PULP AND PERIAPICAL DISEASES

Introduction

- PULP

  " Is a delicate connective tissue liberally interspersed with tiny blood vessels, lymphatics, myelinated and unmyelinated nerves and undifferentiated connective tissue cells"

- PERI-Apical – Surrounding the apex

Introduction

- Development

- Pulp - Dental Papilla
- Periapical – Dental Sac.

PULPITIS

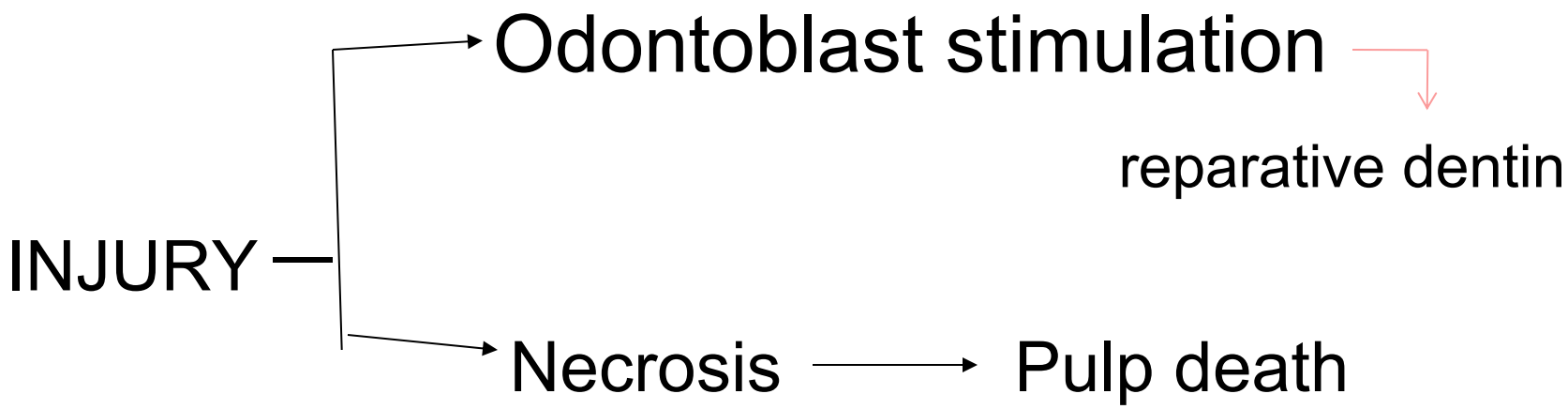
- PULP – ITIS is inflammation of Pulp.
- Principles of inflammation is same as in other body parts but it is unique in Dental pulp because
  - It is encased in hard tissue that does not allow for usual swelling associated with inflammation.
  - Absence of collateral blood supply to maintain vitality
  - Impossibility of direct application of medications without causing necrosis of the pulp.
  - Measure of severity of pulpal inflammation is determined by pain or levels of sensitivity.

PULPITIS

- Diagnostic dilemma due to Referred pain and lack of proprioception .
- Determination of level of inflammation
  - Combination of clinical criteria
    - Electric
    - Heat
    - Cold
    - Percussion along with careful analysis of patient history and clinical examination.

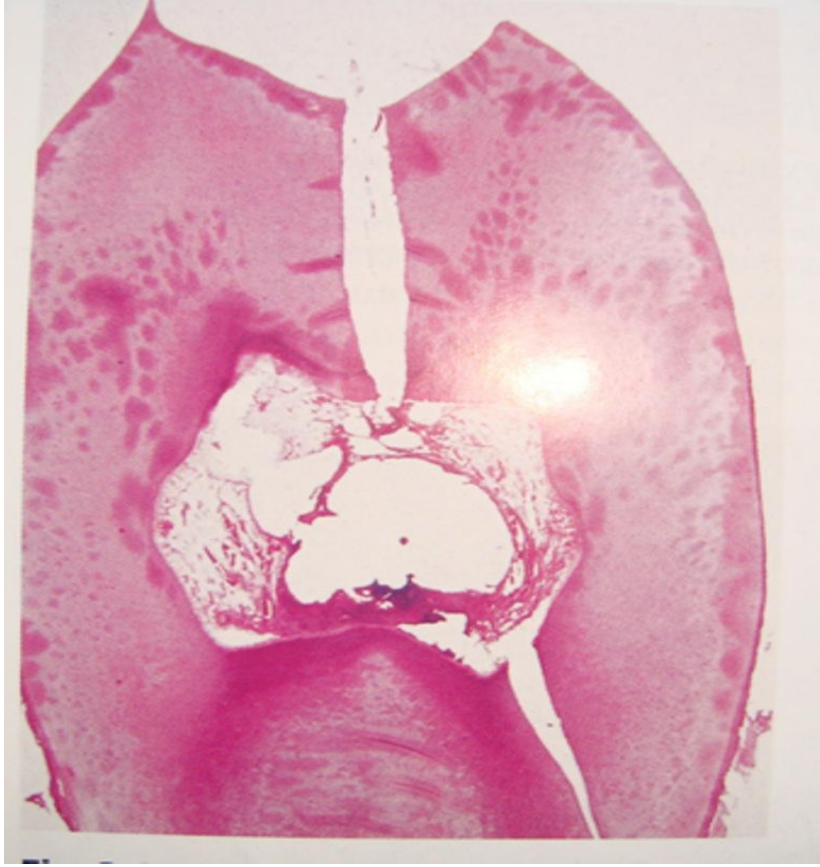
PULPITIS

- Etiology



Etiology

- Nature of injury
  - Caries.
  - Operative procedures.
  - Trauma.
  - Periodontal disease.



Diseases of Pulp - Classification

- Inflammatory
  - Reversible
    - Symptomatic - Acute
    - Asymptomatic - Chronic
  - Irreversible
    - Acute
    - Chronic
      - Asymptomatic
      - Hyperplastic Pulpitis
- Degenerative → Necrosis

- Clinical features
- classification based on clinically related factors that is helpful in treatment and prognosis.

	Pain	Vitality Test	Radiographs
Reversible Pulpitis	Mild	reversible -sensitivity to cold	no change
Acute Pulpitis	severe, constant	hyper response to none	no change
Chronic Pulpitis	Mild, intermittent	Reduced response	no change
Acute Periapical Abcess	severe: pain on percussion	no response	no change
Periapical granuloma	none of slight	no response	lucency
Periapical cyst	none to slight	no response	lucency

Focal reversible Pulpitis

Synonym- Pulp Hyperemia

Clinical Features

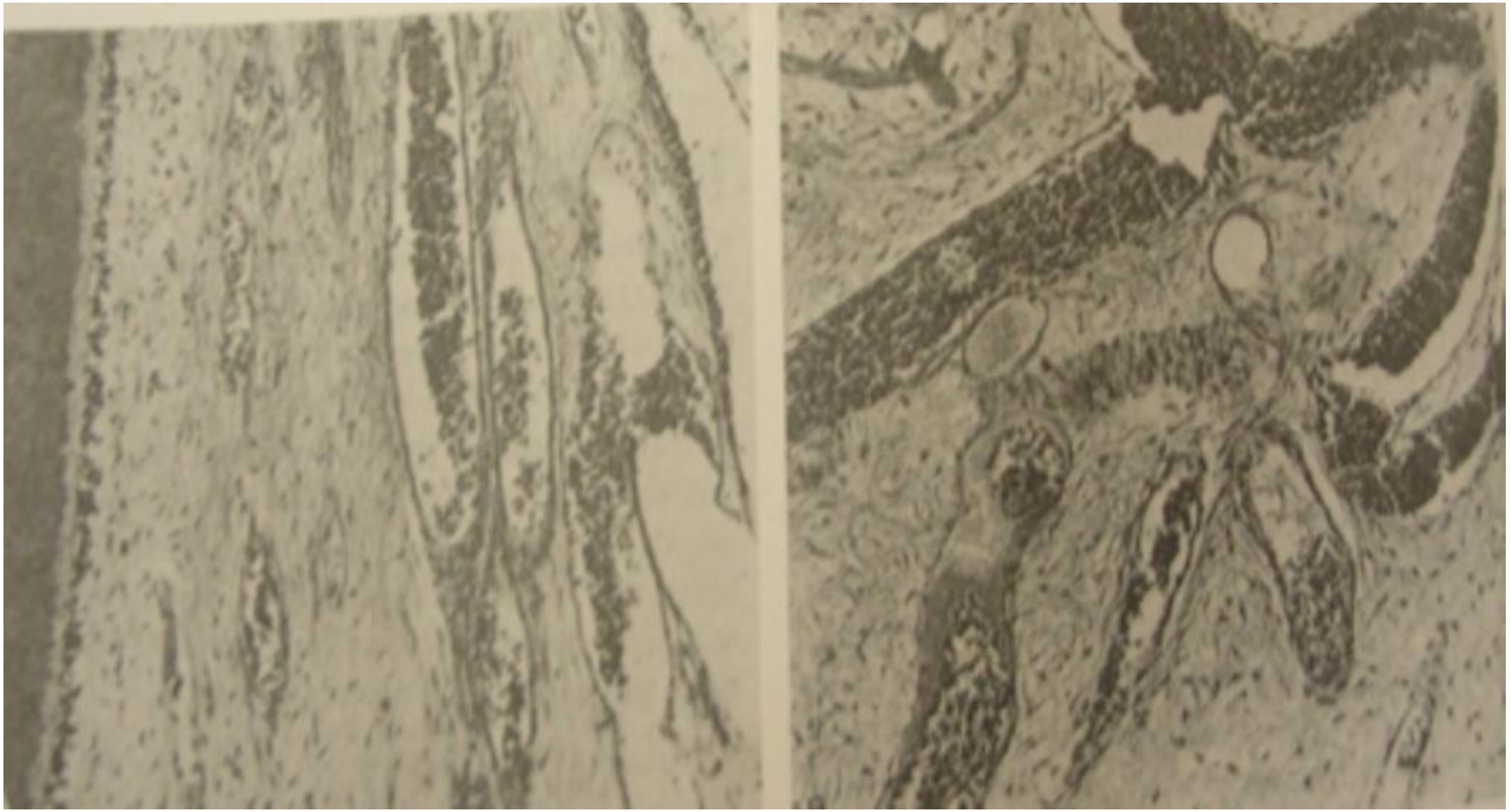
- Mild to moderate pain.
- Intermittent in nature.
- Sensitive to thermal changes – cold
- Tooth responds to low level electric current.
- Due to carious destruction or large restoration.

cont….

Focal reversible Pulpitis

Microscopy

- Dilation and Engorgement of blood vessels (hyperemia).
- exudation of plasma proteins – damage of capillary walls.
- Extravasation of RBC and Diapedesis.
- Thrombosis – due to slowing of blood flow and hemoconcentration.
- Self strangulation is incorrect belief.



Vasodilatation but no extravasation

Focal reversible Pulpitis

Treatment

Reversible – Irritant removed or else “death” of Pulp.

Acute Pulpitis

Etiology

- Progression of FRP
- Acute exacerbation of an established exacerbation.

Clinical Feature

- Large carious lesion or a restoration
- Patient presents with constant severe pain.
- Lancinating pain – due to intrapulpal abcess formation.

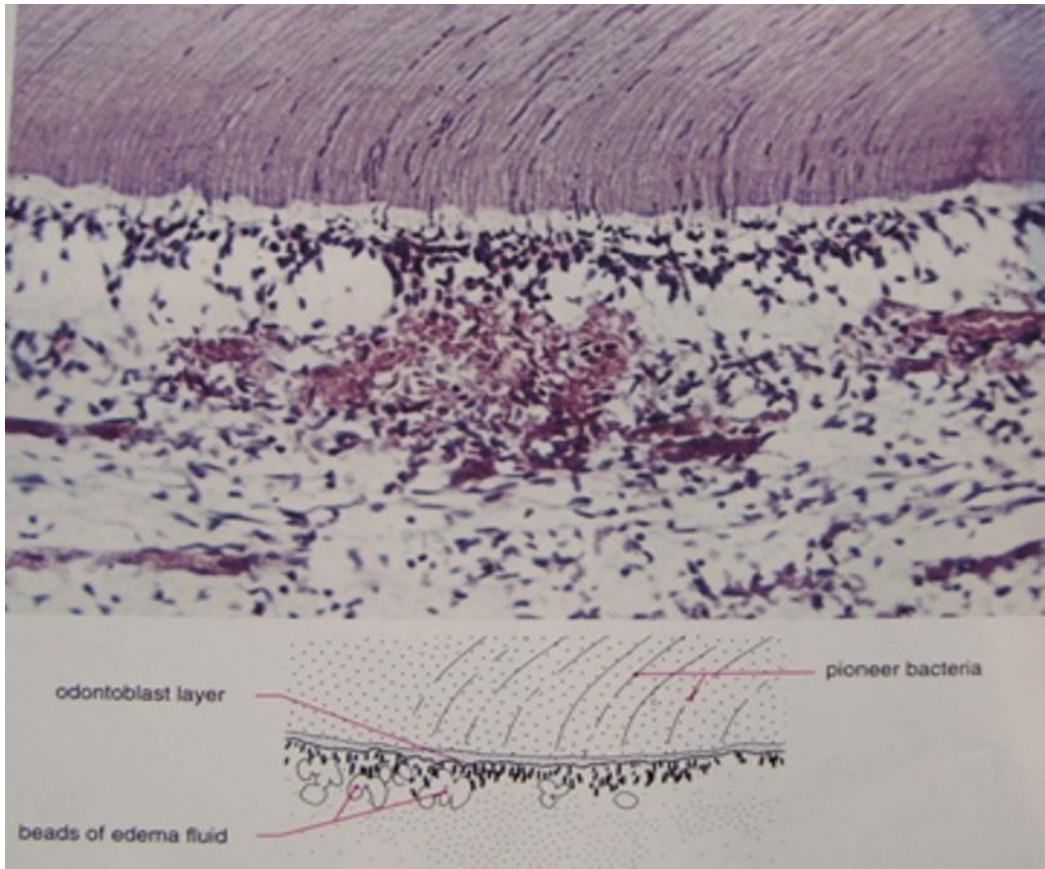
Acute Pulpitis

- Thermal test – positive
- Electric test – positive with lower level electric current.
- Pain persists after cessation of stimulus for few seconds
- Severe pain due to lack of escape route for inflammatory exudate.
- Opening from the pulp reduces symptoms – dull, throbbing ache
- Percussion might elicit less response in early stages.
- Patient extremely uncomfortable and mildly ill.

Acute Pulpitis

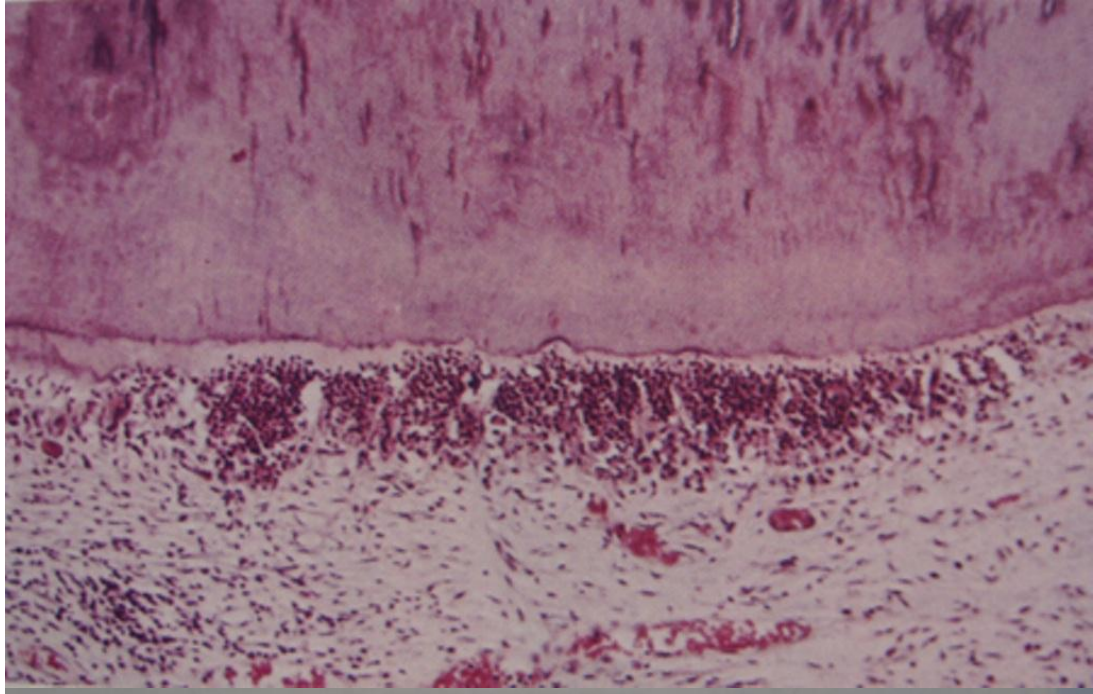
Microscopy

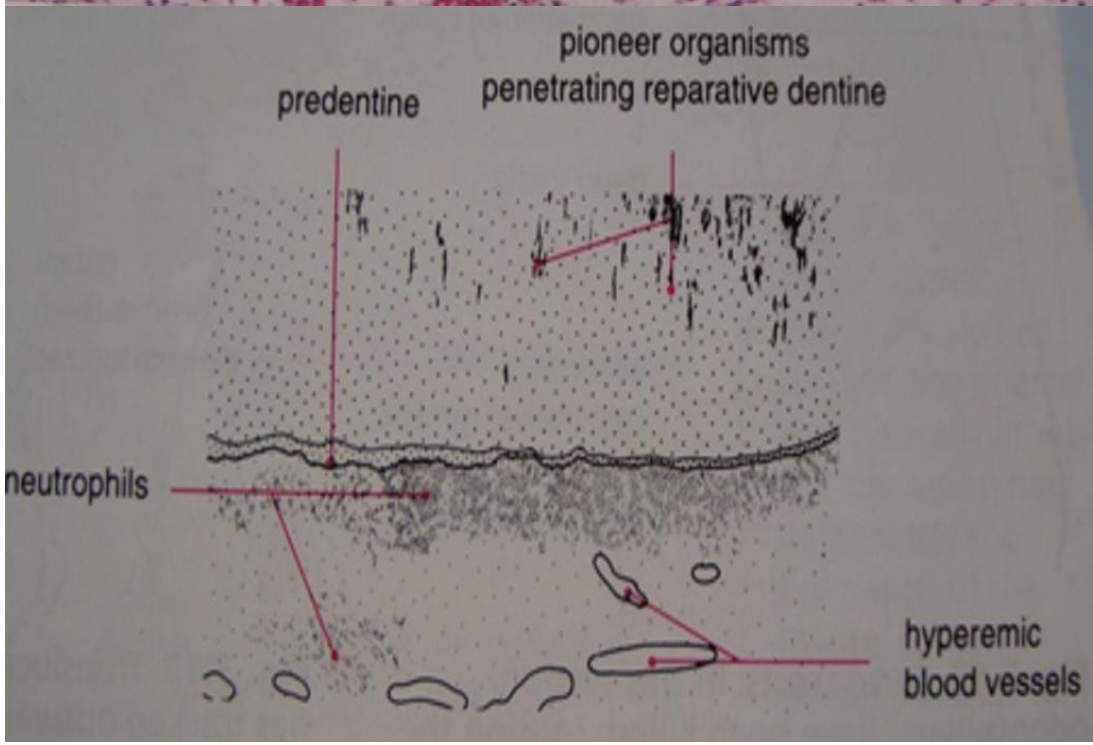
- Vascular dialatation.
- Accumulation of edema fluid.
- Diapedesis.
- WBC found beneath carious lesion.



Acute Pulpitis

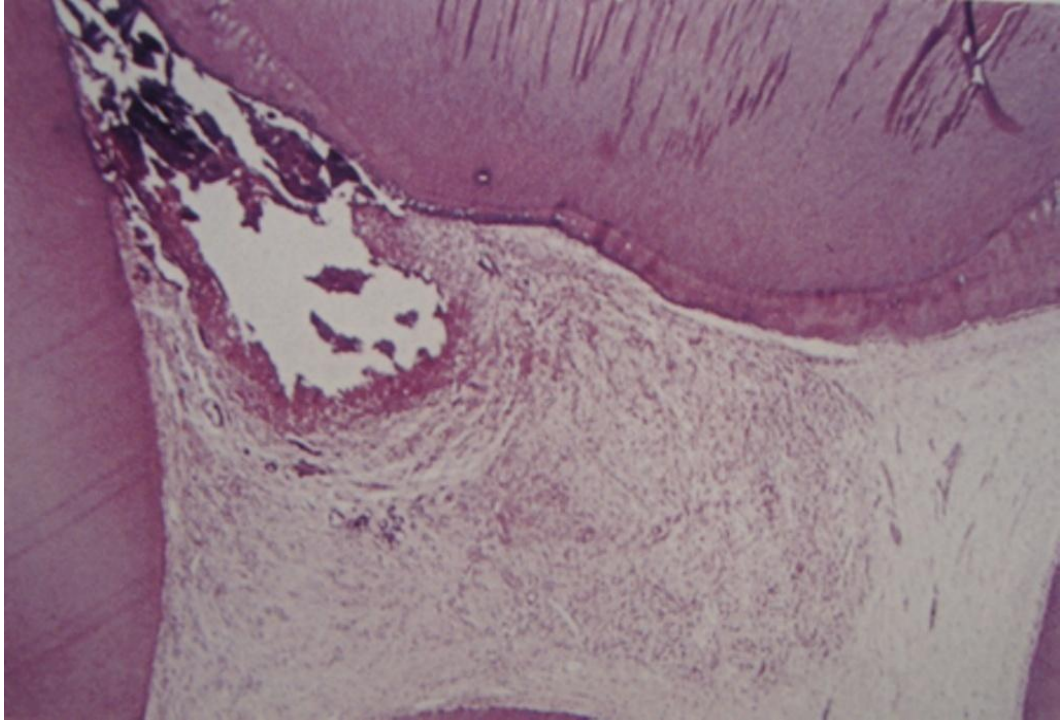
- Early pulpitis





Acute Pulpitis

- Small Pulp Abcess appears as small void sorrounded by dense band of leukocytes.

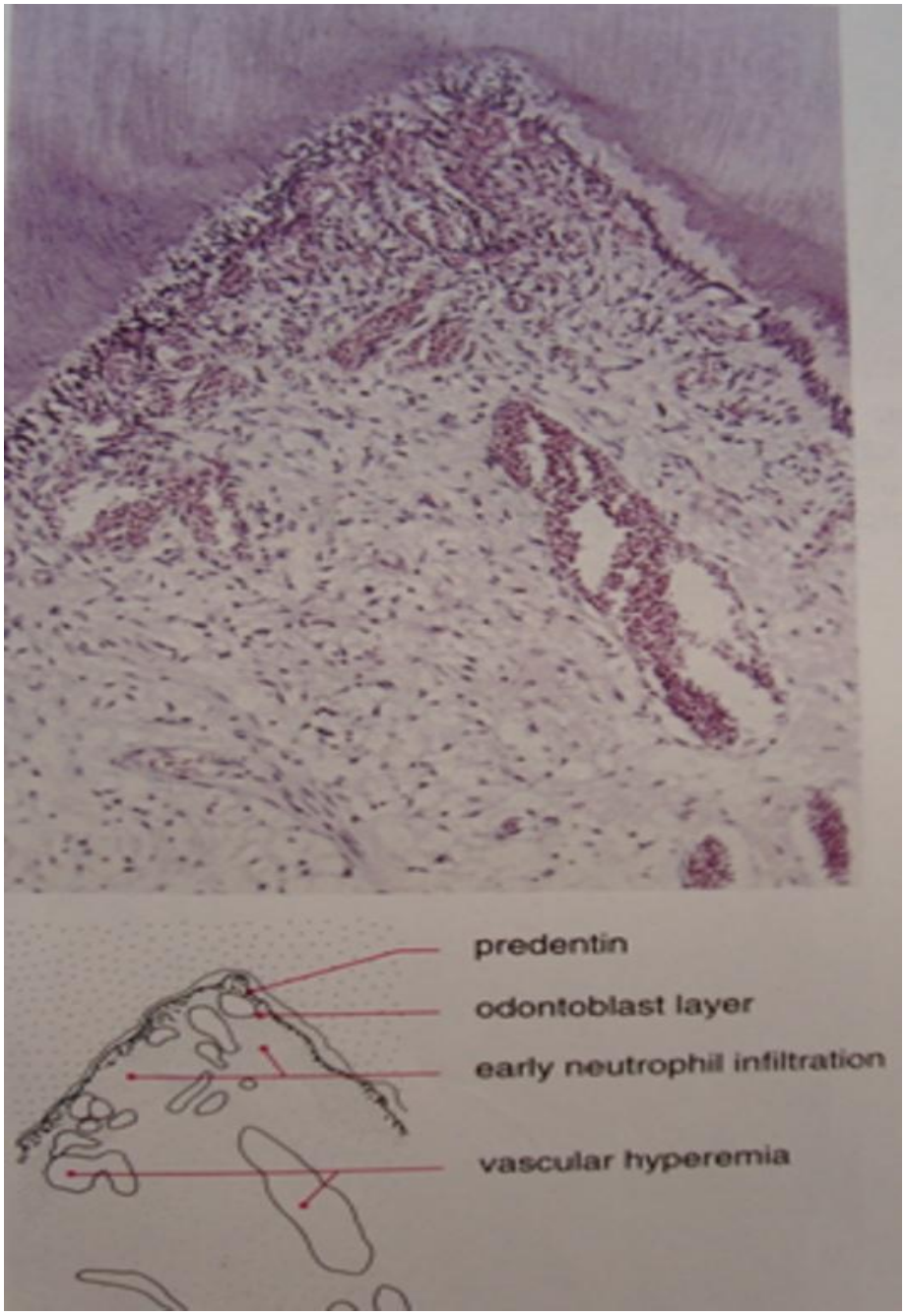


- Later, whole pulp liquefies and necrosis – "Acute Suppurative Pulpitis"
  - Bacterias of mixed population.

Treatment

Pulpotomy.

Root canal treatment.


predentin
odontoblast layer
early neutrophil infiltration
vascular hyperemia

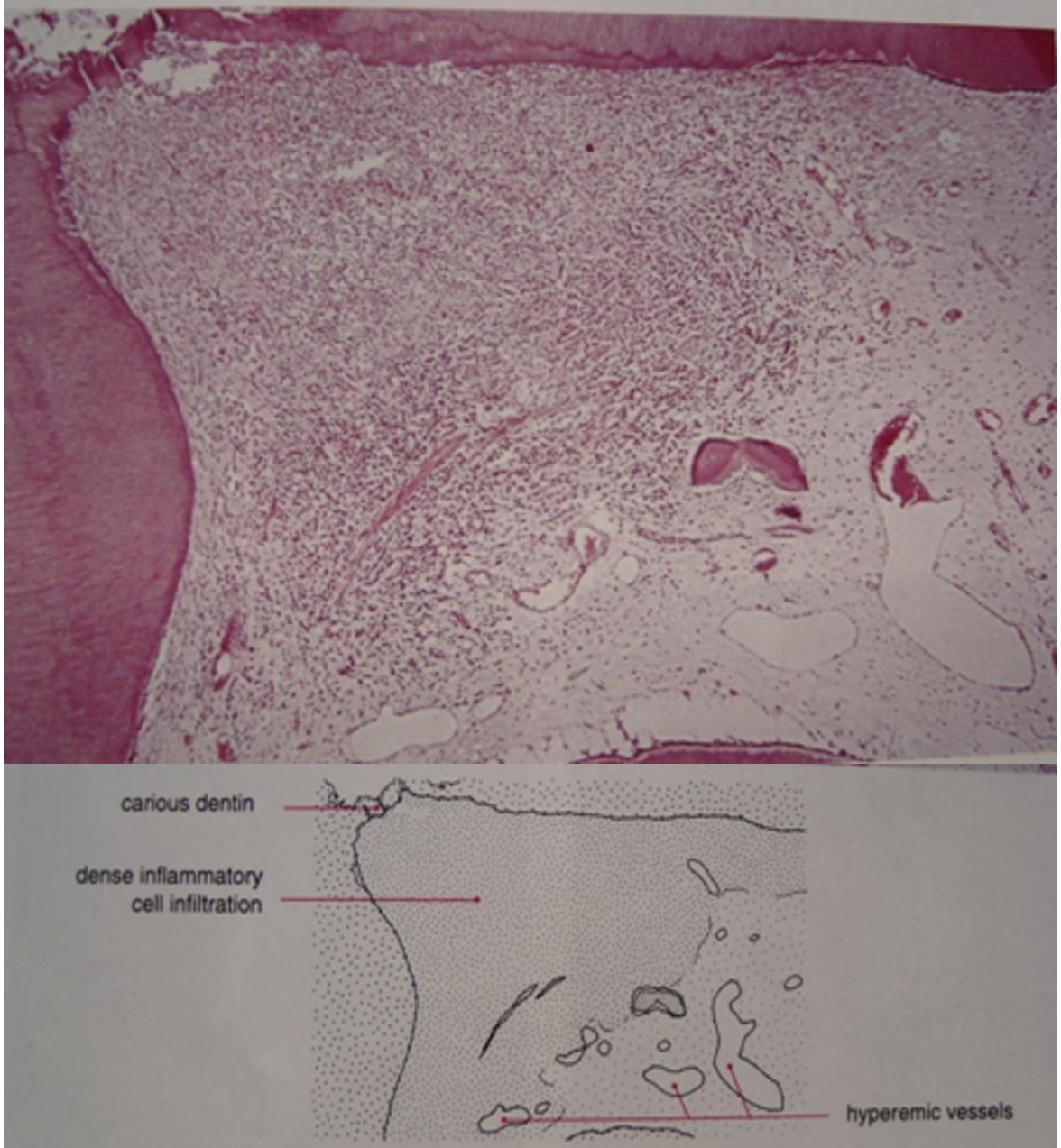


Fig. 3.18 Acute pulpitis, fully developed: the left cornu of the pulp is exposed because of breakdown of the overlying dentin, and the inflammatory infiltrate has spread across three-quarters of the pulp. In the lower right-hand corner, the pulp is relatively normal but shows gross vasodilatation.

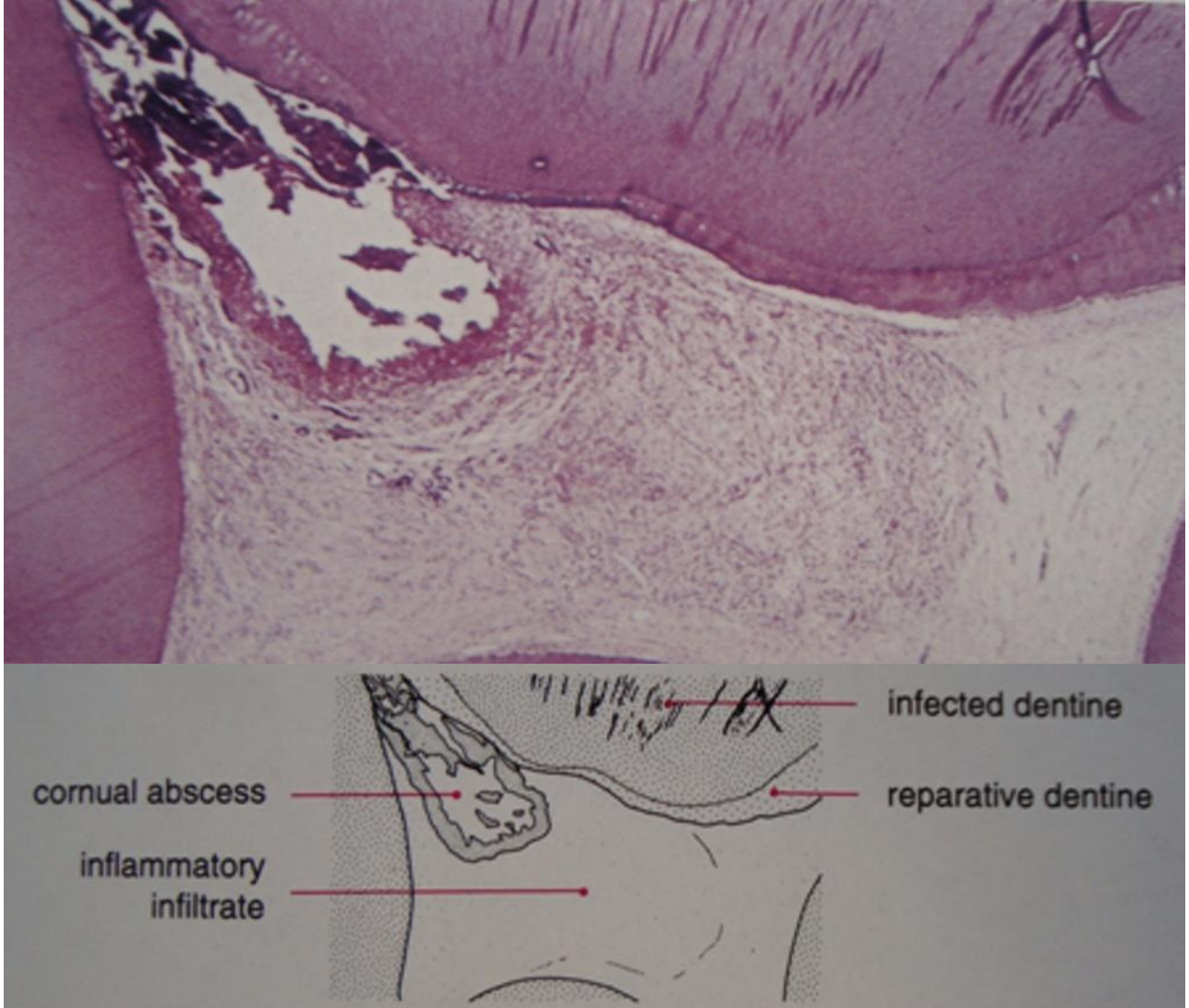
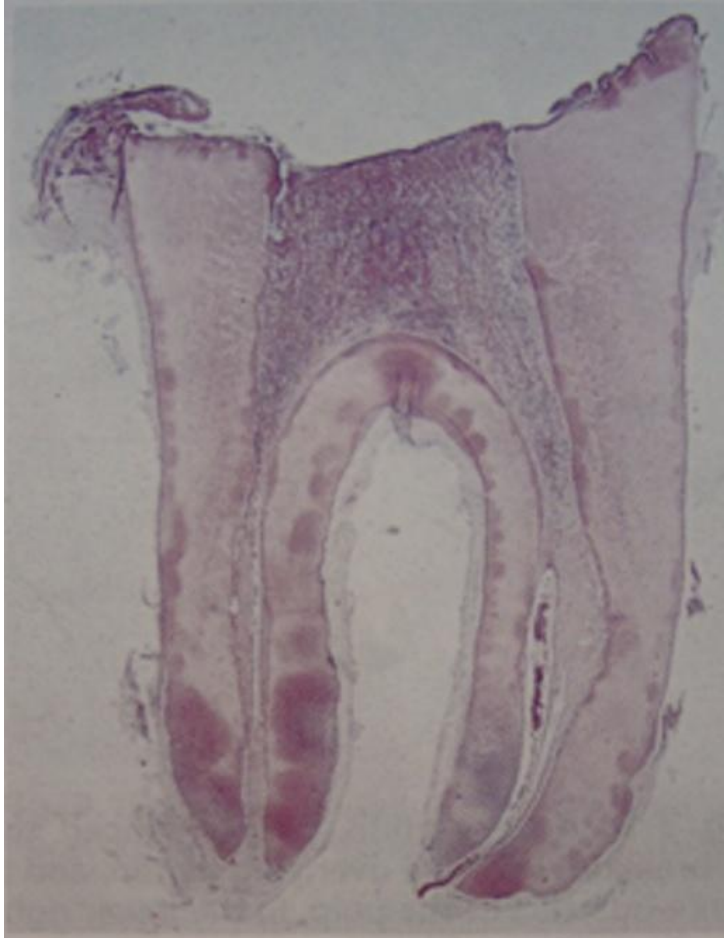
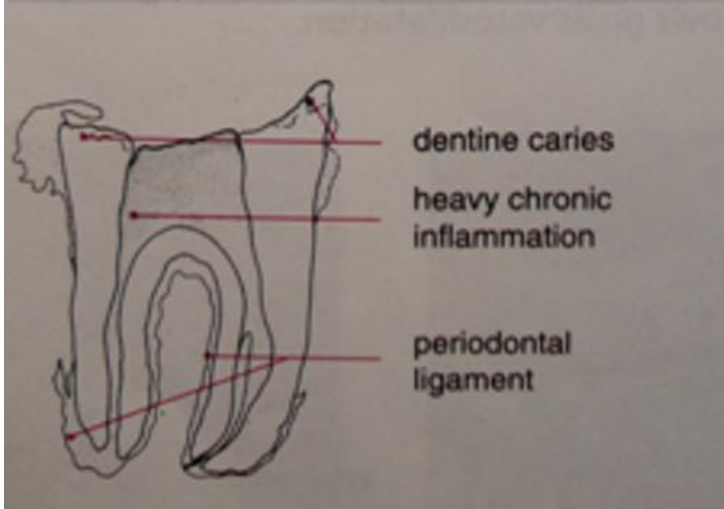


Fig. 3.20 Pulp abscess: a collection of pus in the left cornu has been partially walled off by an inflammatory and fibrous reaction. However, a dense inflammatory infiltrate has spread across the greater part of the pulp chamber and the pulp would not have survived much longer.

Chronic Pulpitis

- Long term, low grade injury.
- Mild, dull ache and intermittent in nature.
- Extended period.
- Responses to thermal and electric stimuli are reduced.
- Wide open carious lesion.
- Manipulation causes bleeding.





Microscopy

lymphocytes, plasma cells, and fibrosis are seen.

In acute exacerbation –

neutrophils are seen

capillaries are prominent

collagen fibers are seen in bundles – defense mechanism.

Microorganisms in wide exposure

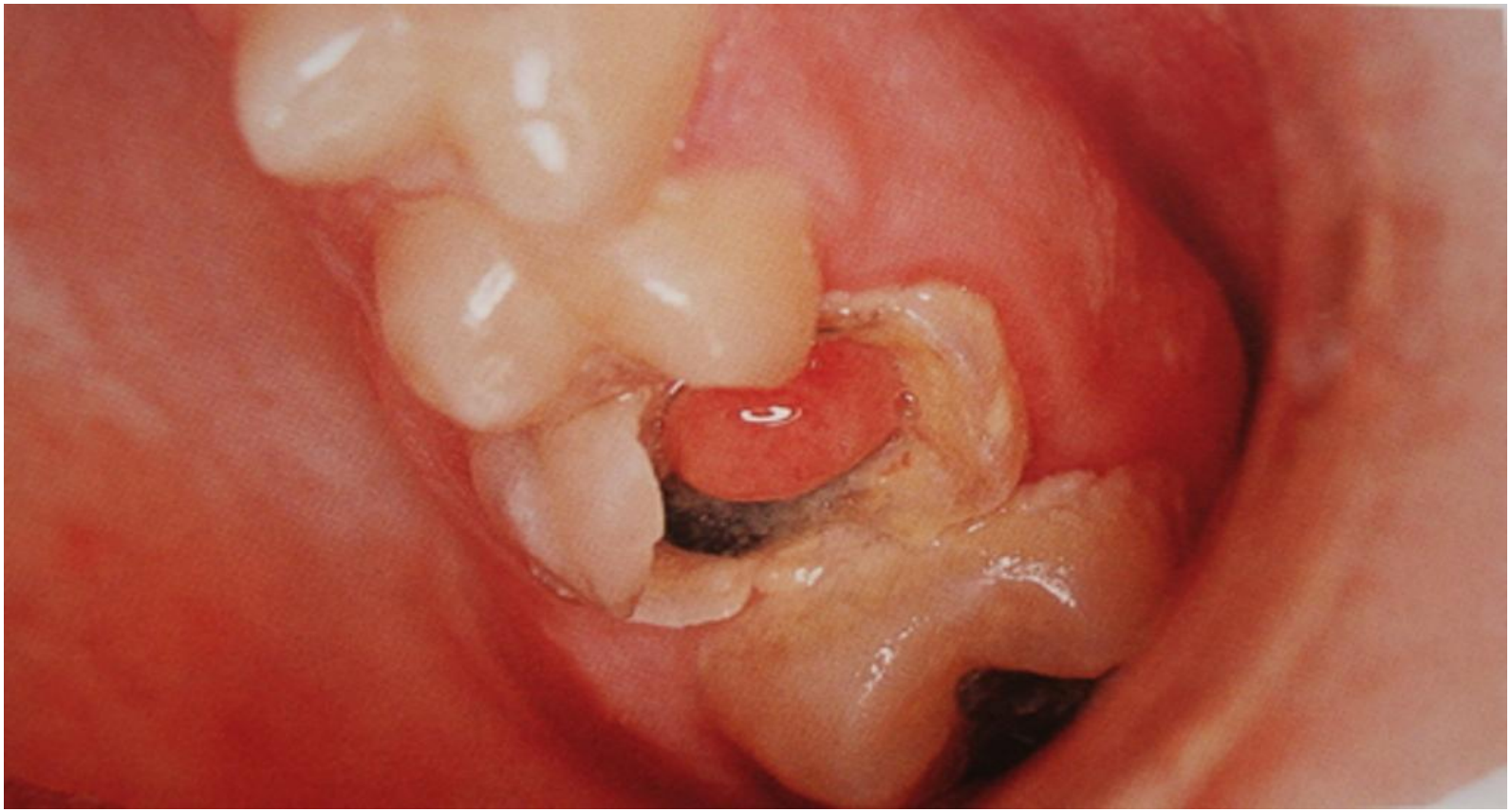
Pyogenic membrane – a fibrous connective tissue wall surrounding a Pulp abcess.

Treatment - RCT or Extraction.

- Chronic Hyperplastic Pulpitis

Synonym – Pulp Polyp

Form of Pulpitis , wherein pulp instead of perishing, reacts by excessive and exuberant proliferation.



Chronic Hyperplastic Pulpitis

Clinical Features

- Large open Carious teeth involved.
- Affected pulp appears as Pinkish – red globule of tissue protruding from pulp chamber.
- Insensitive to manipulation due to fewer nerves.
- Lesion may or may not bleed readily.
- Seen in molar teeth of children and young adults.

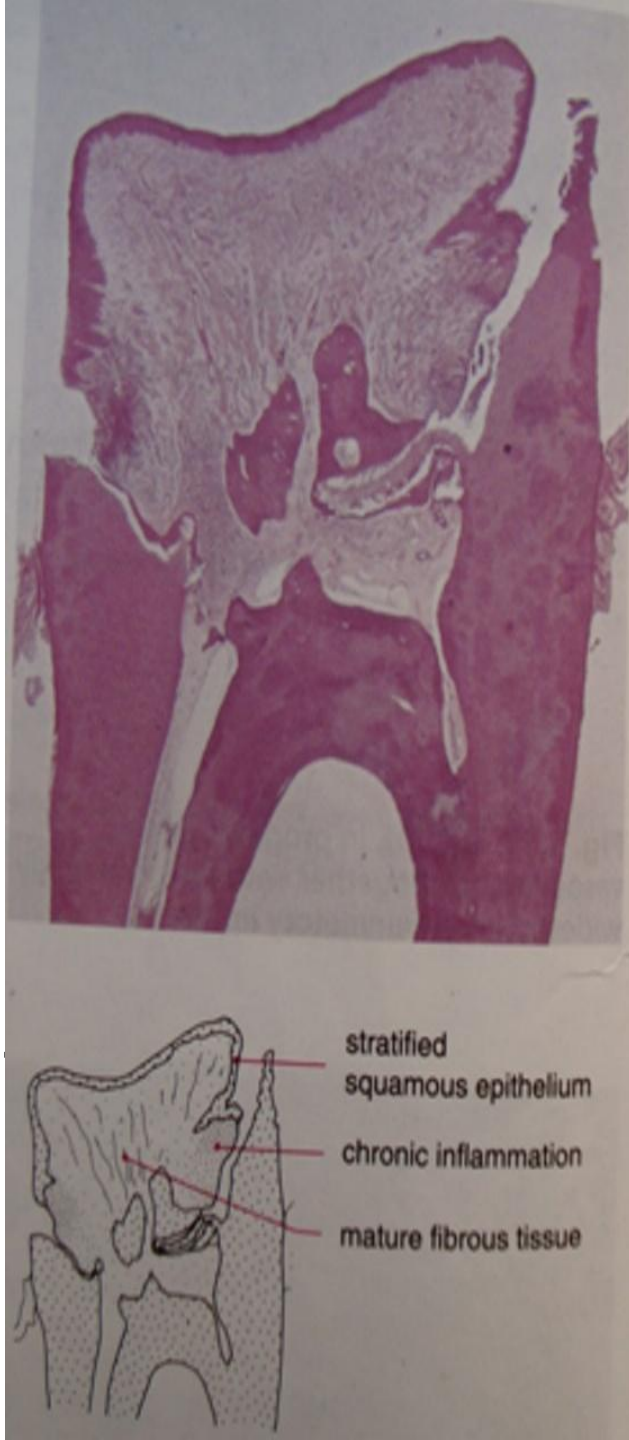
(large open apex and high tissue resistance)

Microscopy

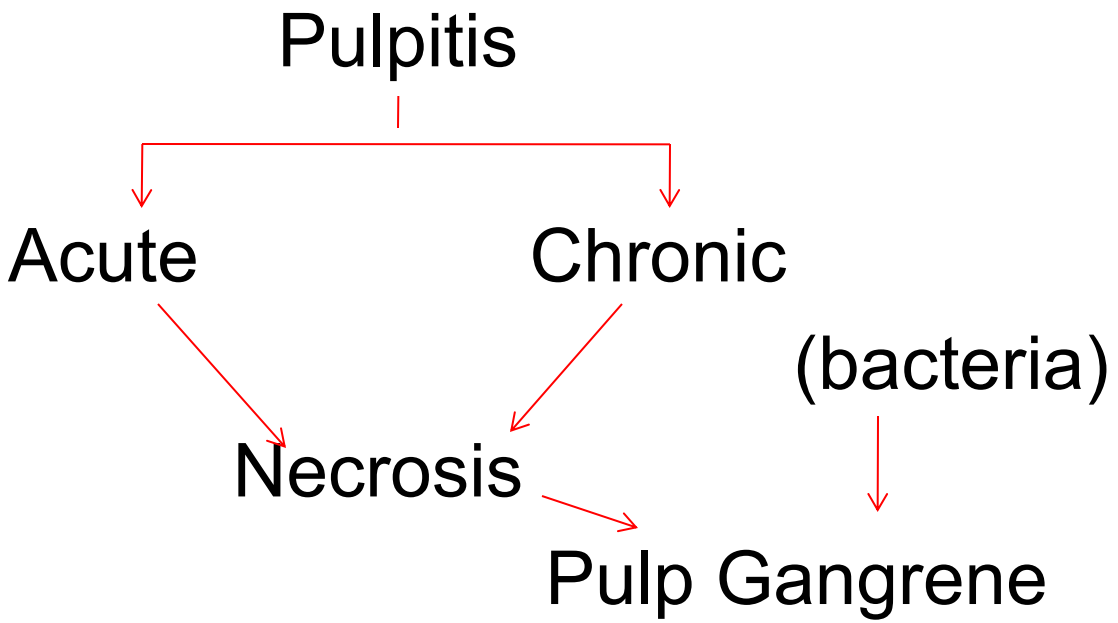
Basically granulation tissue

- Delicate connective tissue fibers
- No. of small capillaries.
- Inflammatory cell infiltration
  - » lymphocytes
  - » plasma cells

- Granulation tissue becomes Epithelialised – Autotransplantation.
- Later, tissue organization leads ↓ decreased vascularity and ↑ fibrosis.



Gangrenous Necrosis of Pulp



Foul odour when opened for RCT

End result of Pulpitis.

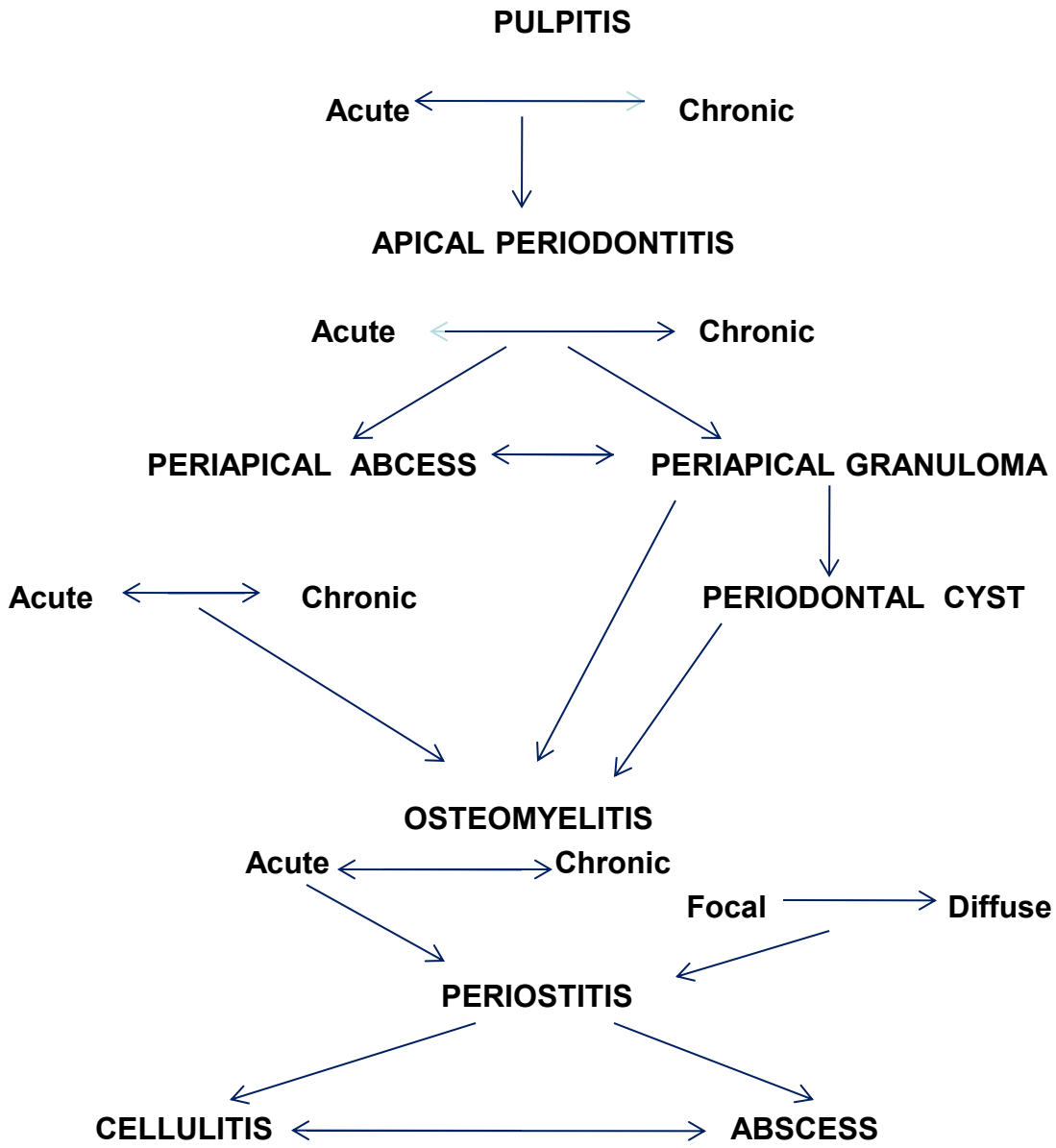
Treatment: Condition is not reversible

Xn or RCT

Treatment

- FRP
  - Identify and eliminate cause.
- Acute Pulpitis
  - Endodontic therapy or Extraction.
- Chronic Pulpitis
  - Removal of stimuli may save vitality.
  - Endodontic or extraction.
- Chronic Hyperplastic Pulpitis.
  - Root canal therapy or Extraction.

Diseases of Periapical lesion



Apical Periodontitis

Dental Pulp

Root canal

Periapical region

Apical Periodontitis

- Inflammation of Pd. Lig. Around root apex.
- Resorption of periapical bone.
- Sometimes root apex.
- Acute or Chronic
    - Virulence of microbes.
    - Type and severity of physical or chemical irritants.
    - Host resistance.

Apical Periodontitis

- Etiology
  - Pulp necrosis
  - Occlusal trauma -High restoration.
  - Biting suddenly on hard object.
  - Endodontic procedure – Over instrumentation.

    Pushing infected material into apical portion.

    Chemical irritation.

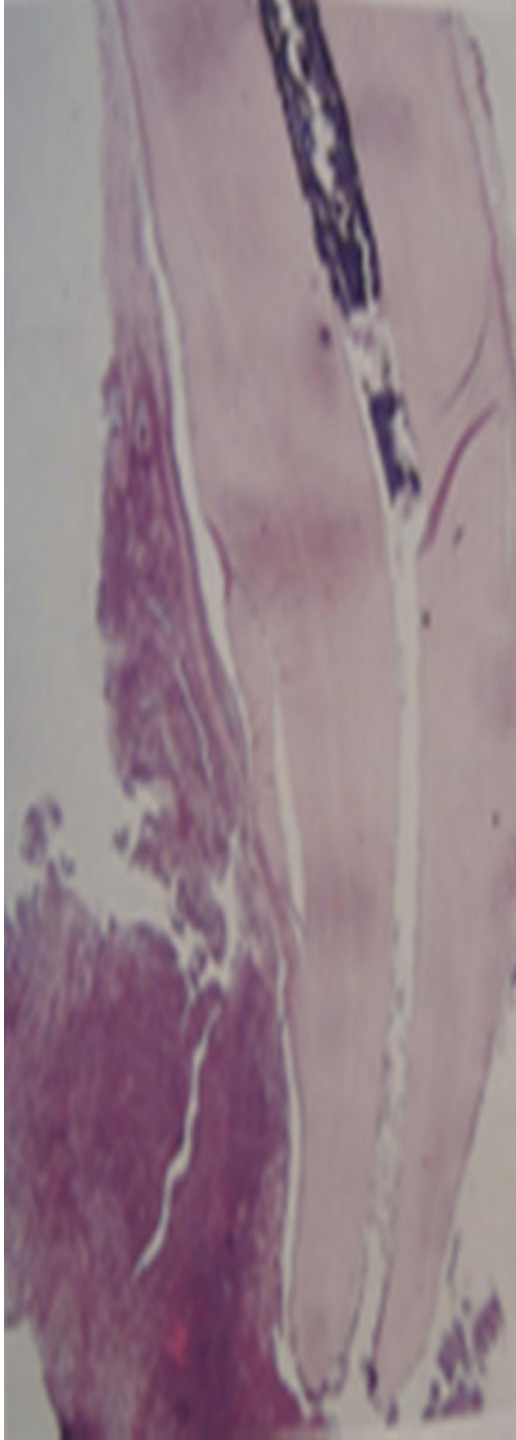
Acute Apical Periodontitis

Acute inflam. process is exudative response

- Clinical features
  - Previous H/O Pulpitis
  - No response to thermal stimuli.
  - Tenderness while biting or even touch.
  - Feeling of elevation in socket – Inflam. Edema
- Radiology
  - Slight widening of Pd. Lig. space

Acute Apical Periodontitis

- Microscopy
  - Inflammation characterized by Vascular dilatation and infiltration of Leukocytes.
  - Initially, changes are localized around root apex.
  - Resorption of surrounding bone, if irritant not removed.
- Treatment
  - Selective occlusal grinding if, occlusal trauma.
  - RCT or Xn if, Pulp Infected.



Chronic Apical Periodontitis

- Synonym – Periapical Granuloma

Chronic process is a proliferative response.

It is essentially a localized mass of granulation tissue formed in response to the infection.

- Etiology
  - low grade infection
  - Sequelae of Pulpitis or Acute Apical Periodontitis.
  - Untreated acute process
- LATERAL GRANULOMA.



Chronic Apical Periodontitis

- Clinical features
  - Nonvital.
  - Slightly tender to percussion.
  - Dull sound on percussion.
  - Pts. Complain of mild pain on biting or chewing.
  - Feeling of elongation on the tooth.
  - Tooth sensitive – edema and inflammation of Apical Pd. Lig.
  - Usually Asymptomatic.

Chronic Apical Periodontitis

- Radiologic Features
  - Thickening of Pd. Lig. At the root apex.
  - Later, bone resorption.
  - Radiolucent area attached to root apex.
  - Radiolucency is well-circumscribed, definitely demarcated from surrounding bone.
  - Thin radiopaque line representing zone of sclerotic bone is seen. – long standing and slowly progressive,
  - Diffuse blending of radiolucent area suggest acute phase of the lesion.
  - Root resorption



Chronic Apical Periodontitis

- Microscopy
  - Inflammatory cell infiltrate
  - Locally increased vascularity
  - Bone resorption adjacent to this area.
  - As bone resorbs, proliferation of fibroblast and endothelial cells are seen.
  - New capillaries lined by swollen endothelial cells.
  - Cells predominantly seen are macrophages, lymphocytes, and plasma cells



Chronic Apical Periodontitis

- Immune granuloma – lymphocytes and plasma cells.
- Non-immune granuloma – macrophages and giant cells and rare plasma cell.
- Bone resorption- T-lymphocytes – OAF (osteoclast activating factor)
- Foam cells – Collection of phagocytes in sheets – ingest lipid material

Chronic Apical Periodontitis

- Cholesterol crystals & hemosiderin – breakdown product of RBC
- Crystals – needlelike clefts are spaces.
- Crystals are associated with multinucleated giant cells.
- Bundles of collagen fibres are seen peripherally separating the granulation tissue from bone.

Chronic Apical Periodontitis

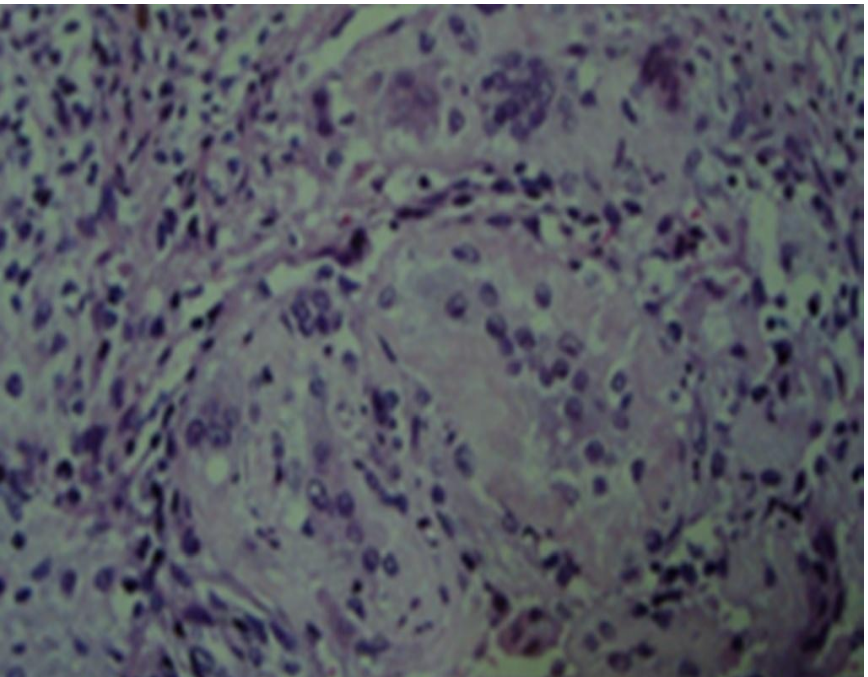
- Presence of epithelium – stratified Sq. epithelium as sheets are anastamosing chords.
- Giant-cell hyaline angiopathy

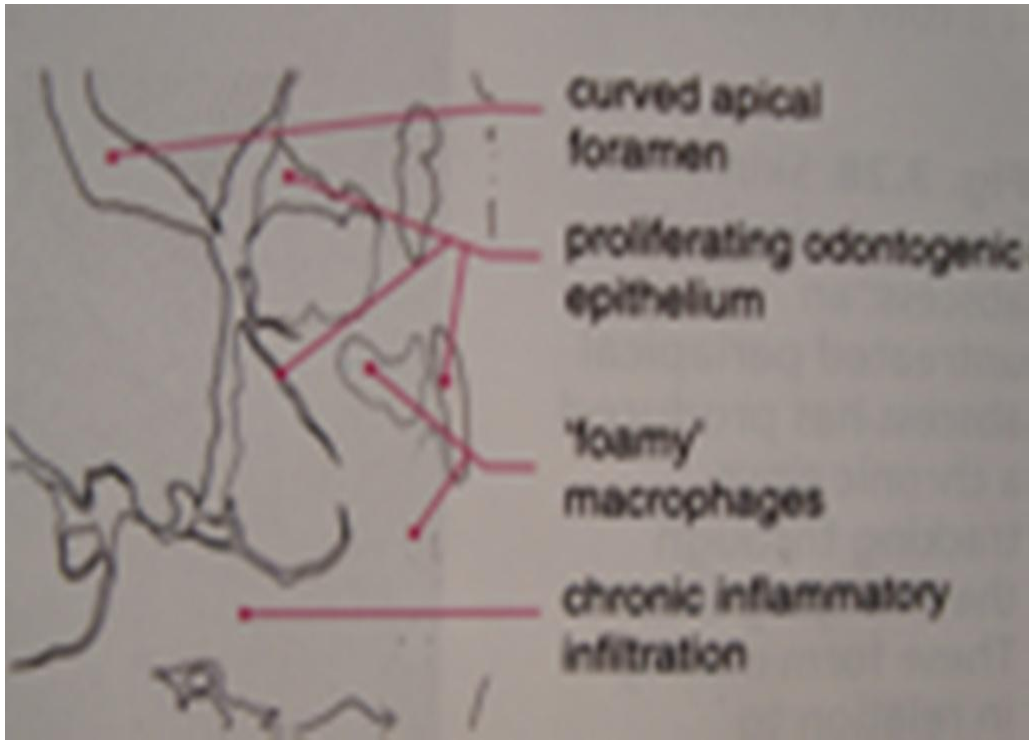
  inflammatory cell infiltrate, collection of foreign body type giant cells, Rushton bodies.

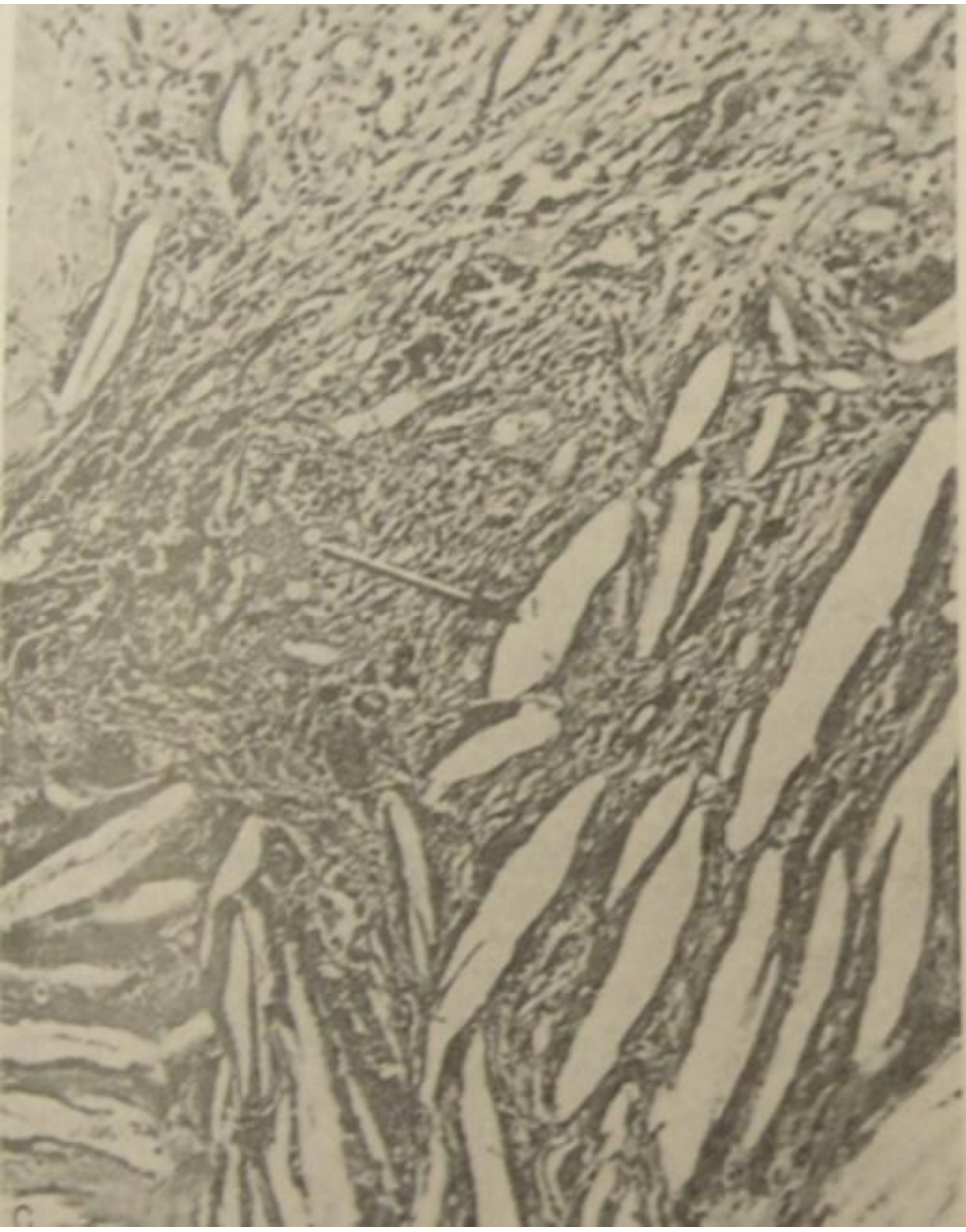
Rushton bodies – ring like structures with eosinophillic material resembling hyalinized collagen.

Chronic Apical Periodontitis

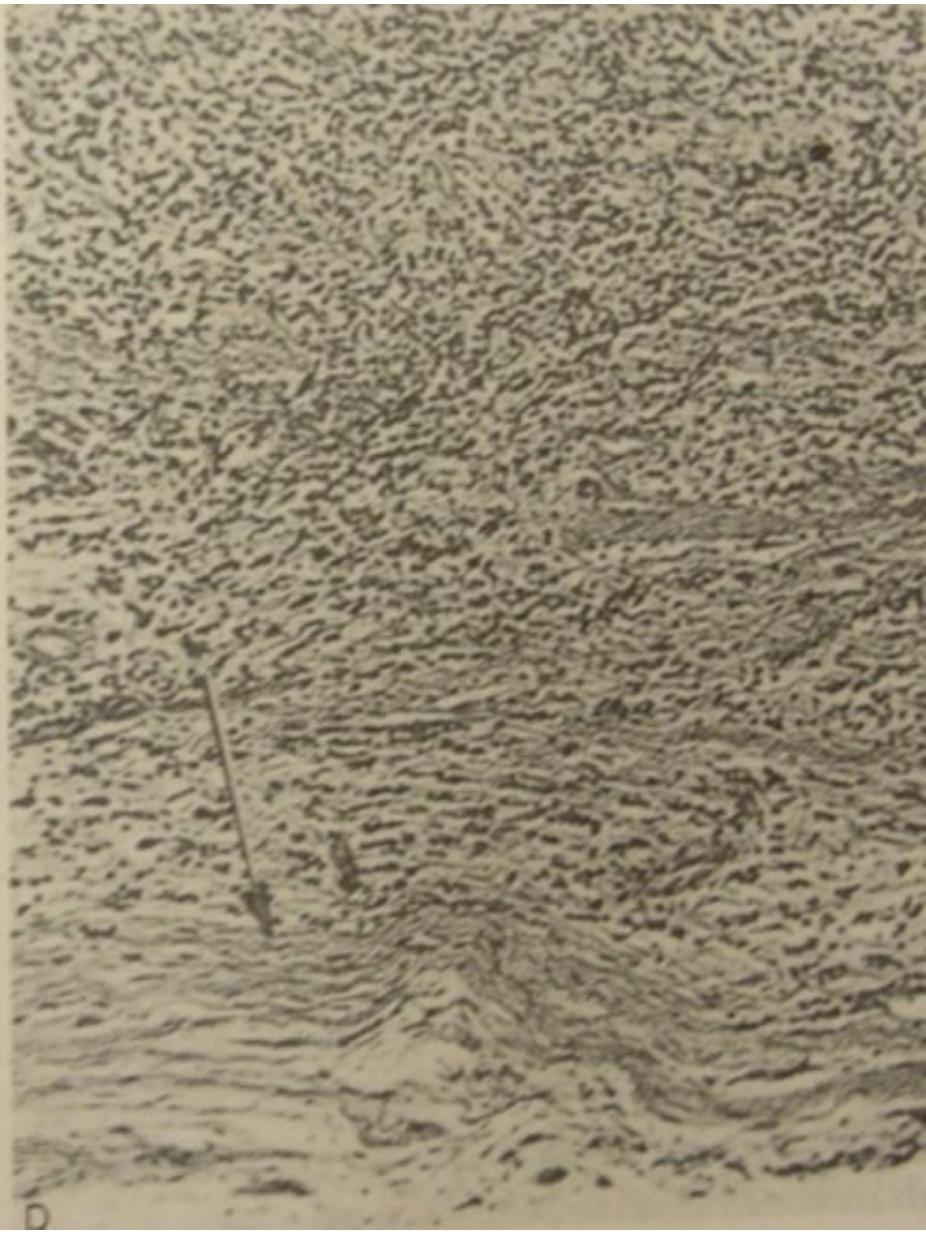
- Pulse granuloma – fragments of foreign material resembling vegetable mater like *legumes.*



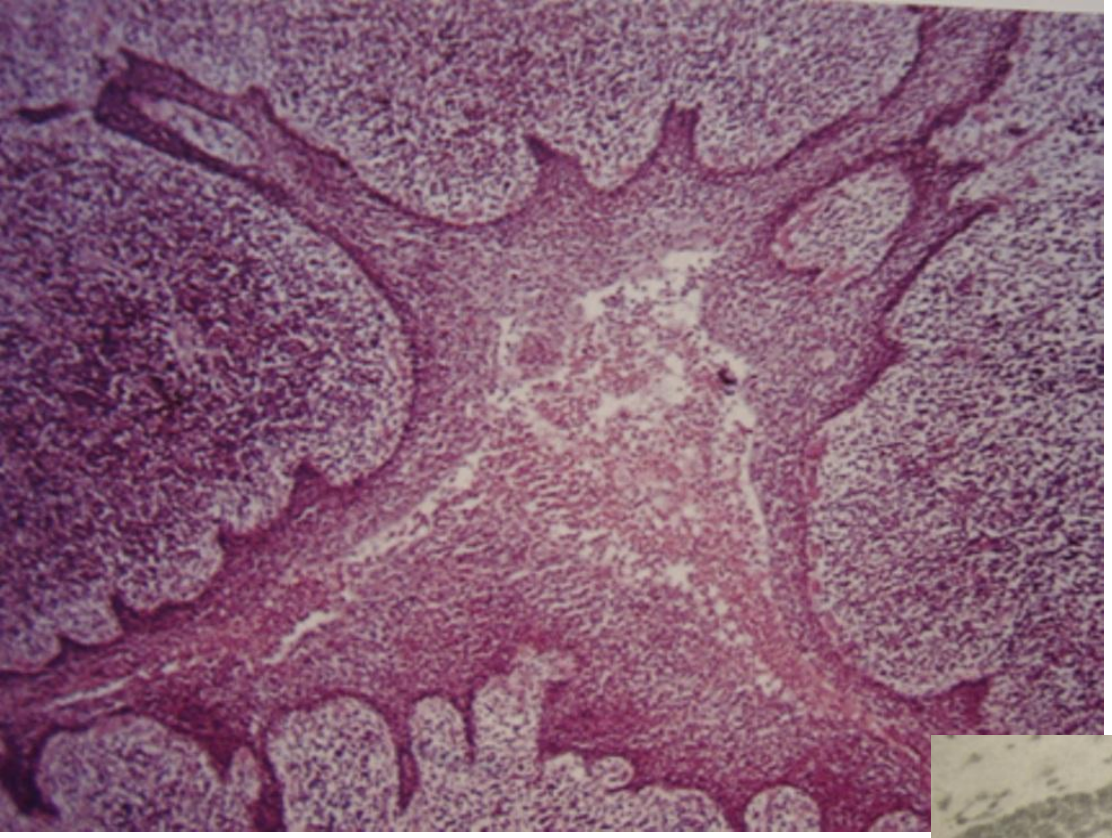


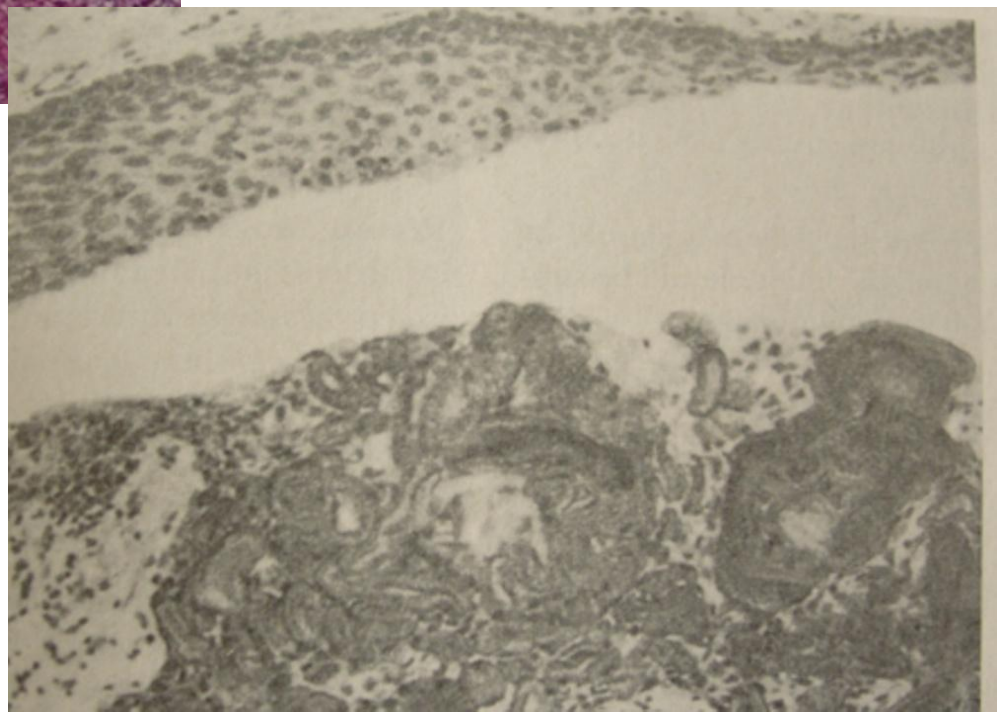


C



D





Chronic Apical Periodontitis

- Microbiology

  cultures – Strep. viridans, Strep. Hemolyticus, Staph. Aureus, Staph. Albus, E.coli. etc.

  HCMV and E-BV are found.

- Treatment

  Xn or RCT with or without apicocectomy

Untreated Granuloma develops into Cyst.

Apical Periodontal Cyst

- Synonym

  Radicular cyst, Periapical cyst, Root end Cyst

Commonest odontogenic cyst

True cyst – Lesion consists of a pathologic cavity that is lined by epithelium and is often fluid-filled.

Apical Periodontal Cyst

- Etiology

  Sequelae of Periapical granuloma

  DC → Necrosis of Pulp → Apical Cyst ← Periapical granuloma

Source of epithelium – cell rests of Malassez, chiefly.

Respiratory epithelium

Oral epithelium from fistula tract

Oral epithelium proliferating apically from Pd. pocket.

Apical Periodontal Cyst

Clinical Features

Majority cases are asymptomatic.

Seen b/w ages of 20 and 60 yrs.

Uncommon in deciduous dentition.

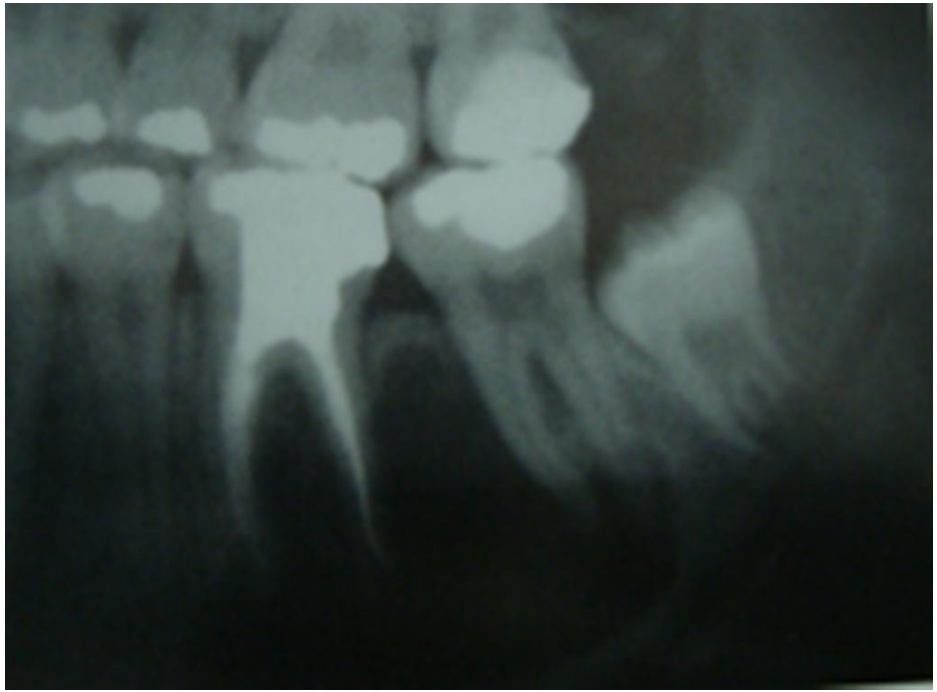
Most commonly involved tooth Max. Ant.

Rarely expands cortical plates, Destruction of bone seen.

Long standing Carious or non-vital tooth.

Apical Periodontal Cyst

- Radiographic Features
  - identical to Periapical granuloma.
  - Size > Periapical granuloma.
  - Thin radiopaque line around the periphery of radiolucent area – reactive lesion.



Apical Periodontal Cyst

Microscopy

Three components Epi., C.T., Lumen

- Epithelium lined lumen
  - St. Sq. epithelium usually.
  - Occasionally Pseudo St. Sq. epithelium or Respiratory epithelium
  - Keratin formation.
  - Newly formed cyst – Epithelial thickness is uneven and shows hyperplasia.
  - Established cyst – regular and even thickness.

Apical Periodontal Cyst

- Rushton bodies/ hyaline body in the epithelium – tiny linear or arc-shaped, amorphous in nature, eosinophillic and brittle in nature.
- Connective tissue
  - Compressed parallel bundles of collagen fibres – form wall of cyst.
  - Inflammatory infiltrate in C.T. adjacent to epithelium – Lymphocytes and plasma cells.
  - Cholesterol slits associated with multinucleated giant cells

Apical Periodontal Cyst

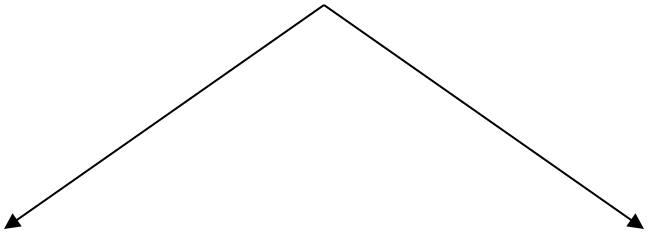
- Lumen
  - Fluid with low conc. of proteins.
  - Cholesterol crystals.
  - Rarely Keratin and blood are found.

- Treatment

  RCT with apicoectomy or curettage

PERIAPICAL ABCESS

- Etiology
  - Results from untreated pulp necrosis.
  - Dependent on virulence of microbes and host defense.
  - Inflammatory process extends into periapical tissues



Chronic

1. Periapical granuloma
2. Periapical cyst

Acute

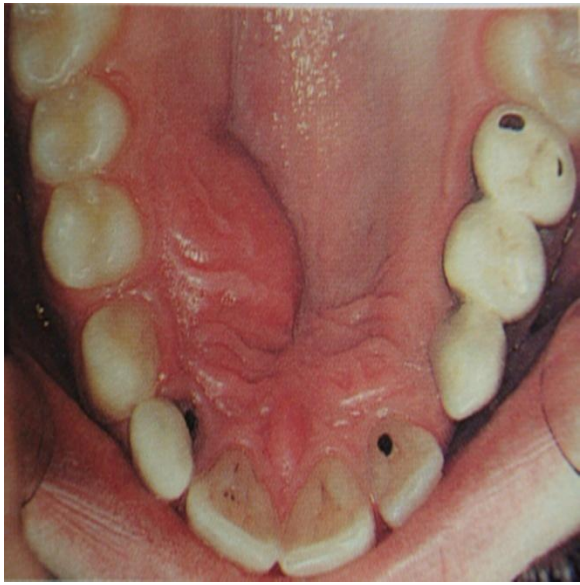
1. Abcess

Necrotic pulpal tissue debris, inflammatory cells, and bacteria(anaerobes) – stimulate and maintain periapical inflammatory process.

PERIAPICAL ABCESS

- Clinical Features.
  - Severe pain – non vital tooth.
  - extrusion of tooth from socket.
  - Pus associated and spreads into adjacent structures.
  - Pain of the associated jaw area.
  - Unresponsive tooth to electric and thermal stimuli.

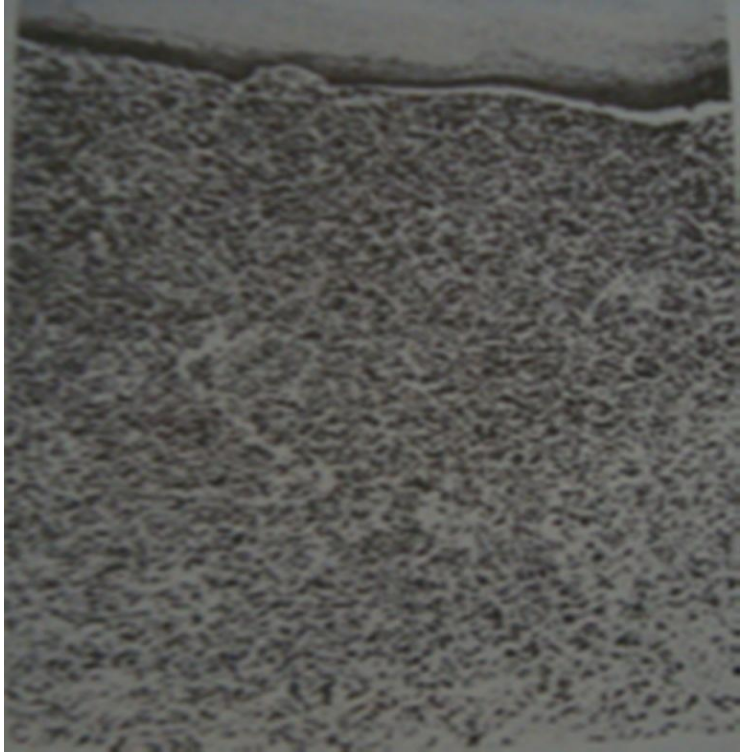




PERIAPICAL ABCESS

- Radiographic feature.
  - radiographic changes are slight.
  - mild thickening of the apical periodontal membrane space.

PERIAPICAL ABCESS



- Microscopy
  - Central area of disintegrating leukocytes,
  - Surrounding viable leukocytes and occasional lymphocytes.
  - Dilatation of blood vessels in Pd. Lig.

Periapical Abscess



- Treatment

Establish Drainage

1.Open pulp chamber and RCT

2. Extraction

Osteomyelitis

- “An inflammation of bone and bone marrow may develop in the jaw as a result of odontogenic infection as well as in other conditions”.

Definition

an inflammatory condition of the bone that begins as an infection of Medullary cavity and the haversian system and extends to involve the periosteum of the affected area.

Classification of Osteomyelitis

- **Anatomic Location**
  - Intramedullary
  - Subperiosteal
  - Periosteal
- **Duration and Severity**
  - Acute
  - Chronic
- **Presence of Absence of Suppuration**

Suppurative

- Acute suppurative osteomyelitis
- Chronic suppurative osteomyelitis
  - Primary
  - secondary
- Infantile osteomyelitis

Classification of Osteomyelitis

- Nonsuppurative
  - Chronic nonsuppurative
    - Focal Sclerosing
    - Diffuse Sclerosing
  - Radiation Osteomyelitis
  - Garre's Osteomyelitis
  - Osteomyelitis due to Specific infection
    - Actinomycosis
    - Tuberculosis
    - Syphilis.

Acute Suppurative Osteomyelitis

- Sequelae of periapical infection resulting in necrosis of bone.

Etiology

Dental infection

# of Jaw

Gunshot wound

Hematogenous spread

Acute Suppurative Osteomyelitis

- It is a well localized lesion.
- Polymicrobial in nature – common organism

  Staphylococcus aureus and staph. albus , various Streptococcus

  Anerobes – Bacteroides, Porphyromonas, Prevotella sp.

Acute Suppurative Osteomyelitis

- Clinical Features

  Acute and Subacute may involve both maxilla and mandible.

  Maxilla localized infection.

  Mandible wide spread and diffuse.

  Severe pain

  Trismus

  Paresthesia of lips

  Elevation of temperature

  Regional lymphadenopathy

Acute Suppurative Osteomyelitis

Mobility of teeth is noted.

Eating food difficult.

Pus may exude from gingival margin.

Fetid odour.

No swelling or reddening of the skin or mucosa.

WBC count is elevated.

Acute Suppurative Osteomyelitis

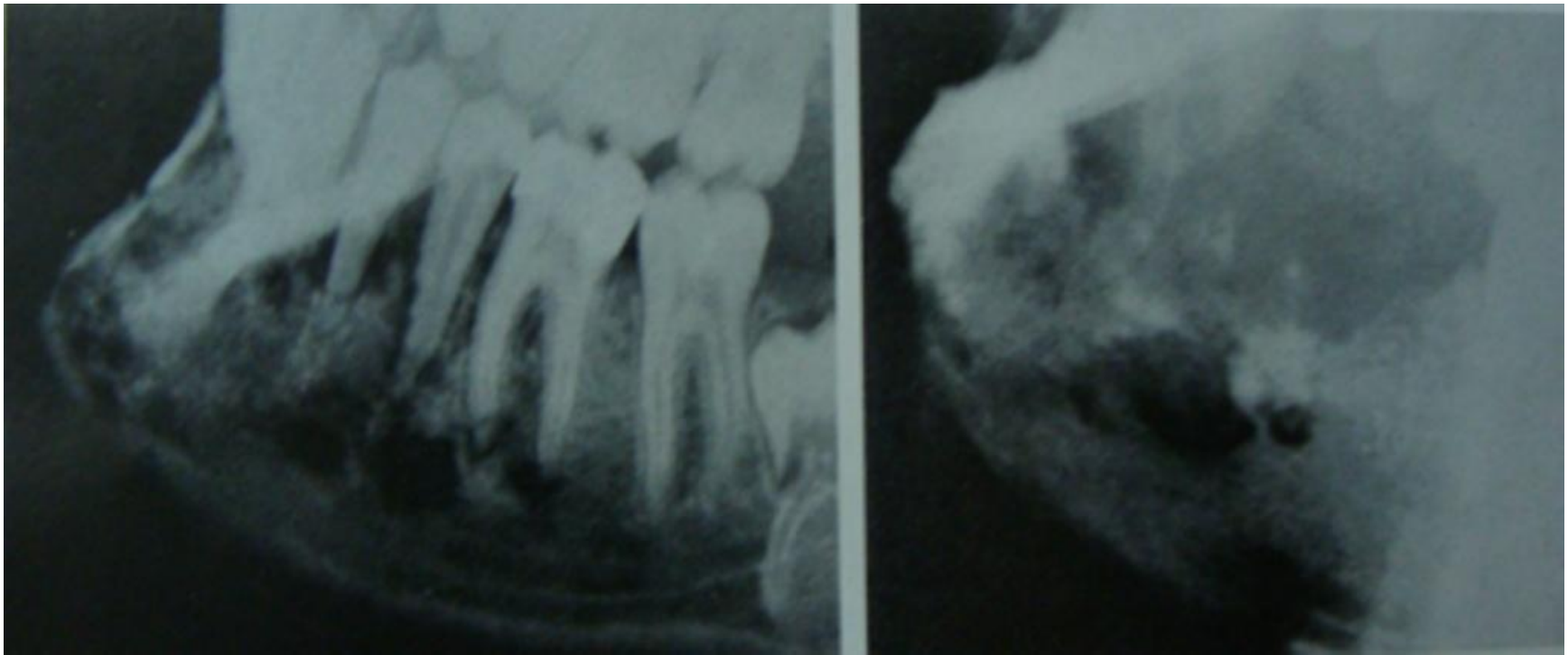
- Radiologic Feature

➢ First two weeks no changes are not seen.

➢ Later, diffuse lytic changes appear.

➢ Bone trabeculae become fuzzy and indistinct.

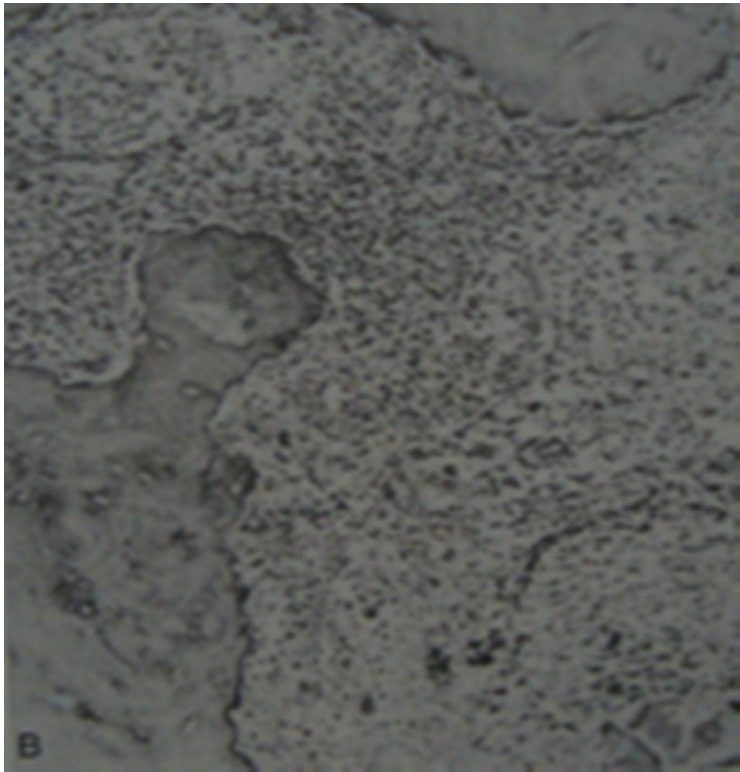
➢ Radiolucent area begin to appear.

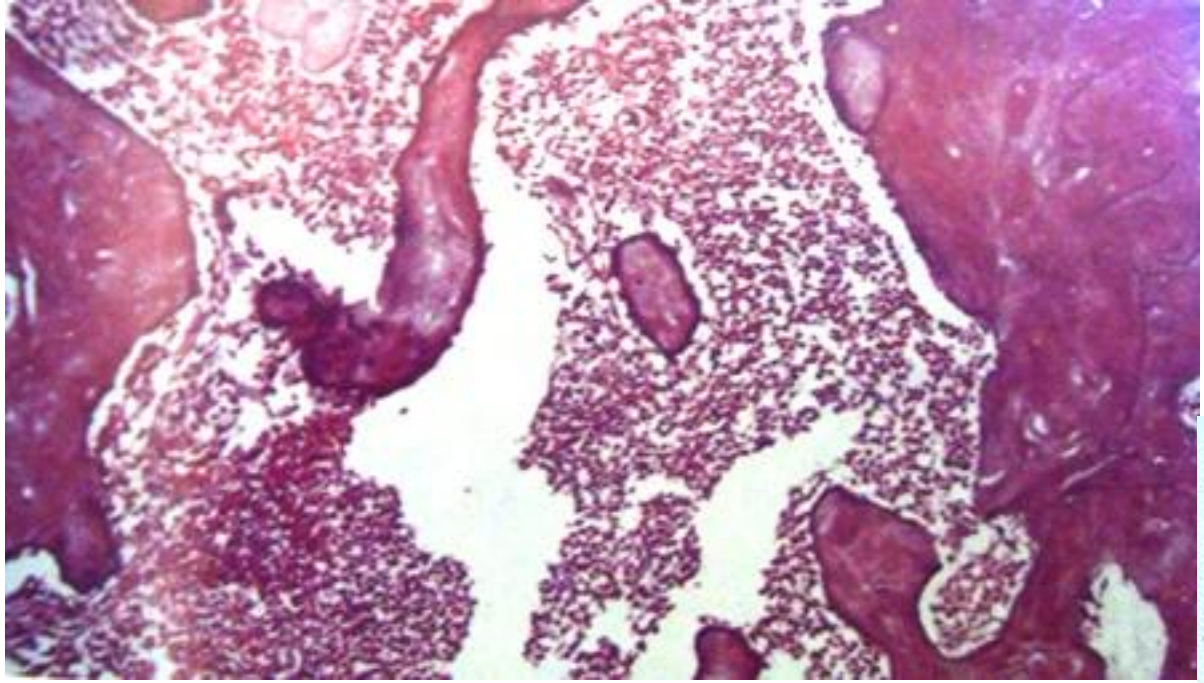


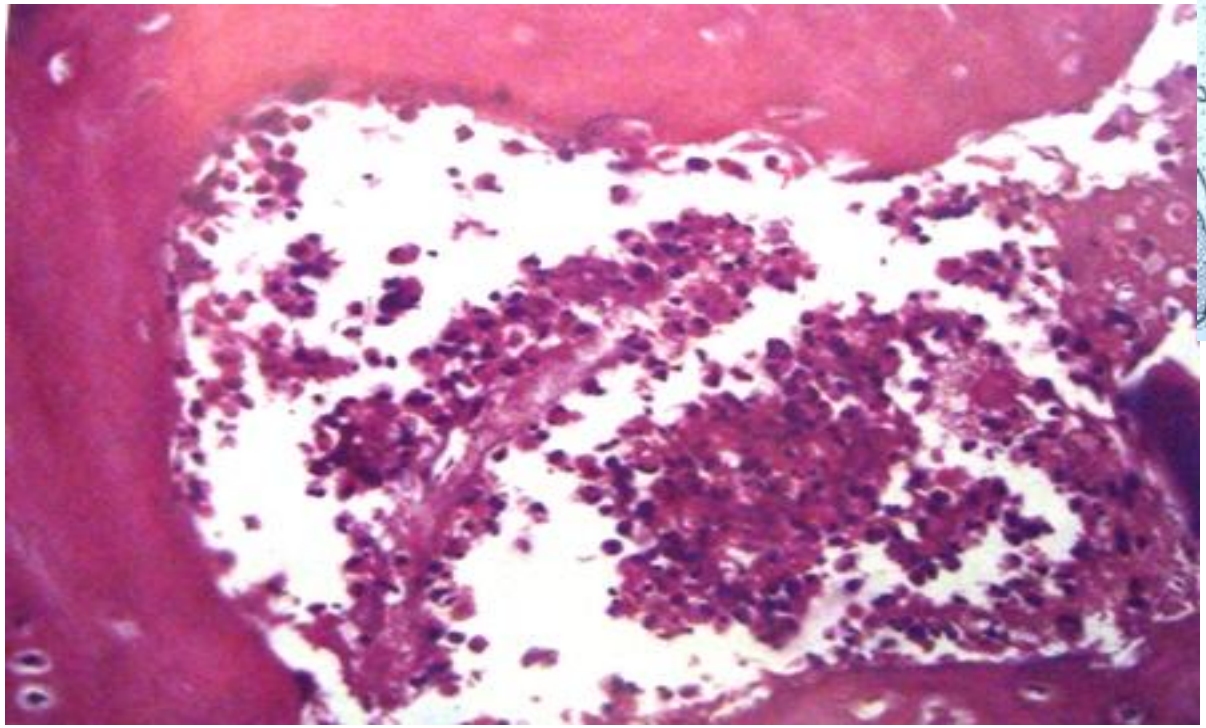
Acute Suppurative Osteomyelitis

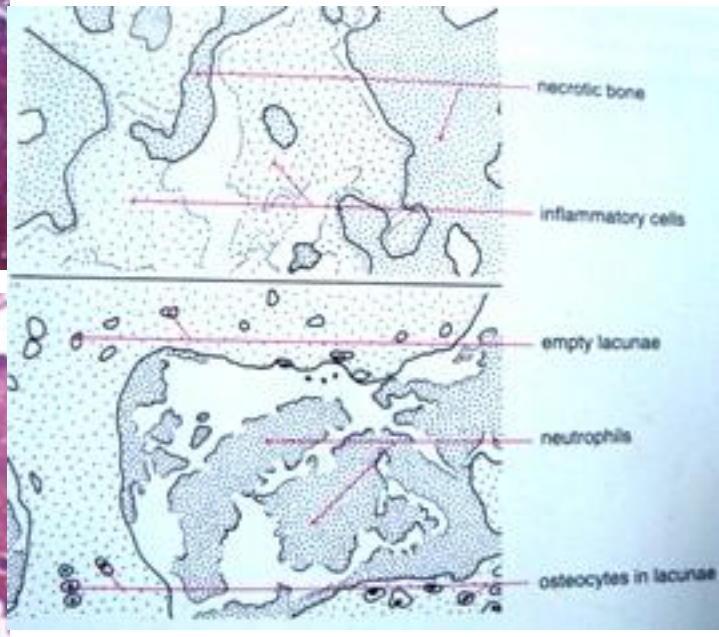
Microscopy

- Medullary spaces filled with inflammatory exudate - Polymorphonuclear leukocytes
- Occasional lymphocytes and plasma cells.
- Osteoblasts bordering bony trabeculae are destroyed.
- Trabeculae undergo slow resorption.








necrotic bone
inflammatory cells
empty lacunae
neutrophils
osteocytes in lacunae

Acute Suppurative Osteomyelitis

- Treatment

General Principles of management

DEBRIDEMENT, DRAINAGE, and ANTIMICROBIAL THERAPY.

Sequestrum , if small – exfoliates.

if large - Surgical removal.

Chronic Suppurative Osteomyelitis

Etiology

- Inadequately treated acute Osteomyelitis.
- Untreated dental infections.
- Complication of irradiation.

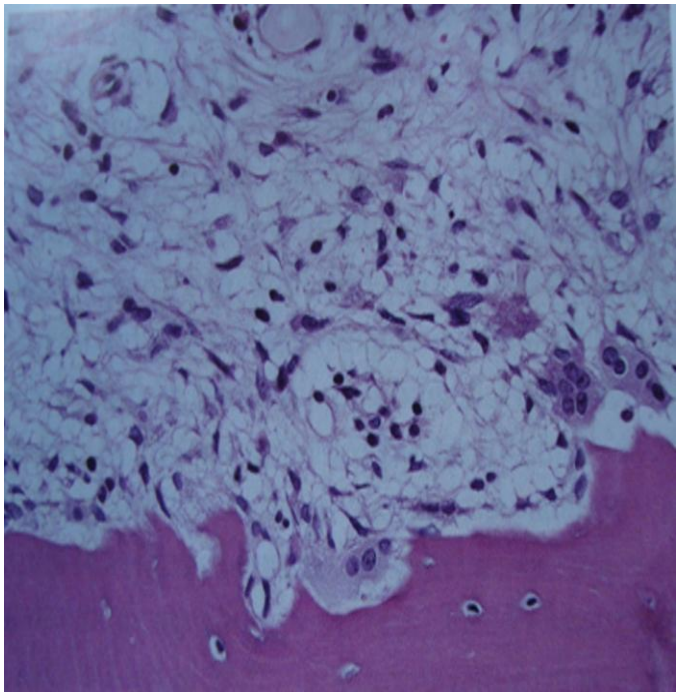
Clinical Features

- Milder signs and symptoms than Acute stage.
- Less severe pain.
- Temperature slightly elevated.
- Perforation of bone and overlying skin or mucosa to form fistula.

Chronic Suppurative Osteomyelitis

Microscopy

- Osteoblastic activity in this area.
- Centrally both Osteoblastic and osteoclastic activity seen.
- Marrow spaces contain fibrous tissue with scattered lymphocytes and plasma cells.



Chronic Focal Sclerosing Osteomyelitis

- Unusual reaction of bone to infection.
- Reaction is proliferation than destruction.
- Infection acts as stimulus.

Clinical Features

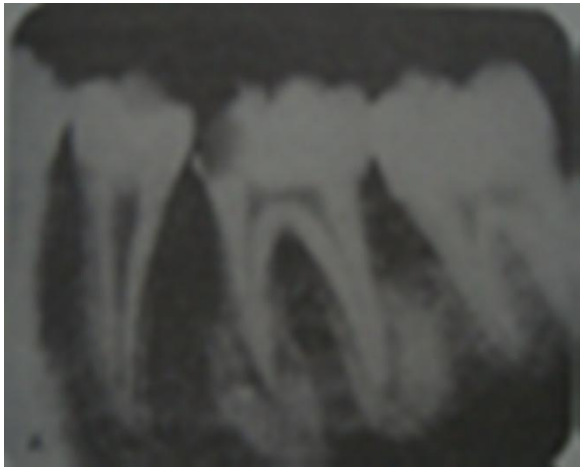
- Common in children and young adults.
- Rare in older individuals.
- Mandibular first molar – commonly involved.
- No signs and symptoms except for mild pain with infected pulp.

Chronic Focal Sclerosing Osteomyelitis

Radiographic Features



- Well-circumscribed radiopaque mass of sclerotic bone surrounding and extending below the apex of one or both roots.

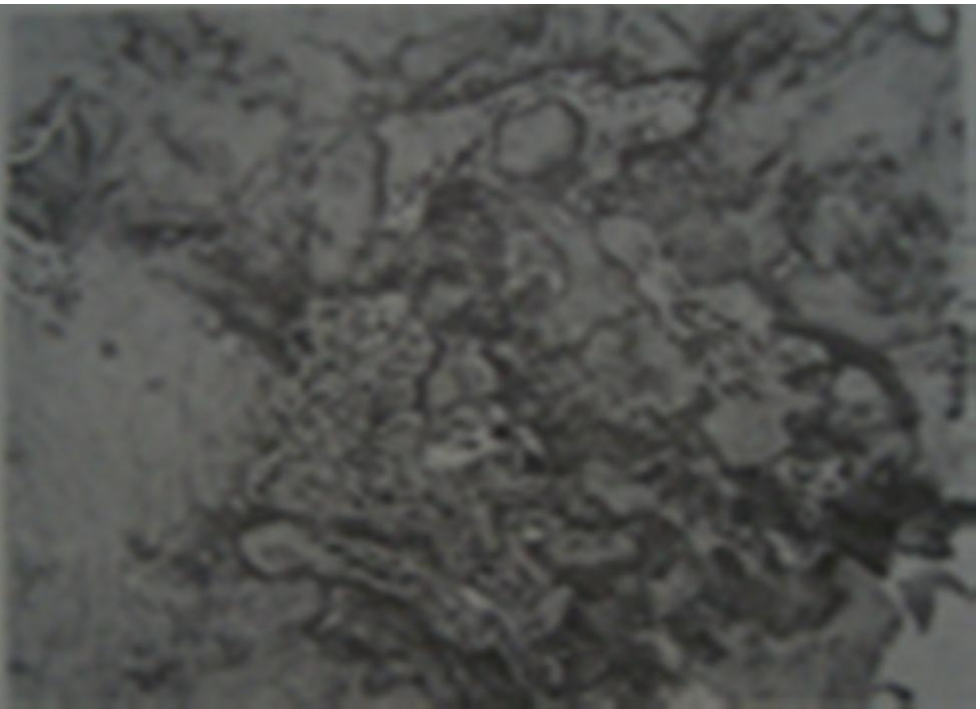


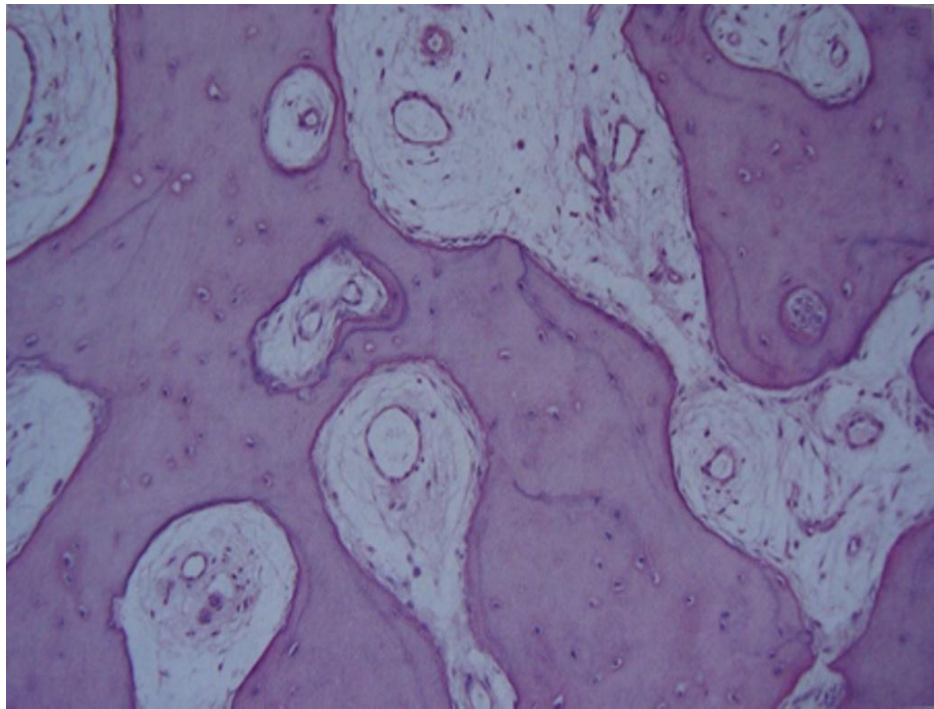
- Root outline is visible , intact Lamina Dura.
- Pd. Lig space widened.
- Border – smooth and distinct or blend into the surrounding bone.

Chronic Focal Sclerosing Osteomyelitis

Microscopy

- Dense mass of bony trabeculae with little marrow tissue.
- Osteocystic lacunae appear empty.
- Trabeculae exhibit Pagetoid appearance
  (reversal and resting lines)
- If marrow tissue is seen – Fibrotic and small no. of lymphocytes.
- Osteoblastic activity almost absent.





Chronic Focal Sclerosing Osteomyelitis

Treatment

- Affected tooth – RCT or Xn.

Chronic Diffuse Sclerosing Osteomyelitis

- Proliferative reaction of the bone to a low grade infection.
- Portal of entry of infection is thro' diffuse periodontal disease.

Chronic Diffuse Sclerosing Osteomyelitis

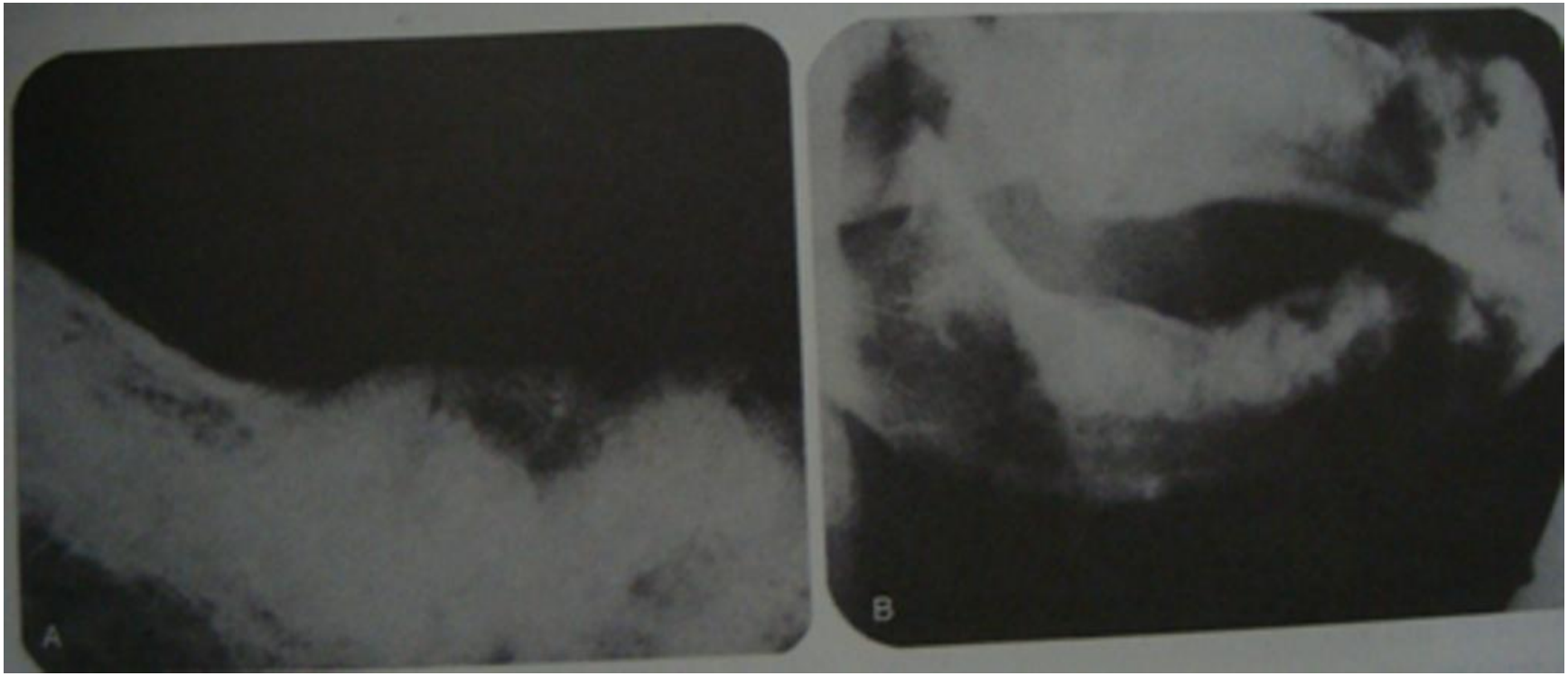
Clinical Features

- May occur at any age
- Most common in older age group esp. edentulous mandibular jaws.
- Acute exacerbation results in mild pain, unpleasant taste and mild suppuration.
- Fistula formation to establish drainage.

Radiologic Features

"Cotton –wool" appearance – diffuse patchy, sclerosis of bone.

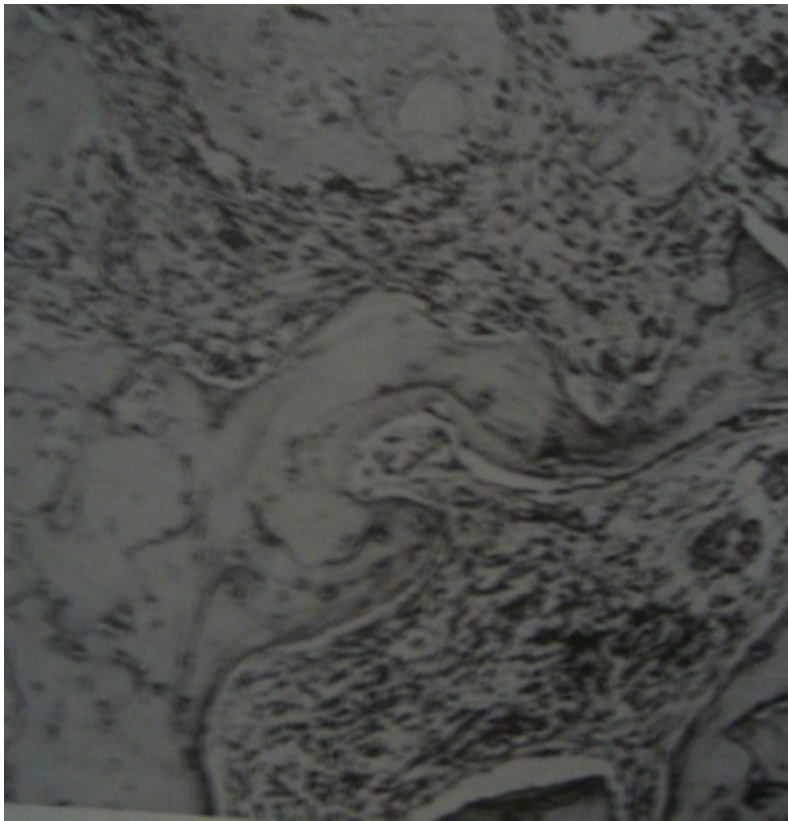
Indistinct border between sclerotic and normal bone



Chronic Diffuse Sclerosing Osteomyelitis

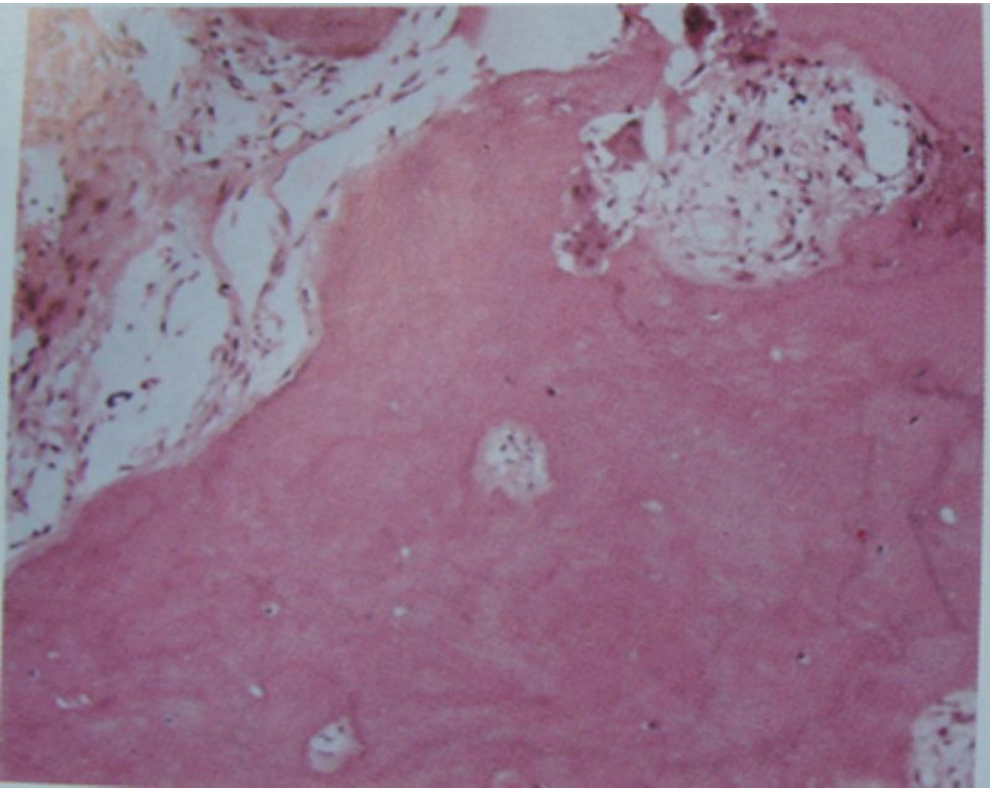
Microscopy

- Dense, irregular trabeculae of bone, some bordered by active layer of osteoblasts.
- Focal areas of osteoclastic activity is seen.
- ‘Mosaic’ pattern of bone - indicating remodelling and repair



Chronic Diffuse Sclerosing Osteomyelitis

- Marrow space is fibrous and show proliferating fibroblasts , small capillaries and lymphocytes and plasma cells.
- Acute phase – leukocytes are seen.



Chronic Diffuse Sclerosing Osteomyelitis

Treatment

- Difficult problem
- Too extensive lesion for surgery.
- Acute episode – antibiotics
- Involved tooth is extracted, if present in sclerotic area.


Everyday
do something
that will
inch you
close to a
better
tomorrow