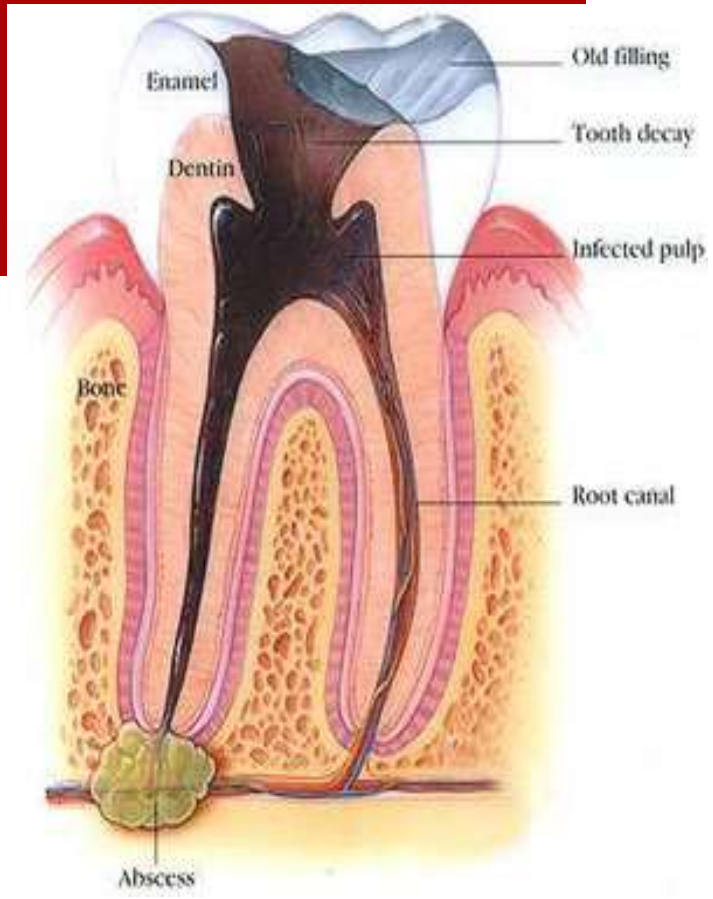
PULP AND PERIAPICAL PATHOLOGIES-1



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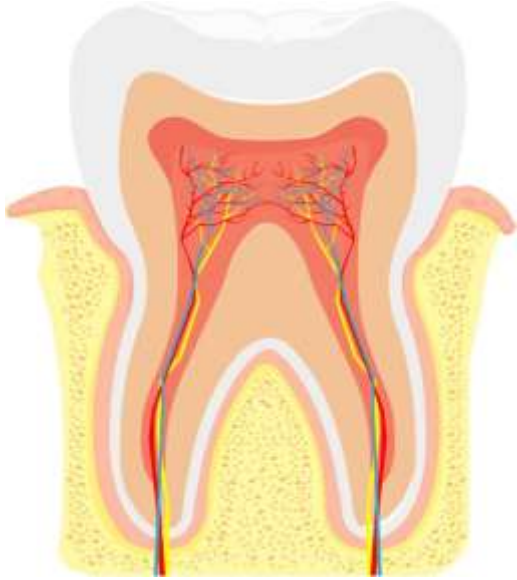
INTRODUCTION

- The dental **pulp** is the part in the center of a tooth made up of living connective tissue and cells called odontoblasts.
- The pulp contains the blood vessels the nerves and connective tissue inside a tooth and provides the tooth's blood and nutrients.
- **Pulpitis** is inflammation of dental pulp tissue



CAUSE OF PULP DISEASE

According to Grossman,

1) PHYSICAL

A) Mechanical

1) Trauma- a) Accidental

b) Iatrogenic dental procedures

2) Pathologic wear

3) Crack through the body of the tooth

4) Barometric changes

B) Thermal

1. Heat during cavity preparation
2. Exothermic heat during setting of cement
3. Conduction of heat and cold through deep restoration with out a protective base
4. Frictional heat during the polishing of restoration

c) Electrical

- Galvanic shock

II) CHEMICAL

A) Phosphoric acid, acrylic monomer

B) Erosion

III) BACTERIAL

A) Toxins associated with caries

B) Direct invasion of pulp from caries or trauma

C) Anachoresis

CLASSIFICATION OF PULP DISEASE

According to Grossman.

- Based on clinical features.

1) Pulpitides (Inflammation)

A) Reversal

1) Symptomatic (acute)
2) Asymptomatic (chronic)

B) Irreversible

1) Acute

a) Abnormally responsive to cold
b) Abnormally responsive to heat

2) Chronic

a) Asymptomatic with pulp exposure
b) Hyperplastic pulpitis
c) Internal resorption

11) Pulp Degeneration

a) Calcific
b) Others

111) Necrosis

FACTORS AFFECTING RESPONSE OF PULP

- Severity and duration of irritant.
- Nature of irritant.
- Health condition of the pulp or pre-existing state of the pulp
- Apical blood flow
- Local anatomy of the pulp chamber
- Host defence

PATHWAYS OF BACTERIAL INVASION OF THE PULP

Most common cause of pulp injury- irreparable

- Opening in dental hard tissue wall
  - caries
  - clinical procedures
  - trauma induced fractures
  - microcracks
- Bacteria from the gingival sulcus/ pocket
- Endodontic reinfection
- Extension of a periapical infection from adjacent infected teeth.

ANACHORESIS

Transportation of microbes through the blood or lymph to an area of inflammation such as tooth with pulpitis

AERODONTALGIA

- Toothache occuring at low atmospheric pressure experienced either during flight or during a test run in a decompression chamber
- Observed in higher altitudes over 5000 feet
- Tooth with chronic pulpitis can be symptomless at ground level, but it may cause pain at high altitudes because of reduced pressure

Treatment:

Lining the cavity with a varnish or a base of zinc phosphate cement with a subbase of ZOE cement in deep cavities

Reversible Pulpitis (Pulp Hyperemia)

Mild to moderate inflammatory condition of the pulp caused by noxious stimuli in which the pulp is capable of returning to the uninflammed state following removal of the stimuli.

Etiology:

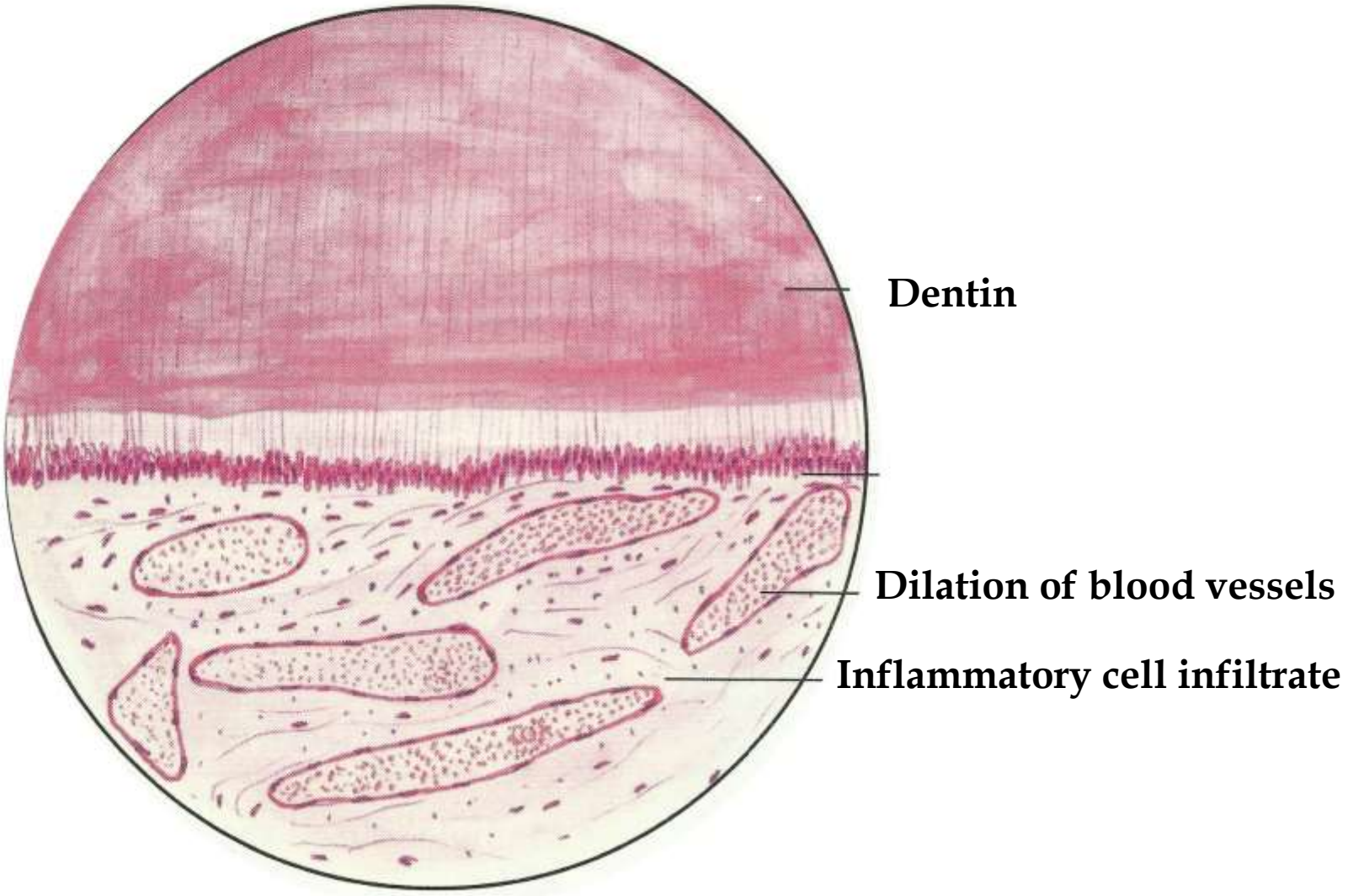
- Trauma
- Thermal shock
- Excessive dehydration of the cavity
- Galvanism
- Bacteria from caries

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.

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.


Dentin
Dilation of blood vessels
Inflammatory cell infiltrate

DIAGNOSIS:

- Based on symptoms
- Clinical test- cold test

TREATMENT:

- Prevention of dental caries
- Early insertion of filling
- Desensitization of neck of teeth where gingival recession occurs
- Cavity varnish or base application before insertion of filling
- Care in cavity preparation and polishing
- If primary cause is not corrected, extensive pulpitis may result in death of pulp.

Acute Pulpitis

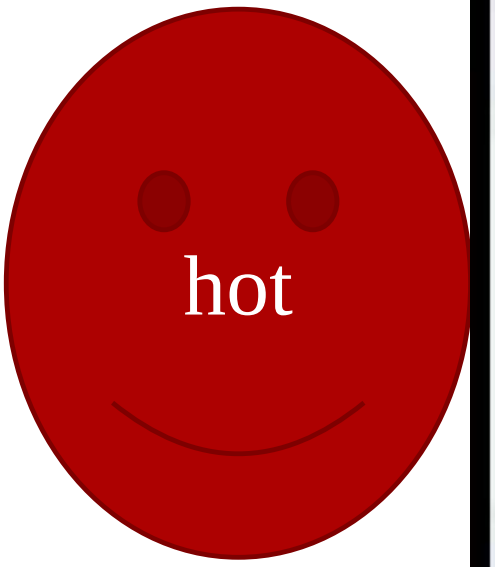
Extensive acute inflammation of the dental pulp is a frequent immediate sequela of focal reversible pulpitis, although it may occur as an acute exacerbation of a chronic inflammatory process

Etiology:

- Bacterial invasion through the dental caries- most common
- Chemical, thermal or mechanical causes
- Reversible pulpitis may deteriorate into irreversible pulpitis

CLINICAL FEATURES:

- Early stages- involves only a portion of the pulp, usually the area just beneath the carious lesions.
- Teeth extremely sensitive to Hot or cold stimuli and cause increase in pain intensity & persists even after the thermal stimulus has been removed.
- 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.



Thermal stimulation

- 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.

Rise in pulp pressure along with inflammatory exudate

↓

Local collapse of the venous part of the circulation

↓

Local tissue hypoxia and anoxia

↓

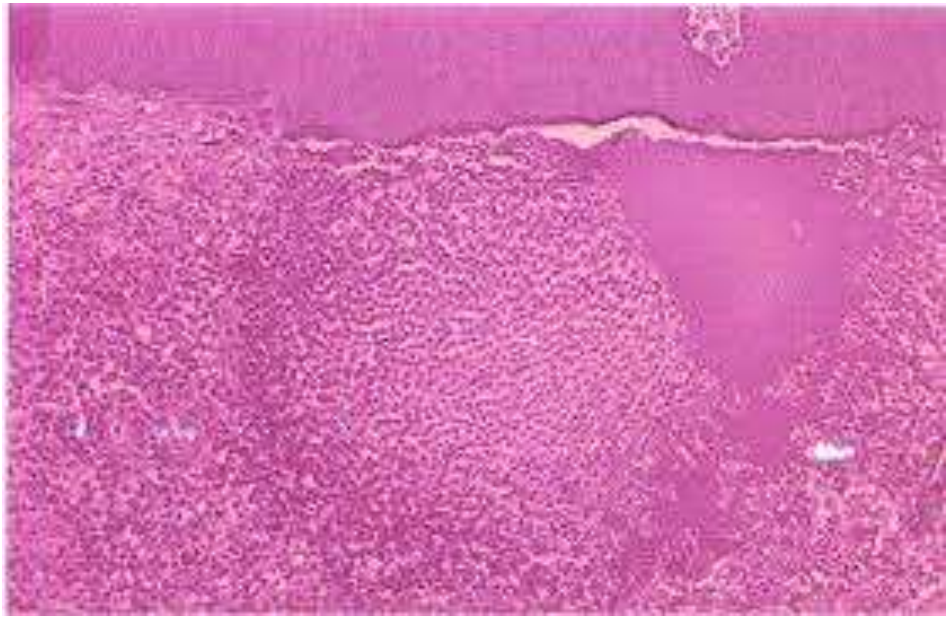
Pulp abscess

Ohnishi T – reported presence of Hepatocyte Growth Factor in acute inflammation of pulp

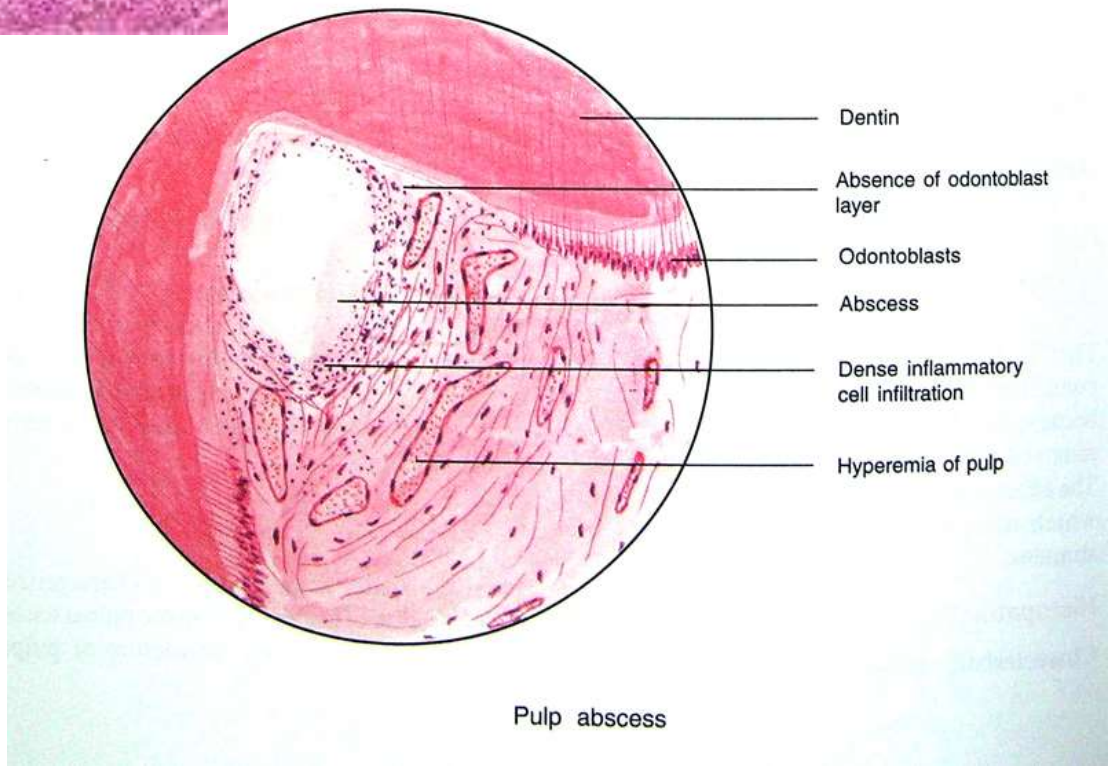
Guo X et al- IL-8 level is higher in acute than in chronic pulpitis

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.
- Abscess consists pus, leukocytes & bacteria.
- Acute suppurative pulpitis- Numerous abscess formation cause pulp liquefaction & necrosis.



Dental pulp exhibiting acute inflammatory infiltrate consisting predominantly of polymorphonuclear leukocytes.



DIAGNOSIS

1) Inspection:

- Discloses a deep cavity
- Pulp exposure

2) Radiography:

- Exposure of the pulp
- Caries under a filling

3) Thermal test

TREATMENT:

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

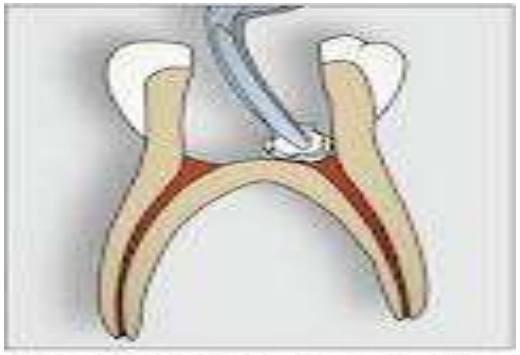
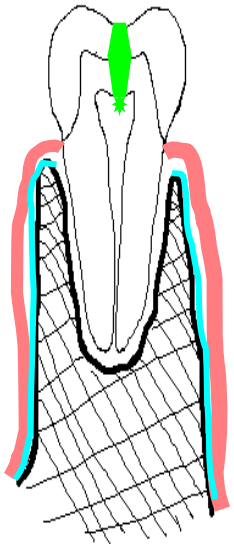


Fig. 7. An illustration of hemorrhage control using a water-dampened cotton pellet.

Chronic Pulpitis



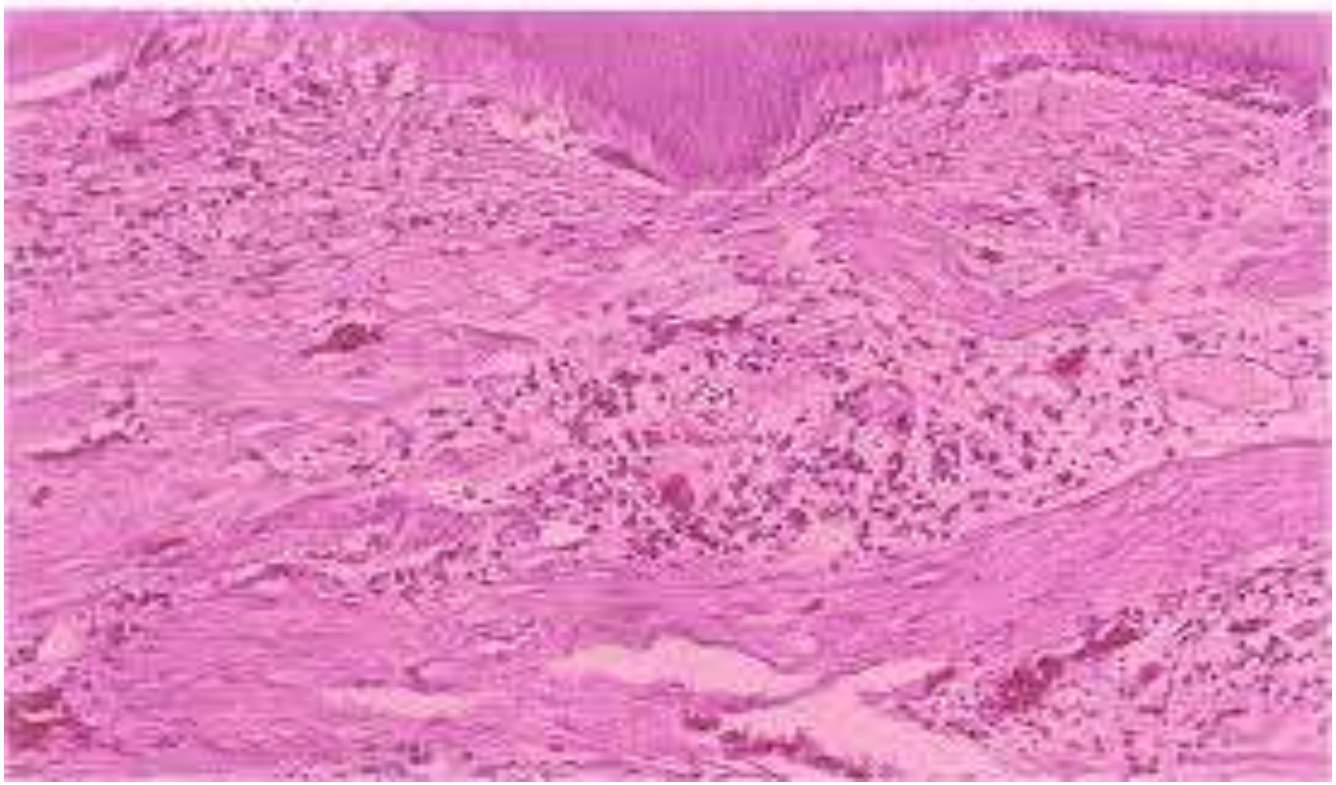
- Persistent inflammatory reaction in pulp with little or non constitutional symptoms.

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

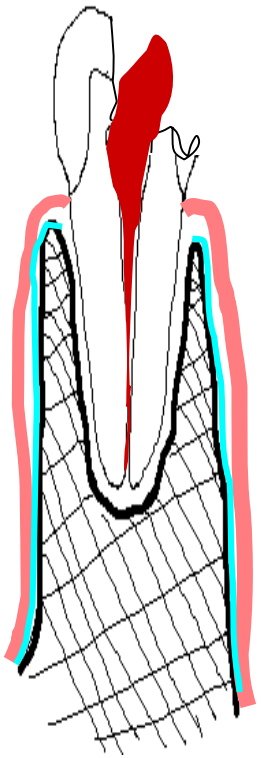


The dental pulp exhibits an area of fibrosis and chronic inflammation peripheral to the *zone* of abscess formation.

TREATMENT :

- Root canal therapy
- Extraction of tooth.

Chronic Hyperplastic Pulpitis (pulp polyp)



- Overgrowth of pulp tissue outside the boundary of pulp chamber as protruding mass.
- Characterized by the development of granulation tissue, covered at times with epithelium and resulting from long standing low grade irritation

ETIOLOGY:

- Slow progressive carious exposure of the pulp
- A large open cavity, a young resistant pulp and a chronic low grade stimulus are necessary

CLINICAL FEATURES:

- Children & young adults with high degree of tissue resistance & reactivity & responds to proliferative lesions.
- Teeth with large , open carious 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

- 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 & PMNLs.
- Fibroblast and endothelial cell proliferation prominent.
- Stratified squamous type epithelial lining resembles oral mucosa with well formed rete pegs.
- Grafted epithelial cells are believed to be desquamated epithelial Cells, which carried by saliva.
- Origin of these cells is unknown. They are degenerated superficial squames, which have lost dividing capacity.

DIAGNOSIS

Clinical Examination:

- Seen in children and young adults
- A freshly, reddish pulpal mass fills most of the pulp chamber or cavity or even extends beyond the confices of the tooth.

Radiography:

- Large open cavity with direct access to the pulp chamber

TREATMENT:

- Extraction of tooth
- 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 seen
- Dry gangrene- pulp dies for unexplained reasons.
- This may be due to trauma or infarct.

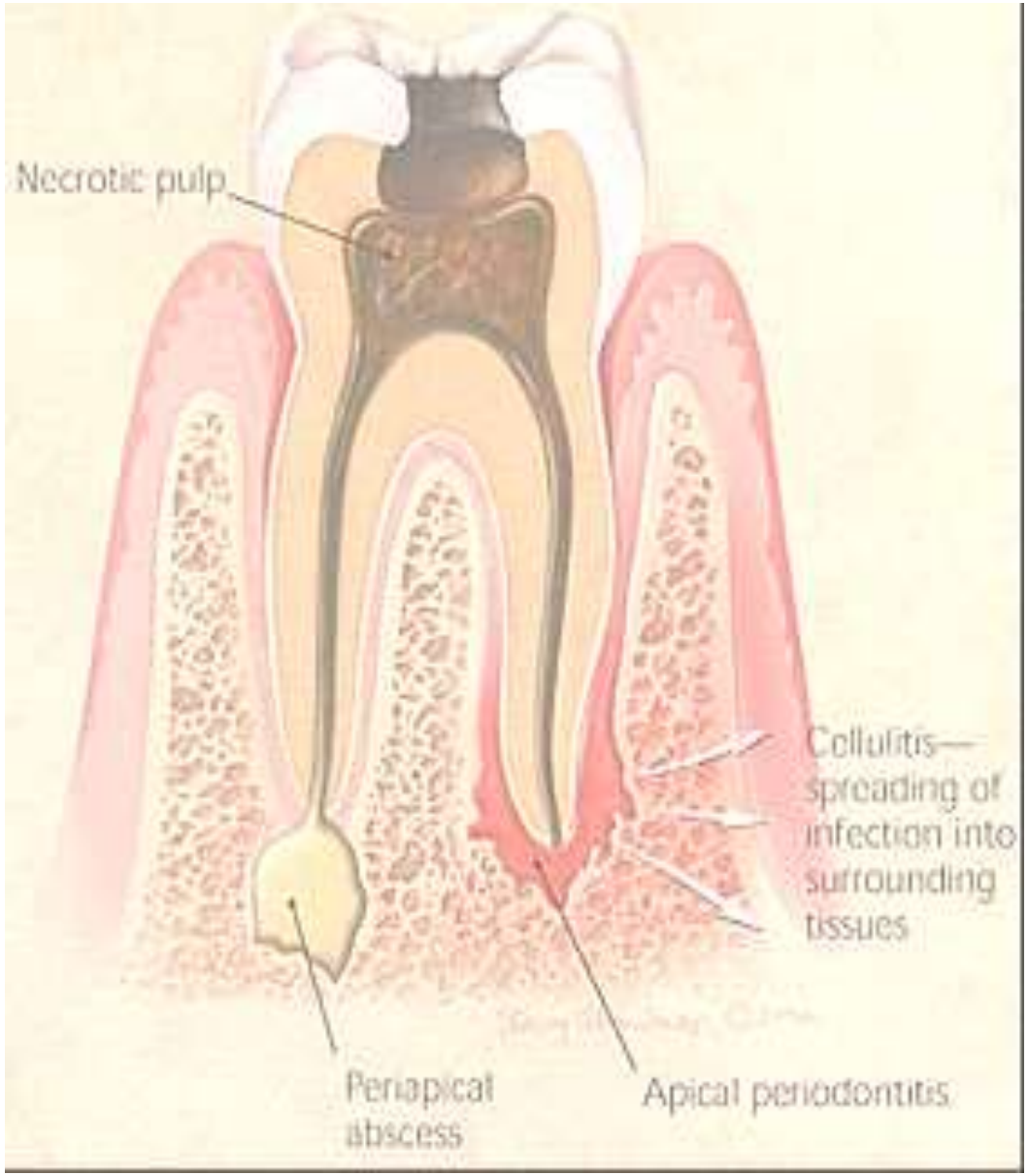
REVERSIBLE PULPITIS

- 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.

IRREVERSIBLE PULPITIS

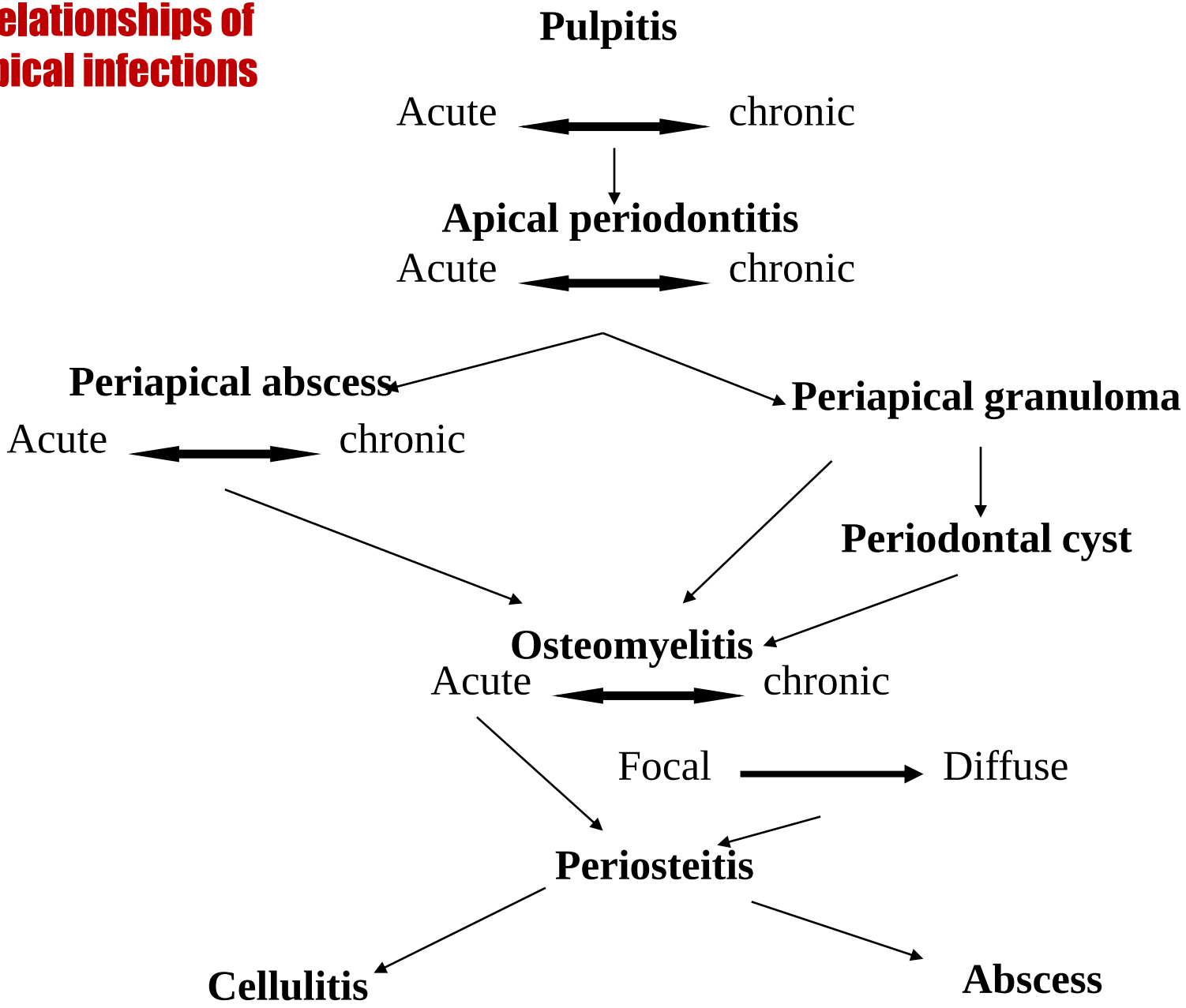
- Sharp, severe, radiating pain of long duration & varying intensity.
- Pain continues even after the stimulus is removed.
- Pain may exacerbate with bending over or lying down..
- Increased by stimulus, like heat & at times relieved by cold although the cold may intensify the pain.
- When infection extends into PDL - apical periodontitis.

Diseases Of Periapical Tissues



- Once infection has established in the dental pulp, spread of the process can be in one direction- through the root canals and into the periapical region.
- Number of different tissue reaction may occur, depending upon a variety of circumstances.
- Subtle transformation from one type of lesion into another type in most cases.

Interrelationships of periapical infections



Apical Periodontitis

- Inflammation of PDL around apical portion of root.
- Types: 1.Acute Apical Periodontitis

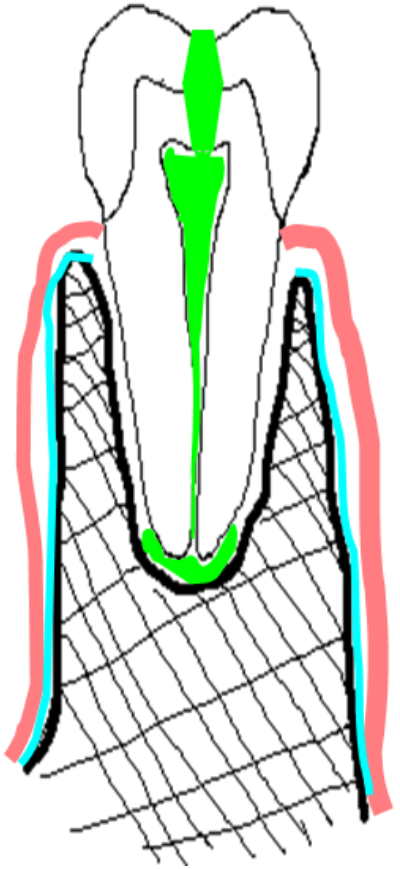
  2.Chronic Apical Periodontitis

Etiology:

- spread of infection following pulp necrosis,
- occlusal trauma,
- Bitting suddenly on high objects
- Inadvertent endodontic procedures
- Pushing the infected material into apical portion
- Chemical irritation from root canal medicaments

Acute Apical Periodontitis

- Painful inflammation of the peridontium as a result of trauma, irritation, or infection through the root canal, regardless of whether the pulp is vital or nonvital.
- Also referred to as symptomatic apical periodontitis.
- Tooth is tender on percussion & pain can be severe making closure of the teeth difficult.



Etiology

VITAL TOOTH	NON VITAL TOOTH
Occlusal trauma	Sequelae of pulpitis
Wedging of foreign body between teeth	During root canal therapy Forcing of irrigating irrigants or medicaments through the apical foramen
Blow on teeth	Extension of obturating material through the apical foramen , Perforation of the root, Overinstrumentation
Orthodontic pressure	

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.

DIAGNOSIS:

CLINICAL DIAGNOSIS:

Tender to percussion

RADIOGRAPHIC FEATURES:

widening of PDL space

HISTOLOGIC FEATURES:

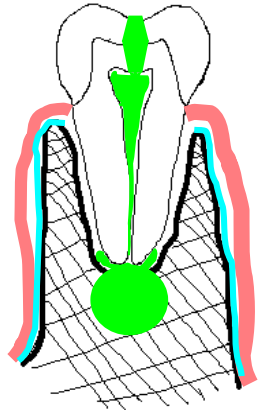
- PDL shows signs of inflammation -vascular dilation

  -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:

- Selective grinding if inflammation due to occlusal trauma.
- Extraction & endodontic treatment be done to drain exudate.

Chronic Apical Periodontitis (Periapical Granuloma)



- A growth of granulomatous tissue continuous with the Periodontal ligament resulting from the death of the pulp and the diffusion of bacterial toxins from the root canals into surrounding periradicular tissue through the apical and lateral canals
- Low- grade infection
- Most common sequelae of pulpitis or apical periodontitis.
- If acute (exudative) left untreated → chronic (proliferative).

- Term is not accurate since it doesn't shows true granulomatous inflammation microscopically.
- Presence of lateral or accessory root canals opening on the lateral surface of the root give rise to **lateral granuloma**

ETIOLOGY

- Death of the pulp
- Irritation of the periapical tissue that stimulates a productive cellular response

CLINICAL FEATURES:

- Tooth involved is non vital / slightly tender on percussion.
- Percussion may produce dull sound instead metallic 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.
- No perforation of bone & oral mucosa forming fistulous tract unless undergoes acute exacerbation.

RADIOGRAPHIC FEATURES:

- Thickening of PDL at root apex.
- As concomitent bone resorption & proliferation of granulation tissue appears to be radiolucent area.
- Thin radiopaque line or zone of sclerotic bone sometimes seen outlining lesion.
- Long standing lesion may show varying degrees of root resorption.

Figure 3-15 · Periapical granulomas. Discrete periapical radiolucencies associated with the apices of the mandibular first molar. (Courtesy of Dr. Garth Bobrowski.)

Figure 3-18 · Periapical granuloma. Ill-defined radiolucency associated with the mandibular first molar, which exhibits significant root resorption.

HISTOLOGIC FEATURES:

- Hyperemia and edema of the PDL ligament with infiltration of chronic inflammatory cells.
- Inflammatory and locally increased vascularity of the tissue are associated with resorption of the surrounding bone adjacent to this area.
- 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.

In some granulomas,

- Large number of phagocytes will ingest lipid material and become collected in groups forming foam cells
- Abundant mast cells may be found
- Deposits of cholesterol as well as hemosiderin are often present and both are probably derived from the breakdown of extravasated RBCs.

Epithelium of Periapical granuloma can be derived from:

- Respiratory epithelium of the maxillary sinus
- Oral epithelium growing in through a fistulous tract
- Oral epithelium proliferating apically from a periodontal pocket or bifurcation or trifurcation involvement by periodontal disease also with apical proliferation.

- **Dunlap and Barker** – termed **Giant-cell hyaline angiopathy.** Consists of inflammatory cell infiltration, giant cells, rushton bodies, eosinophilic material resembling hyalinized collagen.
- 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.

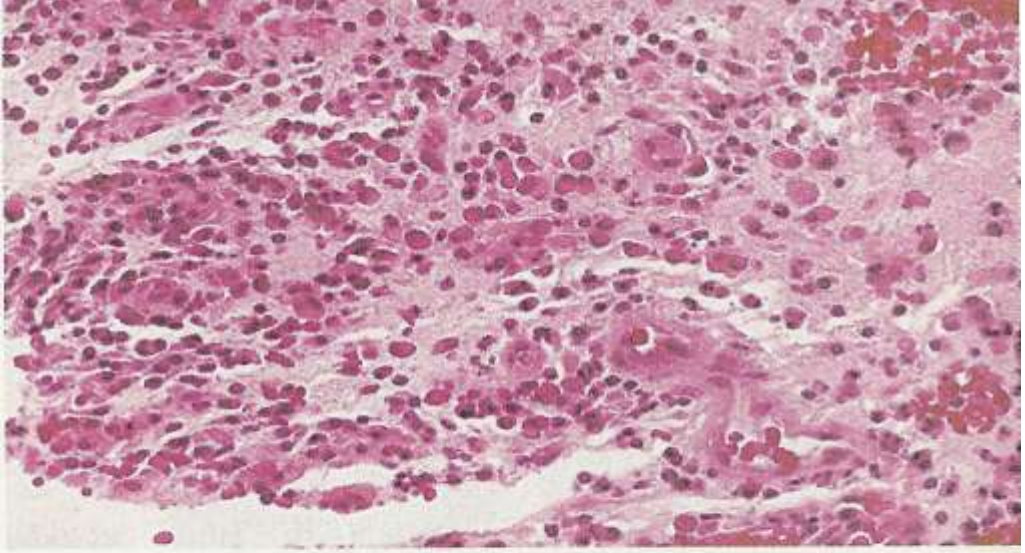
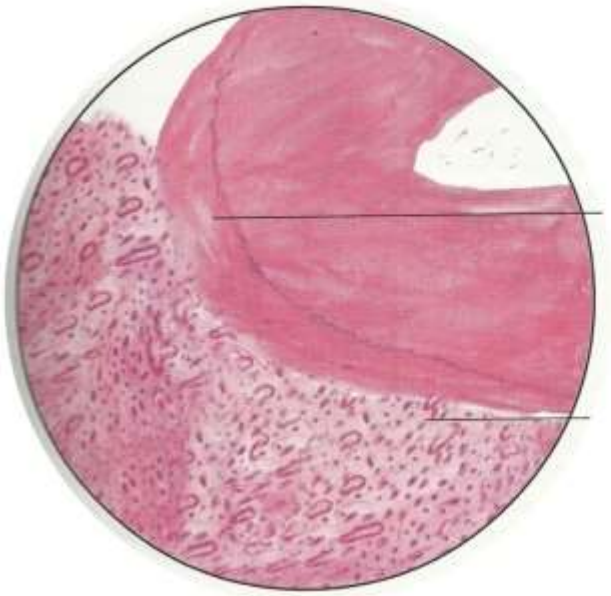


Fig. 3-18 Periapical granuloma. Granulation tissue exhibits mixed inflammatory infiltrate consisting of lymphocytes, plasma cells, and histiocytes.

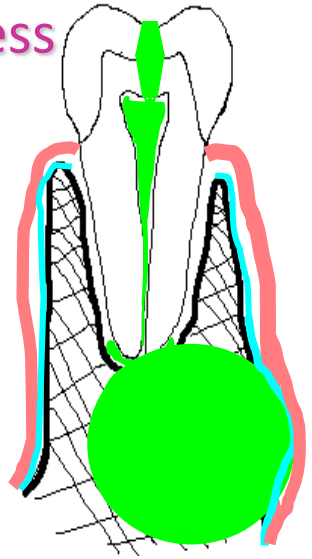


TREATMENT

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

Periapical Abscess
(Dento-Alveolar abscess, Alveolar Abscess)

- Periapical abscess is an acute or chronic suppurative process of the dental periapical region.
- Developed from acute periodontitis / periapical granuloma.
- Acute exacerbation of chronic lesion → Phoenix Abscess

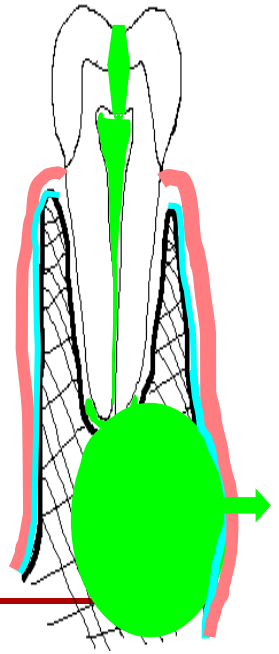


ETIOLOGY:

- traumatic injury
- pulp necrosis,
- irritation of periapical tissues

CLINICAL FEATURES:

- Common findings of inflammation- heat, redness, swelling and pain.
- Tenderness of tooth, which relives after pressure application.
- Extreme painful tooth extrude from socket.
- Systemic manifestations like lymphadenitis & fever may present when confined to periapical region.
- Rapid extension to adjacent bone marrow spaces produces acute osteomyelitis or dentoalveolar abscess.



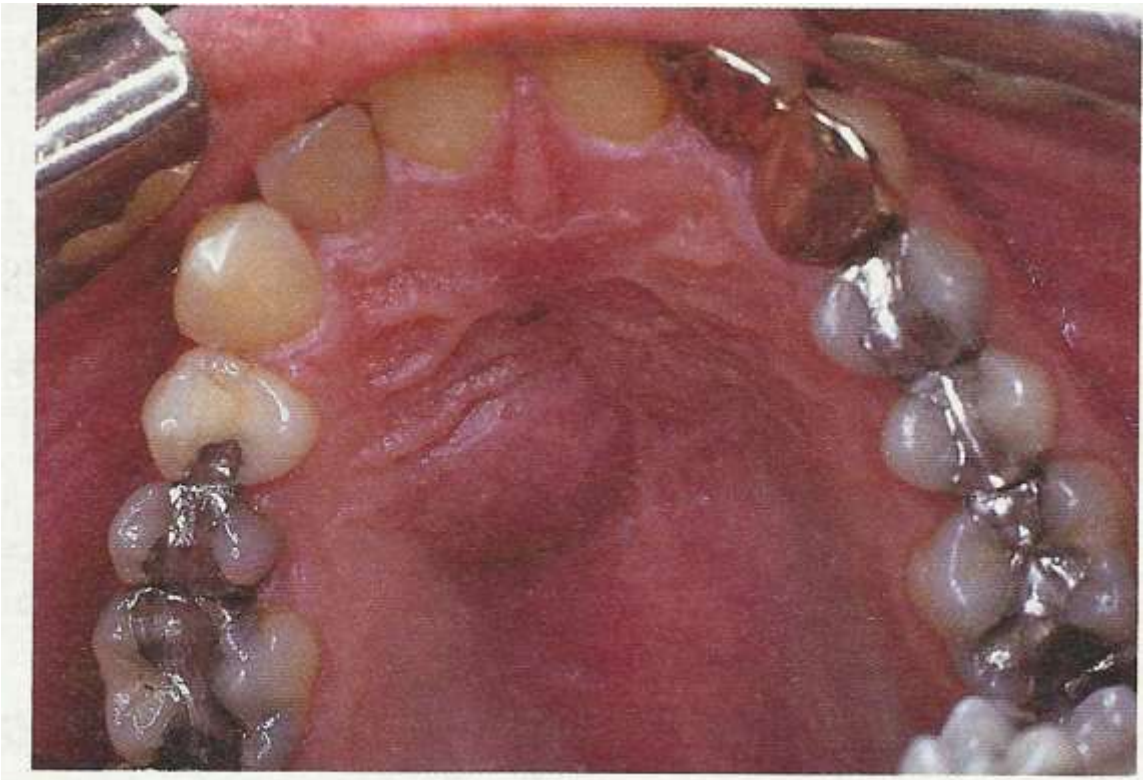
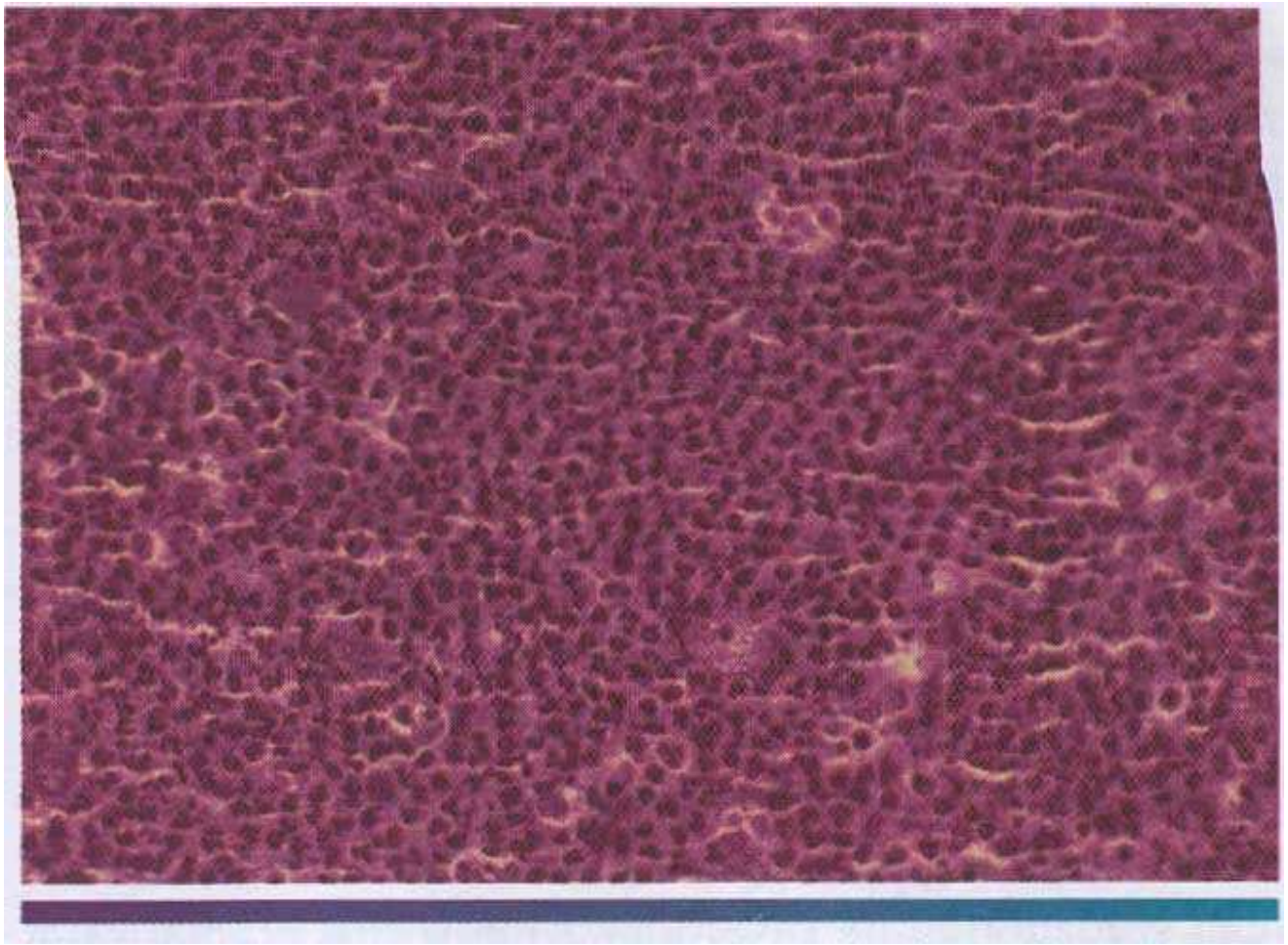


FIGURE 13-4 Palatal abscess representing extension of a periapical abscess.

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.



Sheet of polymorphonuclear leukocytes intermixed with scattered histiocytes

RADIOGRAPHIC FEATURES:

- Slight thickening of PDL space.
- Radiolucent area at apex of root.

TREATMENT:

- Drainage of abscess by opening pulp chamber or extraction.
- Root canal treatment.
- If untreated, causes osteomyelitis, cellulites & bacteremia & formation of fistulous tract opening to oral mucosa.

ACUTE EXACERBATION OF A CHRONIC LESION/ PHOENIX ABSCESS

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

ETIOLOGY:

- Periradicular disease
- Bacteria released from root canals during instrumentation may trigger acute response

Symptoms:

- At onset, tooth is tender to touch
- As inflammation progresses tooth may be elevated in its socket and may become sensitive.
- Mucosa over the radicular area appears red and swollen

Histopathologically, shows areas of liquefaction necrosis with disintegrated polymorphonuclear leukocytes & cellular debris surrounded by macrophages, lymphocytes, plasma cells in periradicular tissues.

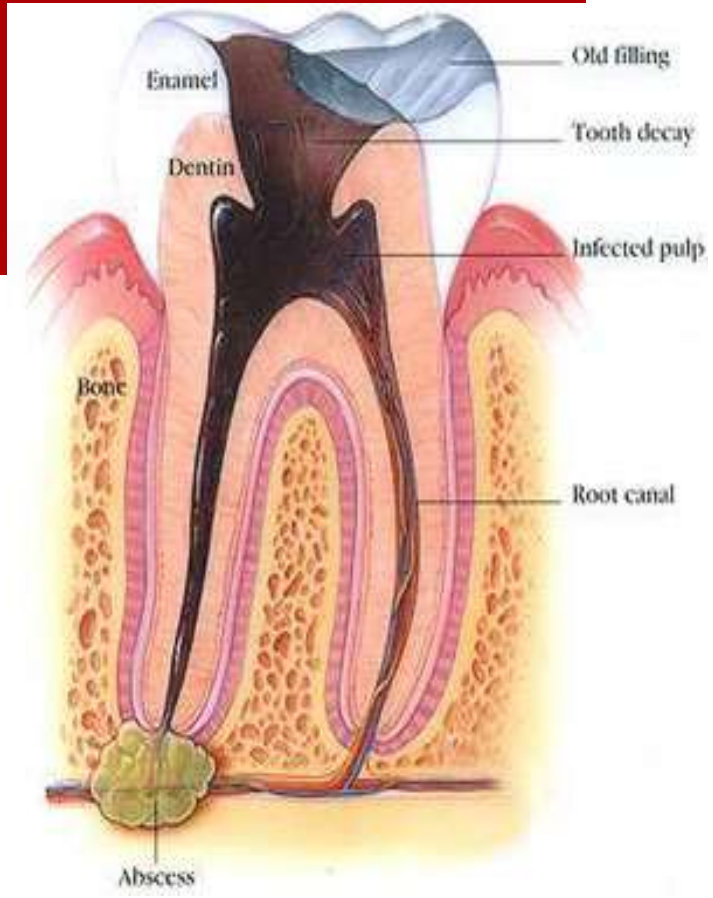
DIAGNOSIS:

- Common findings of inflammation- heat, redness, swelling and pain.
- Most commonly associated with the initiation of RCT
- History of trauma
- Radiographically, well defined periradicular lesion may be present.

TREATMENT:

- Drainage and debriment
- Root Canal Treatment

PULP AND PERIAPICAL PATHOLOGIES- 2



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CONTENTS

RADICULAR CYST

INTRODUCTION

- Radicular cysts are the most common inflammatory cysts and arise from the epithelial residues in the periodontal ligament as a result of periapical periodontitis following death and necrosis of the pulp.
- Most common odontogenic cyst.
- Other names: Periapical cyst, apical periodontal cyst, root end cyst or dental cyst

Periapical Cyst: These are the radicular cysts which are present at root apex.

Lateral Radicular Cyst: These are the radicular cysts which are present at the opening of lateral accessory root canals of offending tooth.

Residual Cyst: These are the radicular cysts which remains even after extraction of offending tooth

ETIOLOGY: Infected tooth, leading to necrosis of tooth

Inflammation stimulates the epithelial rests of melassez found in apical periodontal ligament

↓

Periapical granuloma infected/sterile

↓

Epithelium undegoes necrosis caused by lack of blood supply

↓

Cyst

- **BAY CYST:** Island of squamous epithelium which have developed from the odontogenic rests of Malassez can also be found in a periapical granuloma without cystic transformation.

Pathogenesis

It is convenient to consider the pathogenesis of radicular cysts in three phases:

1. the phase of initiation,
2. the phase of cyst formation and
3. the phase of enlargement.

The phase of initiation

- Epithelial linings of these cysts are derived from the epithelial cell rests of Malassez in the periodontal ligament.

Epithelium may also be derived from:

- Respiratory epithelium of the maxillary sinus
- Oral epithelium growing in through a fistulous tract
- Oral epithelium proliferating apically from a periodontal pocket or bifurcation or trifurcation involvement by periodontal disease also with apical proliferation.

- **Bacterial endotoxins** released from the necrotic pulp- Key factor [Meghji *et al. (1996)]*
- Important role for inflammatory **cytokines** in the proliferation of epithelial cell rests. Immunoglobulin G (IgG) is the predominant class. [Stern *et al. (1981)]*

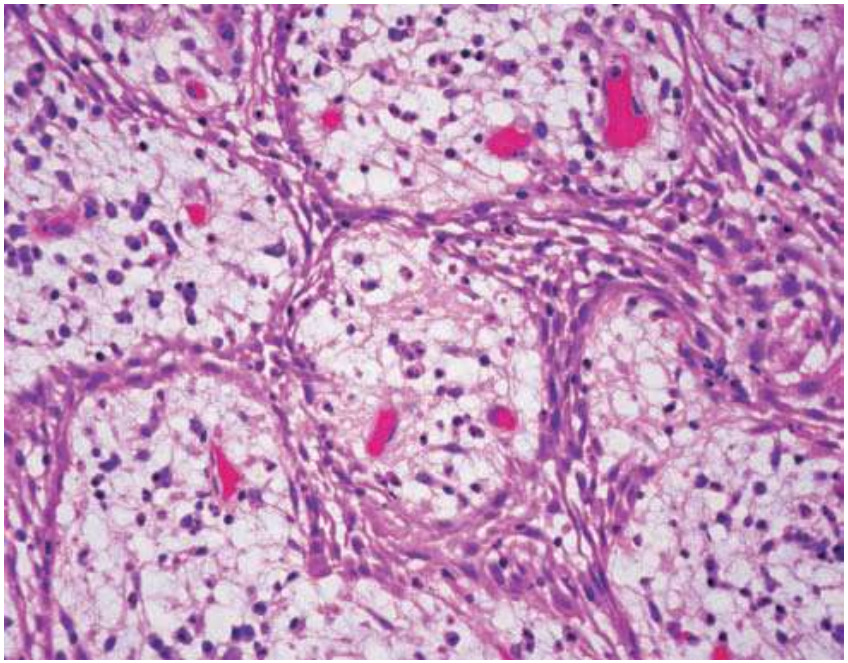
  IL-1β, IL-6, IL-8, TNF-α, γ-interferon (IFN-γ), and transforming growth factor β1 (TGF-β1) and most of these showed increased expression. [Kusumi *et al. (2004)]*

Phase of cyst formation

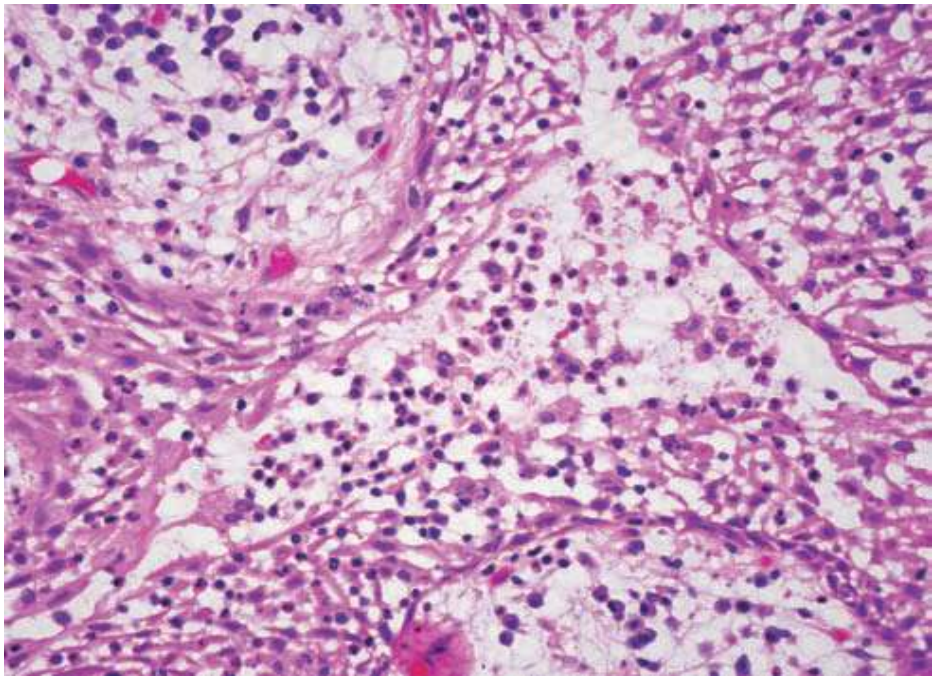
2 CONCEPTS:

1. Epithelium proliferates and 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 (Summers, 1974).
2. **More widely supported theory**→ cyst cavity forms within a proliferating epithelial mass in an apical granuloma by degeneration and death of cells in the centre

The proliferating epithelial masses show **considerable intercellular oedema**. These intercellular accumulations of fluid coalesce to **form microcysts** containing epithelial and inflammatory cells.

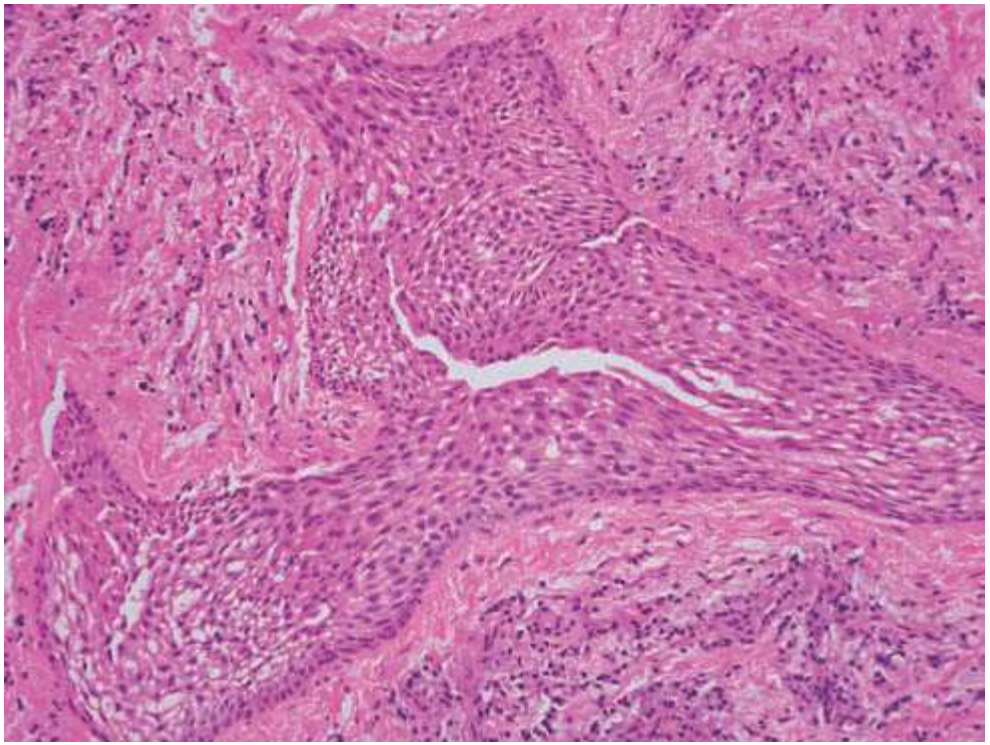


Arcades and rings of proliferating epithelium in an apical granuloma



Degeneration of cells in the centre of a mass of proliferating epithelium in an apical granuloma → **Microcyst**

- In some periapical lesions, sheets of epithelial cells with **distinct clefts** are seen and in certain instances the cyst may be initiated in this way.



Sheet of epithelial cells in a periapical lesion

Growth and enlargement of the radicular cyst

- According to Toller- **osmosis** makes a contribution to the increase in the size of cysts. Lytic products of the epithelial and inflammatory cells in the cyst cavity provided the greater numbers of smaller molecules which raised the osmotic pressure of the cyst fluid.
- According to Harris and Toller (1975)- **epithelial proliferation continues as long as there is an inflammatory stimulus**, and suggested that this contributed to enlargement of the cyst.

- **According to Harris and Goldhaber (1973)-** Growth of the cyst must also be accompanied by **degradation of adjacent connective tissues and bone resorption**.
- The **synthesis of prostaglandins**, their bone resorbing capacity and their possible role in the enlargement of jaw cysts. **(Harris et al 1973)**
- **Collagenases** also contribute to breakdown of the connective tissues and collagenolytic activity→ Expansion
- **MMPs and gelatinase**→ possible role in cyst expansion

CLINICAL FEATURES: -

- **Age incidence:** peak in 3rd, 4th and 5th decade
- **Sex incidence:** Slightly more males.
- **Frequency:** Commonest cystic lesion of jaws.
- Primarily symptom less.
- Discovered accidentally during routine dental X ray exam.
- **Diagnostic criteria** – **associated teeth are non vital**
- Rare in deciduous teeth.

Clinical presentation

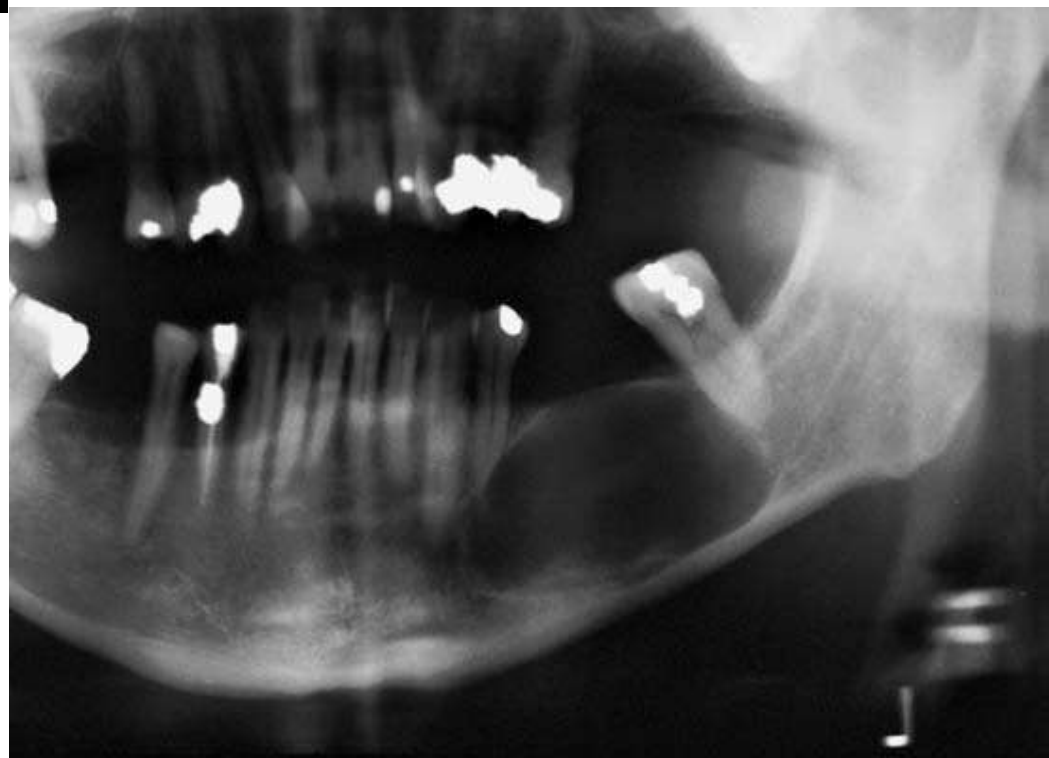
- Symptomless
- At first the enlargement is bony hard but as the cyst increases in size, the covering bone becomes very thin despite subperiosteal bone deposition and the swelling then exhibits **‘egg shell crackling’.**
- Occasionally, a sinus may lead from the cyst cavity to the oral mucosa.

RADIOLOGICAL FEATURES:

- Classically presents as round / ovoid radiolucency with sclerotic borders and associated with pulpally affected tooth.
- Rarely induce resorption of affected teeth

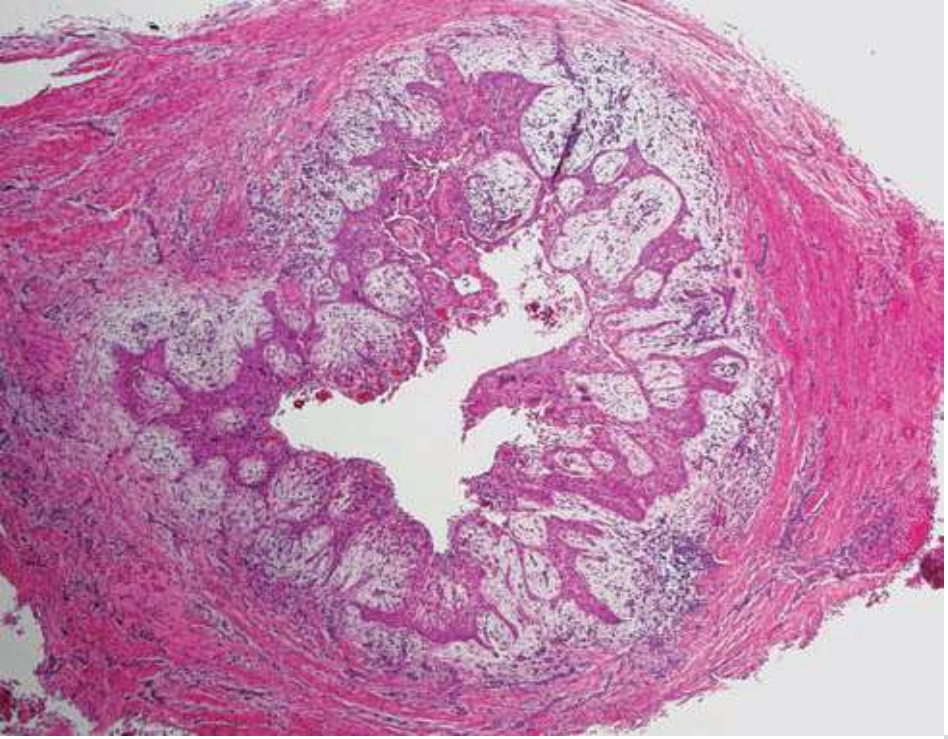
The lesion is a well defined radiolucency associated with the apex of a non-vital root filled tooth.

The lesion is at the site of a previously extracted tooth



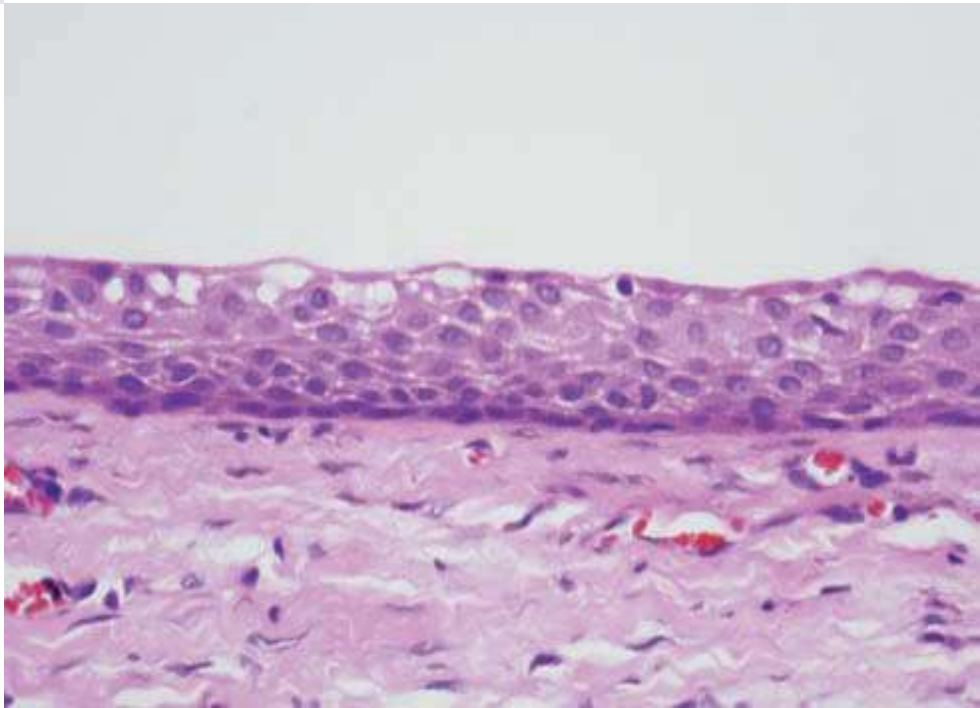
HISTOLOGICAL FEATURES

- Almost all radicular cysts are lined wholly or in part by stratified squamous epithelium.
- These linings may be discontinuous in part and range in thickness from 1 to 50 cell layers. The majority are 6–20 cell layers thick.
- **In early cysts-** the epithelial lining may be proliferative and show arcading with an intense associated inflammatory process .
- **As cyst enlarges-** the lining becomes quiescent and fairly regular with a certain degree of differentiation to resemble a simple stratified squamous epithelium



cyst is lined by proliferating epithelium

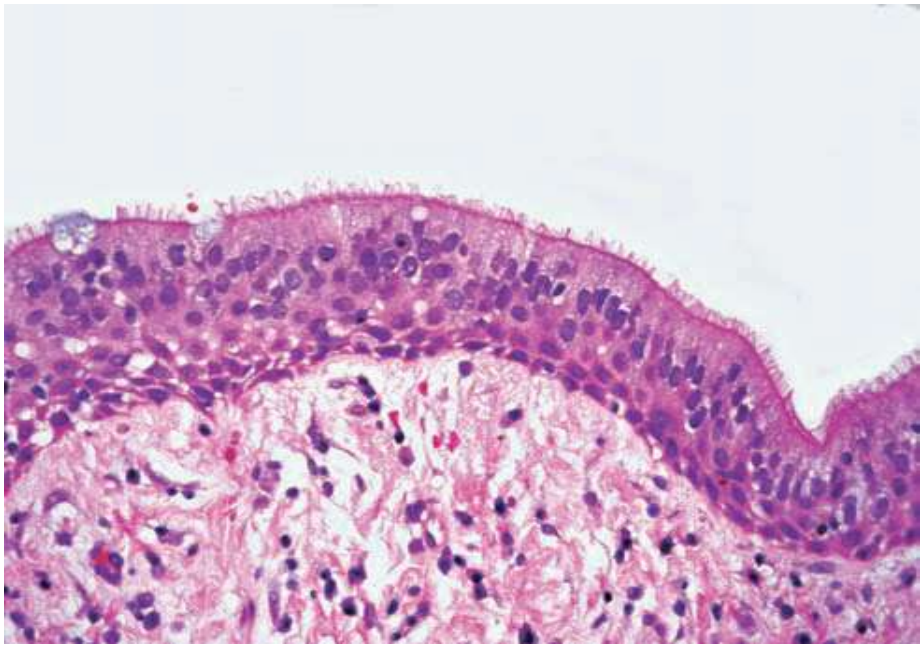
Quiescent epithelium lining a mature, long-standing radicular cyst



- Keratin formation is only seen → 2% of radicular cysts
- Orthokeratinisation is most common, with evidence of a granular cell layer, but parakeratinisation may also be seen (Browne and Smith, 1991).
- Inflammatory cell infiltrate in the proliferating epithelial linings consists → predominantly of polymorphonuclear leucocytes whereas the adjacent fibrous capsule is infiltrated mainly by chronic inflammatory cells (Shear, 1963).
- Proliferating epithelial linings show →degree of spongiosis.
- As the cyst enlarges, the wall may become less inflamed and fibrous

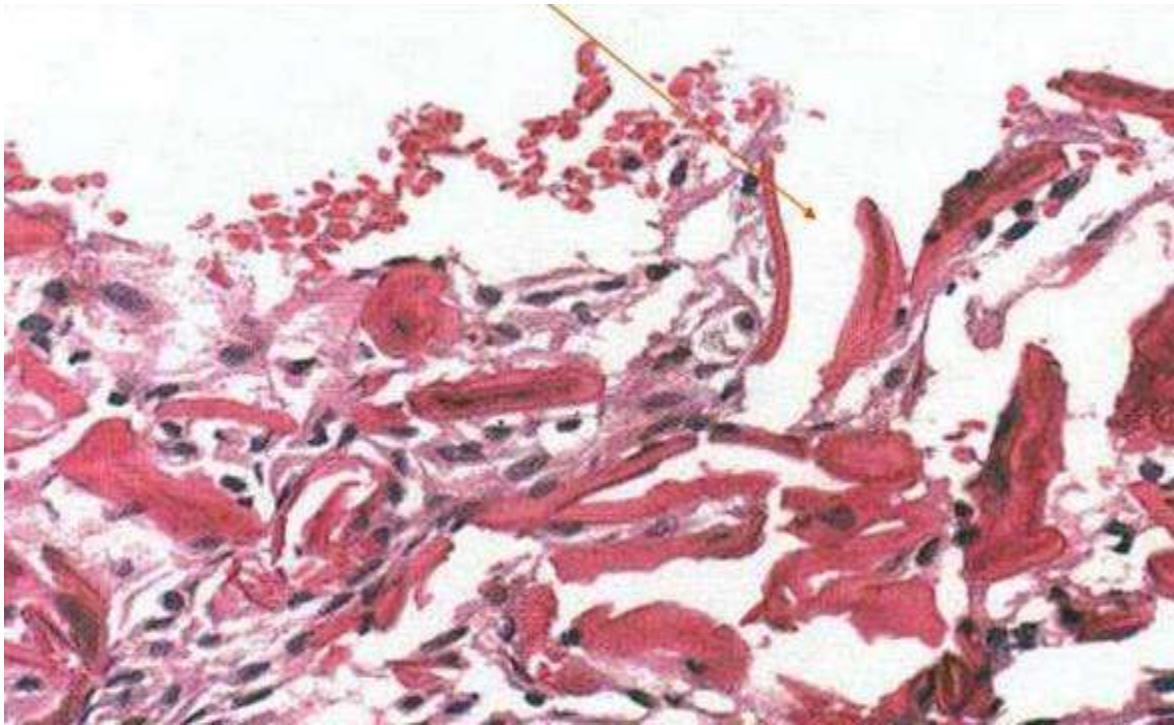
- Metaplastic changes, in the form of mucous cells or ciliated cells, are frequently found in the epithelial linings of radicular cysts (Shear, 1960)

Mucous cells in the surface layer of the stratified squamous epithelial lining of a radicular cyst

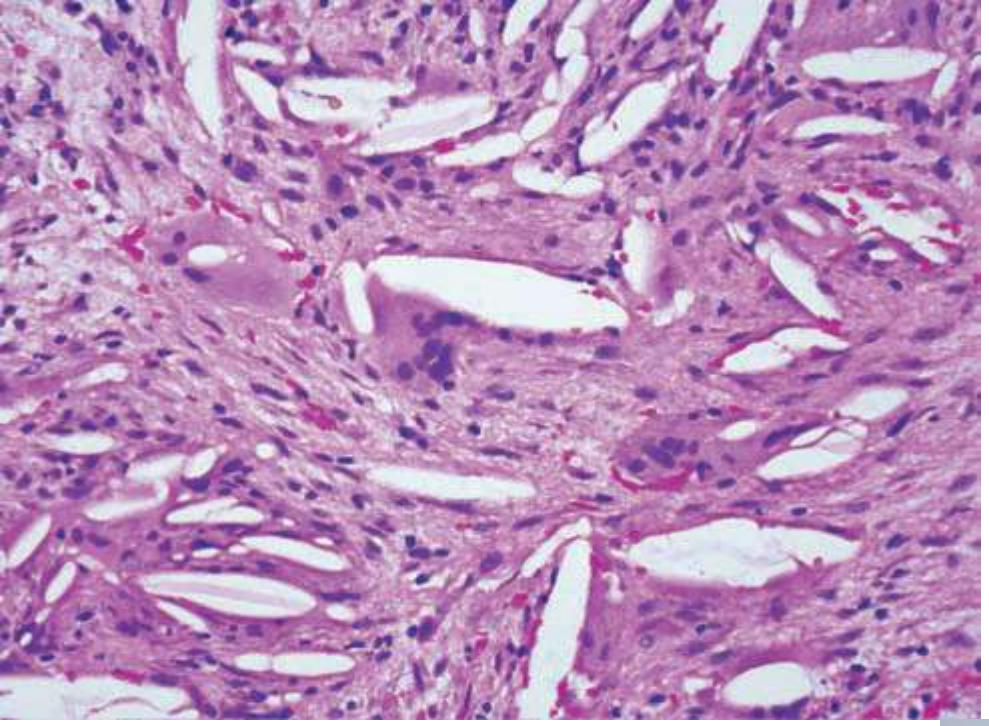


Ciliated epithelium in a radicular cyst

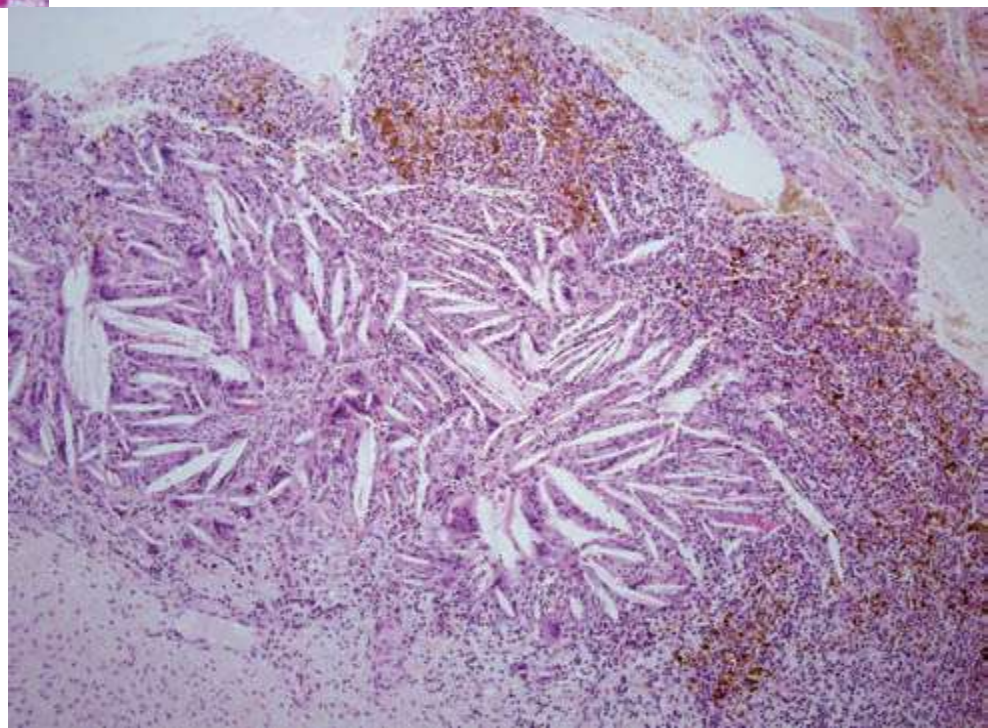
- In approximately **10% of radicular cysts**, hyaline bodies, first described by **Dewey in 1918** and often referred to as **Rushton's hyaline bodies**, are found in the epithelial linings.
- These hyaline bodies are *tiny linear or arc- shaped bodies, generally associated with the lining epithelium, that appear amorphous in structure, eosinophilic in reaction and brittle in nature, since they evidence fracture in some cases*.
- Origin- haematogenous origin



- Deposits of cholesterol crystals are found in many radicular cysts (Shear 1963).
- Main source of cholesterol → disintegrating red blood cells in a form that readily crystallises in the tissues
- Slow but considerable accumulation of cholesterol could occur through degeneration and disintegration of lymphocytes, plasma cells and macrophages taking part in the inflammatory process, with consequent release of cholesterol from their walls.
- The granulation tissue containing the cholesterol protrudes into the cyst cavity and appears macroscopically and microscopically as a ‘mural nodule’.



Multinucleate foreign body giant cells on the surface of cholesterol clefts in the wall of a radicular cyst



- Mast cells have been demonstrated in the epithelium and the connective tissue wall, particularly in the subepithelial zone.
- Haemorrhage is invariably present and haemosiderin deposits are seen in many specimens.
- Calcifications of various kinds are frequently present.

TREATMENT:

- Extraction of the involved tooth + Curettage of the periapical tissue.
- Root canal treatment + Apicocectomy.

OSTEOMYELITIS

- The word “osteomyelitis” originates from the ancient Greek words osteon (bone) and muelinos (marrow)
- Inflammatory condition of the bone, which begins as an infection of the medullary cavity, rapidly involves the haversian systems, and extends to involve the periosteum of the affected area.

- “It define as the inflammation of bone and its marrow contents”. (Shafer)

- “It is an acute or chronic inflammatory process in the medullary spaces or cortical surfaces of the bone that extends away from the initial site of involvement”. (Neville)

CLASSIFICATION

By Topazian RG

I. Suppurative osteomyelitis

1. Acute suppurative osteomyelitis
2. Chronic suppurative osteomyelitis
   - – Primary chronic suppurative osteomyelitis
   - – Secondary chronic suppurative osteomyelitis
3. Infantile osteomyelitis

II. Nonsuppurative osteomyelitis

1. Chronic sclerosing osteomyelitis

– Focal sclerosing osteomyelitis

– Diffuse sclerosing osteomyelitis

2. Garrè's sclerosing osteomyelitis

3. Actinomycotic osteomyelitis

4. Radiation osteomyelitis and necrosis

PREDISPOSING FACTORS

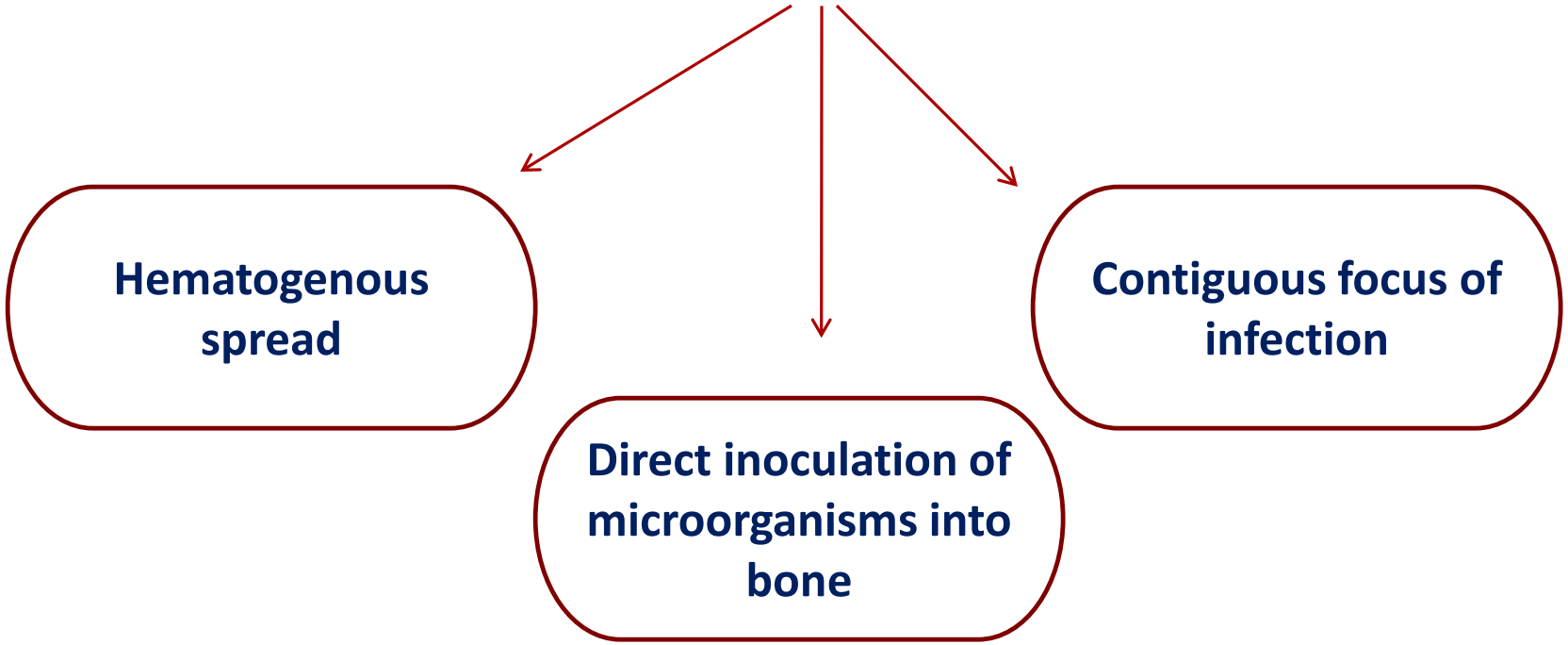
- Fractures due to trauma
- Road traffic accidents
- Gun shot wounds
- Radiation damage
- Paget`s disease
- Osteoporosis

Systemic disease :

- Malnutrition,
- Acute Leukemia,
- Uncontrolled diabetes,
- sickle cell anemia,
- Chronic alcoholism

PATHOGENESIS

Microorganisms may infect bone through one or more of three basic methods



- Osteomyelitis of the jaws is mainly caused by spread of adjacent odontogenic infection followed by traumatic fracture

Occurrance

Sex—more common in men, than women.

Osteomyelitis in maxilla :

Rare occurrance due to-

- Extensive blood supply
- Thin cortical plates
- Abundant medullary spaces

Osteomyelitis in mandible:

An important factor in establishment of osteomyelitis in mandible is *compromise of blood supply*

MICROBIOLOGY

	Facultative anaerobic	Anaerobic
Gram-positive cocci	•*Staphylococcus aureus* •Coagulase-negative staphylococci •*Streptococcus spp*	*Peptostreptococcus spp.*
Gram-positive rods	*Actinomyces spp.* *Corynebacterium spp.* *Lactobacillus spp.*	*Actinomyces israelii* *Eubacterium lentum*
Gram-negative cocci	*Neisseria spp.*	*Veillonella spp*
Gram-negative rods	*Actinobacillus actinomycetemcomitans*	*Fusobacterium spp.* *Porphyromonas spp.* *Prevotella spp.*

ACUTE SUPPURATIVE OSTEOMYELITIS

- Serious sequela of periapical infection that often results in diffuse spread of infection throughout the medullary spaces , with subsequent necrosis of variable amount of bone.
- Poly microbial- Staphylococcus aureus, S. albus, Porphyromonas, Prevotella, Bacteriodes.

ETIOLOGY

- Most common cause : Dental infection
- Infection due to fracture of jaw, gun shot, or
- Hematogenous spread

PATHOLOGY

Acute inflammation of marrow tissues

↓

Spread of exudate along the marrow spaces

↓

Thrombosis of vessels due to compression

↓

Necrosis of bone

↓

Necrotic tissues, dead and dying cell, pus from bacteria → fill the marrow space

↓

Involves cortical bone → Lifting of periosteum causing further necrosis

↓

Finally ,Osteoclastic activity >>>

SEQUESTRUM

CLINICAL FEATURES

- Maxilla : localized
- Mandible : Diffuse and widespread

In infants→ **NEONATAL MAXILLITIS**

- ORIGIN- Hematogenous spread or local oral infection
- Seriously ill and may not survive

In adults,

- Sever pain
- Trismus
- Parasthesia of lips in case of mandibular involvement
- Elevation of temperature
- Regional lymphadenopathy
- Loosening of teeth and exudation of pus from gingiva
- No swelling and redness till periostitis develops

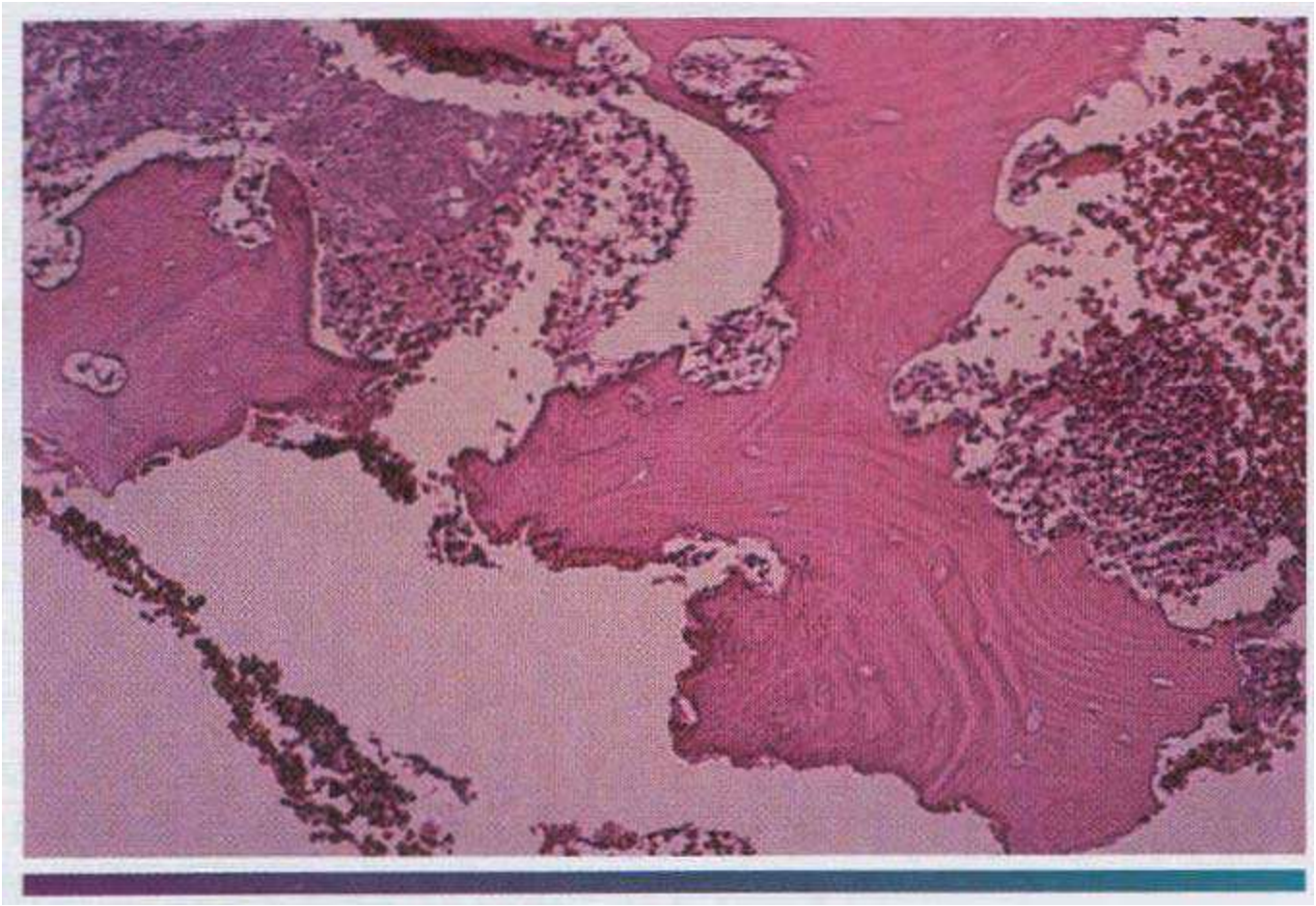
RADIOGRAPHIC FEATURE

- Progress rapidly → little evidence
- Trabeculae becomes fuzzy and indistinct
- Ill defined margins

Ill-defined area of radiolucency of the right body of the mandible

HISTOLOGIC FEATURES

- **Necrotic bone- loss of osteocytes** from their lacunae, peripheral resorption and bacterial colonization.
- **Medullary space→ filled with inflammatory exudates**
- The inflammatory cells are **chiefly PMNs** but may show occasional lymphocytes and plasma cells
- **Osteoblasts bordering the bony trabeculae are destroyed**
- Trabeculae may lose their viability and begin to undergo slow resorption



Nonvital bone exhibits loss of the osteocytes from the lacunae. Peripheral resorption and surrounding inflammatory response alsocan be seen

TREATMENT AND PROGNOSIS

- **3D** >>

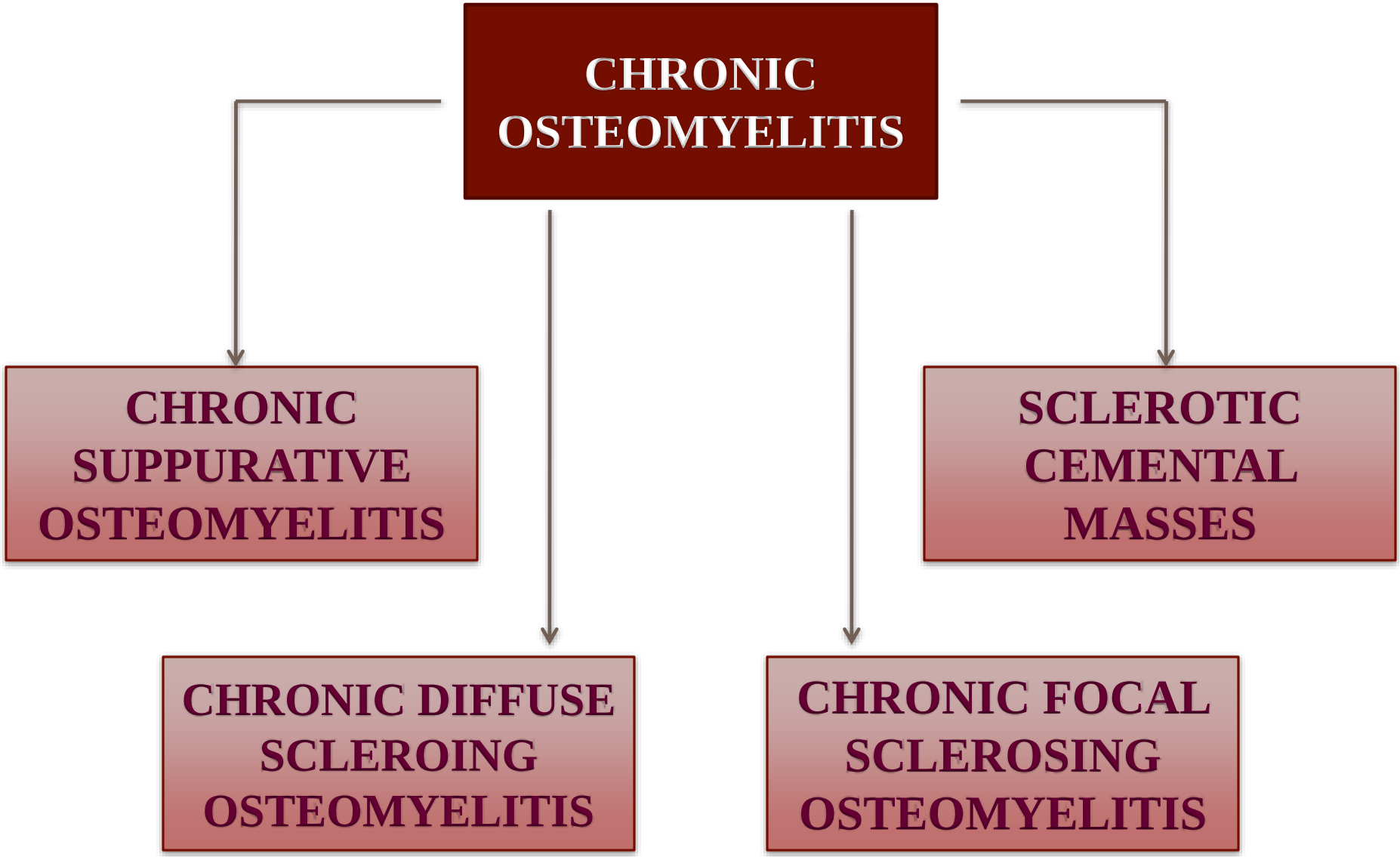
1. **Debridement ,**
2. **Drainage and**
3. **Drugs [Anti-microbial]**

- Sequestrum >> If small, exfoliates through mucosa
  >> If large, surgical removal
- Untreated cases may proceed to development of periostitis , soft tissue abscess or cellulitis

COMPLICATIONS

- **Rare but include:**

Pathological fracture	**Extensive bone destruction.**
Chronic osteomyelitis	**Inadequate treatment.**
Cellulitis	**Spread of virulent bacteria.**
Septicemia	**Immuno-compromised patient.**


CHRONIC OSTEOMYELITIS
CHRONIC SUPPURATIVE OSTEOMYELITIS
CHRONIC DIFFUSE SCLEROING OSTEOMYELITIS
CHRONIC FOCAL SCLEROSING OSTEOMYELITIS
SCLEROTIC CEMENTAL MASSES

CHRONIC SUPPURATIVE OSTEOMYELITIS

- Inadequately treated acute osteomyelitis
- Rarely- complication of irradiation
- Acute exacerbations of chronic stage may occur
- Fistulous tract may form which open to surface

CLINICAL FEATURES

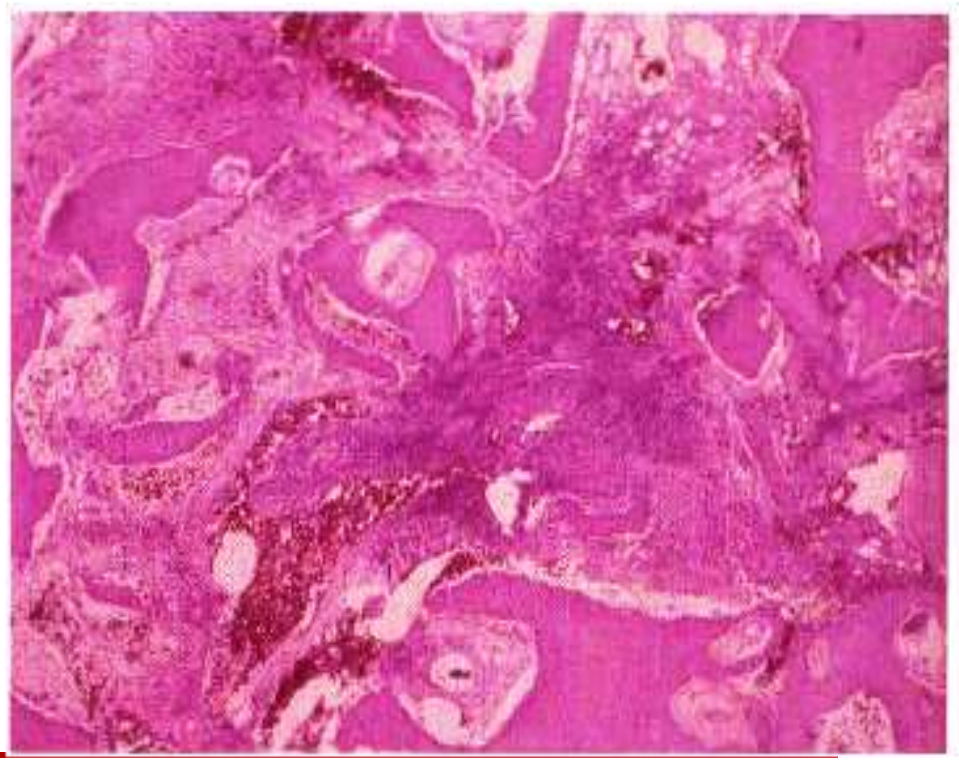
- Swelling
- Pain
- Sinus formation
- Purulent discharge
- Sequestrum formation
- Tooth loss
- Pathologic fracture

RADIOLOGICAL FEATURE

- Patchy, ragged & ill defined radiolucency.
- Often contains radiopaque sequestra.

HISTOLOGY

- Inflammed connective tissue filling inter-trabecular areas of bone.
- Scattered sequestra.
- Pockets of abscess.



Chronically inflamed and reactive fibrousconnective tissue filling the intertrabecular spaces.

TREATMENT

- Difficult to manage medically.
- Surgical intervention is mandatory, depends on spread of process.
- Antibiotics are same as in acute condition but are given through IV in high doses.

SMALL LESIONS

Curretage, removal of necrotic bone and decortication are sufficient.

EXTENSIVE OSTEOMYELITIS

Decortication combined with transplantation of cancellous bone chips.

PERSISTANT OSTEOMYELITIS

Resection of diseased bone followed by immediate reconstruction with an autologous graft is required. Weakened jawbones must be immobilized.

CHRONIC FOCAL SCLEROSING OSTEOMYELITIS (CONDENSING OSTEITIS)

- Unusual reaction of bone to infection
- A reaction to mild bacterial infection entering the bone through a carious tooth in persons who has high degree of tissue reaction and tissue reactivity.
- In some instances- tissue reacts to the infection by proliferation rather than destruction.
- The sclerotic reaction results from good patient immunity and a low degree of virulence of the offending bacteria

ETIOLOGY

- Infection of periapical tissues of a high immunity host by organisms of low virulence which leads to a localized bony reaction to a low grade inflammatory stimulus
- Non- vital tooth

CLINICAL FEATURES

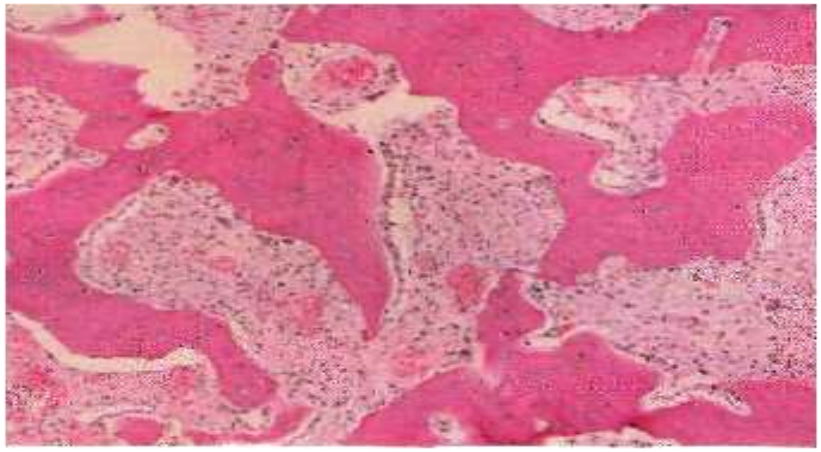
- Commonly affects young adults and children
- Mandibular molar is affected commonly
- Large carious lesions
- Symptoms : mild pain due to infected pulp

RADIOGRAPHIC FEATURES

- Pathognomic ,well circumscribed radiopaque mass of sclerotic bone surrounding and extending below the apex of one or both roots
- PDL space widening.

HISTOLOGIC FEATURES

- Dense bony trabeculae with little interstitial marrow tissue
- Many reversal and resting lines giving pagetoid appearance
- If interstitial soft tissue is present , it is generally fibrotic and infiltrated with small amount of lymphocytes
- lacunae appears empty



TREATMENT

- Root canal treatment
- Extraction

CHRONIC DIFFUSE SCLEROSING OSTEOMYELITIS

- CHRONIC DIFFUSE SCLEROSING OSTEOMYELITIS is the clinical entity characterized by a nonsuppurative, inflammatory process associated with recurrent swelling, trismus and pain
- Common in edentulous mandible
- Proliferative reaction of bone to a low grade infection.
- Portal of entry is diffuse periodontal disease

CLINICAL FEATURE

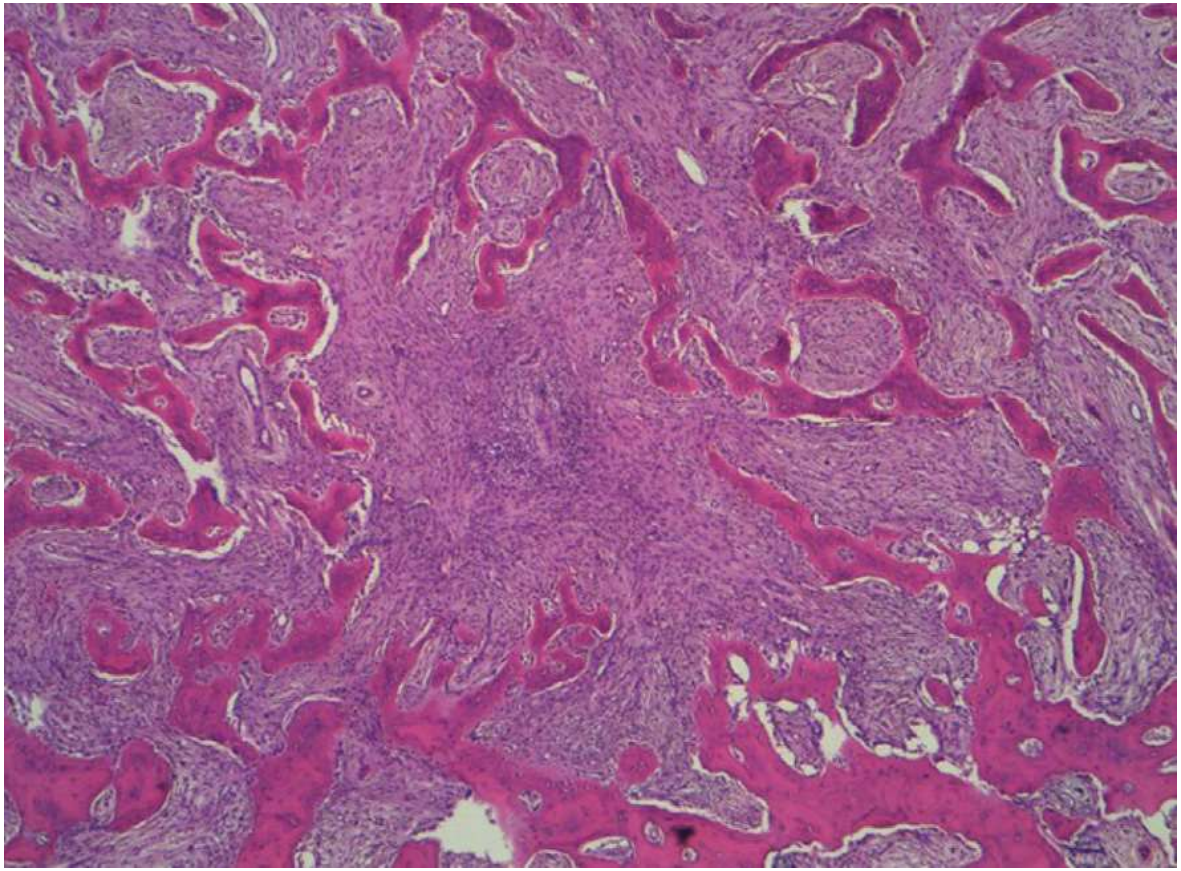
- Most common in older individual with especially in edentulous mandibular jaws or edentulous areas and does not exhibit any gender predominance.
- On acute exacerbation results in vague pain ,unpleasant taste and mild suppuration ,many times with the spontaneous formation of a fistula opening onto the mucosal surface to establish drainage.

RADIOLOGICAL FEATURE

- Diffuse patchy, sclerosis of bone (cotton wool appearance).
- Sometimes bilateral involvement
- Occationally involvement of both maxilla and mandible of same patient.
- Border between the sclerosed bone and normal bone is indistinct.

HISTOLOGIC FEATURES

- Dense , irregular trabeculae of bone bordered by **active layer of Osteoblasts ; focal Osteoclastic area may be present**
- Trabecular bone with the presence of reparative and reactive new bone formation. There was a marked **abundant osteoid and osteoblastic rim**.
- **Mosaic pattern appearance-** indicative of repeated periods of resorption followed by repair
- **Interstitial soft tissue is fibrotic**
- Proliferating fibroblasts and occasional small capillaries as well as small focal collection of lymphocytes and plasma cells



Irregular trabeculae of bone bordered by active layer of Osteoblasts.

Proliferating fibroblasts and occasional small capillaries as well as small focal collection of lymphocytes and plasma cells

TREATMENT

- Lesion is too extensive to be removed surgically
- Sclerotic bone is hypovascular and resistant to antibiotics
- Bell has recommended **extraction of tooth** as a last option utilizing a surgical approach with ***removal of liberal amounts of bone*** to facilitate extraction and *increase* ***bleeding***.
- Antibiotic administration during acute exacerbation may help

SAPHO syndrome

- It was first described by **Chamot et al. in 1987.**
- Rare and of unknown etiology.
- **The synovitis, acne, pustulosis, hyperostosis, and osteitis**
- In 1994, **Kahn et al. (1994**) reported three diagnostic criteria for SAPHO syndrome:

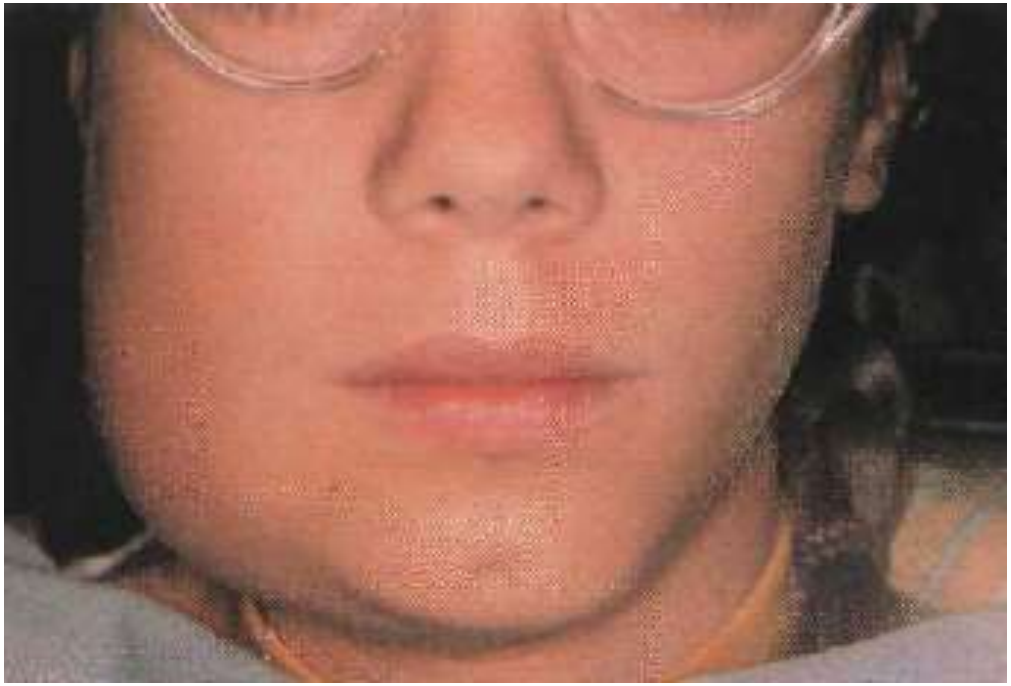
1. Multifocal osteomyelitis with or without skin manifestations.
2. Sterile acute or chronic joint inflammation associated with pustules or psoriasis on the palms and soles, acne, or hidradenitis.
3. Sterile osteitis in the presence of one of the skin manifestations

CHRONIC OSTEOMYELITIS WITH PROLIFERATIVE PERIOSTITIS (Garre's chronic nonsuppurative sclerosing osteitis ,periostitis ossificans)

- Garre's osteomyelitis was first described by Carl Gaffe in 1893 as *"a focal gross thickening of periosteum with peripheral reactive bone formation resulting from infection".*
- Non -suppurating type of osteomyelitis, with a reactive periosteal thickening due to a low-grade irritation or dental infection.

CLINICAL FEATURE

- Young person before the age of 25 years
- In jaws- more common in mandible of children and young adults (most cases occur in bicuspid and molar region)
- Hard swelling over the jaw, producing facial asymmetry with little or no pain.
- This occur as a result of overlying soft tissue infection or cellulitis subsequently involving periosteum.
- The overlying skin was normal, but could occasionally be inflammed

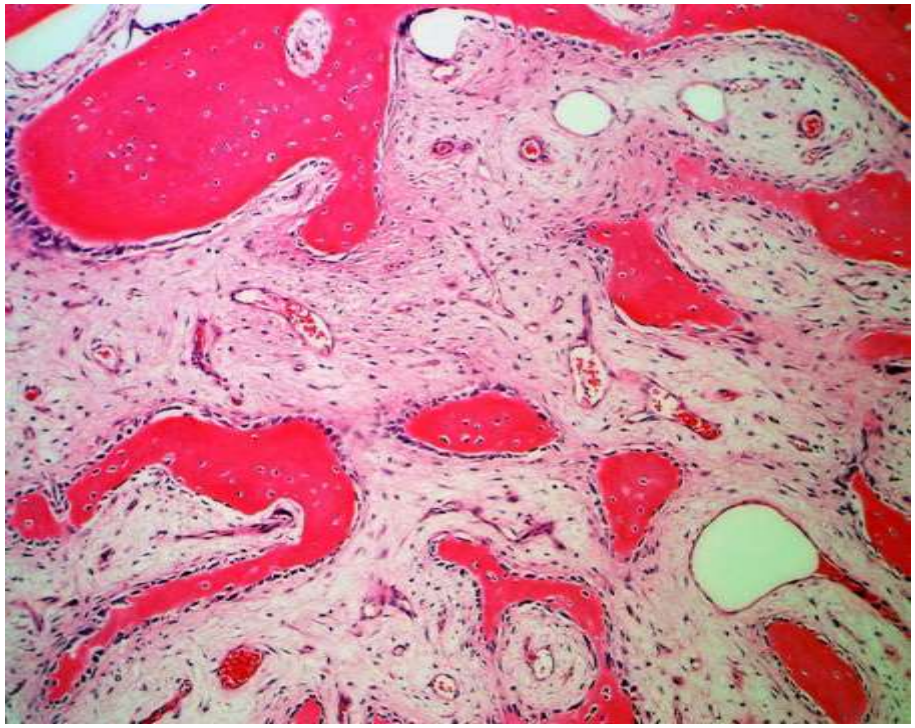


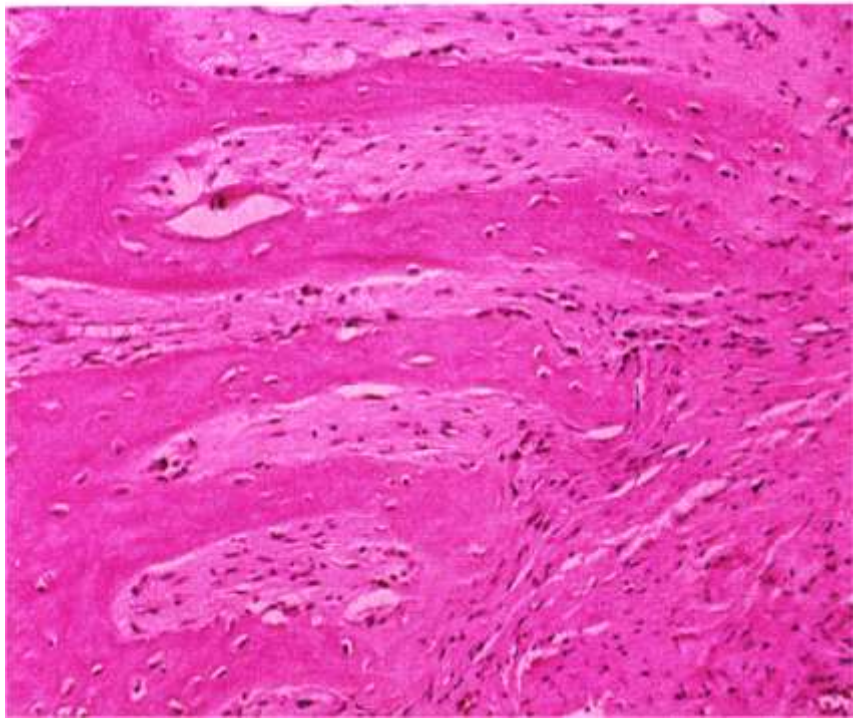
RADIOGRAPHIC FEATURE

- IOPA → reveals an carious tooth opposite the hard bony mass
- Occlusal radiograph →focal overgrowth of bone on the outer surface of the cortex ,which may be described as duplication of the cortical layer of bone .

HISTOLOGIC FEATURES

- Subperiosteal mass is composed of **much reactive new bone and osteoid tissue , with Osteoblasts bordering many of trabeculae**
- **Trabeculae is perpendicular to cortex and parallel to each** other
- Connective tissue is fibrous and shows sprinkling of lymphocytes and plasma cells





TREATMENT

- Extraction or endodontic treatment of the teeth
- No surgical intervention except biopsy to confirm diagnosis
- After extraction the jaws undergo remodeling and facial symmetry is restored
- Neoperiostitis or new periosteum formation may occur in certain conditions.

SCLEROTIC CEMENTAL MASSES

- Multiple symmetric lesions producing pain, drainage or localized expansion
- Common in black females
- Unknown etiology
- Large painless radiopaque mass usually involving several quadrants of the jaws.
- This condition has previously been described as chronic sclerosing osteomyelitis, sclerosing osteitis, or gigantiform cementoma, it appears more appropriate to consider these lesions as part of the spectrum of the benign fibro-osseous lesions of periodontal ligament origin

HISTOLOGIC FEATURES

- Cemental masses have tissues interrupted by the cementum unlike diffuse type which mostly have sclerotic bone
- In some instances ,the cementum is in the form of large solid masses with smooth, lobulated margins often with a globular accretion pattern

CELLULITIS/ PHLEGMON

- **Cellulitis** is a diffuse inflammation of soft tissues which is not circumscribed or confined to one area, but which, in contrary to the abscess, tends to spread through tissue spaces and along fascial spaces.
- If an abscess is not able to establish drainage through the surface of the skin or into the oral *cavity it may* spread diffusely through fascial planes of the soft tissue.

ETIOLOGY

- It occurs as a result of infection by microorganisms that produce significant amount of **streptokinase, hyaluronidase and fibrinolysins** which acts to breakdown or dissolve hyaluronic acid , the universal intracellular cement substance, and fibrin.
- **MICROORGANISMS**- **Streptococci**, Prevotella & Porphyromonas
- Dental infection
- Sequela of periapical abscess or osteomyelitis
- Pericoronitis
- Tooth extraction or injection with a infected needle

PATHOLOGY

Streptococci → potent producers of hyaluronidase

↓

In their growth phase consume local oxygen

↓

Metabolize nutrients to produce → acidic environment

↓

Conductive to the subsequent growth of anaerobic microbes

↓

Prevotella, Porphyromonas destroy collagen

CLINICAL FEATURES

- **Systemic features** →increased body temperature , general fatigue, chills, sweatings, headache, loss of appetite.
- Swelling is because of inflammatory edema.
- If superficial tissue space involved- skin is inflammed, has an orange peel appearance and is even purplish
- If spread of infection in deeper planes- overlying skin is normal
- Regional lymphadenitis present

In maxilla-

- Perforates the outer cortical layer of bone above the buccinator attachment → swelling in the upper half of the face.
- Extension towards eye→ cavernous sinus thrombosis

In mandible-

- Perforates the outer cortical plate below the buccinator attachment → swelling in the lower half of the face.
- Spread to cervical tissue cause respiratory discomfort

- Facial abscess and Fistulous tract may occur

HISTOLOGICAL FEATURE

- A microscopic section through an area of cellulitis shows a diffuse exudation of polymorphoneuclear leukocyte and lymphocyte.
- Considerable serous fluid and fibrins causing separation of connective tissue and muscle fibres.

COMPLICATION

- Cavernous sinus thrombosis
- Ludwigs Angina
- Facial abscess

TREATMENT

- Antibiotics
- Antianaerobics
- Removal of the cause of the infection
- To avoid massaging the affected area to avoid spread

CONCLUSION

- Establishment of proper diagnosis is of utmost importance to carry out the effective clinical procedure for the benefit of patient .
- Review after the treatment is also to be given importance
- It is essential that we understand the progressive nature of the periapical disease process as well as how and why the various stages occur so they can be diagnosed and managed appropriately.

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THANK YOU