

Lecture 18

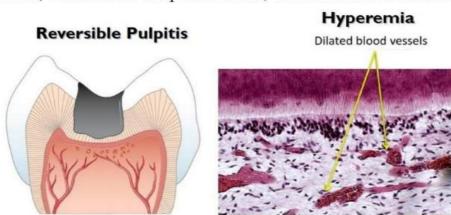
Classification of Pulpal and Periapical Diseases

Pulpal Diseases

Diagnosis of pulp and periapical disease is usually based on patient symptoms and clinical findings. Pathological conditions of the pulp (pulpal disease) can be classified into the following:

I. Reversible pulpitis (Pulpal hyperemia):

A mild-to-moderate inflammatory condition of pulp caused by noxious stimuli in which the pulp can return to a normal state following removal of stimuli. Determination of reversibility is the clinical judgment influenced by the patient's history and clinical evaluation. Actiology includes dental caries, trauma after deep restoration, and thermal and chemical injuries.



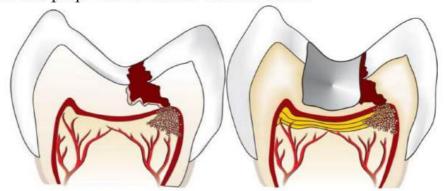
The symptoms are:

- a) Sharp momentary pain caused by cold.
- b) Pain is not spontaneous; it needs an external stimulus and subsides immediately after removing it.
- c) Normal periradicular radiographic appearance on X-ray radiograph.
- **d)** Teeth are not tender to percussion but sensitive to a cold stimulus.

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

II. Irreversible pulpitis:

"It is a persistent inflammatory condition of the pulp, symptomatic or asymptomatic, caused by a noxious stimulus." Irreversible pulpitis usually occurs as a result of more severe insults than reversible pulpitis. It may develop as a progression from a reversible state. Histologically, a pulp with irreversible inflammation is characterized by the presence of an area of liquefaction necrosis compared to a pulp with reversible inflammation.



Irreversible pulpitis is divided into:

a) Symptomatic Irreversible pulpitis (previously known as acute irreversible pulpitis):

This condition is usually associated with acute symptoms, which include:

- Severe pain develops spontaneously or from stimuli that may last minutes to hours after exposure to cold or hot stimuli. Pain is sharp, throbbing, intermittent, or continuous.
- Pain is exacerbated on bending down or lying down due to a change in intrapulpal pressure from stand to supine.
- Pain is so severe that it keeps the patient awake at night.
- · Presence of referred pain.
- In a later stage, heat stimulus increases pain due to the 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.
- It is not tender to percussion and normal radiographic apical region in the early stage.
- A widened periodontal ligament may be seen radiographically in the later stages.

Treatment involves either pulpectomy (removal of the infected pulp) followed by a root canal filling or extraction of the tooth.

b) Asymptomatic irreversible (previously known as chronic irreversible pulpitis):

After the acute phase, the pulp might enter the chronic phase. The symptoms experienced are:

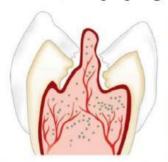
- The patient may tolerate mild to moderate intermittent pain for a long period.
- Thermal tests are of little value.
- Tenderness to percussion and radiographic changes are not seen until infection reaches the periapical region.

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

c) Hyperplastic pulpitis: Hyperplastic pulpitis is a form of irreversible chronic pulpitis known as a pulp polyp. It is an inflammatory response of pulpal connective tissue due to extensive carious exposure to young pulp. It shows the overgrowth of granulomatous tissue into the carious cavity. It is commonly seen in the teeth of children and adolescents because this pulp

tissue has high resistance, and large carious lesions permit a free proliferation of hyperplastic tissue. The symptoms include:

- Usually, it is asymptomatic; fleshy pulpal tissue fills the pulp chamber.
- It is less sensitive than normal pulp but bleeds easily due to a rich network of blood vessels.
- · Sometimes, this pulpal growth interferes with chewing.

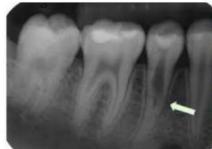




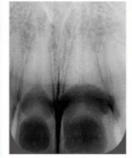


d) Internal resorption: Internal resorption is initiated within the pulp cavity and results in the loss of substance from dentinal tissue. Aetiology is unknown, but occasionally, pulpal inflammation may cause changes that result in dentinoclastic activity. Such changes result in resorption of dentin; clinically, it is asymptomatic unless perforation of the root occurs; a pink spot may be seen in the later stages if the lesion is in the crown. Radiographic examination reveals a continuous radiolucency with the rest of the pulp cavity. Root canal therapy will arrest the resorptive process; however, if destruction is very advanced, extraction may be required.











III. Pulp necrosis:

"Pulp necrosis or death is a condition following untreated pulpitis. Pulpal tissue becomes nonvital, and if the condition is not treated, noxious materials will leak from the pulp space, forming a lesion of endodontic origin. Symptoms include:

- The tooth might be asymptomatic.
- The discolouration of the tooth is due to the extravasation of pulpal blood into the dentin.



Radiograph shows a large cavity or restoration or normal appearance unless there is concomitant apical periodontitis or condensing osteitis. Treatment involves root canal therapy or extraction.

IV. Pulp calcification:

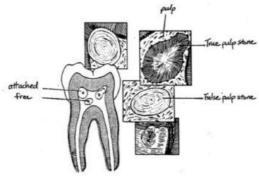
"In which part of the pulp tissue is replaced by calcific material, and mainly there are three types of calcifications are seen in pulp:

- Dystrophic calcifications: The foci of these calcifications can begin in dead and degenerated tissue, blood clots, connective tissue walls of blood vessels and nerves of the pulp because of the inflammation.
- Diffuse calcifications: These are usually linear calcifications present in the root canal.
- Denticles/pulp stones: They are present in the pulp chamber and can be classified according
 to location as either a free stone, embedded stone in dentin, or an attached stone.



Also, pulp stones can be classified according to structure into:

- ♣ True denticle: It is composed of dentin formed from detached odontoblasts or fragments of Hertwig's enamel root sheath, which stimulates the undifferentiated cells to assume dentinoblastic activity.
- ♣ False denticle: Here, degenerated tissue structures act as a nidus for depositing concentric layers of calcified tissues.



Periapical Diseases

Untreated pulpal infection leads to total pulp necrosis. If left untreated, irritants leak into the periapical region, forming periapical pathologies. The severity of periapical inflammation is

related to microorganisms in root canals and the length of exposure to infecting microorganisms. Periapical diseases can be classified into:

- 1. Symptomatic apical periodontitis (acute apical periodontitis).
- 2. Asymptomatic apical periodontitis (chronic apical periodontitis).
- 3. Acute apical abscess.
- 4. Chronic apical abscess.
- 5. Condensing osteitis.
- 6. Acute exacerbation of asymptomatic apical periodontitis.

1. Symptomatic apical periodontitis (acute apical periodontitis):

Symptomatic apical periodontitis is defined as painful inflammation of the periodontium as a result of trauma, irritation, or infection through the root canal, regardless of whether the pulp is vital or nonvital. Etiology includes occlusal trauma, high points in restoration, sequelae to pulpal diseases, over-instrumentation, pushing debris and microorganisms beyond the apex, overextended obturation, and root perforations.



The symptoms include the tooth being tender on percussion, which may present mild-to-severe soreness, dull, throbbing, and constant pain. Pain occurs over a short period, pain on mastication. Diagnosis reveals that the tooth is tender on percussion.

A radiographic picture of a vital tooth may show no change, whereas, in the case of a nonvital tooth, it may show a widening of apical periodontal ligament space and loss of lamina dura. Treatment includes occlusion adjustment in vital teeth and root canal treatment in nonvital teeth.

2. Asymptomatic apical periodontitis (chronic apical periodontitis):

It is the sequelae of symptomatic apical periodontitis resulting in inflammation and destruction of periradicular area due to extension of pulpal infection, characterized by asymptomatic periradicular radiolucency on radiographs. The etiology is pulp necrosis. The tooth is nonvital, usually asymptomatic, but dull and throbbing pain may be present in the acute phase.

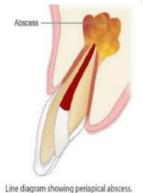
Treatment includes endodontic therapy of the affected tooth; in the acute phase, treatment is the same as acute apical abscess, i.e. cleaning and shaping of canals followed by analgesics if required and extraction of nonrestorable teeth.



3. Aute apical abscess:

It is a localized collection of pus in the alveolar bone at the root apex of the tooth, following the death of the pulp with an extension of the infection through the apical foramen into periradicular tissue. The most common cause of acute apical abscess is an invasion of bacteria from necrotic pulp tissue. Symptoms vary from moderate discomfort or swelling to systemic involvement, such as raised temperature and malaise. The teeth involved are usually tender to both palpation and percussion. The tooth is nonvital, and the pain has a rapid onset and is readily localized as the 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 the removal of the cause as soon as possible. Drainage should be established by opening the tooth or incision into a related swelling. Depending on the patient's condition, an antibiotic may need to be prescribed. Once the acute symptoms have subsided, root canal therapy or extraction may be performed. If the apical abscess is not treated, it will spread to surrounding tissues.







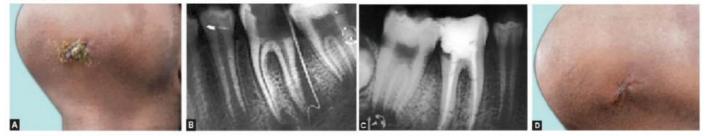


4. Chronic apical abscess:

Chronic periapical abscess is also known as suppurative apical periodontitis, which is associated with the gradual egress of irritants from the root canal system into the periradicular area, leading to the 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.



5. 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 radioopaque area is seen around the offending root. Treatment is only required if symptoms/pulpal diagnosis indicate a need.



6. Acute exacerbation of asymptomatic apical periodontitis:

Also known as "Phoenix abscess," it is an acute inflammatory reaction superimposed on an existing asymptomatic apical periodontitis. Etiology includes the influx of necrotic products from diseased pulp or bacteria and their toxins into chronic periapical lesions that can cause the dormant lesion to react. This leads to the initiation of acute inflammatory response. Lowered body defences also trigger an acute inflammatory response. Symptoms are similar to an acute apical abscess. Phoenix abscess should be differentiated from acute apical abscess by the patient's history, symptoms, and clinical test results. Radiographs show a large area of radiolucency in the apex created by inflammatory connective tissue, which has replaced the alveolar bone at the root apex. Treatment includes drainage and root canal treatment once symptoms subside.