Diseases of Pulp & Periapical Tissues

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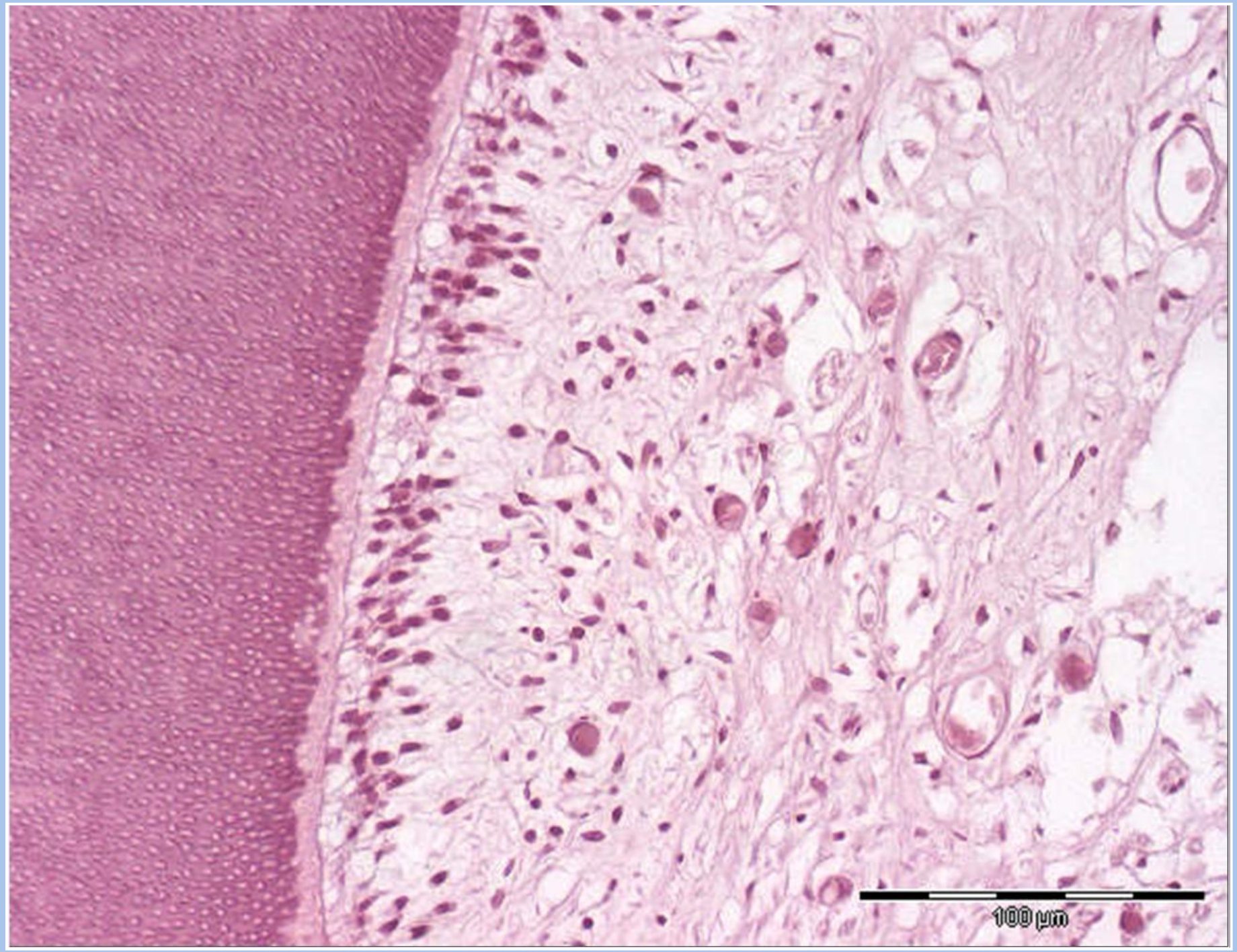
- Nerve distribution
- Pathway of pulpal infection
- Causes of pulpal disease
- Classification
- Description of pulpal disease
- Description of periapical disease

Pulp

- Its a delicate connective tissue interspersed with
  - Tiny blood vessels
  - Lymphatics
  - Nerves
  - Undifferentiated mesenchymal cells
- It responds to bacterial infection or to other stimuli by an inflammatory response


100 µm

Nerve distribution in pulp

A delta fiber

- In coronal just beneath the odontoblastic layer of pulp
- Stimulated by:
  - Thermal test
  - Electric pulp tester
- Pain response is:
  - Sharp
  - Lancinating

C fiber

- In core of pulp
- Stimulated by:
  - Presence of inflammatory toxins
- Pain response is:
  - Dull
  - Throbbing

Repair process in dentin – pulp complex

Caries demineralization

Exposes the biologically active molecule & growth factors

Diffuse through the dentinal tubules

Molecular signaling

Tertiary dentin

Pulpal inflammation

- Extent of pulpal inflammation depends upon:
  - Depth of bacterial invasion
  - Degree to which dentin permeability is reduced by sclerosis & reparative dentin
- If barrier is not sufficient □bacteria invade into the pulp
- Inflammation is defense reaction, but its increase severity can progress to pulp necrosis, pulp death or periapical lesions

Causes of pulpal diseases

I . Physical injuries

A. Mechanical

B. Thermal

C. Electrical

II. Chemical

III. Bacterial

Causes of pulpal diseases

I . Physical injuries

A. Mechanical

1. Trauma: Accidental or Iatrogenic (Cavity or crown prepara)

2. Pathologic wear: Attrition & Abrasion

3. Crack tooth syndrome

4. Barometric changes

Causes of pulpal diseases

I . Physical injuries

B. Thermal

- Heat from cavity preparation
- Exothermic heat from cements
- Deep fillings without a protective base
- Heat caused by polishing a restoration

C. Electrical: Galvanic current from dissimilar metallic restorations

Causes of pulpal diseases

II. Chemical

A. Phosphoric acid, Acrylic monomer

B. Erosion (acids)

III. Bacterial

- toxins associated with caries
- Direct invasion of pulp from caries or trauma
- Microbial colonisation in pulp by blood-borne micro organisms (Anachoresis)

Classification of Pulpal diseases

According to the inflammatory nature:

- Acute pulpitis
- Chronic pulpitis

Depending on the extent of involvement of pulp:

- Partial/ Subtotal pulpitis
- Total/ Generalized pulpits
- Aseptic pulpitis

Classification of Pulpal diseases

Presence/absence of a communication b/w pulp & oral cavity:

- Open pulpitis (pulpitis Aperta)
- Closed pulpitis (Pulpitis clausa)

Classification that guides the appropriate treatment :

- Reversible
- Irreversible

Reversible pulpitis

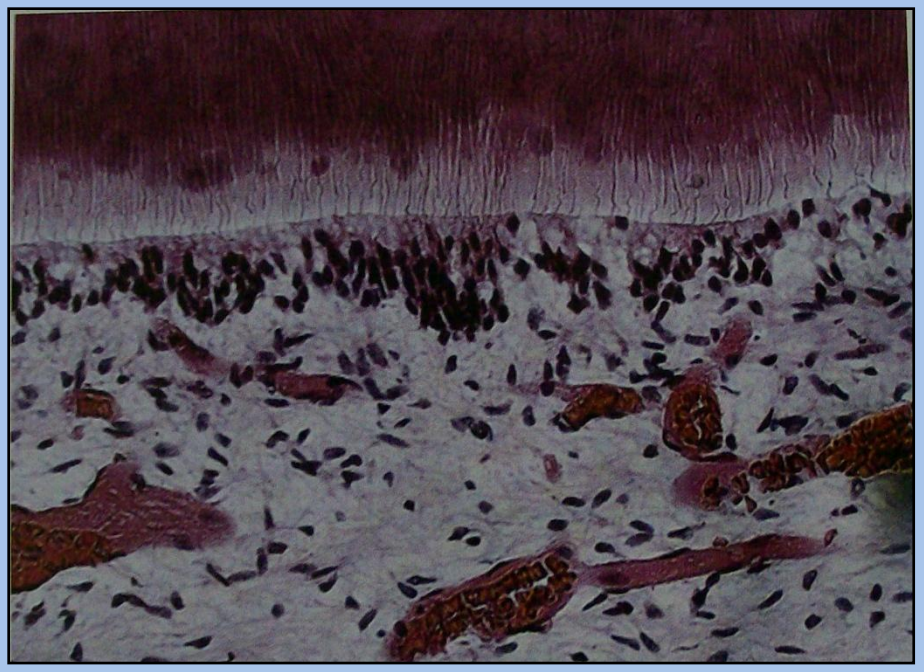
- A mild to moderate inflammatory condition of the pulp caused by noxious stimuli in which the pulp is capable of returning to the uninflamed state following removal of the stimuli.

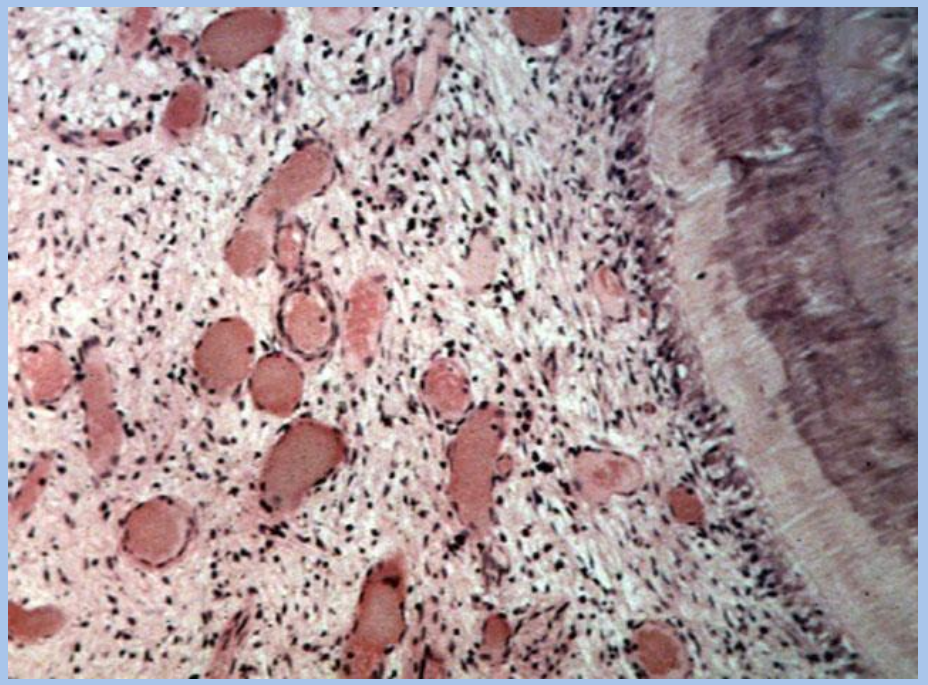
Reversible pulpitis

- **Symptoms**
- Sudden & sharp pain lasting for short duration.
- Tooth responds most to cold stimuli (ice, cold air).
- Does not occur spontaneously
- Does not continue when the cause has been removed
- Asymptomatic reversible pulpitis may result from incipient caries & is resolved on removal of the caries &proper restoration of the tooth

Histopathologic features:

- Dilatation of pulp vessels
- Edema
- Slowing of blood flow &
- Hemoconcentration due to
- Transudation – thrombosis
- Reparative dentin adjacent
- to dentinal wall





Reversible pulpitis

- **Diagnosis :**
- Response to lower level of current in electric pulp tester
- Clinical tests – cold application
- Reacts normally to percussion.
- Periapical tissue normal on radiographic examination.

Treatment :

- Removal of noxious stimuli.
- Early prevention & restoration.

Irreversible Pulpitis:

- Moderate to severe inflammatory condition of the pulp caused by noxious stimulus in which tooth is not capable of returning to its normal uninflammed state after the removal of the stimulus

Irreversible Pulpitis:

Symptoms :

- Pain is of sharp, piercing or shooting and generally severe and radiating type.
- may be intermittent or continuous.
- exacerbates on bending over or lying down
- Pain often continues when the cause has been removed
- Pain is increased by heat & is sometimes relieved by cold
- Tooth responds to electric pulp testing at higher levels of current or demonstrate no response

Irreversible Pulpitis:

Histopathologic features:

- Congestion of venules that results in focal necrosis
- Necrotic zone contains PMN leukocyte & histocytes
- Surrounding pulp tissue exhibits fibrosis & a mixture of plasma cells, lymphocytes, & histocytes

Irreversible Pulpitis:

Diagnosis :

- Inspection generally discloses a deep cavity extending to the pulp .
- Radiographic examination may not show any significant change.
- Electric pulp test induces a response with a marked variation in current from the normal.

Treatment :

Root canal treatment/Extraction

• *Reversible pulpits*	*Irreversible pulpits*
History:	
• of mild or occasional pain	• Constant, intermittent pain
Nature:	
• Momentary, immediate onset, sharp in nature.	• Continuous dull onset of pain .
• Pain disappears on removal of stimuli.	• Pain persist for minute to hour after removal of stimulus
• Pain is localized & not referred	• Not localized, it is diffused
Change in posture:	
• No difference	• Pain increase on lying down /night due to increases inter pulpal pressure

• *Reversible pulpits*	*Irreversible pulpits*
Thermal test:	
• Using heat or cold .tooth responds to cold & heat normally , pain disappear on removal of stimulus	• There is marked response to heat. • Pain persist even after removal of stimulus.
Electric pulp testing:	
• Tooth responds early to an electrical stimulus	• Delayed ,early or mixed response.
Percussion :	
• Negative response	• Positive response
Radio logically:	
• No radiological changes is seen	• It start with widening of periodontal ligament space

Crack tooth syndrome:

- Seen in tooth with large extension of restoration with minimal cuspal support. Their may be minute fracture lines.
- Patient complain of pain while biting & on reliving masticatory force pain disappear

Types:

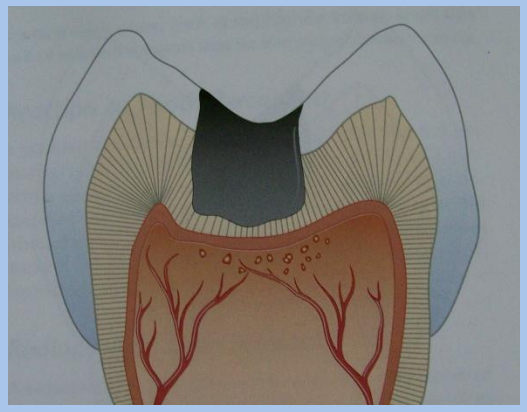
- Incomplete : when fracture line cannot communicate with pulp chamber
- Complete : When fracture line communicate with pulp chamber

Diagnosis :

- Tran illumination(fibro optic light illumination)
- Dyes
- Bite test – patient bites on object like rubber disc ,handle of instrument

Treatment :

- Remove the restoration
- Protect the remaining tooth structure by placing copper band
- Relief the tooth from occlusion



Acute pulpitis:

- Immediate sequela of focal reversible pulpitis
- Or it may occur as an acute exacerbation of a chronic inflammatory process.
- Clinical feature:
  - Tooth with large carious lesion
  - Restoration with recurrent caries
  - Pain is elicited by thermal changes esp. with cold.
  - Pain persist even after removal of stimulus
  - Pain is lancinating type, may be continues & increase when patient lies down.

- Later stage
  - Heat may cause an acute exacerbation of pain
  - Pain is severe due to increase in pressure due to lack of escape of inflammatory exudates .

<u>Histologic features:</u>

Continued vascular dilatation

↓

Edema in connective tissue

↓

PMN leukocytes seen along the walls of vascular channels

↓

Migrates towards the carious lesion.

↓

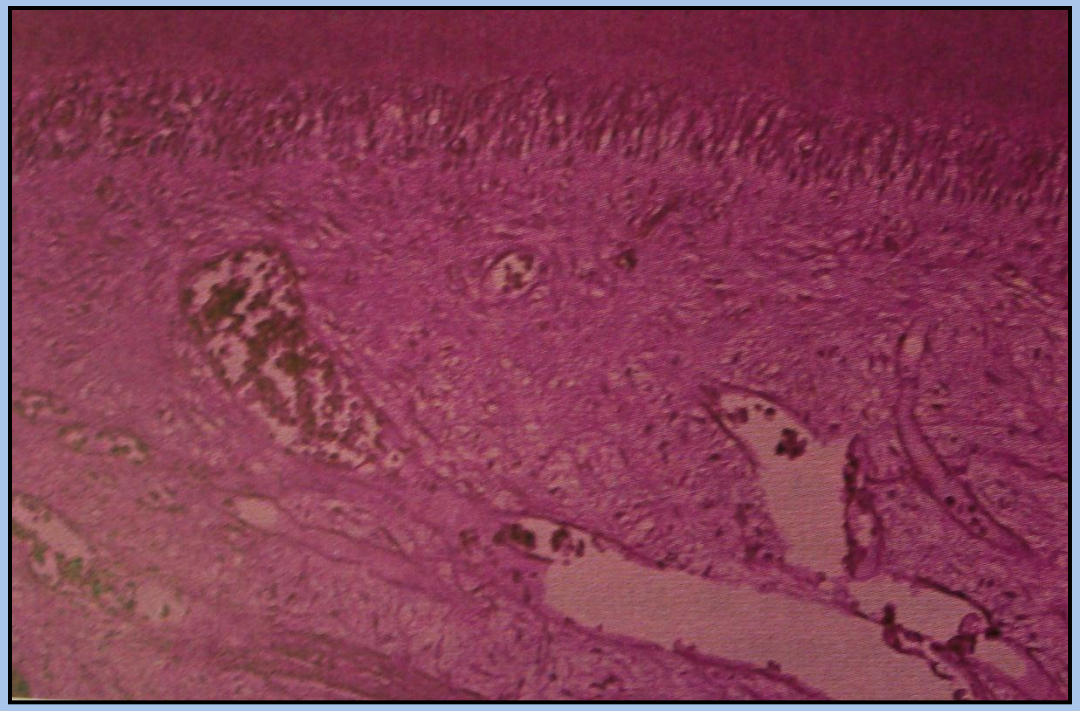
Due to localised destruction of pulp tissue

↓

Formation of small abscess called pulp abscess

↓

In H/E section appears as small voids surrounded by dense bands of leukocyte



- Acute suppurative pulpitis:

Acute inflammation spreads & most of the portion in pulp

↓ Results

Entire odontoblasts layer degenerate

Numerous small abscess may form

↓

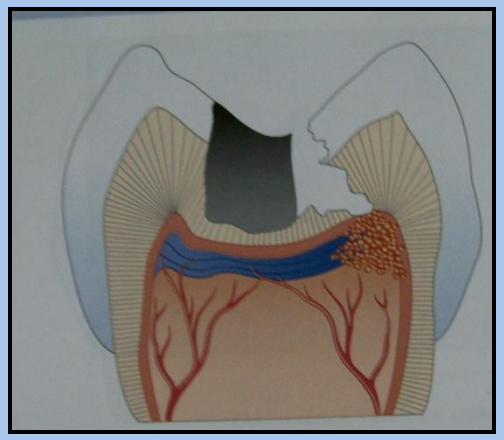
Entire pulp undergoes liquefaction necrosis

↓

Acute suppurative pulpitis

Treatment:

- RCT



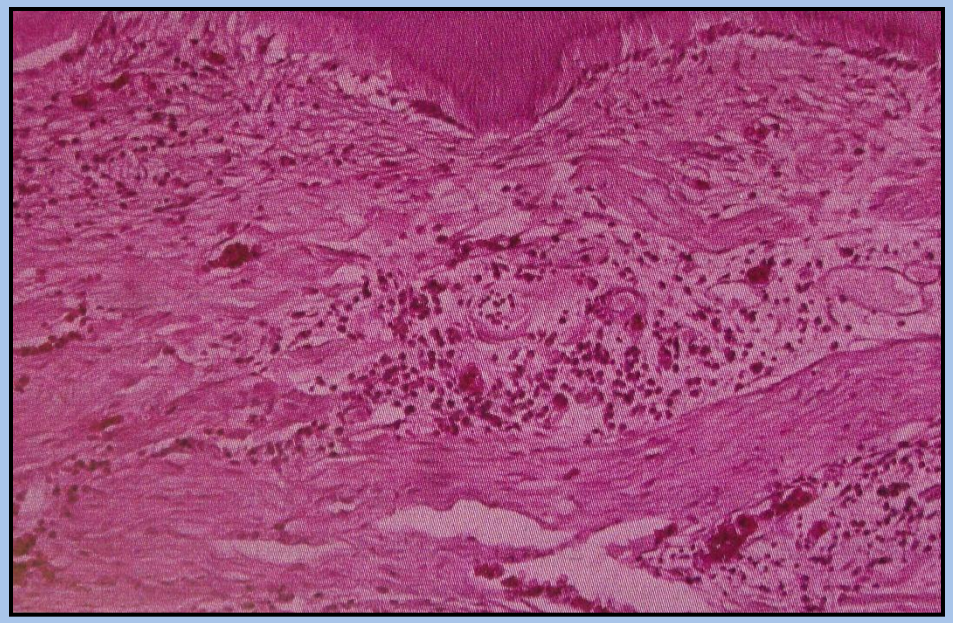
Chronic pulpitis:

- May arise squeal of a previous acute pulpitis
- Occurs as chronic type of disease from the onset

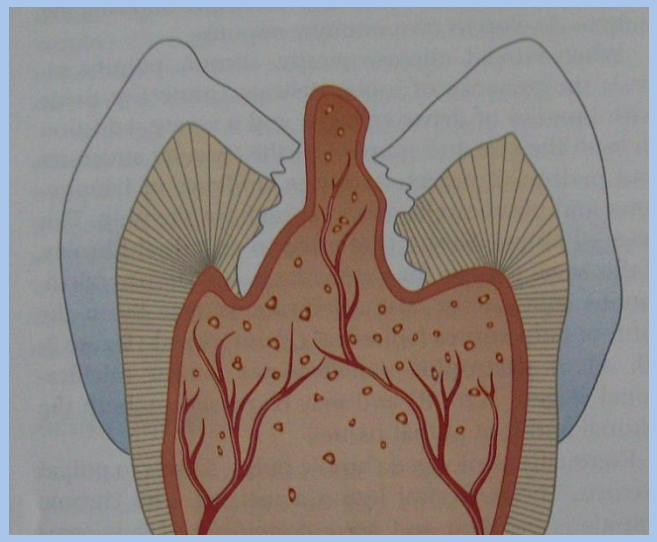
Clinical feature:

- Pain is not prominent feature but patients may c/o mild, dull ache & it is intermittent in nature
- Stimulation to electric pulp vitality tester is increased.
- Carious lesion with wide open & exposure to oral environment shows less pain

- Histological feature:
  - Characterized by infiltration of chronic inflammatory cells.
  - Capillary becomes prominent
  - Granulation tissue
  - Pulp abscess may become quiescent & surrounded by a fibrous connective tissue
  - Micro organism may found in the pulp tissue, esp in the area of a carious exposure.
- Treatment:
  - RCT
  - Extraction



Chronic Hyperplastic pulpitis : ("pulp polyp")



- A productive pulpal inflammation due to an extensive carious exposure of a young pulp. This disorder is characterized by the development of granulation tissue covered at times with epithelium and resulting from long-standing , low grade irritation.

- May occur chronic from onset or chronic stage of a previously acute pulpitis
- Clinical feature:
  - Occurs exclusively in children & young adults who possess a high degree of tissue resistance & reactivity
  - Most commonly involved is deciduous molars & permanent 1st molar
  - It involves in teeth with large, open carious lesions.
  - Pulp appears as a pinkish red globule of tissue protruding from the pulp chamber
  - Contains few nerves, it is relatively insensitive

Symptoms:

Chronic hyperplastic pulpitis is symptomless, except during mastication, when pressure of the food bolus may cause discomfort.

- Histologic feature:
  - Basically granulation tissue made up of delicate connective tissue fibers interspersed with variable No. of small capillaries.
  - Surface may or may not covered with stratified epithelium
    - Epithelium may migrate from
      - Adjacent gingiva
      - Arise from sloughed epithelium within the oral
  - Deeper pulp tissue demonstrate a chronic inflammatory infiltrate.

Diagnosis :

- Generally seen in the teeth of children and young adults.
- The appearance of the polyp tissue is clinically characteristic.
- A fleshy reddish pulpal mass fills most of the pulp chamber or cavity or even extends beyond the confines of the tooth.
- At times, the mass is large enough to interfere with comfortable closure of the teeth.
- This tissue bleeds easily because of a rich network of blood vessels.

- Radiographs generally show a large, open cavity with direct access to the pulp chamber.
- The tooth may respond weak or not at all to the thermal test, unless one uses extreme cold, as from an ethyl chloride spray.
- More current than normal may be required to elicit a response by means of the electric pulp tester.

Differential Diagnosis

- proliferating gingival tissue.

pulp polyp	gingival polyp
Stalk of tissue is from pulp chamber	From gingival
More painful & sensitive	Less
More vascular	More fibrous
Seen mostly long standing carious Lesion in deciduous teeth or in permanent molar	Seen any where on gingival

Treatment :

- Polyp tissue elimination & root canal treatment.

Necrosis of Pulp

- Necrosis is death of the pulp. It may be partial or total; depending whether part of or the entire pulp is involved.
- Necrosis, although a sequel to inflammation, can also occur following a traumatic injury in which the pulp is destroyed before an inflammatory reaction takes place.

- Pulp gangrene:
  - Defined as necrosis of tissue due to ischemia with superimposed bacterial infection.
  - Associated with foul odor when it is opened for endodontic treatment.
  - Dry gangrene occurs when the pulp die for some unexplained reasons. May be due to traumatic injury.

Symptoms:

- A necrotic pulp causes no painful symptoms.
- Frequently, discoloration of the tooth is the first indication that the pulp is dead.
- At times, of examination of the tooth may have a definite grayish or brownish discoloration and may lack its usual brilliance and luster.

Diagnosis:

- Radiographs generally show a large cavity or filling, and a thickening of the periodontal ligament.
- Tooth with a necrotic pulp does not respond to cold, the electric pulp test, or the test cavity.

Treatment:

Preparation and obturation of the root canals.

Disease of the Periradicular Tissues:

I. Acute periradicular diseases

- Acute alveolar abscess
- Acute apical periodontitis

Vital

Non vital

II. Chronic periradicular diseases with areas of rarefaction

- Chronic alveolar abscess
- Periapical Granuloma
- Periapical cyst

Interrelationshios of periapical infection

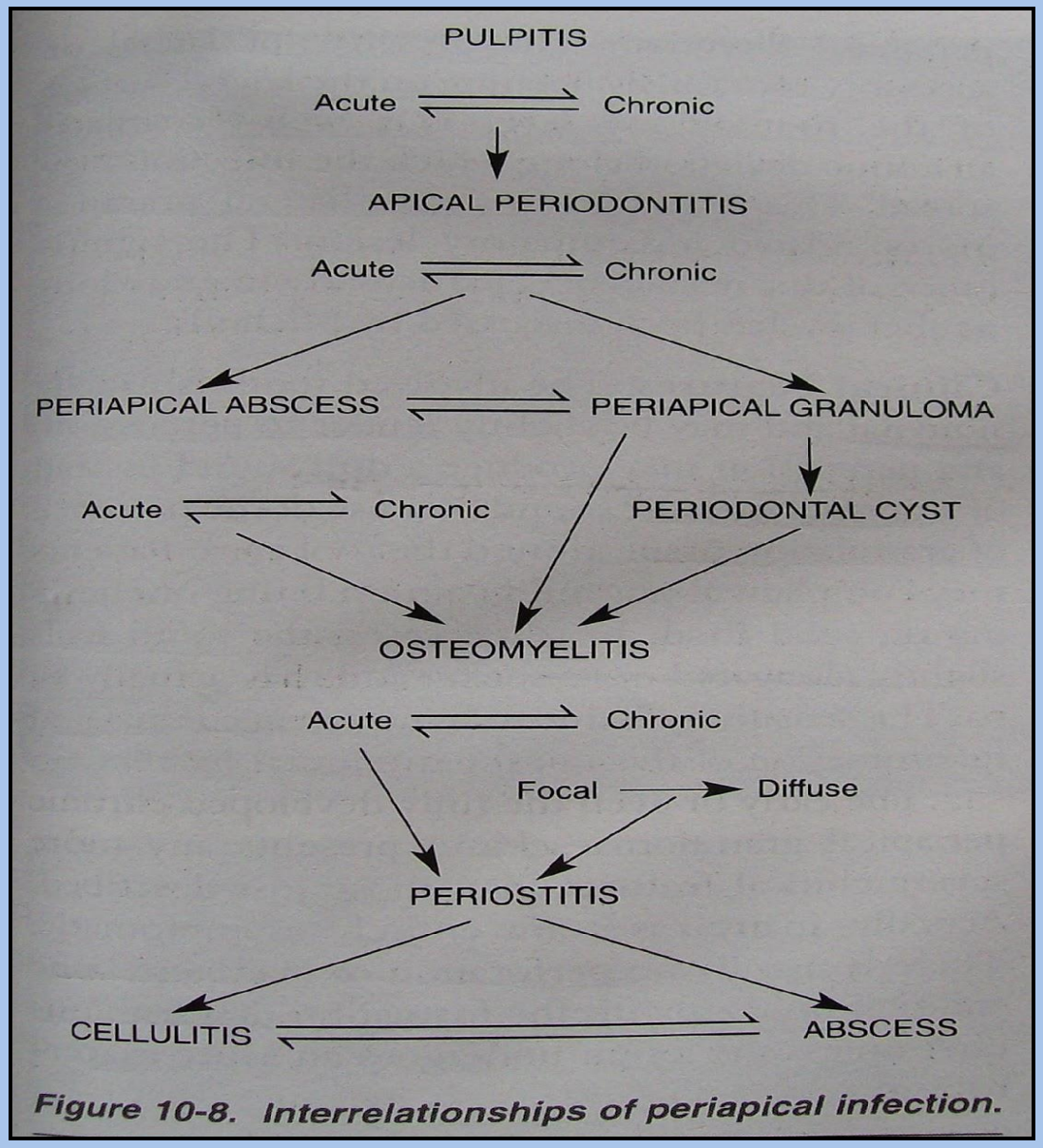


Figure 10-8. Interrelationships of periapical infection.

Acute Alveolar Abscess:

(Acute abscess, acute apical abscess, acute dento alveolar abscess, acute periapical abscess and acute radicular abscess.)

- An acute alveolar abscess is a localized collection of pus in the alveolar bone at the root apex of a tooth following death of the pulp, with extension of the infection through the apical foramen into the periradicular tissues.

Symptoms:

- The first symptom may be a mere tenderness of the tooth that may be relieved by continued slight pressure on the extruded tooth to push it back into the alveolus.
- The patient may have severe, throbbing pain, with attendant swelling of the overlying soft tissue.

- In time, tooth, may becomes more painful, elongated and mobile.
- Swelling is usually seen in the adjacent tissues close to the affected tooth.
- Patient may appear pale, irritable and weakened from pain & loss of sleep.
- Patients may have a rise in temperature.

- Histologic feature:
  - Area of suppuration is composed
    - chiefly of a central area of disintegrating PMN leukocyte,
    - occasional lymphocyte,
    - cellular debris, necrotic material & bacterial colonies
  - Dilatation of blood vessels in the periodontal ligament & adjacent marrow spaces of the bone
  - Marrow space also show inflammatory cell infiltration
  - Tissue around the area of suppuration contains a serous exudate.

Diagnosis:

- clinical examination, and the subjective History given by the patient.
- The affected pulp is necrotic and does not respond to electric current or application of cold.
- The tooth may be tender to percussion.
- The apical mucosa is tender to palpation.
- The tooth may be mobile and extruded.

Treatment:

- Treatment consists of establishing drainage and controlling the systemic reactions.
- When symptoms have subsided, the tooth should be treated endodontically by conservative means.

Prognosis.

- The prognosis for the tooth is generally favorable, depending on the degree of local involvement and the amount of tissue destruction.

Radiographic Differential Diagnosis of Common Periapical Lesions

	Abscess	Granuloma	Cyst
Size	Any size	Not more than 1.6 cms	More than 1.6 cms
Shape	No shape	Round or oval	Round or oval
Outline	Irregular	Regular	Regular
Border	Ill-defined	Well-defined	Well-defined
Other Features	Root resorption	Tooth elevated	Root displacement Expansion of jaw Thinning of cortex

Acute Apical Periodontitis:

- 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 non vital.

- Clinical feature:
  - History of pervious pulpitis
  - Due to collection of edema in the PDL tooth is slightly elevated
  - Tenderness while biting
- **AP may seen in vital tooth**
- With occlusal trauma caused by abnormal occlusion contact
- Recently inserted restoration extending beyond the occlusal plane
- Wedging of the foreign object between the teeth such as tooth pick, food
- Blow to the tooth
- **AP also associated with non vital teeth**
- Squeal of pulpal disease
- Diffusion of bacteria & noxious product from inflamed or necrotic pulp during RCT treatment

- histologic feature:
  - PDL shows signs of inflammation characterized by vascular dilatation & infiltration with PMN leukocyte.
  - Inflammation is transient if cause is removed
  - If not it progresses with resorption of the surrounding bone.
  - Abscess formation may occur.

Diagnosis:

- The tooth is tender to percussion
- The mucosa overlying the root apex may or may not be tender to palpation.
- Radiographic examination may show a thickened periodontal ligament or a small area of rarefaction if a pulpless tooth is involved, and it may show normal periradicular structures if a vital pulp is present in the tooth.

Treatment:

- Treatment of acute apical periodontitis consists of determining the cause and relieving the symptoms. It is particularly important to determine whether apical periodontitis is associated with a vital or a pulpless tooth. When the acute phase has subsided, the tooth is treated by conservative means.

Prognosis:

- The prognosis for the tooth is generally favorable.

Acute Exacerbation of a Chronic Lesion:

(Phoenix abscess.)

- An acute inflammatory reaction superimposed on an existing chronic lesion, such as a cyst or granuloma.

Symptoms:

- At the onset, the tooth may be tender to the touch. As inflammation progresses, the tooth may be elevated in its socket and may become sensitive.
- The mucosa over the radicular area may be sensitive to palpation and may appear red and swollen.

Treatment:

- Treatment consists of establishing drainage and controlling the systemic reactions.
- **Prognosis:**
- The prognosis for the tooth is good once the symptoms have subsided.

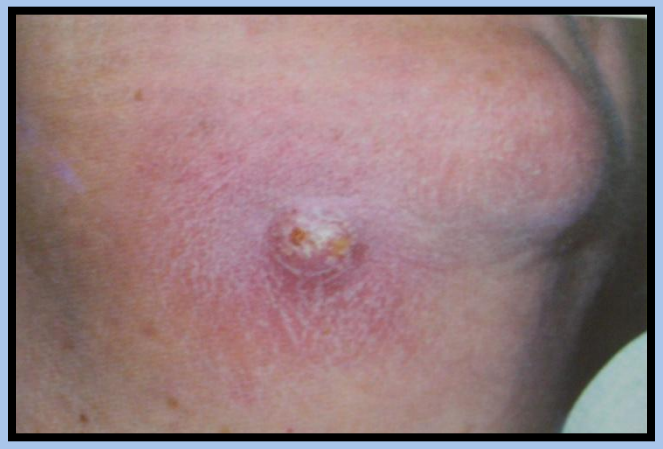
Chronic Alveolar Abscess:

(Chronic suppurative apical periodontitis.)

- A chronic alveolar abscess is a long-standing, low-grade infection of the periradicular alveolar bone.

Symptoms:

- A tooth with a chronic alveolar abscess is generally asymptomatic.
- Detected only during routine radiographic examination or because of the presence of sinus tract.



- The sinus tract usually prevents exacerbation or swelling by providing continual drainage of the periradicular lesion.
- At the intra oral opening of a sinus tract, there often is a mass of subacutely inflamed granulation tissue known as a Parulis (gum boil)
- Dental abscess may also channelize through the overlying skin drain via a cutaneous sinus

Diagnosis:

- The tooth does not react to the electric pulp test or to thermal tests.
- The radiograph often shows a diffuse area of bone rarefaction, but the radiographic appearance of lesion is non diagnostic.
- The periodontal ligament is thickened.
- The rarefied area may be so diffuse as to fade indistinctly into normal bone.

Treatment.

Treatment consists of elimination of infection in the root canal.

Followed by root canal treatment

Periapical Granuloma:

- It is localized mass of chronic granulation tissue formed in response to the infection

- Spread of infection is not always periapical direction, it may spread via lateral or accessory root canals opening & give rise “lateral granuloma”

Clinical feature:

- Involved tooth is non vital
- May be slightly tender to percussion & may produce dull sound
- Mild pain on biting or chewing on solid food
- Slightly tooth is elongated in its socket
- Sensitivity is due to hypermia, edema & inflammation of the apical periodontal liagments.

- Radiological feature:
  - Thickening of PDL at root apex
  - Proliferation of granulation tissue with resorption of bone continues & appear radiolucent area attached to root apex.
  - Well circumscribed lesion with definite demarcation from surrounding bone.
  - e may
  - tion is

- Histologic features:

Begins as hyperemia & edema of PDL with infiltration of inflammatory cells

↓ results

Inflammation & increased vascularity of the tissue

↓ leads

Resorption of bone

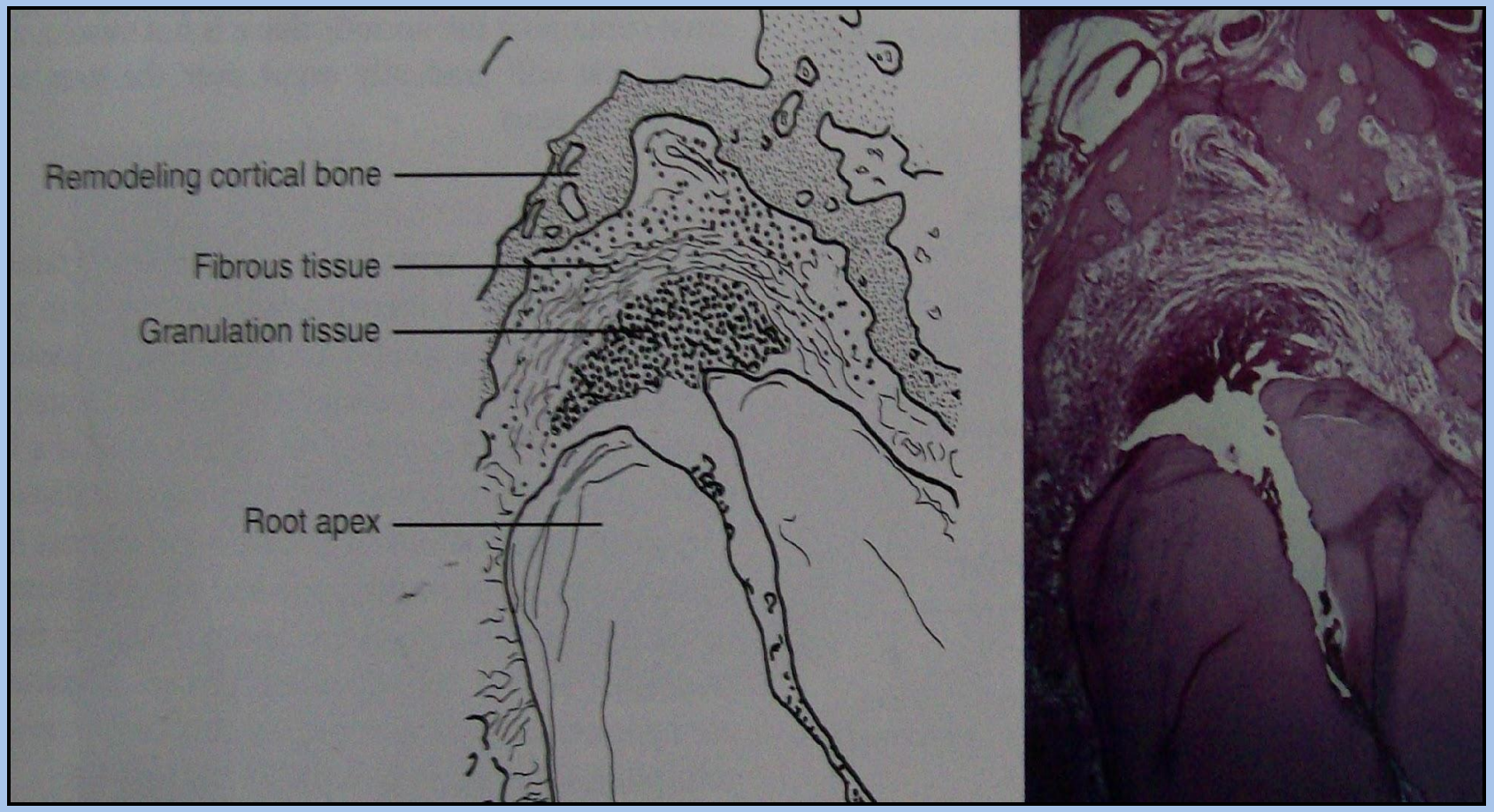
↓

Proliferation of fibroblast & endothelial cells

↓

Formation of more tiny vascular channels & delicate connective tissue

Lesion is composed predominately macrophage, lymphocyte & plasma cell.


Remodeling cortical bone
Fibrous tissue
Granulation tissue
Root apex

- Spear & page stated two type granuloma
  - Immune type:- more lymphocyte & plasma cell
  - Non immune type:- macrophage & giant cells with only a rarely plasma cell.
- 81% lymphocyte were not associated with immunoglobulin production :- T – cell
- 19% lymphocyte associated with immunoglobulin
  - IgG -74%
  - IgA – 20%
  - IgE – 4%
  - IgM – 2%
- T- cell activity accounts for bone & tooth resorption through production of osteoclastic activity factors.

- Other cells:
  - Foam cell:

    Large no. of phagocyte ingest lipid material & become collected in group, forming sheets so called foam cells
  - Cholesterol crystal:

    Appears as clear needle like spaces or clefts due to dissolving the contained cholesterol by the agent used in preparation of the tissue for histologic examination
  - Mast cell
- Connective tissue:
  - At periphery of granuloma, bundles of collagen becomes condensed
    Results in slow expansion & forms continue capsule separating the granulation tissue from bone

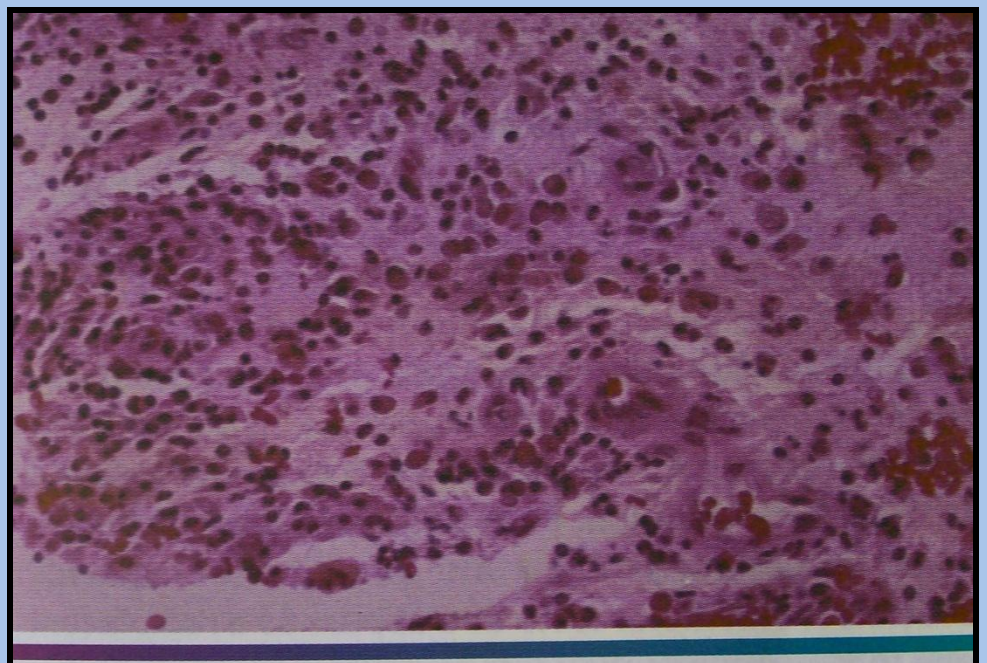
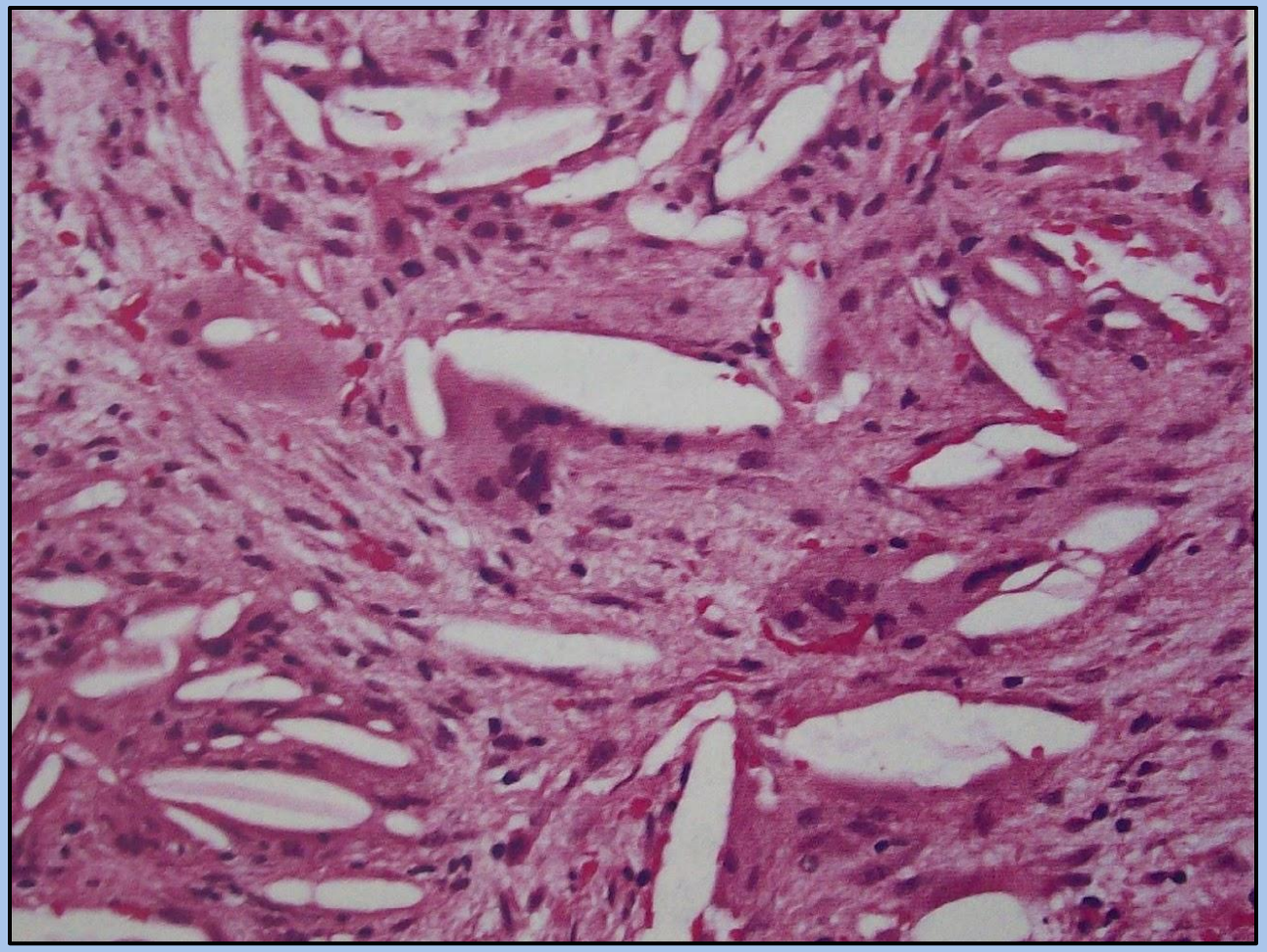
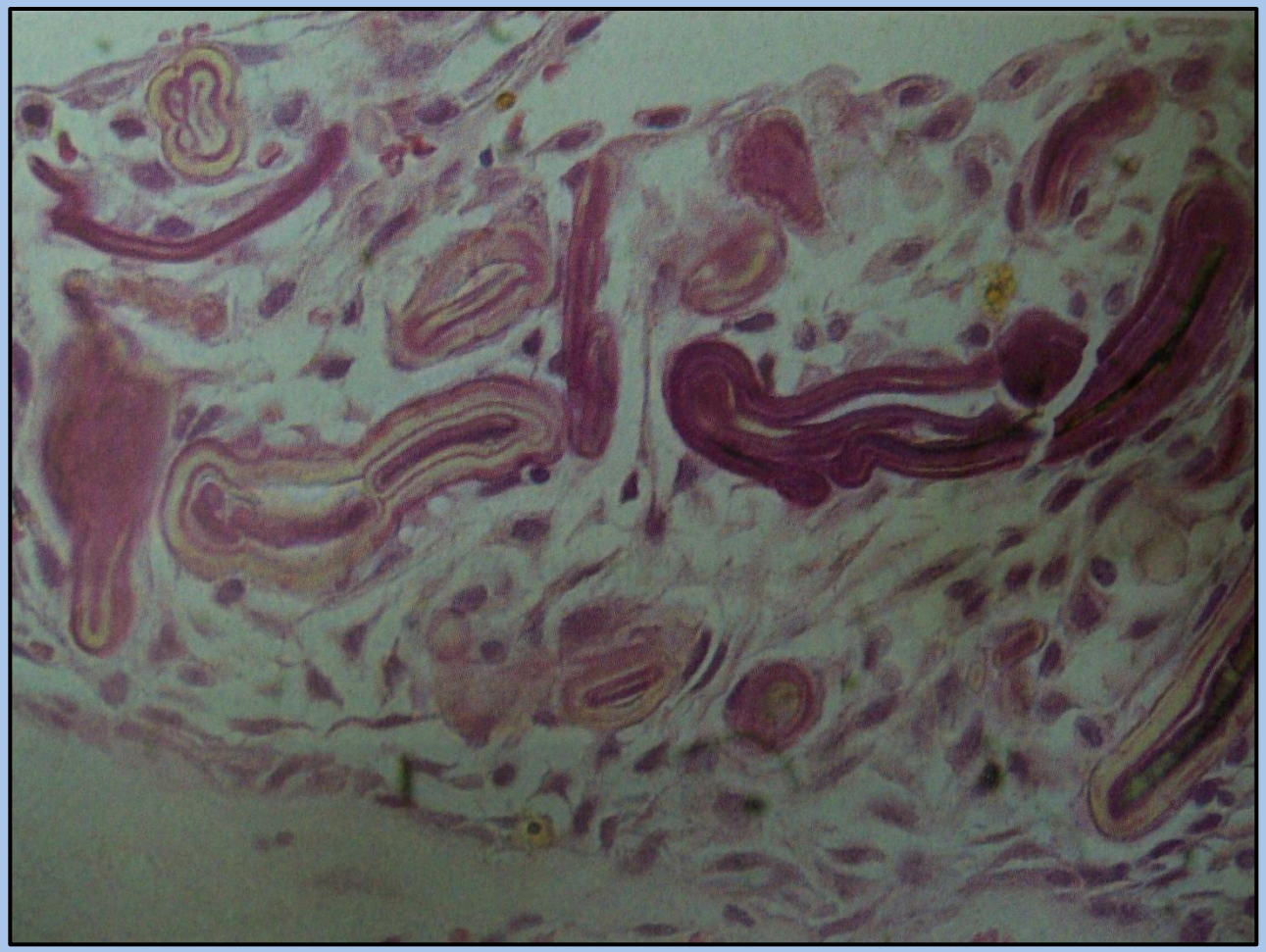


Figure 3-19 • Periapical granuloma. Granulation tissue exhibits mixed inflammatory infiltrate consisting of lymphocytes, plasma cells, and histiocytes.



- Epithelium derived from:
  - Epithelium rest of malassez
  - Occasionally:
    - Respiratory epithelium of maxillary sinus

    (if periapical lesion perforate the sinus wall)
    - Oral epithelium growing in through a fistulous tract.
    - Oral epithelium proliferating apically from periodontal pocket or bifurcation or trifurcation.
- Other finding:
  - Dunlap & Barker:
    - Giant cell hyalin angiopathy: consist of inflammatory cell infiltration , collection of foreign body type giant cell
  - Rushton bodies: composed of an eosinophilic material resembling hyalinzed material
  - pulse granuloma: fragments of foreign material resembling vegetable matter such as legums



Treatment:

Root canal therapy

Extraction on involved teeth

- Radicular cyst:
  - It is true cyst arise from epithelial remnants present normally in periodontal ligament following death of pulp & inflammation of periodontal ligament.

Etiology :

Physical , chemical, or bacterial injury resulting in death of the pulp, followed by stimulation of the epithelial rests of malassez, which are normally present in the periodontal ligament.

Pathogenesis

Considered in 3 phases

- Phase of initiation
- Phase of cyst formation
- Phase of enlargement

Phase of initiation

- Epithelial lining are derived from the epithelial cell rests of malassez which is normally present in PDL.
- These cells present in PDL proliferates & comes to lie in periapical granuloma.
- Shear (1963)

Products of dead pulp

↓ initiates

inflammation

↓ stimulation

Epithelial remnants

↓

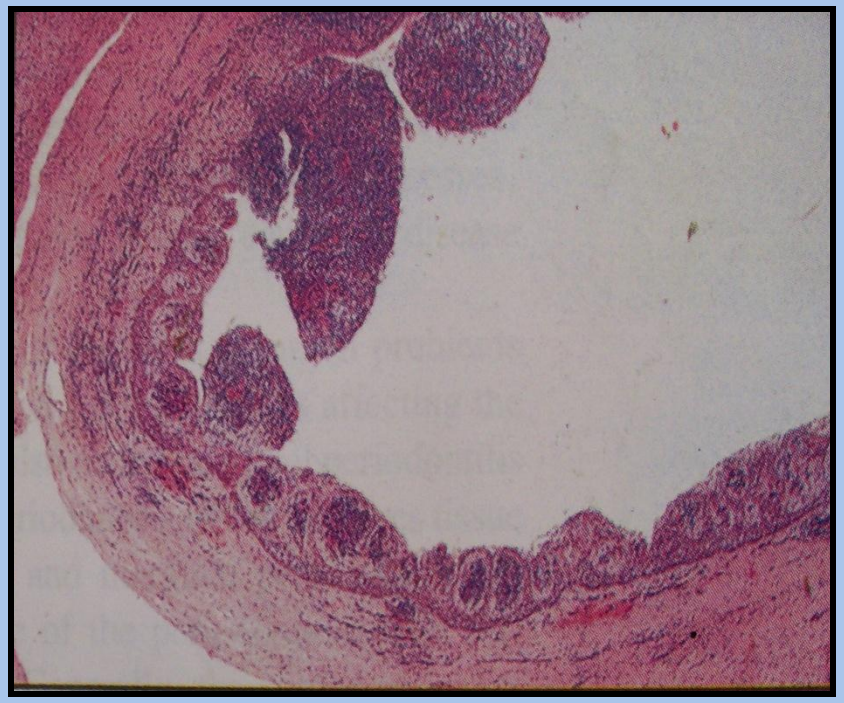
cells proliferate

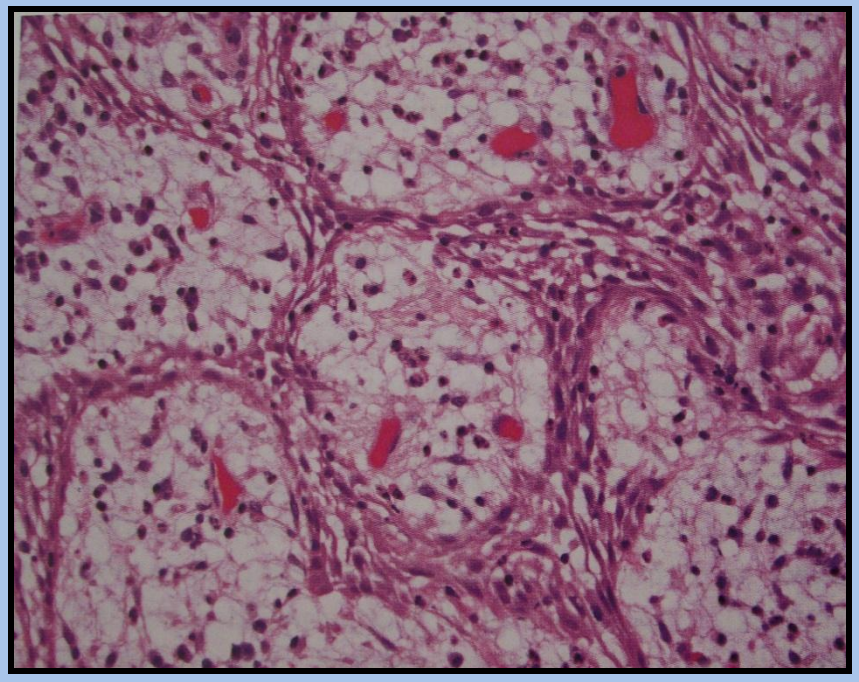
- Proliferating cells shows
  - a decrease in nucleo cytoplasmic ratio, glycogen
  - synthesize neutral lipid and ribonucleic acid and show an increase Glucose6-phosphate dehydrogenase and decrease succinic dehydrogenase activity

- Nilsen & Skang (1983):
  - Complement factors C3c & IgG in basement membrane & IgE with in the epithelium all indicative of chemotactic factors – stimulate the cell proliferation
- Gao et al (1988):
  - They speculated that activated T cell in periapical granulomas produce lymphokines that may acts on the rest of malassez causing proliferation & leading to cyst formation

Arcading & Ring pattern:

- Reason:
  - Consider as 3-D picture:
  - When the epithelial cell proliferate (they do so in different plane) forming a mass rather than sheet or strands
  - Cores of vascular connective tissue extends into the epithelium mass from all direction & resulting appearance in histological section- arcading & rings of epithelial cell surrounding by connective tissue cores
  - Apical granuloma & radicular cyst





Phase of cyst formation

- Lining by the proliferating odontogenic epithelium

One concept (summers,1974)

- epithelium proliferate & covers the bare connective tissue surface of an abscess cavity or a cavity which may occur as a result of connective tissue breakdown by Proteolytic enzyme activity

2nd concept

- Cyst cavity forms with in a proliferating epithelial mass in an apical granuloma by degeneration and death of cells in the centre

Evidence: Tencate 1972; tencate, Grupe & Zandu 1967

Proliferating epithelium mass

↓ shows

Intercellular edema

↓

Fluids coalesce to form microcysts (containing epithelium & inflammatory cell)

↓

Increase in size by coalescence with adjacent microcyst

↓

cyst

- Torabinejad(1983):
  - It is not the lack of blood supply which accounts for the death of the central epithelial cells in an apical lesion, but that the development of cavities in proliferating epithelium & final destruction of these cells are mediated by immunological mediator.

Phase of cyst enlargement:

- Toller's studies states that osmosis makes a contribution to increase in the size of cyst.
- Toller's (1966) proposed hypothesis that the content of cyst cavity are subject to an imbalance with the surrounding tissue because of absence of lymphatic drainage.
- Skang (1973,1976,1977)
  - Confirmed that accumulation of cyst fluid results essentially from inadequate lymphatic drainage of cyst cavity.
- Harris et. Al (1973,1978)
  - They postulated that intraosseous cyst expansion is facilitated by local enzyme or hormone– induces bone resorption & Suggested that active principle is a prostaglandin.

- Uitto et. al 1977
  - Collagenolytic activity:- demonstrated in radicular cyst wall & suggested that it might influence their expanssion.
- Livingston (1927)
  - Estimated radicular cyst growth is about 5mm in diameter annually
- Harris & Toller (1975)
  - Suggested that as long as inflammatory stimulant present their be continues proliferation of epithelium, inturn contributes in enlargement of cyst.
  - Once the stimulate is removed , epithelium able to differentiate to a certain extent, but keratinization is rare.
  - Further increase in the capacity of cyst cavity leads to thinning of the epithelial lining.

Clinical features :

Most common cyst

52.3%-Shear

68%-Killey & Kay (1972)

- Common in 3rd and 4th decades
- Common in males than in females.

Maxilla- 60%

Incisors-62%

cuspids-7%

Premolars-20%

Molars-11%

Mandible - 40%

Distribution is:

- Incisors - 16%
  - Cuspids - 2%
  - Premolars - 34%
  - Molars - 48%

- Majority of cases are asymptomatic & present no clinical evidence of their presence.
- Associated tooth is non vital or show deep carious lesion rarely painful or sensitive to percussion.
- Rarely produce expansion of the cortical plates.
- Pressure of the cyst may be sufficient to cause movement of the affected Teeth. Teeth may also become mobile.
- In long standing cases may undergo an acute exacerbation of the inflammatory process and develop rapidly in to an abscess that may than proceed to a cellulitis.

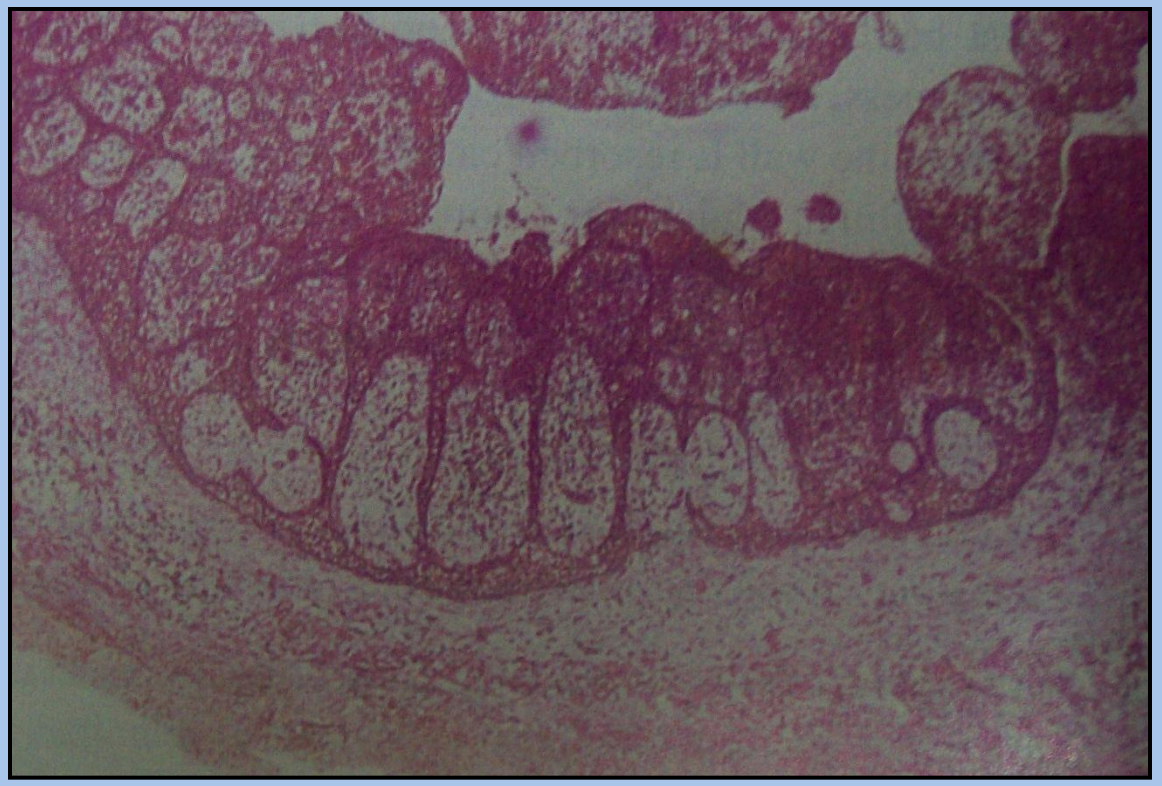
Histopathology

Epithelium lining the apical periodontal cyst is usually stratified squamous in type.

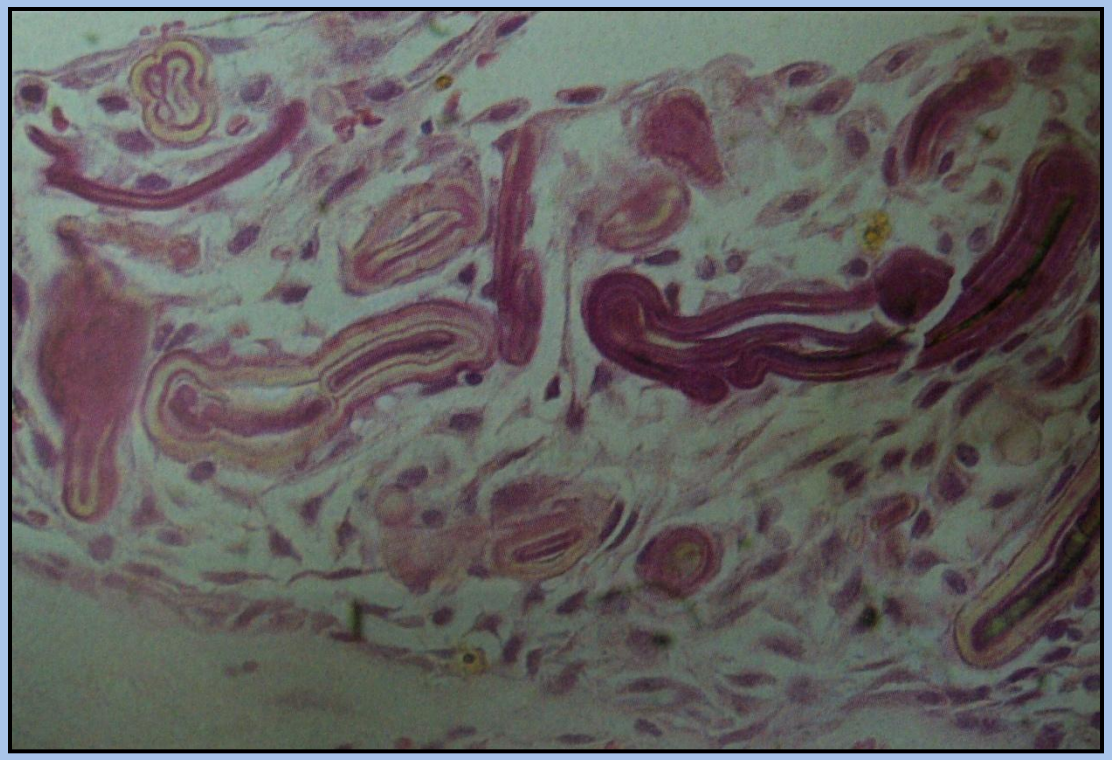
- Rare cases lined with pseudo stratified ciliated columnar or respiratory epithelium.

- Epithelial lining varies, newly formed cyst show hyperplasia & uneven thickness while established cyst shows even thickness.
- Rarely exhibit keratin formation

Epithelial cells proliferates in an arcading pattern or in rings surrounding core of connective tissue.



- Rushton body found in greater number in epithelium.
  - They are tiny linear bodies, generally associated with the lining epithelium, that appear amorphous in structure, eosinophilic in reaction & brittle in nature.
  - No clinical or diagnostic significant.
  - Origin is unknown , thought to arise from thrombus formation in small capillaries.



- Connective tissue:
  - Makes up the wall of cyst
  - Composed of parallel bundles of collagen fibers
  - Variable no. of fibroblast & small blood vessel
  - Inflammatory cell infiltration adjacent to epithelium

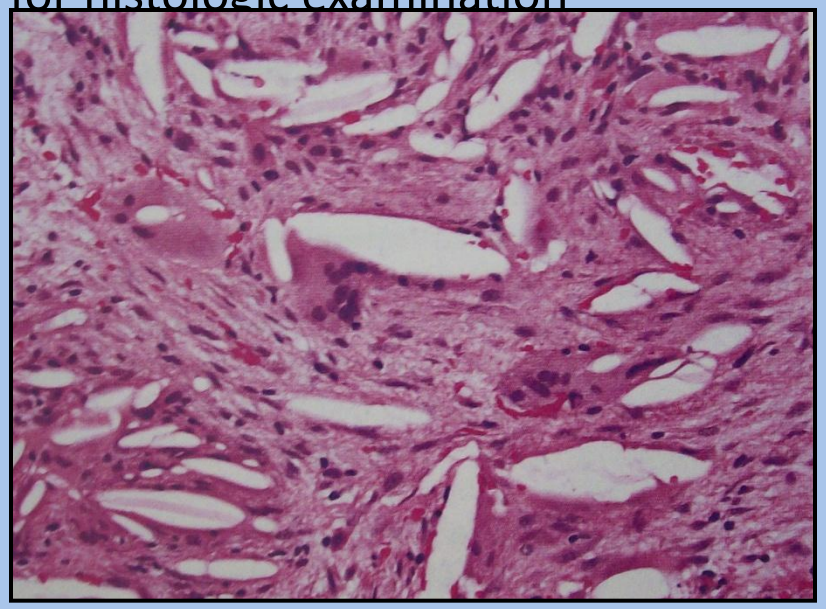
– Cholesterol crystal:

- Seen in lining epithelium

Origin

- From disintegrating RBC
- Serum which accumulate in the tissue because of inaccessibility of normal lymphatic drainage.
- Disintegrating of lymphocyte, plasma cells, & macrophage

Appears as clear needle like spaces or clefts due to dissolving the contained cholesterol by the agent used in preparation of the tissue for histologic examination

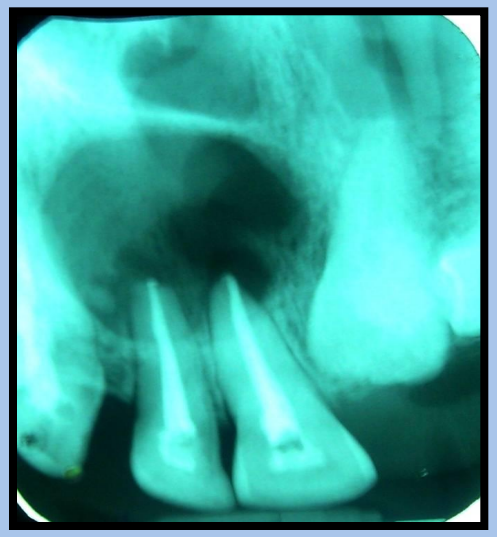


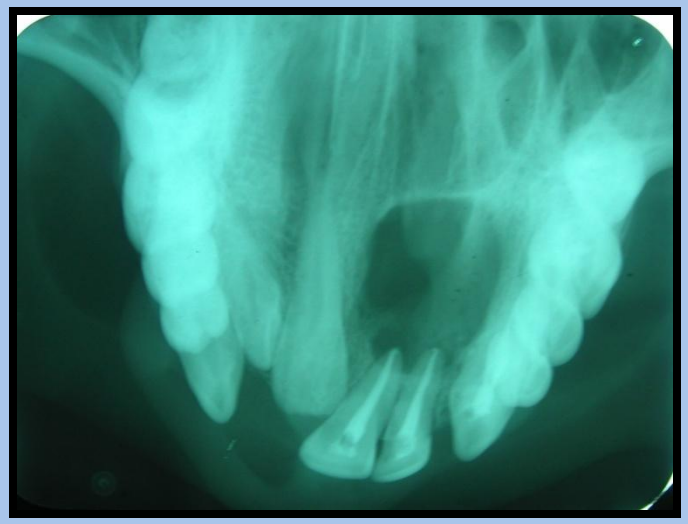
- Calcifications may be also seen in the form of amorphous calcification.
- <u>Contents of fluid:</u>
  - From watery, straw colored, blood tinged fluid to semi solid materials, with a low concentration of protein that stains palely eosinophilic.
  - May contain cholesterol
  - Limited amount of keratin
  - Blood is rare except associated with surgical procedure

- Variants of inflammatory cyst:
  - Residual cyst:
    - After extraction of involved tooth, leaving the periapical pathology untreated or incomplete removal of periapical granuloma or periapical cyst.
  - Lateral periodontal cyst:
    - When radicular cyst arise from lateral aspect of affected tooth, resulting after inflammation that pass through a lateral accessory canal
  - Inflammatory periodontal cyst or inflammatory collateral cyst
    - Cyst arise following inflammation in a periodontal pocket
  - Paradental cyst:
    - A cyst of inflammatory origin can arise from lateral to the tooth root of partially erupted third molar associated with H/O pericoronitis
  - Mandibular buccal infected cyst:
    - Inflammatory origin occurring in children i.r.t permanent 1^{st} & 2^{nd} mandibular molar

Radiographic Features

- Appears as round to ovoid radiolucency which is surrounded by thin radiopaque margin
- Most cases is identical to granuloma but it seen that cyst may be greater in size
- Actual diagnosis were establish by histologic examination.





- Differential diagnosis:
  - Granuloma: radiolucency is less than 1.6cm
  - Periapical scars: nonenlarging radiolucency is more likely periapical scars.
  - Periapical cementoosseous dysplasia (PCOD): in its early lytic & fibroblastic stage.
    - Pulp is vital
  - Traumatic bone cyst:
    - predilection for lower jaw esp. molar-premolar region
    - Associated teeth show vital pulp
    - Intact lamina dura.

Treatment :

- Radicular cysts may be treated by marsupialization or by enucleation .
- Extracted or root canal filled following an apicoectomy.
- The radiographic appearance of the periapical area of an endodontically treated tooth should be checked periodically to make sure that normal healing is occurring.