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.

Numerous sequelae may follow untreated pulp necrosis and are dependent on the nature and behavior of lesions that form at the apex of the tooth.

The factors which may affect these lesions are

1- the presence of open or closed pulpitis.

2- irulence of the involved micro organisms.

3-extent of sclerosis of the dentinal tubules.

4-competency of the host immune response of the individual.

Where these factors are optimal e.g. the presence of an open chronic pulpitis, bacteria of few virulence, and an older tooth with sclerotic dentinal tubules in a healthy ((immune component)) individual, the changes at the apex of the tooth are mild and chronic.

Where the conditions are mostly adverse e.g. the presence of a closed acute pulpitis, large numbers of highly virulent bacteria, and open dentinal tubules of young teeth, the inflammation at the apex of the tooth will rapidly intensify and large amounts of bacterial toxins and autolytic enzymes will be produced and disseminated leading to rapid destruction of the periapical tissue and the surrounding bone ((e.g. acute periapical abscess)).

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.

Aetiology 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.

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 periodentitis (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 osteoclaststo 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 acivity 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 resporption 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 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 micro-organisms 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 facialand 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 liquifactive 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 openong of a sinus tract, which is a mass of subacutely 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

Radicular cyst

Clinical and radiographical features

Apical radicular cyst are the most common cystic lesions in the jaws and are always associated with apex of non vital teeth , they account for about 75% of all radicular cyst. When small they are frequently symptomless and are usually discovered during routine radiographical examination as they enlarge, they produce expansion of alveolar bone and ultimately may discharge through sinus. However the majority of radicular cyst does not grow to large dimension. The expansion of the alveolar bone is due to deposition of successful layers of new bone by overlying periosteium. As the cyst enlarge and cause bone resorption centrally. Increments of new sub periosteoal bone are lead down to maintain the integrity of the cortex. Producing a bony hard expansion. Howeverthe rates of expansion tens to out strip the rate of subperiosteol deposition. Leading to progressive thinning of the cortex which can be default on palpitation producing the clinical signs of oil can bottoming and egg shell is crackling. Eventually the cyst may perforate the cortex and present as a bluish fluctuant sub mucosal swelling. The rate of expansion of radicular cyst has been estimated at a proximately 5 mm diameter per year.

Pain is seldom a feature unless there is an acute exacerbation which may readily progress to abscess formation. The cyst can rise at any age after the tooth eruption but are rare in deciduous dentition. They are most common

between the ages of 20-60. They can occur in relation to ant tooth in the arch although 60% are found in the maxilla where there is a particular high incidence in anterior teeth. In addition to dental caries pulp death from trauma and irritant restorative material is more likely in anterior teeth than at other sites. Pulp death in maxillary lateral incisors may also be associated with an invaginated odontoma in the mandible the majority of cyst occur posterior to the canine tooth. Radiographically the apical radicular cyst presents as a round or avoid radiolucency at the root apex. The lesion is often well circumscribed and may be surrounded by peripheral radio-opaque margins continues with lamina dura of the involved tooth. However whether or not cyst formation has occurred in an apical radiolucency cannot be detected from radiographic appearance alone. The other varieties of radicular cyst are less common. The residual cyst is a radicular cyst that has remained in the jaw and failed to resolve following extraction of the involved tooth. About 20% of radicular cysts are of this type. However it should be noted that most periapical inflammation will resolve after removal of the causative agents. The reasons why some lesion persists as residual cyst are unknown. The lateral type is very uncommon and arises as a result of extension of inflammation from the pulp to into the lateral periodontal along the lateral root canal

Pathogenesis

Radicular cyst arises from proliferation of rest of malassez within chronic periapical granulomas but not all granulomas progress to cyst. The factors which determine why cystic transformation occurs in some and the mechanism involved in the formation of cyst are controversial. Persistence of chronic inflammatory stimuli are derived from the necrotic pulp appears essential since as mentioned above. Most periapical inflammation will resolve spontaneously once the causative agent has removed. It is assumed that the environment within chronically inflamed granuloma. Which is likely to be rich in cytokines

including growth factors? Stimulates the rate of malassez to proliferate strands and sheets of squamous epithelium derived from proliferation of the rest are common finding in the periapical granulomas. The mechanism of formation of an epithelial lined cyst cavity within granuloma is unclear. Two main mechanism have been proposed

1- Degeneration and death of central cells within a proliferating mass of epithelium. Epithelium is a vesicular and transport of metabolites and gaseous exchange occur by diffusion. It argued that when the mass proliferating epithelium within granuloma reaches a critical size. The central cells furthers away from the surrounding vascular bed. Degenerate and die, the micro cyst so formed then continues to expand
2- Degeneration and liquifactive necrosis of granulation tissue. It is suggested that areas of granulation tissue within the granuloma may undergo necrosis due to enclavement by proliferating strands of epithelium or to release toxic products from a dead pulp or from infecting organism. Epithelial proliferation to surround such an area of necrosis results in the formation of cyst.

Histopathology

Radicular cyst are lined wholly or impart by non-keratinized stratified squamous epithelium supported by a chronically inflamed fibrous tissue capsule. In a newly formed cyst the epithelial lining is irregular and may vary considerably in thickness. Hyperplasia is a prominent feature in long anastomosing cords of epithelium forming complex arcades extending into the surrounding capsule. The latter is richly vascular and diffusely infiltrated by inflammatory cells often predominant. In established cyst the epithelial lining is more regular in appearance and fairly even thickness breaks in the linings epithelial discontinuities are common. Metaplasia of epithelial lining may give rise to a mucus cell. Found in about 40% of radicular cyst lining and more

rarely ciliated cells and area of respiratory type epithelium. In approximately of cases the lining contains hyaline eosinophilic bodies Rushton bodies of varying size and shape. They appear to have no clinical or diagnostic significant and they origin is unknown. But they may represent some type of epithelial product. Within time the connective tissue capsule tends to become more fibrous and less vascular and there is reduction in the density of inflammatory cell infiltration, myofibroblast in capsule may help to constrain the tendency of the cyst to expand.

Deposits of cholesterol crystals are common within the capsules of many radicular cysts. In histological sections cholesterol clefts may be few in number of forms large mural nodules in which case they are often associated with epithelial discontinuity and project into cyst lumen. They are the probable of cholesterol crystals found in the cyst fluid; mural cholesterol clefts are associated with foreign body giant cells. As a periapical granulomas the cholesterol probably derived from the breakdown of red blood cells as a result of hemorrhage in the cyst capsule and deposits of hemosiderin are commonly associated with the clefts

Cyst contents

The cyst contents vary from a watery straw color fluid through to semi solid brownish material of paste like consistency. Cholesterol crystals impart a shimmering appearance the composition of cyst fluid is a complex of variable it is hypertonic compared with serum and contents

1- breakdown products of degenerating epithelial cell and inflammatory cell and connective tissue components

2- serum proteins all groups of serum proteins are present in cyst fluid and the soluble proteins level is 5-11 g/dl most are derived as inflammatory exudates.

Compared with serum the fluid contain higher level of immunoglobulin which probably reflect local production of plasma cells in the capsule

3- Water and electrolytes
4- Cholesterol crystals

Cyst expansion

- Cysts expansion is dependent on osteoclastic resorption of surrounding bone. Osteoclasts are derived from haematopoietic precursors and are transported via the blood.
- Osteoclasts are recruited to and activated at sites of resorption by mediators. The cytokines interleukin-1 and interleukin-6 (IL-1, IL-6) tumor necrosis factor and prostaglandin E2 are key mediators in cyst expansion.
- Mediators are generated locally by a variety of cells e.g.: macrophage, lymphocytes, epithelial cells, fibroblast.
- Activated osteoclast attached to the bone surface and release acids resulted in de mineralization. The organic matrix is then degraded by matrix metalloproteinase MMP's, collagenases, and lysosomal proteases.
- MMP's synthesized by other cells in the cyst wall e.g.: fibroblasts, epithelial and inflammatory cells, may contribute to matrix degradation.
- Bone resorption is followed by cyst expansion which may involve hydrostatic pressure.
- Cyst contents are hypertonic. The wall acts as a semi permeable membrane and retains the osmotically active molecules in the lumen creating an osmotic gradient. Water moves into the lumen along the gradient increasing the hydrostatic pressure in the cyst leading to enlargement.

- Enlargement is a complained by growth of the lining and the capsule. IL-1 and IL-6 stimulate epithelial proliferation other epithelial and fibroblast growth factors are also synthesized.

Treatment of radicular cyst:

The treatment of periapical radicular cyst depend on the condition of the tooth as whole, if the tooth is restorable, the root canals can be filled, if the root canals cannot be filled and the apical area is in a location accessible for surgery, an apicoectomy with complete surgical enuculation may be performed to remove the cystic lesion, followed by histopathological examination; otherwise, the tooth is extracted and the periapical cyst is curreted through the toothsocket.