Oral pathology

Periapical Pathology

Inflammation in the periapical part of the periodontal ligament is similar to that occurring elsewhere in the body, but, because of the confined space within which the process develops; a particular feature of inflammation in this site is that the adjacent bone and occasionally the root apex may resorb. However, the periapical tissue heals, if the cause of inflammation is removed.

This potential for complete periapical healing, providing the source of irritation is removed, is the basis of endodontic treatment. The periapical periodontitis is different from pulpitis in the following:

1-the periapical periodontitis differs markedly from pulpits where the potential for healing is very limited.

2-the symptoms are also different in that they are generally well located by the patient to a particular tooth, due to the presence of the properioceptive nerve ending in the periodontal ligament.

Etiology of periapical periodontitis

1-Pulpitis and pulp necrosis:

If pulpitis is untreated, bacteria, bacterial toxins and the product of inflammation will extend down the root canal and through the apical foramina to cause periodontitis.

2-Trauma:

Occlusal trauma either from a high restoration or less frequently associated with bruxism, may result in periapical periodontitis under pressure during orthodontic treatment, a direct blow on tooth insufficient to cause pulp necrosis and biting unexpectedly on a hard body in food may all cause minor damage to the periodontal ligament and localized inflammation.

3- Endodontic treatment

Mechanical instrument through the apex during endodontic treatment as well as chemical irritation from root filling material may result in inflammation in the periapical periodontium. Instrumentation of an infected root canal may be followed by periapical inflammation, due to bacterial proliferation in the root canal or due to bacteria being forced into the periapical tissues.

1-Chronic apical periodontitis (periapical granuloma)

The term periapical granuloma refers to a mass of chronically or sub acutely inflamed granulation tissue at the apex of a non-vital tooth. The term is not totally accurate because the lesion does not show true granulomatous inflammation microscopically. The formation of the periapical granuloma represent a definitive reaction secondary to the presence of

microbial infection in the root canal with spread of related toxic products into the apical zone.

In the early stages of infection, neutrophils predominate, and radiographic changes are not present, this phase of periapical inflammation is termed acute periapical periodontitis.

The neutrophils release prostaglandins which activate osteoclasts to resorb the surrounding bone leading to detectable periapical radiolucency. With time, chronic inflammatory cells begin to dominate the host response. Mediators released by lymphocytes reduce further osteoclastic activity while also stimulating fibroblast and microvasculature.

For this reason chronic periapical granuloma is often asymptomatic and demonstrates little additional changes radiographically.

Clinical features

1-most of periapical granulomas are asymptomatic.

2-pain may develop if acute exacerbation occurs.

3-typically the involved tooth does not demonstrate mobility or significant sensitivity to percussion.

4-the soft tissue overlying the apex may or may not be tender

5-the tooth does not respond to thermal or electric pulp tests unless the pulp

necrosis is limited are limited to a single canal in a multirooted tooth.

Radiographic features

Most lesions are discovered on routine radiographic examination which may

show:

1-variable radiolucenies ranging from very small to 2 cm in diameter

2-affected teeth typically reveal loss of the apical lamina dura

3-the lesion may be circumscribed or ill-defined and may or may not

demonstrate a surrounding radiopaque rim

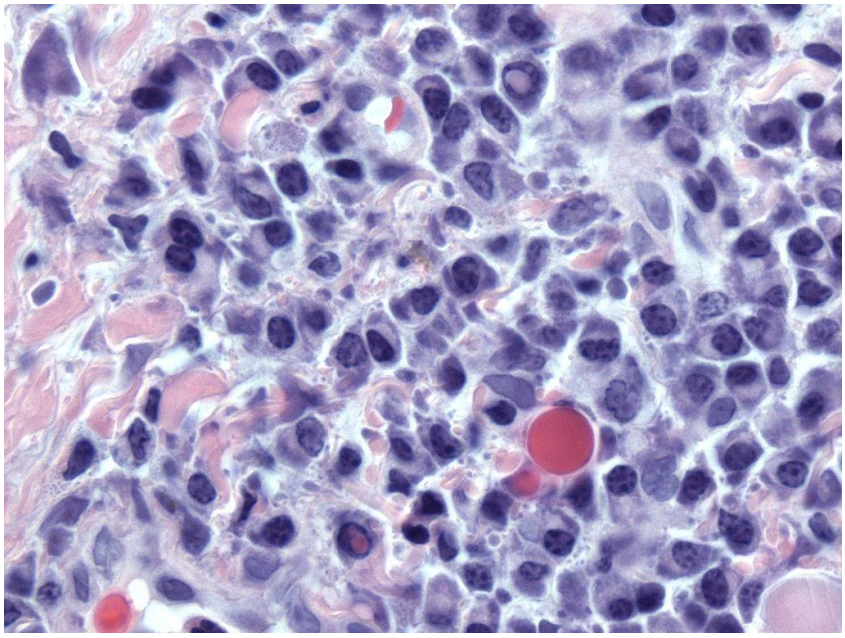
4- root resorption may be seen

The radiographic features are suggested but not diagnostic

Histopathological features

Periapical granulomas consist of an inflamed granulation tissue surrounded by fibrous connective tissue wall. The central part of the lesion contains macrophages with foamy cytoplasm caused by the phagocytosis of cholesterol.

Cholesterol crystals may be present surrounded by multinucleated giant cells. A diffuse infiltrate of lymphocytes and plasma cells. When numerous plasma cells are present, scattered eosinophilic globules of gamma globulin (Russell bodies) may be seen. A frequent finding is the presence of irregular islands of epithelium, a result of prolonged, mild stimulation of the rest malassez, which are remnants of the Hertwig's root sheath.



Treatment and prognosis

Periapical granuloma represents about 75% of apical inflammatory lesions and 50% of these failed to respond to conservative endodontic measures. Treatment depend on the reduction and control of the offending microorganisms or their toxic products in the root canal or apical tissues. A successful treatment depends on the complexity of the canal system and size of the periapical granuloma (more than 2 canals is difficult to be treated by conservative endodontic therapy Non restorable teeth may be extracted, followed by curettage of all apical tissues, with nonsteroidal anti-inflammatory drugs in symptomatic cases. Antibiotic are not recommended unless systemic signs and symptoms are present The teeth after conventional endodontic should be evaluated at 1-3-6 months and 1-2 years, to rule out possible causes of failure which includes

1-Cyst formation

2-Persistent pulpal infection

3-Extraradicular infection ((periapical Actinomycosis)

4-Accumulation of endogenous debris

5-Periapical foreign material

6-Periodontal diseases

7- Sinus penetration

8-fibrous scar formation, which is most frequently seen when both the facial and lingual cortical plates have been lost, which is not an indication for future surgery. If initial conventional therapy is unsuccessful, periapical surgery is indicated which include through curettage of all periradicular soft tissue, amputation of the apical portion of the root and scaling of the lumen of the canal, all tissues should be submitted for histopathological examination to exclude more serious conditions, like neoplastic process.

Sequelae:-

1- Periapical granuloma may continue to enlarge with continued bone resorption

2- Acute exacerbation to an acute periapical periodontitis

3- A suppuration to form an acute periapical abscess

4- Formation of a radicular cyst

5- Low grade irritation may cause osteosclerosis (bone apposition) or cementum

apposition (hypercementosis).

Acute periapical periodontitis

The factors leading to the treatment of an acute periapical periodontitis

include:-

1-young tooth with open tubules

2-rampant caries

3-closed acute pulpitis

4-presence of highly virulent micro-organisms

5-weakened host defense system

Histopathological findings:

Vascular dilatation, exudates of neutrophils, and oedema, in the periodontal ligament situated in the confined space between the root apex and the alveolar bone

Clinically:

Pain is intense when external pressure is applied to the tooth, as the pressure is transmitted through the fluid exudates to the sensory nerve endings. Even light load may be sufficient to induce pain, as the fluid is not compressible; the tooth feels elevated in its socket. Hot and cold stimulation does not cause pain.

The findings are often 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. If

radiological changes are present, they consist of slight widening of periodontal ligament and the lamina dura around the apex.

Sequela and prognosis

The inflammation may transient if it is due to acute trauma rather than infection and the condition seen resolves. If the irritant persist the inflammation becomes chronic and may be associated with resorption of the surrounding bone.

Suppuration may occur associated with necrosis and bacterial infection with continued exudation of neutrophils leading to abscess formation, called acute periapical abscess.

Acute periapical abscess

The accumulation of acute inflammatory cells at the apex of a nonvital tooth is termed a periapical abscess. It is a progression of an acute pulpitis in which exudates extend into the adjacent soft and hard tissue. Because it often contains one or more strains of virulent bacterial organisms, the exudates usually contains potent exotoxins and lytic enzymes capable of rapidly breaking down tissue barriers. Another cause is the acute exacerbation of a chronic periapical granuloma.

Clinical features

Patients have severe pain in the area of the nonvital tooth because of pressure and the effects of inflammatory chemical mediators on nerve tissue. The exudates and neutrophilic infiltrate of an abscess cause pressure on the surrounding tissue, often resulting in slight extrusion of the tooth from its socket.

Pus associated with a lesion, if not focally drained from the tooth ((e.g. by endodontic treatment)), seeks the path of least resistance and spread into contagious structures. The affected area of the jaw may be tender to palpation, and the patient may be hypersensitive to tooth percussion. The tooth is not responding to electric pulp tester, or thermal stimuli, headache, malaise, fever and chills may be present

Radiographic features:-

Abscess may demonstrate a thickening of apical periodontal ligament, an ill-defined radiolucency, or both. However, often no appreciable alterations can be detected because insufficient time has occurred for significant bone destruction.

If the condition is an exacerbation of a chronic periapical periodontitis or periapical granuloma. It could demonstrate the outline of the original chronic lesion with or without the associated bone loss.

Histopathology-Microscopically

A periapical abscess appears as a zone of liquefaction, composed of pertinacious exudates, necrotic tissue and viable and dead neutrophils, (pus).

Adjacent tissues containing dilated vessels and a neutrophilic infiltrate surrounds the area of liquefactive necrosis.

Sequelae

1-with progression, the abscess spreads along the path of least resistance and discharge into the oral cavity through a sinus tract following local penetration of overlying periosteium and mucosa. This is usually not painful. On other occasions the pus may accumulate beneath the mucosa and the patient may complain of a swelling at the intraoral opening of a sinus tract, which is a mass of sub acutely inflamed granulation tissue known as parulis ((Gum boil))

2- May extend through the medullary spaces away from the apical area, resulting in osteomyelitis

3-it may perforate the cortex and spread diffusely through the overlying soft tissue as cellulitis.

4-dental abscesses may discharge through the skin and drain via a cutaneous sinus.

5-periapical infection occasionally spread the blood stream and result in systemic symptoms such as fever, lymphadenopathy and malaise.

6-it may spread diffusely through facial planes of the soft tissues. This acute and edematous spread of an acute inflammatory process is termed cellulitis.

Cellulitis is a misnomer, because the process is not an inflammation of the cells but an acute condition in which purulent forms of bacteria, involve the facial and perioral mucosa.

The most common cause is extension from a periapical abscess. However other causes may also results in cellulitis like fractures.

Occasionally the exudates tracks onto the palate, producing a large swelling, when a periapical abscess erodes into the maxillary sinus, destroying the intervening bone and lining, and the offending tooth is extracted, a communication between the floor of the maxillary sinus and the oral cavity may result. This tract may remain permanently patent, particularly if it becomes lines by epithelium of the maxillary sinus and the oral cavity. This abnormal open communication is called oroantral fistula.

Involvement of the soft tissue and muscle overlying the maxilla usually result in perioral swelling. When the muscle layers overlying the body of the mandible are involved, patients experience a puffy swelling on the side of the face.

Extension of the pus lingually into the tissue spaces of the posterior floor of the mouth may result in swelling of the structures around the epiglottis which is a life threatening, as it restricts the airway and may cause suffocation.

Cellulitis of this area ((submental , submandibular and sublingual spaces)) is called Ludwig's angina.

Another serious complication is the extension of the exudates into the maxillary cavernous sinus area, resulting in thrombophlebitis. From this location fatal forms of brain abscess or acute meningitis are possible unless rapid intervention is undertaken.

Treatment and prognosis

Treatment of periapical abscess consist of drainage and elimination of the focus of infection Localized abscess should be drained by incision and drainage. If the abscess is localized with no systemic features ((fever, lymphadenopathy and malaise)), the patient is healthy, antibiotics are not recommended. However if the patient is compromised (e.g. diabetic) or, systemic symptoms are present antibiotics are recommended.

NSAID is needed if not contraindicated. The tooth should be endodontically treated or extracted. Sinus and fistula tracts if not treated spontaneously after extraction, should be removed surgically