OSTEOMYELITIS

WHAT'S IN THE NAME?

- The word "osteomyelitis" originates from the ancient Greek words osteon (bone) and muelinos (marrow) and literally means infection of medullary portion of the bone.

WHAT IS IT?

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

Chronic osteomyelitis of the left mandible

(a) Extraoral fistula and scar formation

(b) large exposure of infected bone and sequestra

(c) Large sequester collected from surgery

FACTORS PREDISPOSING TO OSTEOMYELITIS

LOCAL FACTORS
(decreased vascularity/vitality of bone)

- Trauma.
- Radiation injury.
- Paget's disease.
- Osteoporosis.
- Major vessel disease.

SYSTEMIC FACTORS
(impaired host defense)

- Immune deficiency states.
- Immunosuppression
- Diabetes mellitus.
- Malnutrition.
- Extremes of age.

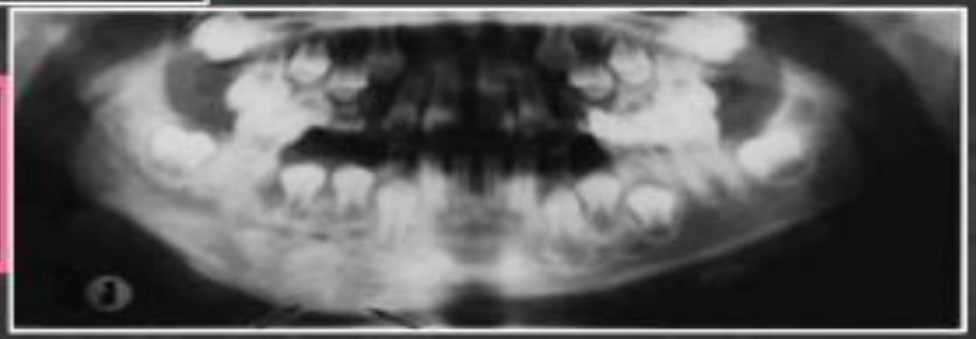
TYPES OF OSTEOMYELITIS

SUPPURATIVE OSTEOMYELITIS

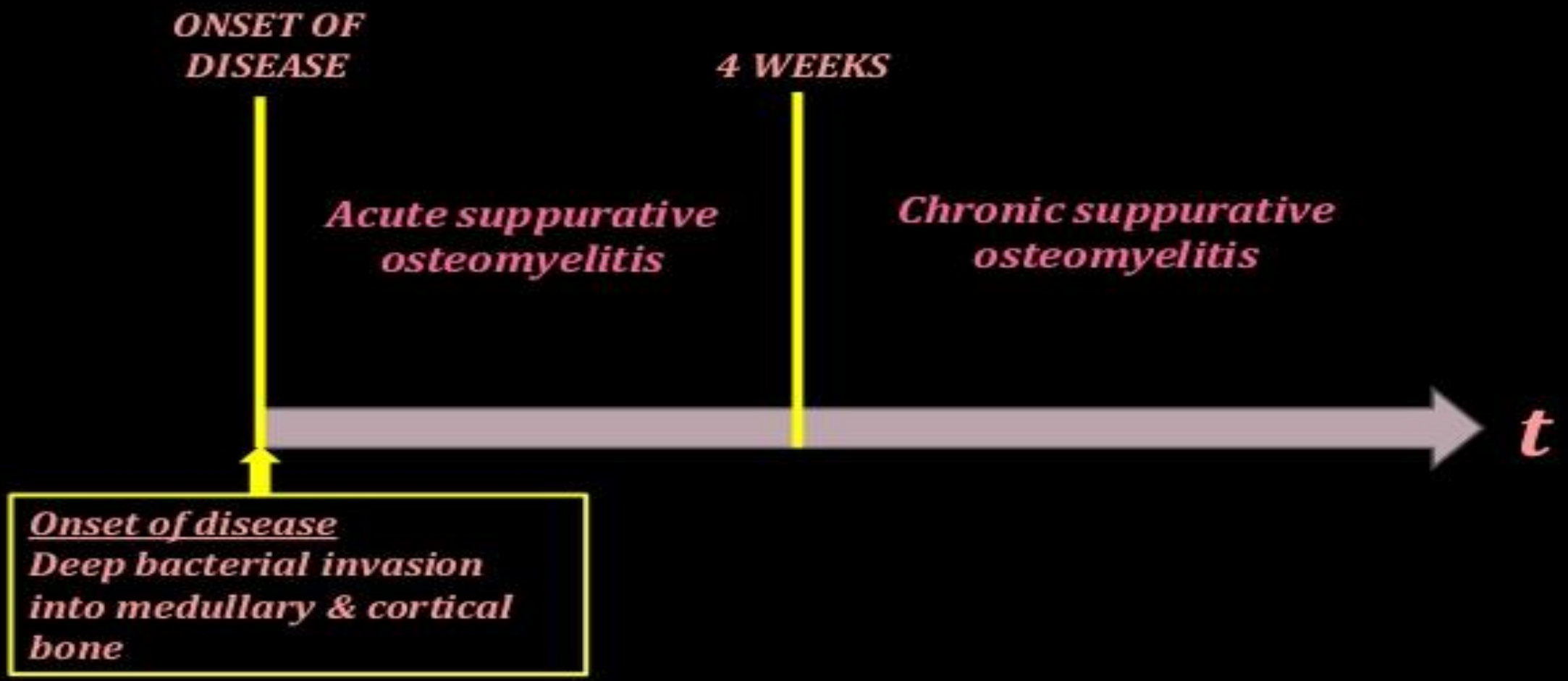
FOCAL SCLEROSING OSTEOMYELITIS

DIFFUSE SCLEROSING OSTEOMYELITIS

PROLIFERATIVE PERIOSTITIS



SUPPURATIVE OSTEOMYELITIS



SUPPURATIVE OSTEOMYELITIS

- *Source of infection is usually an adjacent focus of infection associated with teeth or with local trauma.*
- *It is a polymicrobial infection, predominating anaerobes such as Bacteriods, Porphyromonas or Provetella.*
- *Staphylococci may be a cause when an open fracture is involved.*
- *Mandible is more prone than maxilla as vascular supply is readily compromised.*

Cropped panoramic radiograph of suppurative osteomyelitis at the right side of mandible.

Microbiology

- Aerobs – Streptococcus viridans, Pyogenes
  Staphylococcus
- Anaerobic bacteria - Bacteroides
  Pseudomonas
  Peptostreptococci
- Others – Klebsiella , Proteus , E. Coli
- Specific – M. tuberculosis, T. Pallidum

- Therefore Mixed infection

Antimicrobial Rx

- First choice

  IV Pnicillin G - 2mu/ 6 hr

  O. oxycilline - 1g/ 6 hr

If symptoms improved within 3 days – convert to oral drugs

  O. Penicilline 500mg/6hr

  Cloxacilline 250mg / 6hr – 2/3 weeks

- Second choice

  O. Clindamycine 600mg/ 8 hr – 2/3 weeks

- Third choice

  O. Cefazolin 500 mg/ 8 hr

- Fourth choice

  O. Erythromycine 2g /6hr

ACUTE SUPPURATIVE OSTEOMYELITIS

CLINICAL FEATURES

EARLY :

- Severe throbbing, deep- seated pain.
- Swelling due to inflammatory edema.
- Gingiva appears red, swollen & tender.

LATE :

- Distension of periosteum with pus.

FINAL:

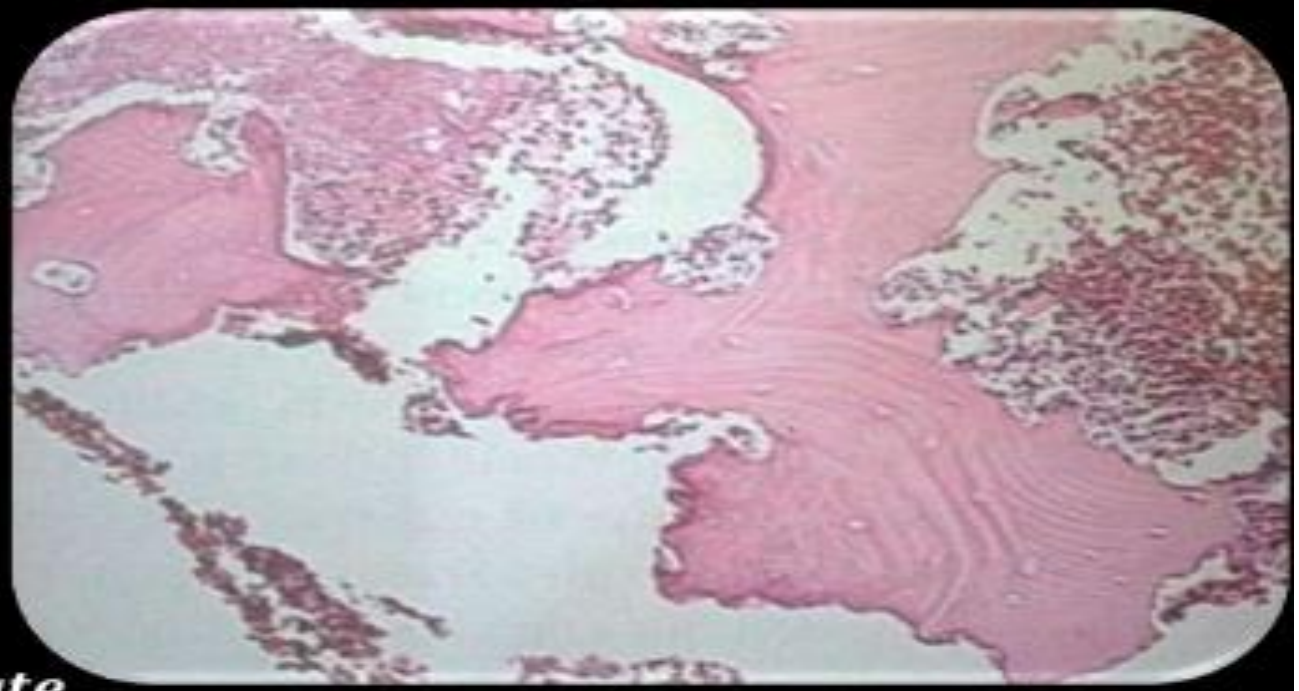
- Subperiosteal bone formation cause swelling to become firm.

ACUTE SUPPURATIVE OSTEOMYELITIS

HISTOLOGY

- *Submitted material for biopsy predominantly consists of necrotic bone & is diagnosed as sequestrum*

- *Bone shows:*
  - *Loss of osteocytes from lacunae.*
  - *Peripheral resorption.*
  - *Bacterial colonization.*
  - *Acute inflammatory infiltrate consisting of polymorphonuclear leukocytes in haversian canals & peripheral bone.*



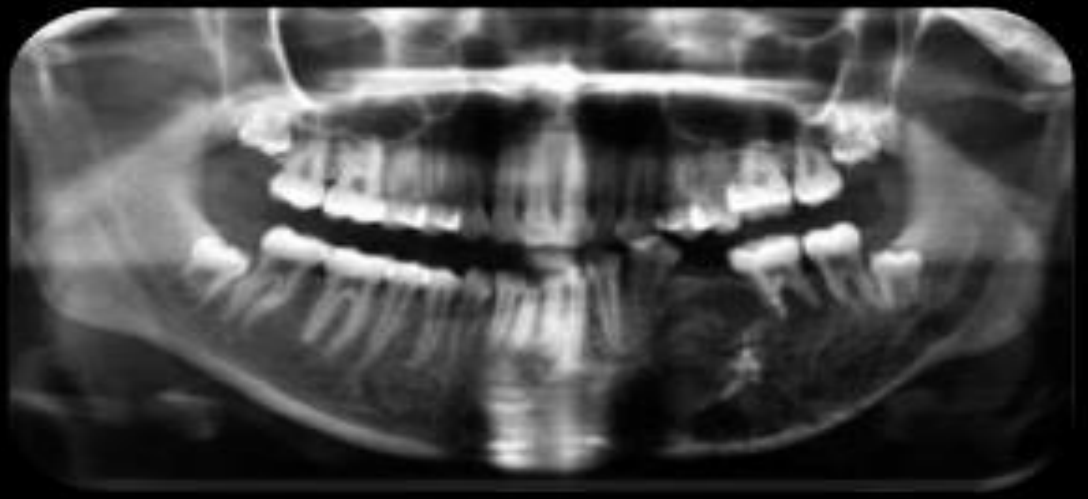
ACUTE SUPPURATIVE OSTEOMYELITIS

RADIOGRAPHIC FEATURES

- *May be normal in early stages of disease .*
- *Do not appear until after at least 10 days.*

Radiograph may demonstrate ill-defined radiolucency.

After sufficient bone resorption irregular, mot-eaten areas of radiolucency may appear.



ACUTE SUPPURATIVE OSTEOMYELITIS

MANAGEMENT

ESSENTIAL MEASURES

- Bacterial sampling & culture.
- Emperical antibiotic treatment.
- Drainage.
- Analgesics.
- Specific antibiotics based on culture & sensitivity.
- Debridement.
- Remove source of infection, if possible.

ADJUNCTIVE TREATMENT

- Sequestrectomy.
- Decortication (if necessary)
- Hyperbaric oxygen.
- Resection & reconstruction for extensive bone destruction.

ACUTE SUPPURATIVE OSTEOMYELITIS

COMPLICATIONS

Rare but include:

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

CHRONIC SUPPURATIVE OSTEOMYELITIS

- Inadequate treatment of acute osteomyelitis
- Periodontal diseases
- Pulpal infections
- Extraction wounds
- Infected fractures

Infection in the medulllary spaces spread and form granulation tissue

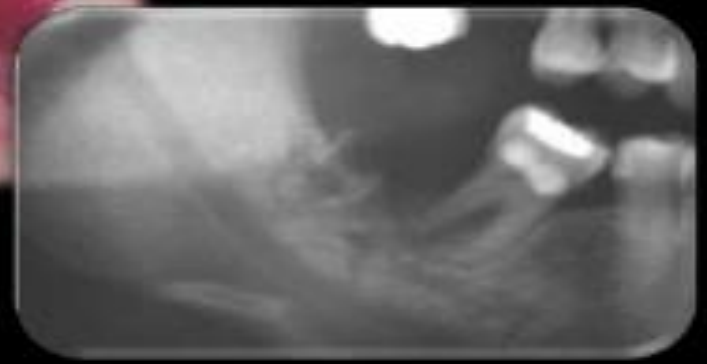
Granulation tissue forms dense scar to wall off the infected area

Encircled dead space acts as a reserviour for bacteria & antibiotics have great difficulty reaching the site

CHRONIC SUPPURATIVE OSTEOMYELITIS

CLINICAL FEATURES

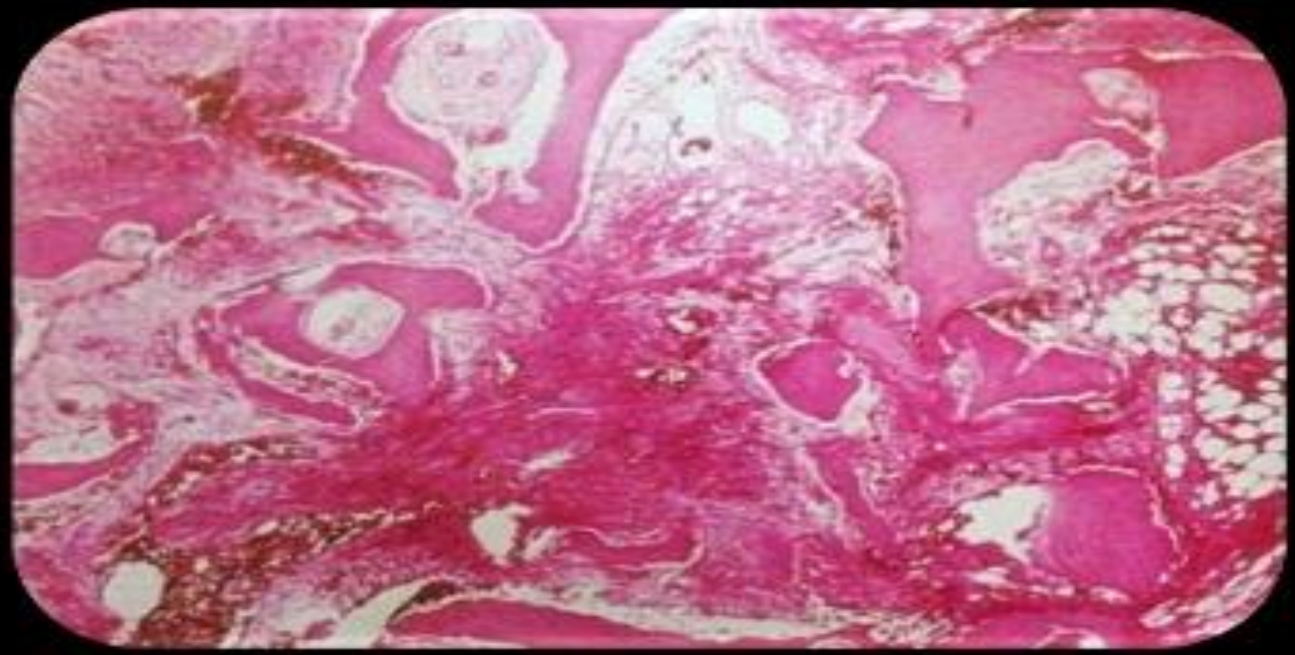
- Swelling
- Pain
- Sinus formation
- Purulent discharge
- Sequestrum formation
- Tooth loss
- Pathologic fracture



CHRONIC SUPPURATIVE OSTEOMYELITIS

HISTOLOGY

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



CHRONIC SUPPURATIVE OSTEOMYELITIS

RADIOLOGY

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

- *Sequestra lying close to the peripheral sclerosis & lower border.*
- *New bone formation is evident below lower border.*

CHRONIC SUPPURATIVE OSTEOMYELITIS

MANAGEMENT

- 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.

FOCAL SCLEROSING OSTEOMYELITIS

- *Also known as "Condensing osteitis".*
- *Localized areas of bone sclerosis.*
- *Bony reaction to low-grade peri-apical infection or unusually strong host defensive response.*
- *Association with an area of inflammation is critical.*

FOCAL SCLEROSING OSTEOMYELITIS

CLINICAL FEATURES

- *Children & young adults are affected.*
- *In mandible, premolar & molar regions are affected.*
- *Bone sclerosis is associated with non-vital or pulpitic tooth.*
- *No expansion of the jaw.*

HISTOLOGY

- *Dense sclerotic bone.*
- *Scanty connective tissue.*
- *Inflammatory cells.*

FOCAL SCLEROSING OSTEOMYELITIS

RADIOLOGY

- *Localized but uniform increased radiodensity related to tooth.*
- *Widened periodontal ligament space or peri-apical area.*
- *Sometimes an adjacent radiolucent inflammatory lesion may be present.*

Increased areas of radiodensity surrounding apices of nonvital mandibular first molar

FOCAL SCLEROSING OSTEOMYELITIS

MANAGEMENT

- *Elimination of the source of inflammation by extraction or endodontic treatment.*
- *If lesion persists and periodontal membrane remains wide, reevaluation of endodontic therapy is considered.*
- *After resolution of lesion, inflammatory focus is termed as bone scar.*

DIFFUSE SCLEROSING OSTEOMYELITIS

- *It is an ill-defined, highly controversial, evolving area of dental medicine.*
- *Exact etiology is unknown.*
- *Chronic intraosseous bacterial infection creates a smoldering mass of chronically inflammed granulation tissue.*

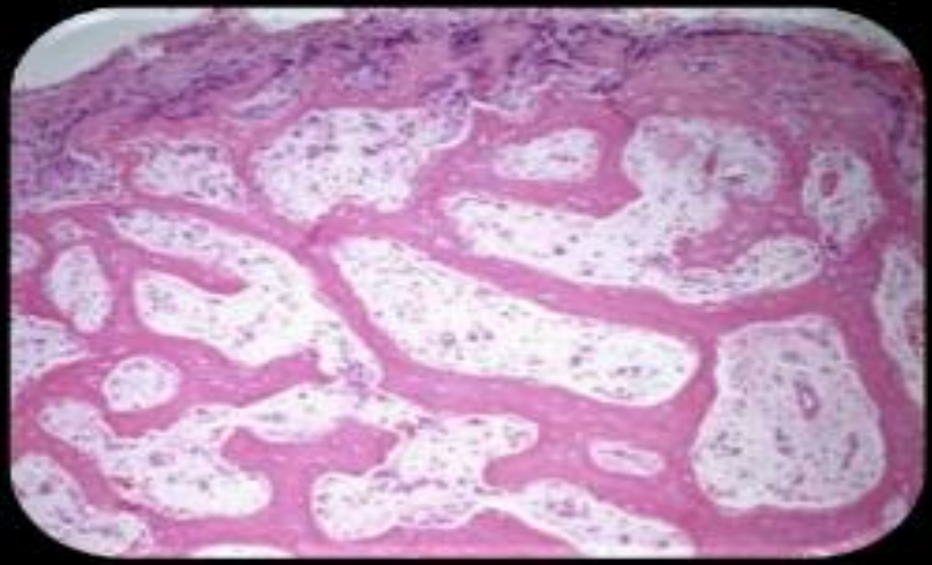
DIFFUSE SCLEROSING OSTEOMYELITIS

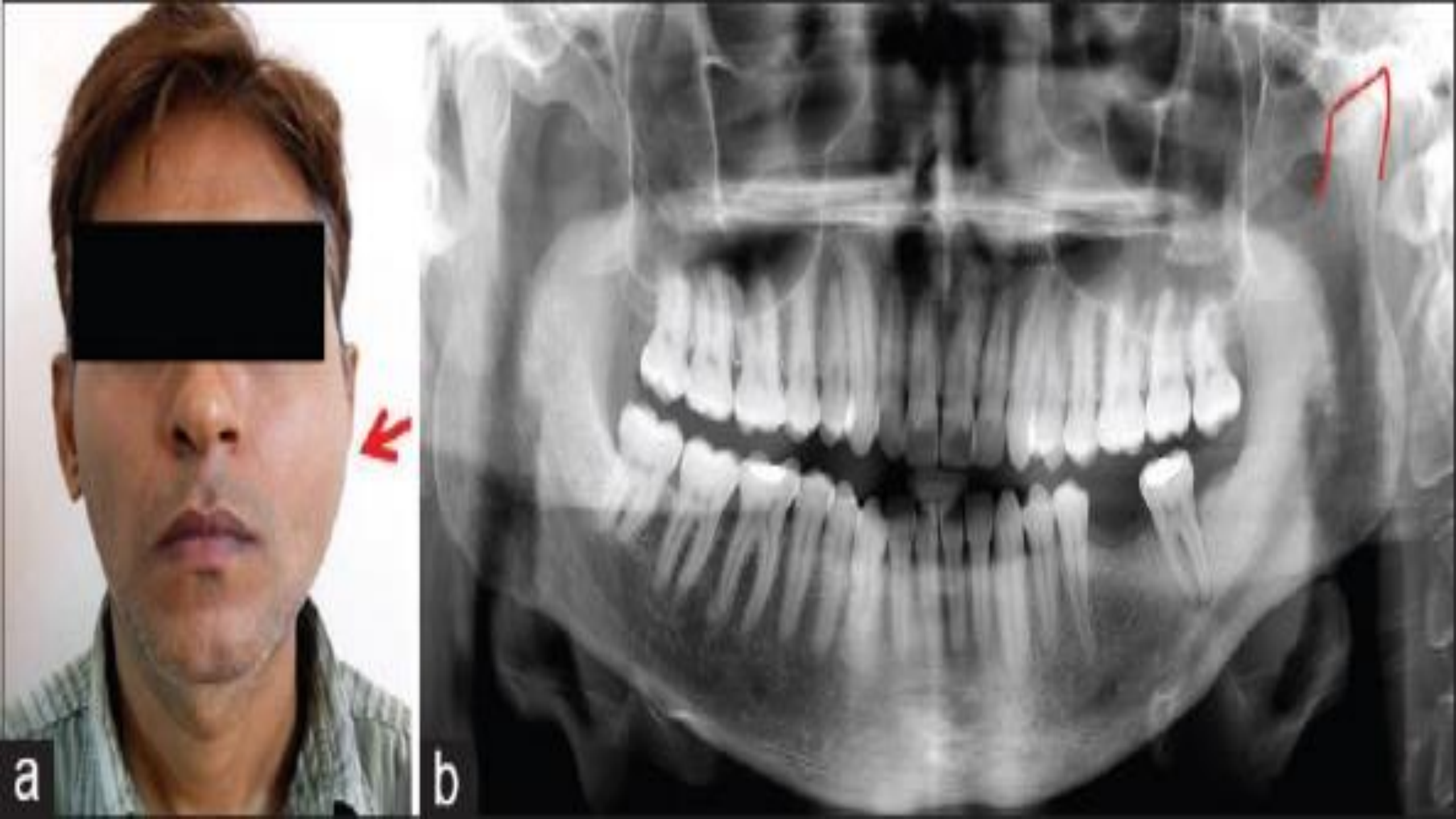
CLINICAL FEATURES

- *Arises exclusively in adult-hood with no sex pre-dominance.*
- *Primarily occurs in mandible.*
- *No pain.*
- *No swelling.*

HISTOLOGY

- *Bone sclerosis and remodling.*
- *Scanty marrow spaces.*
- *Necrotic bone separates from vital bone & become surrounded by granulation tissue.*
- *Secondary bacterial colonization often is visible.*

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DIFFUSE SCLEROSING OSTEOMYELITIS

RADIOLOGY

Increased radiodensity may be seen surrounding areas of lesion.

Diffuse area of increased radiodensity of Rt. Side of mandible

PROLIFERATIVE PERIOSTITIS

- *Also known as " Periostitis ossificans" & "Garee's osteomyelitis".*
- *It represents a periosteal reaction to the presence of inflammation.*
- *Affected periosteum forms several rows of reactive vital bone that parallel each other & expand surface of altered bone.*

PATHOGENESIS

The spread of low-grade, chronic apical inflammation through cortical bone

↓

Periosteal reaction occurs

↓

Stimulates proliferative reaction of periosteum

PROLIFERATIVE PERIOSTITIS

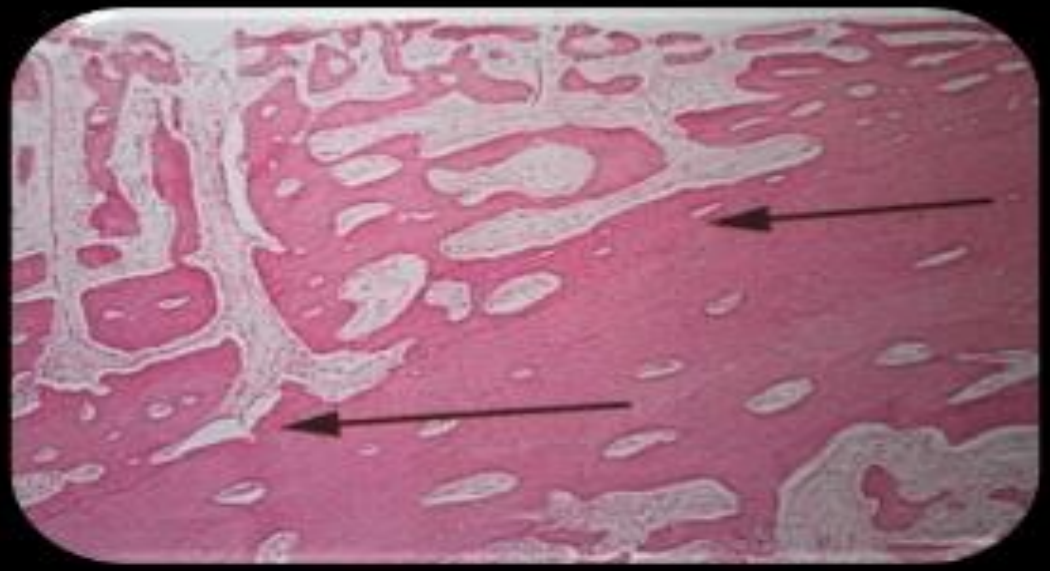
CLINICAL FEATURES

- *Affected patients are primarily children & young adults.*
- *Incidence is mean age of 13 years.*
- *No sex predominance is noted.*
- *Most cases arise in the premolar & molar area of mandible.*
- *Hyperplasia is located most commonly along lower border of mandible.*
- *Most cases are uni-focal, multiple quadrants may be affected.*

PROLIFERATIVE PERIOSTITIS

HISTOLOGY

- Parallel rows of highly cellular & reactive woven bone .
- Trabeculae are frequently oriented perpendicular to surface.
- Trabeculae sometimes form an interconnecting meshwork of bone.
- Between trabeculae, uninflammed fibrous tissue is evident.



PROLIFERATIVE PERIOSTITIS

RADIOLOGY

- *Radiopaque laminations of bone roughly parallel each other & underlying cortical surface.*
- *Laminations may vary from 1-12 in number.*
- *Radiolucent separations often are present between new bone & original cortex.*

Osteoradionecrosis

Osteoradionecrosis is one of the most serious complications of radiation to the head and neck but is seen less frequently today because of better treatment modalities and prevention. The current prevalence rate is less than 4%, whereas the frequency approached 15% less than 20 years ago. Although the risk is low, it increases dramatically if a local surgical procedure is performed within 21 days of therapy initiation or between 4 and 12 months after therapy. Radiation of bone results in permanent damage:' to thc osteocytes and microvasculature system. The altered bone becomes hypoxic, hypovascular, and hypocellular. Osteoradionecrosis is the result of nonhealing, dead bone; infection is not necessarily present.

- It is a chronic, nonhealing wound caused by ***hypoxia, hypocellularity,*** and ***hypovascularity*** of irradiated tissue.

Clinical features

- Severe , deep , boring pain which may continue for weeks or months .
- Swelling of face when infection develops
- Soft tissue abscesses & persistently draining sinuses .
- Exposed bone ; in association with intraoral or extraoral fistulae .
- Trismus .

Bisphosphonate-Associated Osteonecrosis

A similar type of jaw necrosis may be seen as a complication of bisphosphonate therapy (e.g., pamidronate, zoledronic acid). Bisphosphonates are currently used as part of the treatment regimen for patients with multiple myeloma, metastatic cancers to bone (e.g., breast or prostate cancer), Paget's disease, and osteoporosis because of their inhibitory effect on ostcoclastic bone resorption.

Bisphosphonates, taken for an extended period (greater than 1 year), but the patient at risk for non infectious jaw necrosis.

The typical presenting clinical symptom of bisphosphonate-associated osteonecrosis is pain, and the characteristic sign is bone exposure. The lesion usually follows tooth extraction or other form of jaw surgery, although many cases seem to be spontaneous. As with osteoradionecrosis, the mandible is more commonly affected than the maxilla.

Figura 2

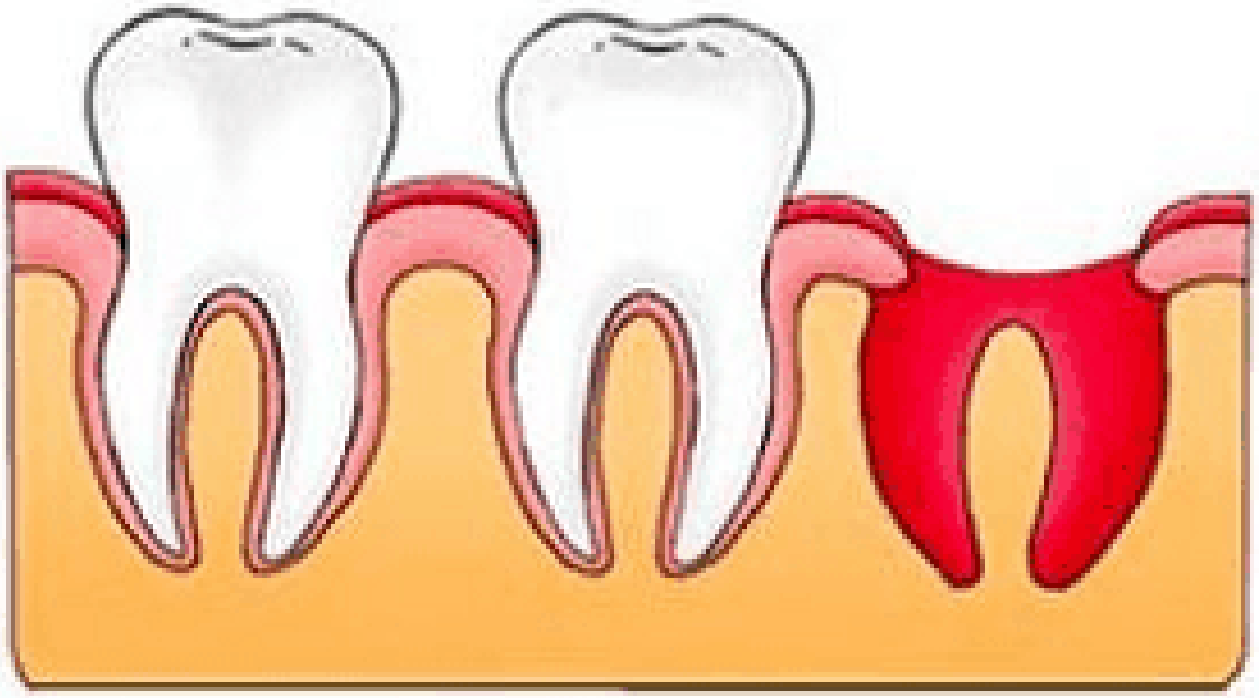
Dry socket

DEFINITION:

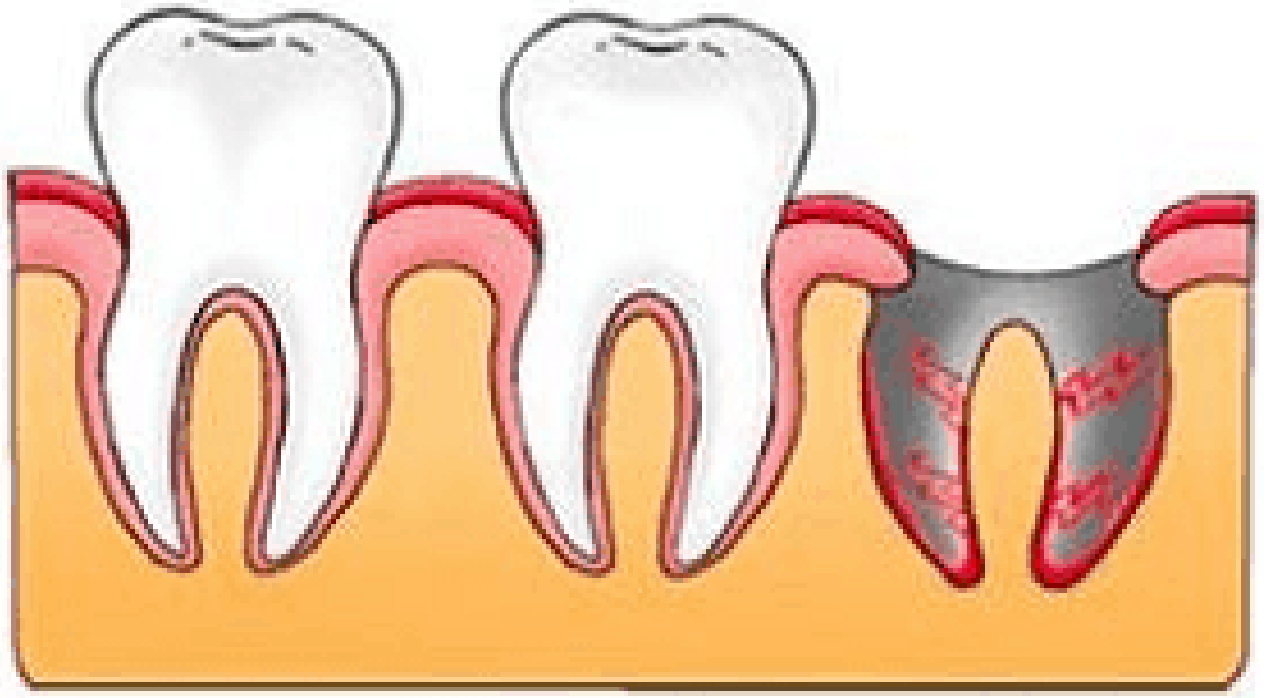
- Dry socket is also called Alveolar Ostitis.
- It refers to the inflammation of the alveolar bone which commonly occurs as a complication of tooth extraction.

AETIOLOGY:

- Smoking.
- Traumatic extraction.
- Poor oral hygiene.
- Patients with history of dry sockets.
- Surgical extraction of wisdom teeth.
- Oral contraceptives.
- Pre-existing infection: (pericoronitis)



FORMATION OF BLOOD CLOT AT TOOTH EXTRACTION SITE TO PROTECT THE BONE AND NERVE ENDINGS



PAINFUL DRY SOCKET OCCURS WHEN BLOOD CLOT IS DISLODGED BEFORE WOUND HAS HEALED

EPIDEMIOLOGY

- A dry socket will occur in only 1% to 3% of all tooth extraction.
- But its more common in the extraction of Mandibular Wisdom teeth.
- In lower impacted wisdom teeth, as many as 25% to 30% of cases will result dry socket.

PATHOPHYSIOLOGY:

- After tooth extraction, a blood clot forms on the socket of the extracted tooth, mainly to reduce blood loss due to the extraction and also to prevent the alveolar bone from getting exposed to the oral environment.
- When this blood clot fails to form or is physically lost from the socket, the alveolar bone gets exposed to the oral saliva, bacteria and food debris.
- This results in localized inflammation of the alveolar bone, leading to a severe throbbing pain that radiates to the jaws, ears and eyes of the affected side.

SIGNS AND SYMPTOMS:

Symptoms

- **Severe pain.**
- Onset : 2 – 3 days after extraction.
- Radiation: Pain radiates to the jaws, ears ,eyes and neck of the affected tooth.
- **Intra-oral Odor (Halitosis)**
- **Bad taste in the mouth.**
- **Regional Lymphadenopathy (Rare).**

SIGNS:

- **Empty socket:-**That partially or totally lacks blood clot.
- **Exposed bone:-** Visible and sensitive to touch
- **Inflammation of the soft tissue around.**
- **Food debris trapped in the socket.**