Diseases Of Pulp & & Periapical Tissues

PULPITIS

- Pulpitis is the most common cause of pain and loss of teeth in younger persons.
- The usual cause is caries penetrating the dentine but there are other possibilities of pulpitis.
- If untreated, is followed by death of the pulp and spread of infection through the apical foramina into the periapical tissue.

https://dentistrykey.com/library/examination-and-diagnosis-of-pulp-root-ca nal-and-periapical-periradicular-conditions/

CAUSES OF PULP DISEASE

- The causes of pulp disease are Physical, Chemical and Bacterial.
- D Physical
 - a. Mechanical
 - Trauma:
 - . Accidental
 - . Iatrogenic dental procedures
 - Pathological wear
 - Crack through body of tooth
 - b. Thermal
 - Heat from cavity preparation
 - Exothermic heat from setting of cements
 - **c.Electrical** (galvanic current from dissimilar metallic filling)

2. Chemical

- Phosphoric acid, acrylic monomer, etc.
- -Erosion (acids)
- 3. Bacterial
- Toxin associated with caries
- Direct invasion of pulp from caries or trauma
- Microbial colonization in the pulp by blood-borne microorganisms.

CLASSIFICATION

I. According to pathological condition: -Focal or acute reversible pulpitis (Pulp hyperaemia) -Irreversible pulpitis II. According to its duration: --Acute pulpitis -Chronic pulpitis III. According to presence of dentin covering the pulp chamber: -

- -Open pulpitis
- -Closed pulpitis

CLASSIFICATION

- IV. According to extension of inflammation in pulp tissue: -
 - Partial pulpitis
 - Complete / total pulpitis
 - V. According to amount of pus formation: -
 - Exudative pulpitis
 - Suppurative pulpitis

Pulp state

No signs of disease:

Clinically normal pulp (based on history, clinical examination, tests, radiographs, etc.)

Pulpitis:

- Reversible pulpitis acute*
 - chronic*
- Irreversible pulpitis acute*
 - chronic*

* Notes:

- Acute—rapid onset and a short, severe course; moderate to severe pain; patient is seeking urgent relief of the symptoms.
- Chronic—long-standing; no pain or only mild and occasional pain; patient not seeking urgent relief.

Pulp necrosis:

- Pulp necrobiosis (part of pulp is necrotic and infected; the rest is irreversibly inflamed)
- Pulp necrosis with NO signs of infection
- Necrotic and infected pulp

Pulpless teeth:

- Pulpless and infected root canal system
- Teeth with previous root canal treatment
 - Root-filled with NO signs of infection
 - ◊ Root-filled and infected root canal system
 - Incomplete endodontic treatment** (e.g., treatment started elsewhere) ** Note: Must also specify whether the root canal system is infected or not
 - Technical standard (based on the radiographic appearance)
 - » Adequate
 - Inadequate
 - Other problems—specify the problem: e.g., perforation, untreated canal(s), fractured instrument, silver point, etc.)

Degenerative changes:

- Pulp canal calcification** (based on radiographic appearance)
 - ** Note: Must also specify the diagnosis/condition of the pulp or root canal system
- Pulp hyperplasia (note: a form of irreversible pulpitis; specify whether acute or chronic)

FOCAL REVERSIBLE PULPITIS (PULP HYPEREMIA)

Mild, transient, localized inflammatory response.

It is a reversible condition .

CLINICAL FEATURES:

Caries - pain from cold test disappeares immediately.

Hyperemia -pain from cold test does not linger more than 30 s. No percussion sensitivity, no spontaneous pain, no heat sensitivity. Affected tooth responds to stimulation of electric pulp tester at lower level of current indicating low pain threshold.

Teeth usually show deep caries, metallic restoration with defective margins.

HISTOLOGICAL FEATURES:

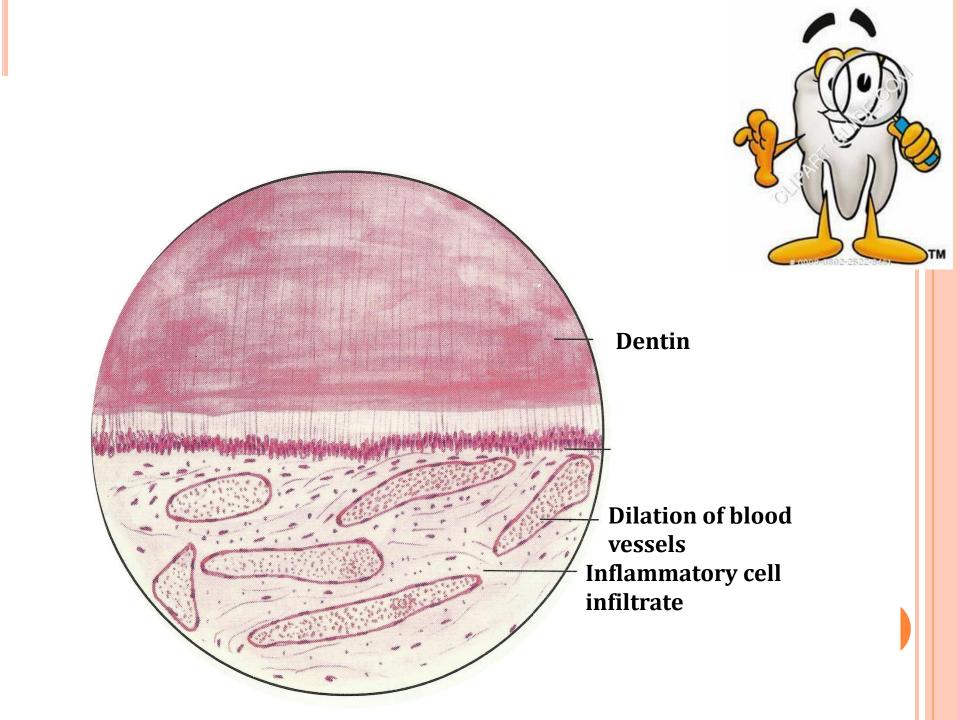
- Dilation of pulp blood vessels.
- Edema fluid collection due to damage of vessel wall & allowing extravasations of RBC or diapedesis of WBC.
- Slowing of blood flow & hemoconcentration due to transudation can cause thrombosis.
- Reparative or reactionary dentin in adjacent dentinal wall.

TREATMENT & PROGNOSIS:

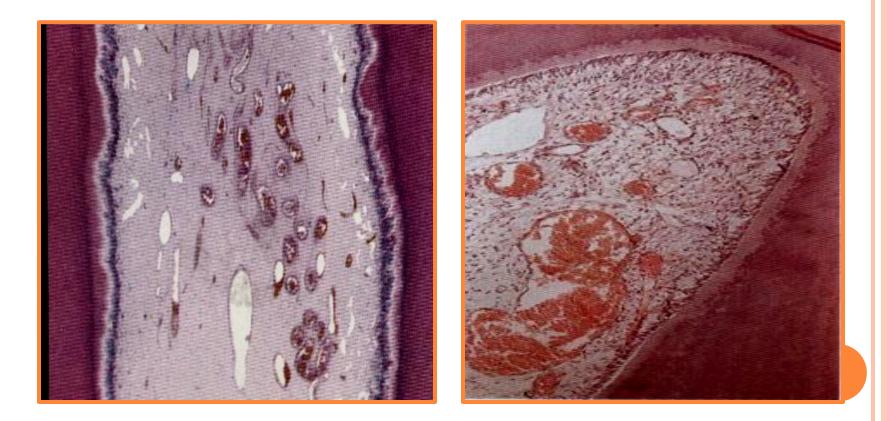
Options for management—with the actual procedure chosen depending on clinical findings once the caries has been removed:

- ◊ Indirect pulp cap
- \Diamond Direct pulp cap
- Ortial pulpotomy
- ◊ Pulpotomy.

If primary cause is not corrected, extensive pulpitis may result in death of pulp.

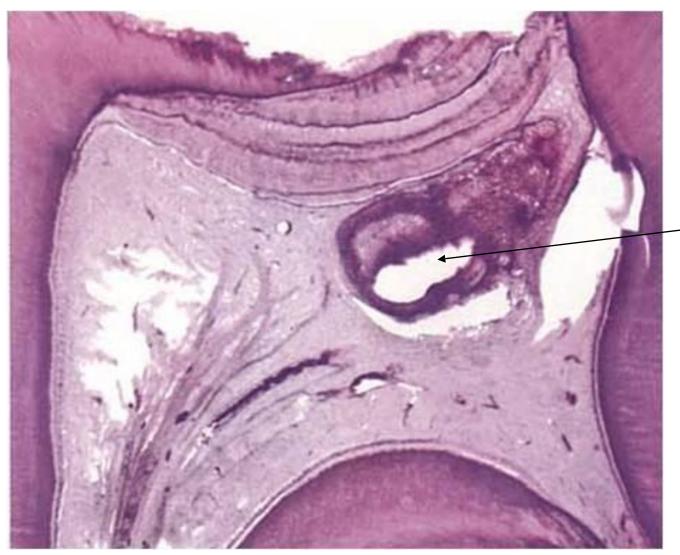






<u>ACUTE PULPITIS s</u>

- Irreversible condition characterized by acute, intense inflammatory response in pulp.
- It is a frequent immediate sequela of focal reversible pulpitis, it may occur as an acute exacerpation of a chronic process.
- Acute pulpitis may be either closed where the dentinal wall of the pulp is intact or open where the dentinal wall is broken.
 CLINICAL FEATURES:
- Pain from cold test lingers more than 30 s. May get pain from heat test May have spontaneous pain. May be percussion sensitive.
 Radiographically or clinically visible deep caries.
- Pain poorly localized since pulp of individual tooth is not represented in sensory cortex.
- Intrapulpal abscess formation cause severe pain lancinating or throbbing type. (10 – 15mins).(acute total pulpitis)
- Intensity of nain can increase when natient lies down



-Acute pulpitis with Intrapulpal abscess



- Pulp vitality test indicates increased sensitivity at low level of current.
- Pulpal pain is due to:

- pressure built up due to lack of exudate escape.

- pain producing substances from inflammation.

- Pain subsides when drainage is established or when pulp undergoes complete necrosis.
- The tooth is not tendered to percussion unless the pulpal inflammation has spread beyond the root apex into the periapical region.
- Closed pulpitis manifests pain that severer than that of open pulpitis .this is because in the closed form, the pressure increases within the pulp as a result of the inability of fluid exudate to escape.

HISTOLOGIC FEATURES:

- Edema in pulp with vasodilation.
- Infiltration of polymorphonuclear leukocytes along vascular channels & migrate through endothelium lined structures.
- Destruction of odontoblasts at pulp dentin border.
- Rise in pressure due to inflammatory exudate local collapse of venous part of circulation Tissue hypoxia
 - & <u>Destruction of pulp & abscess formation.</u>
- Abscess consists of pus, leukocytes & bacteria.
- Numerous abscess formation cause pulp liquefaction & necrosis. (acute suppurative pulpitis)



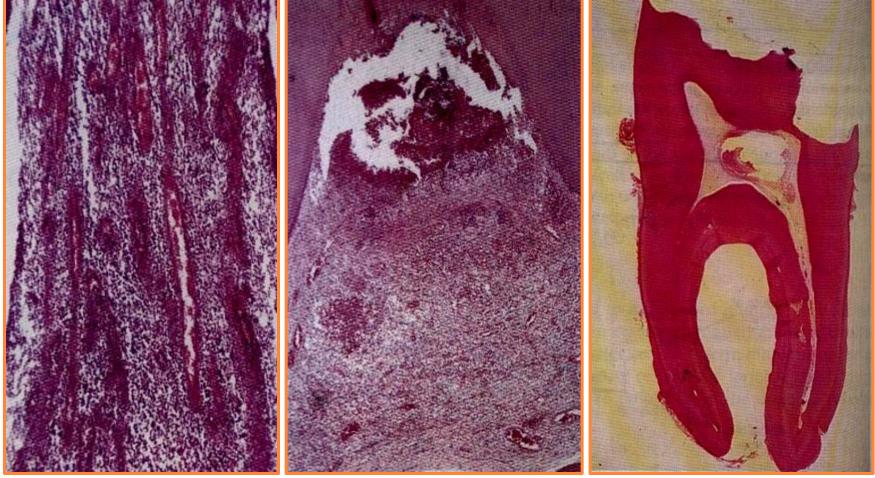




Acute pulpitis. Beneath the carious exposure (top right) a dense inflammatory inflammatory infiltrate is accumulating. More deeply. The pulp is intensely hyperaemic.

Acute pulpitis. Infection has penetrated dentine Causing inflammation to spread down the pulp and pus to form in corner

PULPITIS

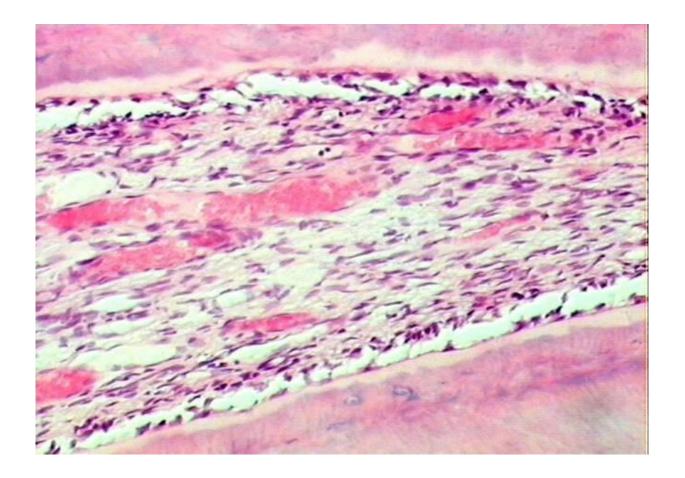


Acute pulpitis stage. The entire pulp has been destroyed and replaced by inflammatory cells and dilated vessels

Acute caries and pulpitis. Infection has penetrated to the pulp. Part of the pulp has been destroyed, and an abscess has formed containing a bead of pus

Localized pulpitis with localized pulp abscess

Acute PULPITIS











TREATMENT & PROGNOSIS:

- Options for management:
- ◊ Extraction
- Oulpectomy and root canal treatment—with the following considerations/variations:
- >> Pulp tissue is present and needs to be removed >> Sodium hypochlorite is used to dissolve pulp tissu remnants
- Anti-inflammatory medicament should be used to reduce periapical nerve sprouting and neuropathic pain, e.g., a corticosteroid/antibiotic compound.
 If the tooth also has acute apical periodontitis—consider postoperative systemic NSAID's and analgesics.

Persistent inflammatory reaction in pulp with little or non symptoms.

It can arise from a previous acute

Pulpitis or occurs as the chronic type from the o

It may be open or closed form.

CLINICAL FEATURES:



- Pain is not prominent, mild, dull ache which is intermittent.
- Reaction to thermal changes is reduced because of degeneration of nerves.
- Response to pulp vitality tester is reduced.
- Wide open carious lesion & with exposure of pulp cause relatively little pain.
- Manipulation with small instruments often elicits bleeding but with little pain.

HISTOLOGIC FEATURES:

- **FEATURES:** Infiltration of mononuclear cells, lymphocytes & plasma cells, with vigorous connective tissue reaction.
- Capillaries are prominent; fibroblastic activity & collagen fibers in bundles.(chronic closed pulpitis)
- When granulation tissue formation occurs in wide open exposed pulp surface – ulcerative pulpitis. (with bacterial stains & micro org. in carious lesion)
- Chronic open ulcerative pulpitis, is characterized by the presence of an ulcer on the exposed pulp surface, with a large number of PMNIs below the surface, there is adense chronic inflammatory cell infiltration with increased fibroblastic activity.





TREATMENT & PROGNOSIS:

- Root canal therapy
- Extraction of tooth.



form of a chronic pulp disease.

Overgrowth of pulp tissue outside the boundary of pulp chamber as protruding mass.

Chronic Hyperplastic Pulpitis (<u>(pulp</u>polyp)

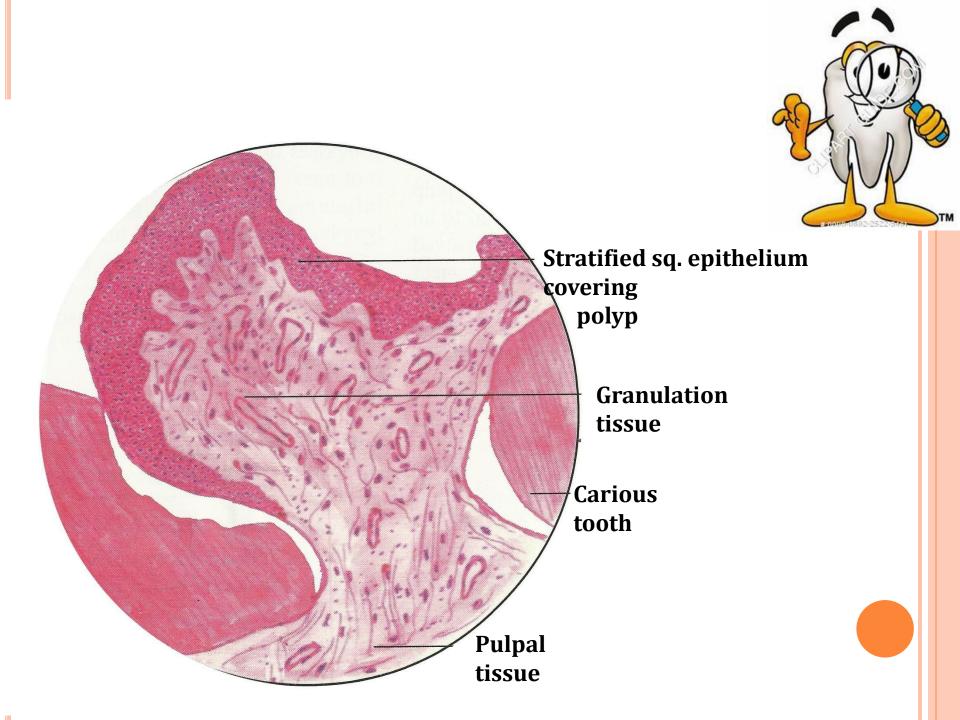
CLINICAL FEATURES:

- It occurs almost exclusively in children & young adults and involves teeth with large open carious cavity.
- Pulp pinkish red globule of tissue protruding from chamber & extend beyond caries.
- Most commonly affected are deciduous molar & Ist permanent molars.
- Pulp is relatively insensitive because few nerves in hyperplastic tissue.

- Lesion bleeds profusely upon provocation.
- Due to excellent blood supply high tissue resistance & reactivity in young persons leads to unusual proliferative property of pulp.
- Some cases, gingival tissue adjacent, may proliferate into carious lesion & superficially resemble hyperplastic pulpitis.
- So careful examination is made to determine whether connection is with pulp or gingiva.

HISTOLOGIC FEATURES:

- Hyperplastic tissue is basically granulation tissue, consisting delicate CT fibers & young blood capillaries.
- Inflammatory infiltrates lymphocytes, plasma cells & polymorphs.



- Stratified squamous type epithelial lining resembles oral mucosa with well formed rete pegs.
- Grafted epithelial cells are believed to be desquamated epith. Cells, which carried by saliva.

TREATMENT & PROGNOSIS:

Extraction of tooth or pulp extripation.



Pulp Polyp











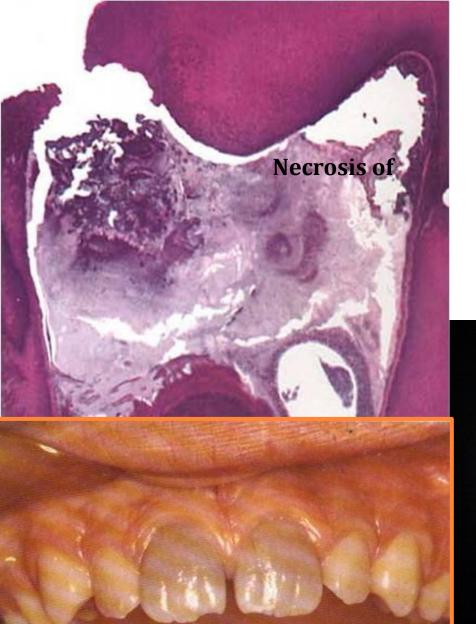




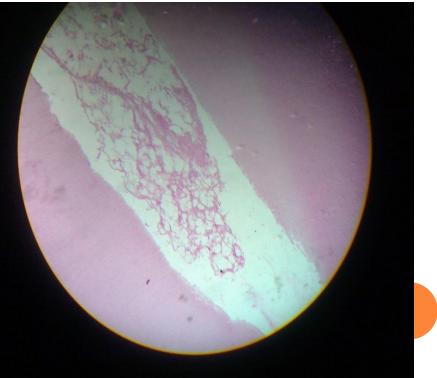
Gangrenous Necrosis of Pulp

- Plated pulpitis results complete necrosis of pulp.
- As this is associated with bacterial infection pulp gangrene.
- It is associated with foul odor when pulp is opened for endodontic treatment.
- In sickle cell anemia, blockage of pulp vessels be defective RBC results pulp necrosis.
- Non vital pulp maintain general histology being non purulent.
- This may be due to trauma or infarct.





pulp



- Mild moderate inflammatory condition.
- Nature of pain is mild & diffuse.
- Brief duration & can be produce cold stimuli that elicits the pain mostly although hot, sweet or sour

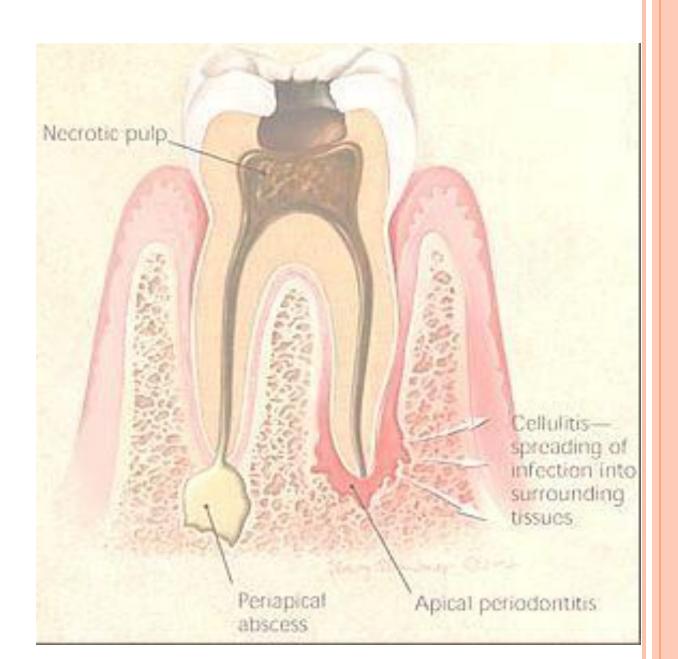
food may also initiate the pain.

- Once stimulus is removed, pain is usually subsides.
- Tooth responds to electric pulp tester at lower currents.
- Reversible pulpitis if allowed to progress can led to irreversible pulpitis.

Sharp, severe, radiating pain of long duration & varying intensity.

- Pain continues even after the stimulus is removed.
- Pain may exacerbate with bending over or lying down.
 It may progress to more severe pain)throbbing(.
 - Increased by stimulus, like heat or without stimulus.
 - When infection extends into PDL - apical periodontitis.

DISEASES OF PERIAPICAL TISSUES

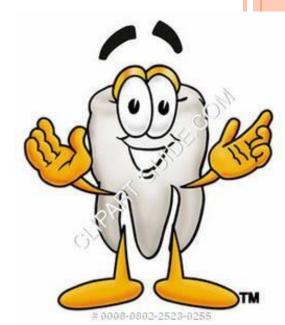


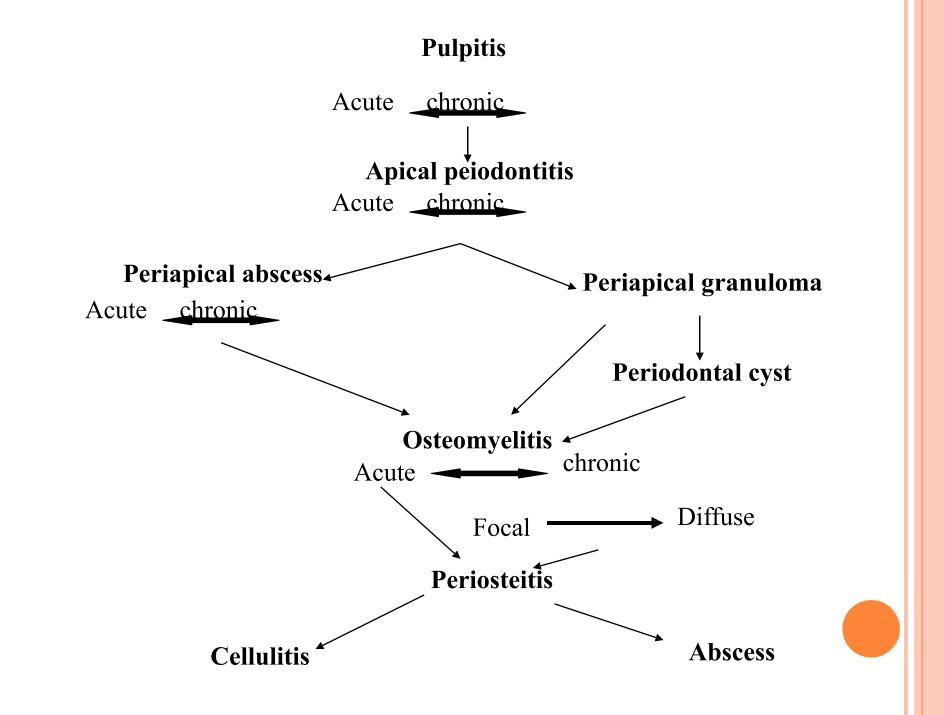
Diseases of the periapical issues

Installation of PDL around apical portion of root.

- Cause: 1. spread of infection following pulp necrosis, 2. occlusal trauma, 3. inadvertent endodontic procedures , 4. infection through the gingival crevice.
- In PDL inflammation the patient can locate the symptoms to a particular tooth due to stimulation of the proprioceptive nerve ending in PDL.
- **Types**: 1.Acute Apical Periodontitis

2. Chronic Apical Periodontitis





<u>Acute Apical</u> Periodontitis <u>cPricadoptitiires:</u>

- Thermal changes does not induce pain.
- Slight extrusion of tooth from socket.
- Cause tenderness on mastication due to inflammatory edema collected in PDL.
- Due to external pressure, forcing of edema fluid against already sensitized nerve endings results in severe pain.

RADIOGRAPHIC FEATURES:

• Appear normal except for widening of PDL space.





Acute Apical Periodontitis

HISTOLOGIC FEATURES:

Description PDL shows signs of inflammation -vascular dilation

-infiltration of PMNs

- Inflammation is transient, if caused by acute trauma.
- If irritant not removed, progress into surrounding bone resorption.
- Abscess formation may occur if it is associated with bacterial infection Acute periapical abscess / Alveolar abscess.

TREATMENT & PROGNOSIS:

• Selective grinding if inflammation due to occlusal trauma. Options for management:

 \Diamond Extraction

◊ Root canal treatment—with the following considerations/variations:

- \gg \gg There is no pulp tissue present
- >>>> Sodium hypochlorite is used as an antibacterial agent (i.e., tissue dissolution less important)
- \gg \gg Antibacterial medicament is required, e.g., calcium hydroxide

If the tooth also has acute apical periodontitis—consider using a corticosteroid/antibiotic compound in conjunction with the calcium hydroxide to reduce periapical inflammation and pain.
 If the tooth also has an acute apical abscess—consider drainage; consider systemic antibiotics.

<u>Chronic Apical</u> Periodontitis <u>(Remioploalt Gris</u>nuloma)

- Most common sequelae of pulpitis or apical periodontitis.
- If acute (exudative) left untreated chronic (proliferative).
- Periapical granuloma is localized mass of chronic granulation tissue formed in response to infection.

CLINICAL FEATURES:

- Tooth involved is non vital / slightly tender on percussion.
- Percussion may produce dull sound due to granulation tissue at apex.

- Mild pain on chewing on solid food.
- Tooth may be slightly elongated in socket.
 Sensitivity is due to hyperemia, edema & inflammation of PDL. In
- many cases, asymptomatic.
- Fully developed granuloma seldom presents more severe clinical symptoms.

RADIOGRAPHIC FEATURES:

Lesion can be either well / ill defined

Thickening of PDL at root apex.

- As concomoitant bone resorption & proliferation of granulation tissue appears to be radiolucent area.
- 👦 Loss of apical lamina dura.

Periapical Granuloma



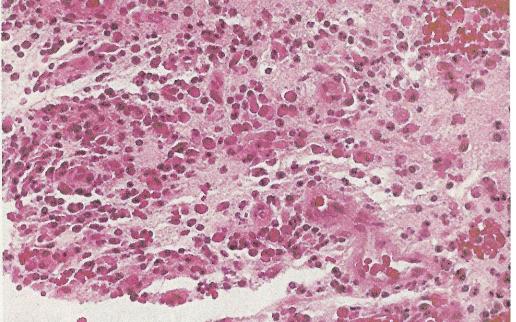




- Thin radiopaque line or zone of sclerotic bone sometimes seen outlining lesion.
- Long standing lesion may show varying degrees of root resorption.

HISTOLOGIC FEATURES:

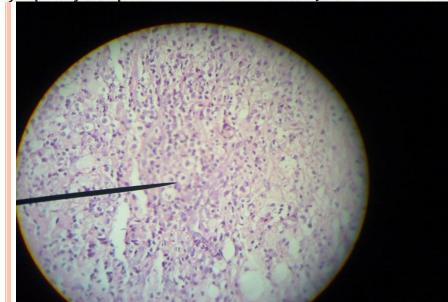
- Granulation tissue mass consists proliferating fibroblasts, endothelial cells & numerous immature blood capillaries with bone resorption.
- Capillaries lined with swollen endothelial cells.
- Its is relatively homogenous lesion composed of macrophages, lymphocytes & plasma cells.



Periapical Granuloma



Fig. 3-18 Periapical granuloma. Granulation tissue exhibits mixed inflammatory infiltrate consisting of lymphocytes, plasma cells, and histiocytes.



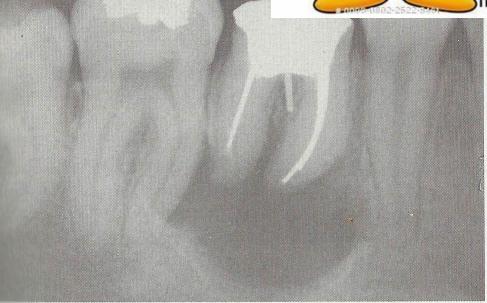
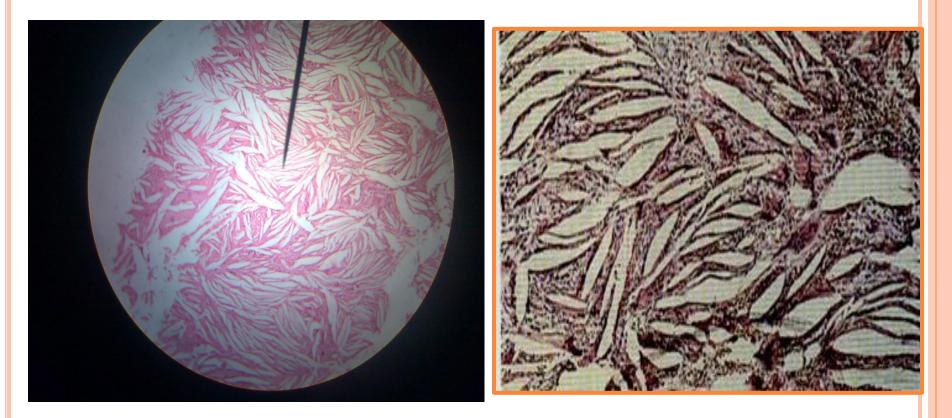


Fig. 3-16 Periapical granuloma. Large, well-defined radiolucency associated with the apices of the mandibular first molar. (Courtesy of Dr. Robert E. Loy.)

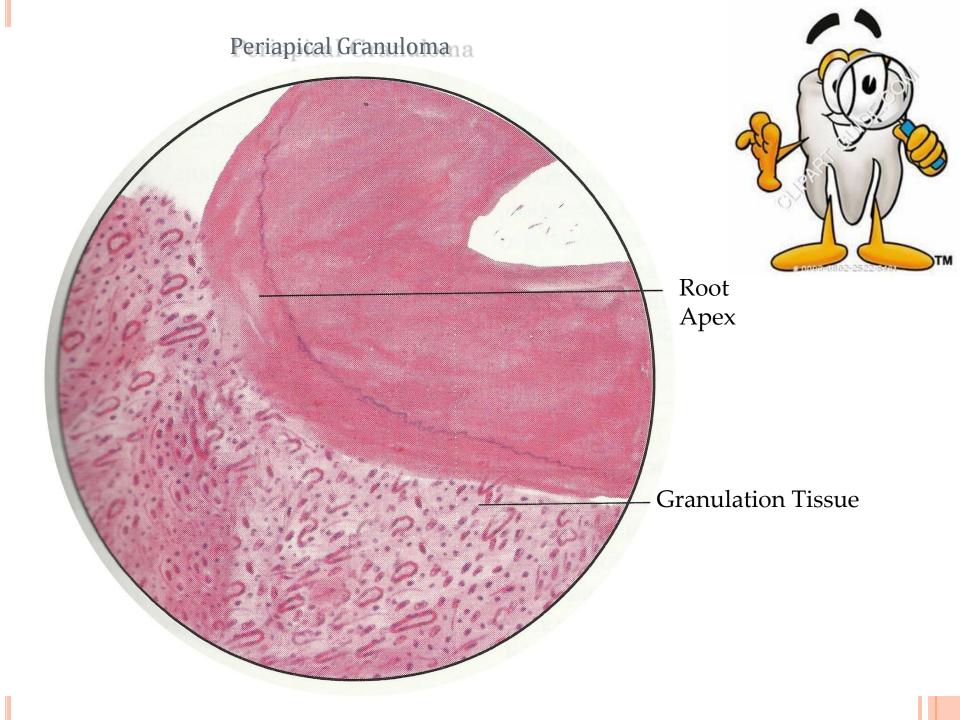
Cholesterol clefts



- © Collection of cholesterol clefts, with multinuclear gaint cells.
- Epithelial rests of Malassez may proliferate in response to chronic inflammation & may undergo cystification.
- The granulation tissue is outlined by a capsule of fibrous tissue that is usually attached to the cementum.

TREATMENT & PROGNOSIS:

- Extraction & RCT with / without apicoetomy.
- If untreated _____ apical periodontal cyst formation.





- 1) The granuloma may continue to enlarge and be associated with resorption of the bone and root apex.
- 2) Acute exacerbation acute apical periodontitis.
- 3) Suppuration may occur **-as**ute or chronic periapical abscess.
- 4) Proliferation of epithelial cells rests of malassez radicular cyst.
- 5) Low grade irritation to the apical tissues bone apposition (osteosclerosis). →
- 6) Low grade irritation to the apical tissues the apposition of cementum on the root surface (hypercementosis).



Abseelssolar abscess, Alveolar Abscess)

It is an acute or chronic localized suppurative process of the dental periapical region.

Developed from acute periodontitis / periapical granuloma.

Acute exacerbation of chronic lesion Phoenix Abscess

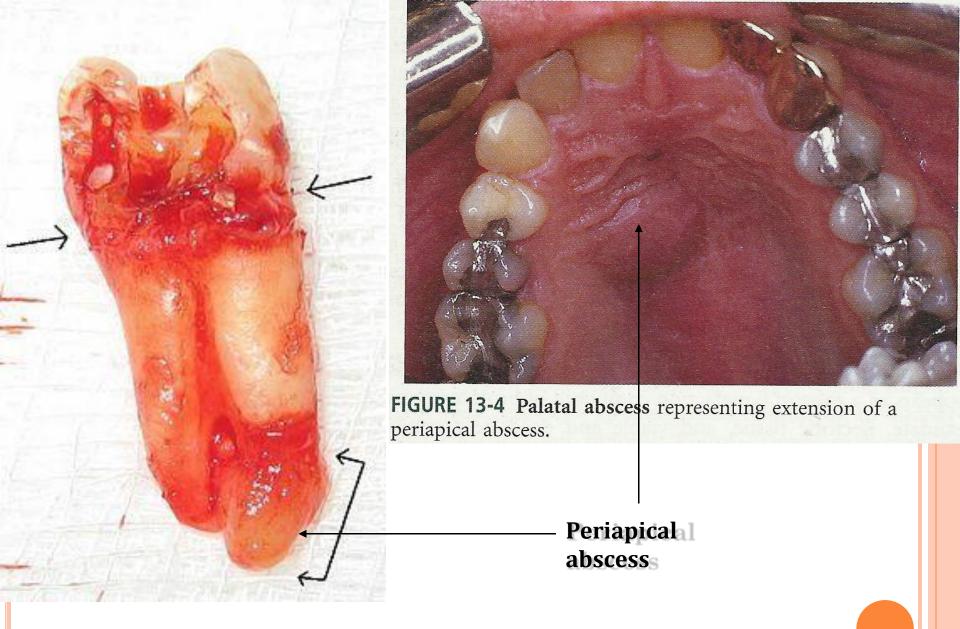
Cause due to – pulp infection, traumatic injury irritation of periapical tissues (endo procedures).

→pulp necrosis,

CLINICAL FEATURES:

- Features of acute inflammation.
- Tenderness of tooth to percussion .
- The tooth is slightly extruded from its socket.

Systemic manifestations like lymphadenitis & fever may present.







The pus tends to track through the cancellous bone and eventually perforates the cortex, it becomes asymptomatic due to lack of collection of pus within the cavity.

- The tooth will not respond to electric or thermal tests.
- Chronic abscess generally presents no features, since it is mild, well circumscribed area of suppuration that shows little tendency to spread.

RADIOGRAPHIC FEATURES:

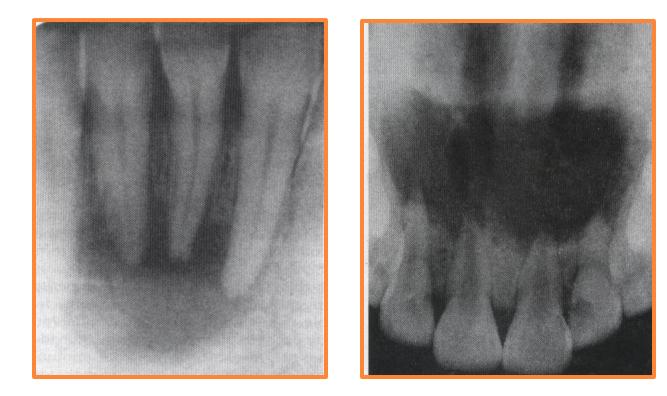
- Slight thickening of PDL space.
- Radiolucent area at apex of root (phoenix abscess).

HISTOLOGIC FEATURES:

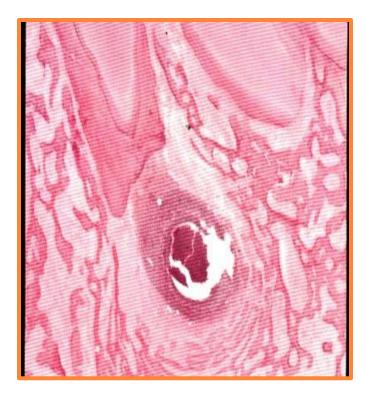


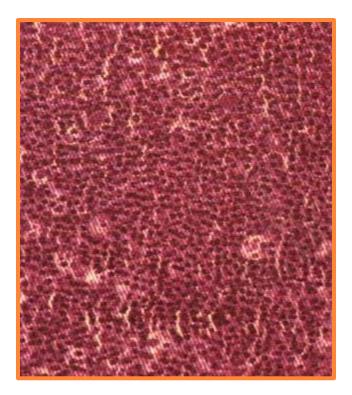
- Area of suppuration composed of PMN leukocytes, lymphocytes, cellular debris, necrotic materials & bacterial colonies.
- It appears as an empty space due to loss of pus during the preparation.

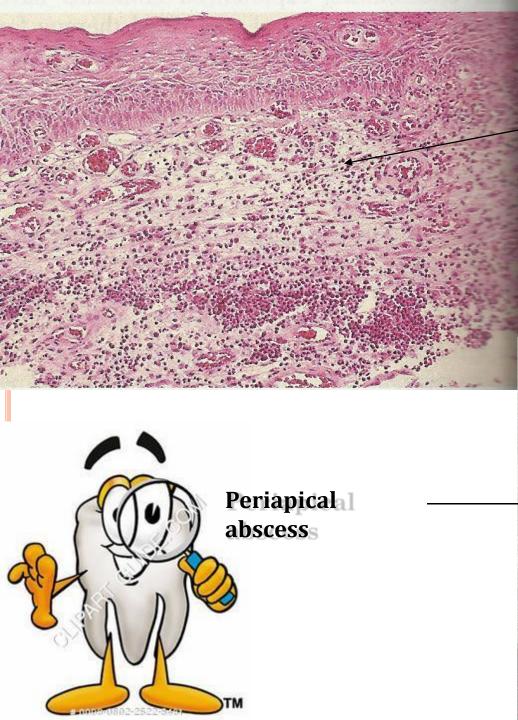




ill defined radiolucency.







Inflammatory infiltrate, cellular debris, necrotic materials etc..

- The abscess cavity is surrounded by acute inflammatory cell and few chronic inflammatory cells.
- Dilation of blood vessels in PDL
- Marrow space show inflammatory infiltrates.
- In chronic periapical abscess, the abscess cavity is surrounded by dense layer of chronic inflammatory cell and few acute inflammatory cells, and surrounded by dense bundle of collagen fibers.

TREATMENT & PROGNOSIS:



Drainage of abscess by opening pulp chamber or extraction.
 RCT.

- If untreated, causes formation of fistulous tract opening to oral mucosa (parulis), osteomyelitis, cellulites & bacteremia.
- Cavernous sinus thrombosis has been reported.

- Abscess may spread along path of least resistance through medullary spaces resulting in Osteomyelitis.
- Can also perforate cortical bone and spread to soft tissues –
 Cellulitis.
- It can also drain through an intraoral sinus tract. Opening of such a tract is usually covered by a granulation tissue Parulis.



COMPLICATIONS

- Facial Cellulitis
- Ludwig's angina
- Osteomyelitis
- Septicaemia
- Menengitis, brain abscess, cavernous sinus thrombosis

CELLULITIS

- It is a rapidly spreading inflammation of the soft tissues characterized by diffuse pus formation, usually associated with malaise and an elevated temperature.
- This happens if an abscess is not able to establish drainage through the skin surface or into oral cavity.

TYPES:

- Cellulitis arising from dental infection and spreading through soft tissues of head and neck can take various forms.
- Mostly, infection spreads through tissue spaces like canine space, infratemporal space, pharyngeal space, buccal space, submental and submandibular space etc.

CELLULITIS

Two especially dangerous forms of cellulitis are:-

cellulitis associated with mandibular teeth into submandibular and cervical tissues may cause

(Ludwig's angina).

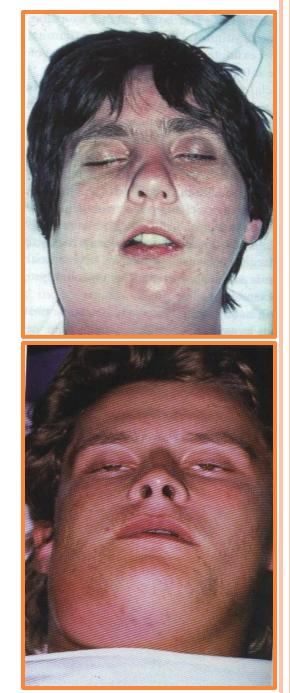
cellulitis associated with maxillary teeth towards the eye may cause(Cavernous sinus thrombosis)

LUDWIG'S ANGINA

- Cellulitis of submandibular region involving sublingual, submandibular and submental spaces.
- In 70% cases develops from spread of infection from mandibular teeth.
- Increased prevalence in immunocompromised patients like AIDS, aplastic anemia, organ transplantation etc.

CLINICAL FEATURES

- It produces a broad –like swelling of the floor of the mouth.
- Involvement of the sublingual space results in elevation and posterior displacement of the tongue, leading to difficulty in eating, swelling (dysphagia) and breathing(dyspnea).
- After reaching submandibular region, infection extends to lateral pharyngeal and retropharyngeal spaces.



- Lateral pharyngeal space involvement may cause respiratory obstruction due to laryngeal edema(suffocation).
- In sever cases tachypnea, dyspnea, tachycardia, may also be noted.
- General signs fever, malaise, leukocytosis, and raised Erythrocyte Sedimation Rate ESR.

TREATMENT

- maintenance of the airway, tracheostomy may be indicated.
- 2. Antibiotic therapy.
- 3. Surgical drainage.

CAVERNOUS SINUS THROMBOSIS

- The infection from the posterior maxillary teeth reach the orbit via the maxillary sinus , while infection from the anterior maxillary teeth reach the orbit via the ophthalmic veins.
- Infection from orbit reaches the cavernous sinus through the communicating veins between them.

CLINICAL FEATURES

- Periorbital edema including lateral border of nose, protrusion and fixation of eyeball.
- Pupil dilatation, lacrimation, photophobia and loss of vision may also occur.
- Pain along distribution of ophthalmic and maxillary branches of Vth cranial nerve.
- Proptosis, chemosis seen in 90% cases.
- Fever, chills, headache, sweating, tachycardia, nausea and vomiting also occur.

Treatment

- 1. High dose of penicillin.
- 2. Extraction and drainage(if fluctuant).
- 3. Corticosteroid and anticoagulant to prevent thrombosis and septic emboli formation.

