Oral Pathology

Periapical Pathology

Periapical periodontitis

Periapical periodontitis (also termed apical periodontitis, AP), is an acute or chronic inflammatory lesion around the apex of a tooth root which is caused by bacterial invasion of the pulp of the tooth. Periapical periodontitis can be considered a sequela of dental caries, irreversible pulpitis and pulpal necrosis, since it is the likely outcome of untreated dental caries. It is often accompanied by resorption of bone, and occasionally the root apex, sufficient to be detected radiographically. Diagnosis usually made on basis of combination of clinical and radiographic features. However, the periapical vascular network has a rich collateral circulation, lymphatic drainage supply in addition to unlimited source of undifferentiated cells, greatly enhancing the ability of the tissue to heal if the cause of the inflammation is removed. This potential for complete periapical healing, providing the source of irritation is removed, is the basis of endodontic treatment.

Etiology

- Bacterial invasion from pulpal disease infection
- Periodontal disease → periodontal pathogens in a pocket find their way to infect the periapical region
- Trauma: Occlusal trauma from, a high restoration, pressure during orthodontic treatment, a direct blow on a tooth, and biting unexpectedly on a hard body in food may all cause minor damage to the periodontal ligament and localized inflammation.
- Chemical irritation: ex: root filling materials
Apical inflammatory lesions formation represents a defensive reaction secondary to the presence of bacteria in the root canal with spread of related toxic products into the apical zone. Initially, the defense reaction eliminates noxious substances that exit the canals. With time, however, the host reaction becomes less effective with microbial invasion or spread of toxins into the apical area.

Whether the response to irritation in the periodontal ligament is principally an acute or chronic inflammation depends on factors such as

1. The number and virulence of the microorganisms involved
2. The type and severity of mechanical or chemical irritant
3. The efficiency of the host defences
**Acute apical periodontitis**

This is characterized by an acute inflammatory exudate in the periodontal ligament within the confined space between the root apex and the alveolar bone.

**Clinical features**

1- Pain is elicited when external pressure is applied to the tooth because the pressure is transmitted through the fluid exudates to the sensory nerve endings.

2- Even light touch may be sufficient to induce pain

3- Unlike pulpitis, pain is generally well localized to a particular tooth due to stimulation of proprioceptive nerve endings in the periodontal ligament.

4- As the fluid is not compressible, the tooth feels elevated in its socket.

5- Hot or cold stimulation of the tooth does not cause pain, as it would in pulpitis.

**Radiographic appearances**

1- Normal as there is generally insufficient time for bone resorption to occur between the time of injury to the periodontal ligament and the onset of symptoms.

2- If radiological changes are present, they consist of slight widening of the periodontal ligament and the lamina dura around the apex may be less well defined than normal
The inflammation may be transient if it is due to acute trauma rather than infection and the condition soon resolves. If the irritant persists the inflammation becomes chronic and may be associated with resorption of the surrounding bone.

-Suppuration may occur if there is severe irritation and tissue necrosis associated with bacterial infection and the continued and massive exudation of neutrophil leucocytes leading to abscess formation.

-Persistent irritation usually derived from bacteria and their products in the pulp chamber and root canals, leads to chronic periapical periodontitis. This is characterized by resorption of the periapical alveolar bone and its replacement by chronically inflamed granulation tissue to form a periapical granuloma.

**Periapical Granuloma (Chronic apical Periodontitis)**

A growing mass of chronically inflamed granulation tissue surrounding the apex of a non vital tooth and arising in response to necrosis of the tooth pulp & spread of related toxic products into the apical zone.

Periapical granulomas may arise
1- After quiescence of a periapical abscess
2- Develop as the initial periapical pathosis.
These lesions are not static and may transform into periapical cysts or may demonstrate acute exacerbations with abscess formation.

**Clinical Features:**
Most periapical granuloma are asymptomatic, but pain & sensitivity can develop if acute exacerbation occurs. The involved tooth does not demonstrate mobility or significant sensitivity to percussion. Tooth does not respond to thermal or electric pulp testing unless the pulpal necrosis is limited to a single canal in a multirooted tooth.
.Most lesions are discovered on routine radiographic examination.

**Radiographic features:** Affected tooth typically demonstrate loss of apical lamina dura. The lesion is well or ill defined radiolucency associated with apex of a tooth, may or may not demonstrate a
surrounding radioopaque rim. The size is variable, ranging from small to lucencies exceeding 2 cm in diameter. Root resorption is not uncommon.

Histopathologic features:

- It consists of inflamed granulation tissue surrounded by fibrous connective tissue. The granulation tissue demonstrates variably dense lymphocytic infiltrate intermixed with plasma cells & neutrophils.

- Collection of cholesterol clefts, and multinucleated giant cells & areas of RBC’s with hemosiderine pigmentation may be present. The cholesterol clefts attract macrophages to degrade them & release inflammatory and bone resorptive mediators. 
- Cholestrol- laden macrophages appear with clear cytoplasm named as foam cells.

- Epithelial rests of Malassez may be identified within the granulation tissue. Epithelial cell rests of Malassez incorporated within the granuloma may begin to proliferate, probably as a result of stimulation by growth factors released by a variety of cells within the granuloma. The
proliferated squamous epithelium forms anastomosing cords, often arranged in loops or arcades, throughout the granulation tissue.

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

Treatment:
- Non restorable tooth can be extracted followed by curettage of all apical soft tissue.
- If the tooth can be maintained then root canal therapy can be performed.
- Periapical surgery is an important tool for resolution of periapical inflammatory disease, but it is reserved for lesions larger than 2 cm or those associated with tooth that are not appropriate for conventional endodontic therapy.

Lesions may fail to heal for several reasons:
♦ Cyst formation
♦ Inadequate endodontics (e.g., poor access design, missed canals, perforated canals, inadequate aseptic technique or instrumentation, leaking fillings)
♦ Vertical root fractures
♦ Periapical foreign material
♦ Associated periodontal disease
♦ Penetration of the adjacent maxillary sinus
Periapical (RADICULAR) Cyst (lateral Radicular Cyst, Residual Cyst)

It is defined as an odontogenic cyst of inflammatory origin that is preceded by a chronic periapical granuloma. Epithelium at the apex of a non-vital tooth can be presumably stimulated by inflammation to form a true epithelium-lined cyst.

The source of the epithelium is:
1. rest of Malassez
2. crevicular epithelium
3. Sinus lining
4. Epithelial lining of fistulous tracts

Classification It is classified as follows:
1) Periapical Cyst: These are the radicular cysts which are present at root apex.
2) Lateral Radicular Cyst: These are the radicular cysts which are present at the opening of lateral accessory root canals of offending tooth.
3) Residual Cyst: These are the radicular cysts which remain even after extraction of offending tooth.

Pathogenesis:

In pre-existing periapical granuloma continuous inflammation stimulates the epithelial rests of Malassez, which are found in the periodontal ligament, resulting in the formation of a cyst since initial reaction leads to proliferation of epithelial rests in periapical area involved by granuloma. As this proliferation continues with the epithelial mass increasing in size by division of the cells on periphery corresponding to basal layer of surface epithelium. The cells of central portion of mass become separated further & further from nutrition in comparison with basal layer due to which they fail to obtain sufficient nutrition, they eventually degenerate, become necrotic & liquify. This creates an epithelium lined cavity filled with fluid.

The lesions can grow into large lesions because they apply pressure over the bone causing resorption. It expands in balloon-like fashion. Cystic fluid is largely inflammatory exudate & high concentration of protein with increase in osmotic pressure & since the cystic wall cavity is
semipermeable membrane allows the penetration of fluids from the surrounding tissues. The toxins & bone resorbing factor (interlukins & prosthoglandines) released by the breakdown of granulation tissue is a common cause of bone resorption.

**Clinical Features**

- Most common type of cyst of the jaws
- Common in the maxilla
- Slowly progressive painless swellings
- Asymptomatic unless there is an acute exacerbation
- Movement & mobility of adjacent teeth as the cyst enlarges
- The associated tooth does not respond to thermal & electrical pulp testing

**Radiographic Features**

- Identical to periapical granuloma
- Round to ovoid with narrow opaque margin that is continuous with the lamina dura of the involved teeth.

**Lateral radicular cyst**

- On occasion, a similar cyst, best termed a *lateral radicular cyst*, may appear along the lateral aspect of the root. Like the periapical cyst, this lesion also usually arises from rests of Malassez, and the source of inflammation may be periodontal disease or pulpal necrosis with spread through a lateral foramen. Radiographically, these cysts mimic developmental *lateral periodontal cysts*. Lateral radicular cysts appear as discrete radiolucencies along the lateral aspect of the root.

**Residual cyst**

appears as round to oval radiolucency of variable size within the alveolar ridge at a site of a previous tooth extraction.
Periapical cyst, Radiolucency associated with the maxillary central incisor, which exhibits significant root resorption

Lateral radicular cyst
Residual periapical cyst, Radiolucency with
Central radiopacity of the right mandibular body

Histopathological features:

*The histopathologic features of all three types of inflammatory cysts are similar.*

- **Epithelial Lining**: Stratified Squamous epith, a defined basal cell layer. Sometime mucous producing cells with ciliated pseudostratified columner epith. May be seen in maxillary periapical cysts derived from maxillary sinus.
- **Cyst capsule & wall**: collagenous fibrous connective tissue. Cholesterol clefts: hemosiderin pigments derived from breakdown of blood cells. Inflammatory cells infiltrate mainly lymphocytes & plasma cells intermixed with neutrophils. Scattered hyaline bodies appear as eosinophilic material surrounded by lymphocytes and multinucleated giant cells.
- **Cyst Fluid**: watery and opalescent, sometimes yellowish; contains cholesterol crystals
- **Rushton Bodies**: occasionally the lining epith. May demonstrate linear or arch-shaped calcification.
Periapical cyst lined by stratified squamous epithelium

Periapical cyst. Squamous epithelial cyst lining exhibits numerous linear and arch-shaped Rushton bodies

Hayaline bodies

Treatment

- Extraction of Non-Vital Tooth and curettage of apical zone → small cysts usually come with the extracted tooth due to its attachment to the PDL
- RCT with apicoectomy → seal roots from the apex
- Surgery (2 Ways of Cyst Removal)
  1. Enucleation
     - Removal of cysts < 2 cm
     - The cyst is removed as a whole by scraping it from the surrounding bone.
  2. Marsipulisation
**Periapical Abscess:**

Numerous sequelae may follow untreated pulp necrosis and are dependent on the virulence of the microorganisms involved & the integrity of the patient’s defense mechanism.

Acute inflammatory lesions with abscess formation may arise

1- As the initial periapical pathosis
2- From an acute exacerbation of a chronic periapical inflammatory lesion.
3- On occasion, however, pulpal death may be trauma related, and the tooth may contain neither a cavity nor a restoration.

The necrotic pulpal tissue debris, inflammatory cell & bacteria all serves to stimulate & sustain the periapical inflammatory process.

**Clinical Features:**

Many investigators subdivide periapical abscesses into acute and chronic types. However, these are misnomers because both types represent acute inflammatory reactions. Periapical abscesses should be designated as **symptomatic** or **asymptomatic** on the basis of their clinical presentations.

Patients with periapical abscesses typically have severe pain in the area of the non vital tooth because of pressure & the effects of inflammatory chemical mediators on nerve tissue. The exudate & neutrophilic infiltrate of an abscess cause pressure on the surrounding tissue, often resulting in slight extrusion of the tooth from the socket. 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 electric & thermal tests because of pulp necrosis.

**Radiographic appearance:**

Because of rapidity with which the lesion develops, there is generally insufficient time for significant amounts of bone resorption to occur. Therefore, radiographic changes are slight & usually limited to 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.
periapical abscess

Histopathology:

A periapical abscess appears as a zone of liquefaction composed of exudate, necrotic tissue and viable & dead neutrophils (pus). Adjacent tissues containing dilated vessels & a neutrophilic infiltrate intermixed with histiocytes, surrounds the area of liquefaction necrosis.

Treatment and prognosis:

- Drainage should be established, either through the tooth itself or through the soft tissue surrounding the jaw if cellulitis has developed.
- Antibiotics directed against the causative organisms is required.
- Endodontic therapy or extraction is required. Once the infection has been resolved the affected bone typically heals.
- If the affected tooth is extruded, reduction of the occlusion is recommended.
- Analgesics are administered in more severe cases.

**Routes of spread**

If the cause of the abscess is not removed, for example by extraction of the tooth, endodontic treatment, or antibiotic therapy, suppuration will continue and the abscess continues to enlarge. With progression, the abscess spreads along the path of least resistance. The increase in hydrostatic pressure within the abscess associated with progressive suppuration causes the pus to track in one of a number of directions.

1. It may drain through the root canal if this is open to the mouth
2. Occasionally it may track through the periodontal ligament to discharge into the gingival sulcus
3. More commonly, the pus tends to track through the cancellous bone and eventually perforates the cortex.

Once the cortical plate is perforated the pus strips up the periosteum and may result in the formation of a **subperiosteal abscess**. More frequently, it penetrates the periosteum after which it may track in various directions. After the pus has perforated the cortical plate its subsequent routes of spread are dictated largely by anatomical factors.

1. The pus may discharge directly into the oral cavity through a sinus following local penetration of the overlying periosteum and mucosa.
2. The dense palatal mucoperiosteum is resistant to penetration by pus. Pus tracking palatally may spread under the mucoperiosteum posteriorly to the junction of the hard and soft palate and present as a palatal abscess.
3. Dental abscesses also may channelize through the overlying skin and drain via a **cutaneous sinus**.
4. Abscesses in the molar region of either jaw may penetrate the buccal cortical plate above (in the maxilla) or below (in the mandible) the attachments of the buccinator muscle. In such cases acute inflammatory oedema and suppuration spread into the soft tissues of the face or neck.

5-Abscesses related to anterior maxillary teeth may perforate the labial bone above the attachment of the levator anguli oris muscle. The infection may then pass medially and upwards towards the inner canthus of the eye, obliterating the nasolabial fold, and into the loose connective tissue of the lower eyelid.

6-Abscesses developing at the root apices of maxillary molars and premolars are very close to the floor of the maxillary sinus and consequently may discharge into the sinus.

**NOTE:** Most abscesses point buccally as the root apices lie closer to the buccal than to the lingual or palatal cortical plates. However, abscesses related to the **apices of the maxillary teeth, particularly lateral incisors and the palatal roots of the molars and premolars**, often track towards and **point on the palate**. Although the apices of the roots of the mandibular second and third molars lie close to the lingual cortical plate, the bone in this area is very dense and is rarely penetrated.

**NOTE:** If a chronic path of drainage is achieved, a periapical abscess typically becomes asymptomatic because of a lack of accumulation of purulent material within the alveolus. Occasionally, such infections are discovered during a routine oral examination after detection of a parulis or drainage through a large carious defect. If the drainage site becomes blocked, signs and symptoms of the abscess frequently become evident in a short period of time.

Sinus opening related to abscess. Palatal abscess related to lateral incisor. Associated with maxillary central incisor.
Abscess related to maxillary canine pointing buccally

Localized extraoral spread of abscess related to a mandibular molar. Scarring associated with chronic extraoral sinus

Cellulitis

Cellulitis is a rapidly spreading inflammation of the soft tissues particularly associated with streptococcal infections. It is not well-localized, in contrast to a circumscribed abscess, and the rapid spread is most likely related to release of large amounts of streptokinase and hyaluronidase which are produced by most strains of streptococci.

Clinically:

1- Diffuse, tense, painful swelling of the involved soft tissues
2- Usually associated with malaise and an elevated temperature. Much of the swelling is due to inflammatory oedema; suppuration and abscess formation if treatment is neglected or delayed.

Cellulitis associated with maxillary teeth initially involves the upper half of the face. Extension towards the eye is a potentially serious complication because of the risk of cavernous sinus thrombosis as
a result of infection involving veins at the inner canthus of the eye which communicate with the cavernous sinus.

Cellulitis associated with mandibular teeth initially involves the lower half of the face; extension into the submandibular and cervical tissues may cause respiratory embarrassment.

Cellulitis spreading into the deeper surgical spaces usually presents clinically as pain and trismus rather than facial swelling.

Cellulitis associated with spread of inflammation from abscess

1-related to a maxillary molar 2-related to an anterior maxillary tooth.

Ludwig's angina

It is severe cellulitis involving the submandibular, sublingual, and submental spaces, usually as a result of initial involvement of the submandibular space. Since the advent of antibiotics it is now rare. The diffuse cellulitis produces a board-like swelling of the floor of the mouth, the tongue being elevated and displaced posteriorly. As a result there is difficulty in eating, swallowing, and breathing. The latter is exacerbated as infection tracks backwards to involve the pharynx and larynx. Odema of the glottis may occur with risk of death by suffocation.