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.

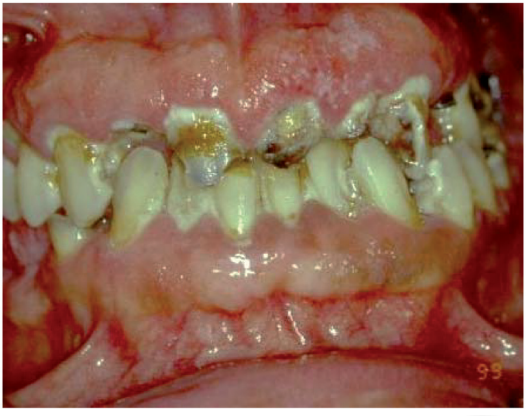
Etiology: Aggressive dental caries is multifactorial, involving inadequate salivary output, frequent consumption of carbohydrates, suboptimal fluoride exposure, previous history of caries activity, and infrequent or ineffective dental care. Inadequate saliva output can arise from systemic disease, disease 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.

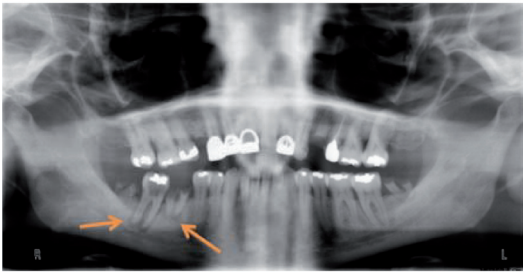
Periapical granulomas should be suspected in about 50% of teeth which have failed to respond to traditional root canal therapy. Most lesions are found during routine radiographic examinations. Granulomas can transform into cysts or abscesses and vice versa without significant radiographic change.

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

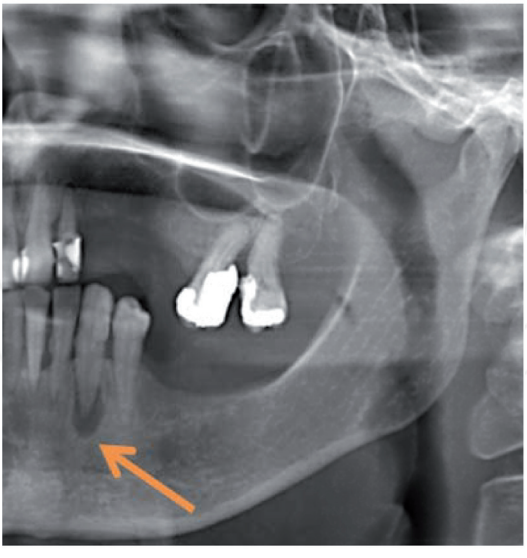
Lateral radicular cysts appear along the lateral aspect of the root and may be a result of inflammatory periodontal disease or pulpal necrosis, spread through accessory canals.Lateral radicular cysts are noted to present as discrete radiolucencies along the lateral tooth root. Loss of lamina dura may 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

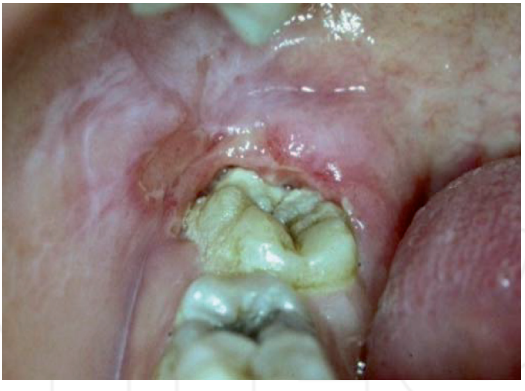
Description: Inflammation of a gingival flap of tissue surrounding the crown of a partially erupted tooth, most often seen with mandibular third molars. The tissue is often erythematous and edematous. Associated symptoms and signs include extreme pain, foul taste, inability to close the jaws due to swelling,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

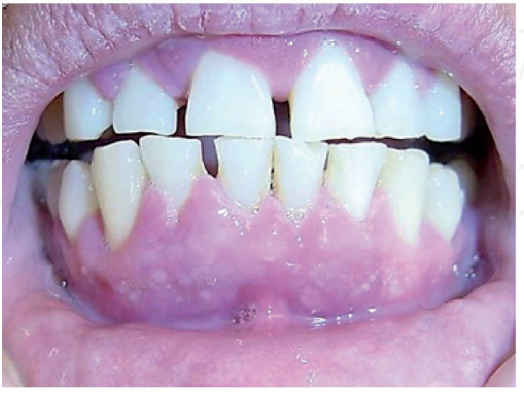
dysfunction without systemic effects. In the localized aggressive disease, there has been found a selective immune dysfunction and specific defect of bactericidal activity toward *A. actinomycetemcomitans*. There is some familial predisposition. The condition begins around the ages of 11 to 13 or circumpubertal. 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.

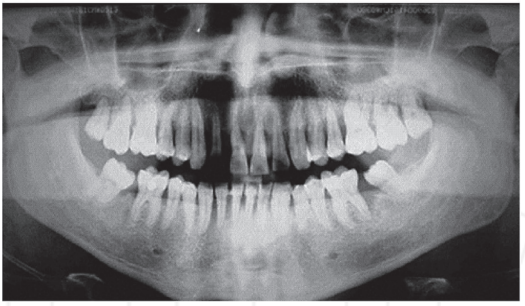
Etiology: There is no association with a systemic disease process; patients are generally otherwise healthy. Organisims commonly found at sites of aggressive periodontal disease include *Actinobacillus actinomycetemcomitans*, *Prevotella intermedia*, *Porphyromonas gingivalis* and others.

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.

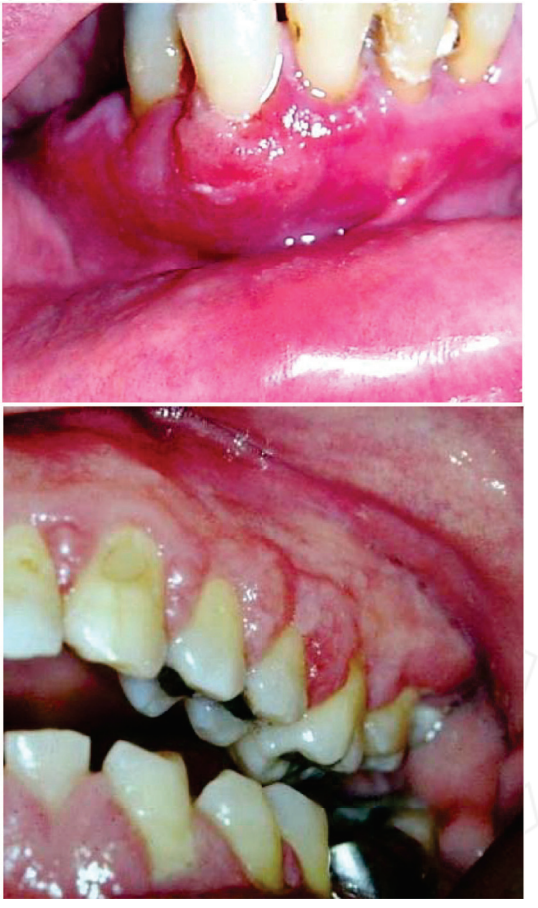
Tissues are generally normal in color and firm, and may be smooth, stippled, or granular in texture. Inflamed tissues may become erythematous and edematous and will bleed easily and become ulcerated and friable. Pyogenic granuloma-like enlargements may be seen in areas of greater inflammation.

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

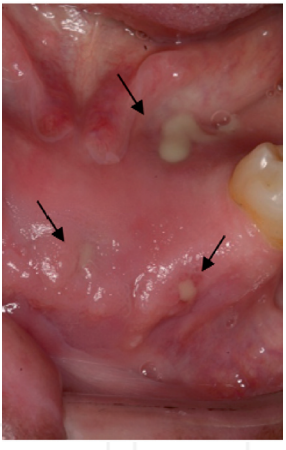
Description: A small erythematous mass of subacutely inflamed granulation tissue, which appears at the intraoral opening of a sinus tract from a periapical abscess. The lesion may be asymptomatic if the infection is chronic and a path of drainage is achieved.

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

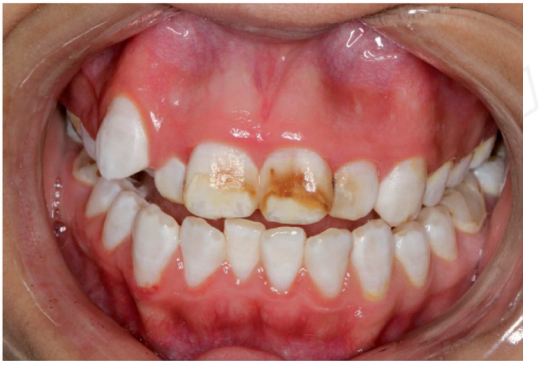
Description: Fluorosis is a condition of hypomineralized enamel, caused by ingestion of excess amounts of fluoride as teeth are developing. Permanent hypomaturation occurs in the developing enamel resulting in increased surface and subsurface porosity and a permanent staining of the teeth. Enamel may 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.

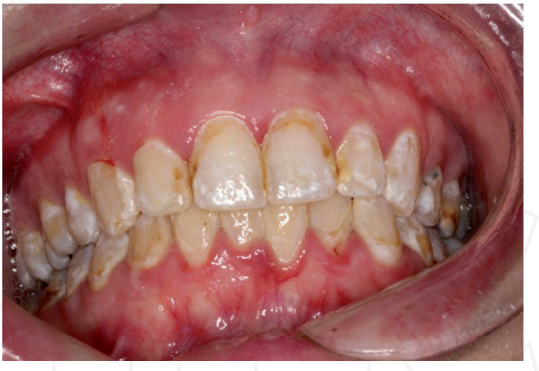
Etiology: Higher than optimal intakes of fluoride during critical periods of tooth development.

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.

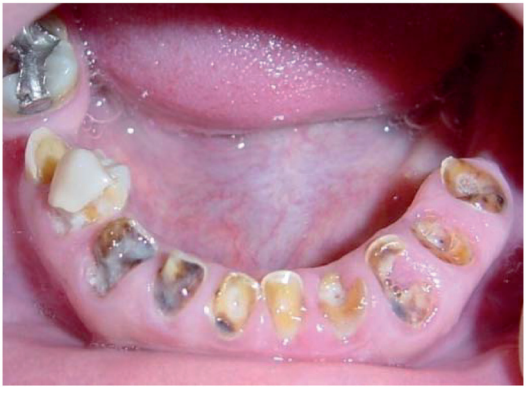
Etiology: Meth users abuse the drug because they feel it gives them greater energy, increased physical ability, and a state of euphoria. Methamphetamine can be smoked, snorted, injected, or taken orally. The majority of users are men between the ages of 19 and 40. The effects of the drug can last up 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.

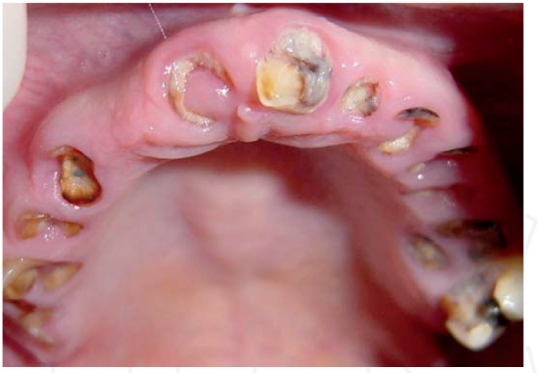
Treatment: Thorough medical and dental history taking is imperitive. Warning signs would include a patient who is emaciated, agitated, and nervous, who exhibits tachycardia, tachypnea, hypertension, hyperthermia, and rampant smooth surface caries. Using local anesthetics containing epinephrine or levonordephrine 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)

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Pericoronitis

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Localized aggressive periodontitis

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

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Parulis

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Fluorosis

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Methamphetamine abuse and its effect on oral health

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