

Endodontics

Lecture 2

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PULP AND PERI-RADICULAR PATHOLOGY

Etiology of pulpal diseases can be broadly classified into:

I. Bacterial irritant

Bacteria, usually from dental caries, are the main source of injury to the pulpal and periradicular tissues and they enter either directly or through dentine tubules.

Modes of entry for bacteria to the pulp are as follows:

- 1-Through the carious cavity.
- 2-Through the dentinal tubules as in contamination during cavity preparation, through exposed root surface, and surfaces with erosion, abrasion and attrition.
- 3-Through the apical foramen as in advanced periodontitis where microorganisms reach the apical foramen and then the pulp.
- 4-Through the blood stream (anachoresis: it is a process by which microorganisms get carried by the bloodstream from another source localize on inflamed tissue). Following trauma or inflammation to the pulp any bacteria in the blood might be attracted to the pulp causing pulpitis.
- 5-Through faulty tooth restoration.
- 6-Through extension of a periapical infection from adjacent infected tooth.

Bacteria most often recovered from infected vital pulps are:

- .Streptococci
- .Staphylococci
- . Diphtheroids, etc.

II. Mechanical irritants

Examples of mechanical irritation include trauma. operative procedures, excessive orthodontic forces, subgingival scaling and overinstrumentation using root canal instruments.

III. Chemical irritants

Pulpal irritation may result from bacterial toxins or some restorative materials/conditioning agents.

Peri-radicular_ irritation may occur from irrigating solutions, phenol-based intra-canal medicaments or extrusion of root canal filling materials.

IV. Radiation injury to pulp

Radiation therapy affects pulps of fully formed teeth in patients exposed to radiation therapy. The pulp cells exposed to ionizing radiation may become necrotic, there may occur vascular damage and the interference in mitosis of cells.

Classification of Pulp Disease

Diagnosis of pulp disease is usually based on patient symptoms and clinical findings. Pulpal disease may result in changes to both the soft and hard tissues.

❖ Soft tissue changes

✚ Reversible pulpitis (Pulpal Hyperaemia): It is a transient condition that may be precipitated by any insult (ex. caries) to the pulp and characterised by increase in vascular vasodilation.

The symptoms are usually:

- Pain needs an external stimulus and it subsides immediately after removal of stimulus.
- Pain is difficult to localise (as the pulp does not contain proprioceptive fibers).
- Normal periradicular radiographic appearance.
- Teeth are not tender to percussion but sensitive to cold stimulus.

Treatment involves covering up exposed dentine, removing the stimulus or dressing the tooth.

✚ Irreversible pulpitis: Irreversible pulpitis usually occurs as a result of more severe insults than in the reversible pulpitis. It may develop as a progression from a reversible state.

1) Acute pulpitis: The pulp experiences increased inflammatory process and intrapulpal pressure. The symptoms experienced are:


- Severe pain develops spontaneously or from stimuli which may last from minutes to hours.
- Heat stimulus increases pain due to expansion of blood vessels therefore increasing pressure in the pulp.
- Cold stimulus decreases pain due to contractile action on the blood vessels therefore lowering intrapulpal pressure.
- Not tender to percussion and normal radiographic apical region
- A widened periodontal ligament may be seen radiographically in the later stages.

Treatment involves either root canal therapy or extraction of the tooth.

2) Chronic pulpitis: After the acute phase the pulp might enter the chronic phase. The symptoms experienced are:

- Mild to moderate intermittent pain may be tolerated by the patient for long period of time.
- Thermal tests are of little value.
- Tenderness to percussion and radiographic changes are not seen until infection reaches the periapical region.

Treatment involves either root canal therapy or extraction of the tooth.

 **Hyperplastic pulpitis:** Hyperplastic pulpitis is a form of irreversible chronic pulpitis and is also known as a pulp polyp. It occurs as a result of proliferation of chronically inflamed young pulp tissue. Treatment involves root canal therapy or extraction.

Pulp necrosis: Pulp necrosis occurs as the end result of irreversible pulpitis; **treatment** involves root canal therapy or extraction.

❖ **Hard tissue changes**

Pulp calcification: Physiological secondary dentine is formed after tooth eruption and the completion of root development. It is a condition in which hardening, or calcification, of pulp tissue results in hypersensitivity and extreme pain because the dental nerves become compressed. A root canal is usually necessary to clear away hardened tissue. Pulp calcification is deposited on the floor and ceiling of the pulp chamber rather than the walls and with time can result in occlusion of the pulp chamber. Pulp calcification may be composed of irregular dentine (true denticle) or due to ectopic calcification of pulp tissue (false denticle). Tertiary dentine is laid down in response to environmental stimuli as reparative dentine where it is deposited directly beneath the path of injured dentinal tubules as a response to strong noxious stimuli. Treatment is dependent upon the pulpal symptomst

Internal resorption: Internal resorption is initiated within the pulp cavity and results in loss of substance from dentinal tissue. Occasionally, pulpal inflammation may cause changes that result in dentinoclastic activity. Such changes result in resorption of dentine; clinically, a pink spot may be seen in the later stages if the lesion is in the crown. Radiographic examination reveals a radiolucency that is seen to be continuous with the rest of the pulp cavity. Root canal therapy will result in arrest of the resorptive process; however, if destruction is very advanced extraction may be required.

External resorption: External root resorption is not a pulp dystrophy for its origin lies within the tissue of the periodontal membrane space. It is the removal of the mineralized and organic components of dental tissues by clastic cells. In the case of external root resorption, this may be a response that may occur following trauma or orthodontic tooth movement and is called **physiological root resorption**. All other forms of external root resorption are **progressive** and **are called Inflammatory (infective) root resorption** and usually results from luxation injury and is caused by the transmission of bacterial toxins from a devitalized and infected pulp via dentinal tubules to an external resorbed root surface.

Clastic cells are stimulated to the region by inflammatory mediators. A diagnosis of **inflammatory root resorption**, which is characterized radiographically by bowl-like radiolucencies in both the tooth and the adjacent bone, is also diagnostic of an infected and probably totally necrotic pulp.

Treatment: Early root-canal debridement and medication with calcium hydroxide paste is recommended.

Classification of periapical disease

Untreated pulpal infection leads to total pulp necrosis. If left untreated, irritants leak into periapical region forming periapex pathologies. Severity of periapical inflammation is related to microorganisms in root canals and the length of exposure to infecting microorganisms.

Acute apical periodontitis AAP

Acute apical periodontitis is defined as painful inflammation of the periodontium because of occlusal trauma, egress of bacteria from infected pulps, toxins from necrotic pulps, chemicals, irrigants or over instrumentation in root canal therapy. Clinically, the tooth is tender to biting. The distinctive features of AAP are dull throbbing constant pain, it occurs over a short period of time. Cold stuff may relieve pain, whereas heat may exacerbate pain. Widening of the periodontal ligament space may be seen on a radiograph.

Treatment depends on pulpal diagnosis it may range from occlusal adjustment to root canal therapy or extraction.

Chronic apical periodontitis

Chronic apical periodontitis occurs because of pulp necrosis. Affected teeth do not respond to pulp sensitivity tests. Tenderness to biting is usually mild however, some tenderness may be noted to palpation over the root apex, radiographic appearance is varied ranging from minimal widening of the

periodontal ligament space to a large area of destruction of periapical tissues. Treatment involves root canal therapy or extraction.

Condensing osteitis

Condensing osteitis is a variant of chronic apical periodontitis and represents a diffuse increase in trabecular bone in response to irritation. Radiographically, a concentric radio_ opaque area is seen around the offending root. Treatment is only required if symptoms/pulpal diagnosis indicate a need.

Acute apical abscess

It is a localized collection of pus in the alveolar bone at the root apex of the tooth, following the death of pulp with extension of the infection through the apical foramen into periradicular tissue. The most common cause of Acute Apical Abscess is invasion of bacteria from necrotic pulp tissue. Symptoms vary from moderate discomfort or swelling to systemic involvement, such as raised temperature and malaise. Teeth involved are usually tender to both palpation and percussion. The tooth is non vital and the pain is being of rapid onset with Readily localized as tooth becomes increasingly tender to percussion.

Radiographic changes are variable depending on the amount of periradicular destruction already present; however, usually there is a well-defined radiolucent area. As in many situations, an acute apical abscess is an acute exacerbation of a chronic situation. Initial treatment of an acute apical abscess involves removal of the cause as soon as possible. Drainage should be established either by opening the tooth or incision into a related swelling. An antibiotic may need to be prescribed, depending on the patient's condition. Once the acute symptoms have subsided, then root canal therapy or extraction may be performed. If the apical abscess is not treated, it will spread to surrounding tissues

Chronic apical abscess

Chronic periapical abscess is also known as suppurative apical periodontitis, which is associated with gradual egress of irritants from root canal system into periradicular area leading to formation of an exudate. In a chronic apical abscess, the abscess has formed a communication through which it discharges. Such communications may be through an intraoral sinus or, less commonly, extraorally. Alternatively the discharge may be along the periodontal ligament; such cases resemble a periodontal pocket. Usually these

communications or tracts heal spontaneously following root canal therapy or extraction.

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