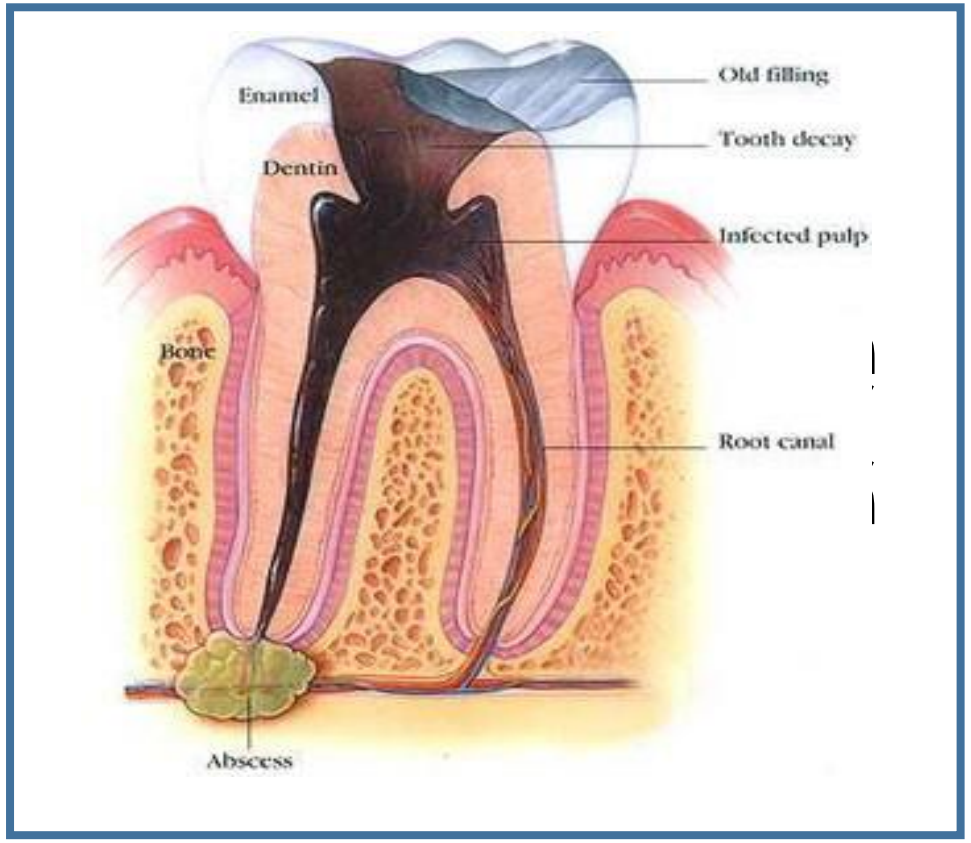

Diseases Of Pulp & Periapical Tissues

Lecture 4 A

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Objectives

- Determine the causes of pulpal disease.
- Classification of pulpal diseases.
- Clinical and Histopathological charactarestics of pulpal diseases.

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Etiology:

FACTORS

PHYSICAL INJURY

Acute Injury

- Injury on tooth
- Cavity preparation without water spray
- Vigorous polishing
- Root planning in PDL therapy
- Restoration – improper insulation

Chronic Injury

- Attrition -abrasive food & bruxism
- Abrasion -abnormal tooth brushing

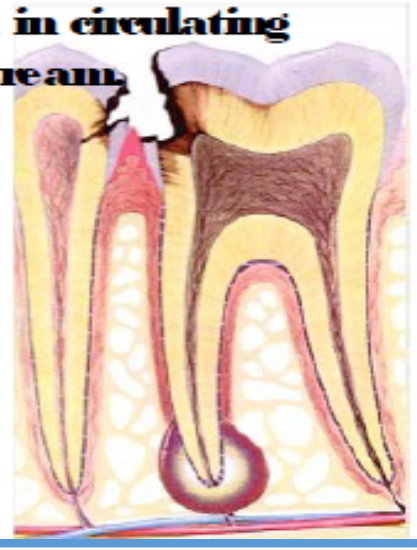
CHEMICAL INJURY

- Medicaments or materials applied to dentin diffuses through dentinal tubules.

MICROBIAL FACTORS

Bacterial invasion by:

- Dental caries
- Fractured tooth where exposed pulp
- Anachoretic infection due to presence of bacteria in circulating blood stream.



Classification Of Pulpitis

1. Acute & Chronic

2. Based on extend

- Partial pulpitis (confined a portion of pulp)

- Subtotal pulpitis

3. i. If inflammatory process confined within a portion:

- Focal /Partial pulpitis

ii. If most of pulp diseased:-Total /Generalized pulpitis

4. Another classification of acute & chronic based on presence or absence of direct communication between pulp & oral environment:

- Open pulpitis (pulpitis aperta) communicated exist.

- Closed pulpitis (pulpitis clausa) no communication exist.

Focal Reversible Pulpitis (Pulp Hyperemia)

Mild, transient, localized inflammatory response.

CLINICAL FEATURES:

•Tooth is sensitive to thermal changes, especially cold.

•Pain - short duration, disappears on withdrawal of thermal irritant.

•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.

Focal Pulpitis

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 totransudation can cause thrombosis.

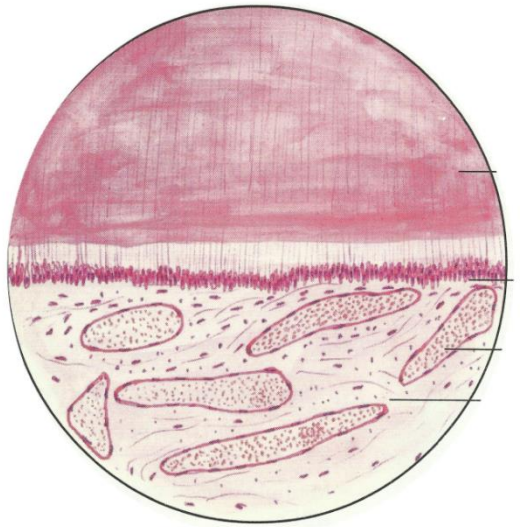
•Reparative or reactionary dentin in adjacent dentinal wall.

TREATMENT & PROGNOSIS:

•Carious lesion should be excised & restored or defective

•filling is replaced.

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



- **Acute Pulpitis**

Irreversible condition characterized by acute,intense inflammatory response in pulp.

CLINICAL FEATURES:

•Teeth extremely sensitive to thermal changes.

•Hot or cold stimuli cause increase in pain intensity &persists.

•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)

•Intensity of pain can increase when patient lies down.

Acute pulpitis

Pulp vitality test indicats increased sensitivity at low level of current.

Pulpal pain is due to:

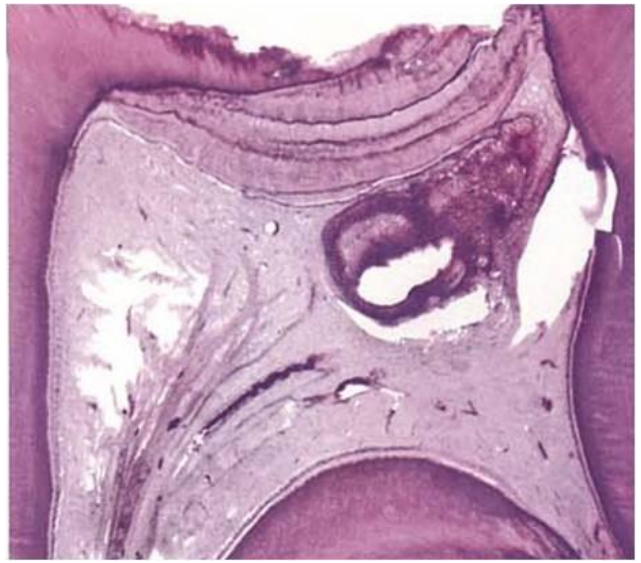
- pressure built up due to lack of exudate escape.
-pain producing substances from inflammation.

Pain subsides when drainage is established or when pulp undergoes complete necrosis.

Note:The tooth is not tendered to percussion unless the pulpal inflammation has spread beyond the root apex into the periapical region.

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 & anoxia Destruction of pulp & abscess formation.
•Abscess consists pus, leukocytes & bacteria.
•Numerous abscess formation cause pulp liquefaction &necrosis. (acute suppurative pulpitis)



TREATMENT & PROGNOSIS:

•Drainage of exudate from pulp chamber.
•Pulpotomy & placing calcium hydroxide over entrance of root canal.
•Root canal treatment.
•Extraction of tooth.

Chronic Pulpitis

Persistent inflammatory reaction in pulp with little or non constitutional symptoms.

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:

•Infiltration of mononuclear cells, lymphocytes & plasma cells, with vigorous connective tissue reaction.

•Capillaries are prominent; fibroblastic activity & collagen fibers in bundles.

•When granulation tissue formation occurs in wide open exposed pulp surface – ulcerative pulpitis. (with bacterial stains & micro org. in carious lesion)

•If pulpal reaction vacillates between an acute & chronic phase causes pulp abscess formation, which is surrounded by fibrous CT wall, which is called Pyogenic Memberane

TREATMENT & PROGNOSIS:

Root canal therapy

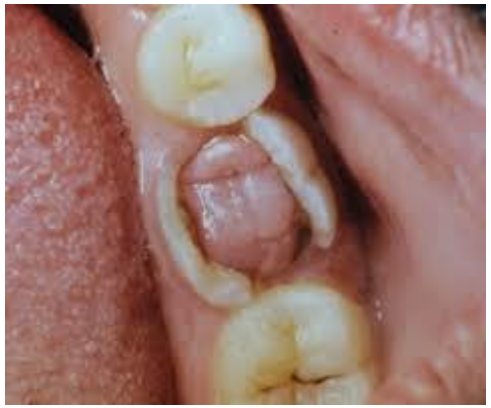
Extraction of tooth.

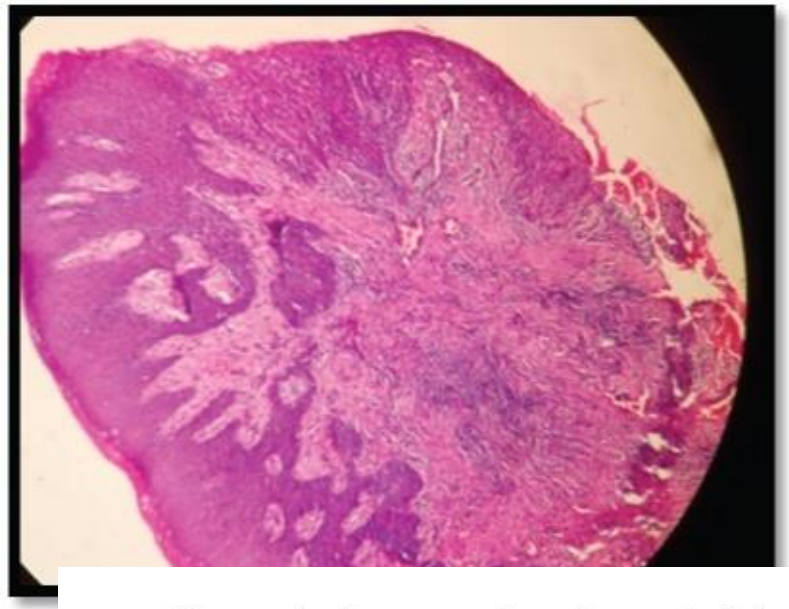
Chronic Hyperplastic Pulpitis (pulp polyp)

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

CLINICAL FEATURES:

•Children & young adults with high degree of tissue resistance & reactivity & responds to proliferative
•lesions.
•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.

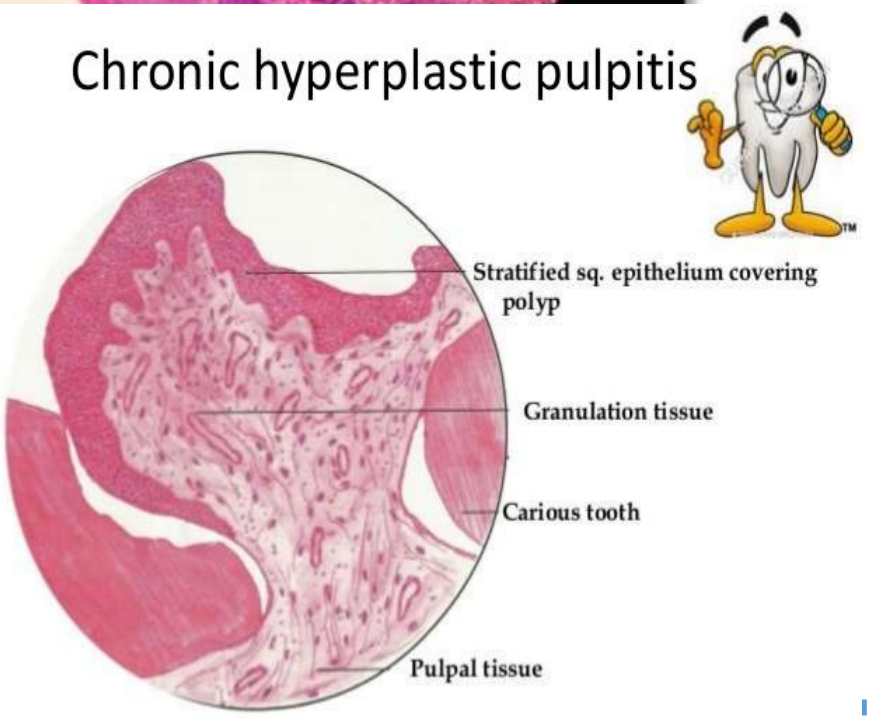
•Stratified squamous type epithelial lining resembles oral mucosa with well formed rete pegs.

•Grafted epithelial cells are believed to be desquamated epith. Cells, which carried by saliva.

•When pulp polyp is present for a long time, persistent rubbing of buccal mucosa may help in grafting of epith. cells.

TREATMENT & PROGNOSIS:

Extraction of tooth or pulp extripation.



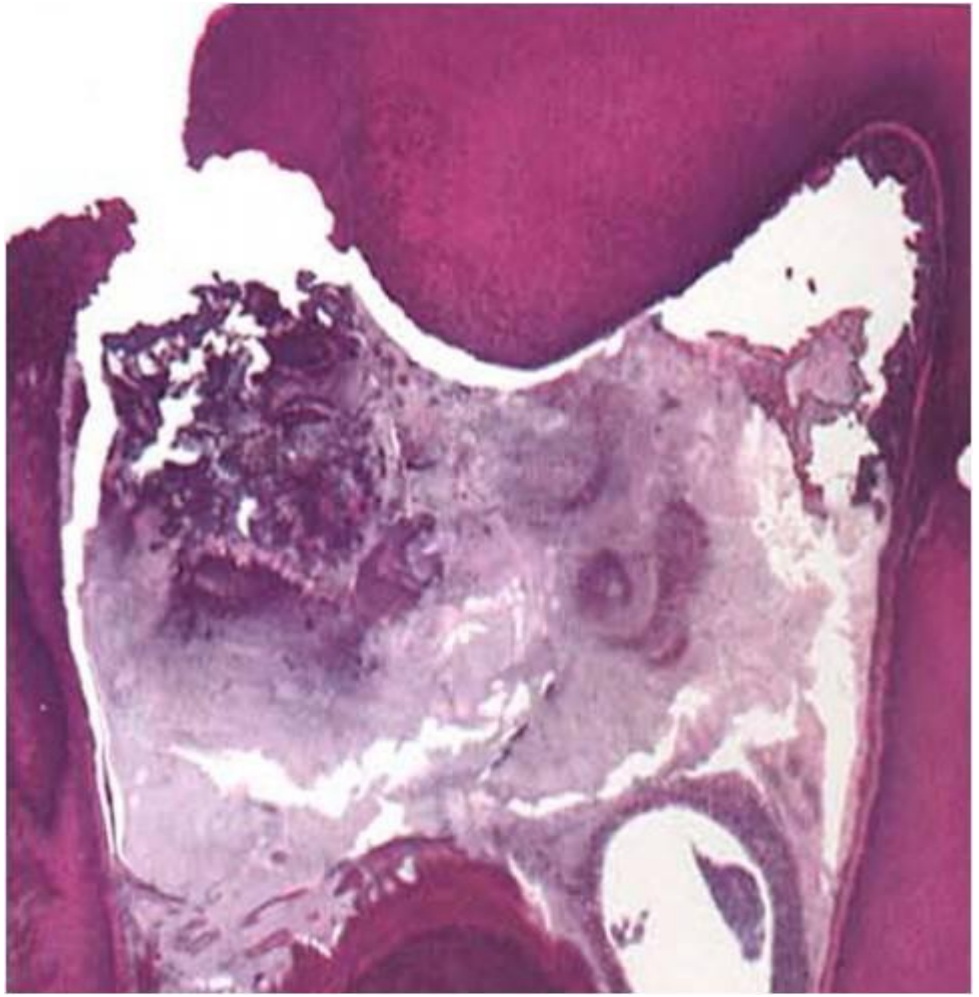
Gangrenous Necrosis of Pulp

•Untreated pulpitis results complete necrosis of pulp.

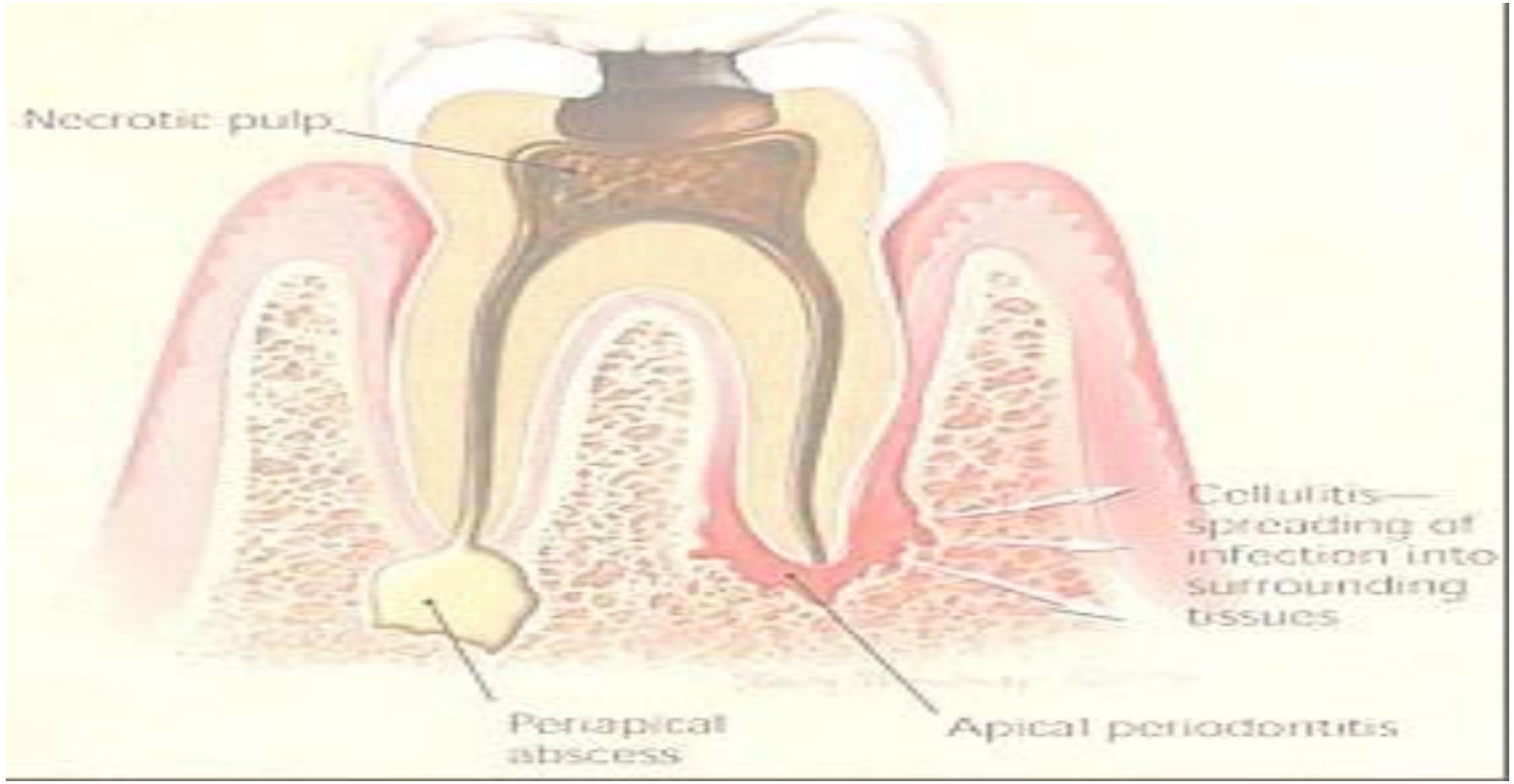
•As this is associated with bacterial infection – pulp gangrene.,It is associated with foul odor when pulp is opened for endodontic treatment.

•In sickle cell anemia, blockage of pulp vessels be defective RBC results pulp necrosis.

•When Non vital pulp maintain general histology being non purulent,This may be due to trauma or infarct.



Diseases Of Periapical Tissues



Apical Periodontitis

Inflammation of PDL around apical portion of root.

Causes: spread of infection following pulp necrosis, occlusal trauma, inadvertent endodontic procedures etc.

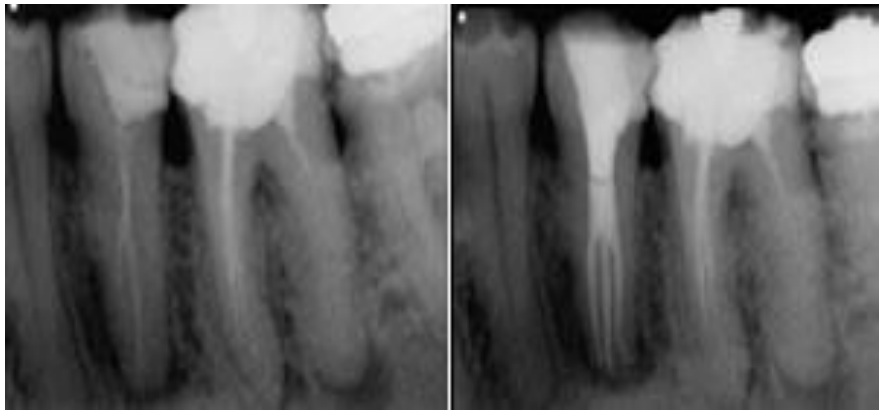
Types:

1. Acute Apical Periodontitis
2. Chronic Apical Periodontitis

Acute Apical Periodontitis

CLINICAL FEATURES:

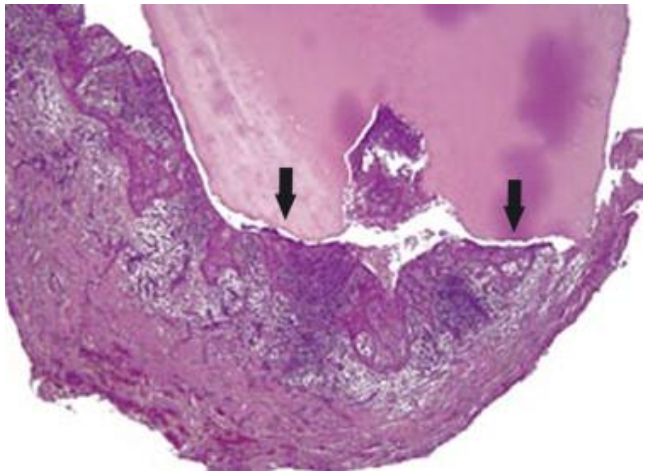
- Thermal changes does not induce pain.
- Slight extrusion of tooth from socket.
- Cause tenderness on mastication due to inflammatory edema collected in PDL.
- Due to external pressure, forcing of edema fluid against already sensitized nerve endings results in severe pain.



RADIOGRAPHIC FEATURES:

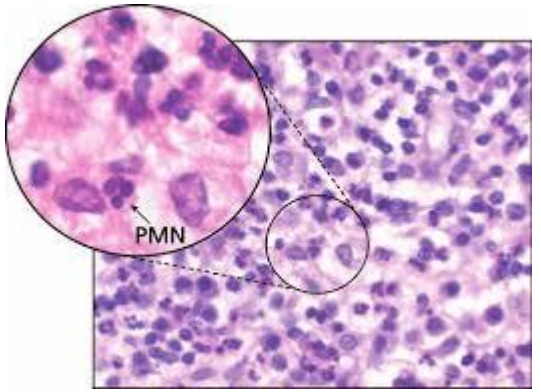
- Appear normal except for widening of PDL space.

Acute Apical Periodontitis



HISTOLOGIC FEATURES:

- 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:

- Extraction or endodontic treatment
- Selective grinding if inflammation due to occlusal

Chronic Apical Periodontitis

(**Periapical Granuloma)**

•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.

•But term is not accurate since it doesn't shows true granulomatous inflammation microscopically.

CLINICAL FEATURES:

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

Clinical features

- 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.
- No perforation of bone & oral mucosa forming fistulous tract unless undergoes acute exacerbation.

RADIOGRAPHIC FEATURES:

- Thickening of PDL at root apex.
- As concomoitent bone resorption & proliferation of granulation tissue appears to be radiolucent area.

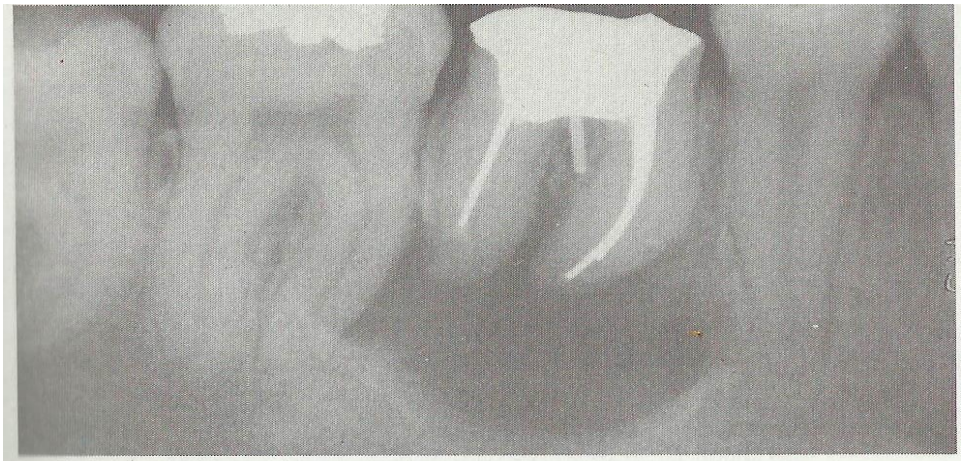


Fig. 3-16 Periapical granuloma. Large, well-defined radiolucency associated with the apices of the mandibular first molar. *(Courtesy of Dr. Robert E. Loy.)*

Hitological features:

•Plasma cells containing Russels body are found extracellularly.

•T lymphocytes produce cytotoxic lymphokines, collagenase & other enzymes & destructive lymphokines.

•Collection of cholesterol clefts, with multinuclear gaint cells.

•Epithelial rests of Malassez may proliferate in response to chronic inflammation & may undergo cystification.

Bacteriologic Features:

Strep. viridans, strep. Hemolyticus, non hemolytic strep, staph. aureus, staph. Albus, E coli & pnemococci are isolated from lesion.

TREATMENT & PROGNOSIS:

Extraction & RCT with / without apicoetomy.

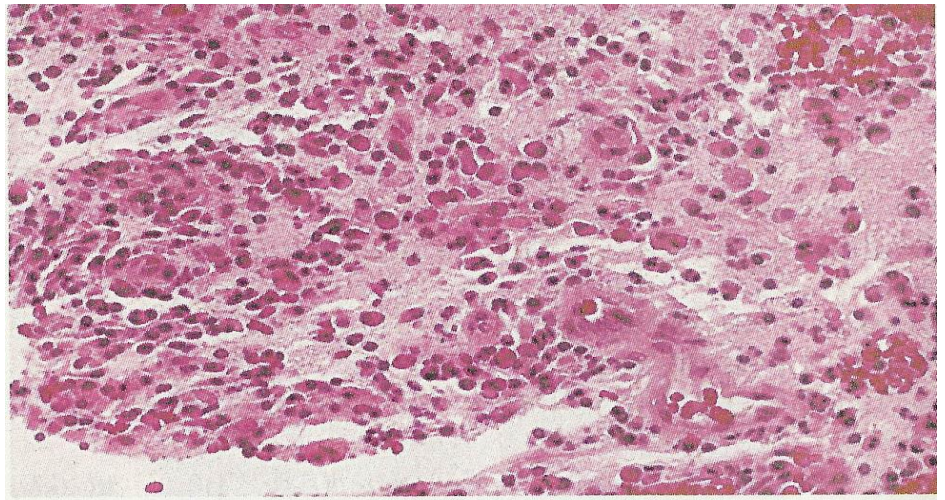


Fig. 3-18 Periapical granuloma. Granulation tissue exhibits mixed inflammatory infiltrate consisting of lymphocytes, plasma cells, and histiocytes.

Periapical Abscess
(Dento-Alveolar abscess, Alveolar Abscess)

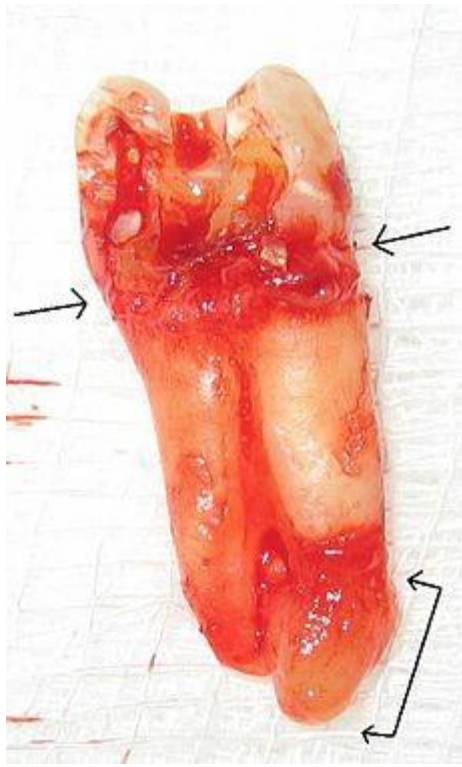
Developed from acute periodontitis / periapical granuloma.
Acute exacerbation of chronic lesion Phoenix Abscess

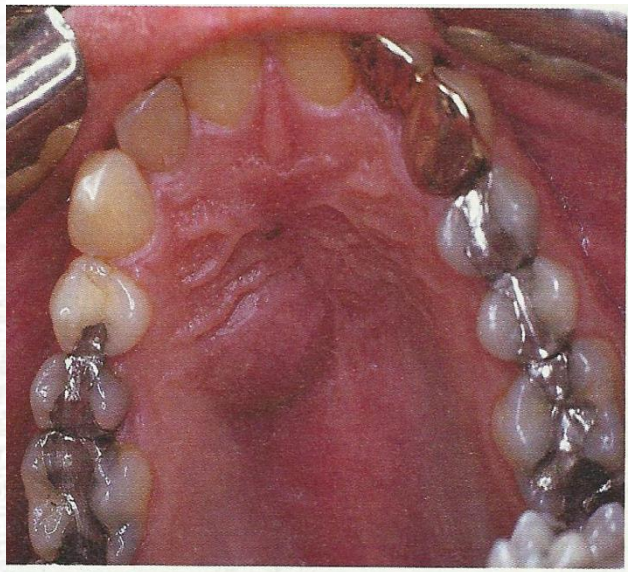
Causes:

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

CLINICAL FEATURES:

- Features of acute inflammation.
- Tenderness of tooth, which relives after pressure application.
- Extreme painful tooth extrude from socket.



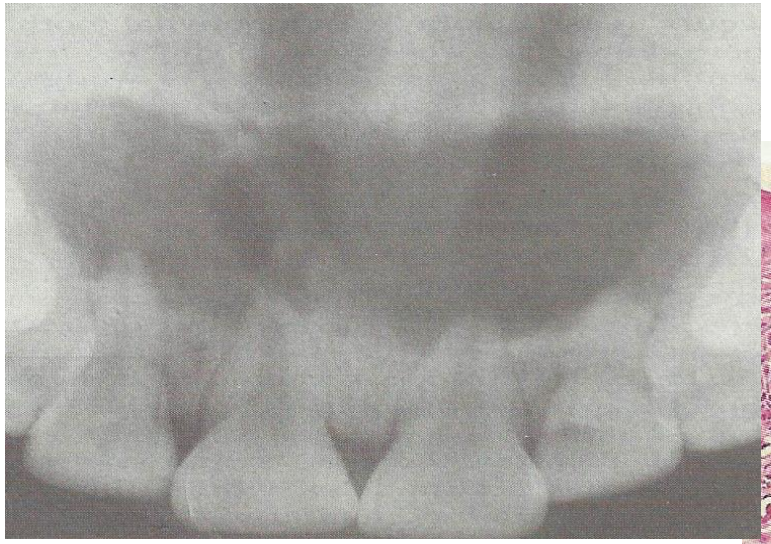


URE 13-4 Palatal abscess representing extension of a
iapical abscess.

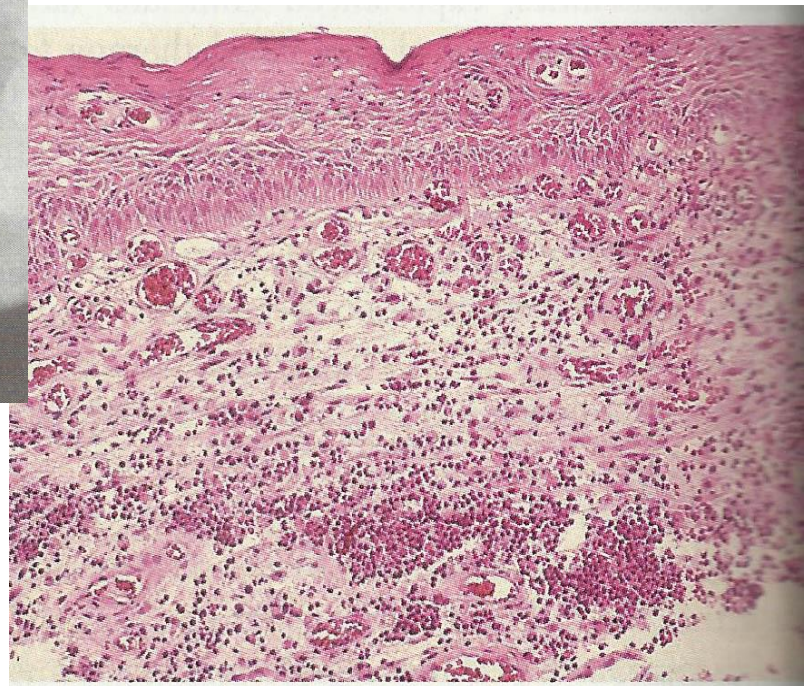
Extension to bone marrow spaces produce osteomyelitis, but clinically considered as Dento-Alveolar abscess –swelling of tissues.

Chronic abscess generally presents no features, since it is mild, well circumscribed area of suppuration which spread from local area.

RADIOGRAPHIC FEATURES:
Slight thickening of PDL space.
Radiolucent area at apex of root.



HISTOLOGIC FEATURES:
•Area of suppuration composed of PMN leukocytes,
•lymphocytes, cellular debris, necrotic materials & bacterial colonies.
•Dilation of blood vessels in PDL & bone marrow space.



•Marrow space show inflammatory infiltrates.
•Tissue around area show suppuration containing serous exudate.

TREATMENT & PROGNOSIS:

•Drainage of abscess by opening pulp chamber or extraction.
•RCT.
•If untreated, causes osteomyelitis, cellulites & bacteremia & formation of fistulous tract opening to oral mucosa.
•Cavernous sinus thrombosis has been reported.