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Review Article

Osteomyelitis of the Jaw Bones and Its Mimics: Resolving the Diagnostic Enigma

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Abstract

Jaw osteomyelitis is a severe inflammatory condition affecting the maxilla or mandible, posing diagnostic and therapeutic challenges. An early and accurate diagnosis is crucial to initiating appropriate treatment and preventing potential complications. Radiological imaging plays a pivotal role in diagnosing and evaluating jaw osteomyelitis, providing valuable insights into the extent of the disease and aiding clinicians in making informed decisions. One of the most important aspects of imaging is to differentiate them from the noninfectious mimics, which can closely mimic their imaging appearance. This article aims to present a comprehensive overview of imaging in jaw osteomyelitis, focusing on the critical clinical and imaging markers that distinguish it from its noninfectious imitators.

Keywords

- jaw osteomyelitis
- mandible
- maxilla
- osteoradionecrosis
- osteonecrosis
- CT
- CRMO
- Caffey disease

Introduction

Osteomyelitis typically refers to an inflammation of the medullary cavity of bone, which can be acute or chronic in clinical presentation. In jaw bones, it occurs more commonly in the mandible than the maxilla, which is related to the reduced blood supply to the mandible. Thick cortical plates and abundant medullary tissues in the mandible contribute to the confinement of the infection within the bone. Osteomyelitis in the maxilla follows the same etiopathogenesis as the rest of the face, while in the mandible, a different etiologic spectrum is observed. Due to its insidious onset and variable clinical presentation, an accurate diagnosis often requires a combination of clinical findings, laboratory tests, and imaging techniques. Also, it is important to distinguish osteomyelitis from conditions that resemble it but are not caused by infection, that is, the noninfectious mimics, to direct appropriate management.

Approach to Jaw Osteomyelitis

Before we describe specific entities, let us review how to manage a suspected case of osteomyelitis based on its location (maxilla or mandible), clinical presentation (acute or chronic), and imaging morphology (mainly lytic or sclerotic).

Mandibular Osteomyelitis

Infectious mandibular osteomyelitis is suspected when imaging shows lysis of the involved bone with discharging sinuses or periosteal reaction, with or without sclerosis and adjacent soft tissue involvement. Clinical information and predisposing factors then play a major role in differentiating between various causes and noninfectious mimics (►**Fig. 1**).

Before labeling osteomyelitis, it is prudent to rule out malignant etiologies like oral cavity carcinoma, primary

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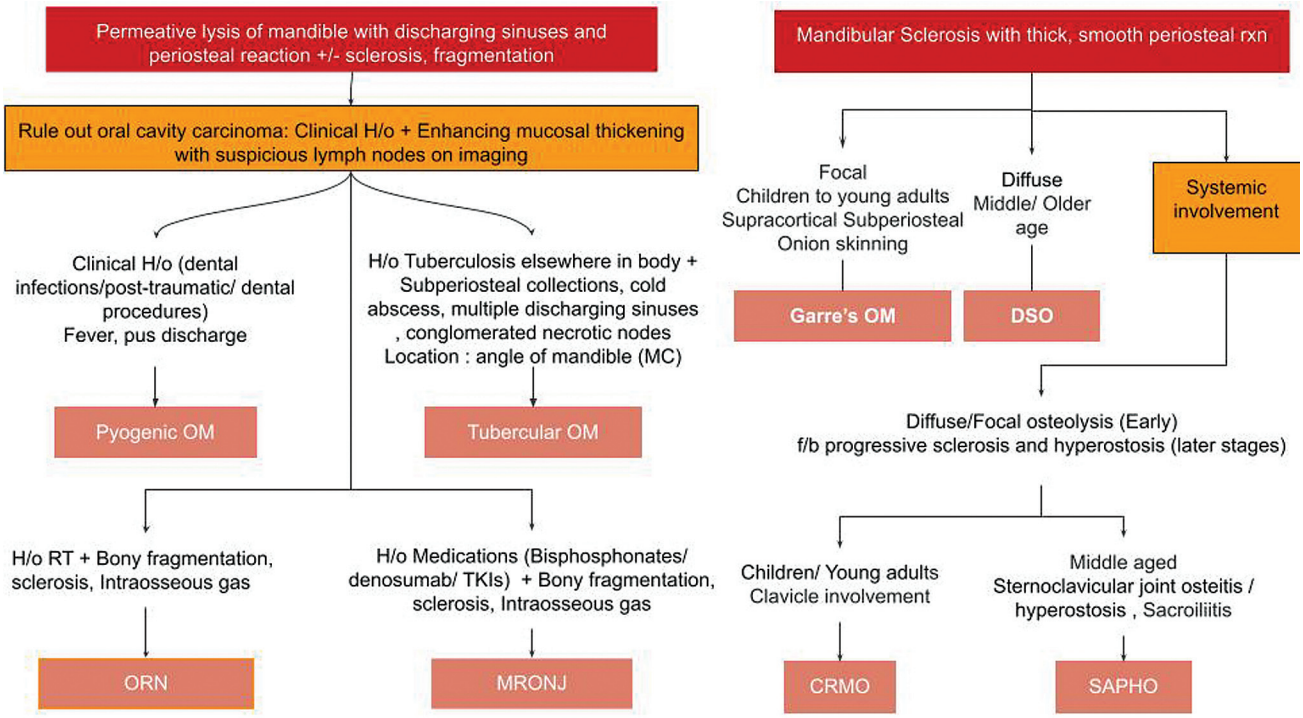

Fig. 1 Algorithmic approach to mandibular osteomyelitis.

bone malignancy, or lymphoma. Broadly, these entities are usually associated with disproportionately enhanced mucosal thickening, permeative lysis, aggressive periosteal reactions, and suspicious lymph nodes. Also, in an appropriate clinical scenario, one should consider noninfectious causes, such as osteoradionecrosis or medication-related osteonecrosis of the jaws (MRONJ).

A unique imaging appearance is when the predominant finding is sclerosis with a thick, smooth periosteal reaction. In these cases, age group, location, and presence or absence of systemic features help narrow the differential diagnosis. While focal sclerosis in the supracortical or subperiosteal region in young patients without systemic symptoms typically indicates Garre's osteomyelitis, diffuse sclerosis in middle-aged or older patients suggests diffuse sclerosing osteomyelitis. In cases of diffuse distribution in children with systemic involvement, conditions such as chronic recurrent osteomyelitis, SAPHO syndrome (synovitis, acne, pustulosis, hyperostosis, osteitis syndrome), etc. are also to be considered.[1,2]

Maxillary Osteomyelitis

Classical maxillary osteomyelitis is typically considered when there is a destructive maxillary lesion with mild soft tissue and a periosteal reaction, with or without palatal involvement, in a clinical scenario suggestive of infection (►**Fig. 2**). If imaging depicts exuberant soft tissue with palatal or nasal septal involvement, then malignant etiologies especially lymphoma should be ruled out first (►**Fig. 3**). A history of inhalational substance use should always be sought to rule out cocaine-related osteonecrosis, while other important differentials include sarcoidosis and granulomatous polyangiitis.[3,4]

Infectious Osteomyelitis

It is also known as classical osteomyelitis or suppurative osteomyelitis. The mandible is most commonly affected by bacterial or tubercular osteomyelitis, as opposed to the maxilla, where fungal infection is the leading cause, often from chronic invasive fungal infections, followed by odontogenic infections of mostly bacterial origin (►**Table 1**).

Most cases of bacterial osteomyelitis are secondary to contiguous odontogenic infections, whereas hematogenous spread is the primary cause of mandibular tuberculosis.

Bacterial: A clinical history of dental infections, recent trauma, or dental procedures should prompt bacterial etiology. The characteristic clinical findings of bacterial osteomyelitis are pain, swelling, and suppuration (from the fistular tract). The symptoms may be severe or mild, and there may be a mixture of severities in any given case.

On imaging, bone resorption is prominent, and radiography shows an osteolytic pattern. In long-standing lesions or cases with preexisting sclerosing osteomyelitis, sclerotic areas may be seen around the osteolytic area. On plain radiographs, the margin of bone resorption is ill-defined but may be well-defined at the site of cortical bone resorption (perforation). The periosteal reaction is usually lamellated and appears as a thin, faint, radiopaque line adjacent to, and almost parallel to, or slightly convex to, the surface of the bone. A radiolucent band separates the new periosteal bone from the bone surface. If the process occurs repeatedly, an "onion skin" appearance is observed, caused by the presence of multiple lamellae. On computed tomography (CT) images, the pattern of osteolytic change in the bone is continuous, not scattered, and spreads to the periosteum through sites of

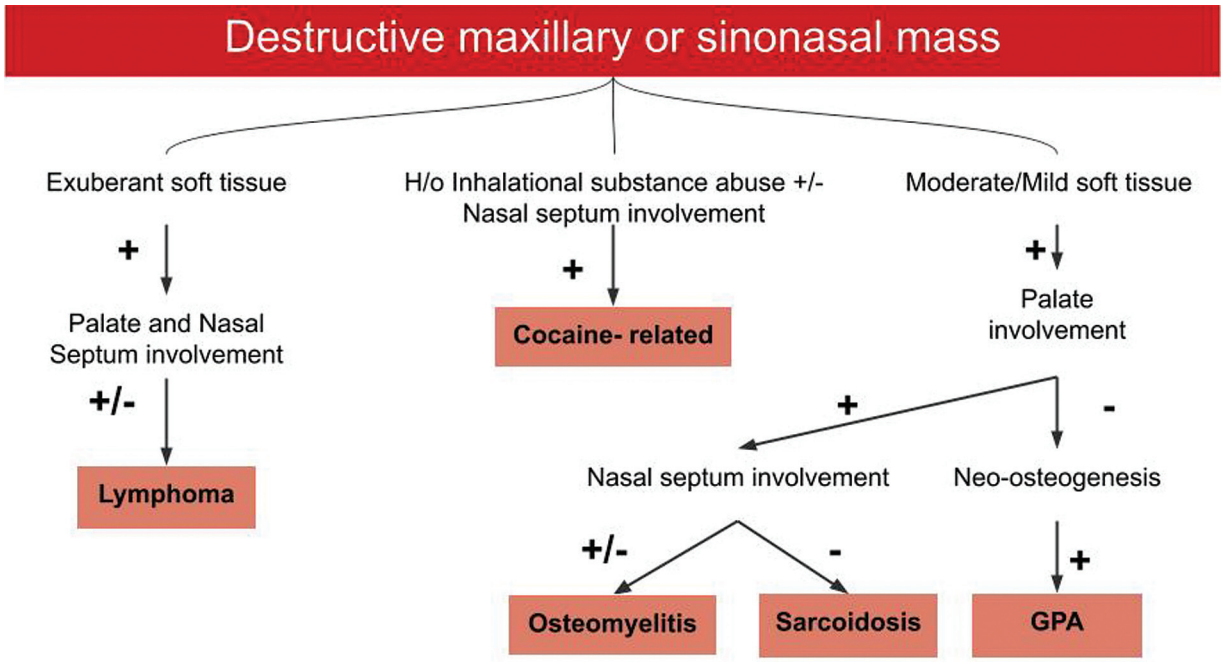

Fig. 2 Algorithmic approach to maxillary osteomyelitis.

perforation of the cortical bone (cloacae). The margin of the zone of cortical bone resorption is moderately well-defined, and the remaining cortical bone appears to be of almost normal density. Periosteal reactions may be seen around the site of perforation (►**Fig. 4**).

Magnetic resonance imaging (MRI) plays an increasing role in the diagnosis of the early acute stage of osteomyelitis. It is the most sensitive and specific modality to image the medullary cavity and can identify soft tissue/joint complications.[5,6] Bone marrow edema is the earliest feature of the acute stage and can be detected as early as 1 to 2 days after the onset of infection.[7] Concordant low signal T1 and high signal on fluid-sensitive sequences are the characteristic appearances on MRI (►**Fig. 5**). In the context of cellulitis, the stage that precedes osteomyelitis, abnormalities of the marrow signal may be subtle on both T2 and T1 images. To be called "osteomyelitis," contrast enhancement with a signal increase is therefore required. In cases where significant edema is present, the extent of T1 and T2 signal abnormalities exceeds the location of contrast enhancement. Fat-suppression techniques allow better discrimination of the area of contrast enhancement from normal bone marrow.[1]

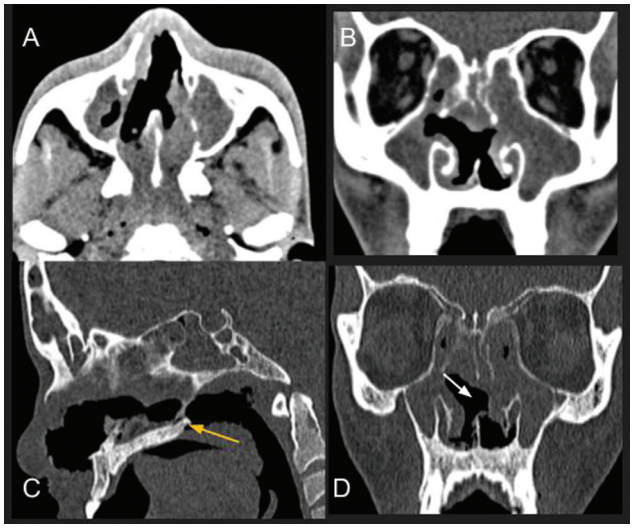

Fig. 3 Soft tissue (**A**, **B**) and bone window computed tomography (CT) images (**C**, **D**) of a 30-year-old male with on- and off-nasal bleeding for 2 months show extensive sinonasal mass-like soft tissue thickening in the nasal cavity and maxillary antrum extending to the nasal skin and subcutaneous tissue with septal and turbinate bony destruction (white arrow in **D**), roomy nasal cavities, and hard palate typically spared (yellow arrow in **C**). Biopsy-proven natural killer (NK) cell lymphoma.

The interpretation of bone marrow abnormalities on MRI images has to take into account the age-related conversion of hematopoietic marrow into fat-containing cancellous bone. This commences within the mandibular body in the second decade of life. The angle and ramus follow in sequence until marrow conversion progressively reaches the mandibular condyle by the third decade.

The "penumbra" sign has been described as an additional finding related to the subacute phase of acute osteomyelitis.[8] It consists of a peripheral zone of slightly elevated signal intensity on T1 images that surrounds the central bony abscess cavity. It corresponds to a layer of granulation tissue that lines the abscess cavity. The penumbra sign is bordered by a zone of peripheral edema and reactive new bone formation.

Tubercular: Primary tuberculosis of the mandible is a rare occurrence. The rarity of mandibular tuberculosis has been attributed to the paucity of cancellous bone in the mandible, with the angle and the alveolar regions being affected most frequently. Mandibular tuberculosis is often insidious, and patients usually cannot recall when the symptoms started. In

Table 1 Infectious causes of jaw osteomyelitis

Characteristic features	Bacterial	Tubercular	Fungal
Clinical h/o	Dental caries, posttraumatic, dental procedures	H/o tuberculosis elsewhere in the body	Immunocompromised, diabetic patients, steroid intake
Location	Mandible > maxilla	Mandible > maxilla	Maxilla > mandible
Spread	Odontogenic > rhinosinogenic	Hematogenous	Contiguous sinonasal infections
Imaging	Areas of increased radiopacity with loss of bone trabeculae (radiolucent) Cortical erosion Sequester formation, fistulae Calcified periosteal reaction Pathological fractures	Same as bacterial Subperiosteal abscess formation Intraorally or externally, multiple discharging sinuses (MC along the inferior border of the mandible) Manifestations of pulmonary or extrapulmonary TB	Mucosal thickening of the paranasal sinuses Ill-defined radiolucencies loss of definition of lamina dura of involved teeth Destruction of the maxilla, zygoma, inferior rim of the orbit, palatal bones, and nasal floor Rarely bony sequestra

Abbreviations: h/o, history of; MC, most common; TB, tuberculosis.

a few cases, it appears as an acute inflammatory swelling that fails to resolve with the use of conventional antibiotics. Since mandibular tuberculosis is rare, clinicians frequently confuse this with a pyogenic abscess, and if a discharging sinus is present, it can be misdiagnosed as actinomycosis. It is suspected when there is a history of tuberculosis elsewhere in the body with subperiosteal collections, cold abscess, multiple discharging sinuses, or conglomerated necrotic nodes.[9,10]

Mandibular tuberculosis begins as an area of rarefaction with trabecular blurring. Gradually, erosion of cortical bone occurs, which is then replaced by soft granulation tissue, and subsequently, subperiosteal abscess formation takes place, culminating in a visible, painful swelling (►**Fig. 6**). The granulation tissue undergoes caseation necrosis, leading to liquefaction, which may burst either intraorally or externally, leading to multiple discharging sinuses, mostly along the inferior border of the mandible or sometimes in the

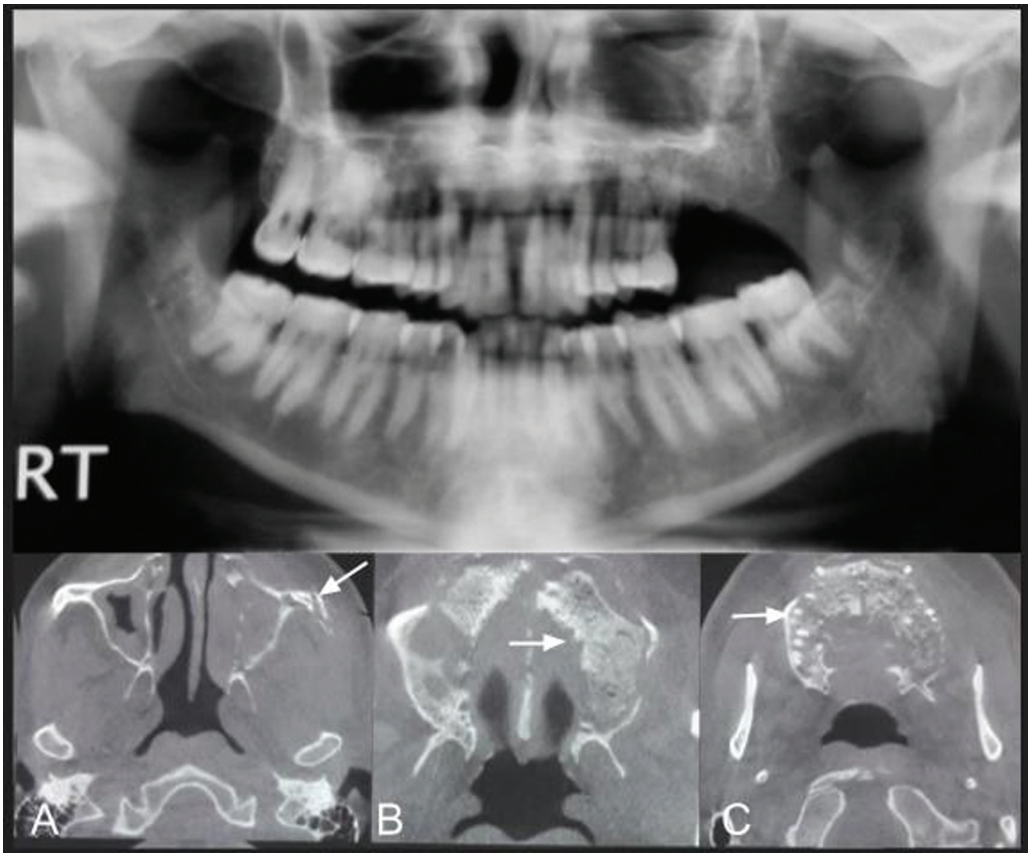

Fig. 4 Bacterial osteomyelitis of maxilla. Orthopantomogram (OPG) and computed tomography (CT) show permeative destruction of the maxillary alveolus (white arrow in **B**), hard palate (white arrow in **C**), left-sided maxillary walls, and left zygomatic arch (white arrow in **A**).

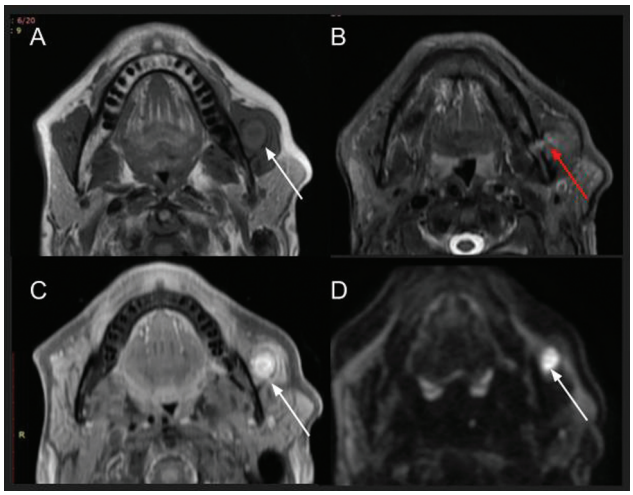

Fig. 5 Bacterial osteomyelitis of the mandible. A 43-year-old male patient presented with an ulcer with discharge for 4 months. Magnetic resonance imaging (MRI) shows ill-defined T1 hypointense (**A**) and short tau inversion recovery (STIR) hyperintense (**B**) marrow involvement of left hemimandible with cortical breach and sinus formation (red arrows) extending to adjacent masticator space causing masseteric abscess (white arrows) showing postcontrast enhancement (**C**) and diffusion restriction (**D**).

preauricular region. Pathological fractures of the mandible, or sequestration, have also been reported. Cavities and pathological fractures can be evident in CT.

Fungal infections are the most common cause of maxillary osteomyelitis. They generally present in an indolent fashion and are devastating to patients if invasive. These are opportunistic infections that frequently enter the body due to a decrease in host defense or through an invasive gateway, such as a dental extraction. Candidal infection is more often encountered when compared with other fungal infections, that is, mucormycosis, aspergillosis, etc. The clinical presentation is often similar to that of bacterial osteomyelitis. Specific delineating features would be the involvement of the maxillary sinus with a complaint of sinusitis in maxillary fungal osteomyelitis. An associated history of diabetes would generally be present in such cases.

Aspergillosis is the second most common fungal infection after candida. It is usually invasive when involving the maxillary sinus, though noninvasive forms have also been reported, and does not cause bone destruction when compared with mucormycosis.

Coronavirus disease (COVID)-associated mucormycosis of the jaw bones is another rare condition seen in greater incidence during the recent pandemic and has to be considered in patients who usually present with a triad of diabetes, steroid therapy, and severe acute respiratory syndrome coronavirus 2 infection (concomitant or recent). The pathogenesis of mucormycosis in the mandible (which is mainly dependent on endosteal blood supply) appears to be different from that of the involvement of the maxilla in Rhino-orbito-cerebral mucormycosis (ROCM). Isolated mandibular mucormycosis is rare, with very few reported cases.[11]

The most frequent CT findings in our cases were mucosal thickening of the paranasal sinuses, bony alterations such as ill-defined radiolucency (bone hypodensity), loss of definition of the lamina dura of involved teeth, erosion, breach, or destruction of bony structures, including cortical borders of the maxilla, the zygoma, the inferior rim of the orbit, the palatal bones, the nasal floor, and maxillary sinus walls, and sometimes bony sequestra (►**Fig. 7**). Other radiographic appearances of infected bones, such as "ground glass" and "salt and pepper," were also observed in our cases. In these cases, the counterpart surgical findings were necrotic, fragile, and had a cheese-like texture to the bone.

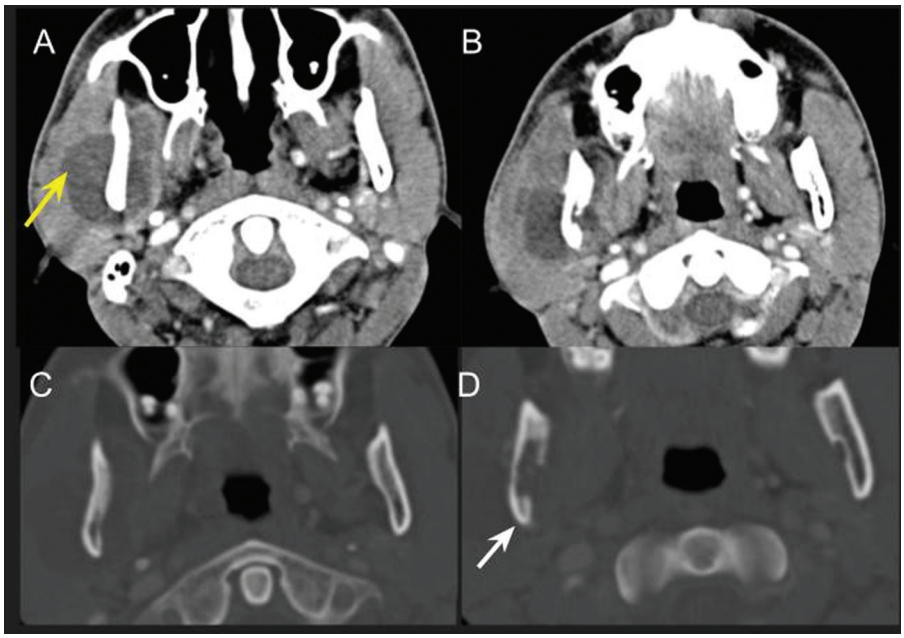

Fig. 6 Tuberculosis of the jaw. Soft tissue (**A**, **B**) and bone window computed tomography (CT) images (**C**, **D**) of a 22-year-old female right preauricular swelling shows lytic lesion with cortical destruction of the right mandibular ramus (white arrow) associated with soft tissue abscess formation in the adjacent masseter and medial pterygoid muscles with an increase in muscle bulk (yellow arrows).

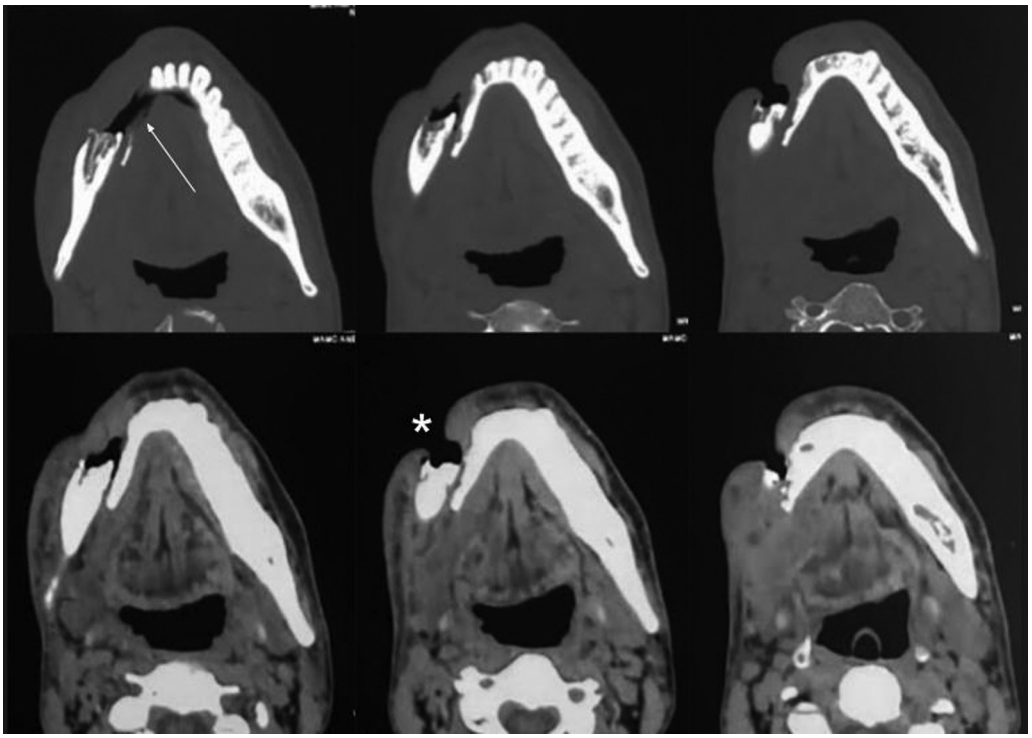

Fig. 7 Mucormycosis mandible with pathological fracture. Computed tomography (CT) shows a bony defect in the right hemimandible (white arrow) with surrounding lytic sclerotic areas and fistulous communication from the skin surface to the right lower alveolus of the mandible (asterisk). Soft tissue thickening and fat stranding are seen in the right buccal space and on the right side of the face.

Similar radiographic findings could also be found in malignant and metastatic bone lesions, osteoradionecrosis, and bisphosphonate-related osteonecrosis. However, metastasis and radiation- and drug-induced osteomyelitis are more common in the mandible. In addition, the patient's history helps differentiate between these conditions.

It is essential to know that all clinical findings in patients with a history of COVID-19 are important and should be considered seriously. In addition, in patients with suspected sinus involvement, bone window CT or cone-beam CT (CBCT) imaging is mandatory to detect any subtle osseous changes in the maxilla and adjacent bony structures in the early stages of the disease, which affects the extent of surgery and final prognosis.

Nonsuppurative and Other Mimics of Osteomyelitis

Based on imaging appearance, the mimics of osteomyelitis can be divided into those that mimic predominantly lytic bony destruction with aggressive periosteal reaction versus those that show diffuse or focal bone sclerosis with a smooth periosteal reaction pattern, that is, predominantly sclerotic pattern.

Before categorizing lesions into these two patterns, one should be diligent in excluding malignant causes like oral squamous cell carcinoma, lymphoma, and metastases, which usually have extensive soft tissue in proportion to bony changes and typical imaging appearances. An adequate history with suspicion of primary malignancy elsewhere in the body must be sought to rule out metastasis. Another important factor to consider is the patient's age group, whether pediatric or adult, which can further narrow down the diagnosis.

Mimics of Lytic Pattern of Osteomyelitis

The conditions that mimic permeative bony destruction with an aggressive periosteal reaction pattern in adults include small vessel vasculitis, mainly granulomatosis with polyangiitis (GPA), osteoradionecrosis of the jaw, and MRONJ. Important differentiating features are summarized in ►**Table 2**.

GPA: A multisystem necrotizing noncaseating cytoplasmic antineutrophil cytoplasmic antibody (c-ANCA) positive vasculitis involving small-to-medium-sized arteries, capillaries, and veins with a predilection for kidneys and respiratory system. It predominantly affects individuals in the 5th to 7th decades of life and involvement of the face is seen in upper respiratory tract disease.

Imaging shows osseous erosion, punctate destruction (75%), mucosal thickening, and neo-osteogenesis (50%). Useful differentiating features include paranasal sinus erosions (anterior ethmoid > posterior ethmoid > sphenoid > maxillary), bilateral symmetrical bony involvement, mucosal thickening maximum in the maxillary sinus, and nodularity (ANCA ±). Osseous changes start in the midline septum, and turbinates spread symmetrically to the antra and then to the rest of the sinuses. Palatal involvement is rare, and the base of the skull is usually preserved. CT shows wavy serpiginous mixed lytic/sclerotic areas (►**Fig. 8**). MRI shows neo-osteogenesis (high T1 signal), heterogeneously hypointense

Table 2 Mimics of lytic-permeative pattern of jaw osteomyelitis

Features	ORN	MRONJ	NK cell lymphoma	GPA
Clinical	Mandible – MC Early (< 2 years from radiation) and late onset (> 2 years from radiation)	Mandible – MC Painful May present with: nonhealing mucosal ulcers, loose teeth , soft tissue, and infections	Maxilla - MC	Maxilla - MC
Imaging	Soft tissue thickening and enhancement Solid/cystic mass (vs. tumor recurrence), lucent pattern of trabecular loss (vs. osteomyelitis): rare Bone fragmentation, intraosseous gas, bone sclerosis, permeative pattern of trabecular loss: more common	Similar to ORN	Extensive soft tissue thickening in the nasal cavity and maxillary antrum Septal and turbinate bony destruction Roomy nasal cavities Hard palate spared	Mucosal thickening of paranasal sinus with erosions Osseous changes start in midline septum, and turbinates spread symmetrically Palatal involvement is rare The base of the skull is usually preserved CT shows wavy serpiginous mixed lytic/sclerotic areas

Abbreviations: CT, computed tomography; GPA, granulomatosis polyangiitis; MC, most common; MRONJ, medication-related osteonecrosis of jaw; NK cell lymphoma, natural killer cell lymphoma; ORN, osteoradionecrosis.

appearance on T1-weighted (T1W) images, and hyperintense appearance on T2W images with enhancement on postgadolinium images.

Osteonecrosis of the jaw: Classically considered a disruption of vascular supply, avascular necrosis, or aseptic necrosis. The causes are enumerated in ►**Table 3**. Osteoradionecrosis may display similarity to chronic osteomyelitis on CT and radiographs (►**Fig. 9**). Osteoradionecrosis of the mandible is a major concern in a patient who underwent radiation treatment for carcinoma of the oral cavity or oropharynx and, after 1 to 3 years, presents with exposed bone, a fracture, or perimandibular swelling. Osteoradionecrosis, even though occasionally called radio-osteomyelitis, is entirely different from true osteomyelitis in pathogenesis and presentation. Osteoradionecrosis is induced primarily by hypovascularity due to vessel obliteration, resulting in hypoxia and hypocellularity. Microorganisms are encountered as surface contaminants only, rather than infection-inducing agents (►**Fig. 10**).

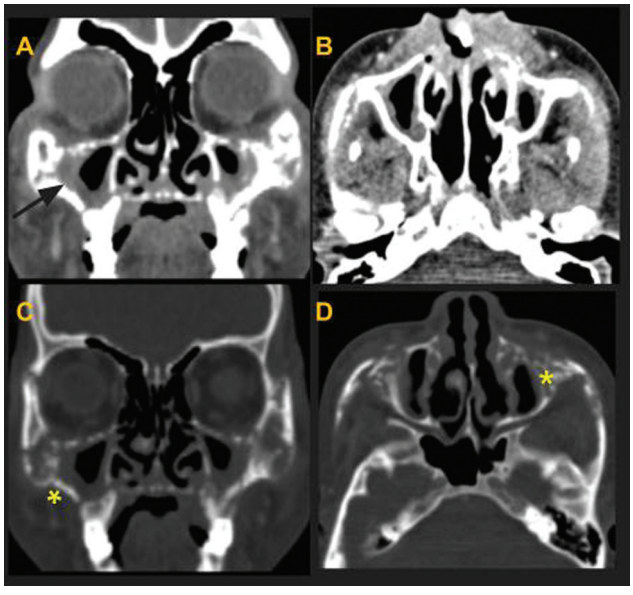

Fig. 8 Wegener's granulomatosis involving the maxilla and paranasal sinuses. A 33-year-old female with a history of incisor caries with a tooth falling off and presented now with pain, fever, and redness of both cheeks for 2 months. Bilateral symmetrical bony involvement with osseous erosion, punctate destruction, mucosal thickening, maximum in the maxillary sinus (black arrow in **A**), and neo-osteogenesis (yellow asterisk in **C**, **D**).

MRONJ has increasingly been recognized as an adverse effect of bisphosphonate treatment, which has evolved into a standard regimen for the treatment of osteolytic bone lesions related to multiple myeloma and osseous metastases of solid cancer, in particular carcinoma of the breast and prostate.

Bisphosphonate incorporation is visualized by the thickening and prominence of the lamina dura and of the cortical confines of the mandibular canal on orthopantomogram (OPG), which is obligatory to assessing the dental status, excludes a fracture at the sites of extraction sockets, and may also be normal. In cases of uncertainty, a comparison with a previous OPG examination, if available, is helpful. The CT findings consisted of marked sclerosis of the cancellous and cortical bone without expansion but commonly with

Table 3 Causes of osteonecrosis of jaw

- Radiation (ORN)
- Medications: high-dose steroid therapy, and medications that disrupt vascular supply or bone turnover in the jaws (bisphosphonate - BRONJ)
- Viral infections, including COVID
- Rare causes - electric shock

Abbreviations: BRONJ, bisphosphonate-related osteonecrosis of jaw; COVID, coronavirus disease; ORN, osteoradionecrosis.

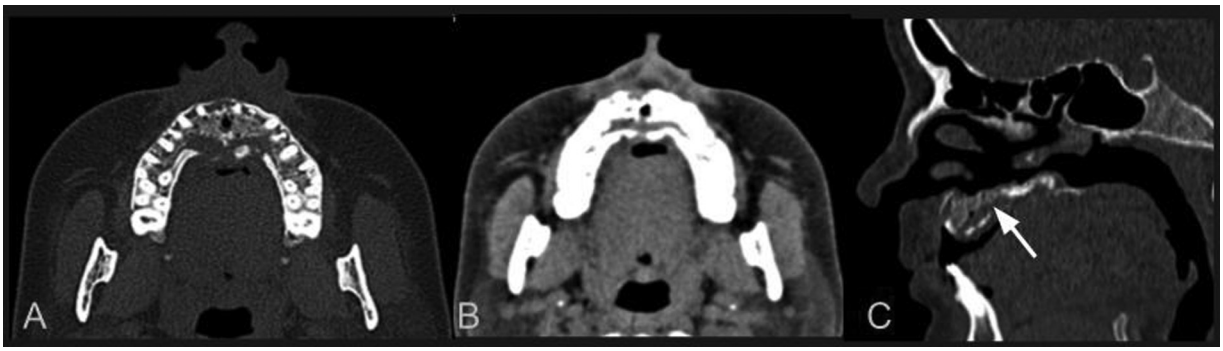

Fig. 9 Osteonecrosis secondary to viral infection in the maxilla. A 40-year-old patient with a history of viral illness presented with loosening of the upper teeth 1 month later and developed oral discharge subsequently. Computed tomography (CT) shows well-marginated areas of osteolysis in the maxillary alveolar process including the anterior hard palate right involving the outer and inner cortices of the bone. Separated pieces of bone within osteolysis are in keeping with bony sequestrum (white arrow). No abnormal periosteal reaction—consistent with osteonecrosis.

sequester-like fragments of bone (►**Fig. 11**). Cessation of bone remodeling and turnover by inhibition of the recruitment and activity of osteoclasts is the presumed mechanism. Marked sclerosis is the principle finding when the maxilla is affected.[12]

Mimics of Sclerotic Pattern of Osteomyelitis

The conditions that show diffuse or focal bone sclerosis with a smooth periosteal reaction pattern include periostitis ossificans, chronic recurrent multifocal osteomyelitis (CRMO), SAPHO syndrome, diffuse sclerosing osteomyelitis, and rarely osseous dysplasia. The first three are most common in children and young adults, while the latter two are mostly encountered in older adults (►**Table 4**)

Periostitis ossificans, also known as Garre's osteomyelitis: This condition is usually nonsuppurative and asymptomatic without signs of inflammation; however, overlying soft tissue involvement can occur rarely (►**Fig. 12**). The underlying pathology is the periosteal reaction to inflammation, which often presents as a localized overgrowth of bone on the outer surface of the cortex. This mass of bone, which is supracortical but subperiosteal, is smooth, fairly calcified, and often described as a duplication of the cortical layer of the mandible. The redundant cortical layering of the bone (onion skinning) is often considered a pathognomonic feature[13]

An important differential in cases of periostitis ossificans of Garre in children is Caffey's disease (infantile cortical hyperostosis) due to a similar radiological appearance. The differences are tabulated in ►**Table 5**.

CRMO: Diffuse bone radiopacity is the most prominent finding in patients with the mandibular manifestation of "CRMO," a relapsing inflammatory disease that is considered a type of seronegative spondyloarthropathy. The prevalence of mandibular involvement is 2 to 8%.[14] It affects a variety of other bones such as the pelvis, sternum, and scapula in addition to the originally described long bones. The disease is rare, accounting for 2 to 5% of all osteomyelitis cases, and primarily affects young girls, with a female/male ratio of 5:1. While radiopacity governs the quiet phase of CRMO, areas of mottled cortical and cancellous bone osteolysis prevail during the episodes of recurrence. Small osteolytic lacunae that contain lymphocytes and plasma cells are potential sites of exacerbation, leading to cortical erosion and periosteal reactions. Additional radiographs (and scintigraphy) aid in detecting synchronous or metachronous involvement of multiple bones, such as the clavicle, humerus, radius, femur, or tibia.

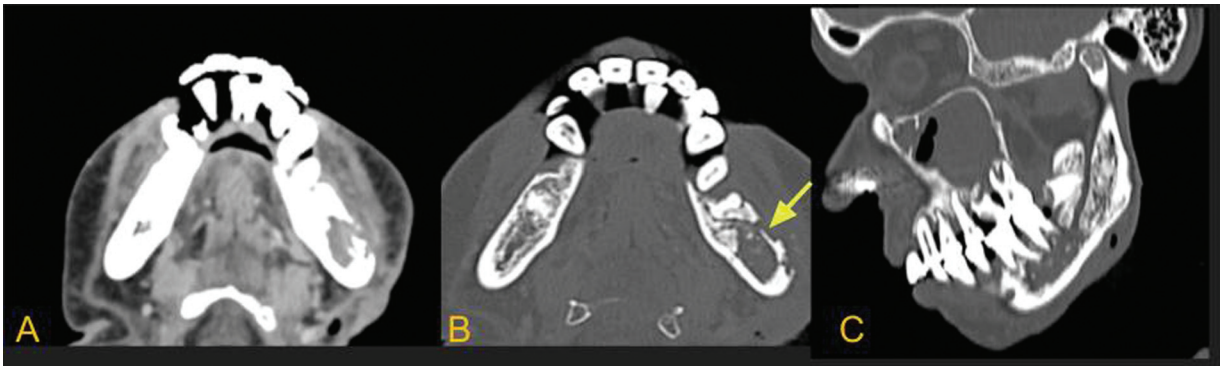

Fig. 10 Osteoradionecrosis (ORN). (**A–C**) Known case of postoperative left parotid carcinoma with history of radiotherapy—shows ill-defined cortical destruction predominantly on the buccal side of the left hemimandible (yellow arrow in **B**) with mixed sclerotic-lucent pattern and an absence of soft tissue mass.

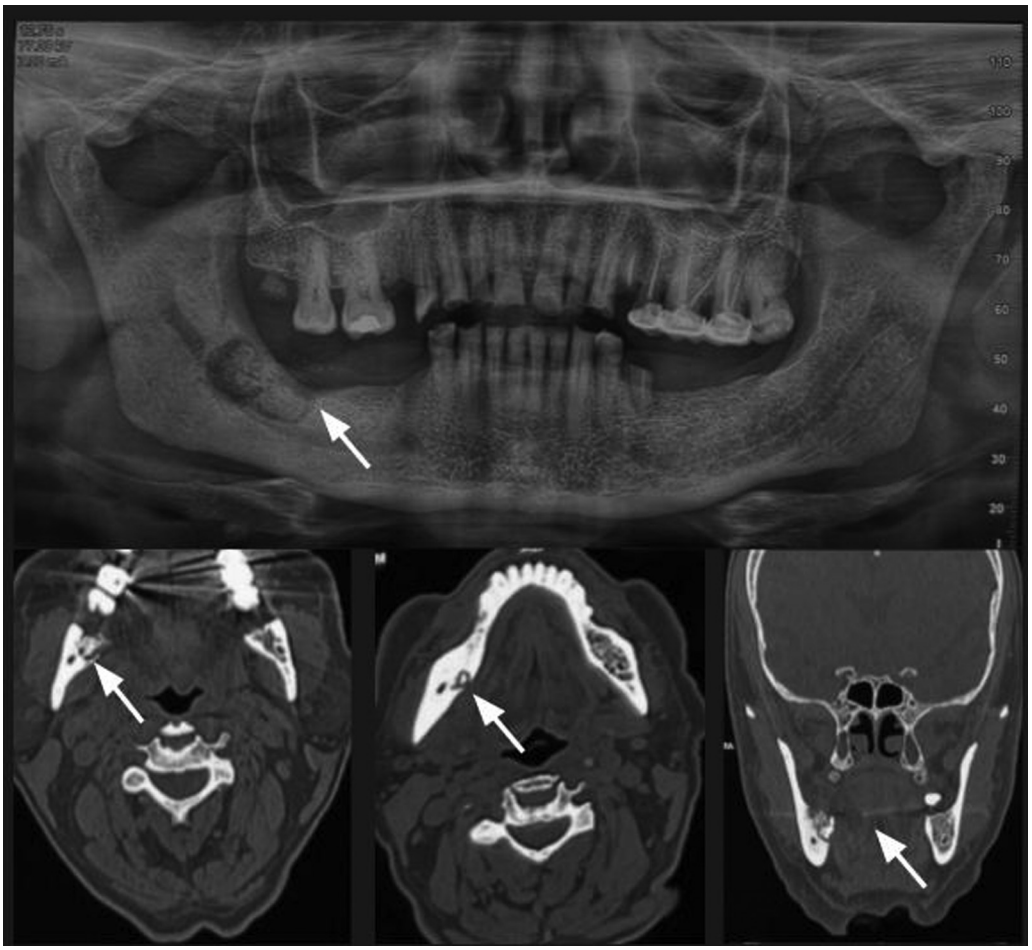

Fig. 11 Medication-related osteonecrosis of the jaw secondary to bisphosphonate. A 68-year-old male patient with multiple myeloma and a treatment history of zoledronic acid 25 mg/day for 2 years. Orthopantomogram (OPG) and computed tomography (CT) images show a well-defined lytic lesion in the body of the right hemimandible with a central sequestrum and generalized sclerosis of the right hemimandible (arrows). No periosteal new bone formation s/o medication-related osteonecrosis of the jaws (MRONJ).

SAPHO syndrome: An affiliation with another spondyloarthropathy called SAPHO syndrome, described by Chamot et al,[15] is characterized by synovitis, acne, pustulosis, hyperostosis, and osteitis. The radiographic appearance of the mandibular manifestation of SAPHO syndrome corresponds to "diffuse sclerosing osteomyelitis."[16]

Clinical features include pain and swelling, but suppuration is never found in this condition. The symptoms may start gradually or suddenly and persist for a long time, with repeated exacerbations and remissions. The lesions are often extensive. Condylar process involvement is not rare, and the entire mandible may be involved.

Diffuse sclerosing osteomyelitis, often termed "chronic diffuse sclerosing osteomyelitis (CDSO), is primarily seen in elderly patients and is thought to be a localized version of CRMO due to its similar clinical course, radiological

Table 4 Mimics of sclerotic pattern of jaw osteomyelitis

Features	DSO	CRMO	SAPHO	Garre's OM
Age group	Older adults	Children and young adults	Young to middle-aged adults	Children and young adults
Imaging	Diffuse sclerosis of the bone Thick periosteal reaction Absence of fistulae and sequestration characteristic	Early stages: osteolysis Later stages: progressive sclerosis and hyperostosis Clavicle involvement is characteristic	Same as CRMO Sternoclavicular joint osteitis/ hyperostosis - characteristic Sacroiliitis Osteosclerosis of vertebral bodies	Supracortical subperiosteal bone formation Smooth and calcified Onion skin periosteal bone formation

Abbreviations: CRMO, chronic recurrent multifocal osteomyelitis; DSO, diffuse sclerosing osteomyelitis; Garre's OM, osteomyelitis; SAPHO, synovitis, acne, pustulosis, hyperostosis, and osteitis.

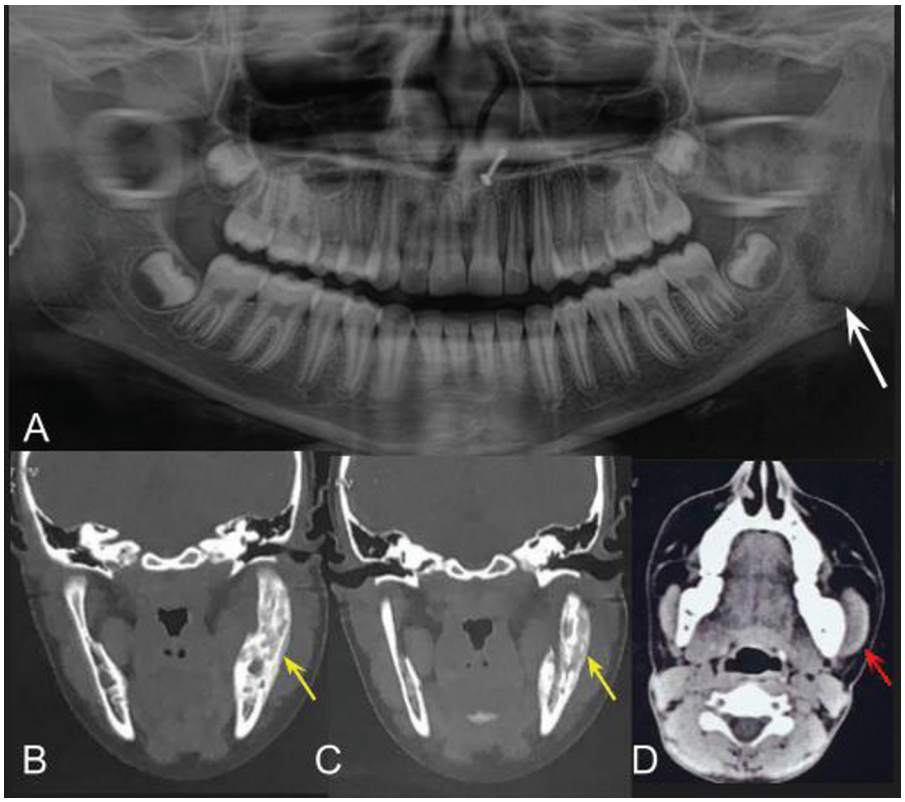

Fig. 12 Garre's osteomyelitis. (**A**) Orthopantomogram (OPG) shows ill-defined sclerosis of left hemimandible predominantly involving the ramus and extending to the posterior body with areas of lysis and cortical perforation (white arrow). Contrast-enhanced computed tomography (CECT) bone window images (**B** and **C**) show: bony expansion, cortical thinning, and diffuse sclerosis of the left mandible with focal new bone formation on the buccal side as well as cortical breakthrough surrounding the lesion (yellow arrows). Edema is present within the masseter muscle which appears bulky compared with contralateral side, better seen in soft tissue window (red arrow in **D**).

appearance, and histological characteristics. Additionally, it has been suggested that CDSO is a mandibular localization of SAPHO syndrome, characterized by condyle ankylosis, medullary sclerosis, and subcortical erosions in addition to skin symptoms.[16] The disease's convoluted progression is attributed to multiple confounding circumstances, making diagnostic isolation challenging. Microorganisms with low virulence mostly cause intramedullary osseous infections that lead to sclerosis.[17,18] Still, results from bone cultures are often inconsistent.[17,19,20]

The patterns most commonly linked with CDSO are seen on CT, which includes different degrees of sclerosis and hyperostosis. The cortical–medullary barrier thickens and sometimes vanishes due to the involvement of the cortical bone. The absence of sequestration and fistulae are among the characteristics. Hyperintense edematous marrow is visible on T2W MRI images. Although MRI reveals the degree of inflammation, CT provides a better depiction of the sclerotic bone, hence it is essential to evaluate the patient using both imaging modalities.[21,22]

Table 5 Differences between Garre's osteomyelitis and infantile cortical hyperostosis

Garre's osteomyelitis	Infantile cortical hyperostosis
Age of presentation: Adolescent, young adult	Age of presentation: 6 weeks to 6 months of age
Nontender Bony hard swelling No fever	Unusual irritability Low-grade fever Soft tissue swelling
Mandible: most common, usually focal No other bone	Mandible: most common, usually bilateral Others: scapula, clavicle, ribs, long bones

By reducing the length of the clinical episode, early treatment with long-term antibiotic therapy can be advantageous.[17,18] However, the best course of action at a chronic stage may involve surgical decortication along with antibiotic therapy. Since reduced oxygen tension and anaerobic infections are likely, hyperbaric oxygen therapy is advised. As bisphosphonates reduce bone resorption and turnover, several reports have indicated encouraging outcomes.[18,23] For several reasons, including the possibility of causing osteonecrosis, bisphosphonates are still not considered conventional treatment for CDSO.

Osseous dysplasia: When a focal sclerotic lesion is restricted to the alveolar process, florid osseous dysplasia is more likely than osteomyelitis.

Miscellaneous

Lastly, two important conditions where both lytic and sclerotic patterns can coexist are secondary infection on a background of primary sclerotic bony pathology like fibrous

dysplasia or osteopetrosis and osteoblastic metastases related to carcinoma of the breast or prostate. True osteopetrosis (Albers–Schönberg) is characterized by increased bone density and diameter due to osteoclast malfunction, and on CT, it resembles primary chronic osteomyelitis. Cancellous bone sclerosis is the dominant finding, and cortical bone is usually thickened. While osteopetrosis is a known predisposing condition for osteomyelitis, osteomyelosclerosis shows similarity to the appearance of primary chronic osteomyelitis, both on radiographs and CT.

Unique Considerations in the Pediatric Age Group

Any disease that causes marrow infiltration, inflammation, or edema can be confused with osteomyelitis. The most difficult differentiation in children is between infectious osteomyelitis and chronic nonbacterial osteomyelitis (CNO), particularly its most severe form, termed CRMO. Both conditions produce bone destruction, primarily affecting the metaphyses and metaphyseal equivalents, and can extend into the physis. With CNO/CRMO, symptoms typically are less acute and involvement is frequently multifocal (more than 80% of cases) and often symmetric. The most common sites of involvement in CNO/CRMO are the pelvis, lower extremities, shoulders, and spine. In the tubular bones, nearly 90% affect the physis. Unlike hematogenous osteomyelitis, CNO/CRMO often involves the clavicle. Lesions of CNO/CRMO in the axial skeleton usually show only mild marrow edema without soft tissue edema, which is unlike bacterial osteomyelitis. Inflammatory markers such as C-reactive protein and erythrocyte sedimentation rate are only mildly elevated in CNO/CRMO.

Next most important are the tumors, which include metastatic neuroblastoma and Langerhans cell histiocytosis (LCH), in children less than 5 years of age, leukemia, Ewing sarcoma, and osteosarcoma in older children. Most of these lesions present with bone destruction, often accompanied by a soft tissue mass. The clinical presentation can overlap, as children with infections and tumors can have fever and localized bone findings. However, a tumor often is associated with longer symptom duration. Although the plain radiographic findings can sometimes overlap, it is important to underscore that radiographically detectable bone destruction is a late finding in osteomyelitis and that patients typically have experienced symptoms for more than a week before findings become apparent. On MRI, osteomyelitis is not associated with a discrete mass. In younger children, LCH can result in dramatic bone destruction and perilesional edema (►**Fig. 13**). On MRI of osteomyelitis, there is abundant perilesional edema that extends along the marrow and into the soft tissues. This extension results in an ill-defined margin between normal and abnormal marrow, which fades away from the center of the infection. Ewing sarcoma and other malignancies such as osteosarcoma usually have a sharper margin between the affected and the unaffected marrow on T1W images (►**Fig. 14**).

Other lesions that can resemble osteomyelitis include sickle cell anemia where both infarction and infection can occur, often coexisting, osteoid osteoma, repetitive or chronic trauma, and septic embolic lesions. On MRI, stress reactions are primarily diaphyseal and the edema is predominantly intramedullary, whereas osteomyelitis usually causes circumferential edema and affects the bones and soft tissues almost equally.

Optimization of Imaging Modalities

In addition to routine imaging modalities, CBCT and scintigraphy also play specific roles in the diagnosis and management of jaw osteomyelitis. CBCT employs a cone-shaped radiation beam that rotates once over the area of interest, as well as a reduced tube dosage, resulting in much lesser radiation exposure. As a result, the risk of artifacts from dental fillings is considerably reduced. Cone-beam technology has various drawbacks, including the inability to depict soft tissue, an extended data collection time of 18 to 36 seconds (up to 75 seconds), and poor contrast resolution within the compact bone. As a result, it is widely employed in implantology to assess impacted third molars and diagnose cysts. Although its significance in osteomyelitis is limited, irregular radiolucencies and osteosclerotic alterations have been proven to be useful in diagnosing the disease.[24]

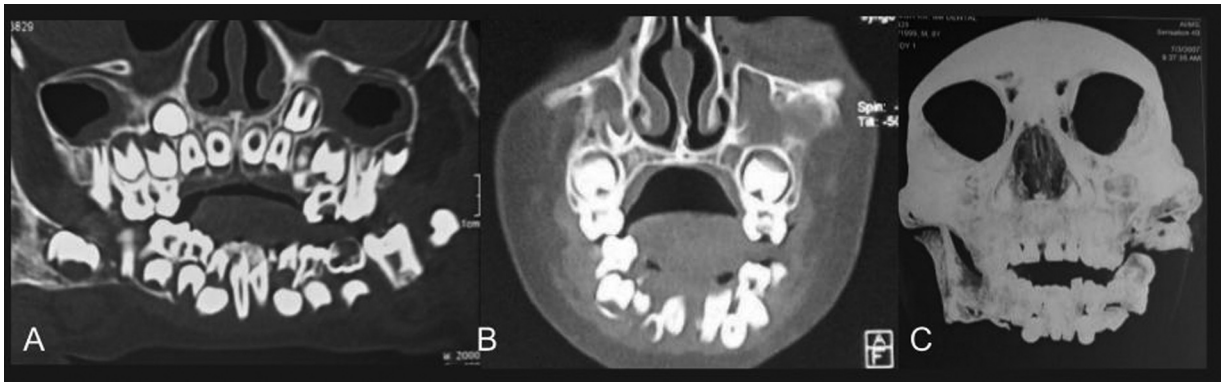

Fig. 13 Langerhans cell histiocytosis (LCH). Panoramic (**A**), coronal (**B**), and volume-rendered reconstruction (**C**) computed tomography (CT) images shows near-complete osteolysis of the mandible with floating mandibular teeth characteristic of this pathology.

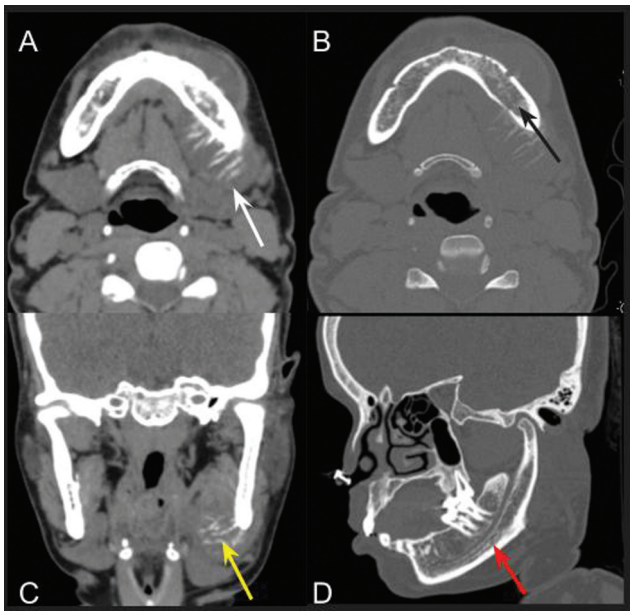

Fig. 14 Osteomyelitis mimics in adults. Case of a 38-year old female with swelling over the left side of the neck around 2 months. Non-contrast computed tomography (NCCT) (**A**–**D**) shows hair on end and sunburst periosteal reaction (white arrow) with permeative lysis and cortical breach in the ramus and body of the left hemimandible mandible (black arrow). Well-defined extraosseous soft tissue lesion in the submandibular region (yellow arrow) and left buccal space displacing submandibular gland posteroinferiorly with focal loss of fat planes. Also, there is widening and irregularity of the wall of the left inferior alveolar canal (IAC; red arrow). Final diagnosis was primary bone tumor—osteosarcoma.

Scintigraphy detects significantly increased bone turnover activity in acute osteomyelitis and has been reported to be an early indicator of the disease.[25,26] Hyperemia could lead to increased activity within 2 to 3 days of symptom onset. The degree of uptake was observed higher when plain films exhibited permeative bone degradation and regions of osteolysis, as opposed to the moth-eaten or sclerotic appearance that occurs in chronic osteomyelitis.[25] Using histology as a reference, the sensitivity in the acute phase is estimated to be close to 100%, with false-negative results ascribed to the examination being performed too early.[26]

The limitations of scintigraphy in acute osteomyelitis stem from the difficulty in distinguishing between soft tissue inflammation and bone involvement. Scintigraphy alone cannot assess the degree of mandibular osteomyelitis in a preoperative setting. It must be supplemented with an additional CT evaluation to provide the necessary comprehensive osseous information. In a follow-up examination, simultaneous indium-111 white blood cell/Tc-99m methylene diphosphonate bone single-photon emission CT scintigraphy proved to revert to normal following effective treatment much sooner than CT.[27]

In chronic osteomyelitis, scintigraphy may be used to monitor disease activity rather than to make a diagnosis.[25] Scintigraphy is the preferred diagnostic modality for detecting extra skeletal symptoms in CRMO and patients suspected of having SAPHO syndrome. Bone scintigraphy detects additional areas of involvement in both diseases, even when no symptoms are apparent.[28] Following conservative therapy, infection recurrence coincides with increasing uptake, while remission correlates with decreasing activity of the radiotracer; nonetheless, even during periods of quiescence, higher activity may remain for up to 4 months after symptoms have resolved.[29] The area of enhanced activity was significantly greater than that suggested by plain films.[25]

Conclusion

In summary, imaging is a crucial diagnostic tool in the assessment of acute and chronic osteomyelitis of the jaws. Before any cross-sectional imaging modality is applied, the panoramic view is the first image to assess the status of dentition, recognize direct radiographic signs of osteomyelitis, narrow the differential diagnosis, and depict potential predisposing conditions such as a fracture or systemic bone disease. The orthopanoramic view is also the first-line image when follow-up examinations are performed. In acute osteomyelitis, the higher sensitivity of MRI concerning the detection of intramedullary inflammation advocates its use as the imaging modality of choice to confirm the diagnosis and provide an estimate of the intraosseous extent and soft tissue involvement. In cases where surgical treatment is planned, high-resolution CT is required to specify the degree of cortical destruction, delineate the presence of sequestra, and define the extent of osseous removal required. In chronic osteomyelitis, the higher sensitivity of CT concerning the detection of sequestered and sclerotic bone changes renders CT the examination of choice to distinguish the usually more uniform and extensive primary chronic osteomyelitis from the more localized type of secondary chronic osteomyelitis. MRI is superior to detecting periosteal inflammation and soft tissue involvement and thus aids in determining the persistence or recurrence of infection. Following surgery, CT is preferred as a follow-up examination for 6 months to distinguish postoperative and reparative changes from recurrent or persistent infection. Complementary information is gained in particular situations by a combination of imaging modalities adapted to the individual patient's course of disease and the panoramic view findings.

Conflict of Interest
None declared.

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