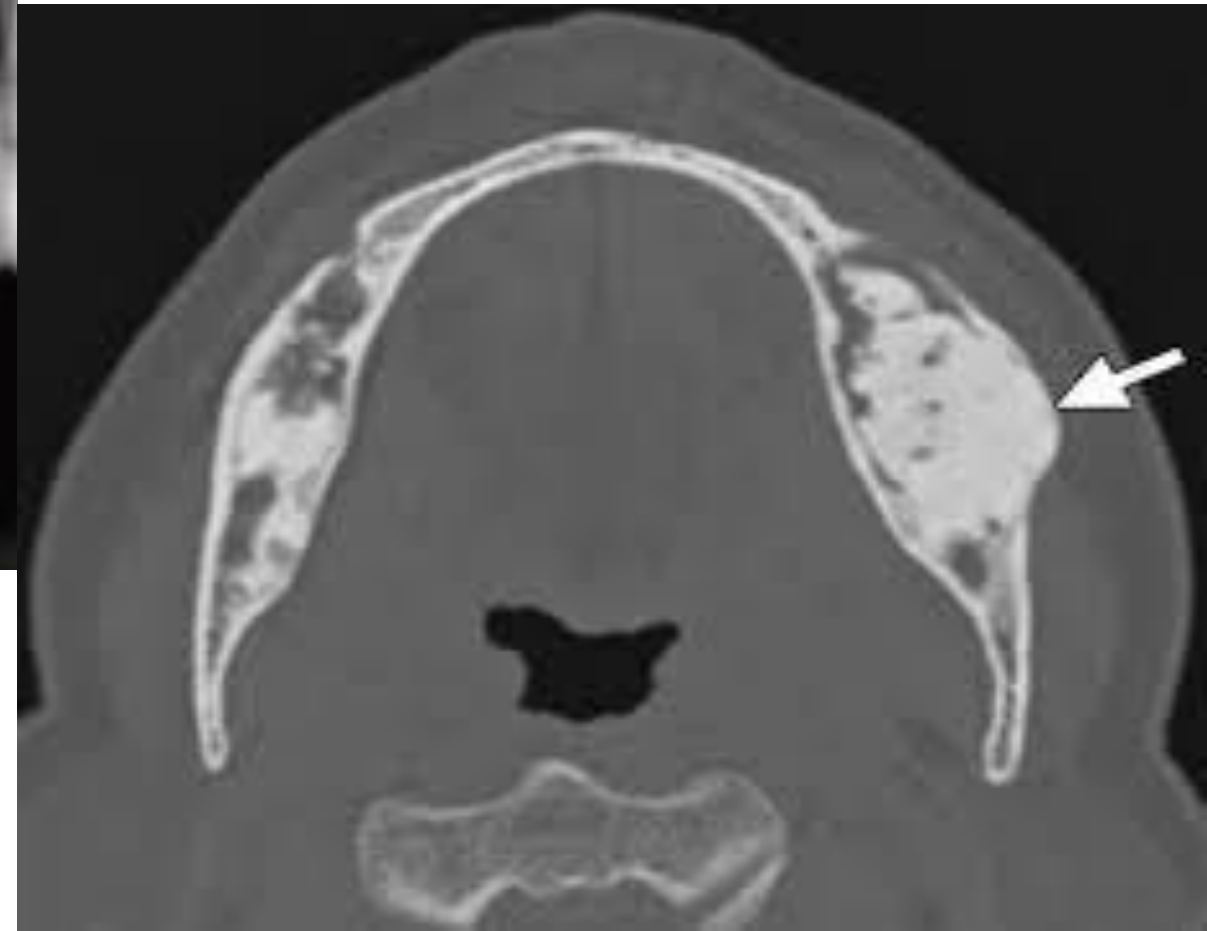
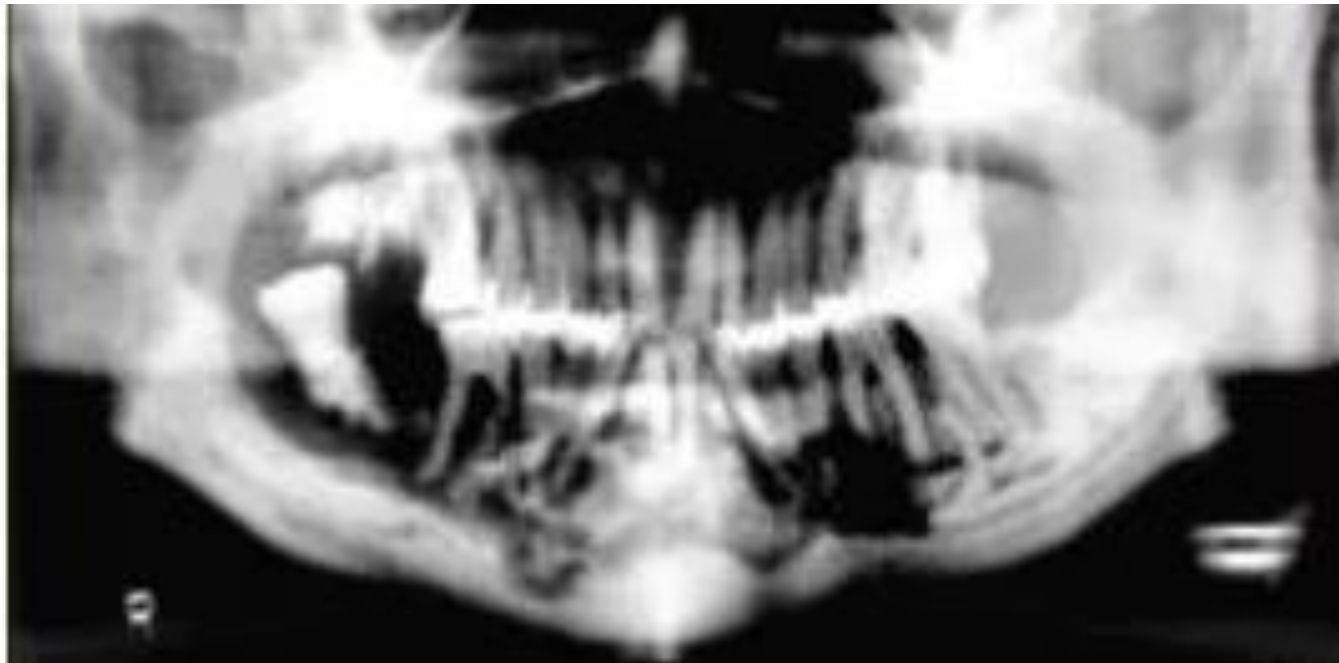


Radiology of inflammatory lesions of the jaws



Inflammation

Inflammation is the most common disease process that the dentist encounters in practice. Whether acute or chronic, localized or generalized, **dentists are responsible for identifying the important radiologic features of inflammation and for determining the extensiveness of bone involvement.**

Imaging plays a key role in this process, and is an important first step in patient diagnosis and management.

Inflammation is the most common pathologic condition arising in the jaws. Unlike the other bones in the skeleton, the jaws are unique in the sense that teeth create a direct pathway for agents or processes, be they pathogenic microorganisms, or chemical or physical agents, to enter bone. **Consequently, the inflammatory response that is mounted by the body attempts to destroy or isolate the injurious stimulus so that an environment can be created for the repair of the damaged tissue.**

The four cardinal signs of inflammation (redness, swelling, heat, and pain) may be observed to varying degrees in the jaws.

Acute lesions tend to have a typically rapid onset, causing pronounced pain. Also, **acute inflammatory** lesions are often accompanied by **fever and swelling.**

In contrast, chronic lesions are generally less severe with a more prolonged course, and the pain is generally less intense. Furthermore, fever may be intermittent and low grade, and swelling may develop gradually. Indeed, low-grade inflammatory lesions may not produce any significant clinical symptoms at all. When the initial source of the inflammatory response is a pathogenic microorganism and a necrotic tooth pulp, the bony lesion that develops is restricted to the tooth root apex.

This condition is referred to as periapical inflammatory disease. When the dissemination occurs through the gingival tissues, the inflammatory response is referred to as periodontal disease if it involves the supporting structures of the teeth or pericoronitis if it involves the crown of a partially erupted tooth. Wider dissemination of microorganisms and the inflammatory response through the bone marrow may be augmented by tissue vascularity and the host immune response. However, in some patients, a pathogenic microorganism or a source of the infection cannot be identified, and hematogenous spread from a distant source is presumed to be the origin. In other cases, the inability to culture a microorganism may be due to prior antibiotic therapy, or the method of bone sampling may be inadequate to reveal small, isolated pockets of infected bone.

When the inflammatory response spreads through the bone via the Haversian system and Volkmann's canals to an adjacent tissue space so that it is no longer localized to the vicinity of the tooth root apex, and there is death or necrosis of bone, **it is called osteomyelitis.**

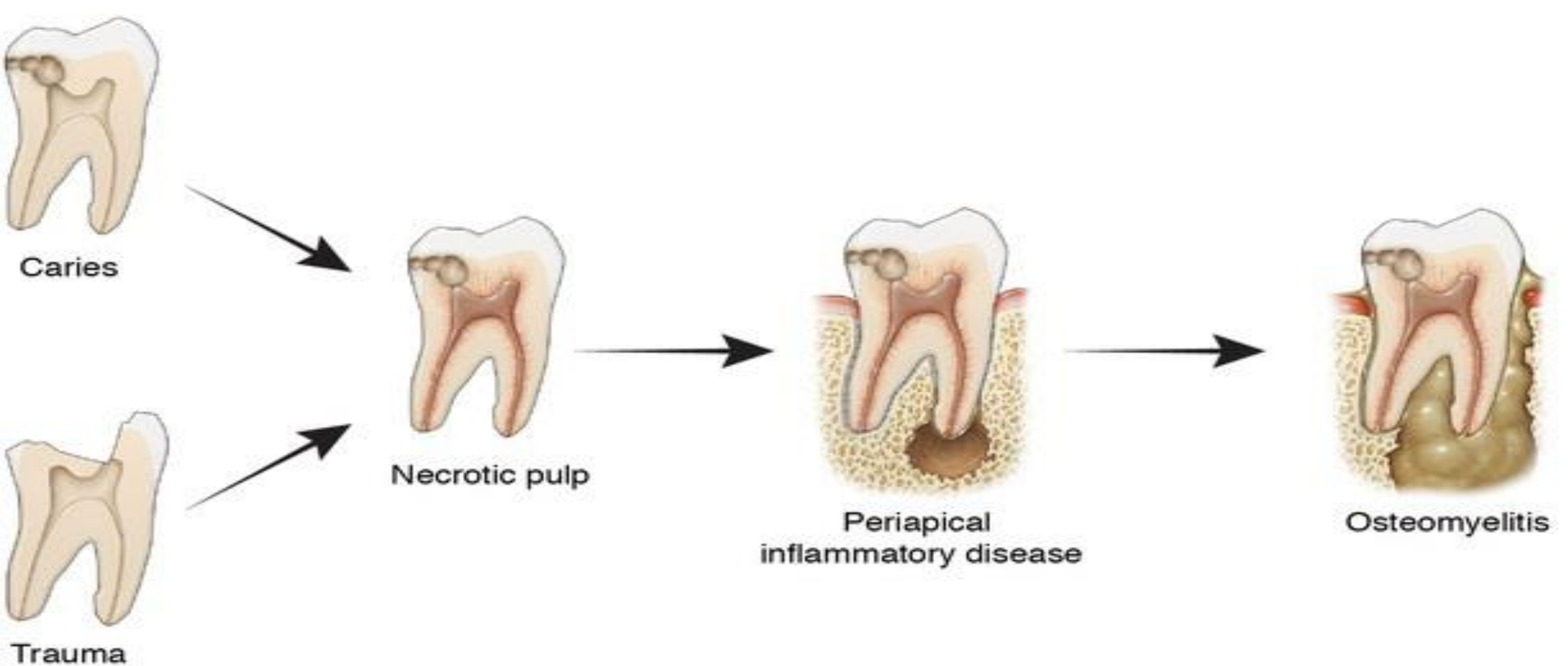
The names of the various inflammatory conditions tend to describe their clinical and imaging presentations and behavior; however, all have the same underlying **disease mechanism, including a common response of the bone to the injury.**

Apical periodontitis (acute and chronic), (periapical, radicular, or periradicular) abscess, (periapical, radicular, or periradicular) granuloma, and (periapical, radicular, or periradicular) cyst are some of the many terms that have been used synonymously to refer to periapical inflammatory disease.



lesion involving the pulp of the second bicuspid without significant change in the **periapical bone** (arrow).

In contrast, note the fusiform lamina dura and periapical bone at the apex of the **mesial root of the second molar**. Also note the subtle halo of sclerotic bone reaction around this apical radiolucency



The development of periapical inflammatory disease at the tooth root apex and dissemination into the adjacent bone

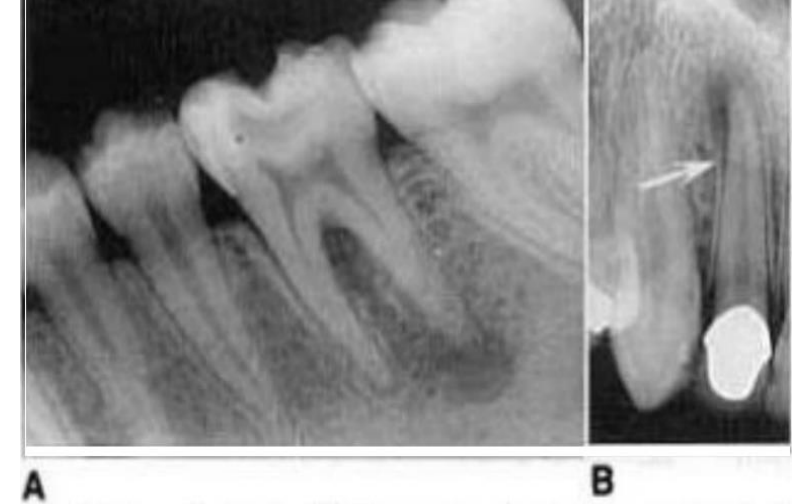
Some of the terms that have been used refer to histopathologic interpretations of disease processes that are associated with characteristic microscopic features that cannot be identified by radiologic imaging. In contrast, the term apical periodontitis is used to describe a disease process that involves the inflammation and destruction of the apical periodontium of pulpal origin (an etiologic link that sometimes cannot be made by simply viewing an image).

“Rarefying osteitis” is the preferred radiologic term when referring to an inflammatory process in bone associated with bone destruction at the apex around a tooth root. In this context, the term “rarefying,” which is derived from the word “rarefaction,” refers to a loss of bone mineralization and the term “osteitis” refers to inflammation of bone.

The net result of this process is an appearance of **increased radiolucency**.

In contrast, the term “**sclerosing osteitis**” is used to refer to an inflammatory process in bone associated with bone deposition around a tooth root. “**Sclerosis**” is a term that refers to a “**hardening**” of the bone and radiologically, this appears as an increase in the radiopacity of bone.

Focal sclerosing osteitis and “**condensing osteitis**” have both been used synonymously to refer to this latter condition, although the use of the term “condensing” as an adjective that describes the biologic response of bone to inflammation should be discouraged



Periapical inflammatory lesions associated with a mandibular first molar (

A) and a maxillary lateral incisor

B) Note that in both cases the epicenter of bone destruction is located at the apex of the root. Also, note gradual widening of the periodontal membrane space (arrow) characteristic of an inflammatory lesion.

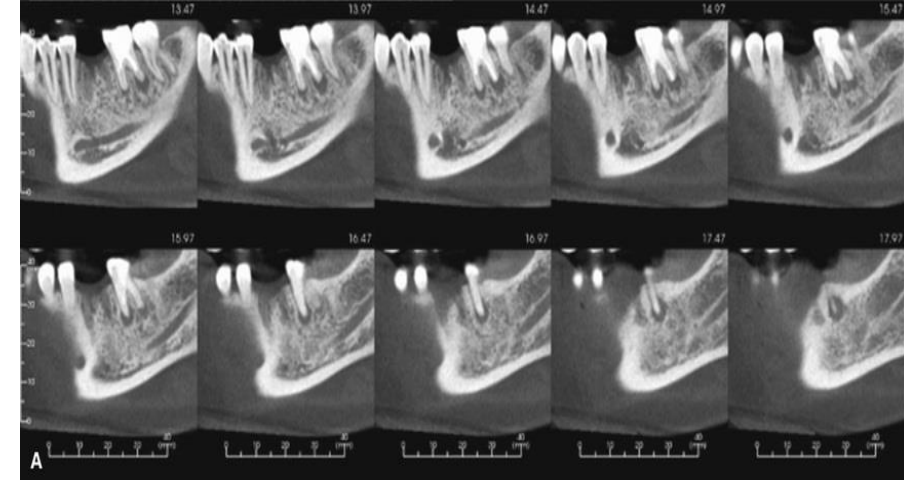
In the early stages of **periapical inflammatory disease** there may be little effect on the adjacent bone. As the disease evolves, bone deposition may be seen around the focus of rarefaction, altering the normal morphology of the trabecular

bone pattern and marrow spaces. The degree to which the appearance of periapical inflammatory disease is radiolucent (rarefying osteitis) or radiopaque (sclerosing osteitis) is variable.

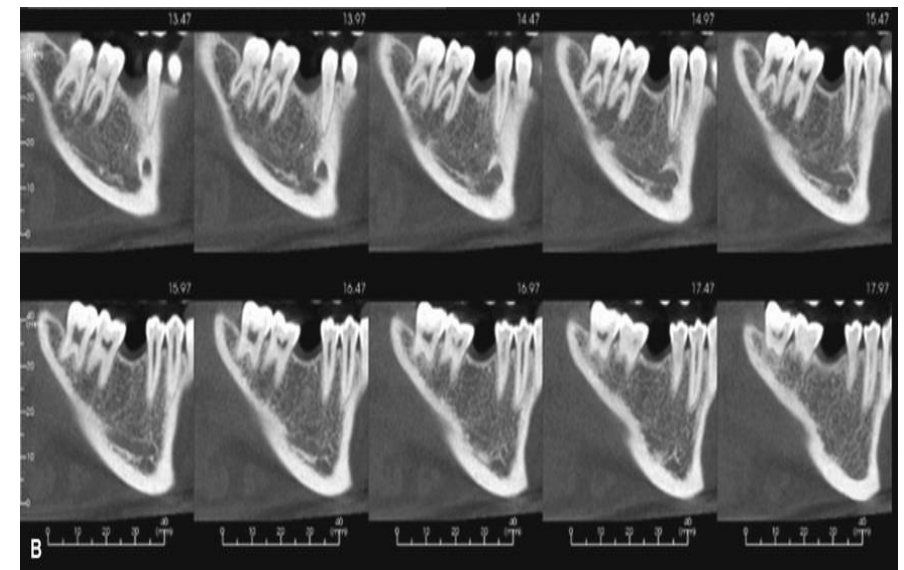
Closer inspection of the peripheral sclerotic regions reveals thicker than normal trabeculae and sometimes an increase in the number of trabeculae per unit area. With time, the new bone formation may result in a very dense sclerotic region of bone, obscuring individual trabeculae and reducing the size of the marrow spaces.

Occasionally, the lesion may appear to be composed entirely of sclerotic bone (sclerosing osteitis), but usually some evidence exists of apical periodontal ligament space widening that may persist

In some instances, the sclerotic reaction of the bone may be localized to a small region around the tooth root apex. However, given the aggressiveness of the pathogenic microorganism and the exuberance of host immune response, as well as tissue vascularity and chronicity, the sclerotic response may extend to adjacent teeth or to a bone border.



Corrected sagittal cone beam computed tomography images of rarefying and sclerosing osteitis affecting the roots of a mandibular left second molar. Note the loss of normal trabecular bone pattern (A) compared with the unaffected side (B). The sclerotic response extends to the inferior cortex of the mandible. This change gives a particular prominence to the inferior alveolar canal



Several examples of a mixture of rarefying and sclerosing osteitis. Note the similarity of the pattern, composed of a radiolucent region at the apex of the tooth sur rounded by a radiopaque reaction of sclerotic dense bone. Also note that most often a gradual transition occurs from the sclerotic bone reaction to the more normal surrounding bone pattern.



Periostitis emanating from the floor of the maxillary antrum that arises secondary to apical inflammatory lesions. A, Laminated type of periostitis (arrow), B, Periostitis and mucositis.

The mucositis is characterized by a slight radiopaque band (arrow).

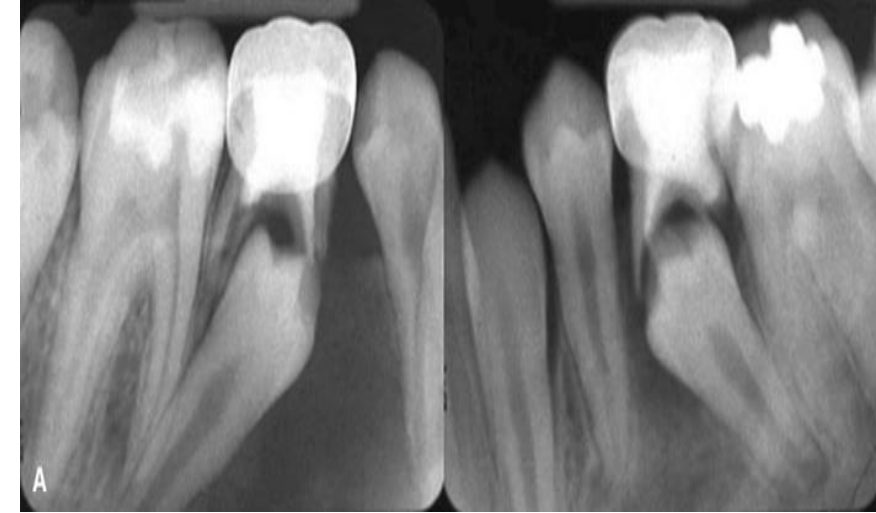


Periapical Sclerosing Osteitis Associated With the First Molar. This is called a sclerosing lesion because most of the lesion is bone forming, resulting in a very radiopaque density. Note, however, the small region of rarefaction at the root apex and the widening of the periodontal ligament space

Effects on Adjacent Teeth:

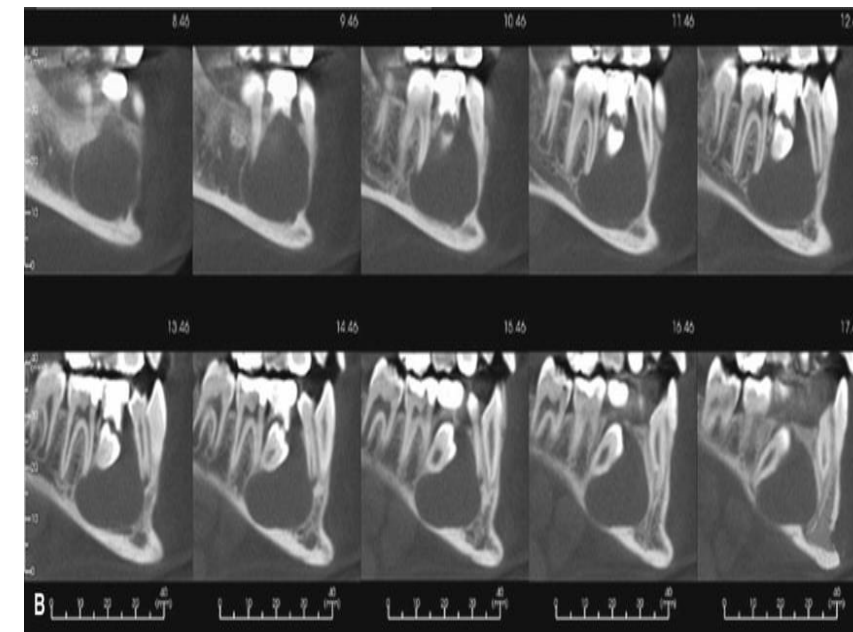
In many regards, the response of a tooth to periapical inflammatory disease, irrespective of whether or not the tooth is the reason for the inflammatory response or an adjacent tooth, closely mirrors the response of the bone to inflammation. In some instances, tooth roots can undergo external resorption and this may be seen on an image as a change in the normally smooth, tapering surface contour of the root. Alternatively, tooth roots may undergo hypercementosis, and this may be visualized as bulbous-shaped roots. The response of the tooth root, whether external resorption or hypercementosis, may be asymmetric and not uniform around the circumference of the root. When rarefying osteitis involves deciduous teeth, eruption of the underlying permanent dentition may be disrupted

The diagnosis may rely solely on the **clinical examination**, which often includes a **test of tooth pulp vitality**. Depending on the **age of the patient** and the **chronicity of the periapical inflammatory disease**, the pulp chamber of the involved tooth may be larger than the adjacent teeth. More mature PCOD lesions may show the development of a radiopaque focus centrally within the radiolucent area, which helps in the differential interpretation since the radiopacity seen with periapical inflammatory disease occurs at the periphery of the radiolucent focus. External root resorption is also more commonly identified in inflammation than in PCOD



Periapical images of two areas of rarefying osteitis associated with the mandibular right and left second deciduous molars that have undergone pulpectomy procedures.

Corrected sagittal and buccolingual cross-sectional





Fibrous Scars After Successful Orthograde Endodontic Treatment. An alternating pattern of concentric radiolucent and radiopaque rings extends inward from the periphery of the healed periapical inflammatory lesion (A). A pattern of internally radiating or spoke-like radiopaque lines also can be seen (B).

Two early lesions of periapical cemento-osseous dysplasia located at the apical region of the mandibular central incisors. Note the similarities to rarefying osteitis



Infiltrate of chronic lymphocytic leukemia appearing as a periapical radiolucent lesion

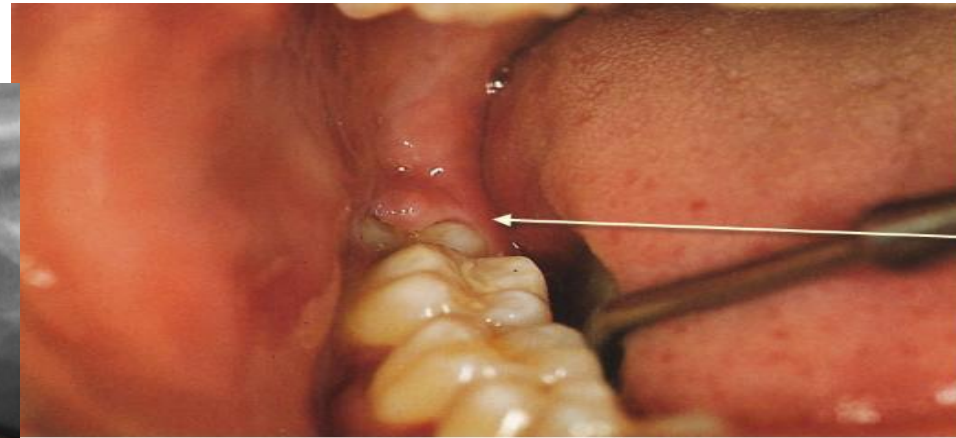
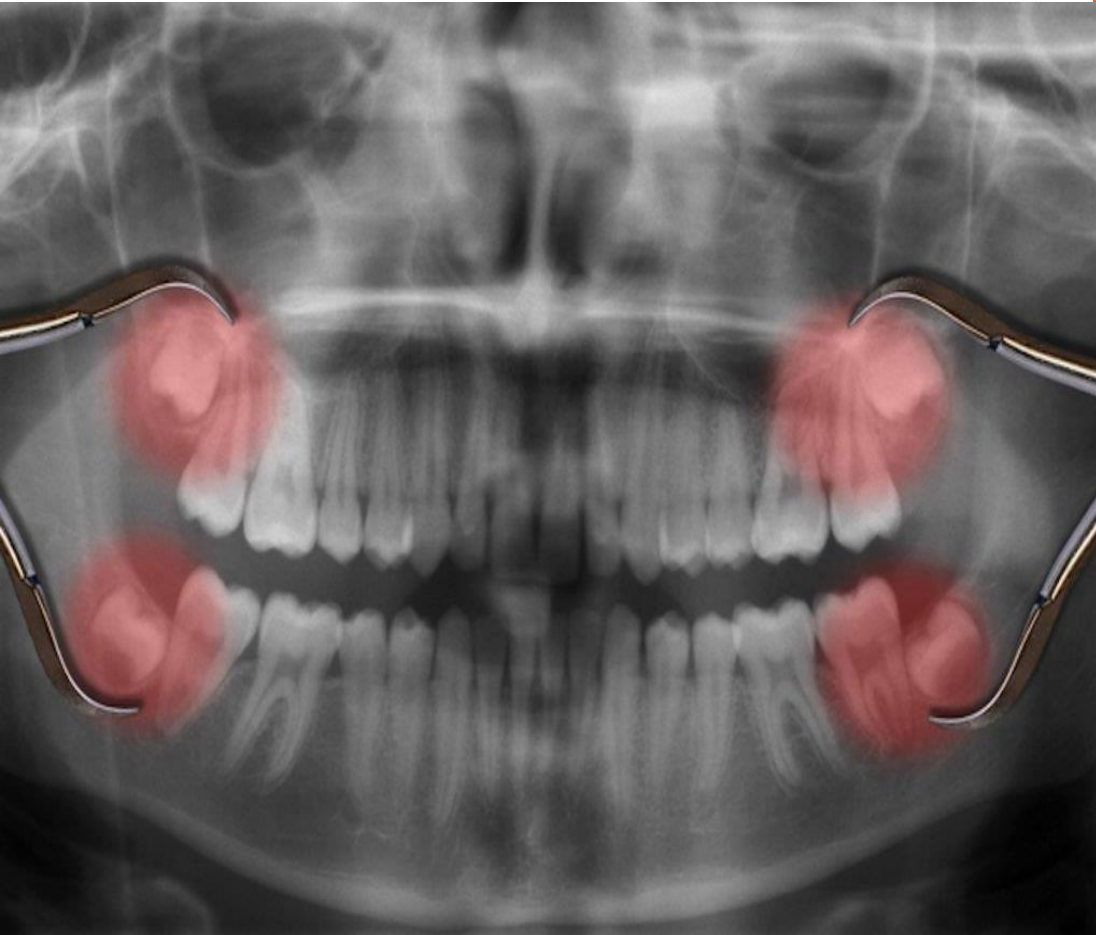
In rare cases, metastatic lesions and blood-borne malignancies, such as leukemia, may develop within the periapical portion of the periodontal ligament space.

Close inspection of the surrounding bone may reveal other bone changes, which may include small regions of cancellous bone destruction

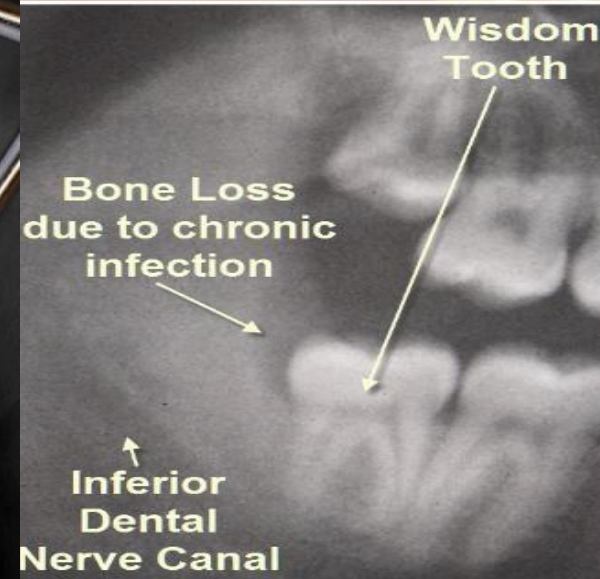
Pericoronitis

Pericoronitis is **swelling and infection of the gum tissue around the wisdom teeth**, the third and final set of molars that usually appear in your late teens or early 20s. It is most common around the lower wisdom teeth.

Pericoronitis occurs around a wisdom tooth that has failed to come in or has only partially erupted. A partially-erupted wisdom tooth can leave a flap of gum tissue that collects food particles and other debris—an ideal breeding ground for bacteria.



LR8 - Partly erupted, inflamed & swollen operculum



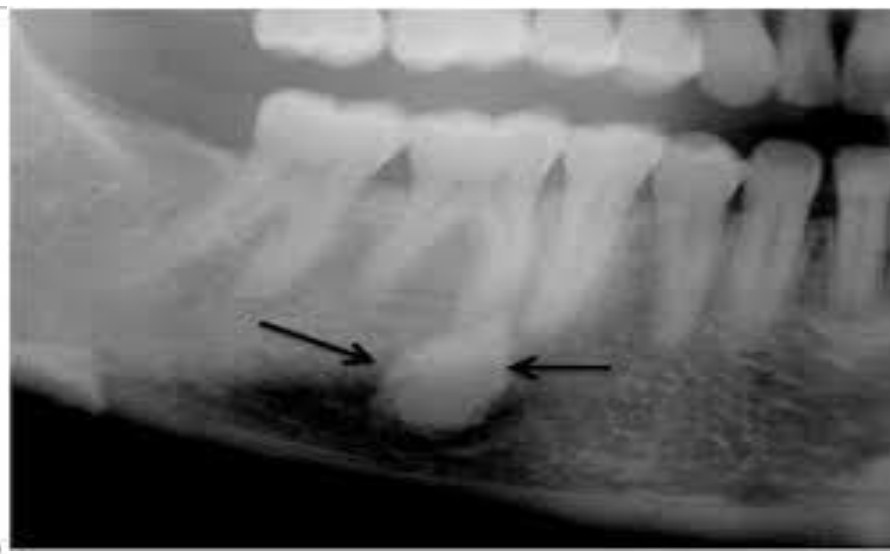
Pericoronitis - Clinical & Radiographical Appearances

LR8 - X-Ray showing an 'arcuate' zone of radiolucency behind the wisdom tooth, indicating chronic infection

Enostosis:

A bone island (enostosis) is a **noncancerous (benign) lesion that rarely causes symptoms.**

It's a tiny, dense piece of bone that **grew within another section of the jaw bone or any bone** specifically, a piece of compact (**cortical**) bone within cancellous bone (a network of spongy bone tissue).



Enostosis (dense bone island) in periapical positions.

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Panoramic view of a case of pericoronitii related to a partially erupted third molar. Note the sclerotic bone reaction adjacent to the follicular cortex (black arrow) and the periosteal reaction (white arrow).



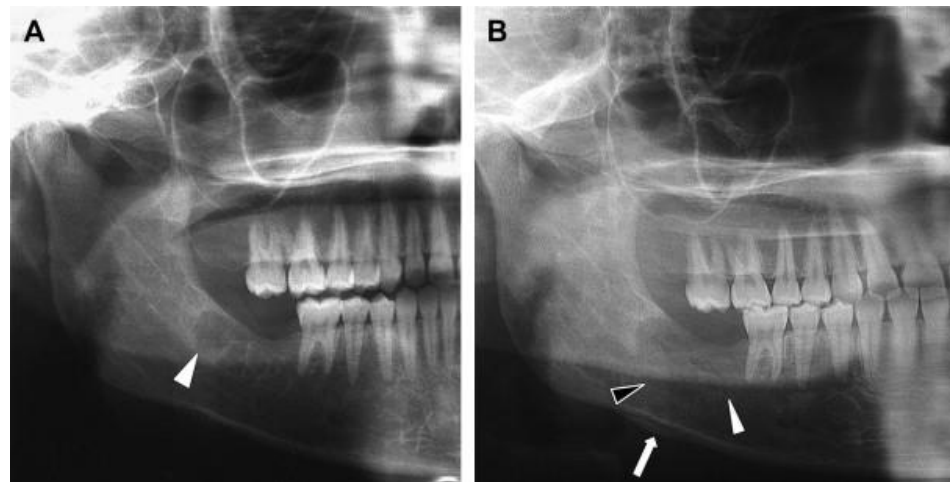
Osteomyelitis:

The root “myel” in the term osteomyelitis refers to bone marrow, and therefore the disease process osteomyelitis is used to describe inflammation of the bone marrow. It is noteworthy that rarefying osteitis can also involve the bone marrow, so most authors use the term osteomyelitis to refer to a more widespread response of the bone to inflammation that includes not only the marrow, but also the cortical and cancellous bone as well as the periosteum. However, even this description may be inadequate. As we have seen in the previous section, even lesions of rarefying osteitis can extend to a bone periphery to involve the cortex and periosteum.

Where a localized focus of rarefying osteitis becomes more widely disseminated involves some subjectivity. A hallmark feature of osteomyelitis is the identification of a sequestrum (pl. sequestra)—one or more fragments of diseased bone that are losing or have lost their blood supply, and have undergone necrosis as a consequence of this ischemic injury.

Osteomyelitis may resolve spontaneously or with appropriate antibiotic therapy. However, if the condition is not effectively managed, the infection agent may persist and continue to spread in some patients; particularly those with preexisting chronic systemic diseases, immunosuppressive states, and disorders of decreased vascularity (e.g., osteopetrosis, sickle cell disease, and HIV/AIDS).

OSTEOMYELITIS

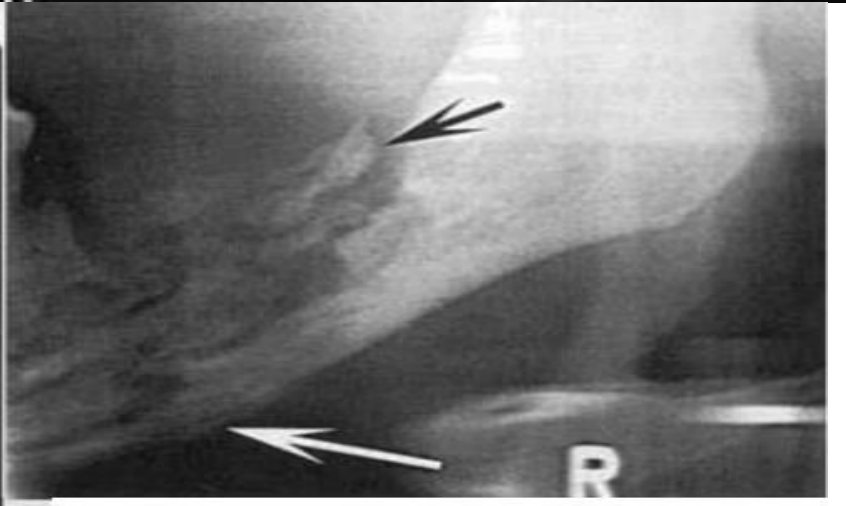




Acute osteomyelitis involving the body of the right mandible, with initial blurring of bony trabeculae



A and B, Proliferative the multiple layers of new bone on onion-skin appearance



Examples of sequestra. A, Occlusal film demonstrates small sequestra as radiopaque islands of bone in radiolucent regions in the chronic phase of osteomyelitis (arrows/), Panoramic film reveals large sequestra (Black arrow) and a periosteal reaction at the inferior border of the mandible in a case of chronic osteomyelitis (white arrow).

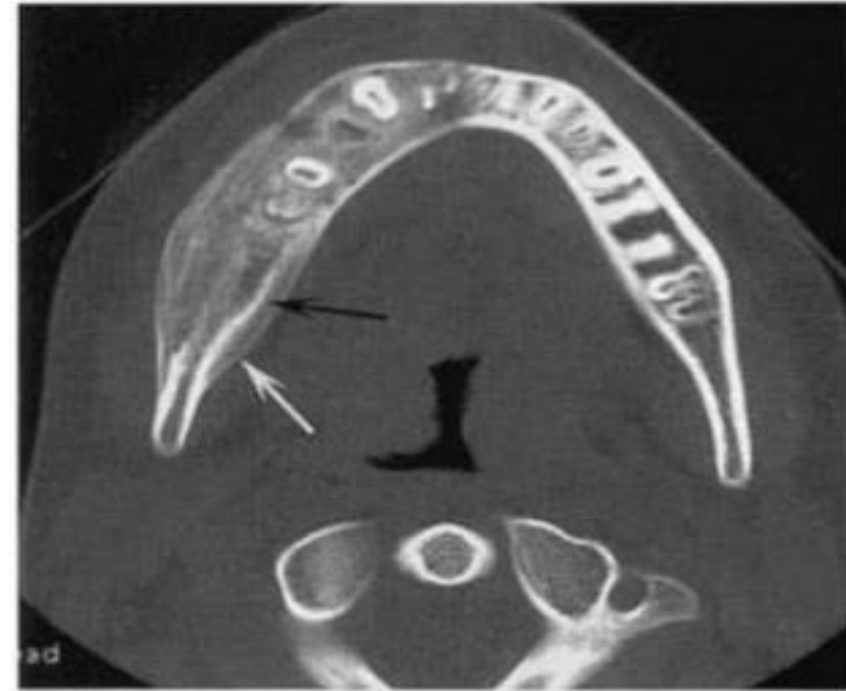
Chronic osteomyelitis. A, This panoramic film demonstrates chronic osteomyelitis of the patient's right mandible. Note the increase in density and size of the right mandible compared with the left side B, An axial CT image using bone window of the mandible of the same case. Note the increase in bone density, width of the mandible and the new periosteal bone formation (*black arrow*) and evidence of the original cortex (*block arrow*).



A



A



A

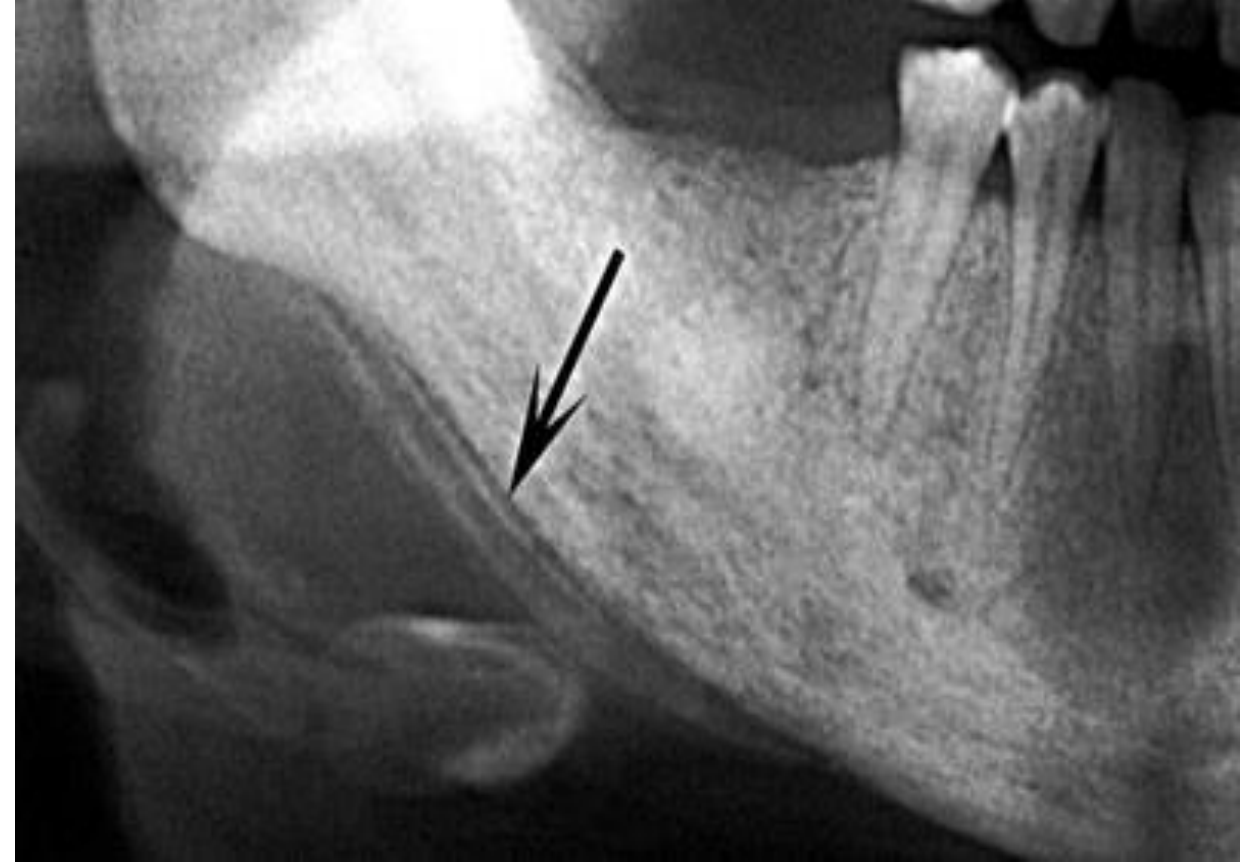
Periapical image (A) showing a mixed radiolucent and radiopaque pattern at the apices of a mandibular right first molar. The cross-sectional occlusal image shows periosteal new bone formation on both the buccal and lingual surfaces of the mandible (B).



B

In some instances, the new bone may be very faint depending on its degree of mineralization. As the lesion develops into a more chronic phase, new bone (bone arcades) may continue to extend into the adjacent underlying connective tissue to support the new surface. The cyclic and periodic acute exacerbations may lift the periosteum again, stimulating the outer osteogenic layer to form a second layer of bone.

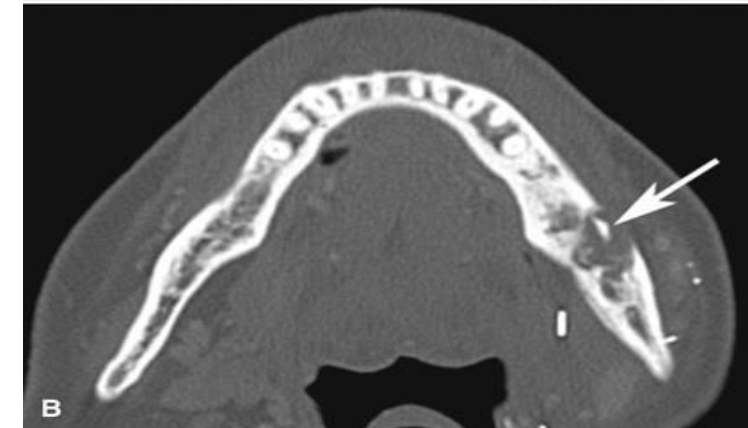
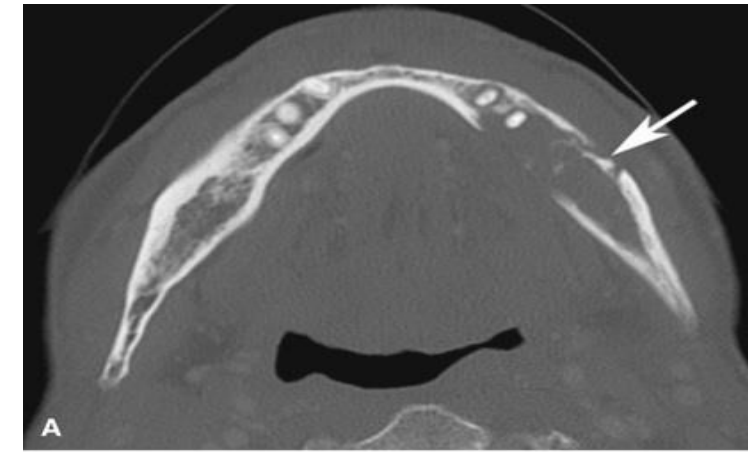
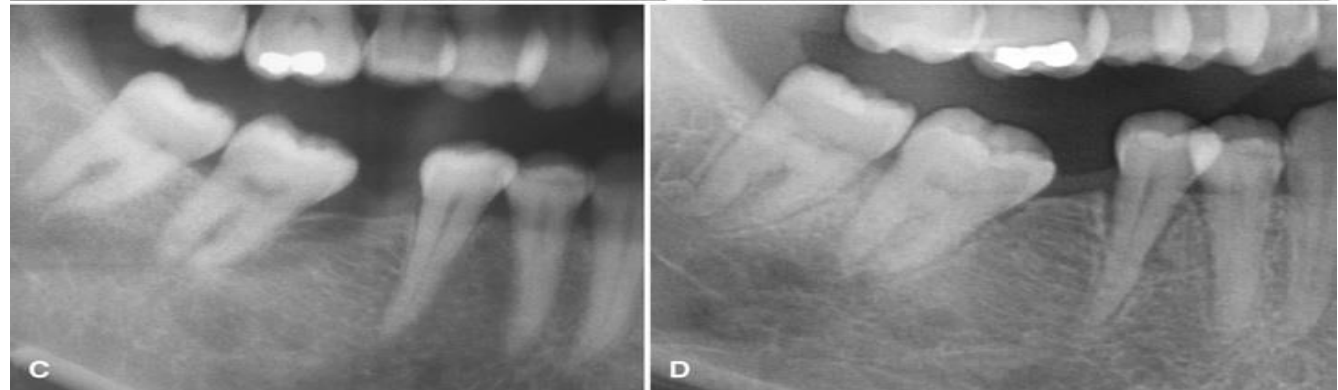
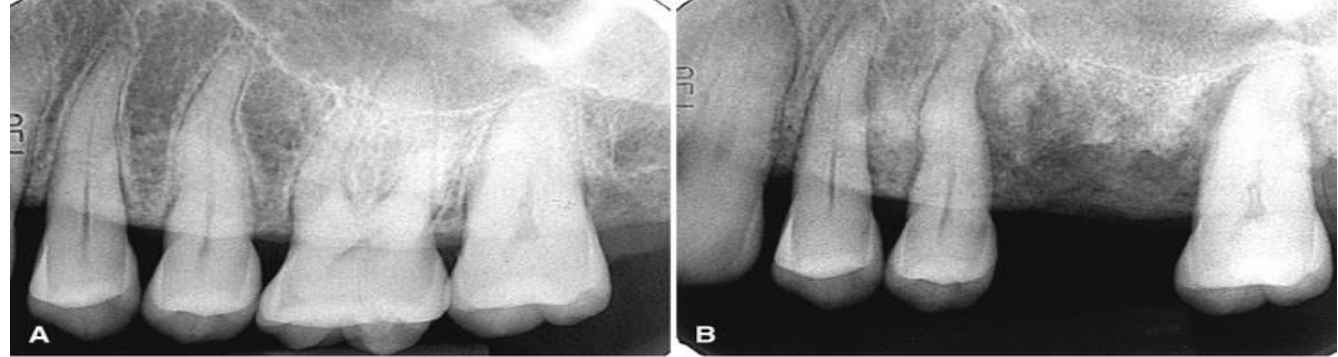
This second layer can be detected in the image as a second radiopaque line almost parallel to the first and separated from it by a radiolucent band. This process may continue and may result in several lines (the “**onion-skin**” appearance), and eventually a massive amount of new bone may be formed adjacent to the native bone surface. This condition is referred to as proliferative periostitis and is seen more often in children. The exuberance of the periosteal response in children reflects a thicker and more loosely attached periosteum than in adults, and the higher osteogenic potential of periosteum in younger people



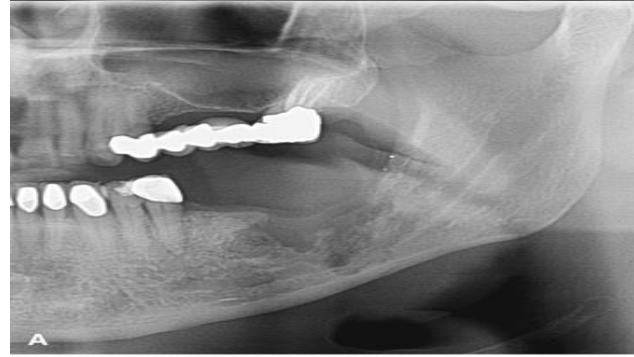
Osteomyelitis of the Mandible With a Periosteal Reaction Located at the Inferior Cortex. Note the radiolucent line (arrow) between the inferior cortex of the mandible and the first layer of periosteal new bone. A second radiolucent line separates the second layer of new bone from the first layer

Changes in the Maxillary Dentition After Therapeutic Radiation Exposure.

Periapical images were made prior to (A) and within 6 months of receiving the radiation (B). Note the combination of bone destruction and sclerosis, and the nonconcentric widening of the periodontal ligament space. In another case, the cropped panoramic image (C) shows the condition of the bone and teeth prior to radiation and thenonconcentric widening of the ligament space after radiation (D).



Examples of Radiation Induced Necrosis (Osteoradionecrosis). Axial multidetector computed tomography images showing extensive bone resorption and sequestra (arrow) (A). The sclerotic response is more prominent in (B) as is the periosteal response on the lingual surface of the left mandible



Cropped panoramic image of a patient with bone resorption secondary to therapeutic radiation exposure (A) and the development of a fracture secondary to the loss of bone integrity in the same patient

