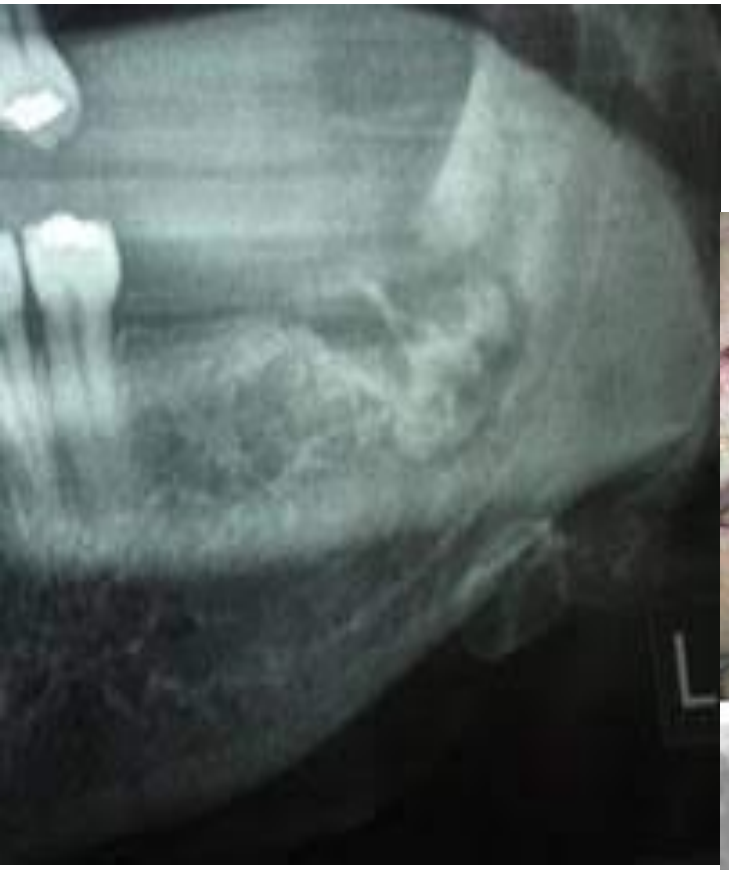
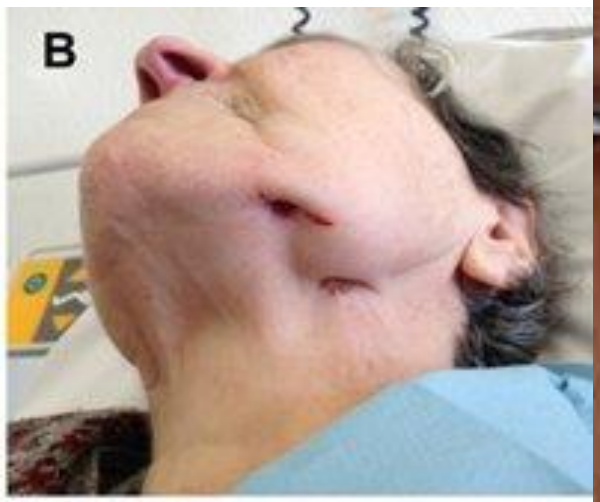
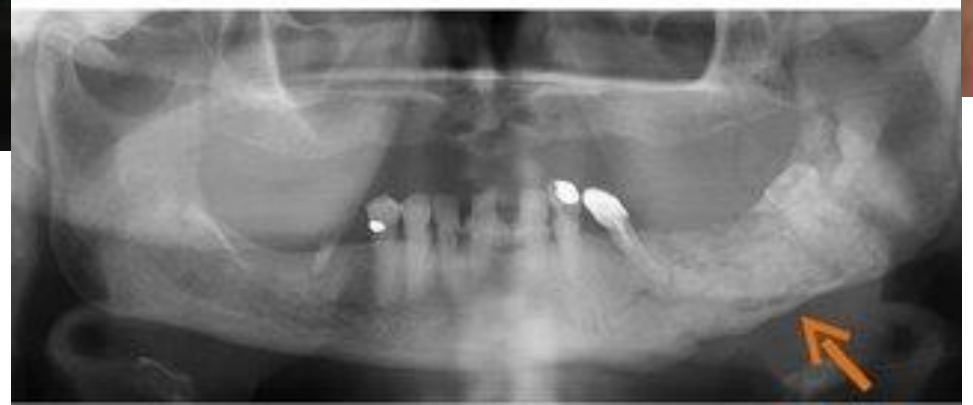
# Osteomyelitis and osteonecrosis of the jaw





Professor Abbas AY Taher

# ***The bone:***

Bones belong to the connective tissue. Their main function is the internal support and source of inorganic ions. Bones have an organic matrix that is secondarily calcified with calcium salts, mainly hydroxyapatite.

The organic matrix consists of a huge extent of type I collagen. Only 5% are other proteoglycans

and non-collagenous proteins .

There are **two major bone parts**: The outer compact and the interior cancellous part. In the compact bone, the collagen fibrils form concentric lamellae around a central canal that is called the Haversian canal. These canals harbor vessels which are interconnected by further vessels lying in the Volkmann's canals. The fibrils in neighboring lamellae have a perpendicular orientation resulting in higher stability of the bone. In between the calcified lamellae are therefore concentric orientated osteocytes. Their main function seems to be the mineralization of the bone .

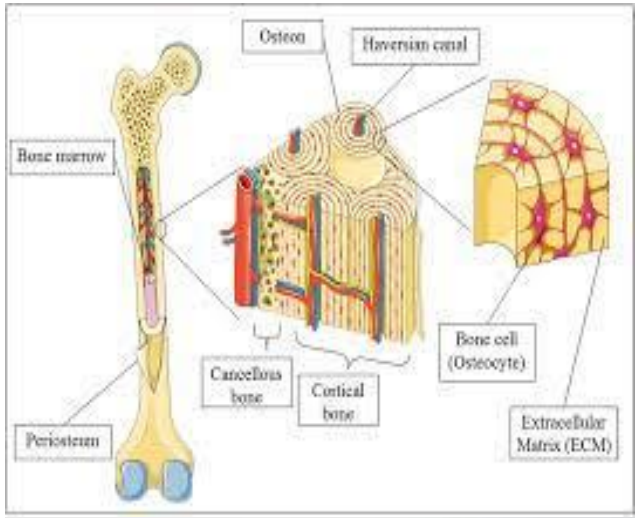
The compact bone is responsible for approximately 80% of the entire bone weight

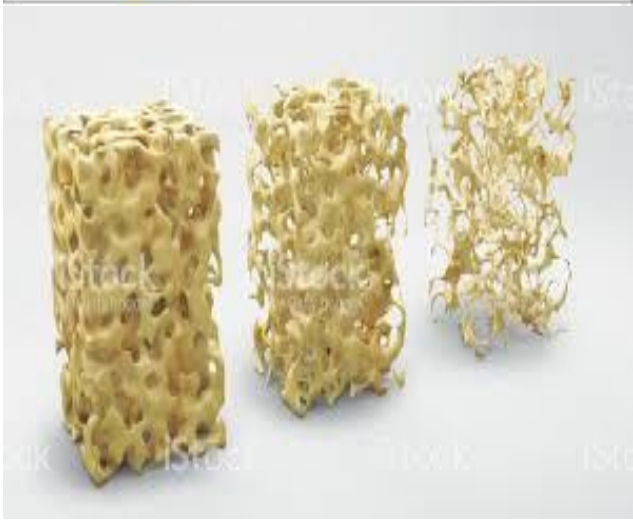
The main function of the compact bone is mechanical stability whereas the cancellous bone mainly has a metabolic function .

The cancellous bone consists of small lamellae and has a surface 10 times bigger than the compact bone .

On the outside of the compact bone is the periosteum, on the inside the endosteum.

There are **three kinds of bone cells**: The osteoblasts, theosteocytes, and the osteoclasts.



## ***Osteoblasts***

Osteoblasts derive from a multipotential stem cell that differentiates via an osteoprogenitor cell into osteoblasts. Osteoblasts form new bone by the production of the inorganic matrix that mineralizes eventually. After a cycle of bone resorption and consecutive bone formation, most osteoblasts become lining cells covering the surface of the bone .

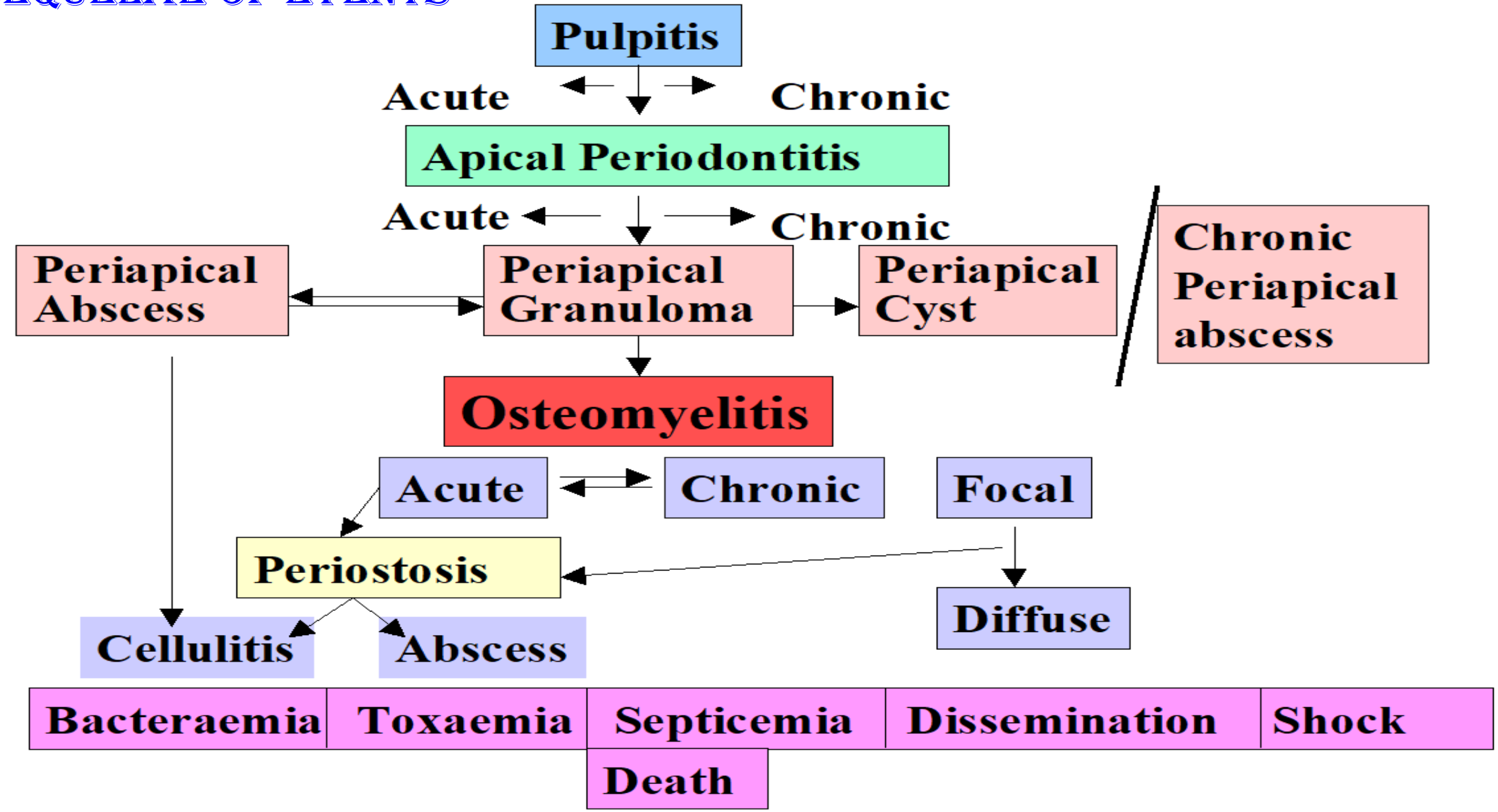
## ***Osteocyte***

During the course of bone formation, approximately 10% of the osteoblasts build themselves into the bony structure and become osteocytes . It is assumed that the number of osteocytes is 10 times higher than the number of osteoblasts in the adult human body. The osteocytes are stellate cells that have lots of slim processes that are connected to surrounding cells. Osteocytes with their three-dimensional network seem to play the key role in bone remodeling

## ***Osteoclast***

From all bone cells osteoclasts represent the smallest fraction. **Osteoclasts** are multinucleated giant cells that resorb bone. They derive from the monocyte macrophage line. Their only function is to resorb mineralized tissue as it is necessary for bone growth, remodeling, and tooth eruption. Most bone diseases are associated with an increased function of the osteoclasts. Therefore, osteoclasts are often the pharmaceutical target in the therapy of bone diseases such as malignancies or metabolic diseases as osteoporosis.

# SEQUELAE OF EVENTS



# 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. That's what differentiate it from dry socket (which is local). Historically, osteomyelitis of the jaws was a common complication of odontogenic infection (infections of the teeth).

***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.**

# Predisposing factors of osteomylitis

## A-LOCAL FACTORS

**Poor oral hygiene , smoking , decreased vascularity/vitality of bone ,trauma. radiation injury. Paget 's disease, oteoporosis. major vessel disease.**

## B- SYSTEMIC FACTORS

***impaired host defense , Immune deficiency states. Immunosuppression, diabetes mellitus, Malnutrition, extremes of age, leukemia, severe anemia, IV- drug abuse, chronic alcoholism, malignancy,autoimmune disease, arthritis, agranulocytosis***

## Classification:

**A-Suppurative osteomyelitis:**

1. Acute suppurative osteomyelitis
2. Chronic suppurative osteomyelitis

C-Osteoradionecrosis /Radio osteomyelitis

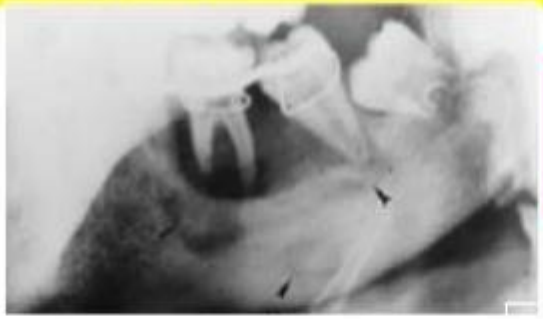
**B-Nonsuppurative osteomyelitis**

1.Chronic focal sclerosing osteomyelitis
2.Chronic diffuse sclerosing osteomyelitis
3.Garre's chronic sclerosing osteomyelitis
4. Proliferative osteomyelitis
5. Rfrectory osteomyelitis

Due to the different classifications and terms used for the entire group of osteomyelitis, it is hard to give general data regarding its epidemiology.

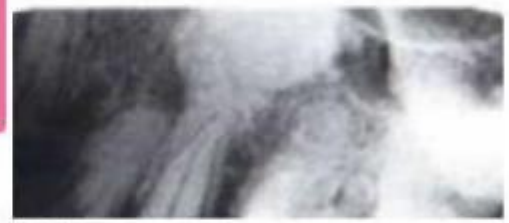
Approximately 17% of all osteomyelitis cases belong to the group of the acute osteomyelitis, 70% to the secondary chronic osteomyelitis, and 10% to the primary chronic osteomyelitis [4]. The average age at the time of diagnosis is a little bit over 40 years for the acute and the secondary chronic osteomyelitis . Because of the inhomogeneity of the secondary chronic osteomyelitis, a general age group cannot be given.

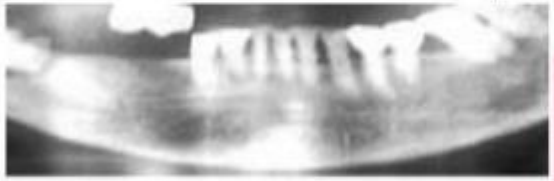
# TYPES OF OSTEOMYELITIS



SUPPURATIVE OSTEOMYELITIS

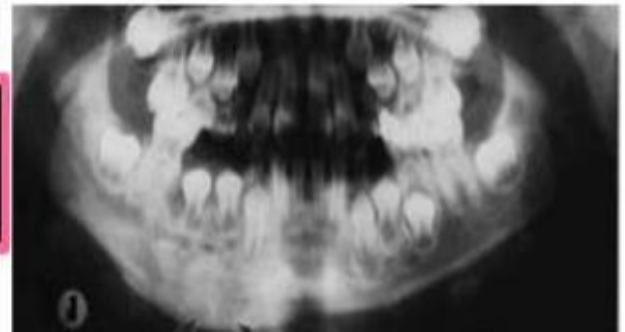
FOCAL SCLEROSING OSTEOMYELITIS



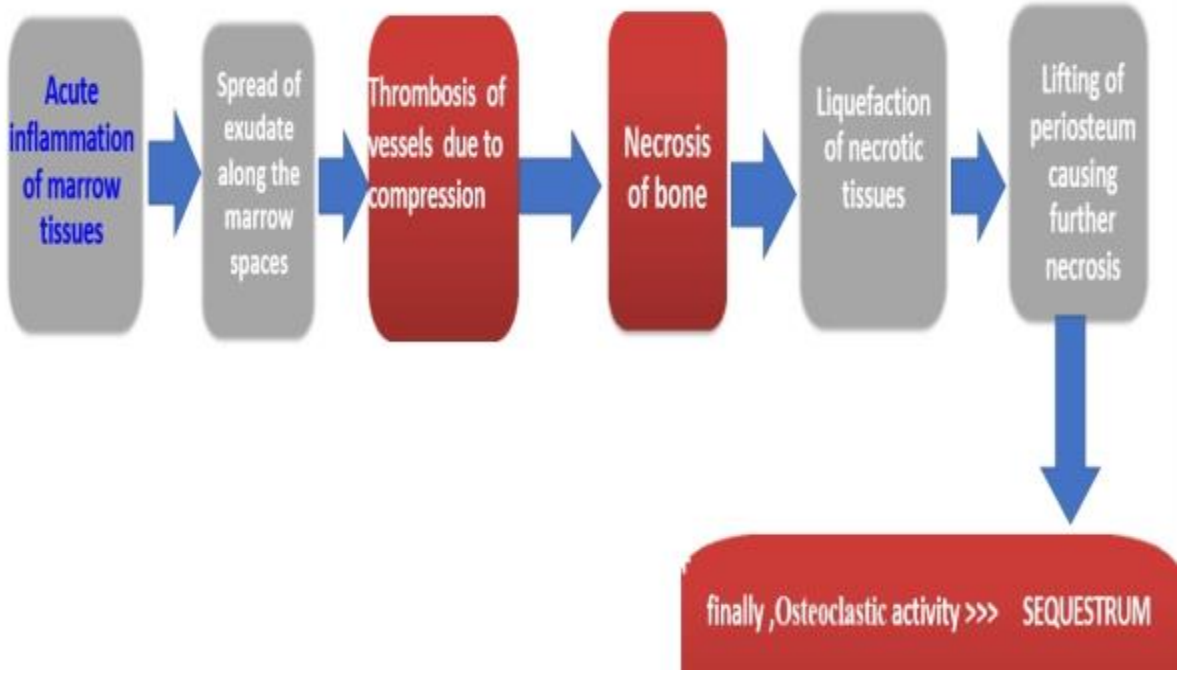


DIFFUSE SCLEROSING OSTEOMYELITIS

PROLIFERATIVE PERIOSTITIS



## Osteomyelitis pathology



## Pathogenesis

The acute osteomyelitis and secondary chronic osteomyelitis are caused by a local infection due to bacteria from the oral cavity. The likelihood of the development of the infection depends on the virulence and number of bacteria and the quality of the local immune response and the blood flow .

Therefore, **general diseases affecting the immune system are risk factors in the development of osteomyelitis**, e.g., diabetes, autoimmune diseases, or anemia. A typical course of the **acute and secondary chronic osteomyelitis** is the contamination of the bone with bacteria. The bacteria proliferate and colonize the bone marrow and reach via the Haversian and Volkmann canals the periosteum. The edema under and in the periosteum disturbs the blood flow resulting in ischemic bone parts and potentially
sequestrum building.

***Inflammatory process of entire bone including cortex & periosteum, not just confined to endosteum***

***Inflammatory condition beginning in medullary cavity & havarsian system & extending to involve periosteum of affected area***

***Local factors decreases the vitality of bone***

***Systemic conditions comprises the defense system of the host***

# Suppurative osteomyelitis

**ONSET OF DISEASE**

**4 WEEKS**

***Acute suppurative osteomyelitis***

***Chronic suppurative osteomyelitis***

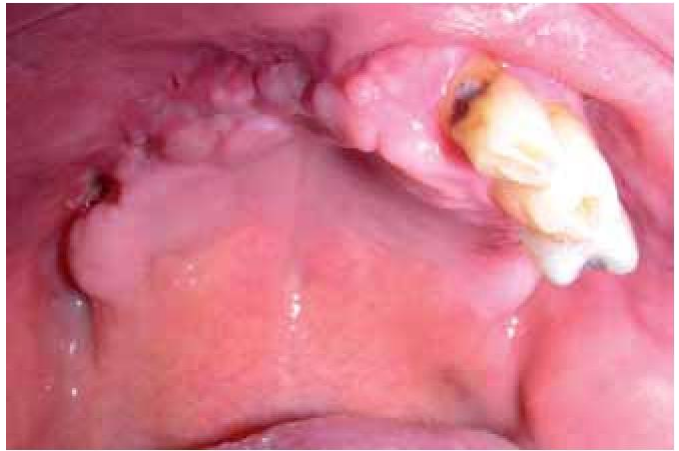
***Onset of disease***
***Deep bacterial invasion into medullary & cortical bone***

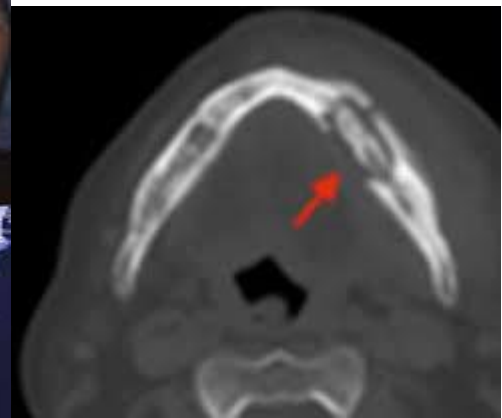
***CHRONIC OSTEOMYELITIS (Suppurative)***

- Follow Acute [failure to respond to therapy or patients with severe immunosupprusive conditions].
- 30 days – 4 months
- Jaw pain, swelling, suppuration
- Less sever than acute, BUT
- Risk of pathological fracture large fistula formation increase.

## *Acute osteomyelitis*

Pain, Trismus, Purulent discharge Fever and Malaise
Change and disturbance in sensation.
By time teeth Loosening., Adenopathy. Fistula formation.



# ACUTE SUPPURATIVE OSTEOMYELITIS

***Organisms entry into the jaw, mostly mandible, compromising the vascular supply***

↓

*Medullary infection spreads through marrow spaces*

↓

*Thrombosis in vessels leading to extensive necrosis of bone*

↓

*Lacunae empty of osteocytes but filled with pus , proliferate in the dead tissue*

↓

*Suppurative inflammation extend through the cortical bone to involve the periosteum*

↓

*Stripping of periosteum comprises blood supply to cortical plate, predispose to rther bone necrosis*

↓

*Sequestrum is formed bathed in pus, separated from surrounding vital bone*

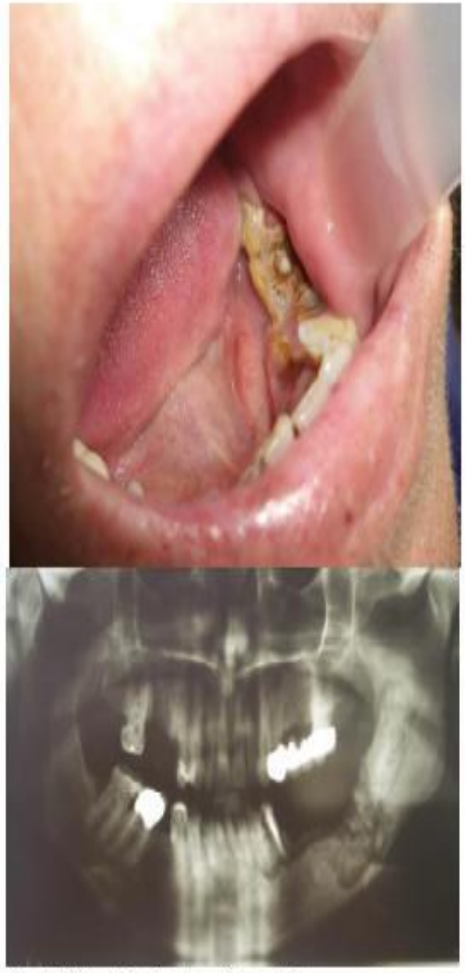
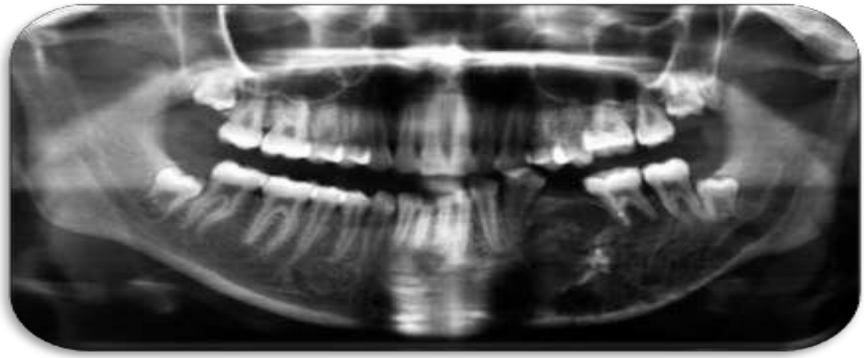


Fig. 1. Pathologic fracture of mandible.

## 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.*



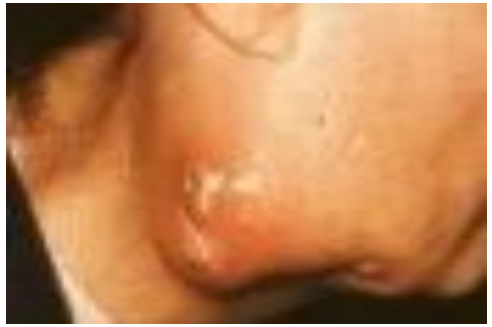
*After sufficient bone resorption irregular, mot-eaten areas of radiolucency may appear.*

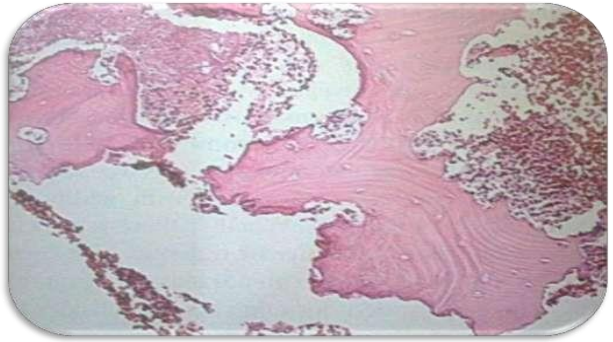
## HISTOLOGY

*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*

# MANAGEMENT

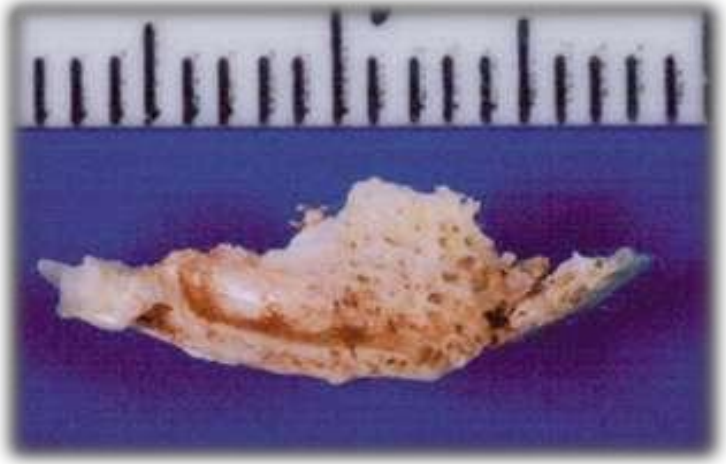
## ESSENTIAL MEASURES

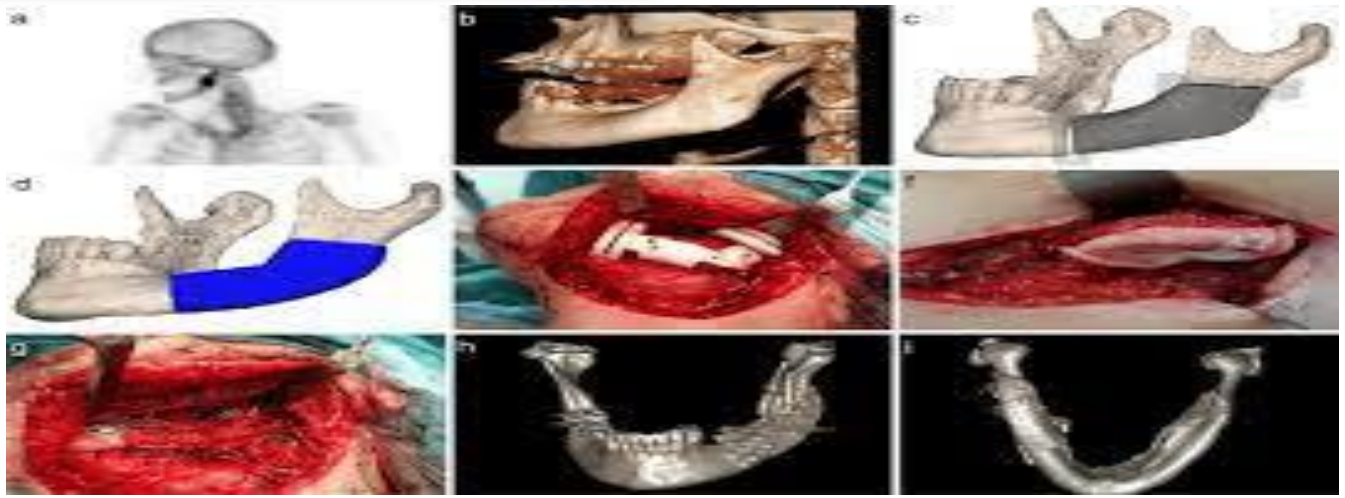
- ***Bacterial sampling & culture, Emperical antibiotic treatment. ,Drainage.***
- ***Analgesics,Specific antibiotics based on culture & sensitivity***
- ***Debridement. ,Remove source of infection, if possible Debridement. Remove source of infection, if possible***

## ADJUNCTIVE TREATMENT

- ***Sequestrectomy, Decortication (if necessary) , Hyperbaric oxygen.***

*,Resection & reconstruction for extensive bone destruction*





## 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 (CSO)***

Chronic suppurative osteomyelitis represents **a progressive inflammatory process caused by pathogens** , resulting in bone **destruction** and **sequestrum formation** . It may present with periods of quiescence of variable duration, whereas its occurrence, **type** , **severity** and **prognosis is multifactorial.**

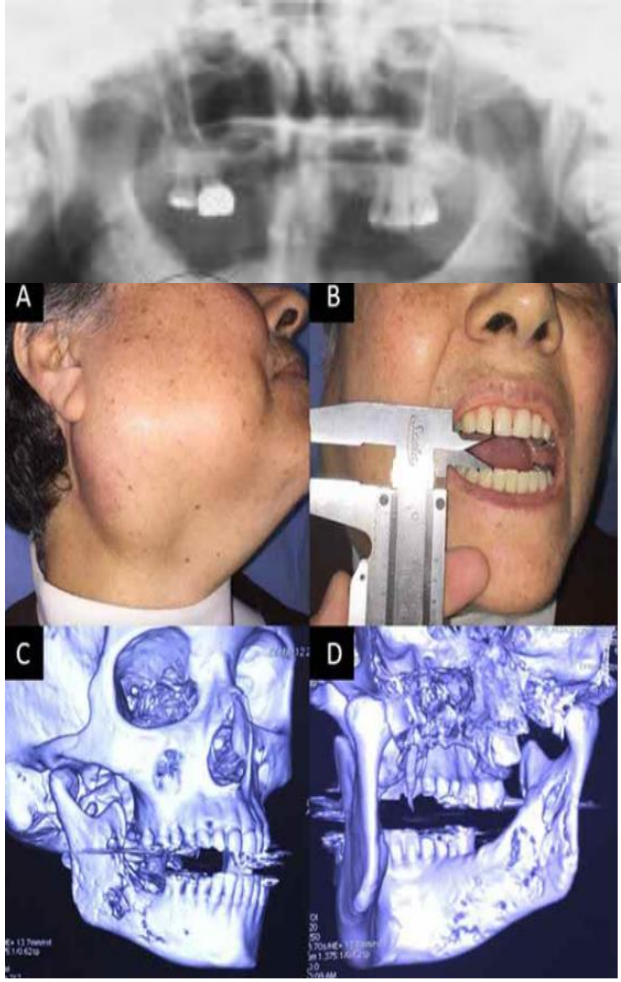
**Chronic suppurative osteomyelitis** may present as a **recurrent** or **intermittent disease.** *Prompt diagnosis and aggressive management of chronic osteomyelitis are critical to the prognosis and final outcome* .

**Chronic suppurative osteomyelitis** is secondary to **direct inoculation of pathogens into the bone at the time of trauma** , as a result of **surgical trauma** (i.e. following open reduction and internal fixation of fractures), from **chronic overlying open wounds or contiguous soft tissue infections** .

**Chronic suppurative osteomyelitis (CSO**) of the maxillofacial region is **primarily caused by infections of odontogenic microorganisms**. It may also arise as a complication of dental extractions, maxillofacial trauma, inadequate treatment of a fracture and irradiation to the mandible.

The ‘gold standard ’ for the **diagnosis of chronic osteomyelitis** is the presence of **positive bone cultures** and **histopathologic examination of the bone** .

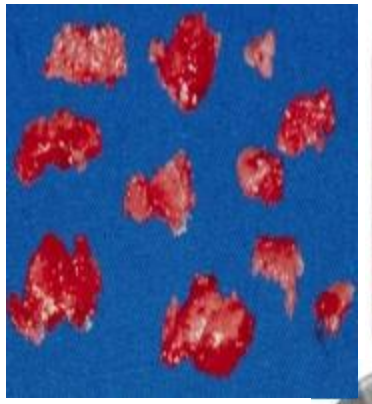
Its management remains challenging to the **treating physician** , with a multidisciplinary approach involving **radiologists, microbiologists** with expertise in **infectious diseases**, OMF surgeons .

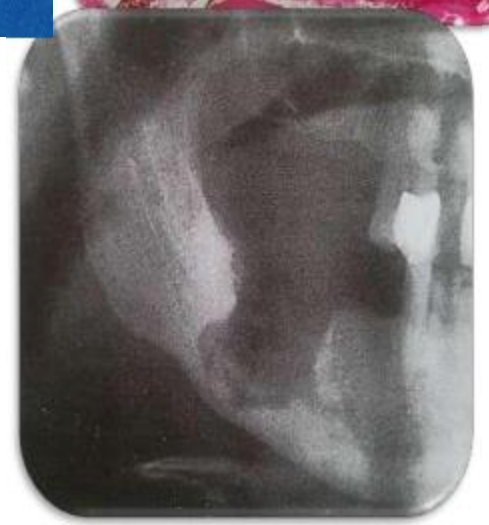



**(A) Side view, where a submandibular increased of volume of indurated consistency and fluctuate can be appreciated. (B) Limited mouth opening due to masseteric contracture, (C and D) CT scan with 3D reconstruction evidence osteolytic lesions in body and right mandibular branch.**

## Rdiograph appearance:

- Patchy, ragged & ill defined radiolucency. *Often contains radiopaque*
- *Sequestra lying close to the peripheral sclerosis & lower border.*
- *New bone formation is*





***HISTOLOGY***

***Inflammed connective tissue filling inter-trabecular areas of bone.***

***.Scattered sequestra. Pockets of abscess.***

# 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.*

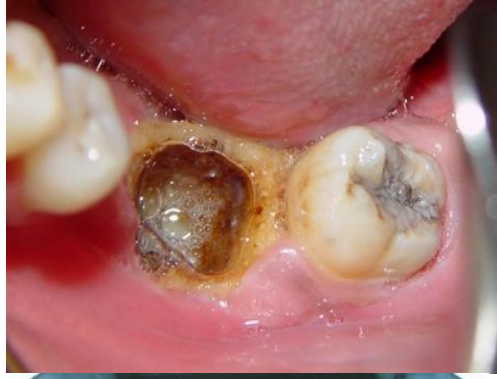
***EXTENSIVE OSTEOMYELITIS***

*Decortication combined with transplantation of cancellous bone chips.*

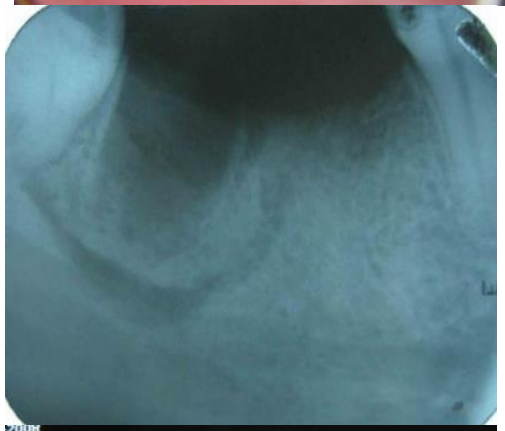
***PERSISTANT OSTEOMYELITIS***

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

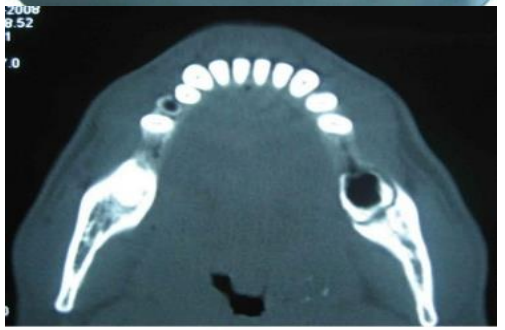
**Treatment:** ***Treatment*** should be tailored to each patient **according the severity** and **duration of symptoms** , as well as to the **clinical and radiological response to treatment**
A combined **antimicrobial** and **surgical treatment** should be considered in all cases, including appropriate **dead space management** and **subsequent reconstruction**.
•Relapse can occur, even following an **apparently successful treatment**, which has a major impact on the quality of life of patients and is a substantial financial burden to any healthcare system.



***The nonsuppurative osteomyelitis*** is characterized by the absence of the formation of **pus**, **fistula**, and sequestration. These forms of **osteomyelitis of the jaw include osteoradionecrosis**, **bisphosphonate-related osteonecrosis of the jaws (BRONJ)** , **chronic recurrent multifocal osteomyelitis of children**, and **chronic sclerosing osteomyelitis**.



Diffuse Sclerosing Osteomyelitis (DSO) is a radiographic term that has been used to describe the radiographic pattern associated with both PCO (Primary Chronic Osteomyelitis )and CSO (chronic sclerosing osteomyelitis).



Chronic recurrent multifocal osteomyelitis (CRMO) is a **nonautoimmune disorder** that mostly affects children and is characterized by **periods of exacerbations** and **remissions over many years .**
It causes **periodic bone pain**, fever, and the appearance of **multiple bone lesions** that can occur in any skeletal site.

**SAPHO syndrome(**Synovitis, acne, pustulosis, hyperostosis, osteitis )is the adult version of **Chronic recurrent multifocal osteomyelitis(CRMO),. CRMO**, which is associated with **synovitis**, **acne**, **pustulosis**, hyperostosis, and **osteitis**

**Primary Chronic Osteomyelitis (PCO)** is a **nonodontogenic** and **nonsuppurative chronic inflammatory** condition of **unknown origin** . It is highly possible association between **primary chronic osteomyelitis(PCO)** of the jaw and other syndromes, such as **CRMO.** The aetiology of PCO remains uncertain and theories include **bacterial infection, autoimmune response** , and **vascular insufficiency due to localized end arteries, especially in the mandible** . There is increasing evidence for the theory that PCO is genetically driven . The importance of autoinflammatory response (interleukin-10, IL-10) in the development of PCO has been discussed by several authors. An impaired gene expression of IL-10 with subsequent disruption of the anti-inflammatory balance might explain part of the clinical presentation of PCO.

## *Radiological Analysis and Findings*

Several imaging modalities have been used for diagnostic of **PCO**, including panoramic radiograph, CT scan, CBCT scan, MRI, and skeletal scintigraphy.

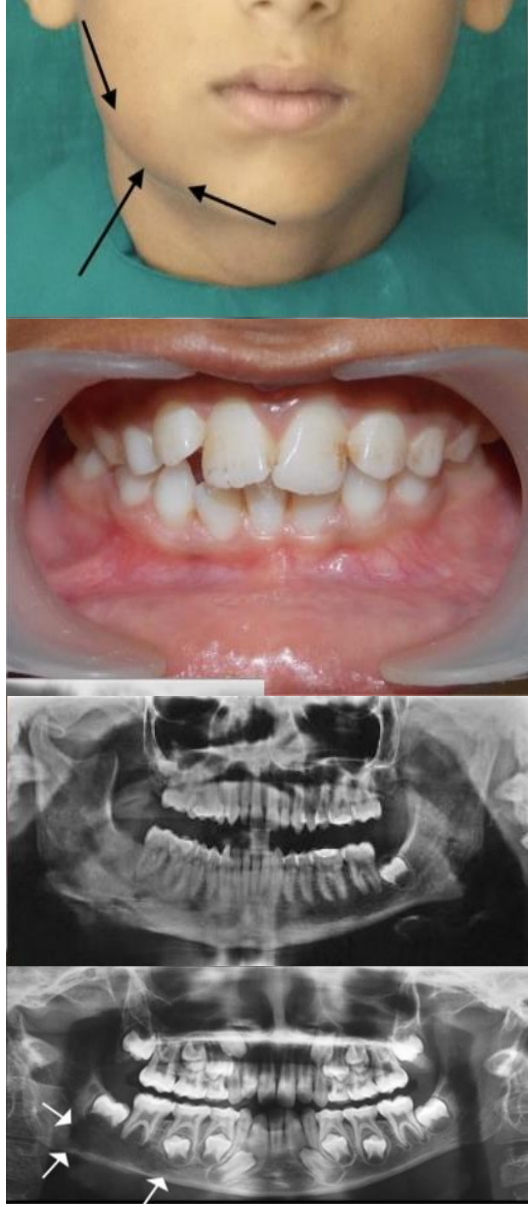
Radiological examination reveals the **presence of radiolucent areas** , **bony sequester**, and **laminations of periosteal new bone .**

## *DSO(Diffuse sclerosing osteomyelitis ).*

•Sclerosing osteomyelitis has traditionally been divided into **focal** and **diffuse** types:

•**The focal type** , also known as **condensing osteitis**, describes a relatively common, limited, well circumscribed radiopaque alteration of bone **surrounding the apex** of a root of a tooth, the latter often with an inflamed or **necrotic dental pulp**

•**Diffuse sclerosing osteomyelitis**, on the other hand, is an uncommon condition affecting a large portion of the bone, usually mandible, and is associated with mandibular expansile remodeling and clinical symptoms



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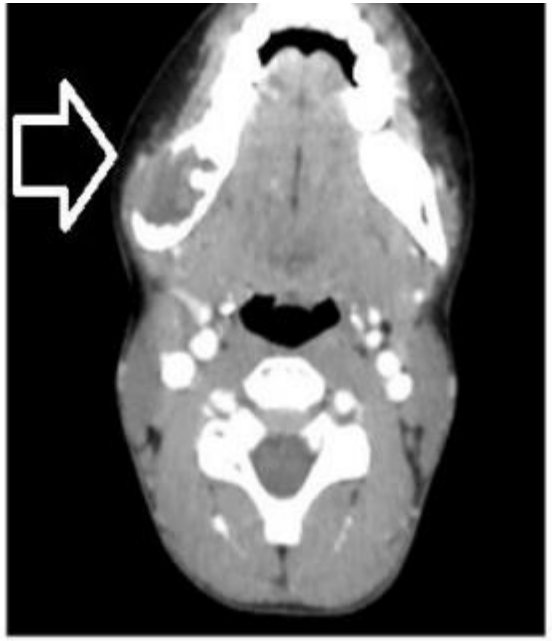
contrast-enhanced computed tomography (CECT).)a, b) showed lytic lesion with expansion of buccal cortex and erosion of outer table at angle of mandible and abnormal enhancing soft tissue within the lytic cavity without showing any periosteal reaction giving an impression of Ameloblastoma.
a Axial CECT image showing lytic lesion with expansion of buccal cortex and erosion of outer table at angle of mandible and abnormal enhancing soft tissue within the lytic cavity without showing any periosteal reaction. b Coronal CECT image showing the center of the lytic lesion



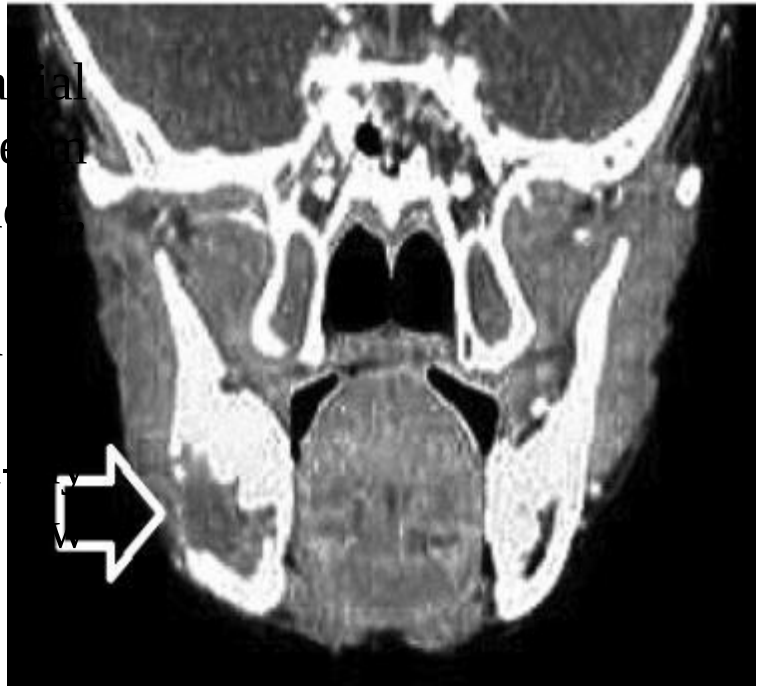
Although **panoramic images** provide an excellent overview of the facial skeleton and teeth, the reliability of measurements has been under criticism. Panoramic images can vary widely as they depend on both position of the patient and the operator .

**Computed tomography** is increasingly used in evaluating osseous pathology in the maxillofacial skeleton. It is an excellent tool for assessing the relative distribution of cortical and cancellous bone and can assist in identifying appropriate location for bone biopsy.

The typical appearance of osteomyelitis of the jaw on CT scan images is that the axial slices can reveal a thickening of the bone with strong periosteal reaction. Cone beam computed tomography (CBCT) technique can be used to ensure a low radiation dose, especially in children .

MRI examination generates no ionizing radiation,it shows both bone reactions and possibly involvement of the adjacent soft tissues .

MRI may even show the extent of the lesions before reactions are seen in X-ray images. MRI weighted images are the best as the pathologic process creates low signal intensity in the normally bright signal of fat contained in the marrow.



# Treatment:

Initially, antibiotics are often used empirically to prevent any bacterial invasion in **acute** and **secondary chronic osteomyelitis**. However, chronic infection remains an **unproven theory** for primary chronic osteomyelitis. **Nonsteroidal anti-inflammatory drugs** and **corticosteroids** are reported to have beneficial effects in reducing symptoms such as extraoral **swelling and trismus**

The importance of **decortication and removal of necrotic bone tissue in primary chronic osteomyelitis of the** mandible have been discussed by several authors .

Few cases have been reported regarding the **treatment of primary chronic osteomyelitis** of the mandibular condyle in children.

Surgical intervention of condyle is a possible method in adults. However, in children, this may cause a disturbance of the mandible growth .

It has been suggested that PCO is genetically driven and this condition acts on the basis of an autoinflammatory respons.

**Nonsteroidal anti-inflammatory** drugs and **corticosteroids** were also used as **first-line options** in other **autoinflammatory bone diseases** including **familial chronic multifocal osteomyelitis** which is also referred to as **Majeed syndrome**, **sporadic chronic recurrent multifocal osteomyelitis** (CRMO), and **Synovitis, Acne, Pustulosis, Hyperostosis, and Osteitis (SAPHO) syndrome** .

**Second-line options** include **methotrexate**, **anti-TNF** , **sulfasalazine**, or **bisphosphonate** .

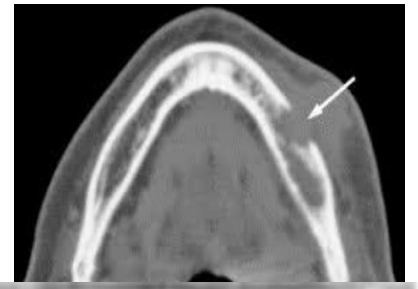
**Bisphosphonate therapy in chronic noninfectious inflammatory bone lesions is based on its anti-inflammatory and antiresorptive properties** . However, because of the side effects of **long-term treatment in children**, these drugs should be used in severe cases and in cases resistant to conventional therapy .

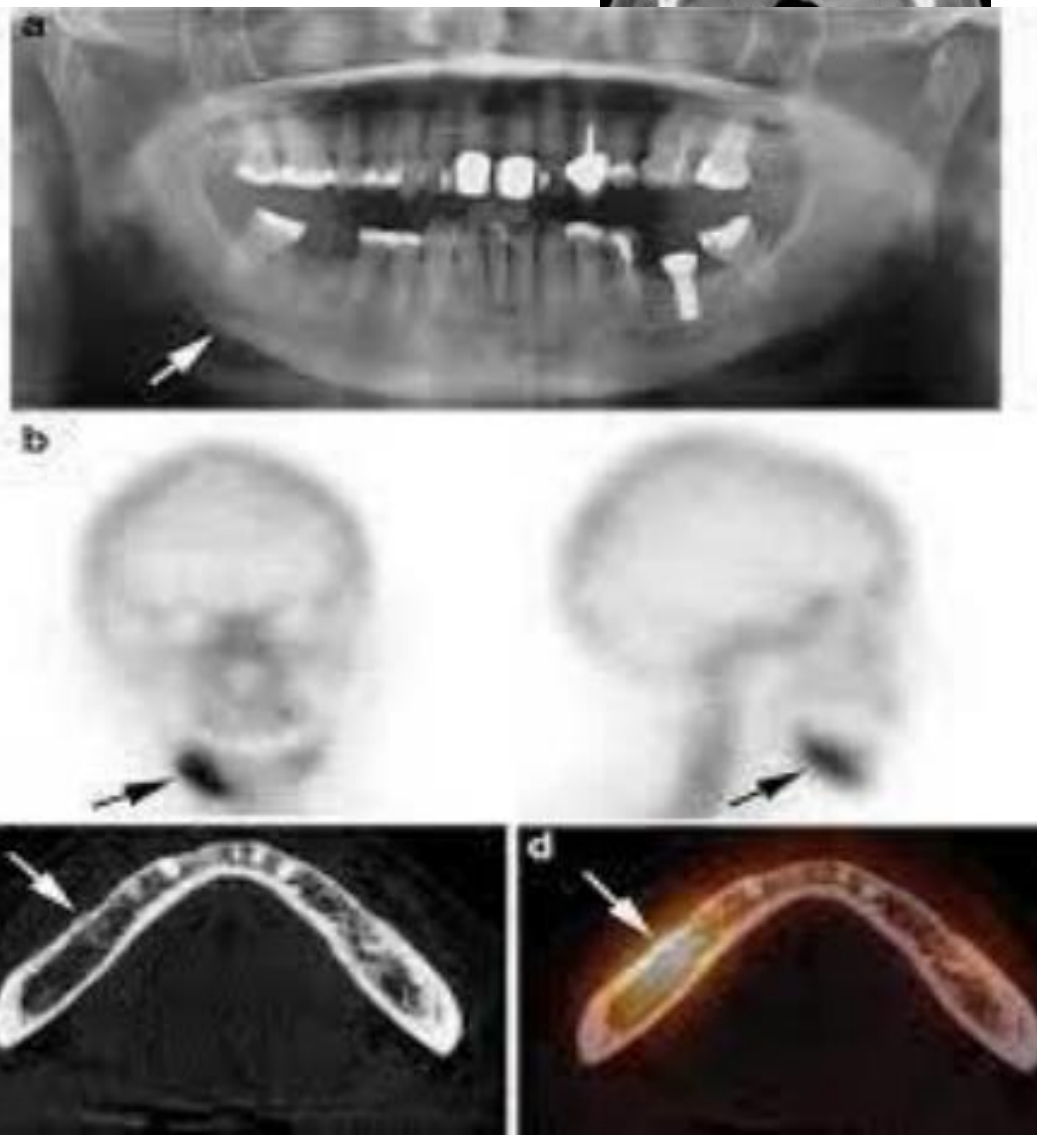
However, MRI examination and waiting time are longer for the patient, and MRIs are not as commonly available in public health care.

Bone scintigraphy is highly sensitive to bone abnormalities and is a useful method especially in patients with suspected or known **inflammatory disease or malignant tumors** .

This radiological method is generally used in combination with other diagnostic imaging techniques, due to low specificity and low spatial resolution.

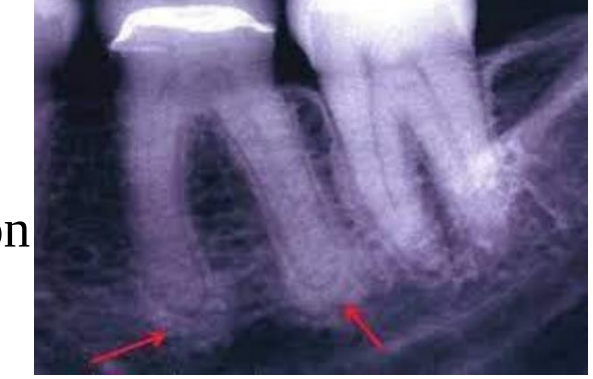
A bone scan image provides a functional display of skeletal activity and is rapidly positive within the **first 24–48 hours after the onset of symptoms** As functional change in bone occurs earlier than structural change, the **bone scan image gives us a hint where the activity is increased and where the next onset of symptoms will occur**





# Chronic Sclerosing Osteomyelitis

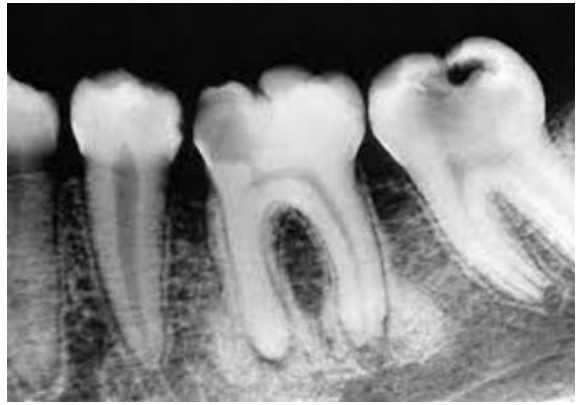
**Focal, Diffuse**



**Chronic focal sclerosing osteomyelitis** is a periapical lesion that involves reactive osteogenesis evoked by chronic inflammation of the **dental pulp**. In most cases, this lesion develops in the **mandibular molar region** in response to a **low-grade infection** of the pulp that results from a deep carious lesion. The disease are two types ,focal and diffuse

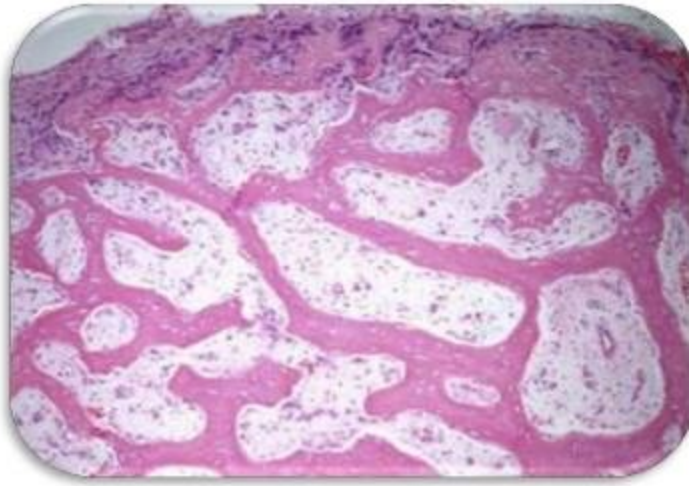
***Clinical features:***

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



## *RADIOLOGY*

Typical appearance of a tooth with chronic focal sclerosing osteitis (CFSO) or condensing osteitis around the root ends (red arrows). While this tooth responded to pulp sensibility testing, pulpal degeneration is obvious



## *HISTOLOGY*

***Bone sclerosis and remodling. Scanty marrow spaces.***

***Necrotic bone separates from vital bone & become surrounded by granulation tissue.***

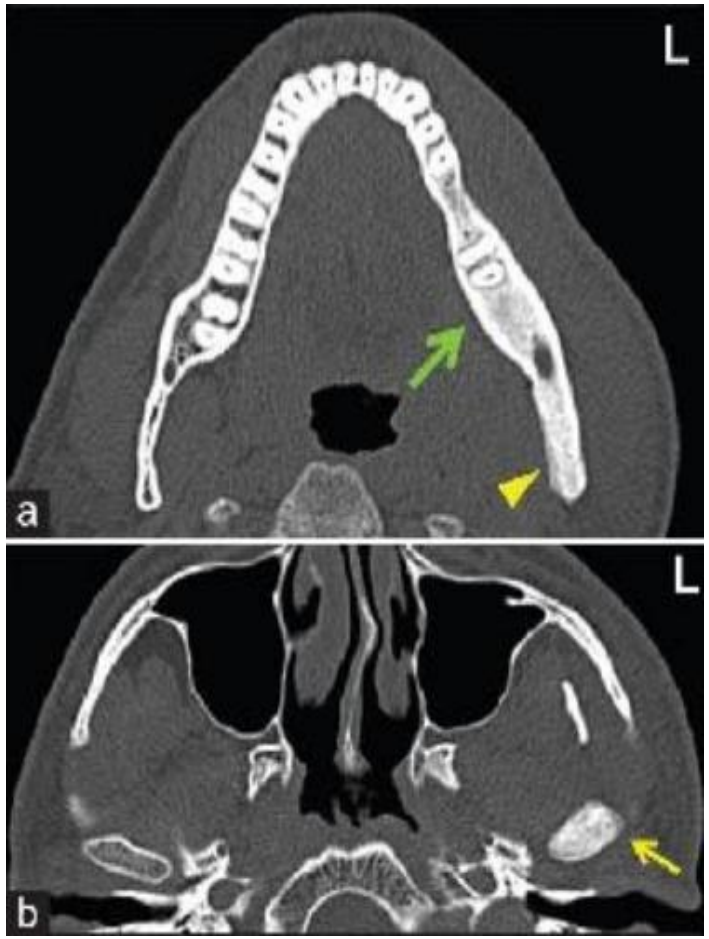
***Secondary bacterial colonization often is visible.***

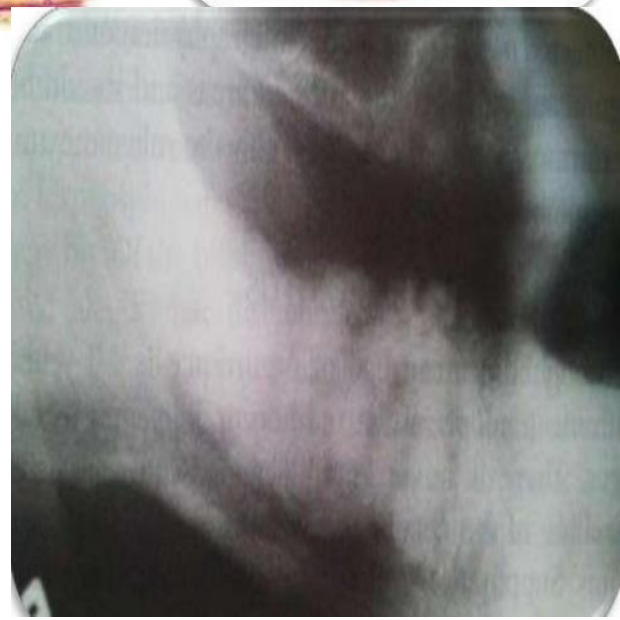
# ***MANAGEMENT***

***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.***

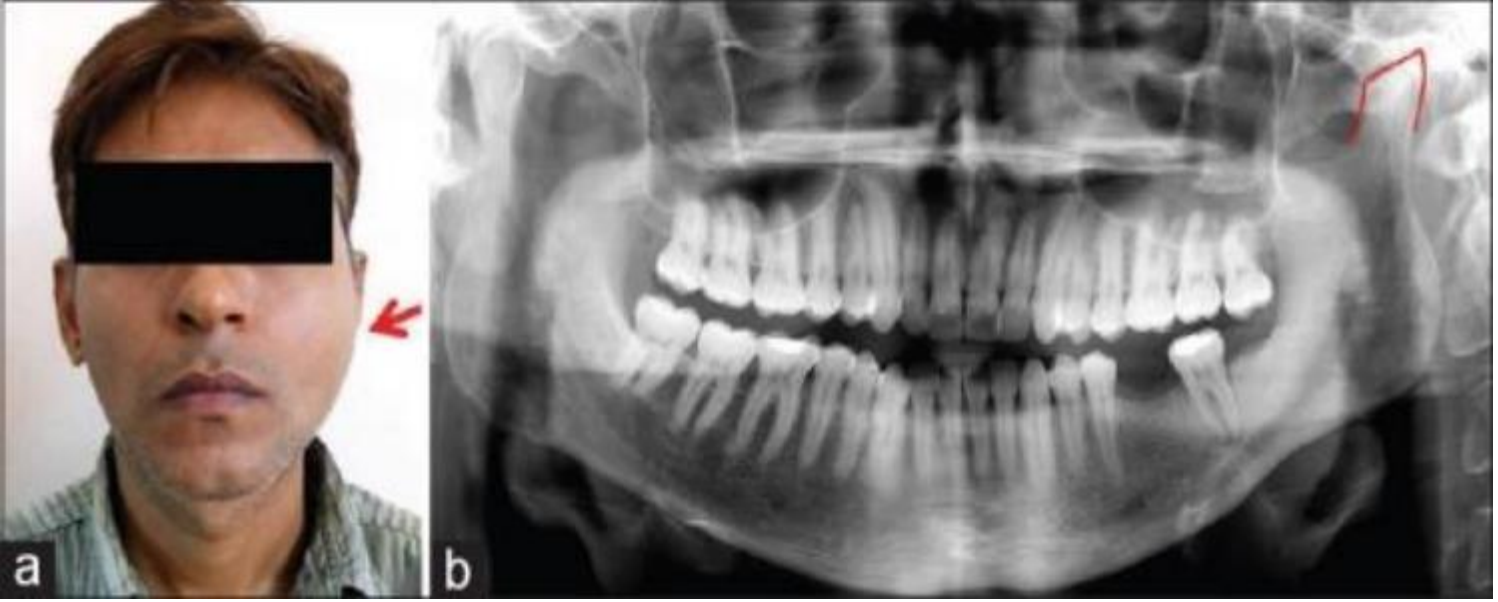
*Diffuse sclerosing osteomyelitis of the jaws* is an **inflammatory condition** that is a consequence of **vascular changes** and **nonspecific bacterial infection** in which **bone deposition** rather than bone resorption occur.This disease presents with unusual clinicoradiographic picture creating **diagnostic** and **therapeutic problems**. Involvement of the **temporomandibular joint** is not frequently observed in **DSO. DSO** commonly affects the **mandibular body**; however, the present case demonstrated radiographic evidence of **endosteal sclerosis extending** up to the condylar head, leading to significant resorption.



***Increased radiodensity may be seen surrounding areas of lesion.***

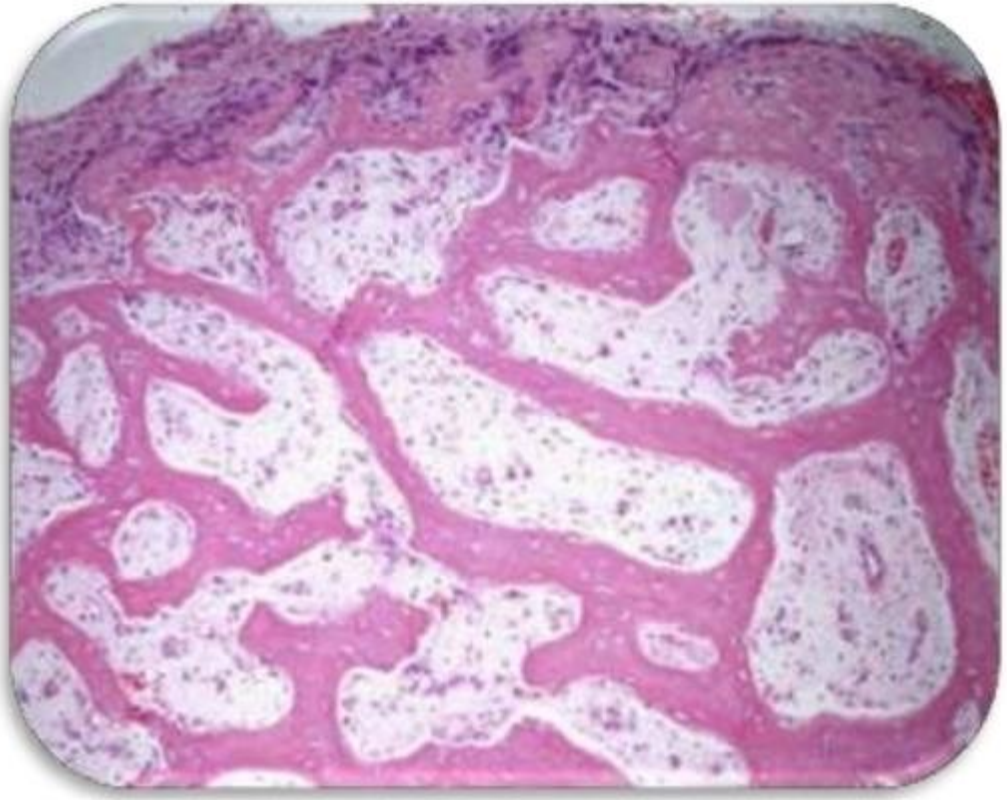
***Diffuse area of increased radiodensity of Rt. Side of mandible***




**(a) Frontal view of a 37-year-old male with unilateral swelling over the left masseter region (arrow) extending superoinferiorly from the level of the zygomatic arch to the inferior border of the mandible and anteroposteriorly extending from the malar region to the tragus. (b) Panoramic radiograph demonstrating unilateral homogeneous sclerosis (ground-glass appearance) of the left hemimandible**

## ***HISTOLOGY***

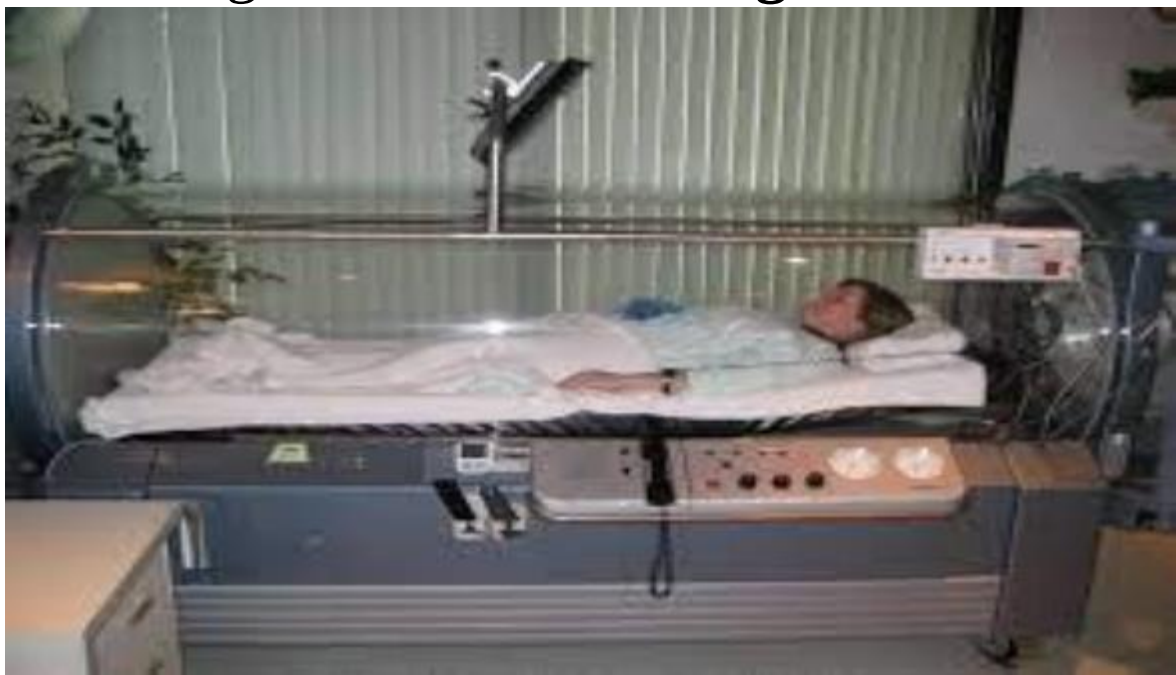
***Bone sclerosis and remodling. Scanty marrow spaces. Necrotic bone separates from vital bone & become surrounded by granulation tissue. Secondary bacterial colonization often is visible.***



**The management** with **long-term antibiotic therapy** in early stages of the disease can have a beneficial effect by **shortening the clinical episode**.In chronic stage, however, **surgical decortication combined with antimicrobial therapy might be the most effective treatment**. Surgical treatment was not recommended as the bone in DSO is hypovascular and healing will be protracted.

**Hyperbaric oxygen therapy is recommended** as lowered oxygen tension and anaerobic infection are **presumed**.

**Several reports** have suggested promising results with **bisphosphonates as they decrease bone resorption and bone turnover**.However, bisphosphonates are still far from being a standard of care in the treatment of DSO due to **several reasons**, one being the risk of **inducing osteonecrosis**



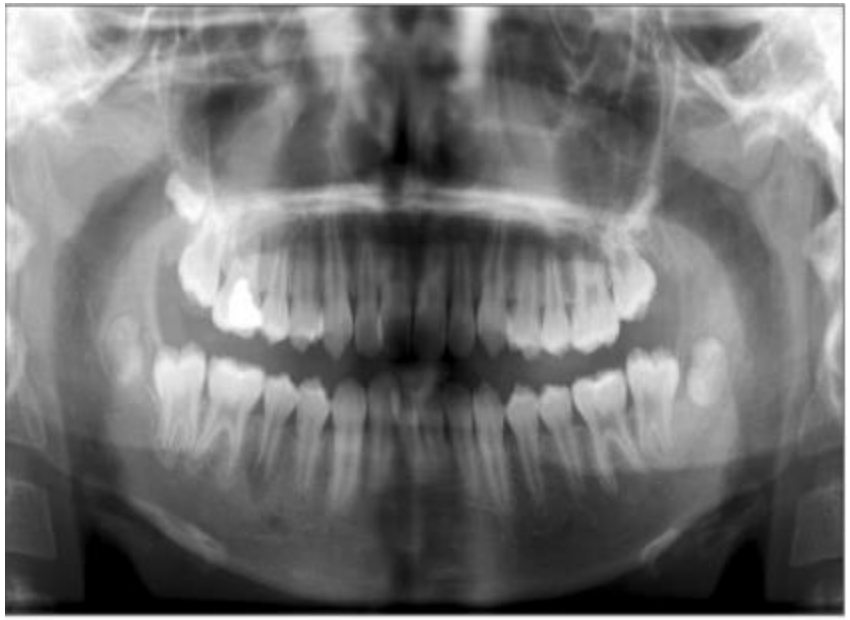
# *Proliferative periostitis:*

Proliferative periostitis is a **rare form of osteomyelitis** that is characterized by **new bone formation** with **periosteal reaction** .

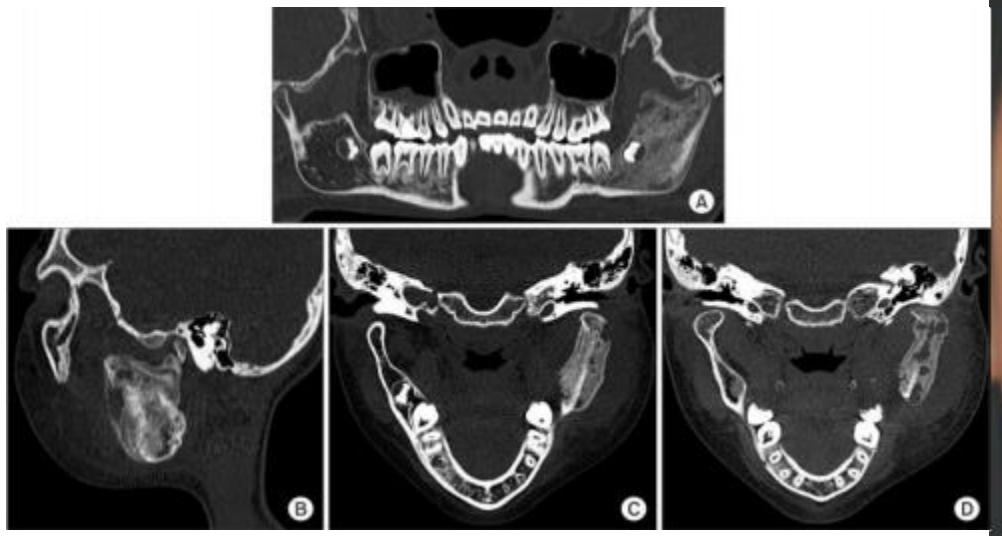
Common causes of **proliferative periostitis** are **dental caries**, **periodontitis**, **cysts** , and **trauma** . While proliferative periostitis typically presents as a **localized lesion**,

The most prevalent areas are the **premolar and molar areas of the mandible.**

**Radiographically** , radiopaque lamination is observed parallel to the surface of the **cortical bone** . The number of laminations varies from 1 to 12, and **radiolucent separation is seen between the newly formed bone** and the **original cortical bone** . Less frequently, **osteolytic radiolucency** and **sequestra** are observed in the **newly formed bone** . Most lesions are localized around the buccal cortex of the teeth which the periostitis originates.



Panoramic radiographs show the radiolucent appearance around the unerupted third molar on the left mandible



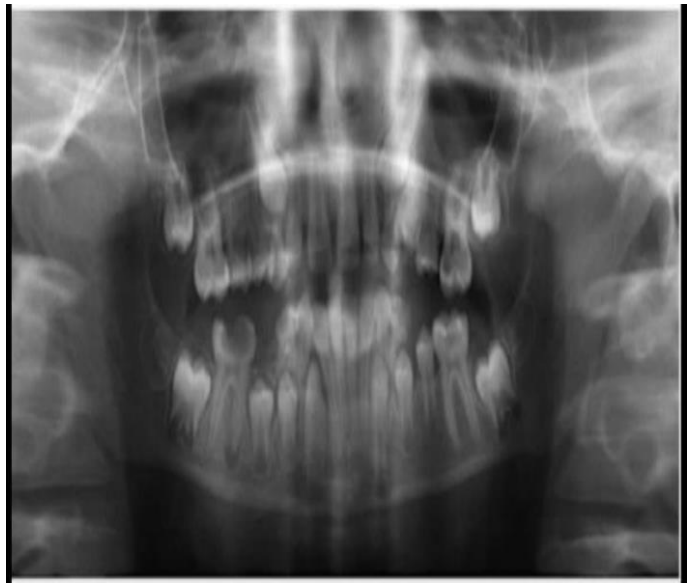

**Computed tomography image. Osteolytic and sclerotic bone lesion. A. The ramus and condyle. B. Mandibular angle. C. Periosteal reaction in the left mandibular ramus and condyle. D. Osteolytic change inside the newly formed bone**

***Garre's osteomyelitis*** is a distinctive type of chronic osteomyelitis associated with gross thickening of the periosteum of the bones and peripheral reactive bone formation resulting from mild irritation or infections. Garre's Osteomyelitis, which was first described by **Carl Garre in 1893**, is a chronic nonsuppurative sclerotic bone inflammation characterized by a rigid bony swelling at the periphery of the jaw . **It is most commonly seen The condition is seen exclusively in children or young adults**

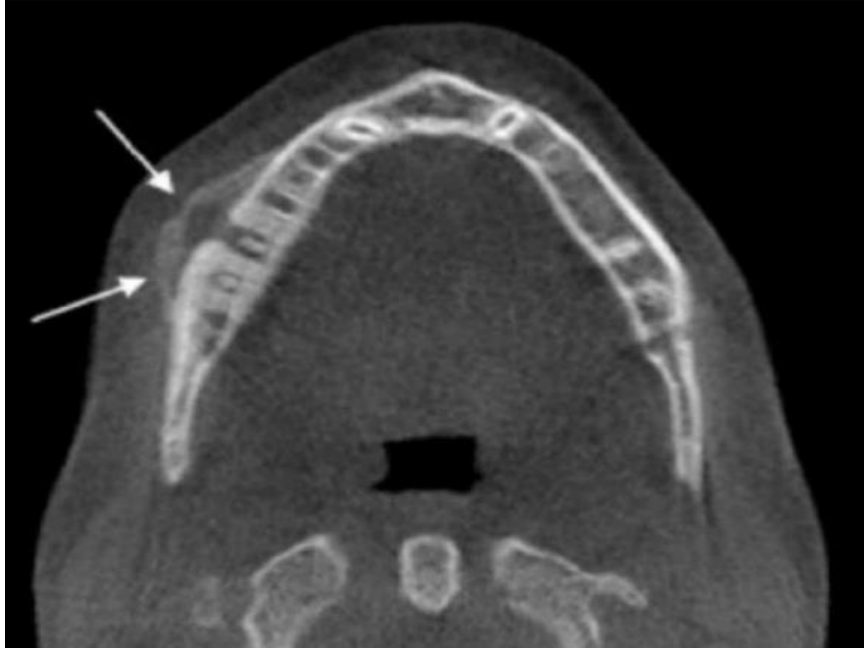
The mandible is more often affected than the maxilla, and it is most generally seen at the lower margin of the mandible in the mandibular first molar region. There is typically a nontender swelling on the medial and lateral sides of the jaw. The size of the swelling may vary from 1-2 cm to the involvement of the entire length of the jaw on the affected side; the thickness of the cortex can reach 2-3 cm .

Clinically, Garre's osteomyelitis results in facial asymmetry, since the lesion unilaterally expands to the outer surface of the bone . Pain is not a characteristic finding, although severe pain can occur if the lesion is secondarily infected. While it is referred to as nonsuppurative,

**Garre's osteomyelitis has sometimes been seen to result in a fistula on the skin** . The other symptoms are **fever**, **lymphadenopathy**, and **leukocytosis**.There is no **macroscopically suppurative lithic area in cases of Garre's osteomyelitis, although histopathological examinations have detected microabscesses and microsequesters.** The radiographic appearance varies with the duration of the lesion and the degree of calcification. During the early period, a thin crust-like convex layer appears over the cortex. As the event continues, the cortex is **thickened as a result of successive new bone deposits**. This lamellar structure is referred to as "**onion skin**" on radiographs. The adjacent spongiosa bone may exhibit a mixed structure, with some **osteolytic areas within the sclerotic field** , normal, or sclerotic area .



**Orthopantomographic image showing a deep caries cavity in the right mandibular first molar tooth, a radiolucent area in its mesial root, and subperiosteal new bone formation below the lower border of the mandible**

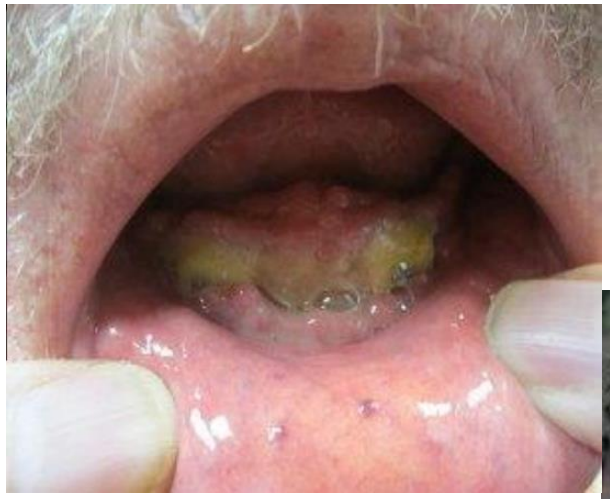


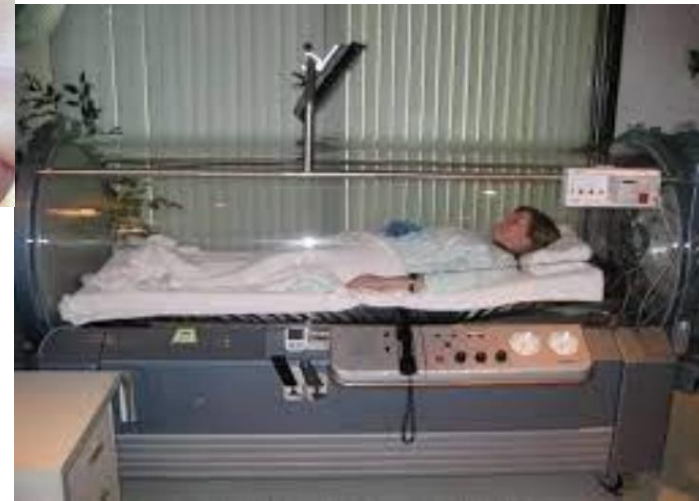
**CBCT showing new bone formation and a tunnel-like defect in the vestibule cortical surface of the inflamed bone starting from the apical region of tooth number LR6**

## *Refractory Osteomyelitis*

Refractory osteomyelitis is a persistent or recurrent bone infection despite appropriate surgical and medical therapy, such as debridement and **intravenous antibiotics**. In some individuals, conditions are worse when co-morbidities like **vascular disease** or **diabetes** is present.

**Treatments** can include a course of I.V. antibiotics, **hyperbaric oxygen therapy or both.**





# Hyperbaric oxygen therapy

**Recurrent bone infections can progress to refractory osteomyelitis**

# *Osteomyelitis of Maxilla*

Osteomyelitis of the facial skeleton is a rare condition. It tends to occur more commonly in the mandible than in the maxilla as the maxilla has a significant collateral blood flow, thin cortical bones, and bone marrow with struts which make it less prone to infection .

Osteomyelitis of the maxilla is a rare entity with the **widespread use of antibiotics** , early **diagnosis**, and **intervention guided by new imaging modalities**

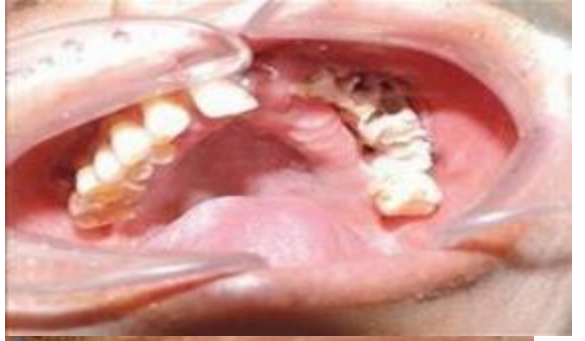
Maxillary osteomyelitis can be classified based on the following causes : **traumatic** , **rhinogenic** , and **odontogenic** .

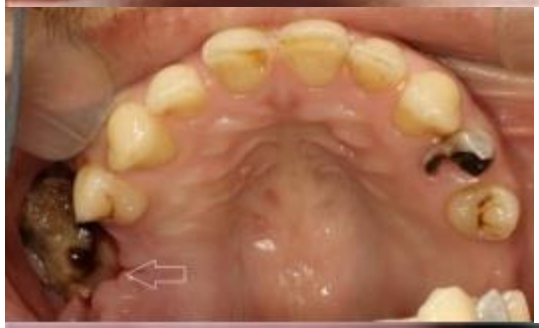
Factors which contribute to osteomyelitis are **systemic diseases** which compromise the **immune system** of an individual such as **diabetes mellitus**, **HIV**, **malnutrition** , and use of **chemotherapeutic agents.**

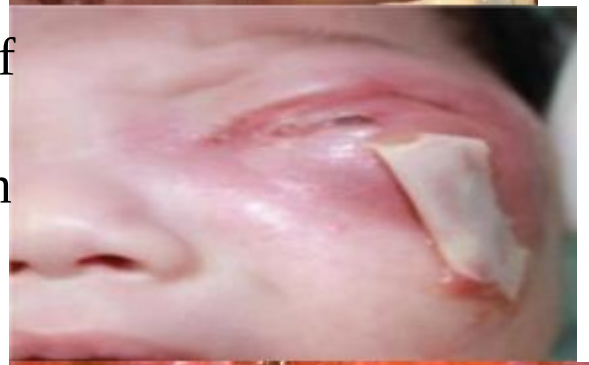
The most common forms of trauma is the dentoalveolar trauma observed in association with maxillofacial trauma were **crown fracture**, **root fracture**, **subluxation , avulsion** , **intrusion** , and tooth **concussion.**

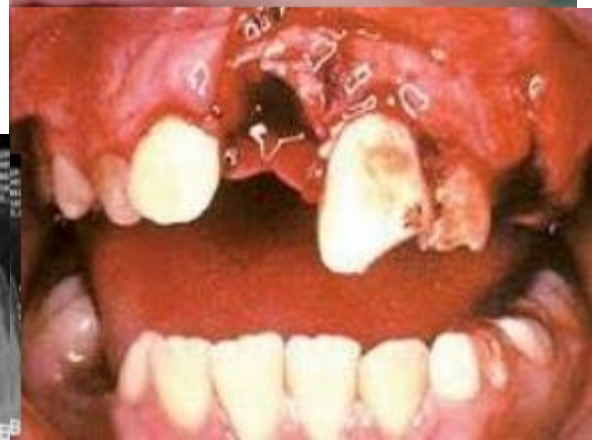
A possible complication of **the trauma** may be **chronic osteomyelitis**, defined as inflammation of the bone primarily caused by **odontogenic bacteria** ; trauma is the second leading cause of **chronic osteomyelitis**

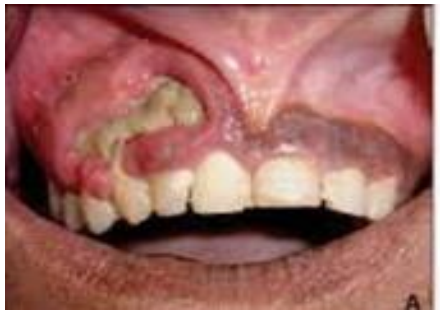
Extensive maxillary osteomyelitis **following tooth extraction in a patient with osteopetrosis**

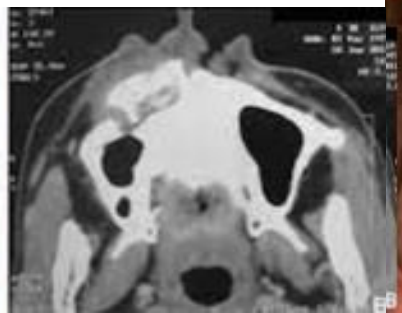




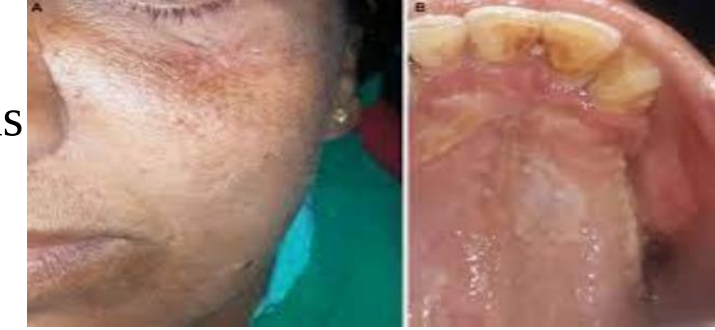


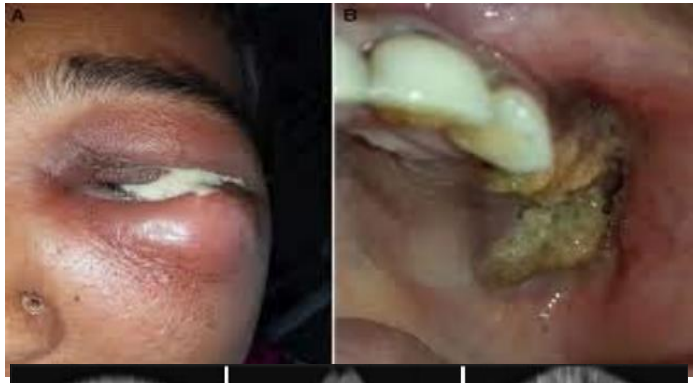


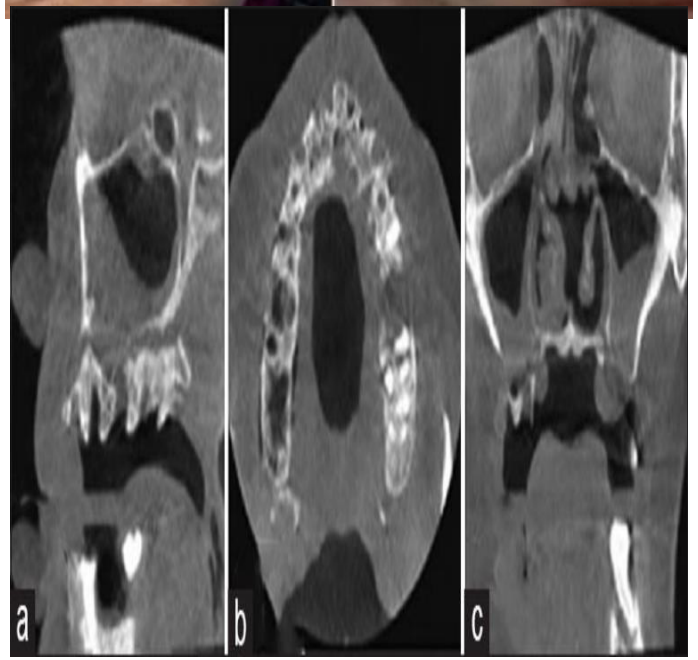




CBCT scan was taken with Field of view 10 × 10 and reconstructions were made in axial, coronal, and sagittal planes. Multiplanar reformation with reformatted OPG was also done. CBCT scan revealed an **extensive mixed density lesion involving the entire alveolar process of the maxilla with a moth-eaten appearance** .
In addition, bilateral breach of the maxillary sinus and floor of nasal cavity by the lesion with soft-tissues intensity of both sinuses suggestive of mucosal thickening and resultant blocked ostium were found



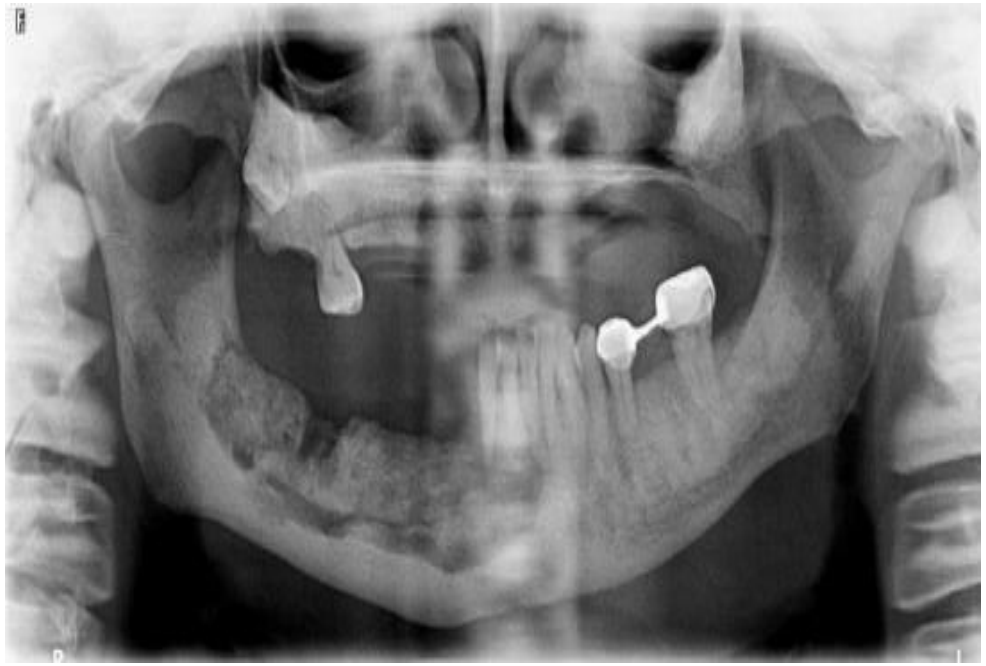





**Cone beam computed tomography scan in**
**(a) sagittal section-mucosal thickening of the maxillary sinus.**
**(b) Axial section-mixed density lesion with moth-eaten appearance.**
**(c) Coronal section-bilateral breach of maxillary sinus and floor of the nose with blocked sinus ostium**

**The treatments for maxillary osteomyelitis** range from a noninvasive approach to a more invasive radical treatment A combination of antibiotic treatment with surgery has shown to be effective in treating the condition.
***Surgical treatment*** involves removal of loose teeth and sequestra, debridement, decortication, resection, and reconstruction . It is important to consider osteomyelitis in immunosuppressed individuals as it is a difficult entity to treat. It may progress to involve infection of the cranial cavity and brain. It is imperative to suspect the diagnosis early and offer **treatment with antibiotics**. Optimal **glycaemic control** in diabetics is **mandatory to prevent such infections**

## *Osteopetrosis (OP) causing osteomyelitis ,*



Osteopetrosis, also know as Albers-Schönberg disease, osteopetrosis generalisata, or “marble bone disease”, is a rare genetic disease characterized by a generalized sclerosis of bone with a significant reduction in bone marrow spaces, due to an impairment of osteoclast activity that results in imbalance in bone remodeling .

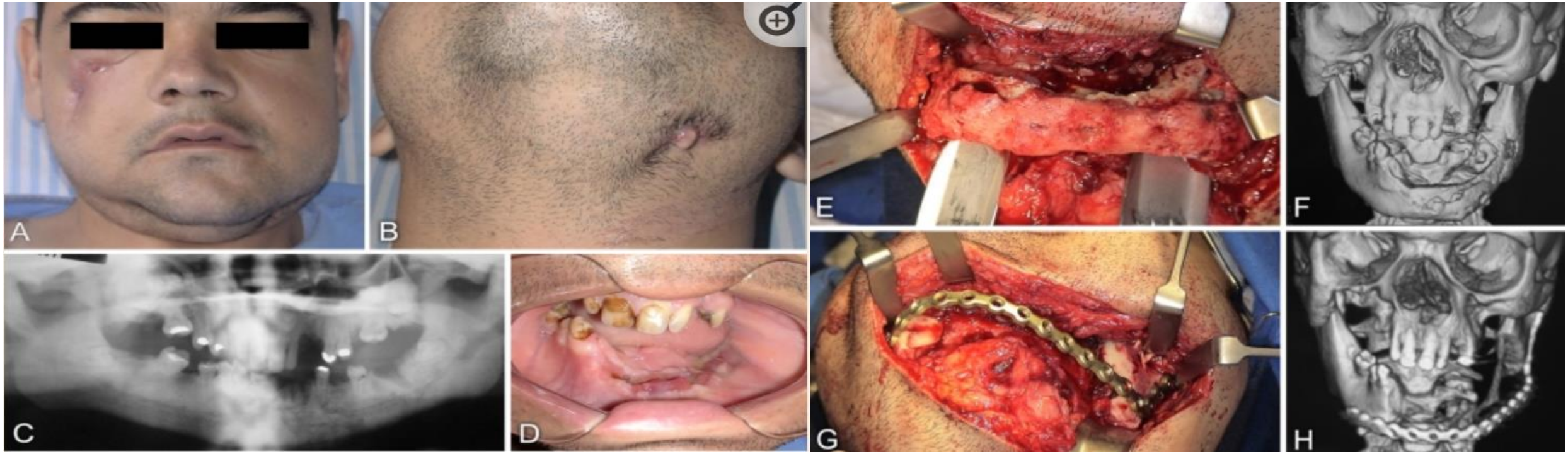
The prevalence of the disease is estimated to be about 0.005% of the population Classical osteopetrosis exhibits a vast spectrum of clinical, physiologic, and genotypic expressions and has been **classified into three clinically distinct forms:**

1) an infantile malignant autosomal recessive form, (2) an intermediate mild autosomal recessive form, and (3) an adult benign autosomal dominant form Autosomal dominant osteopetrosis (ADO) results from ineffective osteoclast-mediated bone resorption caused by inactivating mutations in the chloride channel 7 (ClCN7) gene, likely disrupting acidification of the osteoclast resorption lacunae Clinical manifestations in ADO include mostly **pathological fractures** and **osteomyelitis in** addition to **cranial nerve palsies**, **anemia**, **developmental abnormalities of the teeth**, and **other complications .**

The diagnosis of ADO is often made at the time of the diagnosis of other disease processes or during routine or specific radiological examination.

In patients with **osteopetrosis**, **osteomyelitis** may take place in **jaw bones**, almost exclusively in the mandible; the rare occurrence of osteomyelitis in maxilla has been attributed to its thin cortical bone and rich collateral bloody supply .

Tooth extraction and mild trauma are known factors that contribute to the development of osteomyelitic lesions in facial bones affected by osteopetrosis .**These have been treated using several therapies, including pharmacologic agents, hyperbaric oxygen, local wound care, and surgical procedures**




**a** Facial view of a 25-year-old man with osteopetrosis complicated with mandibular osteomyelitis, showing a fistula in the *right* zygomatic area. **b** The fistula of the *left* submandibular region drained purulent secretion. **c** Panoramic radiograph taken prior to tooth extractions, showing areas of increased bone density and empty tooth sockets (*lower left* incisor and molar areas). **d** Intraoral view prior to the first surgical procedure, revealing edentulous areas on the maxilla and mandible with bone exposure at the sites of tooth extractions in the *left* hemimandible. **e** Appearance of the affected mandible during surgical exposure. **f** Three-dimensional computerized tomography (CT) scan reconstruction reveals an irregular contour of the buccal surface of the *left* hemimandible, suggestive of periosteal bone formation. **g** Photograph of the titanium plate implanted to reconstruct the mandible without any bone grafting. **h** Three-dimensional CT scan reconstruction taken after surgery and showing the implanted titanium plate

# *Infantile osteomyelitis (IO)*

***Infantile osteomyelitis (IO)*** is an **urgent** and **serious disease** with quick changes in systemic condition. Osteomyelitis in the jaws of new born infants occurs almost exclusively in maxilla. This critical disease is often **misdiagnosed** and thus requires a selective **diagnostic approach**.

***Etiology:*** IO is affected by various factors, such as **genetic**, **toxic** , and **environmental factors** . Trauma through break in mucosa cause during delivery. Infection of maxillary sinus, infection from the nose ,hematogenous spread through streptococci & pneumococci.

***Clinical features:*** Fever, anorexia & intestinal disturbances., swelling or redness below the inner canthus of the eye in lacrimal region, Followed by marked edema of the eyelids on the affected side, alveolus & palate in region of first deciduous molar becomeswollen, Pus discharge from affected sites

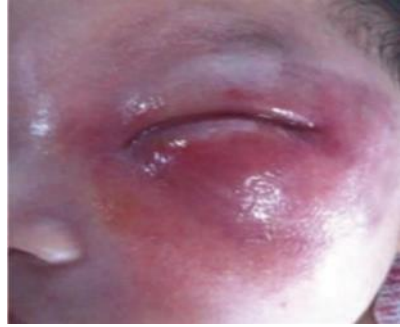
## *D/D for Infantile Osteomyelitis*

Dacrocystitis neonatarum, Orbital cellulitis, Ophthalmia neonatarum Infantile cortical hyperostosis

### *TREATMENT*

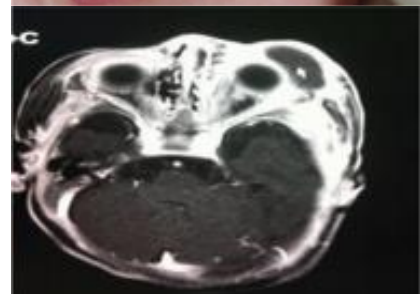
Intravenous antibiotics, preferably penicillin.Culture & sensitivity testing Incision & drainage of fluctuant areas, Sequestrectomy, Supportive therapy **Commonly, the pathogen of IO is *S aureus*** , and the **first-choice antibiotics are penicillin and cephalosporin** . When the clinical symptomsare improved, **antibacterial treatment should be used for another 1 to 2 weeks to prevent the recurrence of inflammation**.

Termination of antibiotic administration could be considered if **hypersensitive .**



**Facial appearance in the first visit. Slight ecstrophy was found in the lower eyelid — conjunctival congestion. The entire left periorbital region was puffy .**

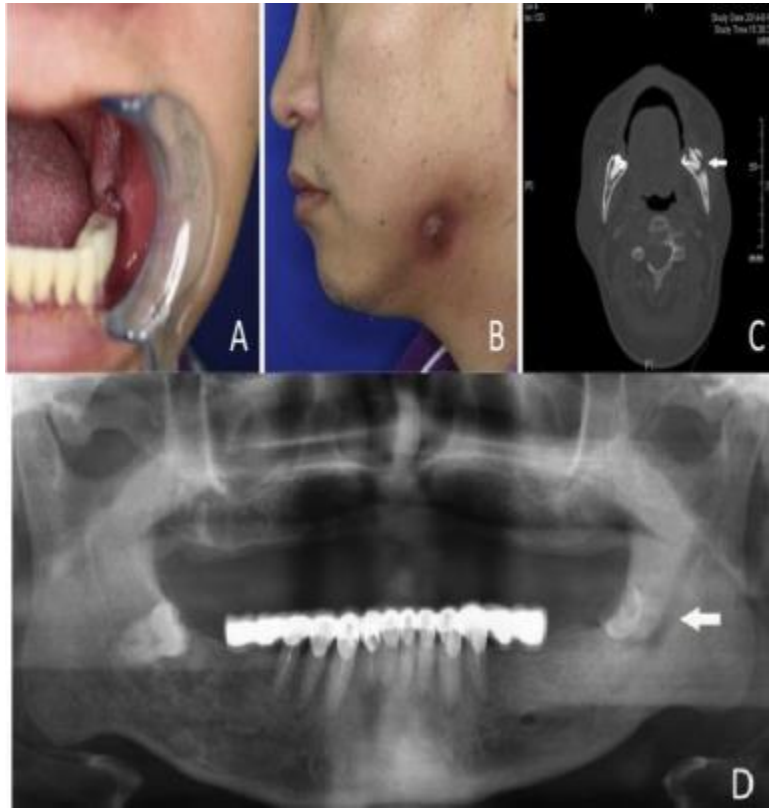
Intraoral, 2 draining fistulas were present in the left alveolar region, with exuded yellow-white pus (indicated by arrows ).



Enhanced MRI showed swelling soft tissue around left orbit, and abscess (indicated by arrows). The signal on bone marrow of external wall of left orbit was intensified. MRI, magnetic resonance imaging.

# *Osteoradionecrosis (ORN)/Radio osteomyelitis*

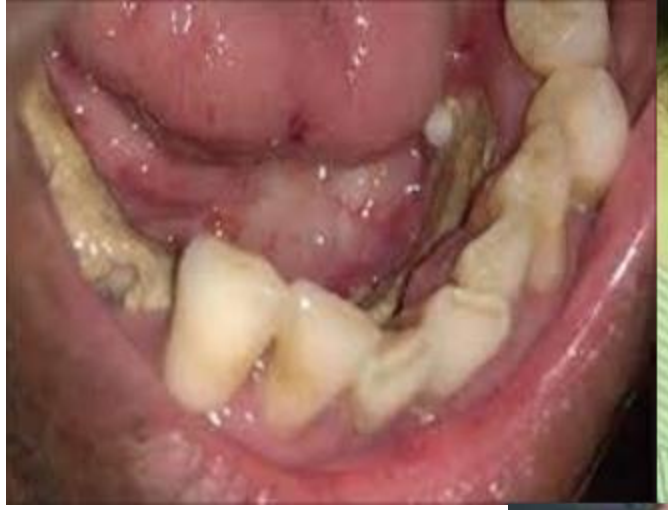
Osteoradionecrosis (ORN) of the jaws is **a pernicious complication of radiation therapy for head and neck tumours** . This article aims to provide an update on data related to the definition, epidemiology, staging, and clinical and radiological findings of ORN of the jaws. Osteoradionecrosis of the jaw (ORNJ) is among the most serious late complications observed in patients with head and neck squamous cell carcinoma (HNSCC) treated with radiation therapy (RT). While ORNJ was **first described in 1922**, the definition, mechanism of pathogenesis, incidence, risk factors, and clinical staging, as well as treatment protocols associated with the disease require further investigation.
**Osteoradionecrosis** is rarely seen in patients who received **less than 6000 centiGrays (cGy) of radiation** and may occur years or even decades after **radiation is concluded** . The resulting chronic infections can lead to osteomyelitis and soft tissue orocutaneous fistulae




## *Evaluation OF ORN*

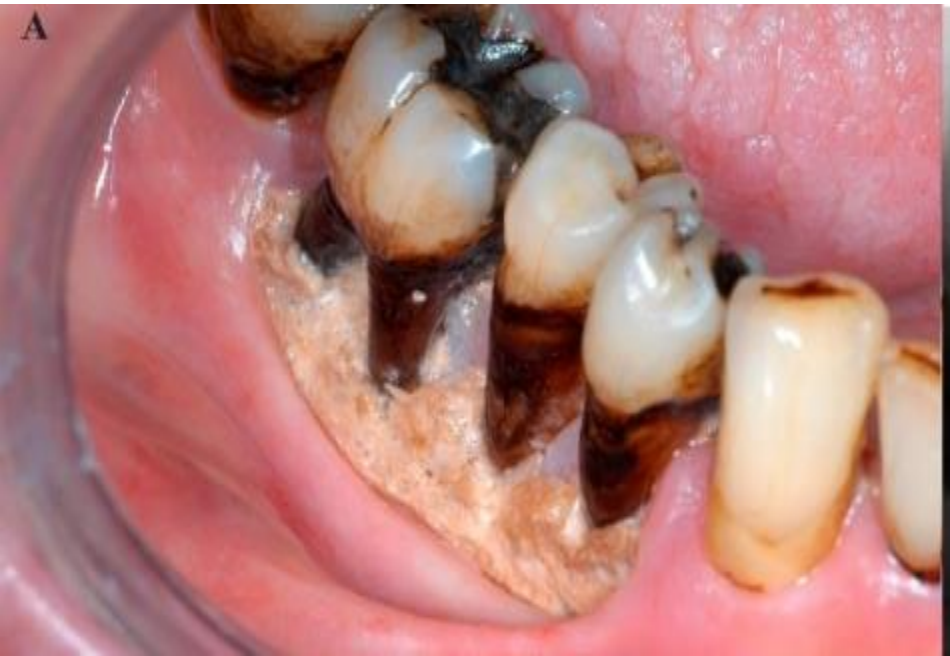
Imaging such as radiographs, panorax images, computed tomography (CT), magnetic resonance imaging (MRI) should be reviewed. There are no lab studies that are diagnostic for osteoradionecrosis (ORN) of the jaw per se other than biopsy. Sedimentation rate and C-reactive protein (CRP) may be elevated in osteomyelitis. **A thorough exam of the oral cavity using a dental mirror and tongue depressor is indicated** . The condition of the teeth should be noted as well as any that are already missing or are going to need extraction.
**The tongue and gingiva should be inspected** . Any exposed bone should be measured and documented. Any draining sinuses or fistulae should be documented.

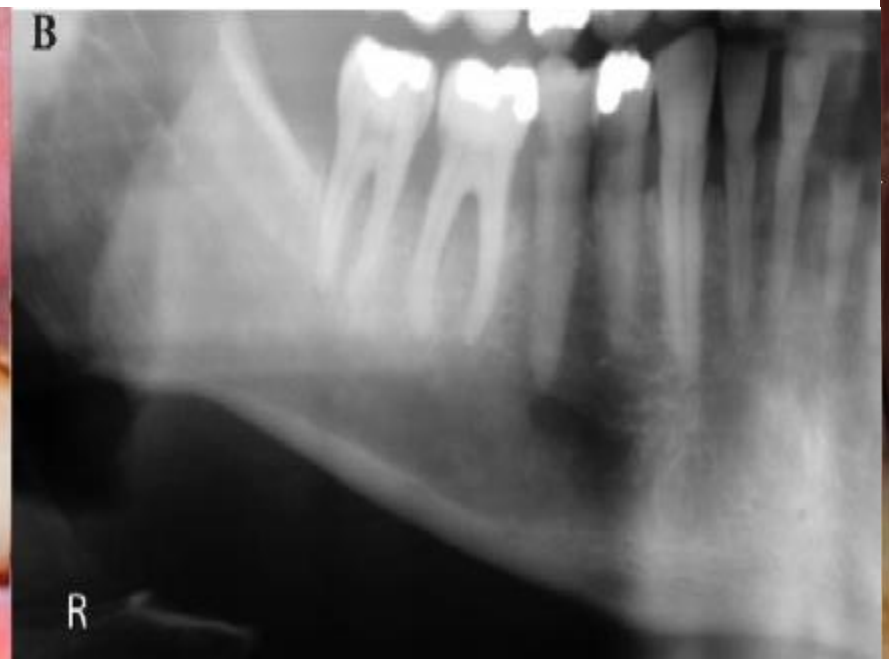
**Palpation of the cervical, posterior auricular, and axillary lymph nodes should be done and results documented**. The timing and dose of radiation should be requested from the radiation oncologist. Any chemotherapy given should be documented as well as start and completion dates noted. **The presence of xerostomia, dysphagia, dysphonia and ageusia should be noted**

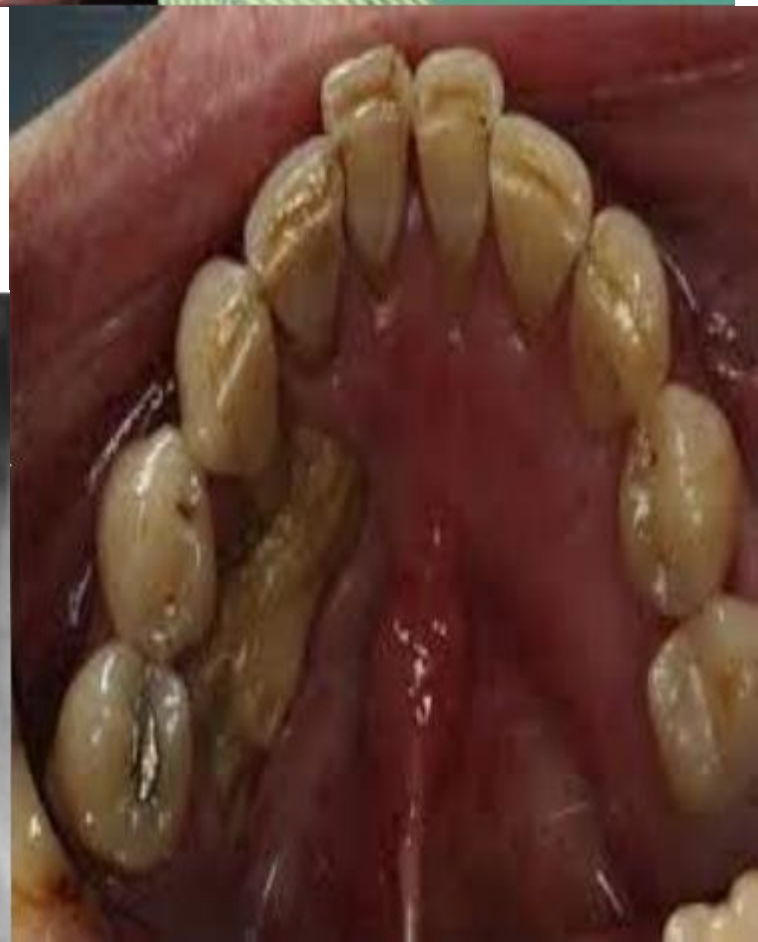


## *Scale of Osteoradionecrosis*

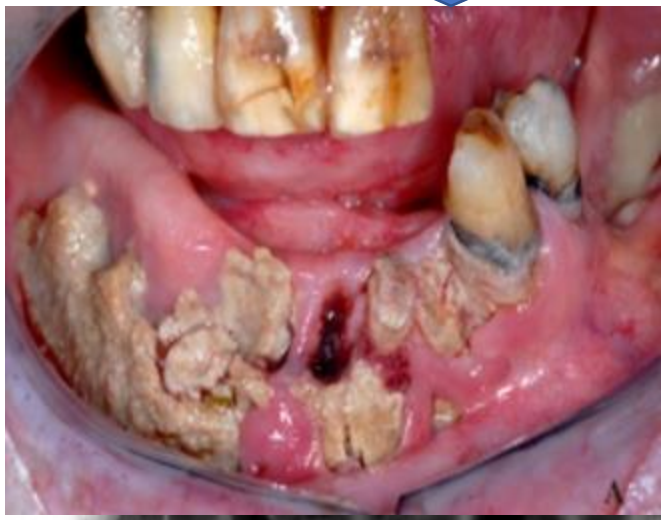
Stage I: Patients with exposed bone that has been chronically present or which developed rapidly. Patients are treated with **30 hyperbaric** treatments preoperatively followed by bony debridement. Postoperatively they are given an additional ten treatments



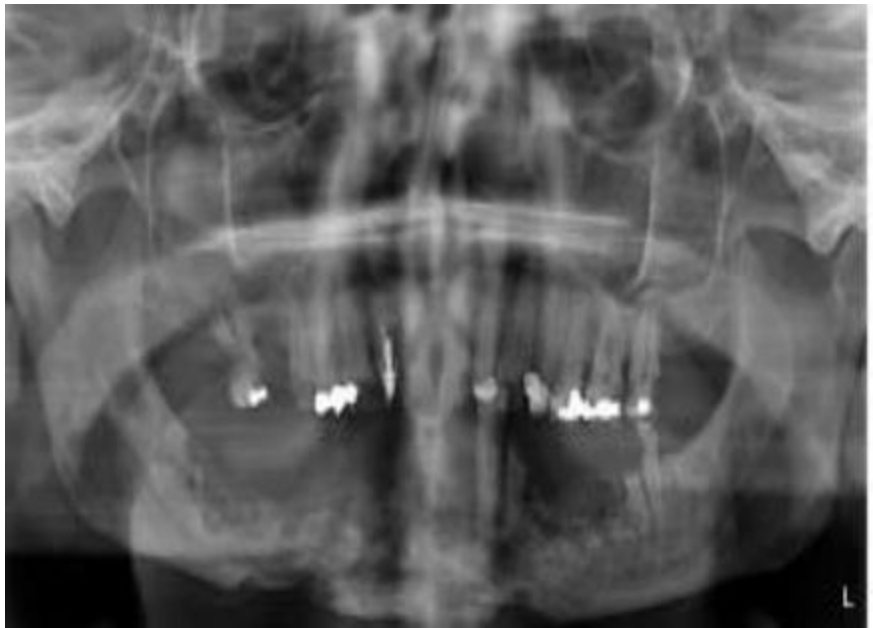



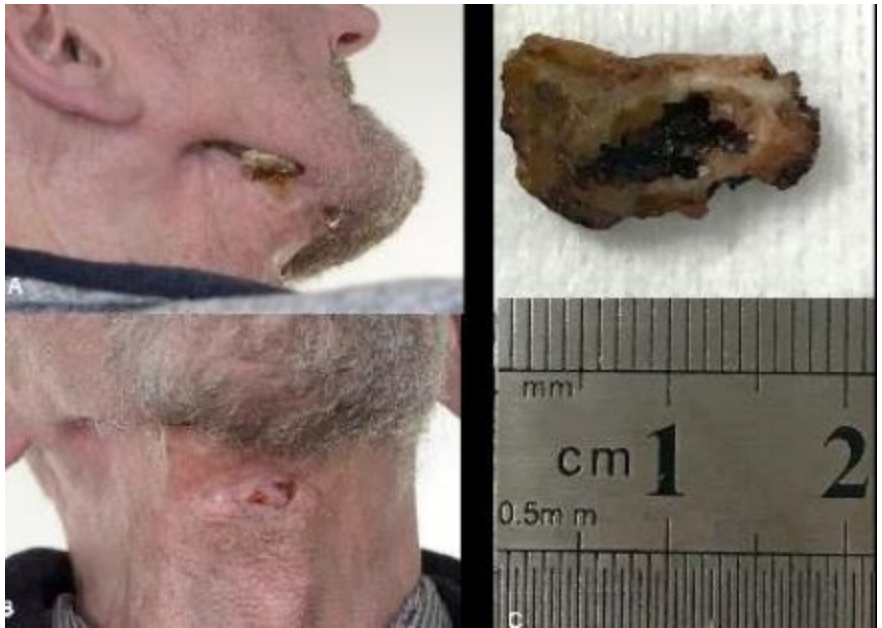



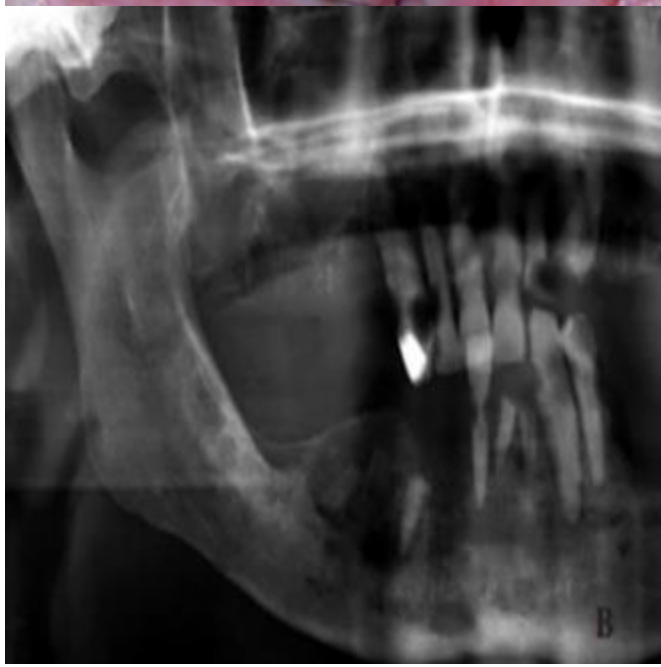
***Stage II:*** These are patients who do not respond favorably to 30 pre-operative treatments, or when a more **major operative debridement is required** . Surgery for stage II osteoradionecrosis patients must be focused on preserving the integrity of the mandible. If mandibular resection is anticipated, patients are advanced to **Stage III.**



***Stage III:*** Along with patients who have progressed from stages I and II, patients with stage III osteoradionecrosis have serious and potentially grave prognostic findings such as **pathologic fracture**, **percutaneous fistulae** , and **lytic lesions that extend to the inferior border of the mandible** . For patients with stage III osteoradionecrosis, mandibular resection is planned as part of the surgical treatment. It is critical that all necrotic bone be debrided and removed **in stage III** patients. Stage III osteoradionecrosis **patients receive 30 treatments preoperatively** followed by ten **hyperbaric oxygen treatments**





**ORN** as long as there is healthy, intact soft tissue. There are three factors in the pathogenesis of ORN:

**exposure of radiotherapy above a critical dose** ,

**local injury,** and

**the development of infection** .

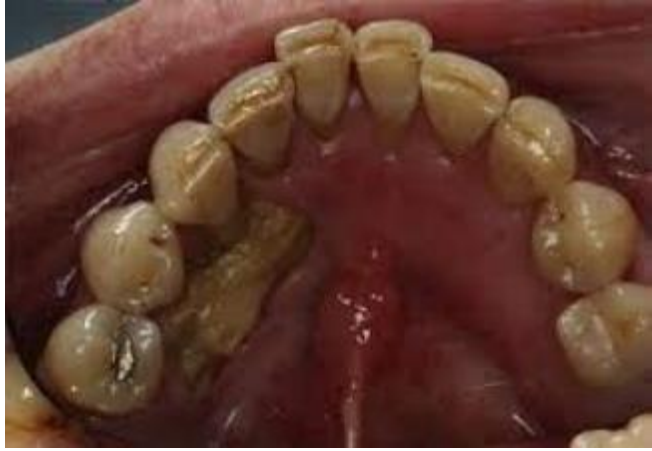
**The vascular changes in the field of radiation that were seen as early as 1.5 weeks into radiation treatment**. The mandible, with its **centrally located inferior alveolar vessels**, compared to **the maxilla, with its rich vascular plexus** . it was the **hypovascular** , **hypoxic** and hypocellular environment that resulted in poor healing and the development of ORN .

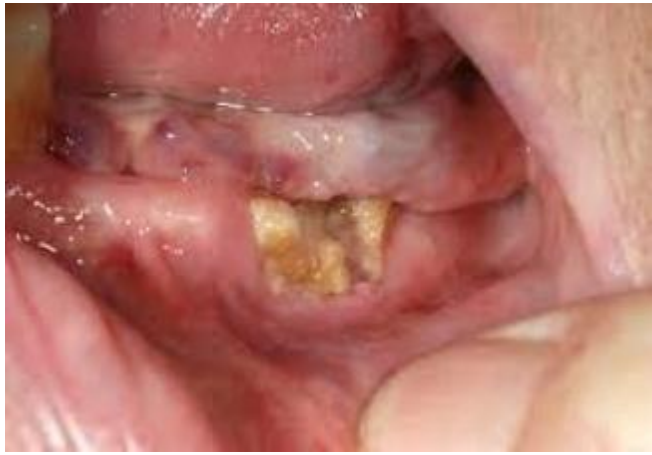
This was the accepted theory of the pathogenesis of ORN for decades, and led to hyperbaric oxygenation (HBO) as the treatment for **prevention and management of ORN.**

***HYPERBARIC OXYGENATION:***

The use of HBO for patients with ORN undergoing dental extractions and implant placement has been accepted for decades. **Patients with ORN were subjected to 30 dives at 2.4 atmospheres for 90 minutes, followed by 10 dives**. **There is no clear evidence in the literature that HBO is effective in the treatment and prevention of ORN** .

A Cochrane review of patients receiving dental implants concluded there is **no evidence for or against the effectiveness of HBO in improving dental implant outcomes in radiated patients**





## *TREATMENT STRATEGIES*

Various studies have reported 10% to 100% resolution of symptoms **after simple, conservative therapy** . When treating patients who are suspected of having ORN, the possibility of **recurrent disease must always be ruled out** . These are cancer patients and, despite being treated, there is a possibility of recurrence. Verification can be done with a biopsy of the affected site. Once recurrence has been ruled out, treatment can proceed.

**There are no universally accepted guidelines for the treatment of ORN today, and protocols for HBO that were once accepted as the standard of care in managing ORN are no longer recommended**.

Proponents of the fibroatrophic theory use two oral medications to help manage ORN. A patient can take 800 mg of pentoxifylline and 1000 IU vitamin E at least two weeks prior to a procedure, and complete a two-month course of this regimen. Vitamin E (alpha-tocopherol) is an antioxidant that scavenges free radicals generated during oxidative stress. Pentoxifiylline has been used for the management of vascular disorders, such as intermittent claudication, and has anti-inflammatory mediators, such as TNF- α.

In addition to these medications, **for early stage ORN, the dentist could consider surgical debridement** ; and **passive closure following the surgical procedure is obligatory** .

The patient may require **oral antibiotics** whenever there is an **overlying infection**. At times, it is difficult to obtain primary closure, and wound dehiscence is common because the soft tissue is contracted from the frequent infections and fibrotic from the radiation.

**If the wound is left open** , the patient should be counseled in proper oral hygiene and counseled to have patience. With time, **a sequestrum will form and can be easily removed, leaving healthy granulation tissue beneath** .

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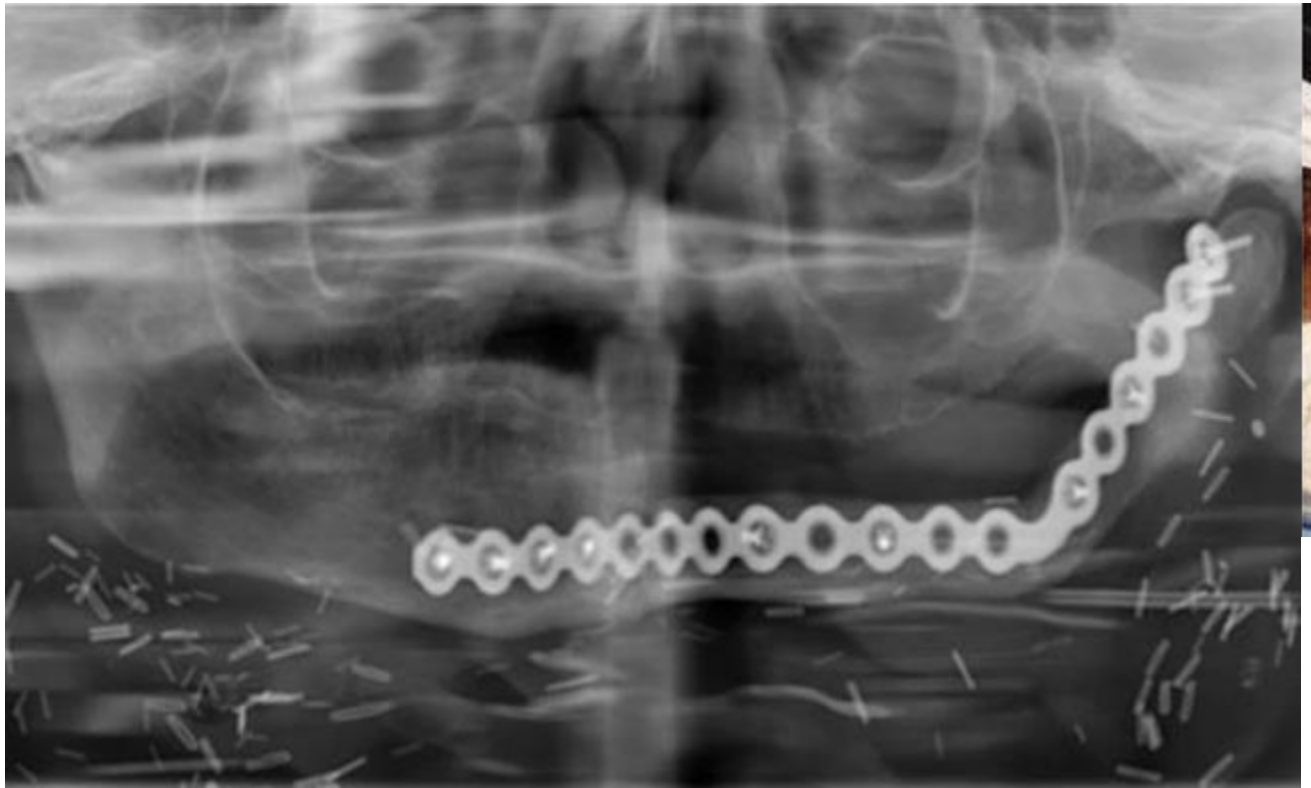
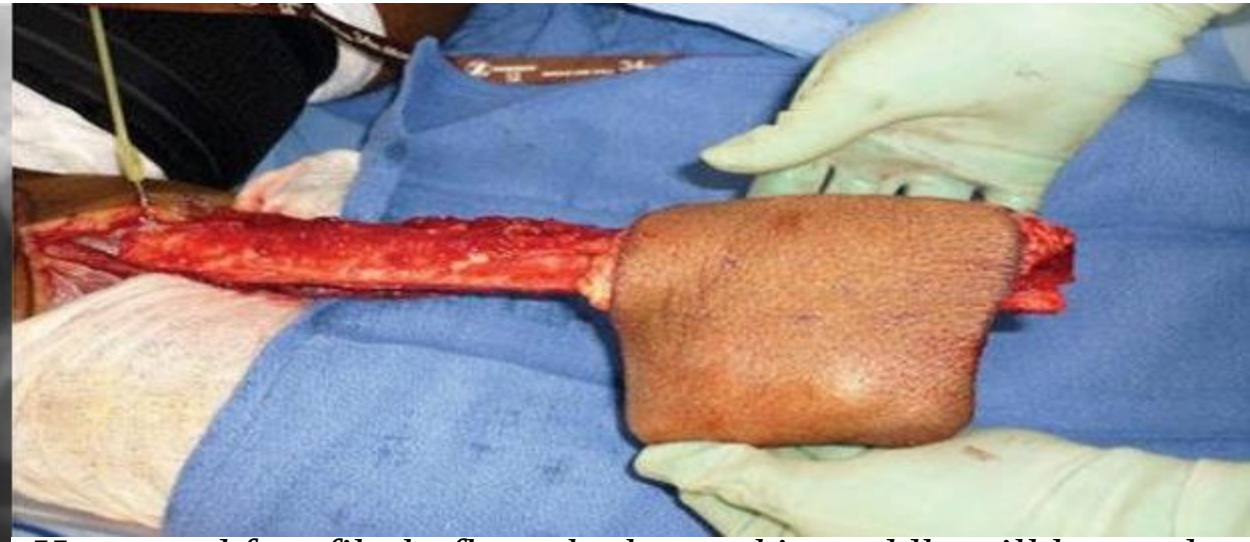
Unfortunately, some patients will progress to ***Stage III ORN***. For these individuals, complete resolution of pain, **infection** and **exposed bone** can only be accomplished by using ***free vascularized tissue to rebuild and replace the damaged jaw*** and ***missing soft tissue*** .These are lengthy surgeries, followed by **long hospital stays** and **prolonged rehabilitation** — with the majority of these patients **requiring gastric feeding tubes**





*Harvested free fibula flap: the large skin paddle will be used to reconstruct the missing soft tissue intraorally, and help close the neck incision and bone to reconstruct the mandible*

Dental providers should understand it is possible to successfully extract teeth and place dental implants in patients who have **previously received radiation therapy** . However, the risks and benefits of these procedures need to be **thoroughly discussed — and informed consent obtained** — ***before providing therapy***.

This is important because the sequelae to tooth extraction or implant placement often brings negative results.

**The psychological impact of surviving cancer** , only to be handicapped by the treatment sequelae, can be crippling. As oral health professionals, treating patients who have had radiation therapy can prove challenging. These patients have survived a momentous hurdle in their lives, and fear of cancer recurrence always lurks in the back of their minds.

## *Complications leading to Osteoradionecrosis*

Osteoradionecrosis often results in the following complications:

- Ulceration and necrosis of the mucosa with exposed bone, inevitably leading to infection and necrotic bone.
- Exposed bone often leads to irritation of surrounding oral soft tissues.
- Pathologic fractures can form in the weakened bone.

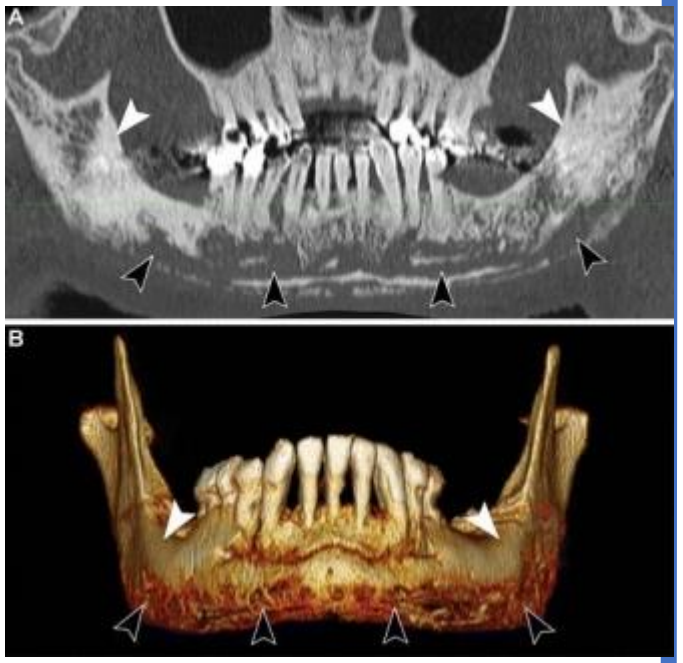
**Fistulae** are a sign of spreading local infection, and can ultimately lead to systemic infection and even sepsis if not appropriately managed.

# Medication related osteonecrosis of the Jaw

**Medication-related osteonecrosis of the jaw** is **a serious complication of treatment with drugs used to prevent skeletal events associated with bone metastases and osteoporosis**. These drugs, such as **bisphosphonates** or **antiresorptive monoclonal antibodies** (eg, denosumab and romosozumab), **inhibit osteoclasts**

Medication-related osteonecrosis of the jaw has also been described in response to **antiangiogenic drugs** such as **anti-endothelial vascular growth factor monoclonal antibodies** or **tyrosine kinase inhibitors** . **Medication-related osteonecrosis** of the jaw can occur in up to **7% and 2% of patients with cancer treated with high-dose bisphosphonates** or **denosumab** , respectively, but is **very uncommon** with low-dose regimens used in **osteoporosis**. Dental check-ups **before** and **after treatment are mandatory** . Patients must be aware about the risk of this condition and must **immediately report any oral symptoms to their physician** . Treatment includes measures to **eliminate pain**, **control infection** , and **minimize the progression of bone necrosis with surgical debridement in advanced cases** .

**Several adjuvant therapies** are under investigation, including **hyperbaric oxyge** n, **laser treatment**, **fluorescence-guided bone resection**, and **recombinant human parathyroid hormone administration**




CT images in 75-year-old woman with breast cancer who received denosumab for bone metastases and presented with intraoral lesion with exposed necrotic bone and submental abscess.
**(A)** Curved reformatted image and **(B)** volume-rendered image show extensive osteolysis (black arrowheads) compared with normal bone (white arrowheads). Appearances are consistent with medication-related osteonecrosis of the jaw

# Medication-related osteonecrosis of the jaw (MRONJ)

| | Primary Indication | Nitrogen Containing | Dose | Route |
|---|---|---|---|---|
| Alendronate (Fosamax®) | Osteoporosis | Yes | 10 mg/day<br>70 mg/week | Oral |
| Risedronate (Actonel®) | Osteoporosis | Yes | 5 mg/day<br>35 mg/week | Oral |
| Ibandronate (Boniva®) | Osteoporosis | Yes | 2.5 mg/day<br>150 mg/month<br><br>3 mg every 3 months | Oral<br><br>IV |
| Pamidronate (Aredia®) | Bone Metastases | Yes | 90 mg/3 weeks | IV |
| Zolendronate (Zometa®)<br><br>(Reclast®) | Bone Metastases<br><br>Osteoporosis | Yes | 4 mg/3 weeks<br><br>5 mg/year | IV<br><br>IV |
| Denosumab (Xgeva®)<br><br>(Prolia®) | Bone metastases<br><br>Osteoporosis | No<br><br>Humanized monoclonal antibody | 120 mg/4 weeks<br><br>60 mg/6 months | SQ<br><br>SQ |

| Drug | Mechanism of action | Primary indication |
|---|---|---|
| Sunitinib (Sutent®) | Tyrosine kinase inhibitor | GIST, RCC, pNET |
| Sorafenib (Nexavar®) | Tyrosine kinase inhibitor | HCC, RCC |
| Bevacizumab (Avastin®) | Humanized monoclonal antibody | mCRC, NSCLC, Glio, mRCC |
| Sirolimus (Rapamune®) | Mammalian target of rapamycin pathway | Organ rejection in renal transplant |

*Abbreviations:* GIST gastrointestinal stromal tumor; RCC renal cell carcinoma; pNET pancreatic neuroendocrine tumor, HCC hepatocellular carcinoma; mCRC metastatic colorectal carcinoma; NSCLC non-squamous non-small cell lung carcinoma; Glio Glioblastoma; mRCC metastatic renal cell carcinoma

Medication-Related Osteonecrosis of the Jaw – 2014 Update AAOMS Positioning Paper Pg. 18 – 19.

# *Medication-related osteonecrosis of the jaw (MRONJ) – Risk Factors*

***1.Medication related risk factors***

The risk of ONJ among cancer patients exposed to zolendronate and denosumab ranges between 50-100 times higher than cancer patients treated with placebo.

Cumulative incidence 0.7 – 6.7% (~1%)

Stay in the bone for many years ~10 years

***2.Osteoporosis Patients***

Oral BPs: 0.1% (10 cases per 10,000) which increased to 0.21 (21 cases per 10,000) among patients with greater than 4 years of oral BP exposure

***3.Duration of Medication Therapy***

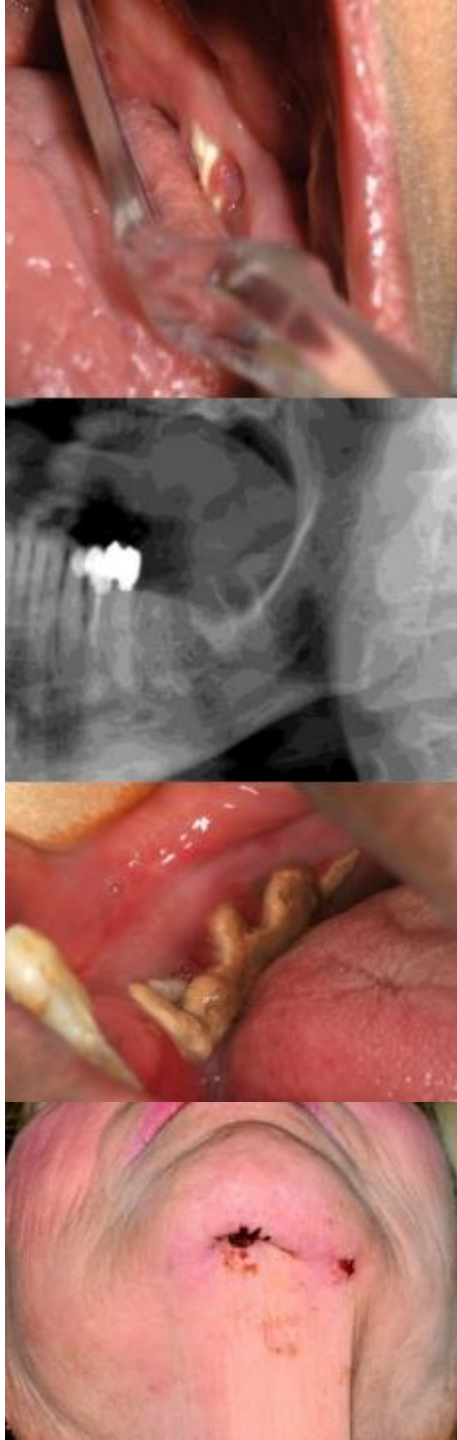
IV therapy – risk increased each year for the first 3 years

Oral BPs – 0.21% after 4 years or more (median duration 4.4 years, minimum 2.6 years)

***4.Others***

Soft tissue toxicity by BP

Innate or acquired immune dysfunction



# *Medication-related osteonecrosis of the jaw (MRONJ) – Risk Factors*

***1.Operative Treatment***

- Dentoalveolar Surgery (extraction, implant placement, periodontal surgery)
- Tooth extraction - 52 to 61%, 16 - 33 folds increased risk (cancer patient on IV BP)
- No data for the risk for ONJ for other procedures.
- AAOMS: dental implant placement and endodontic or periodontal procedures that require exposure and manipulation of bone are comparable to the risk associated with tooth extraction

***2.Anatomic factors***

- Mandible (73%), Maxilla (22.5%), Both (4.5%)
- Dentures

***3.Concomitant oral disease***

- Periodontal disease, Periapical pathology
- Associated with tooth extraction

***4.Demographic, Systemic factors and other Medication factors***

- Age, Sex
- Anemia (hemoglobin < 10g/dL ), Diabetes
- Corticosteroids, Anti-angiogenic drugs
- Smoking

***5.Genetic factor***

# *Bisphosphonates*

Can be given orally or parentally

Prevention and treatment of osteoporosis and osteopenia

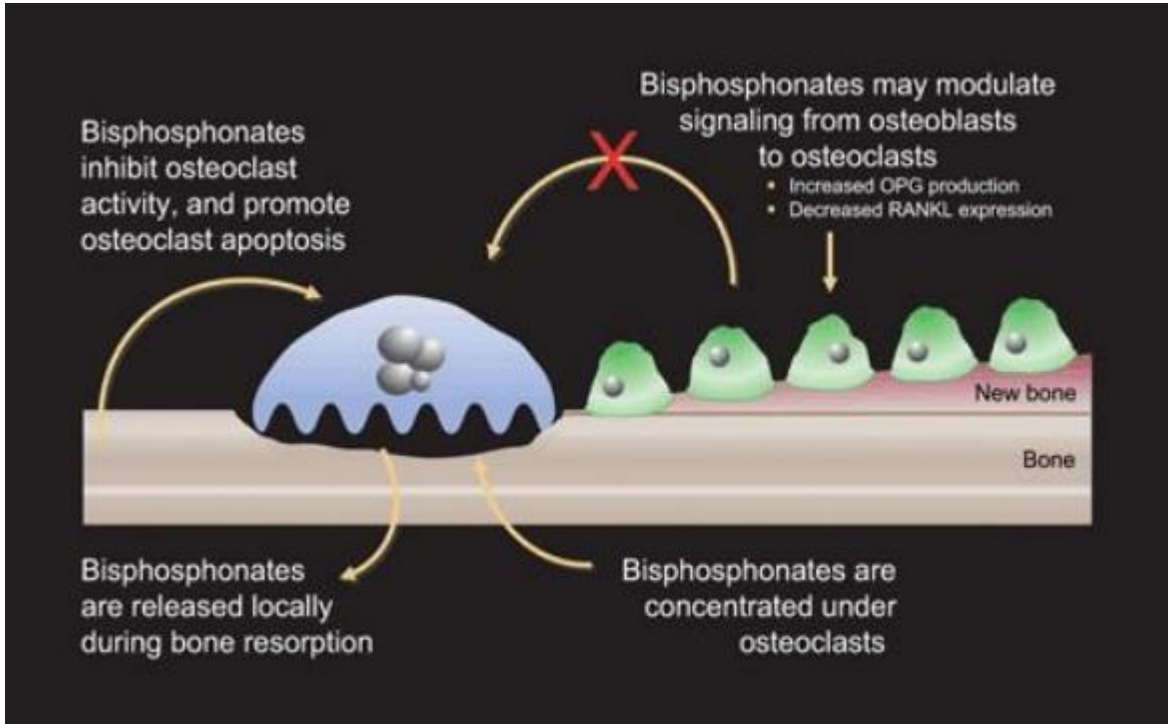
- Oral Bisphosphonates
- IV Bisphosphonates - once yearly infusion of zolendronate (Reclast®) and a parenteral formulation of ibandronate (Boniva®) administered every three months

Managing cancer-related conditions including:

- hypercalcemia of malignancy, skeletal-related events (SRE) associated with bone metastases in the context of solid tumors such as breast cancer
- prostate and lung cancers

Managing of lytic lesions in the setting of multiple myeloma

- IV bisphosphonates




Despite the strong association between jaw necrosis and bisphosphonates and other antiresorptive medications and antiangiogenic drugs, the pathophysiology of MRONJ is not completely understood. Hence, an effective and appropriate therapy for the condition is still to be decided. It is crucial to have a collaborative approach involving dentists, prescribing doctors, and pharmacists to prevent the development of MRONJ

## ***Management***

Before starting drug therapy, Oral health assessment+ Necessary dental work before starting bisphosphonate therapy
Patients who are on **bisphosphonate therapy.** Oral or IV ,For what reason, Duration
Risk of **Medication-related osteonecrosis of the jaw (MRONJ) – Local and systemic factors**

## ***AAOMS Recommendations***

The medical use of **bisphosphate drugs**, **both I.V. and oral, along with other antiresorptive and antiangiogenic drugs for the treatment of cancer and osteoporosis, has increased rapidly over the past 20-plus years**. There is increasing awareness of these drugs' possible adverse side effect of Bisphosphate-associated Osteonecrosis of the Jaw (BONJ). **This is a severe adverse drug reaction, consisting of progressive bone destruction in the maxillofacial region of the patient.**
Patients about to initiate **intravenous antiresorptive** or ***antiangiogenic treatment for cancer therapy***
**Patient education**
**Non-restorable teeth** and **those with a poor prognosis should be extracted** .
Complete the **necessary elective dentoalveolar surgery**
Delay the antiresorptive or antiangiogenic therapy if **systemic conditions permit**, until the extraction site has **mucosalized** (**14-21 days** ) or **until there is adequate osseous healing**
Dental prophylaxis, caries control and conservative restorative dentistry are critical to maintaining functionally sound teeth

**Examine full** or **partial dentures for areas of mucosal trauma** , especially along the **lingual flange region** Patients receiving intravenous antiresorptive or ***antiangiogenic treatment for cancer therapy***

Oncology Patients Receiving Monthly **Antiresorptive Therapy** : Tooth extraction should be avoided if possible If ONJ develops the **oncologist** may consider discontinuing antiresorptive therapy until soft tissue closure has occurred, **depending on disease status.**

**Biopsy only if metastasis is suspected.**

**Avoid elective surgery including Implants**

Routine Dentistry: **restorations**, **local and atraumatic periodontal therapy with RCT to avoid extractions**

Consider placement of **protective stent to *protect exposed bone***, *or when such bone causes trauma to adjacent tissue or is further insulted with normal function.*

Management issues relative to removable prostheses: must be hygienic, cleaned daily and removed at night, well-fitting with no risk of soft tissue trauma.

**Amoxicillin 500mg tid 14 days or Clindamycin 300mg tid for 15 days or Azithromycin 250mg qd for 10 days**

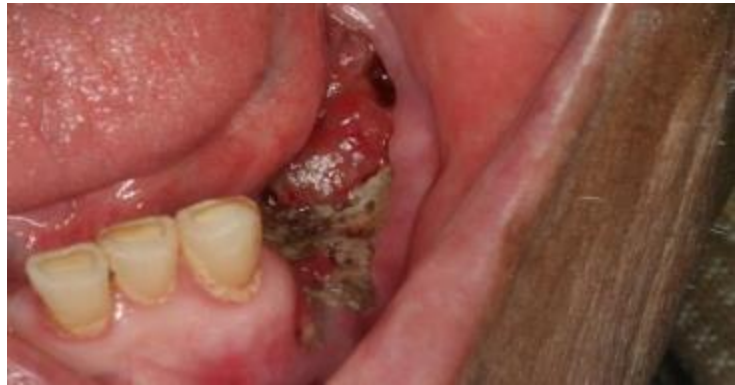
Other anti-microbial therapy: **Intermittent or continuous based on findings of cultures:** quinolones, doxycycline, erythromycin.

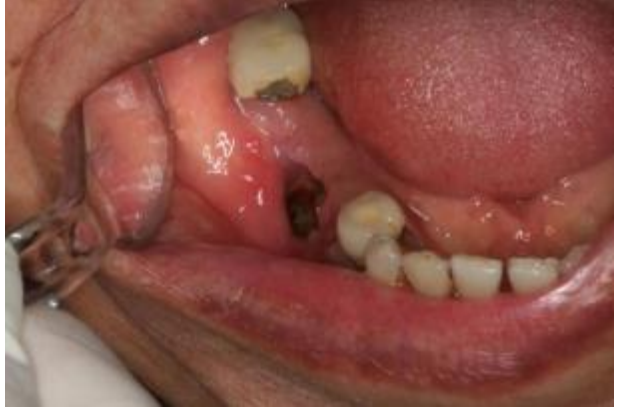
Refractory cases may **require combination AB therapy**; long-term AB maintenance; or intravenous AB therapy.

Anti-fungal medications may be needed.

Recall Schedule: **monitor every 3 months unless additional problems develop.**

**Hyperbaric Oxygen: *not shown to be effective.***





75 YO Asian female patient, Fosamax once a week for 1 and half years

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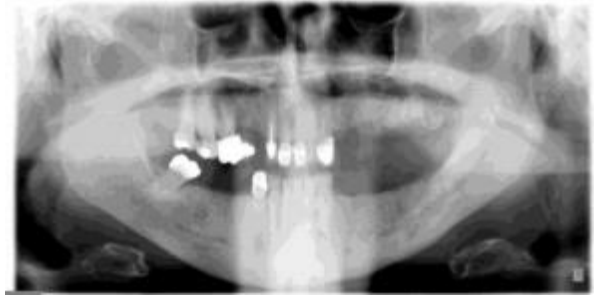

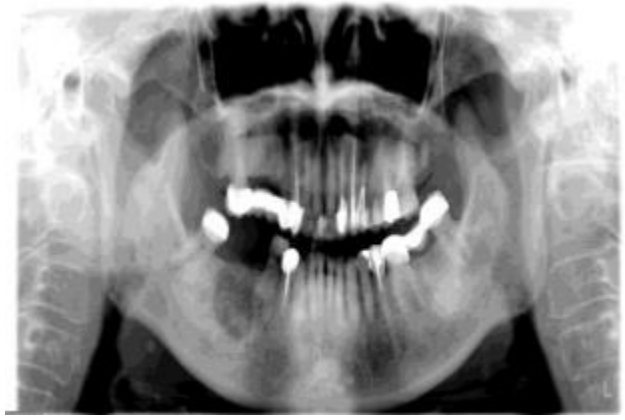

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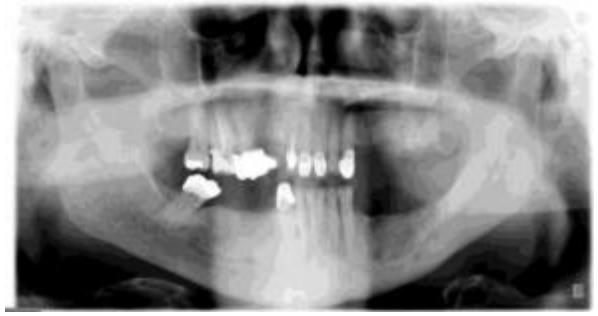

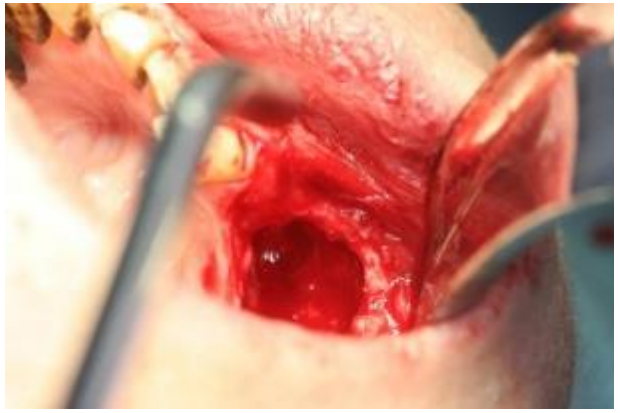

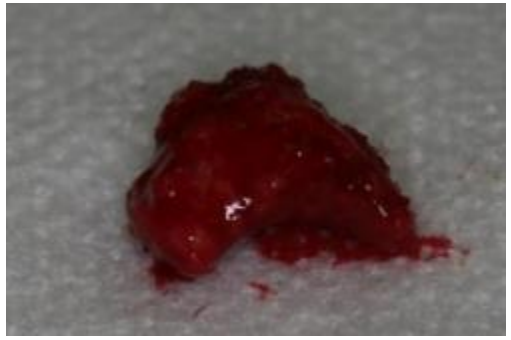

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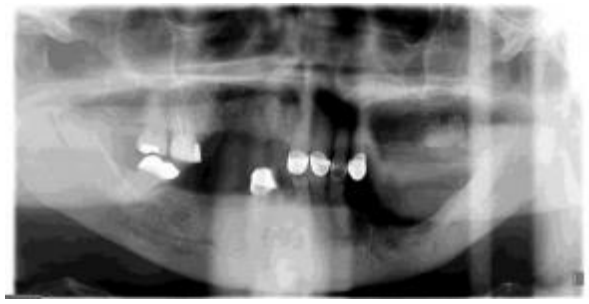

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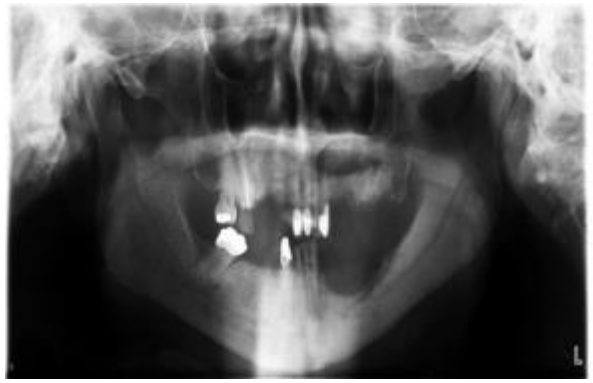


# Protocol for Prevention of BONJ

## Patient at risk before oral surgery/extraction

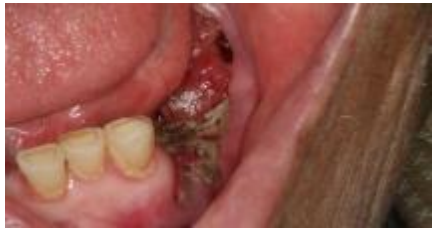
Patient education and Consent form
0.12% Chlorhexidine Gluconate Rinse (3 times/day) 1 week pre-op to until complete healing
Systemic antibiotic 3 days pre-op and 4 days post-op (Amoxicillin 500mg tid or Clindamycin 300mg bid)
Nystatin rinse (3 times/day) 1 week pre-op in high risk patient
Immediate pre-op Chlorhexidine rinse
Weekly or biweekly follow up until complete healing to assess and manage the wound

## Patient with active localized BONJ

Early: 0.12% Chlorhexidine Gluconate Rinse and 1:100,000 units Nystatin (3 times/day) until complete healing, systemic antibiotic if necessary

## Advanced: Surgical therapy and/or systemic antibiotic

Oral medicine consult
Regular follow up



# THANK YOU