Pulpitis and periapical diseases

Pulpitis

All the principles of inflammation that apply to any other body organ apply to lesions of the dental pulp but the dental pulp has some unique features that make it unusually fragile and sensitive.

1. It is encased by hard tissue (dentin/enamel) that does not allow for the usual swelling associated with the exudate of the acute inflammatory process.
2. There is no collateral circulation to maintain vitality when the primary blood supply is compromised.
3. Biopsies and direct applications of medication are impossible without causing necrosis of the entire pulp.
4. Pain and increasing levels of sensitivity are the only features that can be used to determine the severity of pulpal inflammation.

Because of referred pain and the lack of proprioceptors (position sensors) in the pulp, localizing the problem to the correct tooth can often be a considerable diagnostic challenge. In addition, there may be a poor correlation between clinical symptoms and pathologic changes occurring in the pulp. The level of pulpal inflammation is determined through a combination of clinical criteria. Results of electric, heat, cold, and percussion tests must be added to the patient history, clinical examination, and clinician experience to arrive at the most appropriate diagnosis for the correct tooth. Generally, the more intense the pain and the longer the duration of symptoms, the greater the damage to the pulp. Severe symptoms usually indicate irreversible damage. The response of pulp to injury is inflammation. This response includes stimulation of odontoblasts to deposit reparative dentin at the site to help protect the pulp. If the injury is severe, the result is necrosis of these cells.

Etiology:

1. Caries is the most common form of injury that causes pulpitis. Pulpal microbiology adjacent to carious dentin demonstrates a diverse flora, including gram-positive anaerobes with low numbers of lactobacilli.
2. Operative dental procedures associated with cavity and crown preparations may also trigger an inflammatory response in the dental pulp. The heat, friction, chemicals, and filling materials associated with restoration of teeth are all potential irritants.
3. Trauma, especially when it is severe enough to cause root or crown fracture
4. Periodontal disease that has extended to an apical or lateral root foramen.

Focal Reversible Pulpitis: - Focal reversible pulpitis is an acute, mild inflammatory pulpal reaction that typically follows carious destruction of a tooth or placement of a large metallic filling without an insulating base. It causes hypersensitivity to thermal and electrical stimuli. The pain is mild to moderate and is typically intermittent. As the name implies, the changes are focal (subjacent to the injurious agent) and reversible if the cause is removed. Microscopically, the predominant feature is dilation and engorgement of blood vessels (hyperemia). Exudation of plasma proteins also occurs, but this is difficult to appreciate in microscopic sections.

Acute Pulpitis. The inflammatory response of acute pulpitis may occur as progression of focal reversible pulpitis, or it may represent an acute exacerbation of an already established chronic pulpitis. Constant, severe, tooth-associated pain is the usual presenting complaint. Pain is intensified with the application of heat or cold, although in cases in which liquefaction of the pulp has occurred, cold may in fact alleviate the symptoms. If there is an opening from the pulp to the oral environment, symptoms may be lessened because of escape of the exudate that causes pressure on and chemical irritation of pulpal and periapical nerve tissues.

Chronic Pulpitis: Chronic pulpitis is an inflammatory reaction that results from long-term, low-grade injury or occasionally from quiescence of an acute process. Symptoms, characteristically mild and often intermittent, appear over an extended period. A dull ache may be the presenting complaint, or the patient may have no symptoms at all. As the pulp deteriorates, responses to thermal and electrical stimulation are reduced.

Chronic Hyperplastic Pulpitis. This special form of chronic pulpitis occurs in the molar teeth (both primary and permanent) of children and young adults. Involved teeth exhibit large carious lesions that open into the coronal pulp chamber. Rather than undergoing necrosis, the pulp tissue reacts in a hyperplastic manner, producing a red mass of reparative granulation tissue that extrudes through the pulp exposure. This type of reaction is believed to be related to the open root foramen, through which a relatively rich blood supply flow. Symptoms seldom occur because there is no exudate under pressure, and generally no nerve tissue is proliferating with the granulation tissue. Although the pulp tissue is viable, the process is not reversible, and endodontic therapy or tooth extraction may be necessary

Treatment and Prognosis

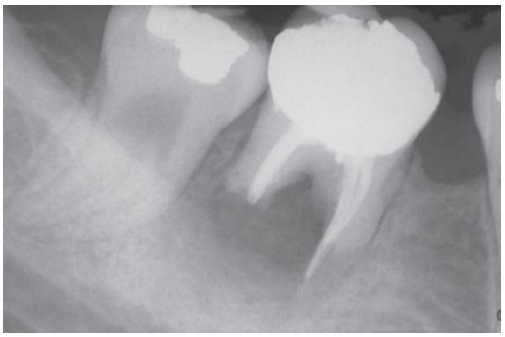
If the cause is identified and eliminated, focal reversible pulpitis should recede, returning the pulp to a normal state. If inflammation progresses into an acute pulpitis with neutrophil infiltrates and tissue necrosis, recovery is unlikely, regardless of attempts to remove the cause. Endodontic therapy or tooth extraction is the only available treatment at this stage. With chronic pulpitis, pulpal death is the characteristic end result. Removal of the cause may slow the process or occasionally may save the vitality of the pulp. Endodontic therapy or extraction is typically required. Chronic hyperplastic pulpitis is essentially an irreversible end stage that is treated with pulp extirpation and an endodontic filling or extraction.

Periapical granuloma: is a mass of chronically or subacutely inflamed granulation tissue at the apex of a nonvital tooth.

Clinical features: Most periapical granulomas are asymptomatic (most of these lesions are discovered on routine radiographic examination) but pain and sensitivity can develop if acute exacerbation occurs. Typically, the involved tooth does not demonstrate mobility or significant sensitivity to percussion. The soft tissue overlying the apex may or may not be tender. The tooth does not respond to thermal or electric pulp tests unless the pulpal necrosis is limited to a single canal in a multirooted tooth. Periapical granulomas represent approximately 75% of apical inflammatory lesions and 50% of those that have failed to respond to conservative endodontic measures.

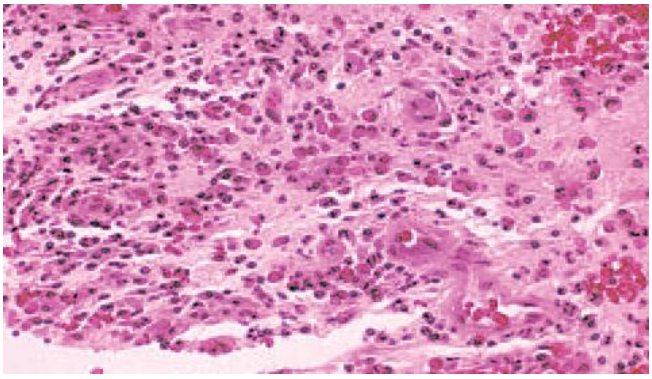
Radiographical features: - In radiograph the periapical granulomas appear as a radiolucencies with variable size ranging from small, hardly noticeable lesions to lucencies exceeding 2 cm in diameter. Affected teeth typically reveal loss of the apical lamina dura. The lesion may be circumscribed or ill-defined and may or may not demonstrate a surrounding radiopaque rim. Root resorption may be seen.

Periapical granuloma. Ill-defined radiolucency associated with the mandibular first molar, which exhibits significant root resorption.



Histopathological features:

Periapical granulomas consist of inflamed granulation tissue surrounded by a fibrous connective tissue wall. The granulation tissue demonstrates a variably dense lymphocytic infiltrate that is intermixed frequently with neutrophils, plasma cells, histiocytes, and, less frequently, mast cells and eosinophils. When numerous plasma cells are present, scattered eosinophilic globules of gamma globulin (Russell bodies) may be seen. In addition, clusters of lightly basophilic particles (pyronine bodies) also may be present. Both of these plasma cell products are not specific for the periapical granuloma and may be found within any accumulation of plasma cells. Epithelial rests of Malassez may be identified within the granulation tissue. Collections of cholesterol clefts, with associated multinucleated giant cells and areas of red blood cell extravasation with hemosiderin pigmentation, may be present.



Periapical granuloma. Granulation tissue exhibits mixed inflammatory infiltrate consisting of lymphocytes, plasma cells, and histiocytes.

Treatment and prognosis: -

If the tooth can be preserved, root canal therapy is recommended. Non restorable teeth must be extracted, followed by curettage of all apical soft tissue. Periapical surgery remains an important tool for resolution of periapical inflammatory disease, but often it is reserved for

lesions larger than 2 cm or those associated with teeth that are not appropriate for conventional endodontic therapy. Periapical surgery should include thorough curettage of all periradicular soft tissue, amputation of the apical portion of the root, and sealing the foramen of the canal.

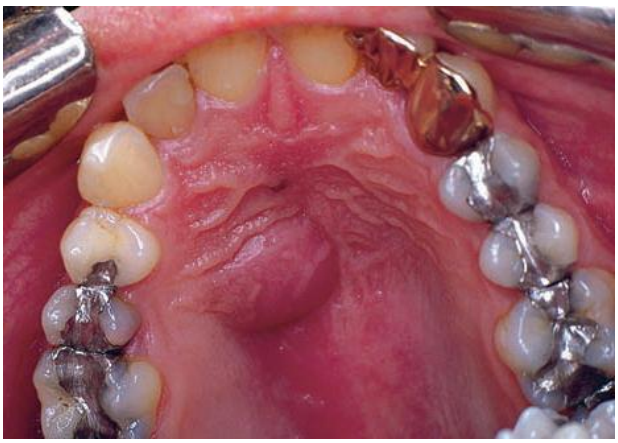
Periapical Abscess

Etiology: Numerous sequelae may follow untreated pulp necrosis and are dependent on the virulence of the microorganisms involved and the integrity of the patient's overall defense mechanisms. From its origin in the pulp, the inflammatory process extends into the periapical tissues, where it may present as a granuloma or cyst (if chronic) or an abscess (if acute). Acute exacerbation of a chronic lesion may also be seen.

Clinical Features

Patients with periapical abscesses typically have severe pain in the area of the nonvital tooth caused by pressure and the effects of inflammatory chemical mediators on nerve tissue. The exudate and neutrophilic infiltrate of an abscess put pressure on surrounding tissue, often resulting in slight extrusion of the tooth from its socket. Pus associated with a lesion, if not focally controlled, seeks the path of least resistance and spreads into contiguous structures. The purulence may extend through the medullary spaces of the bone away from the apical area, resulting in osteomyelitis, or it may perforate the cortex and spread diffusely through the overlying soft tissue (as cellulitis). The affected area of the jaw may be tender to palpation, and the patient may be hypersensitive to tooth percussion. The involved tooth is unresponsive to electrical and thermal tests because of pulp necrosis. The purulent material can accumulate in the connective tissue overlying the bone and can create a sessile swelling or perforate through the surface epithelium and drain through an intraoral sinus. At the intraoral opening of a sinus tract, a mass of subacutely inflamed granulation tissue often is found, known as a parulis (gum boil). Occasionally, the nonvital

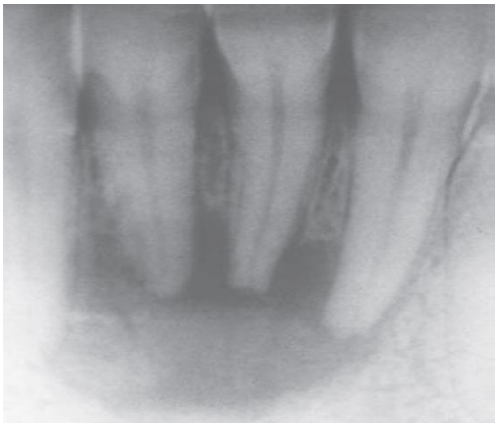
tooth associated with the parulis may be difficult to determine, and insertion of a gutta-percha point into the tract can aid in detection of the offending tooth during radiographic examination. Dental abscesses also may channelize through the overlying skin and drain via a cutaneous sinus.



Palatal abscess

Radiographical Features: -

The rapidity of development makes time insufficient for significant amounts of bone resorption to occur. Therefore, radiographic changes are minor and usually limited to mild radiographic thickening of the apical periodontal membrane space. However, if a periapical abscess develops as a result of acute exacerbation of a chronic periapical granuloma, a radiolucent lesion is evident.



Histopathology

Microscopically, a periapical abscess appears as a zone of liquefaction composed of proteinaceous exudate, necrotic tissue, and viable and dead neutrophils (pus). Adjacent tissue containing dilated vessels and a neutrophilic infiltrate surrounds the area of liquefaction necrosis. With chronicity, an abscess develops into a granuloma, which is composed of granulation tissue and fibrous tissue infiltrated by variable numbers of neutrophils, lymphocytes, plasma cells, and macrophages. Acute flare of a periapical granuloma would show an abundant neutrophilic infiltrate in addition to granulation tissue and chronic inflammatory cells.

Treatment and Prognosis

1. Drainage is required through an opening in the tooth itself or through the soft tissue surrounding the jaw, if cellulitis has developed.

2. Antibiotics directed against the offending organism.

Delayed or inappropriate treatment can be life threatening. Spread of an abscess may occur through one of several avenues. It may progress through the buccal cortical bone and gingival soft tissue, establishing a natural drain or sinus tract. The same type of situation may occur in the palate or skin; this depends on the original location of the abscess and the path of least resistance. If a drain is not established, the purulent exudate can cause an abscess or cellulitis in the soft tissues of the face, oral cavity, or neck. Cellulitis is an acute inflammatory process that is diffusely spread throughout the tissue rather than localized, as with an abscess. This variant is a result of infection by virulent organisms that produce enzymes that allow rapid spread through tissue. Bilateral cellulitis of the submandibular and sublingual spaces has been called Ludwig's angina. A dangerous situation occurs when acute infection involves major blood vessels, possibly resulting in bacteremia. Also, retrograde spread of the infection through facial emissary veins to the cavernous sinus may

set up the necessary conditions for thrombus formation. Cavernous sinus thrombosis is often fatal emergency situation.