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Periapical Periodontitis

Elitsa Deliverska, Milena Petkova

Departmennt of Oral and Maxillofacial surgery, Faculty of Dental medicine, Medical university- Sofia

Abstract: ***Lesions most commonly found at the apices of non-vital teeth are the periapical granuloma and radicular cyst. The treatment and prognosis may differ according to the lesion present. Many studies to determine the diagnostic features and incidence of these lesions have failed to reach a consensus view. The aim of this review is to enlight the treatment option of periapical lesion, whether surgery or not, necessitate precise diagnosis of the lesion as being acute periapical periodontitis, chronic periodontitis (granuloma), true cyst, or pocket cyst.***

Keywords: periapical inflammation, management

1. Introduction

Periapical inflammation is usually due to **spread of infection** following death of the pulp and accumulation of acute inflammatory cells at the apex of a nonvital tooth. It is termed **periapical abscess [1,2,3,4].** There is no evidence that necrotic pulp tissue per se can elicit periapical inflammation, in the absence of bacteria.[5]

It characteristically causes tenderness of the tooth in its socket.

2. Classification

Tissue response to pulpal inflammation caused by microorganism invasion is mainly from [4]:

- Caries
- Trauma
- Medical induced
- Allergy.
- There is no evidence that necrotic pulp tissue can elicit periapical inflammation, in the absence of bacteria.

Infection is far the most common **cause.** The usual sequence of events is caries, pulpitis, death of the pulp and periodontitis. Frequently, the source of the infection is obvious.

On occasion, however, pulpal death may be **trauma** related, and the tooth may contain neither a cavity nor a restoration. The pulp in this case dies from the blow (trauma) which damages the apical vessels. The necrotic pulp probably becomes infected by bacteria from the gingival margins, leading to apical periodontitis. A high filling, or biting suddenly on a hard object, sometimes cause an acute but usually transient periodontitis. During endodontic treatment, instruments may be pushed through the apex or side of the root, damaging the periodontal membrane and carrying infected debris from the pulp chamber into the wound.

Medical induced: irritant antiseptics used to sterilise a root canal can escape through the apex and damage the surrounding tissues. A root-canal filling may also extend beyond the apex with similar effect.

Allergic periodontitis due to allergic reaction to some agents used in root canal treatment.[4]

Periapical periodontitis must be distinguished from **marginal periodontitis**, in which infection and destruction of the margins.

Many investigators subdivide periapical abscesses into **acute** and **chronic** types.

On the basis of their clinical presentations, periapical abscesses should be designated as **symptomatic** or **asymptomatic**. Periapical abscesses become symptomatic as the purulent material accumulates within the alveolus.

Diagnosis of periapical inflammation is made by interpretation of a combination of symptoms and clinical and radiological signs[1,2,3,4]

3. Acute Periapical Periodontitis

Acute inflammatory lesions with abscess formation may arise as the **initial periapical pathosis** (de novo).

In the earliest stage of all forms of periapical inflammatory disease, the periapical periodontal ligament (PDL) fibers may exhibit acute inflammation but no frank abscess formation. This localized alteration, best termed **acute apical periodontitis,** may or may not proceed to abscess formation.

Although this process often occurs in association with a nonvital tooth, acute apical periodontitis may be found in vital teeth secondary to trauma, high occlusal contacts, or wedging by a foreign object.

4. Clinical Features

The tooth should be **non-vital** to simple tests- it **does not respond** to cold and hot or electric pulp testing (as the periapical inflammation is usually provoked by a dead and/or infected pulp) although, particularly with multirooted teeth, some vital response may still be elicited. There is often a large carious cavity or filling in the affected tooth, or it may be discoloured due to death of the pulp earlier. The patient may give a history of pain due to previous pulpitis. Periodontitis could be divided to serous and purolent.[1,2,3,6]

There are four stages of acute purulent periapical periodontitis: periodontal, endosteal, subperiosteal, submucosal, depends on the progression of the disease and the pathway of exudate.

The initial stages produce **tenderness** of the affected tooth- **even to mere touch-** that often is **relieved by** direct application of **pressure**. With progression, the **pain** becomes more **intense**, often with **extreme sensitivity to percussion** and **extrusion of the tooth** (due to the escape of exudate into the periodontal ligament). The affected tooth is **painful to touch.**

As inflammation becomes more severe and pus starts to form, **pain** becomes intense and **throbbing in character**. It may be difficult for the patient to determine whether an upper or lower tooth is affected.

At this stage the **gingiva over the root is red and tender**, but there is **no swelling while inflammation is confined within the bone**.

With progression, the abscess spreads **along the path of least resistance**. The purulence may **extend through the medullary spaces** away from the apical area, resulting in **osteomyelitis,** or it may **perforate the cortex** and spread diffusely through the overlying soft tissue (as **cellulitis**). Once an abscess is **in soft tissue**, it can cause cellulitis or may channelize through the overlying soft tissue. The cortical plate may be perforated in a location that permits entrance into the oral cavity. 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. [1,2,3]

The **regional lymph nodes may** be enlarged and tender, but **general symptoms are usually slight or absent**. Inflammation typically remains localised.

Acute periapical periodontitis may also occur **after trauma** or endodontic treatment to a tooth. In such cases, the history should lead to the diagnosis.

5. Radiology

Often no appreciable alterations can be detected because insufficient time has occurred for significant bone destruction. At this point, radiographs **give little information**. [1,2]

Immediately round the apex the lamina dura may appear **slightly hazy (loss of the lamina dura) and the periodontal space may be slightly widened**. When there is acute periapical abscess due to **exacerbation of a chronic infection**, the original lesion can be seen as **an area of radiolucency** at the apex.[3]

6. Pathology

Acute apical periodontitis is a typical acute inflammatory reaction with engorged blood vessels and packing of the tissue with neutrophil leukocytes and macrophages. These changes are initially localised to the immediate vicinity of the apex as a consequence of the rich blood supply to this area. The immediately adjacent lamina dura becomes resorbed and an abscess cavity may form if not aborted by treatment .[1,2,3,6]

Key features:

- Dull throbbing constant pain
- Pain on biting or percussion- most severe in subperioseat stage
- Palpation – pain in subperiosteal and submucosal stage.
- Facial asymmetry- in subperiosteal and submucosal stage.
- Negative or delayed vitality test response
- Not associated with apical radiolucency
- Widening of PDL space

7. Treatment

If tooth is in hyper occlusion, relieve occlusion

Treatment plan

1. If tooth is infected, initiate endodontic therapy or extraction
2. Incision and drainage should be done in subperiosteal and submucosal stage

8. Chronic Apical Periodontits

Chronic periodontitis is a **low-grade infection**. It may follow an acute infection that has been inadequately drained and incompletely resolved. It can occure **acute exacerbation** of a pre-existing chronic periapical inflammatory lesion.

Clessification[4]

Periodontitis chronica granulomatosa diffusa cum/ sine fistula

Periododntitis chronica granulomatosa localisata:

- Granuloma simplex
- Granuloma epithelisatum
- Granuloma cysticum

Periodontitis chronica fibrosa

Periodontitis chronica granulomatosa exacerbata

9. Clinical Features

There may be few or no symptoms. The tooth is non-vital and may be **slightly tender to percussion** but otherwise **symptoms may be minimal**.[1]

If a **chronic path of drainage** is achieved, a chronic periodontitis 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. Occasionally, the associated nonvital tooth with 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. If the **drainage site becomes blocked**, then signs and **symptoms of the abscess** frequently become

evident in a short time. Those periodontitis associated with a patent fistulous tract may be asymptomatic but, nevertheless, should be treated.[1,2,3]

Lymphadenopathy, headache, malaise, fever, and chills may be present.

The risk of dissemination through the bloodstream appears to be less for periapical abscesses that drain freely.

Complications, such as cavernous sinus thrombosis, mediastinitis, cervical necrotizing fasciitis, and cerebral abscess, can be life threatening.

10. Radiology

The initial sign is widening of the periodontal ligament space with preservation of the radio-opaque lamina dura. This naturally progresses with time to form a rounded periapical radiolucency with a well-defined margin - a periapical granuloma. Ultimately, this may undergo cystic change to radicular cyst. Differentiation between a large granuloma and a small radicular cyst is not possible on purely radiological grounds, but lesions greater than 1 cm diameter are often assumed to be cysts until histopathological diagnosis is established. [2,3]

A further radiological sign frequently seen in chronic periapical periodontitis is sclerosing (or condensing) osteitis. This appears as a fairly diffuse radioopacity, usually around the periphery of a widened periodontal ligament or a periapical granuloma.[1,2]

11. Pathology

Chronic periapical periodontitis is characterised by the formation of granulation tissue derived from the periodontal ligament, the periapical granuloma, surrounding the apex of a tooth. Chronic inflammatory cells infiltrate the granuloma in variable numbers. Often plasma cells predominate because of multiple antigenic stimulations from pulpal infection. Foamy macrophages, cholesterol clefts often rimmed by mulrinucleate giant cells, and deposits of haemosiderin are also frequent findings. Remnants of Hertwig's root sheath, the cell rests of Malassez, may proliferate as a result of release of inflammatory mediators. Neutrophil infiltration within this epithelium may be one factor leading to cavitation and formation of a radicular cyst.[1,6]

Progression of disese:
Caries, trauma, periodontal disease--- Pulp necrosis---- Apical bone inflammation---- Granuloma formation----- Stimulation, then proliferation of epithelial cell rests of Malassez----- Cystification

Differntial diagnosis of acute and chronic periapical periodontitis. Radiolucent and radiopaque lesions of non-endodontic lesion mimic the radiographic appearance of endodontic lesion. Pulp vitality test is important aid. Differential diagnosis could be made with: pulpitis osteomyelitis of jaws, maxillary sinuitis, denticle, neuralgia, lateral periodontal cyst, globulomaxillar cyst, osteoblastoclastoma, odontogenic fibroma, myxoma, ameloblastoma etc.[1,2,3,4]

Local **(periapical)** periodontitis must be distinguished from chronic (**marginal**) periodontitis, in which infection and destruction of the supporting tissues spread from chronic infection of the gingival margins, and the pulp is vital.

12. Treatment of Periapical Periodontitis

Treatment of the patient with a periapical abscess consists of **drainage and elimination of the source of infection.**

With localized periapical abscesses, the signs and symptoms typically diminish significantly within 48 hours of initiation of appropriate drainage. When the abscess causes clinical expansion of the bone or soft tissue adjacent to the apex of the affected tooth, **incisional drainage of the swelling** should be considered because this technique appears to be associated with more rapid resolution of the inflammatory process when compared with drainage through the root canal. If the affected tooth is extruded, then **reduction of the occlusion** is recommended because chronic occlusal trauma has been shown to delay resolution of the inflammatory process. [1,2,3]

Tretament of chronic diffuse periapical periodontitis include extraction and curettage of apical zone if the tooth is not prospective.

If the tooth is prospective: RCT **with or without** apicoectomy.(with retrograde filling: MTA, Bio dentine, amalgam etc.).

Tretament of chronic localised periapical periodontitis include extraction and curettage of apical zone if the tooth is not prospective.

If the tooth is prospective: RCT **with** apicoectomy.(with retrograde filling: MTA, Bio dentine, amalgam etc.).

Also in treatment plan in chronic periapical periodontitis could be perform hemisection, root amputation and bicuspidation.[4]

Unless contraindicated, treatment with **NSAIDs** usually is appropriate preoperatively, immediately postoperatively, and for subsequent pain control. Typically, use of **antibiotic medications** for a well-localized and easily drained periapical abscess in a healthy patient is unnecessary. Antibiotic coverage should be reserved **for the medically compromised and patients with signifi cant cellulitis or clinical evidence of dissemination** (i.e., fever, lymphadenopathy, malaise). Patients with significant cellulitis must be treated aggressively and monitored closely.

Once the infection has been resolved by extraction or **appropriate endodontic therapy**, the affected bone typically heals. Persistence of chronic periodontitis after root canal treatment is usually due to technical faults, and **apicectomy** may be required.

Usually, a **sinus tract** resolves spontaneously after the offending tooth is extracted or endodontically treated. Sinus tracts that persist are thought to contain sufficient infectious material along the fistulous tract to maintain the surface granulation tissue, and surgical removal with curettage of the tract is required for resolution.[1,2,3]

In cases of **post-traumatic acute periapical periodontitis**, the inflammation may resolve with splinting and time. [1]

Healing process occurs after removal of irritants, inflammatory response tissue organization and maturation occur. Level of healing is proportional to extend of tissue injury and nature of tissue destruction.

Bone that is resorbed is replaced by new bone; resorbed cementum and dentin are repaired by cellular cementum. PDL that is first affected is restored in the last to normal.

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