia. Herpes zoster will involve the dermatome supplied by the sensory nerves. Often, a painful prodrome will develop prior to the vesicular outbreak. Lesions are intensely painful and typically show an abruptly unilateral distribution. The vesicles rupture, leaving a crust which will usually heal in 2-3 weeks. While symptoms typically resolve along with resolution of the exanthem, a number of unfortunate patients continue to experience pain, a condition known as post-herpetic neuralgia. Ocular involvement is associated with significant morbidity.

The oral lesions or herpes zoster will follow a similar unilateral anatomic distribution and be characterized by painful ulcerations of the mucosa.

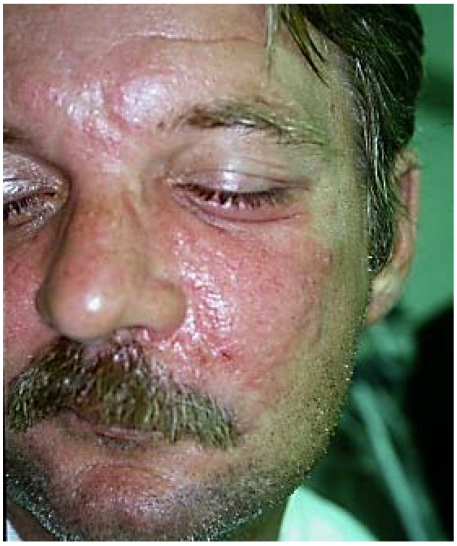
Etiology: Herpes zoster is caused by the varicella-zoster virus (VZV), a member of the human herpesvirus family (HHV-3).

Treatment: A vaccine for herpes zoster is currently available and recommended for those with a history of varicella-zoster infection. For those experiencing the recurrence, antiviral medications are sometimes effective in shortening the duration of the outbreak, especially when initiated within the first 3 days. For post-herpetic neuralgia, topical Capsaicin has shown some

efficacy. Other therapeutic regimens may include systemic medications such as tricyclic antidepressants, anticonvulsants or gabapentin.

Prognosis: For the immune competent and those spared post-herpetic neuralgia, the prognosis is generally favorable. For the immunocompromised, herpes zoster can be associated with significant morbidity. Post-herpetic neuralgia can also be problematic for a year or more in a small percentage of patients.

Differential diagnosis: The distribution of lesions is often diagnostic for herpes zoster. However, in mild cases, the symptoms may mimic neurological conditions such as trigeminal neuralgia. Contact dermatitis may also be considered, although the discomfort associated with herpes zoster is not typically associated with this condition.



3. Candidiasis

Description: Candidiasis refers to infection with the dimorphic fungal organism, *Candida albicans*. The infection is primarily superficial, although rare invasive or disseminated cases may be seen in the severely immunocompromised. The organism normally resides as a yeast in a substantial percentage of the population. Under conditions favorable to the organism, such as broad spectrum antibiotic therapy, corticosteroid therapy or xerostomia, transformation to the hyphael form and progression to an infectious state may occur. Candidiasis infection most often occurs in one of four clinical presentations: pseudomembranous, or "thrush;" erythematous, which may include denture stomatitis; hyperplastic; and angular cheilitis (next topic).

Pseudomembranous candidiasis: sometimes known as "thrush," is characterized by the development of white, curd- or plaque-like deposits that are easily removable. While underlying erythema may be seen, ulceration is rare. Patients may complain of sensitivity to spicy foods or bad taste, but symptoms are typically mild. Pseudomembranous candidiasis is seen most commonly in infants, the immunocompromised or patients undergoing corticosteroid therapy.

Erythematous candidiasis: is simply characterized by erythema and shows a number of recognized forms, including denture stomatitis and central papillary atrophy. Denture stomatitis is seen in patients with removable prostheses. The area covered by the prosthesis is erythematous and tends to clearly follow the outline of the denture or partial denture. Typically asymptomatic, denture stomatitis is often diagnosed during routine dental visits. Contributing factors to the development of denture stomatitis include xerostomia, 24 hour denture wear and/or poor denture hygiene.

Central papillary atrophy: was previously diagnosed under the term median rhomboid glossitis and thought to represent a developmental abnormality of the dorsal surface of the tongue. It is characterized by a variably-sized area of papillary atrophy and erythema. It is often asymptomatic, although patients may complain of sensitivity to spicy foods. Long-term cases may show an inflammatory hyperplastic response of the oral mucosa. Without treatment, the area of papillary atrophy may progress to involve the entire dorsal surface of the tongue. Contributing factors are similar to other forms of candidiasis.

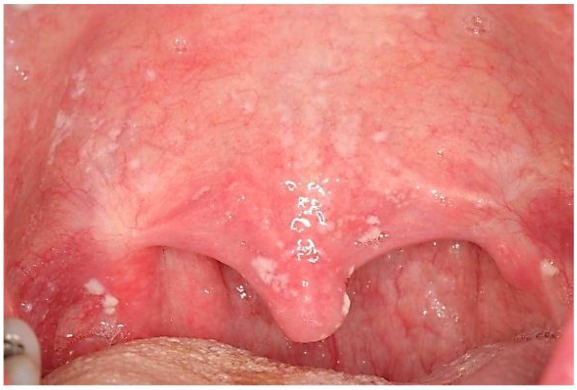
Hyperplastic candidiasis (candidal leukoplakia): is an uncommon form of infection that may be difficult to differentiate from the more serious process, leukoplakia. It is characterized by a white plaque that cannot be removed. While any mucosal surface may be involved, the anterior buccal mucosa is a relatively common site. Antifungal therapy is often necessary to rule out preneoplasia.

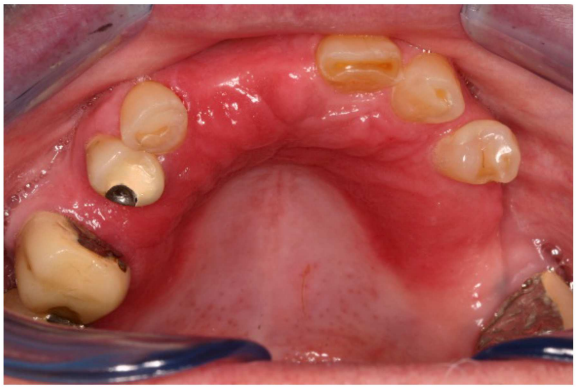
Etiology: Most candidiasis infections are associated with *Candida albicans.*

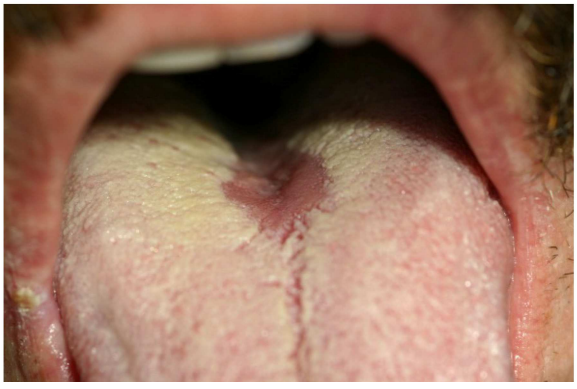
Treatment: Typically, outbreaks are superficial and effectively treated with topical antifungal agents such as Nystatin oral suspension or clotrimazole oral troches. Systemic agents, such as fluconazole, may be used in patients who experience recurrences or in cases that are refractory to the standard treatments. It is important to remember that the treatment of denture stomatitis is not limited to elimination of the organism from the oral cavity. Disinfection of the prosthesis is also necessary to reduce the potential for recurrence.

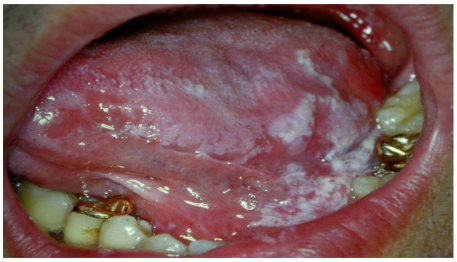
Prognosis: The prognosis for most patients is excellent. For the immunocompromised, the prognosis is more dependent upon their immune status than candidiasis infection.

Differential diagnosis: It may be necessary to differentiate denture stomatitis from allergy to dental materials. However, a thorough clinical history will often assist the clinician in differentiating the two conditions. Hyperplastic candidiasis is often difficulty to differentiate from the more serious premalignant lesion, leukoplakia. For these patients, re-evaluation after appropriate antifungal therapy will typically result in the accurate diagnosis.









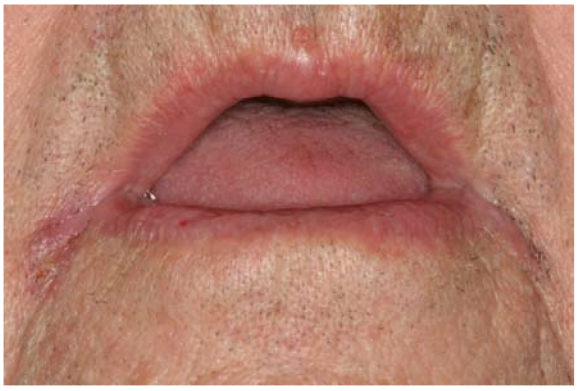
4. Angular cheilitis

Description: Angular cheilitis is a chronic condition affecting the commissural areas of the mouth. It is within the spectrum of diseases associated with candidiasis infection, although a concomitant bacterial component is often present. The lesions of angular cheilitis present as ulceration or cracking at the commissures. Lesions have a tendency to wax and wane, although they may be present for years without treatment. Contributing factors are similar to those of other forms of candidiasis infection. A reduced vertical dimension of occlusion may also be a contributing factor.

Etiology: *Candida albicans*, often in combination with staphylococcal component.

Treatment: Topical agents, such as a formulation of hydrocortisone and iodoquinol, are often effective in resolving the lesions of angular cheilitis.

Prognosis: Excellent



Differential diagnosis: The condition is typically unique in its presentation and easily diagnosed based upon the clinical features. Some vitamin or iron deficiencies are sometimes associated with a similar clinical presentation.

5. Atrophic glossitis

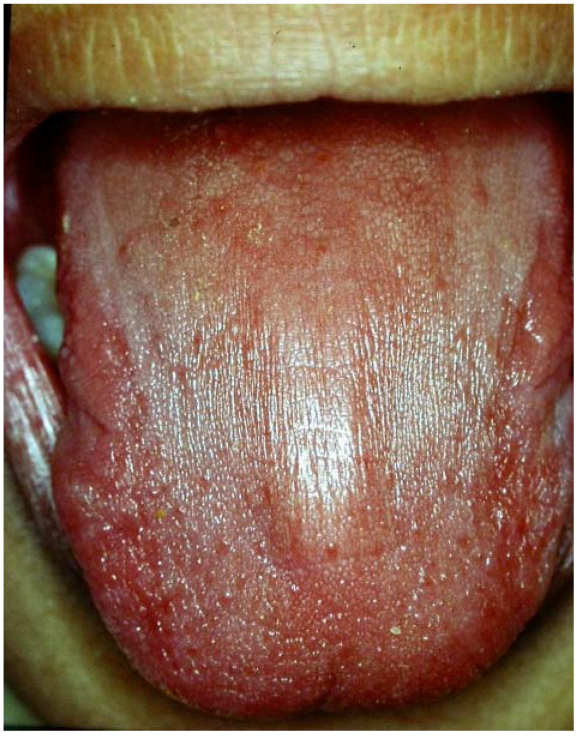
Description: Atrophic glossitis, also known as "smooth tongue" is one of the most frequent tongue conditions associated with systemic disease and requires thorough evaluation. The mucosa of the tongue is characterized by a smooth appearance, which is due to atrophy of filiform papillae, with an underlying pink to reddish color and may be painful.

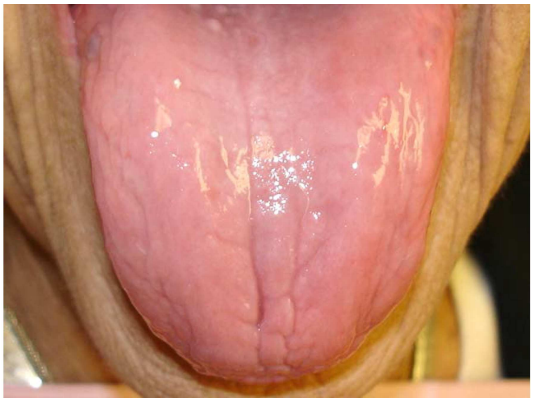
Etiology: Atrophic glossitis may be due to xerostomia, with or without co-existing candidiasis, that is often caused by medication use or a systemic disease such as Sjogren's disease. Other causes include deficiencies of Vitamin B, folic acid, niacin, riboflavin, or iron. Systemic diseases associated with atrophic glossitis include sarcoidosis, syphilis, kwashiorkor and celiac disease. It may also be associated with medication use, such as antibiotics or inhaled corticosteroids used for treatment of asthma which can lead to Candida infection.

Treatment: Treatment is aimed at elimination or palliation of the underlying condition.

Prognosis: The prognosis is good with treatment of the underlying condition.

Differential diagnosis: Median rhomboid glossitis, geographic tongue, strawberry tongue (Scarlet fever)





Candidiasis/denture stomatitis: Oral candidiasis, a fungal infection is a very common complication of dry mouth. The decreased amount of saliva and protecting factors change the local immunity of the oral tissues facilitating the overgrowth of the fungus. C. albicans is the most common specie. Ill-fitting dentures can facilitate the overgrowth of fungus. Figure … shows a common presentation of denture stomatitis. In patients with dry mouth, wearing dentures may be difficult. Saliva is an important vehicle in the process of denture retention in the oral cavity.

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Atrophic glossitis

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Chapter 7

Common Lesions of Varied Etiology

Amalgam tattoo

Nicotine stomatitis

Irritation fibroma/traumatic fibroma

Pyogenic granuloma

Peripheral ossifying fibroma

Peripheral giant cell granuloma

Mucocele

Smoker's melanosis

Frictional hyperkeratosis (Morsicatio buccorum/linguarum)/frictional keratosis

Salivary stones (sialolithiasis)

Squamous papilloma

1. Amalgam tattoo

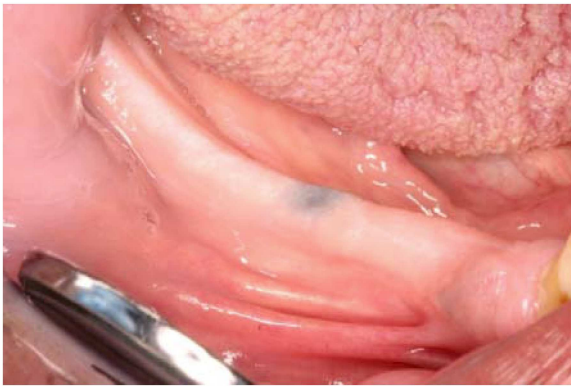
Description: Amalgam tattoo is an exogenous pigment associated discoloration of the oral mucosa. The exogenous pigment is granules of silver amalgam used in the filling of dental cavities. The amalgam tattoo appears as a gray-black macule of few millimeters in size. The border may be well-defined or blend imperceptibly with the surrounding mucosa. Common locations include the alveolar ridge, interdental papillae, alveolar mucosa, floor of the mouth and the vestibule. The amalgam tattoo is asymptomatic and does not increase in size. Multiple amalgam tattoos may be seen in the oral cavity in those with more dental restorations. If adequate amount of metal is present, an amalgam tattoo may be seen on routine radiographs. Microscopic examination of an amalgam tattoo may reveal presence of large amounts of dark metal or brown pigment often coursing along reticulin fibers in the connective tissue especially around blood vessels.

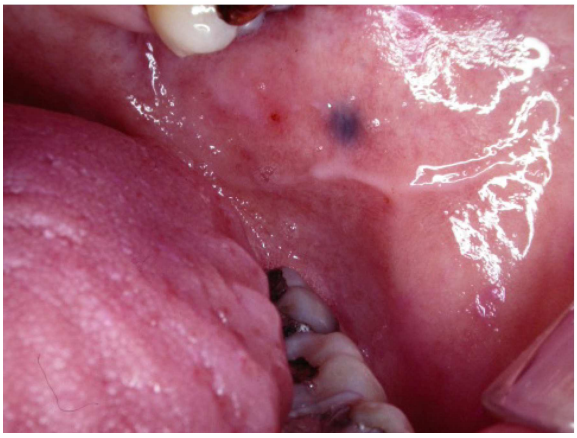
Etiology: Silver amalgam from a heavily restored tooth undergoing extracation may splinter and pieces may embed in the extraction socket. Also, the superficial trauma secondary to removal of cotton sponges and gauze following a dental restorative procedure may result in embedding of a few pieces of silver amalgam in the connective tissue.

Treatment: No treatment is required but if the clinical diagnosis is in doubt, a scalpel biopsy may be performed to ensure complete removal.

Prognosis: There is little to no response by the host tissue to the embedded silver amalgam and the prognosis is excellent.

Differential diagnosis: The diagnosis of an amalgam tattoo is straight forward but often may require differentiation from other benign or potentially harmful lesions such as a benign pigment cellular nevus or even a melanoma.





2. Nicotine stomatitis

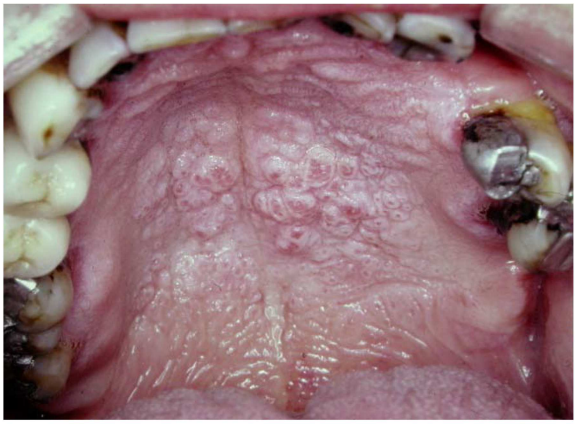
Description: This condition presents acutely as a diffuse whitening of the posterior hard palate with variable extension into the soft palate. The whitened mucosa often appears 'cracked' and demonstrates elevated 'crater-like' red points that represent the inflamed openings of ducts of minor mucous glands of the palate. The severity of the findings is highly variable and patients are usually asymptomatic.

Etiology: This condition is seen more commonly in pipe smokers and is more likely associated with the heat of combustion of the tobacco products rather than due to its nicotine content.

Treatment: No treatment is necessary because the condition fades when smoking and the associated heat generation is discontinued.

Prognosis: The prognosis is very good as the condition resolves upon withdrawal from the smoking habit. It does not have a premalignant potential.

Differential diagnosis: The history of acute onset, the clinical appearance of the lesion and the habit of smoking are diagnostic for this condition of the palate. A differential diagnosis may include palatal petechiae and inflammatory papillary hyperplasia.



3. Irritation fibroma / traumatic fibroma

Description: The irritation or traumatic fibroma is considered to be a reactive proliferation of collagenous connective tissue. Most traumatic fibromas are 1cm or less in size and are most

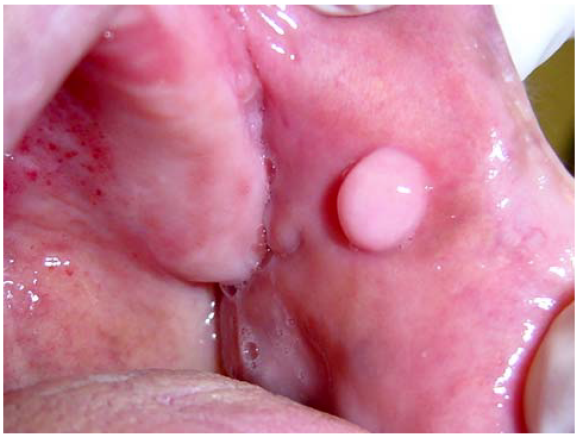
frequently located at the anterior buccal mucosa followed by the anterior lateral tongue although other areas may be infrequently affected. They are slow growing, asymptomatic masses often an annoyance to the patient. Non-ulcerated fibromas are pink to pale pink in color. They may be polypoidal with a pedunculated base or sessile and dome-shaped. On palpation, they may feel soft or firm in consistency depending upon the density of the collagen.

Etiology: The oral irritation/traumatic fibroma is not considered to be a true neoplasm. It forms in response to trauma from the teeth, sharp end of an appliance, or other source of irritation.

Treatment: Excisional biopsy is curative.

Prognosis: The prognosis is excellent with no inherent tendency to recur.

Differential diagnosis: 'soft' fibromas of the buccal mucosa may resemble a lipoma. Sclerosing mucoceles of the lower lip may be mistaken for fibromas and a fibroma of the gingiva may resemble a peripheral ossifying fibroma.



4. Pyogenic granuloma

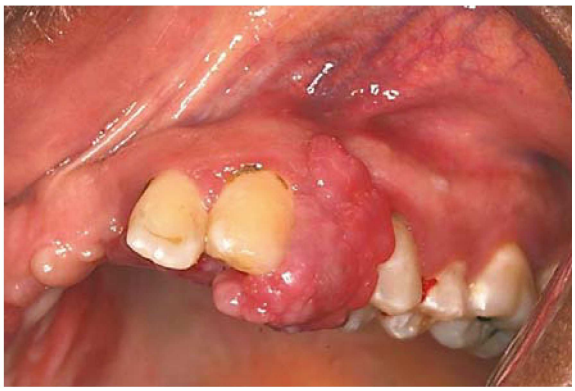
Description: The pyogenic granuloma is a painless, slow growing, organized mass of granulation tissue. Pyogenic granulomas may affect any skin or mucosal surface. Oral pyogenic granulomas present as red, well- circumscribed masses of the gingiva mostly. Infrequently, they may also affect the tongue, buccal mucosa and the lower vermilion. The incidence of pyogenic granulomas is higher during pregnancy and has led to the usage of the term 'pregnancy epulis'. The arrangement of the blood vessels as noted histopathologically, has also led to the usage of the term 'lobulated capillary hemangioma', especially by pathologists.

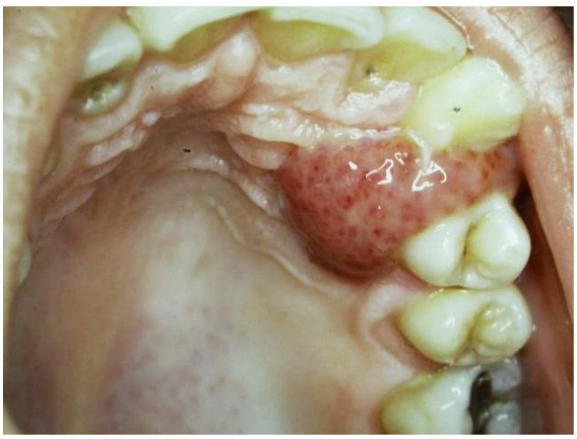
Etiology: The pyogenic granuloma arises due to chronic friction at a site easily affected by inflammation and formation of granulation tissue. The angiogenic potential of estrogen, especially during pregnancy is the reason for a higher incidence of pyogenic granulomas in response to subgingival calculus or a sharp edge of a prosthesis or restoration in women.

Treatment: Excisional biopsy followed by the removal of any chronic irritant is usually curative.

Prognosis: Following complete removal and elimination of any potential irritant, the prognosis is excellent. A recurrence rate of approximately 15% has been reported.

Differential diagnosis: The differential diagnosis of a gingival pyogenic granuloma includes a peripheral giant cell granuloma and other benign peripheral odontogenic and soft tissue tumors of the gingiva.





5. Peripheral ossifying fibroma

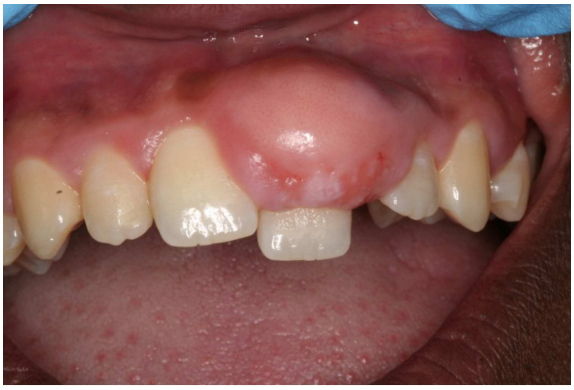
Description: This is an asymptomatic, slow growing, benign, well circumscribed growth of the gingiva. It is seen more commonly in young adult females. It is pale pink in color and firm to hard in consistency.

Etiology: It is thought to arise from the periodontal ligament due to chronic irritation, probably from subgingival calculus.

Treatment: Excisional biopsy followed by removal of any source of chronic irritation is curative. An excisional biopsy requires the removal of the stalk of the lesion from the periodontal ligament space.

Prognosis: The prognosis is excellent and under treatment results in a recurrence rate of approximately 15%.

Differential diagnosis: A differential diagnosis of this benign gingival mass includes inflammatory fibrous hyperplasia/fibroma of the gingiva, osteoma and other benign peripheral odontogenic tumors.



6. Peripheral giant cell granuloma

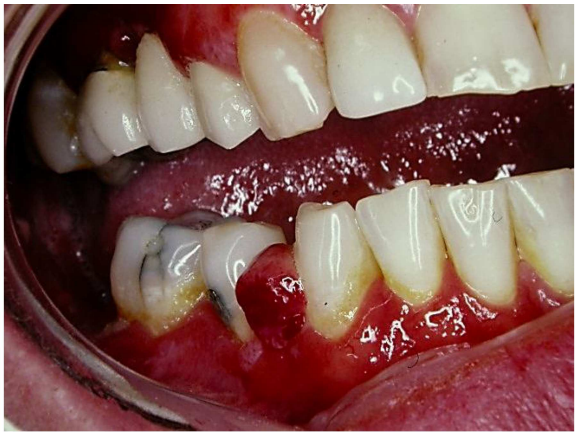
Description: The peripheral giant cell granuloma presents as a painless, well circumscribed, reddish purple mass usually less than 1cm in size. It is found on the gingiva and usually anterior to the molar teeth. Demographically, the peripheral giant cell granuloma is seen more commonly in young female patients.

Etiology: It is suggested that the peripheral giant cell granuloma arises from the periodontal ligament space and the giant cells are of odontoclastic origin.

Treatment: An excisional biopsy including the origin of the lesion within the periodontal ligament space is the treatment of choice.

Prognosis: The prognosis is excellent. A recurrence rate of approximately 15% is associated with incomplete removal.

Differential diagnosis: Lesions that are more apically positioned may be mistaken for a parulis. Other lesions in the differential diagnosis include the pyogenic granuloma and a focal inflammatory fibrous hyperplasia.



7. Mucocele

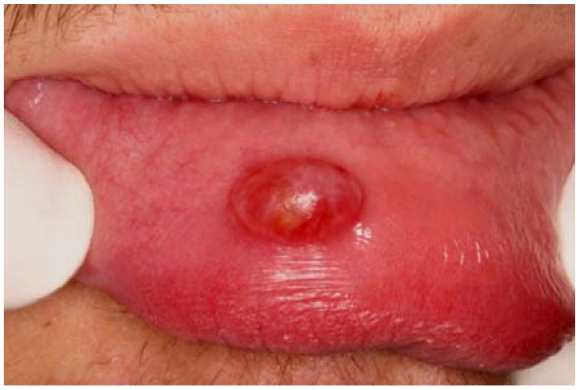
Description: This lesion presents as a dome-shaped, fluid-filled mass of the lower lip. Superficial lesions are fluctuant and translucent while deep seated lesions may have a normal color. There is often a history of the swelling increasing in size and rupturing. Superficial mucoceles may affect other parts of the oral cavity where minor mucous glands are present. A mucocele of the floor of the mouth is known as a ranula. It presents as a unilateral swelling elevating the tongue to the opposite side.

Etiology: Mucoceles are a result of mucus extravasation secondary to traumatic severance of the mucus duct. The mucus pools within the connective tissue resulting in a fluctuant, sometimes blood tinged blister.

Treatment: Excision of the mucocele with surgical margins deep enough to include the lobules of minor mucus glands contributing to the mucus pool.

Prognosis: Very good. Recurrence is related to a simple decapitation of the blister and mucus discharge and incomplete removal of the minor mucus gland lobules.

Differential diagnosis: The diagnosis of a mucocele is straight forward. Benign salivary tumors tend to affect the upper lip mostly. A blood tinged mucocele may resemble a varicosity. Ranulas are soft unilateral swellings of the floor of the mouth while dermoid cysts are midline lesions and feel doughy in consistency.



8. Smoker's melanosis

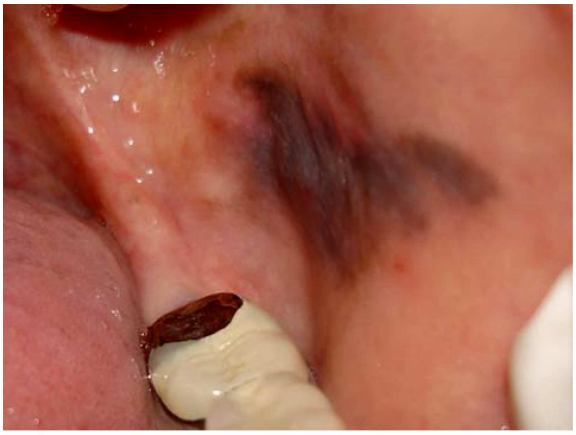
Description: Melanin pigmentation in response to heavy tobacco smoking is prominent over the anterior facial gingiva. Pipe smoking results in pigmentation of the commissures and buccal mucosae. The pigmentation is diffuse, macular and fairly uniform in intensity. The pigmentation may be intense in ethnically dark skinned races. Smoker's melanosis is the most common cause of oral pigmentation in otherwise fair skinned races.

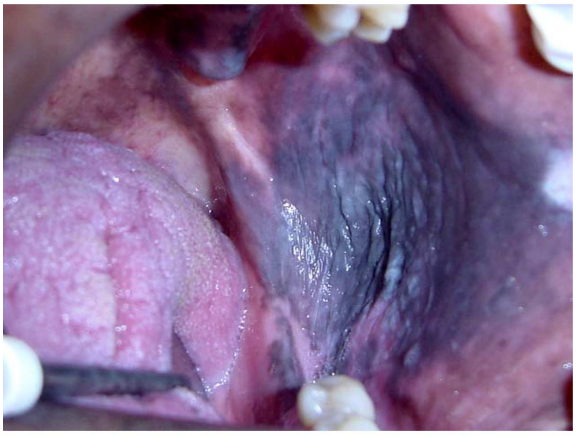
Etiology: Melanocytes are stimulated to produce melanin in the presence of polycyclic aromatic hydrocarbons like nicotine and benzopyrene (tobacco combustion products).

Treatment: Smoking cessation results in gradual fading of the pigmentation over a period of months to several years.

Prognosis: The pigmentation may be esthetically unappealing. Irregular pigmentation, ulceration with or without swelling may necessitate a biopsy. In itself, smoker's melanosis is harmless.

Differential diagnosis: The clinical appearance combined with the smoking history is diagnostic. Occasionally, other causes of oral pigmentation including reactive chronic trauma associated pigmentation, drug-induced pigmentation, pigmentation in a setting of Addison's disease, hemochromatosis, Peutz-Jeghers syndrome, and Café-au-lait pigmentation of neurofibromatosis or fibrous dysplasia may need to be considered in the differential diagnosis.





9. Morsicatio buccarum / labiorum/ linguarum (chronic cheek, lip, and tongue chewing)

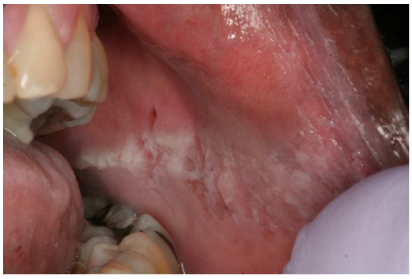
Description: Chronic cheek chewing may be unilateral or bilateral. It may be associated with a lip chewing and a tongue chewing habit. The surface of the buccal mucosa appears rough, shredded and irregular instead of smooth and moist. The changes are more prominent along the occlusal plane and towards the anterior portions of the buccal mucosa. The lower lip is chronically chewed more than the upper lip. Heavy chewing may result in erosion or ulceration.

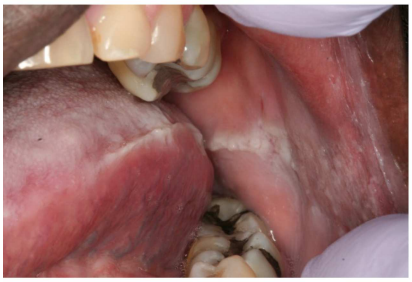
Etiology: A chronic cheek and/or lip chewing habit is subconscious and involuntary and a manifestation of stress or a psychological condition. The clinical appearance is due to shredding of surface layers of parakeratin often colonized by bacteria.

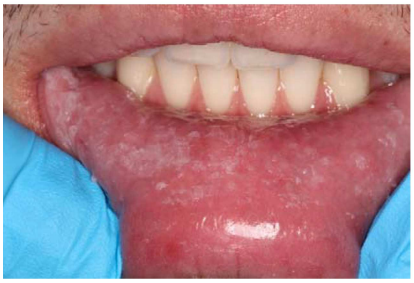
Treatment: Acrylic shields prevent the access to the mucosae and result in healing.

Prognosis: The changes associated with chronic cheek, lip and tongue chewing may be esthetically displeasing but are harmless and the prognosis is excellent.

Differential diagnosis: The diagnosis of morsicatio buccarum/linguarum or morsicatio labiorum is straight forward. Occasionally it may require differentiation from oral cinnamon reaction, a localized allergic response to artificial cinnamon flavoring agents found in many food products as well as oral hygiene products. An isolated lesion affecting the lateral tongue in a HIV positive individual may require differentiation from oral hairy leukoplakia.







10. Chemical burn

Description: Patients with this condition may present with severe pain and burning. The symptoms are of recent onset, typically under 24-48 hours. Careful questioning reveals either

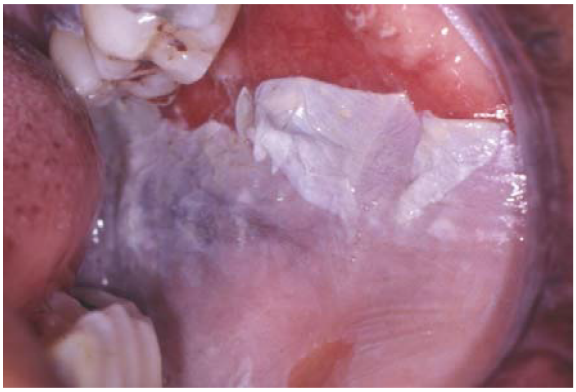
self-medication in the form of placement of an aspirin tablet in the vestibule adjacent to a painful tooth or a very recent visit to a dental clinic. Careful examination of the affected oral mucosa reveals a pseudomembranous white lesion of variable extent. Underlying the pseudomembrane, the mucosa is erythematous and tender.

Etiology: self-medication in the form of aspirin, sodium perborate, hydrogen peroxide, rubbing alcohol, eugenol, turpentine, etc results in oral mucosal chemical burns. Many of the materials used in the dental office are also caustic and prolonged contact with the mucosa may result in chemical burns. Examples of such agents include silver nitrate, sodium hypochlorite, chromic acid, formocresol, dental cavity varnishes, acid etch, etc.

Treatment: Prevention is the best way to avoid chemical burns. Patient education prevents self-inflicted burns. Minimizing contact of dental materials with the mucosa and the use of dental rubber dam can reduce the incidence of chemical burns of the oral mucosa. Upon discontinuation of the offending agent, reepithelization occurs from within 1 to 2 weeks' time. In the meanwhile, salivary substitutes and emollients like hydroxypropyl cellulose help protect the affected mucosa. Topical anesthetic rinses or viscous lignocaine applications will provide temporary pain relief. The pseudomembrane itself provides cover to the exposed connective tissue and should be retained as far as is possible.

Prognosis: The prognosis is excellent if the offending agent is removed.

Differential diagnosis: The diagnosis of this condition is straight forward and is dependent on the history and the clinical appearance of the affected mucosa.



11. Salivary stones (sialolithiasis)

Description: These are calcified masses of varying size found either within the duct or the substance of the salivary gland itself. Salivary stones within the Wharton's duct of the submandibular salivary gland are the commonest. The parotid (Stensens) duct is infrequently

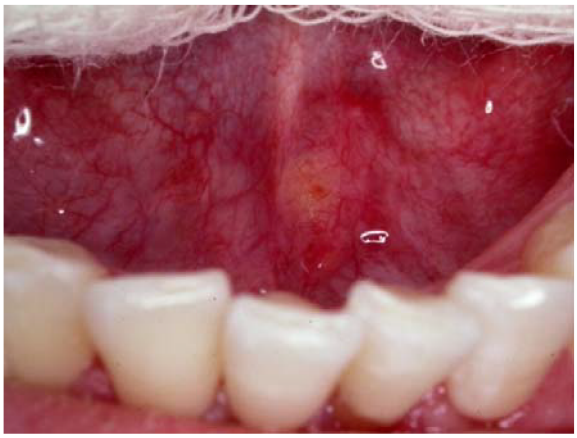
affected. Sialoliths may also form within the minor salivary glands of the oral cavity. If ductal obstruction is moderate to severe, patients may complain of pain and swelling of the affected gland especially at meal times. The hard mass may be superficially located including at the orifice of the duct or may be deep and felt by bimanual palpation. Well calcified sialoliths are easily demonstrable on radiographs.

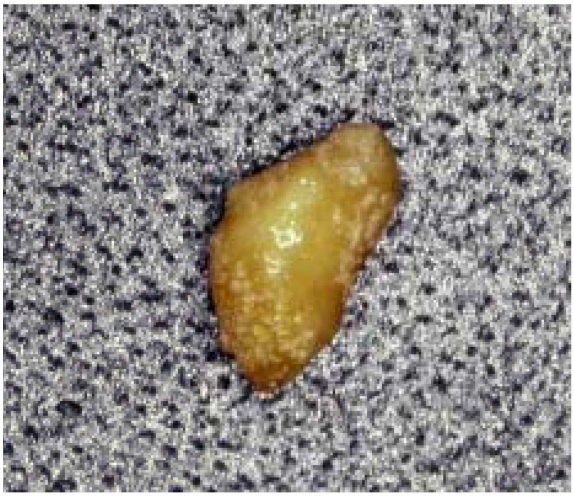
Etiology: Salivary stones form by concentric deposition of calcium salts around a nucleus of necrotic debris including ductal epithelial cells, bacteria, mucus, food debris and other vegetable particulate matter entrapped within the ductal system. Over a period of time, the mass hardens and enlarges sufficiently to register on x-rays, be palpable and cause symptoms of obstruction.

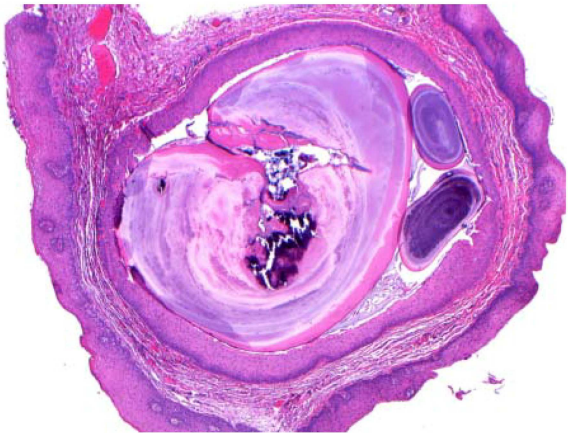
Treatment: Small, superficially placed stones may be gently 'milked' out of the ductal system. Bulkier stones will require surgical removal. Chronic swelling and infection of the affected gland may also necessitate removal of the gland.

Prognosis: Removal of the stone and restoration of the flow of saliva results in recovery of the patient. The prognosis is good.

Differential diagnosis: Obstruction of the flow of saliva may be secondary to tumors of salivary gland origin. These are soft tissue masses and do not calcify. Other rare causes of obstruction of salivary flow may include stricture formation secondary to radiation, sclerosing sialdenitis, Sjogren's syndrome, granulomatous disease and infections of the salivary gland.







12. Squamous papilloma

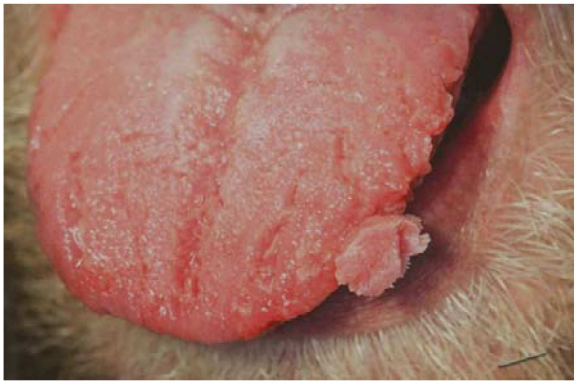
Description: A squamous papilloma is a solitary, benign, wart-like neoplasm of the surface epithelium. While any part of the oral cavity may be affected, the tongue, lips and the soft palate are more frequently involved. The squamous papilloma may rarely exceed over a centimeter in size. The stalk-like origin gives it a pedunculated appearance and the surface may show numerous well keratinized finger-like projections or may show numerous rounded projections giving it a 'cauliflower-like' appearance.

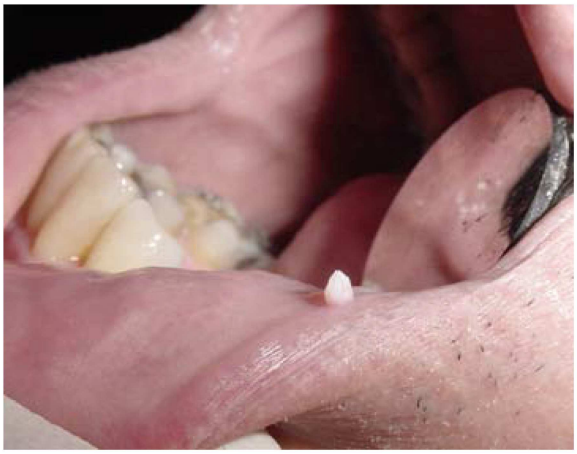
Etiology: The exact etiology is not known but the role of human papilloma virus strains 6 and 11 has been implicated in its causation.

Treatment: Excisional biopsy using a scalpel, laser or electrocautery is curative.

Prognosis: Long standing, untreated squamous papillomas do not enlarge appreciably and do not undergo malignant transformation. Removal is recommended for either esthetic reasons or to prevent habitual traumatization. Multiple lesions are uncommon because of the long incubation period and the very low infectivity of HPV 6 and 11. Prognosis is therefore good.

Differential diagnosis: Heavily keratinized papillomas may be confused with verruca vulgaris (common wart). Cutaneous common warts may be present along with oral verruca vulgaris. Squamous papillomas may also resemble the venereal wart condyloma acuminatum. The condyloma is a sexually transmitted condition and is contagious. Autoinnoculation results in multiple lesions. Condylomas are less keratinized and appear pink in color.





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Chapter 8

Dry Mouth and Associated Conditions

Description: Dry mouth or xerostomia can be defined as a symptom due to less saliva production or not enough saliva in the mouth. Patients can complain that they feel like having sand in the mouth. This is an oral problem that affects populations worldwide. Saliva maintains the oral cavity ph and buffering capacity. This prevents the formation of an acidic environment that would favor tooth demineralization. In addition, saliva provides a supply of calcium and phosphate, two key substrates for dental remineralization. Saliva contains proteins such as antibodies that enhance local defenses against infection. One of the most important functions of saliva is to provide moistening of the oral mucosa and to promote and facilitate food digestion. Therefore, less or no saliva in the mouth can severely affect oral functions and decrease quality of life.

There are two main reasons for the development of dry mouth. Patients can have a decrease in saliva secretion or develop pathological dysfunctions of the salivary glands.

Decrease in saliva secretion: this can be a physiological effect of sleep and aging. However, more severe factors include the use of medication, diabetes and other hormonal changes, and radiation therapy involving the salivary glands **(this will be specifically discussed in Chapter 11)**.

Pathological dysfunctions of the salivary glands: infections, tumors and autoimmune diseases such as Sjoegren's syndrome are the main conditions affecting saliva secretion.

How to diagnosis dry mouth? The diagnosis of dry mouth can be suspected when taking patient history. Patients commonly complain that there is a sensation of having sand in the mouth, mouth burning, lack of taste, difficulty in chewing and swallowing, difficulty in sleeping, and having to drink water all day long. Table 1 shows examples of questions that can be asked during patient interview.

Do you sip liquids to aid in the swallowing of foods?
Does your mouth feel dry when eating a meal?
Do you have difficulty swallowing any foods?
Does the amount of saliva in your mouth seem too little?

Table 1. Specific questions regarding the presence of dry mouth.

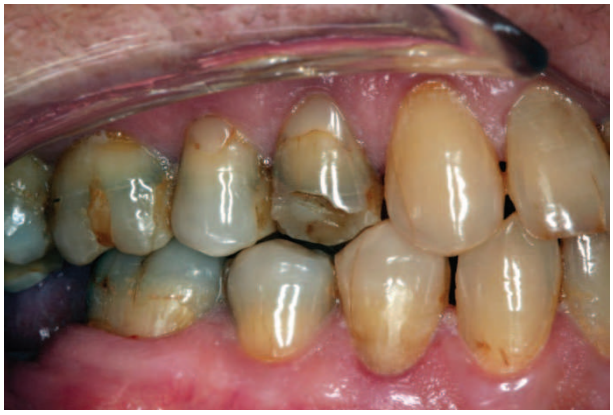
In addition to the history, clinical examination can also help in finding signs that would indicate dry mouth:

Ropey saliva
Dry tissues, pale or red, and atrophic
Tongue may be devoid of papillae, fissured, inflamed
Multiple cervical caries

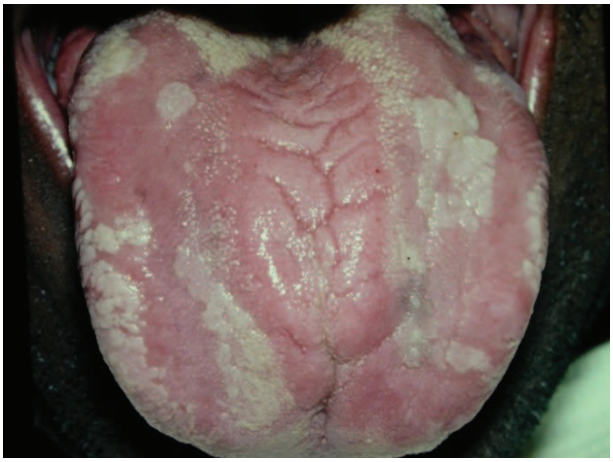
The quantity and quality of the saliva can also be helpful. One can determine salivary gland output and observe the characteristics of the salivary secretion, looking for color, consistency (serous or mucous), and turbidity. Normal salivary secretion is clear and mostly fluid. Changes in consistency and turbidity could indicate the presence of infection.

Whole Saliva Flow Rates (ml/min)		
	Normal Flow Rates	**Abnormal Flow Rates**
Unstimulated (resting) Whole Saliva*	0.3-0.4	<0.1
Stimulated Whole Saliva*	1-2	<0.5
*Whole saliva is the total output of saliva from the major (parotid + submandibular + sublingual) and minor salivary glands plus the gingival crevicular fluid		

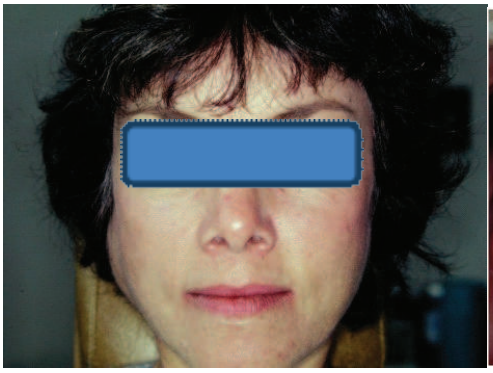
Excessive buccal caries in a patient taking xerostomic medication and with poor oral hygiene and cariogenic diet

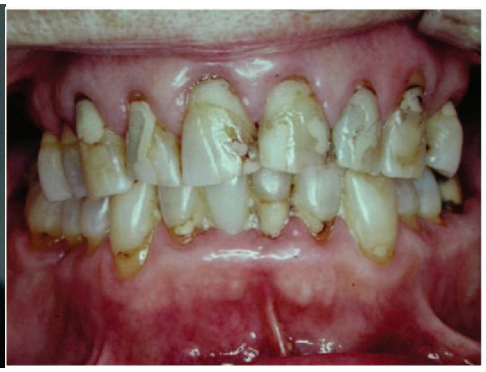


Tongue depapillation in a patient with undiagnosed diabetes and dry mouth. There was also presence of candidiasis



Sjoegren's patient. Notice swelling of the right parotid gland and severe dryness of the oral tissues with evidence of excessive decay





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Chapter 9

Pre-Cancer and Cancer

Actinic cheilitis

Tobacco pouch keratosis (Smokeless tobacco lesion, snuff dipper's lesion)

Leukoplakia

Erythroplakia

Speckled leukoplakia (Erythroleukoplakia)

Proliferative verrucous leukoplakia

Squamous cell carcinoma

1. Actinic cheilitis (actinic cheilosis)

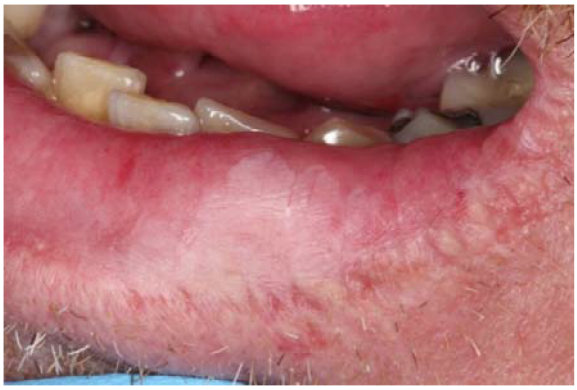
Description: Actinic cheilitis is an irreversible precancerous change of the lower lip vermillion. Adult males are the predominant demographic group affected and fair-skinned individuals or those who have an outdoor occupation are at greatest risk. Early changes include the development of blotchy, pale areas and an indistinct margin between the lip vermillion and skin. Rough, scaly areas, leukoplakia, and ulceration may develop as the lesion progresses.

Etiology: Chronic sun exposure

Treatment: Patients should be encouraged to use lip sunscreens or broad-brimmed hats to protect the lip from further damage when outdoors. Scaly, ulcerated, or indurated areas should be biopsied to evaluate for the possibility of transformation to squamous cell carcinoma. Extensive involvement may require vermillionectomy, a procedure where the entire lip vermillion is removed and the labial mucosa is pulled forward.

Prognosis: Good. Actinic cheilitis behaves similarly to actinic keratosis of the skin. Over time, a small proportion of lesions will undergo malignant transformation, which is generally amenable to surgical excision.

Differential diagnosis: Cheilocandidiasis, lip chapping, traumatic ulcer



2. Smokeless tobacco keratosis (tobacco pouch keratosis, snuff dipper's lesion)

Description: Smokeless tobacco keratosis is a reversible alteration of the oral mucosa in immediate contact with a smokeless tobacco product (chewing tobacco, moist snuff, dry snuff). The characteristic appearance is a gray/white mucosal discoloration with a wrinkled or fissured surface texture. The most common locations are the lower labial or buccal vestibule, where the product is held. Gingival recession around teeth in the area of contact is frequently seen.

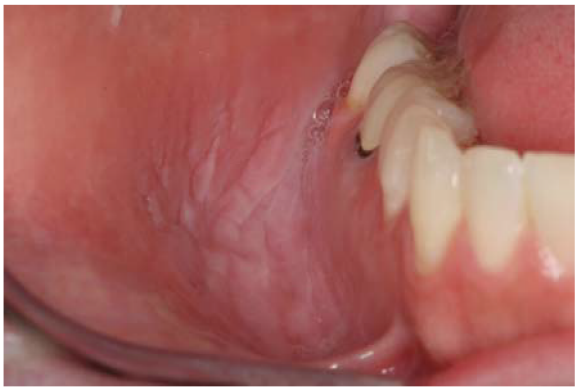
Etiology: Reactive mucosal change in response to habitual use of smokeless tobacco products.

Treatment: The patient should be encouraged to discontinue smokeless tobacco use. If the patient is unwilling to quit, moving the product to a different intraoral location is recommended. Lesions should resolve after two weeks of having no smokeless tobacco product in contact with the site. Biopsy is recommended for persistent lesions.

Prognosis: Controversial. The potential dangers of chronic smokeless tobacco use have been debated for many years. While verrucous carcinoma and squamous cell carcinoma have been known to develop within smokeless tobacco keratosis, the risk of developing oral cancer is often overstated. An extensive review of published studies demonstrated a significant but weak association between smokeless tobacco use and oral cancer. When the data was limited to studies published since 1990 and those which adjusted for smoking, the relative risk of developing oral cancer was close to 1.0 compared to individuals who had never used smokeless tobacco. Based on the available evidence, modern day smokeless tobacco products seem to

impart a much lower risk than those used in the past in the U.S. and the associated risk for oral cancer is undoubtedly less than that from smoking.

Differential diagnosis: Leukoplakia, hyperkeratosis



3. Leukoplakia

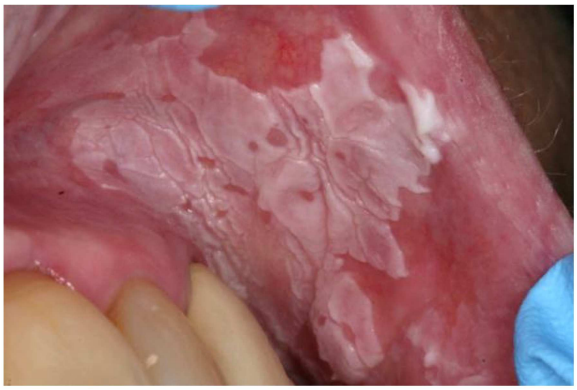
Description: Leukoplakia is defined as an adherent white plaque that cannot be explained or diagnosed clinically as any other pathologic entity. It is seen most often in adults over age 40 and may occur in any intraoral location. Leukoplakia is a clinical term used only after exclusion of other characteristic white lesions. While it does not imply a specific histopathologic diagnosis, leukoplakia is considered to be potentially precancerous. Approximately 85% of all oral precancerous lesions present as a leukoplakia.

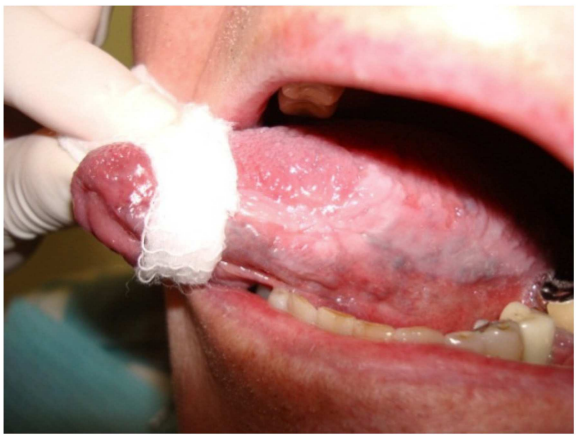
Etiology: The precise cause is unknown and probably varies among individuals. Many potential causes have been proposed such as trauma, smoking, alcohol, and sanguinaria.

Treatment: Because leukoplakia is a clinical term only, biopsy is required to establish a definitive diagnosis. The histopathologic findings are the most important factor in determining the appropriate treatment. The size of the lesion, location, and other patient factors may also be considered in guiding the treatment protocol. Lesions on the tongue, lip vermillion, and floor of mouth account for over 90% of leukoplakias that are dysplastic or malignant. Unfortunately, no diagnostic test exists at this time to help identify which lesions are likely to transform to squamous cell carcinoma and which are not. The management of mild epithelial dysplasia is controversial, with some experts advising close clinical observation, particularly for large lesions that would be difficult to excise. Leukoplakias which demonstrate epithelial dysplasia that is moderate or worse in severity should be removed entirely if possible. Long-term clinical follow-up is important because leukoplakia often recurs or may develop in a new location.

Prognosis: The prognosis of leukoplakia is dependent on both the clinical features and the histopathologic findings. Thin, homogenous lesions are less likely to undergo malignant transformation than those that are more thickened, granular, or heterogeneous in appearance. Microscopically, the greater the severity of epithelial dysplasia, the greater the risk for malignant transformation. Valid data regarding the precise risk of transformation to squamous cell carcinoma may never be available as many leukoplakias are treated when diagnosed. Furthermore, a long-term study in which no treatment is given to patients with a precancerous lesion would be considered unethical.

Differential diagnosis: Frictional hyperkeratosis, hyperplastic candidiasis, chemical burn, lichen planus, squamous cell carcinoma





4. Erythroplakia

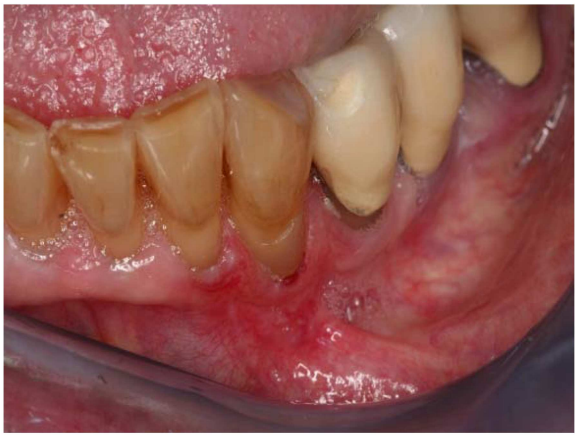
Description: Erythroplakia is defined as a red patch that cannot be explained or diagnosed clinically as any other pathologic entity. It is seen most often in middle-aged or older adults and occurs most frequently on the tongue, floor of mouth, and soft palate. Erythroplakia is much less common than leukoplakia, but much more likely to be precancerous.

Etiology: Multiple potential etiologic agents due to several diseases having the clinical appearance of an erythroplakia. For lesions that prove to be precancerous or cancerous on biopsy, the etiology and risk factors are thought to be similar to those of oral squamous cell carcinoma.

Treatment: If there is suspicion for a reactive lesion, removal of any potential etiologic factors and follow-up in 2 weeks is acceptable. Persistent lesions or erythroplakia in a high-risk location (floor of mouth, ventral or lateral tongue) should be biopsied. Similar to leukoplakia, the treatment is determined by the histopathologic findings.

Prognosis: Guarded. As with leukoplakia, the prognosis depends on the definitive microscopic diagnosis. However, 90% of oral erythroplakias demonstrate severe dysplasia, carcinoma in situ, or superficially invasive squamous cell carcinoma at the time of biopsy.

Differential diagnosis: Non-specific mucositis, erythematous candidiasis, anemia, vascular lesions, submucosal hemorrhage



5. Speckled leukoplakia (erythroleukoplakia)

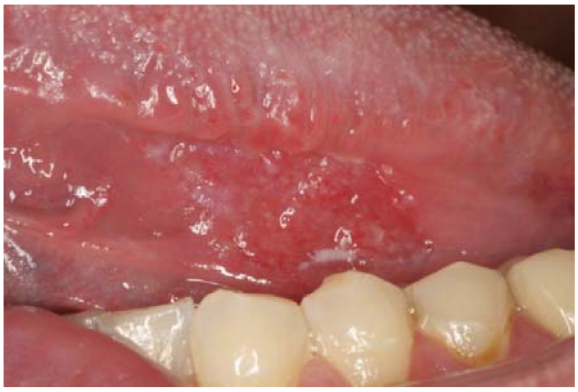
Description: Speckled leukoplakia is a mixed red-and-white precancerous lesion considered to be a type of leukoplakia. Red areas within a leukoplakia often represent locations where the epithelial cells are so poorly-differentiated that they are no longer able to produce keratin.

Etiology: Same etiology and risk factors as squamous cell carcinoma

Treatment: Same as erythroplakia. Speckled leukoplakia often demonstrates significant epithelial dysplasia when biopsied.

Prognosis: Guarded. As with leukoplakia and erythroplakia, the prognosis depends on the definitive microscopic diagnosis.

Differential diagnosis: Lichen planus, trauma



6. Proliferative verrucous leukoplakia

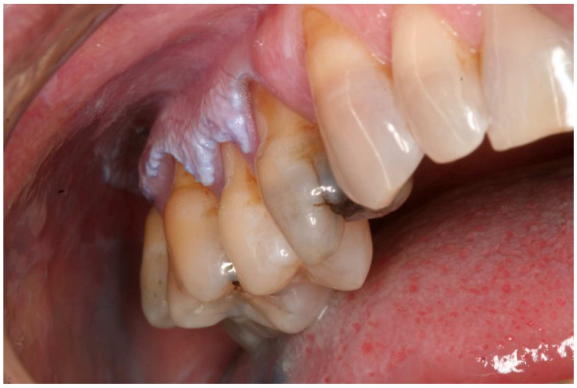
Description: Proliferative verrucous leukoplakia (PVL) is a precancerous condition characterized by the development and progression of multiple intraoral leukoplakias. Any combination of locations may be affected, but the buccal mucosa, tongue, and gingiva are common sites of involvement. Lesions begin as thin leukoplakias that slowly evolve a thicker, verrucous surface texture. PVL is three to four times more common in females compared to males, with most patients diagnosed in the 7th decade.

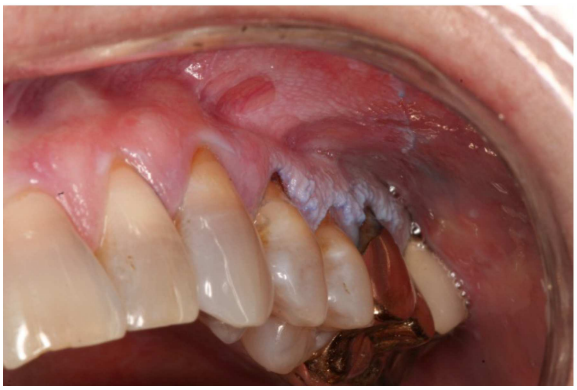
Etiology: Unknown. The majority of PVL cases are not associated with tobacco smoking or other suspected risk factors for traditional leukoplakia and squamous cell carcinoma.

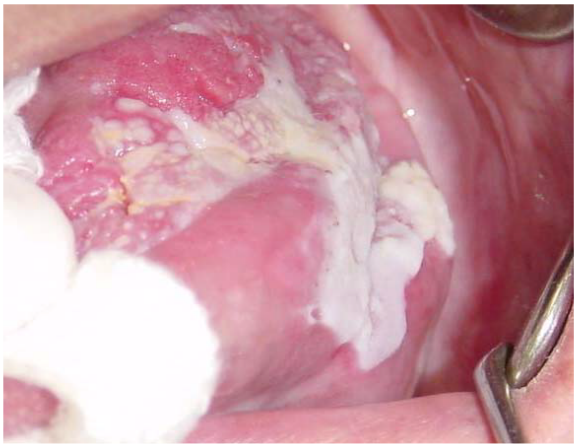
Treatment: Due to the multifocal and often extensive nature of PVL, complete removal of all lesions is not feasible in most cases. Periodic biopsy to assess for dysplasia is appropriate, with selective excision of areas that appear most suspicious clinically. Frequent, long-term re-evaluation is critical due to the persistent and progressive nature of the condition.

Prognosis: Poor. A recent review found that 74% of patients developed squamous cell carcinoma after an average follow-up period of 7-8 years.

Differential diagnosis: Lichen planus, hyperplastic candidiasis, white sponge nevus







7. Squamous cell carcinoma

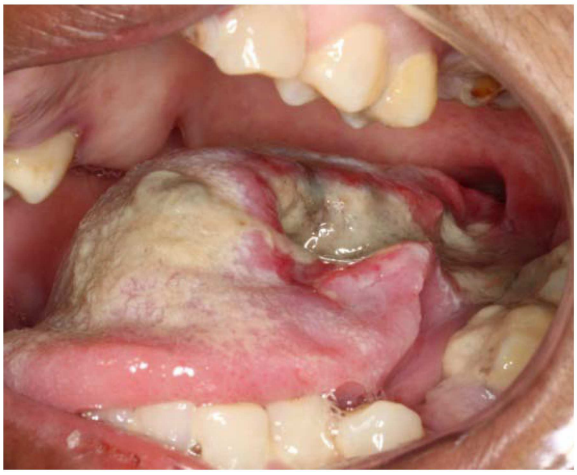
Description: Squamous cell carcinoma (SCC) is a malignancy derived from the surface epithelial cells that line the oral cavity. SCC accounts for over 90% of oral cancers. The risk of developing SCC increases with age and is over twice as common in males compared to females. Early lesions are usually asymptomatic. The lateral and ventral tongue are the most frequent sites of involvement, followed by the floor of mouth, soft palate, gingiva, and buccal mucosa. SCC is variable in appearance. The color may be red, white, or mixed and lesions can be exophytic or endophytic in nature. Ulceration is common and most lesions will feel firm upon palpation (indurated).

Etiology: Multifactorial. Several known or suspected risk factors have been identified. Tobacco smoking is a major etiologic agent in the majority of SCCs. Alcohol abuse, x-irradiation, and genetic mutations have also been implicated. SCC of the lip vermillion is similar in pathophysiology to cutaneous SCC, with sunlight being the primary cause. Human papillomavirus (HPV) infection has recently been established as an independent risk factor for SCC of the oropharynx (tonsils and base of tongue), but not for cancers of the oral cavity. High-risk HPV-16 or HPV-18 subtypes can be identified in the majority of oropharyngeal SCCs. Importantly, 20-25% of oral SCCs are not associated with any of the aforementioned risk factors.

Treatment: SCC of the lip vermillion is treated by surgical excision. The treatment of other oral SCCs is guided by the stage, which is a quantification of the size of the tumor and the extent of its spread (if any) to regional lymph nodes or distant locations. PET/CT imaging is performed to help determine the stage. Surgery, chemotherapy, and radiation therapy (or any combination of these) may be performed, as well as neck dissection if regional lymph node metastasis is suspected. Targeted drug therapy that affects only the cancer cells is being used with increasing frequency and causes much fewer side effects than traditional chemotherapy.

Prognosis: SCC of the lip vermillion behaves similarly to SCC of the skin and has a very good prognosis. The prognosis of other oral SCCs is highly dependent on the stage and also somewhat on location. The overall 5-year survival rate (considering all stages) is estimated to be around 60%. Oral SCC and its treatment can also cause significant morbidity among surviving patients such as facial disfigurement, speech problems, and difficulty eating.

Differential diagnosis: Histoplasmosis, tuberculosis, syphilis, traumatic ulcer



Additional reading

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American Cancer Society website (www.cancer.org)

Chapter 10

AIDS/HIV Associated Lesions and Management

Candidiasis: pseudomembranous, erythematous, angular cheilitis, candida leukoplakia

Kaposi's sarcoma

Oral hairy leukoplakia

Linear gingival erythema

HIV periodontitis

Necrotizing ulcerative gingivitis

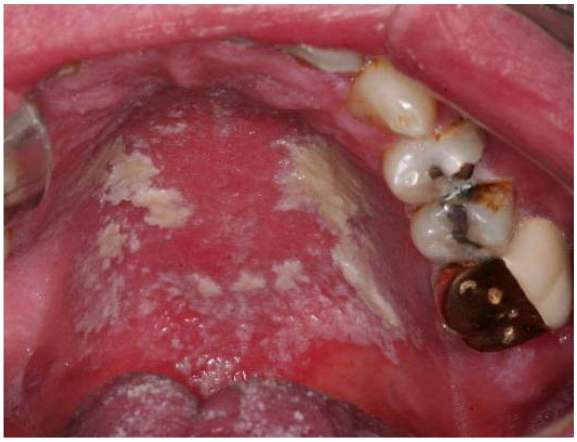
Major aphthae

Infection with the human immunodeficiency virus (HIV) is now a well-recognized chronic disease that leads to severe immunosuppression and acquired immunodeficiency syndrome (AIDS). Since the early days when the disease was first recognized, one of the common complications secondary to the immunosuppression was the presence of a variety of oral lesions and conditions that required constant care. Patients had an increased load of circulating viruses and their lymphocytes, characterized by the phenotype CD4, were being destroyed by the virus and presented in the peripheral blood in very low numbers. With the advent of a combination of antiviral drugs called highly active antiretrovirus therapy or HAART, it became possible to partially restore immunocompetence by decreasing the viral load and increasing the number of circulating CD4 cells. With this advance in therapy, common oral lesions observed in this patient population became more difficult to find, as long as the patients were responding well to HAART. However, there are still many infected individuals who have not yet been diagnosed with HIV and individuals who fail HAART who could develop oral lesions. Therefore, it is very important that the clinician be aware of some of the common oral complications of HIV infection and AIDS. Following we will describe some common oral complications of HIV infection and AIDS.

1. Pseudomembranous candidiasis

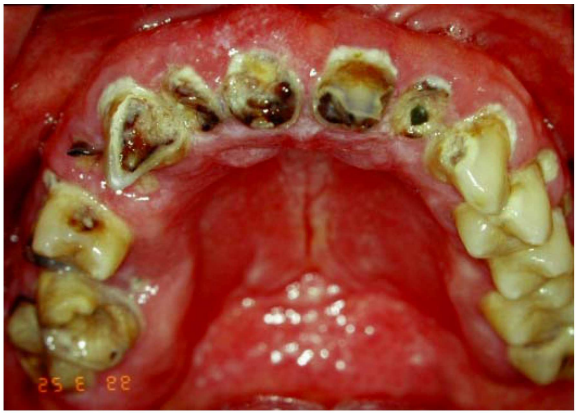
The patient in the photograph presented with white plaques of candida organisms on the palate. These areas can be scraped off with gauze and may leave an erythematous surface underneath. Patients may complain of burning, discomfort, lack of taste and difficulty in

swallowing if the infection migrates to the oropharynx. Symptoms may be the same in all forms of candidiasis.



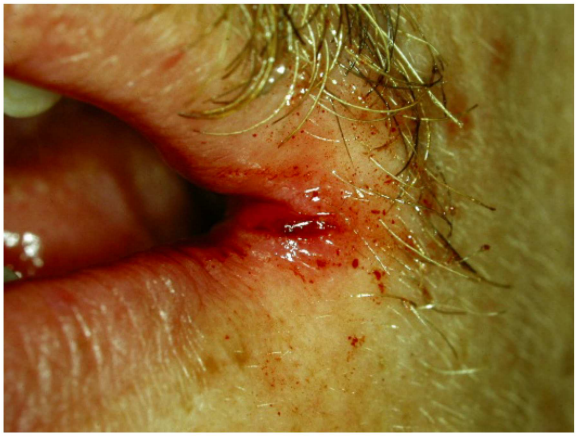
2. Erythematous candidiasis

Note the areas of erythema generalized over the oral mucous membranes.



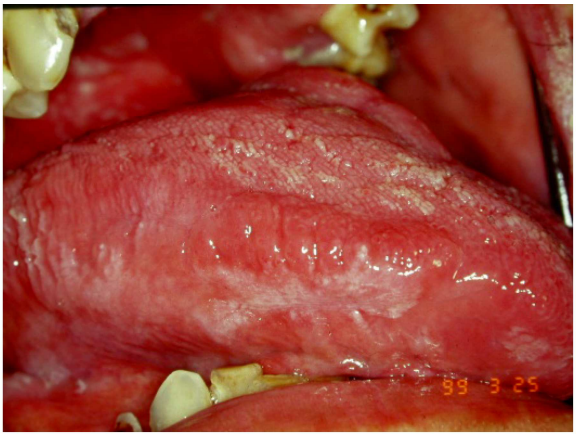
3. Angular cheilitis

Note the cracking of the lip commissure with bleeding as a result of the patient opening the mouth.



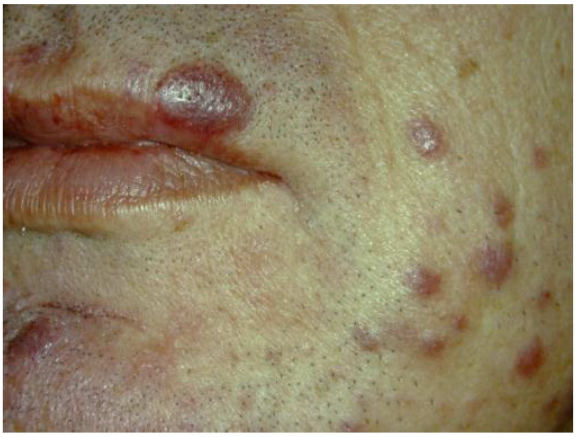
4. Candidal leukoplakia

Note the white plaque on the lateral border of the tongue. This lesion cannot be removed by scraping because the candida organisms are intra-epithelial and the infection is causing the growth of the keratin layer.



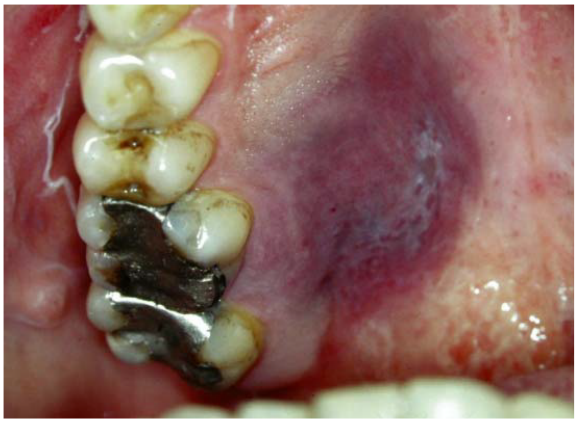
5. Kaposi's sarcoma (KS)

Note the multiple purple macules on the patient's face representing individual lesions of KS. The lesions are asymptomatic, but represent a serious cosmetic problem for the patient. Today we know that this tumor is associated with a herpes type of virus, the Human Herpes Virus type 8 (HHV-8) that can infect the entire body of the patient if not treated.



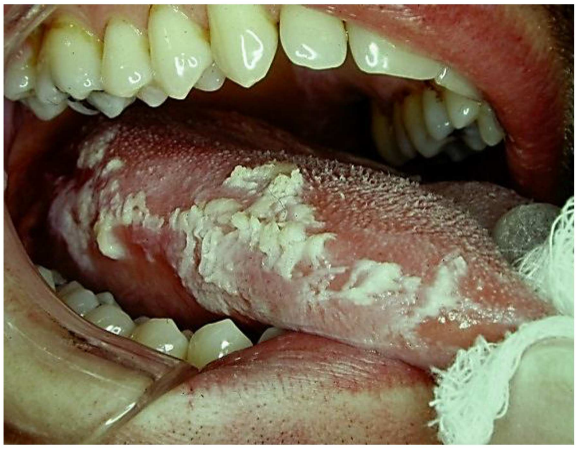
6. Kaposi's sarcoma of the palate

Notice the purple/bluish lesion on the hard palate. This lesion is asymptomatic and can acquire an exophytic growth pattern if not treated.



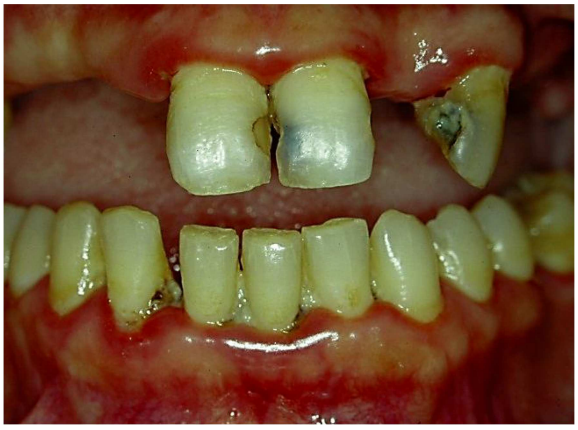
7. Oral hairy leukoplakia

Note the white lesion with a corrugated surface that does not scrape off and is associated with the Epstein Barr Virus. It is asymptomatic.



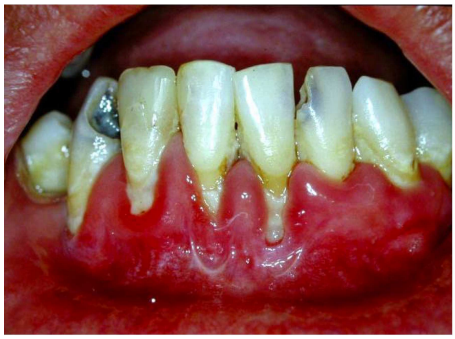
8. Linear gingival erythema

Note the linear erythema along the marginal gingival border. This clinical change is associated with the microbial flora of the periodontium, including *C.albicans*, in the HIV infected patient. It is asymptomatic.



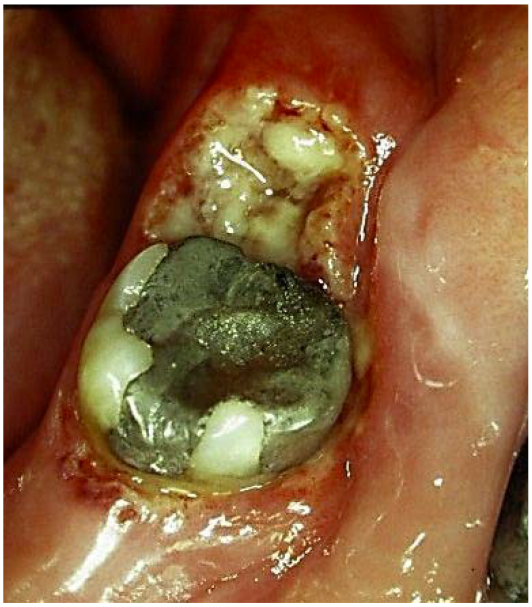
9. HIV periodontitis

Note severe periodontal destruction and inflammation associated with severe immunosuppression in an individual infected with HIV.



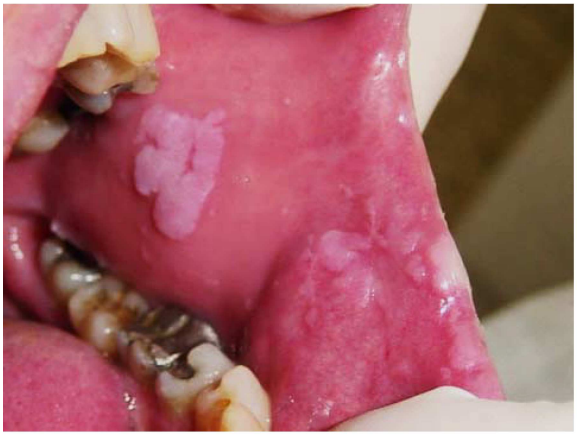
10. Necrotizing ulcerative gingivitis

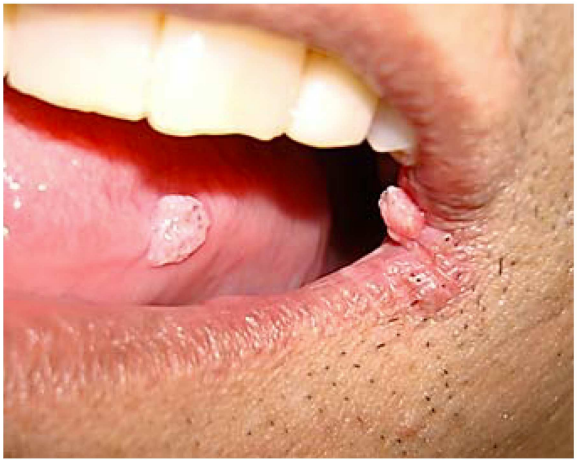
Note the severe and painful ulceration on the retromolar pad, extending to the marginal gingiva around the molar. Such lesions may be associated with many different bacterial microorganisms and require local debridment and topical and systemic antibiotics.



11. Condyloma acuminatum

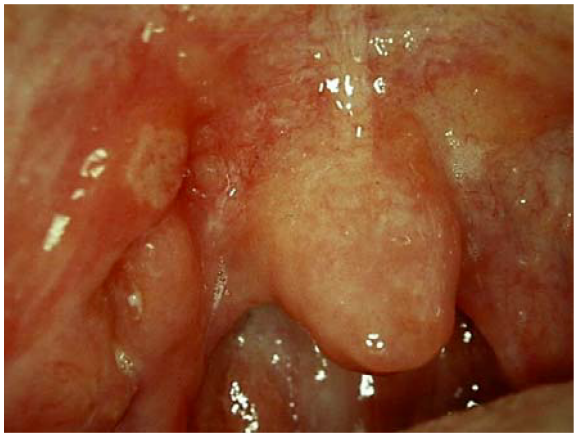
Note the different clinical presentations of condylomas associated with the Human Papilloma Virus (HPV). These lesions are sexually transmitted and associated with HPV types 6 and 11. Although asymptomatic, they require surgical excision and have a high recurrence rate.





12. Major aphthae

Individuals infected with HIV may develop aphthous-like lesions in the oral mucosa. Some may be of the major type, being over 1 cm in diameter. These lesions are very painful and can significantly alter the patient's quality of life. They require therapy with topical steroids if they are single or with systemic steroids when they are multiple.



Additional reading

http://www.hivdent.org/. Extensive information about HIV/AIDS and the oral cavity. Last accessed on Aug 13, 2013

Chapter 11

The Cancer Patient/Oral Complications of Cancer Therapy and Management

With the advance in early diagnosis and new modalities of therapy, cancer patients survive for many years after completion of therapy. However, cancer therapies are also toxic to normal tissues and organs in the human body and can cause both acute and chronic complications. Some of these complications occur in the oral cavity and require that the dental clinician be competent in diagnosing and managing the patients. Acute complications occur during cancer therapy and chronic complications are called late effects because they occur after completion of therapy and can persist for many years. There are different modalities of cancer therapies and some of them are used in combination. Depending on the toxicity of each therapy, the risk of oral complications can increase. Table 2 shows types of cancer therapy and the estimated risk of oral complications.

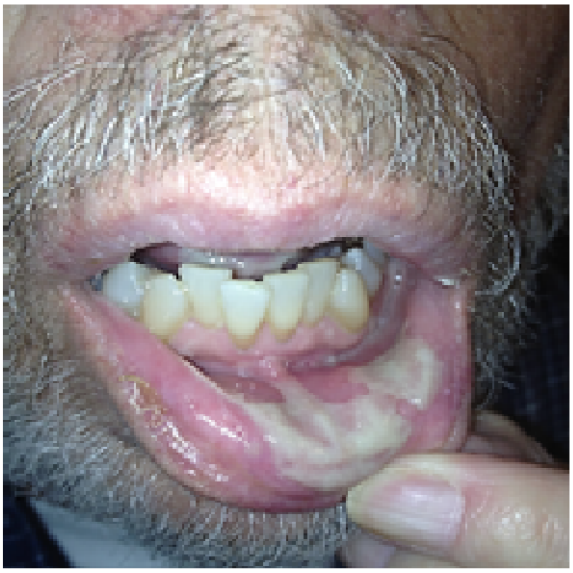
Cancer Treatment Modality	% of patients at risk for oral complications
Head & neck radiation therapy with salivary glands in radiation field	**100**
Hematopoietic Stem Cell Transplantation	**80**
Chemotherapy (primary)	**40**
Adjuvant (additional treatment with) chemotherapy or immunotherapy	**10**

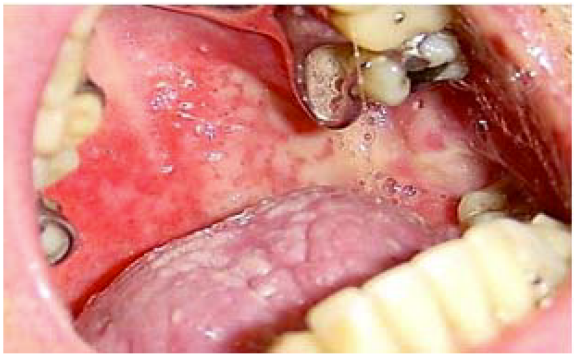
Table 1. Cancer treatment modalities and risk of oral complications

Dentists in private practice may be referred cancer patients in two common situations: a patient recently diagnosed with cancer that needs dental treatment prior to starting cancer therapy. These usually are head and neck cancer patients who will be treated with radiation therapy and will be problematic to receive dental surgical treatment because of the risk of infection and osteoradionecrosis. The other type of referral will be a patient who has completed cancer therapy and may now have oral complications such as dry mouth and need continued dental treatment and follow-up. It is important to keep in mind that such oral complications as dry mouth can be permanent and place the patient at risk of severe caries and periodontal disease. Therefore, maintenance of good oral health and hygiene will be very important for the rest of the patient's life. Some specific oral hygiene protocols for this patient population are discussed in chapter 12. Following, we will describe some of the oral complications of cancer therapy.

1. Mucositis

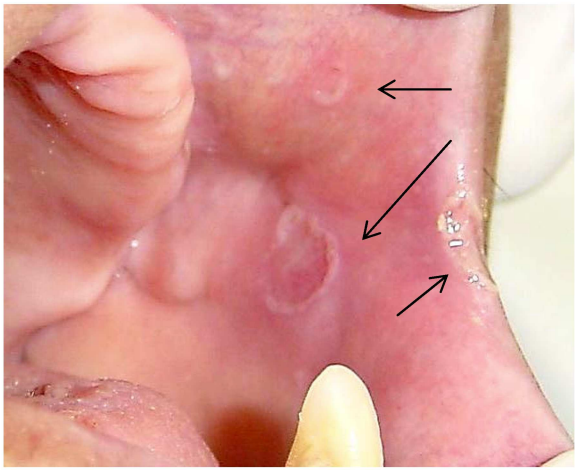
Oral mucositis is an acute complication in the oral cavity of patients treated with radiation and chemotherapy. It is characterized by oral mucosal ulcerations that can start as erythema and progress to large ulcerative lesions that are very painful. The ulcers result from the toxicity of the therapy and usually have an onset around the 7th through the 11th day after starting therapy and can remain active until the effects of therapy end. Patients are usually managed symptomatically, as there is no effective prevention or treatment for this condition. The ulcers can become infected with Candida and/or bacteria. Oral rinses with steroids such as dexamethasone can be associated with antifungals like nystatin and topical xylocaine. Oral bacterial and viral infections can also be included in the differential diagnosis of oral mucositis.





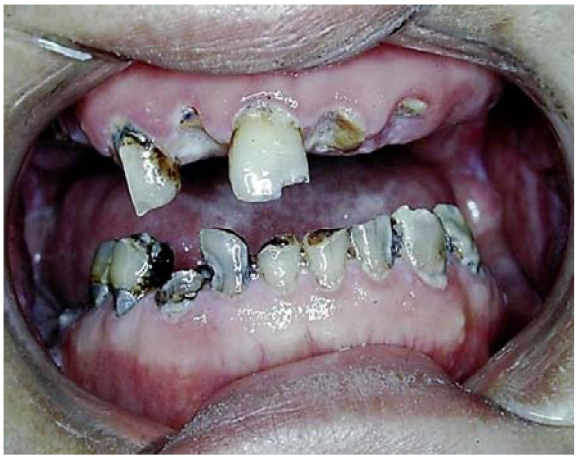
2. Herpetic infection

Herpes infection can be reactivated during cancer therapy. This patient was receiving chemotherapy and developed sudden onset of severe pain that prevented her from eating, drinking and performing oral hygiene. Observe that the herpetic lesions have an unusual clinical presentation of an ulcerated area that can appear anywhere in the oral mucosa, with raised and somewhat keratotic borders. Systemic antiviral therapy with valacyclovir is recommended.



3. Hyposalivation/xerostomia

The photograph shows a patient with dry mouth and foamy saliva post radiation therapy of a tumor in the oral cavity. The decreased amount of saliva in the mouth, associated with poor oral hygiene, leads to the development of severe and rampant decay. Because part of the alveolar bone was included in the fields of radiation, the extraction of teeth could lead to the development of osteoradionecrosis. Such involvement of teeth and mouth tissues with disease could have been prevented with a good oral hygiene protocol and high content fluoride toothpaste, rinse, or fluoride gel in a tray that the patient uses once a day.

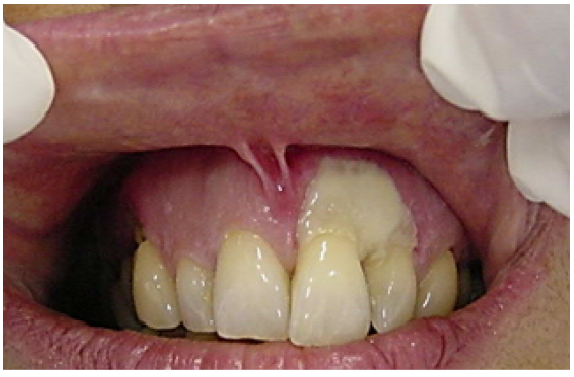


4. Infections/bleeding

Because of changes in saliva quantity and quality, and because of effects on the bone marrow, cancer patients may develop infections in the oral cavity. The quick diagnosis and management of these complications is of utmost importance. Patients are susceptible to fungal, bacterial and viral infections. Photographs below demonstrate examples of infections of various etiologies. The required therapy will be adjusted according to the type of infecting agent.

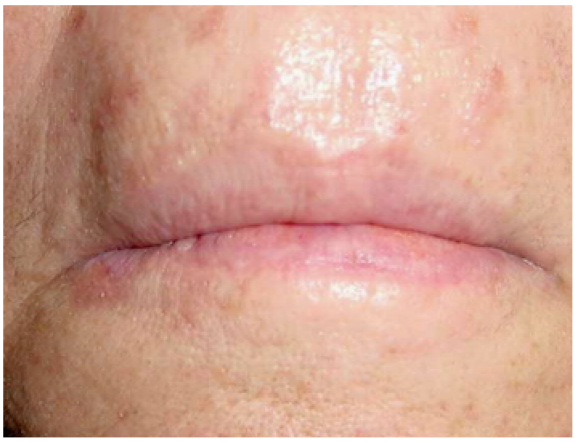
5. Chemotherapy patient with *pseudomonas aeruginosa* infection

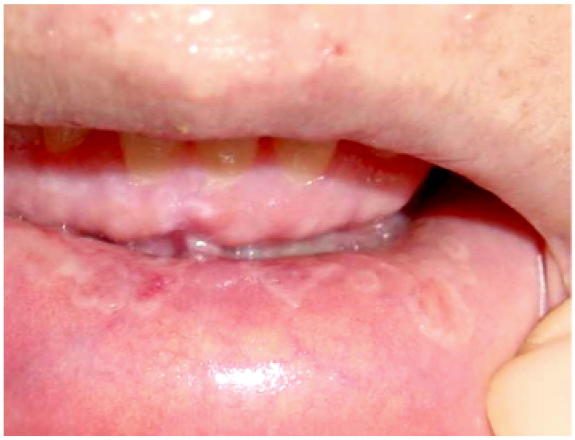
Observe areas of necrotic tissue on the buccal gingiva and vestibule. The patient was in pain and needed immediate care. The infectious agent was sensitive to ciprofloxacin and clindamycin. Topical rinses with chlorhexidine complemented the treatment.



6. Chemotherapy patient with herpetic outbreak

The patient below developed an overnight onset of distressing pain and discomfort in the mouth. She presented with a large number of ulcerated lesions distributed throughout the oral mucosa. The ulcers were painful and presented with a whitish and raised periphery. The diagnosis of a herpetic infection was suspected based on the rapid onset, the severe pain, and the clinical appearance of the lesions. The patient was immediately treated with acyclovir tablets and responded well to therapy.

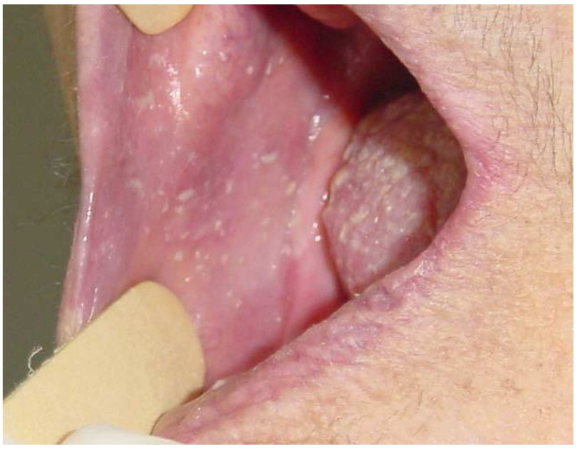




7. Chemotherapy patient with oral candidiasis

The patient in the photograph presented with the lip and oral lesions following a course of chemotherapy from an abdominal malignancy. She developed a general discomfort in the oral

cavity and could no longer taste food. She complained of a burning sensation. Clinically, she presented with areas of white plaques and colonies that could be scraped with gauze suggesting oral candidiasis. The lesions extended to all walls of the mouth. She was treated with an antifungal agent, fluconazole, and responded well to therapy.

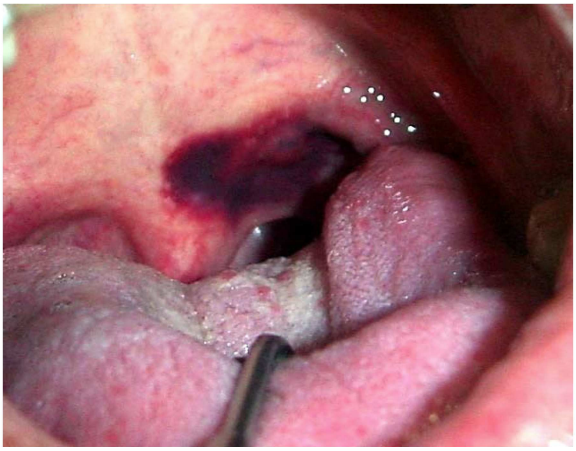


8. Dental treatment of myelosuppressed patients

Some types of chemotherapy can alter bone marrow function leading to suppression. This will place the patient at risk for infections and bleeding. During periods of marrow suppression, patients should not receive elective dental treatment. The treatment of dental emergencies should be planned with the oncology team and precautions should be in place to prevent the initiation and dissemination of mouth infections and bleeding. For this reason, dental disease should be ideally treated prior to the start of cancer therapy. Therefore, good communication between the oncology team, the dental provider and the patient should be established so information can be shared and patient safety can be in place to prevent complications. Stages of immunosuppression occur during high dose chemotherapy and hematopoietic stem cell transplantation. Immunosuppression of transplanted patients persists for years in order to prevent organ rejection. Thus, patients will be at risk of complications for prolonged periods of time. Dental care for these individuals should only be provided when there is good communication amongst the team caring for the individual.

9. Submucosal bleeding in a patient with bone marrow suppression

This chemotherapy patient had a minor trauma while eating and developed submucosal bleeding due to platelet deficiency.



10. Osteoradionecrosis (ORN)

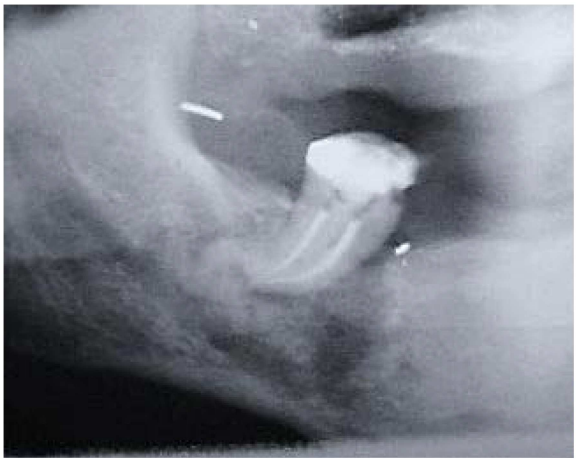
Radiation therapy of the head and neck area can increase the risk for serious complications. One of the late effects of radiation is ORN. Bones included in the field of radiation can develop deficient healing capability because of the effects of radiation on vascularization and cells. It is commonly said that irradiated jaw bones will be avascular, acellular, and hypoxic due to lack of blood vessels. Trauma or chronic infection in irradiated areas can be the stimuli for the development of necrosis because the bones cannot remodel, leading to the formation of ORN. This is a serious complication of radiation and is dependent on the dose of radiation to the bones and the patient's oral health. This complication is usually started by a dental extraction or a periodontal infection and can be non-responsive to antibiotic therapy and local measures. In more severe cases, surgical resection of the affected area is indicated. Patients at risk for this complication usually have received about 5,000 centigrays of radiation and the risk is life-long.

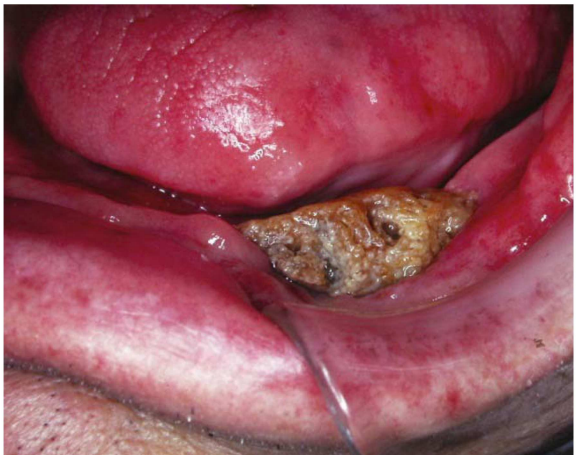
The mandible is the most commonly affected area. Patients complain of pain, partial or complete loss of sensation, infection and fistula formation. In advanced cases, bone fracture can occur.

The management of ORN is multidisciplinary and should involve the oncology team, dental providers and physical therapists. In areas of bone that received high doses of radiation, care must be taken prior to doing invasive dental procedures such as dental extractions or any type of surgery. The determination of radiation dose to the area that needs to be surgically treated must be determined with the help of the radiation oncologist. Only with this information can safer surgical procedures be assured. It has been proposed that prior to doing surgery in irradiated areas in the oral cavity, hyperbaric oxygen therapy (HBO) can be used to increase tissue oxygenation. However, this type of therapy is expensive and there is no scientific prove that it can decrease the risk of ORN. Areas of established ORN can be managed conservatively by local and systemic measures. Broad spectrum systemic antibiotics and topical antiseptic

rinses can be helpful. Partial debridement of bone can decrease the risk of local trauma to the soft tissues. Pain medication can help control symptoms effectively.

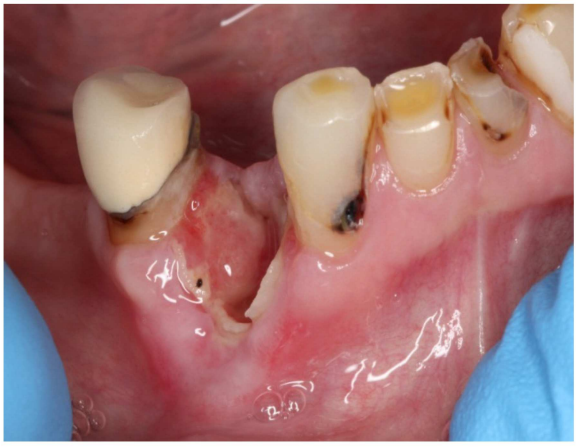
The photographs show the radiographic appearance of an early case of ORN in a patient who was treated with radiation for a tumor of the right cheek. Note that the endodontic therapy of the involved molar was not enough to control the progress of the local infection leading to the bone necrosis. The clinical photograph shows a more advanced area of necrotic bone that has been uncovered by the oral mucosa. The area is very painful and surgical resection will be required in order to control the process of ORN.

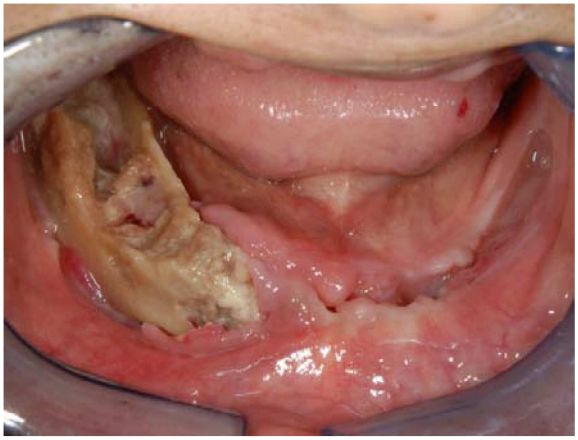




11. Osteonecrosis of the jaws (ONJ)

This is a new oral complication in cancer patients associated with the use of medications that inhibit osteoclasts by various mechanisms. With the inhibition of bone resorption by the osteoclasts, bone cannot be renewed. More recently, new medications with antiangiogenic effect have also been reported to cause ONJ. This complication is defined as the presence of necrotic bone exposed to the oral environment in a patient who is taking an antiresorptive or an antiangiogenic medication and who has not had radiation therapy in the head and neck area. Medications associated with ONJ include the bisphosphonates and denosumab (antiresorptives), and bevacizumab and sorafenib (antiangiogenics). Patients may complain of pain and malodor, loss of sensation and discomfort. There may be evidence of active infection with swelling and pus secretion.





ONJ does not usually respond to standard therapy and in more advanced cases may require resection and primary closure of the surgical defect. Early cases can be controlled with topical measures such as mouth rinses with chlorhexidine. When there is active infection and pain or loss of sensation, systemic antibiotics should be used. More advanced cases that do not respond to therapy should be managed by providers with expertise in the field.

The photographs above show: an early case with a non-healing alveolus after the extraction of a mandibular premolar. Observe the exposed necrotic bone and granulation tissue; this is an advanced case in which about one half of the mandible is necrotic and not covered by oral mucosa.

Additional reading

http://www.cancer.gov/cancertopics/pdq/supportivecare/oralcomplications/HealthProfessional/page1

Hupp W, Brennan M, Migliorati CA. Co-Editors: The Dental Management of the Cancer Patient, 1st edition. American Academy of Oral Medicine, 2012.

Chapter 12

Concepts of Oral Hygiene Maintenance that Would Apply for the Different Groups of Patients

Maintaining good oral health is essential to maintaining good overall health. The goal of proper oral hygiene is to remove or prevent formation and buildup of plaque and tartar, to prevent dental caries and periodontal disease, and to decrease the incidence of halitosis. [1] Results of patient surveys demonstrate that many are unaware of the importance of practicing good oral hygiene and its connection to overall health. For example, results from a May 2012 survey conducted by the American Dental Association regarding oral health found that many people are not certain of basic information regarding proper dental care, recommended replacement time frame for toothbrushes, and causes of dental caries. In addition, new findings from the Centers for Disease Control and Prevention show that approximately 50% of people 30 years and older have some degree of periodontal disease. Routine oral health is important, but only an estimated 44.5% of people obtain professional dental care on a regular basis. Clinical studies have shown that those with poor oral hygiene are at increased risk of developing various oral health problems. Poor oral hygiene is directly responsible for increased incidence of dental caries, periodontal disease, halitosis, oral pain, and discomfort for denture wearers. In addition, some clinical studies have indicated an association between some oral cancers and poor dental and oral hygiene. [1]

1. Oral hygiene guidelines

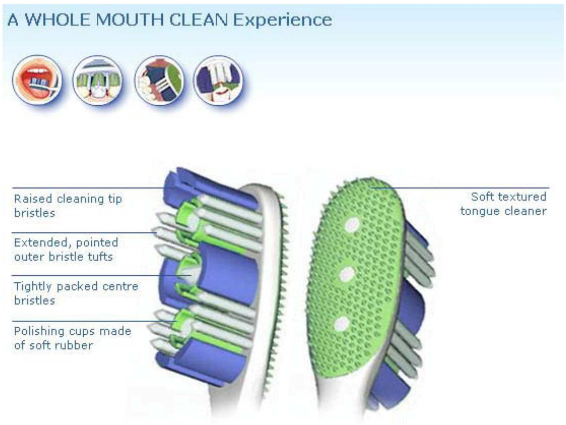
It is recommended that patients see their dentist and dental hygienist at least twice a year. However at home dental care is an integral part of maintaining good oral health. [2-4] Brushing with fluoride toothpaste is recommended at least twice a day to remove plaque and remnants of food and drink from teeth and gums. Flossing once a day is an essential practice to avoid gum disease and preventing tooth decay. [4-5] An oral rinse can also help promote good oral hygiene as it will reduce oral discomfort, provide moisture to oral tissues and help with bad breath. Additional therapeutic oral rinses can be anti-plaque, anti-cavity, anti-tarter, and anti-bacterial and are all good at preventing oral health problems. [2]

2. Over-the-counter preventive and therapeutic oral products

A range of products effective in treating a number of oral health diseases and meeting patients' desires are available in the marketplace. [6] Self-medication with over the counter medicines has long been a feature of the lay health system. Due to the existence of a wide variety of OTC dental products, now the public has access to treatments previously available only through the dentist. [6]

3. Toothbrushes

Mechanical cleaning of teeth and implants can be achieved with the use of a manual or electric toothbrush. [7, 8] The brush head should be worked over the tooth surfaces with light pressure (maximum 100 g). Toothbrushes with soft bristles are recommended because they cause less trauma to the gingiva and abrasion of the dental enamel. The dentist/dental hygienist must choose the ideal technique for the patient from the wide range of tooth-cleaning methods available and must practice and regularly check that technique. The tooth-cleaning routine should be carried out for 2–3 minutes twice a day. Mechanical tooth-cleaning should be avoided immediately after consumption of acidic drinks and foods to ensure that erosion of the dental enamel cannot take place. The American Dental Association recommends replacing a toothbrush every 3 to 4 months. [5, 8]



Toothbrushes are a critical part of biofilm removal. Toothbrushes, like the Colgate® 360° toothbrush, combine unique bristle design and rubber cups with a tongue cleaner to provide superior biofilm removal

3.1. Technique of toothbrushing

Several toothbrushing techniques have been proposed, such as vertical and horizontal scrubbing, the roll technique and the Bass technique. [9] The scrubbing technique, both vertically and horizontally, cleans convex surfaces well but plaque is left in the interdental region. Additionally, scrubbing can cause dental abrasion and gingival recession, unlike the gentler roll technique which does not. The roll technique involves placing the toothbrush against the side of the tooth with the bristles pointing apically, then gently sweeping the bristles downwards for maxillary teeth and up for the mandibular teeth. The shortcoming of this method is that it fails to clean the junction of the tooth with the gingival margin and the gingival crevice. The Bass technique superseded the roll technique owing to its superior cleaning of the gingival crevice. In this, the bristles of the toothbrush are held at about 45° to the long axis of the tooth, pointing towards the gingiva. The brush is pressed against the gingiva and moved with a small circular motion so that the bristles go into the crevice and between the teeth. This is currently the most effective method for the removal of plaque. Regardless of the technique used, it is the onus of the individual to ensure that brushing is undertaken systematically and that no areas of the dentition are overlooked. [9, 10]

3.1.1. Children

Tooth-cleaning with a toothbrush, which should always have soft bristles in the case of children, should start with the eruption of the first primary tooth. A parent or caregiver should ideally perform the tooth-cleaning routine until the child is about 2-3 years old. Parents should

monitor brushing until such time that the child can demonstrate good brushing habits. The brush head should be small and can be modified as in the triple-head toothbrush so that the occlusal, inner and outer surfaces of the teeth can be cleaned at the same time. Electric or battery powered toothbrushes with a timer and the option of playing tunes are ideal motivation aids, encouraging children to brush for the recommended cleaning time of 2 min. [8]

3.1.2. Elderly and disabled patients, patients with limited motor skills and patients requiring care

The use of an electric toothbrush is helpful for people who need support as it requires less effort for the individual or caregiver. These can be divided into rotary/oscillating toothbrushes and sonic toothbrushes. Sonic brushes remove soft bacterial deposits and slight discolorations gently and thoroughly. As a result of their hydrodynamic effect, they can also get rid of subgingival biofilm. If people with impaired fine motor skills use a manual toothbrush, the handle can be individually enlarged with the assistance of an occupational therapist or a physiotherapist. The toothbrush handle can be customized with plasticine material, making it easier to grip. The tooth-cleaning technique should be simple and practical. It can be performed efficiently in a short time with an (electric) triple-head toothbrush. [8, 10]

4. Oral hygiene at the bedside/patients during/after radiotherapy or chemotherapy

If tooth-cleaning with a toothbrush is impossible, food particles and deposits on teeth, mucosa and tongue can be cleaned with a gauze pad, a swab stick or a foam tooth-brush (dental swab). If required, these can be dampened with mouthwash. However, some studies have demonstrated that toothbrushes are far superior to these aids in terms of cleaning the teeth and mucosa so that a toothbrush should be the method of choice whenever possible. The use of a soft (electric) toothbrush is recommended. [8]

5. Toothpastes

The use of toothpaste is imperative for daily tooth-cleaning. Fluoride toothpastes typically contain the following ingredients:[6, 8]

- Fluorides (sodium fluoride, amine fluoride, sodium monofluoride) inhibit the loss of minerals, remineralize the dental enamel and reduce the formation of acid-forming plaque, which helps to prevent caries. Conventional fluoride toothpastes usually have a fluoride content of 0.15% (1,500 p.p.m.)
- Cleaning substances such as silicates, magnesium and calcium carbonate, aluminium oxides, sodium hydrogen carbonate or sodium hexametaphosphate ensure the mechanical removal of plaque and discolorations from the tooth surface. In order to avoid that the surface is mechanically damaged too harshly, toothpastes with a moderate or low relative

dentine abrasion value between 30 and 70 are recommended. Values over 100 are classified as strongly abrasive and should only be used in exceptional cases (thick deposits) not more than once a week

- Binding agents such as alginate or various types of cellulose act as stabilizers and thickening agents
- Foaming agents/surfactants, such as sodium lauryl sulphate, promote wetting of the tooth surfaces, help to spread the toothpaste evenly and loosen plaque. This improves the cleaning action even in hard-to-reach places
- Wetting agents such as glycerine, sorbitol or xylitol give the toothpaste its smoothness and protect it against drying out
- Flavoring agents (oil of peppermint, clove, aniseed or fennel; menthol, cinnamon)
- Preservatives (parabens)
- Sweeteners (aspartame, saccharine)
- Colorings (titanium oxide, patent blue, chlorophyllin, quinoline yellow)

Some toothpastes contain additives such as pyrophosphate or zinc citrate, which inhibit the mineralization of plaque, thereby delaying the formation of dental calculus (tartar). Potassium nitrate, strontium chloride, arginine/calcium carbonate and especially amine fluoride are used to protect sensitive necks of teeth. [8]

Triclosan along with a copolymer has been added to toothpaste to provide 12 hour antibacterial protection. A number of studies have been conducted that demonstrate the effectiveness of triclosan/copolymer in preventing plaque and gingivitis, tartar build up, retarding the progression of periodontal disease and reducing mucositis around dental implants. 6

Colgate® Total™ toothpaste combines the effectiveness of 1100 ppm fluoride with triclosan/copolymer for 12 hour protection against plaque and gingivitis, with additional benefits of tartar control, caries prevention and inhibition of oral malodor.

Fluoride gels have a considerably higher fluoride level of 1.25% (12,500 p.p.m.) than toothpastes and are used for intensive caries prevention. They are applied once a week with a toothbrush in addition to daily tooth-cleaning. [8]

Prescription level fluorides (5000 ppm) are recommended when caries control is critical. Some products have been specially formulated for patients with dentin hypersensitivity or dry mouth.

5.1. Children

Children should be introduced to tooth brushing with a pea-size amount of low-fluoride toothpaste at two years of age. [4] In children younger than two years, parental brushing without toothpaste is recommended. [4] After the age of six, children can safely use regular fluoridated toothpaste. [4, 8]

5.2. Bedridden patients

Non-foaming toothpastes (without sodium lauryl sulphate) should be used for patients who have no opportunity to rinse or spit out. Furthermore, unlike conventional toothpastes, these non-foaming pastes do not have a dehydrating effect on the oral mucosa, which is often a problem in elderly patients. [8]

5.3. Patients after radiotherapy/chemotherapy or with sensitive oral mucosa

To avoid mucosal irritation resulting from ingredients, toothpastes without menthol, cinnamon, peppermint oil and sodium lauryl sulphate should be used. [8]

As salivation is reduced because of radiotherapy, the caries-protective effect of saliva as an important factor in remineralization is diminished. Thermoformed splints should therefore be prepared before the start of radiotherapy. These are coated with fluoride gel 3 times a day, which is allowed to act on the teeth for 5 minutes. This procedure has to be followed throughout life because, without sufficient caries protection, the teeth rapidly develop deep caries and have to be extracted, which carries a risk of osteoradionecrosis. [8]

6. Interdental cleaning

Interdental spaces, which account for 40% of the tooth surface, are hard to reach by normal cleaning with a toothbrush. As a result, bacteria multiply there, leading to caries, gingivitis and periodontitis. Various aids are suitable for interdental cleaning, although their use should be adapted to the individual; patients should be taught how to use them and practice using them. Healthcare professionals should then check that the aids are being used correctly. [8]

The use of interdental brushes has been shown to greatly improve the removal of biofilm, food and other debris from the interproximal areas. Interdental brushes are very useful for those patients with fixed prosthodontics and implants.

6.1. Dental floss

Dental floss is usually made of nylon thread or folded polytetrafluoroethylene strands. It is available as waxed or unwaxed dental floss. Where teeth are crowded, interdental cleaning is easier to manage with waxed than unwaxed floss. It is important to avoid applying too much pressure because this can damage the gingiva. [8]

Superfloss dental floss, as a development of conventional floss, is stiffened with plastic at one end and is easy to thread between the teeth. This stiffened end is then followed by a spongy nylon thread and unwaxed dental floss. This form of interdental tooth-cleaning is particularly suitable for patients with a fixed orthodontic appliance, with dental implants or bridgework. [8]

6.2. Interdental brushes

Interdental brushes are made up of different lengths of nylon bristles arranged radially around a wire core. The wider the gaps between the teeth, the larger the diameter of the interdental brushes should be. If needed, the brushes can be placed in a holder for easier handling. The advantages of interdental brushes are their ease of use, the wide choice of sizes and differently shaped ergonomic handles or holders as well as their great effectiveness. These brushes should be worked through the interdental space, again without exerting too much pressure because that might injure the gums. This cleaning aid is suitable not only for patients with wide interdental spaces, a fixed orthodontic appliance or a dental prosthesis (dental implants, bridges) but also for manually impaired patients. Cleaning should be done without toothpaste in order to avoid mechanical trauma to any root surfaces that may be exposed. [8]

6.3. Dental sticks

These have a triangular shape that matches the interdental space and are suitable for removing food particles and plaque. They are straightforward to use. It is proven that plaque removal is more thorough if toothbrushes plus interdental brushes are used than if the teeth are merely cleaned with a toothbrush. Dental floss or sticks remove plaque less effectively than interdental brushes. Gingival bleeding as well as the depth of periodontal pockets are reduced more markedly after interdental brushes are used than after the use of dental floss. Plaque-induced interdental gingival inflammation declines when dental sticks are employed. [8]

6.4. Children

Flossing in toddlers is valuable for caries prevention and should be commenced as soon as primary teeth establish proximal contacts. At this time, the incidence of proximal caries and gingivitis increases significantly. Manual dexterity and training are needed for effective flossing and since this is not expected of children under 8, parents should floss for young children. Floss incorporating sodium or amine fluoride can promote fluoride uptake in vitro by molar proximal surfaces and demineralized primary enamel. [11]

6.5. Elderly and disabled patients: patients with limited motor skills and patients requiring

6.5.1. Care

Electric dental floss attachments can be used for interdental cleaning to compensate for a decline or lack of fine motor skills. These are supplied as an accessory with the new generation of electric toothbrushes. Vibration of the fixed plastic thread simplifies cleaning of the interdental spaces, although improper use does carry a certain risk of injury. [8]

6.5.2. Patients during chemotherapy

Owing to the possible risk of injury from using dental floss, interdental cleaning with dental floss should be avoided during this phase because of the lower platelet count and the resulting increase in the tendency to bleed. [8]

7. Mouthwashes

7.1. Antiseptic products

Mouthwashes reduce the quantity of bacteria in the mouth, inhibit the growth of bacteria in plaque and thereby prevent gingivitis and periodontitis. Chlorhexidine, cetylpyridinium chloride, amine fluoride, zinc fluoride, triclosan and essential oils are ingredients with a clinically proven action. [8]

Over the counter mouthrinses with CPC have been proven very effective as adjuncts in oral hygiene and biofilm control after toothbrushing.

Chlorhexidine remains one of the most effective antimicrobial mouthwashes because it acts not only against gram-negative bacteria, but also against yeasts and gram-positive bacteria. It is particularly suitable for the inhibition of plaque formation as it has the ability to maintain

effective concentrations for prolonged periods of time, by way of binding to soft and hard tissues. [9] However, longer-lasting rinses with chlorhexidine can cause discoloration of teeth and tongue, increased tartar formation, transient changes in the sense of taste and altered oral flora. [8, 9]

7.2. Fluoride mouthwashes

Mouthwashes containing fluoride enhance the protection against caries by increasing the fluoride reserve built up by toothpaste. Furthermore they also act in the interdental spaces that cannot be reached by the toothbrush. [8]

7.2.1. Mouthwashes with added ingredients

Amine fluoride, arginine calcium chloride or stannous fluoride reduce pain sensitivity. In the process they act on dentine permeability and have anti-erosive effects on the enamel. [8]

7.2.2. Mouthwashes for wetting

The causes of dry mouth are many and varied. They can be divided into iatrogenic causes, diseases affecting the salivary glands and dehydration. Treatment of xerostomia and hyposalivation is very often purely symptomatic and aims to bring subjective relief of dry mouth and improve lubricating ability. In addition, the hard dental substances need to be protected against demineralization. Mucin, carboxymethylcellulose and hydroxymethylcellulose, xanthan, linseed oil and polyethylene oxide are used with the aim of improving viscosity and wettability. Immunologically active ingredients have recently come onto the market, although their mechanisms are still not fully explained. [8]

Rinses for wetting the oral mucosa are mainly used in the elderly population and for patients who have undergone radiotherapy and chemotherapy, but also in rheumatic-type diseases. [8]

7.2.3. Children

Mouthwashes are only recommended after the age of 6 because of the risk of swallowing. [4, 8]

7.2.4. Elderly patients

The cause of dry mouth in elderly frequently lie in the (multiple) medication they take, which can often result in drug-induced xerostomia as a side effect of those medicines. The xerostomia can be minimized by a reduction of the daily dose or by a change of active substance. [8]

8. Oral hygiene at the bedside

If patients are at all able to rinse and spit out, an antibacterial rinse can be beneficial. [Lindenmuller/p107/col…] [this one has a reference too] However, mouthwashes containing chlorhexidine cannot be used as a long-term measure because of their side effects. Therefore

switching to rinses based on amine or stannous fluoride is an option. These may have an additional positive effect on fungal colonization. [8]

The fact that, in addition to their antibacterial action, some amine and stannous fluorides have an antifungal effect and display additional antierosive and desensitizing properties might be beneficial for elderly patients who are at higher risk of fungal infection, caries and sensitive necks of teeth because of increasing morbidity and insufficient oral hygiene. [8]

8.1. Patients during/after radiotherapy or chemotherapy

Mouthwashes containing alcohol and chlorhexidine should be avoided because of the sensitive oral mucosa experienced during/after radiotherapy or chemotherapy, which is often affected by mucositis. Instead an easy-to-make and inexpensive salt-and-soda solution or rinsing with 0.5% lignocaine is favored in cases of chemotherapy-induced mucositis because, in comparison with a 0.12% chlorhexidine solution, no difference has been found in terms of symptoms and time to resolution of mucositis. According to the guidelines of the Multinational Association for Supportive Care in Cancer/International Society for Oral Oncology, as well as effective oral hygiene, a saline or sodium bicarbonate rinse but not chlorhexidine solution should be used. They argue for topical application of benzydamine (Bucco-Tantum®), which has an anti-inflammatory, analgesic and antimicrobial effect. [8]

Following cancer treatment, patients develop transient or persistent xerostomia and hyposalivation. This arises from the reduced quantity of saliva caused by damage to the parenchyma of the salivary glands resulting from exposure to radiation. This in turn leads to worsening of existing tissue inflammation and an increased risk of infection. Chewing and swallowing are made more difficult by the increased viscosity of the saliva. In addition, burning sensations in the tongue and mouth, bad breath, speech disorders, taste disturbances, poor denture grip and resulting ulcerations of the mucosa, caries and oral thrush may occur. [8]

9. Tongue-cleaning

In order to reduce micro-organisms and avoid halitosis, daily cleaning of the surface of the tongue should also be incorporated into dental and oral hygiene. The cleaning action of tongue scrapers is superior to that of a toothbrush. Mouthwashes can also be used temporarily in addition to mechanical tongue-cleaning. However, these should contain scientifically proven ingredients such as chlorhexidine, cetylpyridinium chloride, chlorine dioxide, zinc, hydrogen peroxide, triclosan, zinc chloride, amine fluoride, stannous fluoride or essential oils (Listerine™). [8]

10. Chewing gums

The use of sugar-free chewing gum as a mechanical salivary stimulant after eating can accelerate the clearance of dietary substances and microorganisms, promote buffers to

neutralize plaque acids and provide antibacterial substances. [11, 12] Chewing sugar-free gum for 20 minutes after eating reduces the fall in plaque pH and rapid recovery ensues. This action reduces the time for demineralization and enhances the potential for remineralization of early carious lesions. The saliva flow rate is stimulated three- to tenfold above the resting level and may be prolonged for over 30 minutes. [11, 12]

This approach may enhance saliva function in those with low flow rates such as elderly sufferers of xerostomia or provide symptomatic relief from dry mouth. However, effects on gingivitis or calculus formation have not been demonstrated. [11]

11. Care of complete or partial dentures

As bacteria, fungi, plaque and tartar can become deposited on removable dentures, these must be taken out after at every meal and cleared of food remnants under running water. Denture wearers should also rinse their mouths with water. Furthermore, the oral mucosa covered by the denture, including the palate, should be cleaned at least once a day with a soft toothbrush in order to get rid of remnants of adhesives as well as micro-organisms and food particles. Mechanical denture-cleaning is also done once a day under running water with the aid of a denture brush without toothpaste. Thorough working of the occlusal surface as well as the inside and outside surfaces of the denture is required. [8]

11.1. Patients during/after radiotherapy or chemotherapy

Dentures must not be worn if patients have ulcerations or neutropenia, except while eating. Non-fixed appliances should be removed before the mouth is rinsed with a recommended cleansing agent. [8]

11.2. Supportive periodontal therapy

With at-risk patients, self-care becomes a key behavior in long- term periodontal health maintenance. [13] Inadequate control of bacterial plaque by the patient predisposes the patient to disease recurrence. [13] Therefore, self-care must focus on decreasing the bacterial load daily to prevent the re-establishment of a pathogenic microflora. [13] This can be accomplished by the motivated individual using a variety of oral cleaning devices. [13] Interdental cleaning devices should be recommended and used routinely based upon the type of embrasure spaces present in the patient's mouth. [13]

12. Dry mouth (xerostomia)

Dry mouth (xerostomia) is a complaint that is the most common salivary problem and is the subjective sense of dryness which may be due to reduced salivary flow (hyposalivation) and/or changed salivary composition. [14, 15]

12.1. Causes

Medications are the most common cause of dry mouth. More than 400 medications (prescribed and over-the-counter) list dry mouth as a potential adverse effect. Among them are antihistamines, decongestants, painkillers, diuretics, high blood pressure medications, muscle relaxants, drugs for urinary incontinence, Parkinson's disease medications, antidepressants and many others. [16]

Dry mouth can be caused by other factors, such as emotional stress, anxiety disorders, radiation treatment for head and neck cancers, salivary gland disease, endocrine disorders, diabetes, Parkinson's disease, Alzheimer's disease, stroke, AIDS and Sjögren's syndrome. Hormone changes that take place during pregnancy or menopause also have been associated with dry mouth. Snoring or breathing with an open mouth can contribute to dry mouth. [14, 16]

Drying irritates the soft tissues in the mouth, which can make them inflamed and more susceptible to infection. Without the cleansing effects of saliva, tooth decay and other oral health problems become much more common. [16]

Very rarely, children are born missing salivary glands — so-called salivary gland aplasia or agenesis. Most salivary gland dysfunction how- ever is acquired (Table 1). [14]

12.2. Clinical features

Common symptoms of dry mouth include:[16, 17]

- A sticky, dry feeling in the mouth or throat
- Limited saliva that seems thick or stringy
- A burning sensation in the mouth
- Trouble in chewing, swallowing or speaking
- An altered sense of taste
- A rough, dry tongue
- Cracked lips, sores or split skin at the corners of the mouth
- An infection in the mouth
- Increased plaque (a thin film of bacteria), tooth decay and gum disease
- Bad breath

Additionally, patients with hyposalivation may have difficulty in: controlling dentures. The patient with hyposalivation may complain of a dry mouth or these sequelae alone, or also complain of dryness of the eyes and other mucosae (nasal, laryngeal, genital). Those with eye complaints have blurring, light intolerance, burning, itching or grittiness, and sometimes an inability to cry. Systemic features such as joint pains may be suggestive of Sjogren's syndrome. Complications of hyposalivation can include dental caries, candidosis, halitosis, and ascending sialadenitis. [14]

12.3. Manage symptoms [17]

- Local treatments: simple measures include sucking on pineapple slices, frequent sips of cold orange squash or semifrozen fruit juice, and sugar-free chewing gum.
- Artificial saliva: sprays, lozenges, and gels to use before meals
- Patients with their own teeth can use saliva preparations containing fluoride. Gel preparations are useful for overnight use as they last longer.
- Cracked lips can be very sore—treat with petroleum jelly.
- Pilocarpine (for systemic saliva stimulation) is sometimes used after radiotherapy and to treat the symptoms of Sjögren's syndrome, but it can have considerable side effects.
- Lemon juice should be avoided as it depletes the salivary glands of saliva.
- Dry mouth is associated with dental caries, so referral to a dentist may be required.

12.4. Management of hyposalivation

Any underlying cause of xerostomia should if possible be rectified; for example, xerostomia-producing drugs may be changed for an alternative, and causes such as diabetes should be treated. Patients should be educated into efforts to avoid factors that may increase dryness, and to keep the mouth moist. [14]

Salivary substitutes may help symptomatically. A variety are available including: water or ice chips; frequent sips of water are generally effective, synthetic salivary substitutes (Table2). [14]

As patients with objective xerostomia are at increased risk of developing caries it is important that they take a non-cariogenic diet and maintain a high standard of oral hygiene. The regular use of topical fluoride agents forms an important component of their long-term care. [14]

Salivation may be stimulated by using diabetic sweets or chewing gums (containing sorbitol or xylitol, not sucrose). Cholinergic drugs that stimulate salivation (sialogogues), such as pilocarpine, or cevimeline should be used only by a specialist. Oral complications should be prevented and treated. [14]

Iatrogenic	Drugs
	Irradiation
	Graft versus host disease
Disease	Dehydration
	Psychogenic
	Salivary gland disease
	Sjogren's syndrome
	Sarcoidosis
	Salivary aplasia

Table 1. Causes of dry mouth [14]

Rinse with water after meals.
Keep water at bedside.
Replace missing saliva with salivary substitutes, eg Artificial Saliva, (Glandosane, Luborant, Biotene Oralbalance, AS Saliva Orthana, Salivace, Saliveze).
Alcohol-free mouthrinses (BioXtra and Biotène), or moisturising gels (Oralbalance, BioXtra) may help.
Stimulate saliva with:
• sugar-free chewing gums (eg EnDeKay, Orbit, Biotène dry mouth gum or BioXtra chewing gum) or
• diabetic sweets or
• Salivix or SST if advised or
• drugs that stimulate salivation (eg pilocarpine [Salagen]) if advised by a specialist.
Always take water or non-alcoholic drinks with meals and avoid dry or hard crunchy foods such as biscuits, or dunk in liquids.
Take small bites and eat slowly.
Eat soft creamy foods (casseroles, soups), or cool foods with a high liquid content — melon, grapes, or ice cream.
Moisten foods with gravies, sauces, extra oil, margarine, salad dressings, sour cream, mayonnaise or yogurt.
Pineapple has an enzyme that helps clean the mouth.
Avoid spices.
Avoid anything that may worsen dryness, such as:
• drugs, unless they are essential (eg antidepressants)
• alcohol (including in mouthwashes)
• smoking
• caffeine (coffee, some drinks such as colas)
• mouth breathing.
Protect against dental caries by avoiding sugary foods/drinks and by:
• reducing sugar intake (avoid snacking and eating last thing at night)
• avoiding sticky foods such as toffee
• keeping mouth very clean(twice daily toothbrushing and flossing)
• using a fluoride toothpaste
• using fluoride gels or mouthwashes (0.05%fluoride) daily before going to bed
• having regular dental checks.
Protect against thrush, gum problems and halitosis by:
• keeping mouth very clean
• keeping mouth as moist as possible
• rinsing twice daily with chlorhexidine (eg Chlorohex, Corsodyl, Eludril) or triclosan (eg Plax)
• brushing or scraping your tongue
• keeping dentures out at night
• disinfecting dentures in hypochlorite (eg Milton, Dentural)
• using antifungals if recommended by specialist.
Protect the lips with a lip salve or petroleum jelly (eg Vaseline).
Avoid hot dry environments — consider a humidifier for the bedroom.

Table 2. Tips for managing a dry mouth [14] Drink enough water, and sip on water and other non-sugary fluids throughout the day.

Additional reading

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DIAGNOSIS AND MANAGEMENT OF ORAL LESIONS AND CONDITIONS: A RESOURCE HANDBOOK FOR THE CLINICIAN

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