

Radiopaque Jaw Lesions: An Approach to the Differential Diagnosis¹

Joel K. Curé, MD • Surjith Vattoth, MD, DNB, FRCR • Ritu Shah, MD

ONLINE-ONLY CME

See www.rsna.org/education/learn/rg

LEARNING OBJECTIVES

After completing this journal-based CME activity, participants will be able to:

- Review the classification of jaw bone lesions on the basis of lesion attenuation, margination, and the relationship of the lesion to the teeth.
- Identify the different sclerotic jaw lesions.
- Describe the characteristic imaging features of the various sclerotic jaw lesions and their differential diagnoses.

TEACHING POINTS

See last page

Radiopaque jaw lesions are frequently encountered at radiography and computed tomography, but they are usually underevaluated or underdescribed in radiology reports. A systematic approach to the evaluation of radiopaque jaw lesions is necessary to diagnose the lesion or at least provide a meaningful differential diagnosis. To evaluate a radiopaque jaw lesion, the first, most important step is to categorize the lesion according to its attenuation, its relationship to the teeth, and its location with respect to the tooth. These basic observations are essential to the evaluation of any type of jaw lesion. Once these observations have been made, it is easy to create a proper differential diagnosis. The presence of important characteristics, such as margination, a perilesional halo, bone expansion, and growth pattern, as well as whether the lesion is sclerotic, has ground-glass attenuation, or is mixed lytic and sclerotic, further narrows the differential diagnosis. It is important to note that some radiopaque jaw lesions may be entirely lucent early in their evolution. Awareness of the demographic distribution of these lesions and their associated clinical features, as well as the radiologic approach, is important to explore the “terra incognita” of radiopaque jaw lesions.

Introduction

For many radiologists, radiopaque jaw lesions are terra incognita—Latin for “unknown land.” Jaw lesions that are predominantly radiolucent, such as periapical (radicular) cysts, follicular (dentigerous) cysts, keratocystic odontogenic tumors (previously known as odontogenic keratocysts), and ameloblastomas, are well described in the radiology literature. However, radiopaque jaw lesions have received less attention. In this article, we review benign odontogenic and nonodontogenic jaw lesions that may have a sclerotic, ground-glass, or mixed lytic and sclerotic imaging appearance. Herein, the term *ground-glass appearance* refers to lesions with mostly relatively homogeneous, intermediate attenuation between that of normal cortical bone and soft tissue, an appearance that is analogous to the obturating surfaces of glass stoppers used in laboratory glassware.

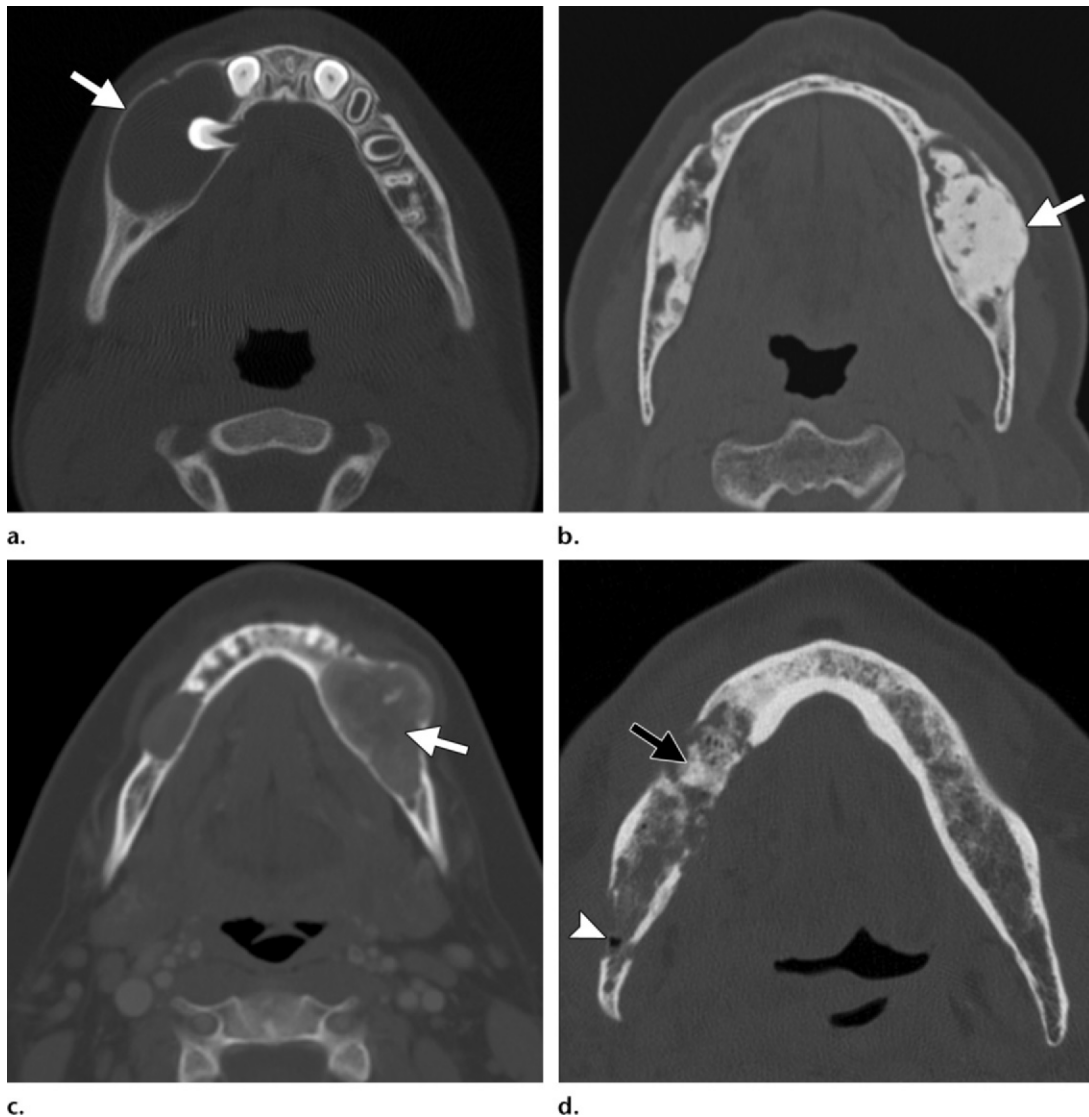


Figure 1. Various jaw lesions. (a–c) Axial computed tomographic (CT) images show jaw lesions (arrow) that demonstrate lysis (a), sclerosis (b), and ground-glass attenuation (c). (d) Axial CT image shows a mixed lytic (arrowhead) and sclerotic (arrow) lesion.

Approach to Image Interpretation in Patients with Jaw Lesions

The radiologic diagnosis of jaw lesions is informed by imaging features such as attenuation, margination (a narrow or wide transition zone), and the relationship of the lesion to adjacent teeth. Although cystic jaw lesions are not discussed in this article, these basic observations are essential when analyzing any type of jaw lesion. The first question to be answered is whether a lesion is lytic, sclerotic, or mixed or if it has ground-glass attenuation (Fig 1).

The second question is whether the transition zone between clearly normal and clearly abnormal bone is narrow or wide. Although it is usually used to help differentiate benign, slow-growing lesions (those with a narrow transition zone) from more aggressive lesions (those with a wide transition zone), the transition zone may also be used to differentiate lesions with otherwise similar appearances, such as ossifying fibromas and fibrous dysplasia.

The third question is whether the lesion is related to a tooth (ie, if it is intimately associated with a single tooth and in a tooth-bearing area of the jaw). Tooth-related lesions commonly surround a

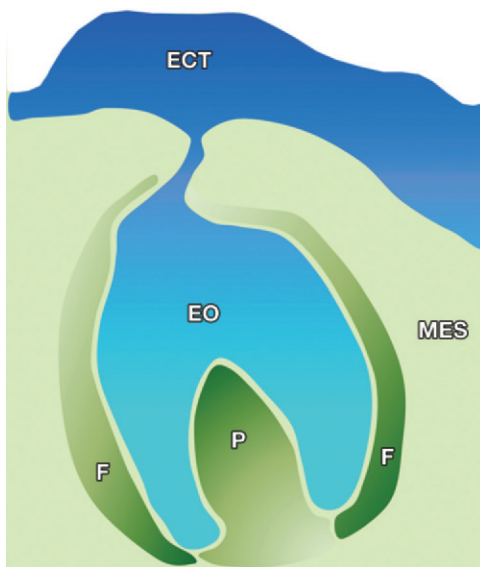


Figure 2. The tooth bud. Illustration shows the three components of the tooth bud. The enamel organ (EO) arises from the ectoderm (ECT), which derives from the first branchial arch. Both the dental papilla (P) and the dental follicle (F) arise from ectomesenchyme (MES) originating from the neural crest. The orientation of jaw lesions with respect to the teeth is influenced by the origin of their cellular constituents within or around the developing tooth bud.

component (eg, the crown or apex) of the tooth and are usually, but not always, odontogenic in origin. A tooth-related lesion may also arise or persist at the site of a congenitally absent or extracted tooth. Lesions that are clearly not tooth-related usually indicate a lesion of osseous origin. Large nonodontogenic lesions may abut adjacent teeth, and it may be difficult to determine whether they are tooth related or not.

Teeth arise from an embryologic structure known as the tooth bud (Fig 2). The constituent cells of the tooth bud arise from the ectoderm of the first branchial arch and ectomesenchyme from the neural crest and organize into the three important generative components of the tooth anlage: the enamel organ, dental papilla, and dental follicle. The enamel organ arises from the branchial ectoderm and endomes the developing dental papilla. Ameloblasts develop within the enamel organ and elaborate the enamel that will cover the crown of the tooth. The dental papilla is of ectomesenchymal origin; odontoblasts that arise in dental papilla produce dentin, the domi-

Table 1
Sclerotic Lesions of the Jaw

Type	Lesion
Tooth-related*	
Periapical	Cementoblastoma, cemento-osseous dysplasia, condensing osteitis
Pericoronal	Odontoma
Non-tooth-related†	
Nonexpansile	Idiopathic osteosclerosis
Expansile	Osteoma
Exophytic	Exostoses, tori

*Tooth-related lesions are usually odontogenic.

†Non-tooth-related lesions are usually related to a bone condition.

nant “hard” component of the tooth. Other mesenchymal cells in the dental papilla form the neurovascular structures of the dental pulp. The third component is the dental follicle, which also arises from ectomesenchymal precursors. The dental follicle surrounds the tooth bud and generates cementoblasts, which form dental cementum; osteoblasts, which form the alveolar bone; and fibroblasts, which form the periodontal ligaments that connect the cementum-covered dental apices (roots) to the adjacent alveolar bone.

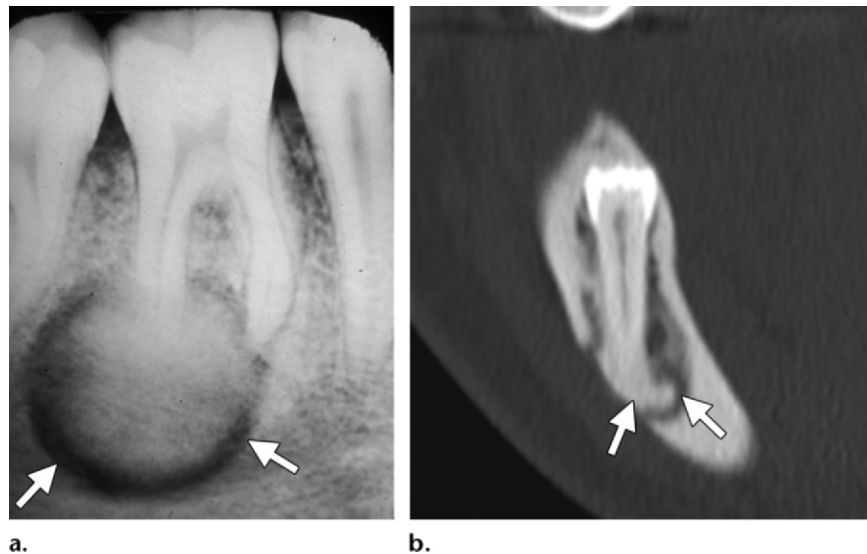
Tooth-associated lesions may be characterized as periapical (around the apex of the tooth), interapical (between apices of two adjacent teeth), or pericoronal (around the crown of the tooth). Pericoronal lesions may arise from the ectoderm-derived components of the enamel organ (ameloblasts). Periapical lesions commonly arise from areas of endodontal or periodontal inflammatory disease or the ectomesoderm-derived components of the dental papilla (odontoblasts) or follicle (cementoblasts, fibroblasts, and osteoblasts) (1).

Tooth-related Sclerotic Jaw Lesions

Cementoblastoma

If a lesion is periapical, the differential diagnosis includes cementoblastoma, cemento-osseous dysplasia, and condensing osteitis. If it is pericoronal, odontoma should be considered (Table 1). Cementoblastoma, a rare benign periapical

Figure 3. Cementoblastoma. Bite-wing radiograph obtained in an adult patient (a) and coronal CT image obtained in a 34-year-old woman (b) show a periapical sclerotic lesion with sharp margins and a lucent or low-attenuation halo (arrows) that is fused to the root of the tooth. Cementoblastoma arises in the molar or premolar region in 90% of cases.



lesion, represents less than 1% of all odontogenic tumors. More than 75% of cementoblastomas occur in the mandible; of those, 90% develop in the molar or premolar region. Cementoblastomas are most common in children and young adults, with 50% occurring before age 20 and 75% occurring before age 30. Although most cementoblastomas are associated with an erupted permanent tooth, they may occur near an impacted or unerupted tooth. At imaging, cementoblastomas appear as a periapical, sclerotic, sharply marginated lesion with a low-attenuation halo. They directly fuse to the root of the tooth (Fig 3). Some cementoblastomas may fuse to more than one tooth root or invade the root canal and pulp chamber (2). Management of cementoblastomas typically involves complete removal of the associated tooth to reduce the likelihood of recurrence (3).

Cemento-osseous Dysplasia

Cemento-osseous dysplasia represents a hamartomatous process that is usually associated with tooth apices. Its clinical forms vary, and the associated terminology may be confusing. Periapical cemento-osseous dysplasia typically arises in the anterior mandible and involves one or only a few teeth. Another limited form that occurs in the posterior jaw and typically involves molar teeth is called focal cemento-osseous dysplasia. Florid cemento-osseous dysplasia typically involves two or more jaw quadrants (4).

Cemento-osseous dysplasia has a strong gender and racial predisposition, with most lesions occurring in black women and women of Asian descent who are in the 4th or 5th decade of life. Only the focal variant is more commonly

reported in white women. Cemento-osseous dysplasia usually produces no symptoms, but it may cause a dull ache. Florid cemento-osseous dysplasia may be complicated by osteomyelitis and drainage of necrotic bone debris into the oral cavity or to the skin surface through osteocutaneous sinus tracts.

At imaging, lesions are initially lytic, with a mixed lytic and sclerotic appearance seen later, often with a central area of calcification. Early (lytic) lesions may be confused with periapical inflammatory lesions, such as cyst, granuloma, and abscess. However, in contradistinction to these inflammatory lesions, cemento-osseous dysplasia is typically associated with a vital, nonrestored tooth and an intact lamina dura and periodontal ligament, and a central area of calcification may be seen (5). Late cemento-osseous dysplasia lesions are periapical, sclerotic, and sharply marginated. A low-attenuation halo may be seen, but unlike cementoblastomas, cemento-osseous dysplasia lesions do not fuse directly to the tooth root (Figs 4, 5). Cemento-osseous dysplasia may occur in the tooth-bearing jaw after dental extraction, and when multiple lesions are present, adjacent periapical lesions may coalesce. Cemento-osseous dysplasia lesions may be expansile when associated with bone cysts or osteomyelitis (Fig 6). The florid variant may involve the entire mandible (6). **Both cementoblastoma and cemento-osseous dysplasia are periapical, sclerotic, sharply marginated lesions with a low-attenuation halo. However, cementoblastoma occurs in children and young adults and fuses directly to the tooth root, whereas cemento-osseous dysplasia is more common among black women and women of Asian descent who are in the 4th or 5th decade of life and does not fuse to the tooth root.**

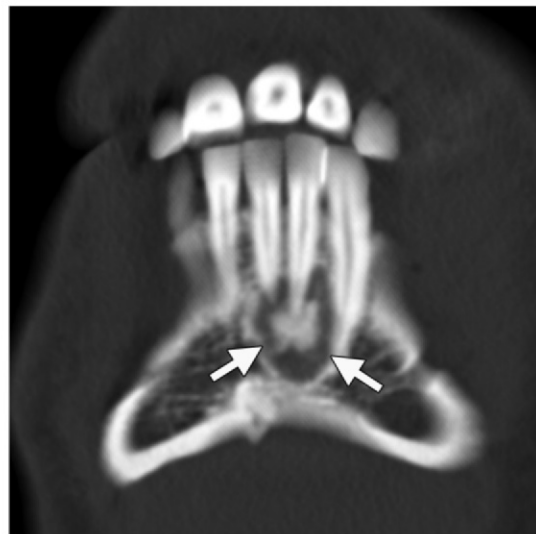
Teaching Point



4a.



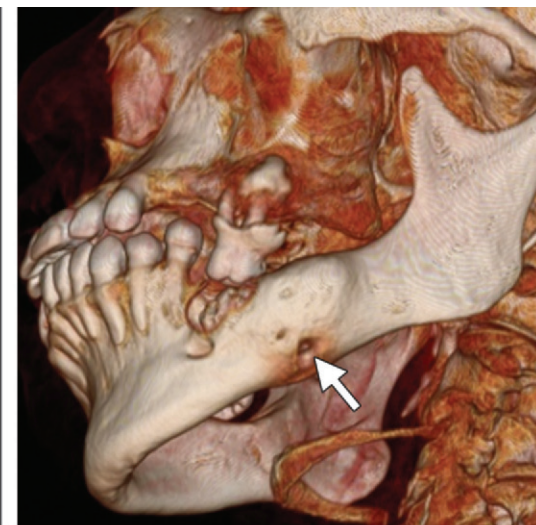
4b.



5.



a.



b.

Figure 6. Cemento-osseous dysplasia with osteomyelitis in a 40-year-old woman. Coronal reformatted (a) and volume-rendered (b) CT images show an expansile cemento-osseous dysplasia lesion with associated osteomyelitis. Arrow = sinus tract.

Figures 4, 5. (4) Cemento-osseous dysplasia. (a) Three-dimensional volume-rendered CT image shows multifocal periapical sclerotic lesions with sharp margins (arrows). Note the coalescence of adjacent lesions. (b) Coronal reformatted CT image shows a cemento-osseous dysplasia lesion (arrows), which does not fuse to the tooth root, unlike cementoblastoma. (5) Early cemento-osseous dysplasia. Coronal CT image shows a low-attenuation periapical lesion with central calcification (arrows), a finding indicative of early cemento-osseous dysplasia and that may be confused with a periapical inflammatory lesion. However, periapical inflammatory lesions are unlikely in vital teeth (ie, with no caries or restoration).

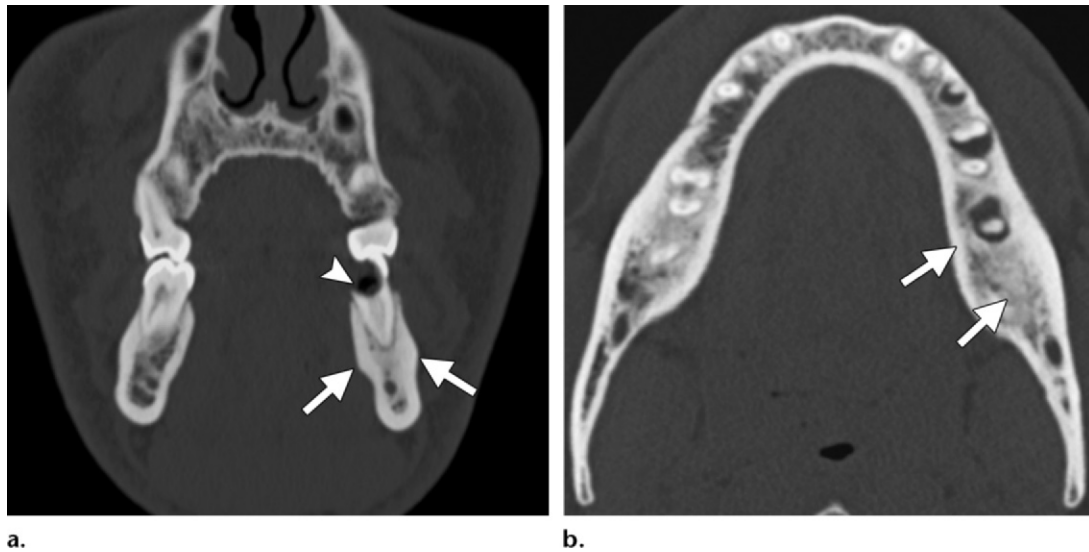


Figure 7. Condensing osteitis in two patients. **(a)** Coronal CT image shows a poorly marginated non-expansile sclerotic lesion (arrows), a finding indicative of condensing osteitis. The lesion is associated with a carious lesion (arrowhead). **(b)** Axial CT image obtained in a different patient shows two poorly marginated nonexpansile sclerotic lesions (arrows) associated with periapical inflammatory lesions, a finding indicative of condensing osteitis.

Condensing Osteitis

Condensing osteitis occurs in children and young adults, usually in the premolar and molar areas of the mandible. It is a localized form of reactive osteitis and sclerosis that surrounds the apices of teeth with pulpitis or pulpal necrosis. The adjacent tooth usually has a thickened periodontal ligament or periapical inflammatory lesion (eg, granuloma, cyst, or abscess). At imaging, condensing osteitis is seen as a periapical, poorly marginated, nonexpansile, sclerotic lesion associated with a carious tooth, and it may be uni- or multifocal (Fig 7) (7).

Odontomas

Odontomas are the most common odontogenic tumor. The result of a developmental anomaly (hamartoma), they may obstruct tooth eruption and are most commonly seen in children. Approximately one-half of all odontomas are associated with an impacted tooth, although they may develop before or after tooth eruption. At imaging, odontomas are usually pericoronal, sharply marginated, and sclerotic, with a low-attenuation halo. They may be expansile or appear purely lucent in their evolution.

Odontomas are classified as simple, compound, or complex. Simple odontomas appear

as supernumerary teeth. Compound odontomas consist of multiple small toothlike structures called denticles and most commonly arise in the anterior maxilla. Complex odontomas appear as an amorphous hyperattenuating conglomerate mass of enamel and dentin, most commonly in the molar regions the jaws (Fig 8). A complex odontoma may be confused with an osteoma; the low-attenuation halo that surrounds odontoma may help differentiate these lesions (2,8). Occasionally, odontoma is associated with dentigerous or calcifying odontogenic cysts (4).

Idiopathic Osteosclerosis

Idiopathic osteosclerosis is a focal solitary sclerotic lesion that arises in the late 1st or early 2nd decade of life. Its cause is unknown. It is asymptomatic, is not associated with inflammation, and may remain static or demonstrate slow growth that usually stops when the patient reaches skeletal maturity. In 90% of patients it occurs in the mandible, usually near the first molar or second molar or premolar. At imaging, idiopathic osteosclerosis has sharp margins and is small and round or oval, sclerotic, periapical, and tooth related, occasionally with peripheral spiculation. In 20% of patients, it is not related to a tooth. Idiopathic osteosclerosis is not expansile and has no low-attenuation rim (Fig 9) (9,10). Some patients may have multiple lesions.



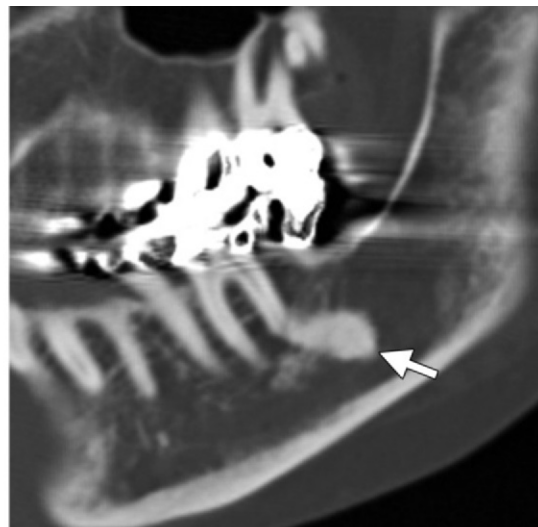
Figure 8. Odontoma in three patients. **(a)** Three-dimensional volume-rendered CT image obtained in a child shows several simple odontomas (arrows) that resemble supernumerary teeth. **(b)** Axial CT image shows multiple small toothlike structures (arrows), a finding indicative of a compound odontoma. **(c)** Sagittal reformatted CT image shows a conglomerate mass of enamel and dentin surrounding the crown of the tooth (arrows), a finding indicative of a complex odontoma. A low-attenuation halo is also seen, a finding that may help differentiate compound odontoma from osteoma.

a.



b.

c.



a.

b.

Figure 9. Idiopathic osteosclerosis in two patients. Axial **(a)** and sagittal reformatted **(b)** CT images show a sclerotic eccentric tooth-related lesion (arrow) with sharp, spiculated margins. No low-attenuation rim is seen.



Figure 10. Osteoma in two patients. **(a)** Axial CT image shows a well-circumscribed sclerotic mass with smooth margins in the mandibular ramus (arrows). The mass is associated with mild bone expansion. No low-attenuation halo is seen. In the absence of bone expansion, it may not be possible to differentiate osteoma and idiopathic osteosclerosis. **(b)** Sagittal volume-rendered CT image obtained in a different patient shows an osteoma (arrowheads) in the posterior mandibular body and ramus with associated expansile simple bone cysts (arrows).

Sclerotic Non-Tooth-related Lesions

Osteomas

Sclerotic non-tooth-related lesions include expansile lesions, such as osteomas, and exophytic lesions, such as exostoses and tori. Osteomas are benign tumors composed of mature compact or cancellous bone. They most commonly arise in the craniofacial bones. The most common location in the jaw is the posterior mandibular body or condyle. Multiple osteomas may be associated with Gardner syndrome (11). At imaging, osteomas appear as a non-tooth-related circumscribed sclerotic mass. Bone expansion may be present.

No perilesional halo is seen, and it may not be possible to differentiate osteomas from idiopathic osteosclerosis if no bone expansion is present.

Osteomas may demonstrate exophytic growth, and they may be associated with simple bone cysts (Fig 10) (12).

Tori and Exostoses

Tori and exostoses are protuberances of dense cortical bone that most commonly arise in adults. Occasionally, they contain a small amount of marrow. They are covered by a thin poorly vascularized mucosa and are characterized by slow growth that usually arrests spontaneously. Their cause is unclear, although genetic and environ-

mental influences may play a role. Tori and exostoses usually manifest with no symptoms, except in the case of trauma, and they may complicate denture fitting. Such lesions include buccal exostosis, which arises from the buccal cortex of the maxilla; torus mandibularis, which arises above the mylohyoid line, along the lingual surface of the mandible; torus palatinus, which arises from the midline hard palate; and torus maxillaris, which arises from the lingual surface of the posterior maxilla (Fig 11) (13).

Jaw Lesions with Ground-Glass Attenuation

Ossifying Fibroma

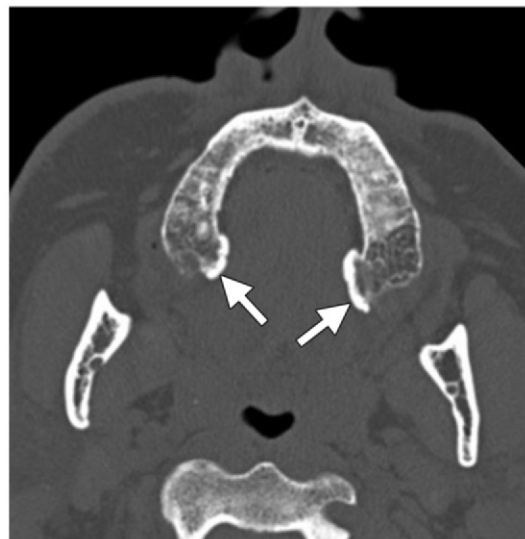
Although lesions with ground-glass attenuation may contain lytic or sclerotic areas (depending on how evolved they are and the degree of mineralization), they are characterized by dominant areas of ground-glass attenuation. Such lesions include ossifying fibroma, fibrous dysplasia, and renal osteodystrophy (Table 2). Ossifying fibroma includes the following subtypes: cementifying fibroma, cemento-ossifying fibroma, and juvenile ossifying fibroma (including its psammomatoid and trabecular variants). Overall, ossifying fibroma is more common in women than in men, and it typically manifests with painless swelling around the mandible. The premolar and molar areas are most commonly affected. Although ossifying fibroma may occur in patients with a wide age range, its incidence peaks in the 3rd and 4th decades of life.



a.



b.



c.

Figure 11. Exostoses and tori. CT images obtained in three different patients show torus mandibularis (arrows in **a**), torus palatinus (arrows in **b**), and torus maxillaris (arrows in **c**).

Table 2
Jaw Lesions with Ground-Glass Attenuation

Type	Lesion
Unifocal	Ossifying fibroma*, monostotic fibrous dysplasia
Multifocal	Florid cemento-osseous dysplasia, multiple ossifying fibromas, brown tumors (hyperparathyroidism)
Diffuse	Renal osteodystrophy, fibrous dysplasia

*Ossifying fibroma may also manifest as a low-attenuation non-tooth-related lesion with fuzzy margins.

Ossifying fibroma is composed of fibrous tissue, bone trabeculae, and cementum-like spherules that arise from a single progenitor cell. In contradistinction to fibrous dysplasia, the bone

spicules in ossifying fibroma are rimmed by osteoblasts. Because ossifying fibroma may occur outside the jaw, some contend that the cementum-like material found within some lesions represents a variant of bone rather than odontogenic products (ie, true cementum). These neoplasms have significant potential for centrifugal growth perpendicular to the long axis of bone.

Juvenile ossifying fibroma, especially the trabecular form, is aggressive and destructive and most commonly occurs in boys younger than 15 years old. It may arise in either the maxilla or mandible. The psammomatoid variant of juvenile ossifying fibroma predominantly involves the extragnathic craniofacial bones (eg, the periorbital, frontal, and ethmoid regions).

At imaging, ossifying fibroma typically appears as a solitary well-defined unilocular focally expansile lesion with sharp margins, ground-glass



Figure 12. Ossifying fibroma. Axial CT images obtained in two different patients show well-defined, focally expansile, sharply marginated lesions with predominantly ground-glass attenuation. The presence of a narrow zone of transition (arrows) helps differentiate ossifying fibroma from fibrous dysplasia.

attenuation, and no low-attenuation halo. Occasionally, a sclerotic border may be seen. Ossifying fibroma typically appears radiolucent early in its evolution because it contains nonmineralized osteoid. Later, it becomes more radiopaque as the matrix becomes more mineralized. Areas of soft-tissue enhancement may be seen, and tooth displacement and erosion are common (Fig 12) (14,15).

Hyperparathyroidism–jaw tumor syndrome may be associated with multiple ossifying fibromas, renal cysts, and Wilms tumors and results from mutation of the tumor suppressor gene *HRPT2*. Parathyroid adenomas or carcinomas may also arise in patients with hyperparathyroidism–jaw tumor syndrome (16).

Some radiopaque jaw lesions may be entirely radiolucent early in their evolution. For example, early cemento-osseous dysplasia may be confused with a periapical inflammatory lesion; however, cemento-osseous dysplasia is associated with a vital tooth with an intact lamina dura and can demonstrate a central area of calcification. Likewise, early ossifying fibromas may be radiolucent and become more radiopaque with maturation.

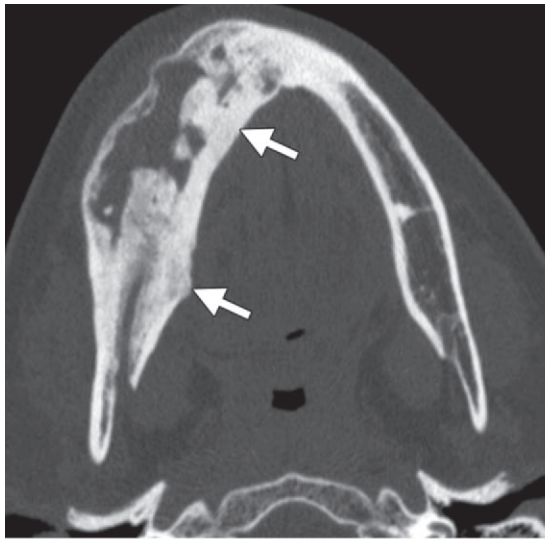
Fibrous Dysplasia

Fibrous dysplasia tends to expand along the longitudinal axis of the affected bone, which demonstrates enlargement, with less focal alteration to its general shape than is typically seen with ossifying fibromas. Fibrous dysplasia is composed of cellular fibrous tissue and woven bone trabeculae, with no

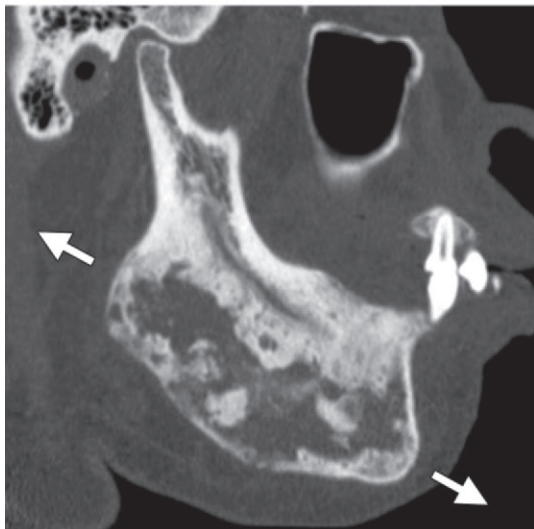
osteoblastic rimming (unlike ossifying fibroma). Activating missense mutations of the gene that encodes a subunit of the stimulatory G protein are consistently found in fibrous dysplasia lesions but not in other fibroosseous lesions (14). Traditionally, it was believed that fibrous dysplasia becomes quiescent at skeletal maturity. However, enlarging fibrous dysplasia may be encountered in adults. The craniofacial region is the most common site of malignant degeneration of fibrous dysplasia. The native shape of the bone is usually maintained unless cystic changes predominate. Most craniofacial fibrous dysplastic lesions are monostotic.

There are three different types of polyostotic fibrous dysplasia that may affect multiple craniofacial bones: craniofacial fibrous dysplasia, Lichtenstein-Jaffe fibrous dysplasia, and Albright syndrome. Craniofacial fibrous dysplasia only affects craniofacial bones. In the Lichtenstein-Jaffe type of fibrous dysplasia, polyostotic disease affects both craniofacial and noncraniofacial bones. Patients may present with cutaneous café au lait spots and rare endocrinopathies. Albright syndrome is characterized by severe polyostotic fibrous dysplasia (mostly unilateral), cutaneous café au lait spots, and various endocrinopathies (typically in girls with precocious puberty) (17). In Mazabraud syndrome, fibrous dysplasia is associated with soft-tissue myxomas (14,18).

At imaging, fibrous dysplasia is seen as a heterogeneous lesion with ground-glass attenuation and a wide, ill-defined transition zone, a feature that helps differentiate it from ossifying fibroma (Figs 13, 14). Its cortex remains intact and is often



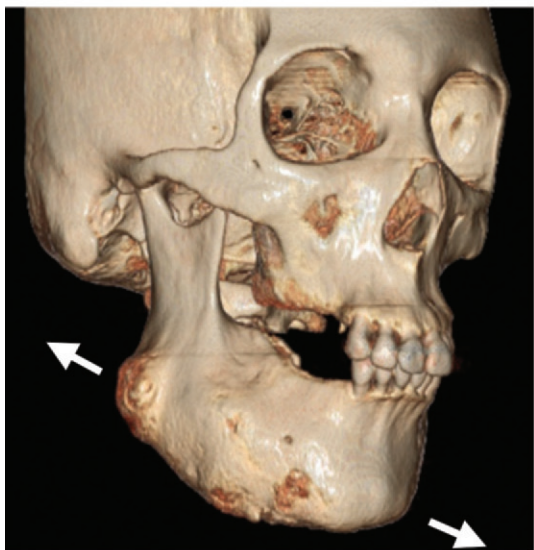
13a.



13b.



14a.



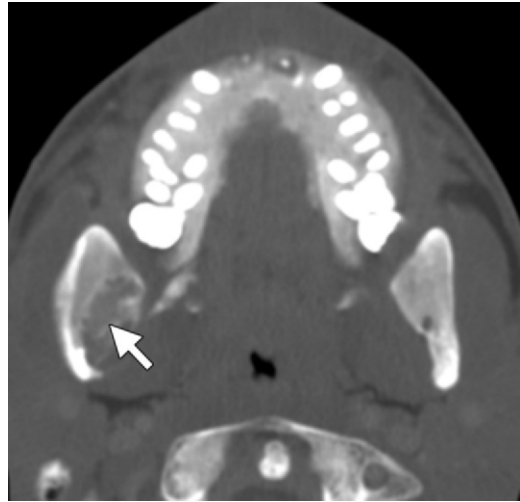
13c.



14b.

Figures 13, 14. (13) Monostotic fibrous dysplasia. (a) Axial CT image shows an expansile lesion with heterogeneous ground-glass attenuation. The transition zone that separates clearly normal bone from abnormal bone is wide and ill defined, and marked cortical thickening is seen along the lingual mandibular cortex (arrows), a finding more commonly seen in fibrous dysplasia than ossifying fibroma. (b, c) Sagittal reformatted (b) and volume-rendered (c) CT images show that expansion of the lesion (arrows) is greater in the longitudinal direction than in the transverse expansion. (14) Polyostotic (craniofacial) fibrous dysplasia. Sagittal reformatted (a) and volume-rendered (b) CT images show diffuse expansion of multiple facial and cranial bones, which demonstrate ground-glass attenuation, a finding indicative of fibrous dysplasia.

Figure 15. Renal osteodystrophy in a patient with chronic renal failure. Axial CT image shows an area of diffuse ground-glass attenuation and bone expansion involving the maxilla and mandible and a low-attenuation area in the right mandible (arrow). Although diffuse ground-glass attenuation similar to that in fibrous dysplasia is seen, the maxillary cortex is nearly imperceptible in many places (rather than thickened), a finding indicative of renal osteodystrophy.



thickened and sclerotic. The dental lamina dura may be involved and altered to the fibrous dysplasia pattern (a result of “field effect”). The teeth are often nondisplaced, unlike in ossifying fibroma, and fibrous dysplasia often encroaches on the orbit, sinuses, and vascular and neural canals (19).

Both ossifying fibroma and fibrous dysplasia demonstrate ground-glass attenuation at imaging. The presence of a well-defined narrow transition zone differentiates ossifying fibroma from fibrous dysplasia, which has an ill-defined wide zone of transition. Other features that indicate fibrous dysplasia are a longitudinal growth pattern, nondisplaced teeth, and crossing of sutures. At pathologic examination, osteoblastic rimming is seen in ossifying fibroma but not fibrous dysplasia.

Renal Osteodystrophy

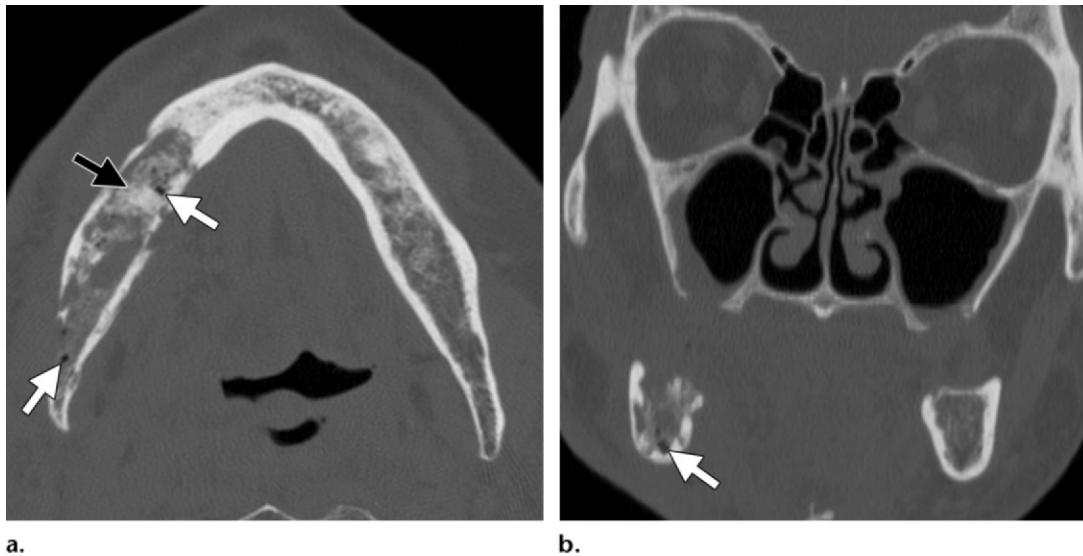
Craniofacial bone lesions may occur in patients with primary or secondary hyperparathyroidism, but benign fibroosseous lesions are more common in patients with renal osteodystrophy, which affects 90% of patients undergoing dialysis. Renal osteodystrophy comprises a spectrum of bone diseases caused by metabolic derangements that are associated with renal

Table 3
Mixed Lytic and Sclerotic Lesions of the Jaw

Osteoradionecrosis
Bisphosphonate-related osteonecrosis
Mandibular osteomyelitis

insufficiency and secondary hyperparathyroidism (14,20). Patients with chronic renal failure develop secondary hyperparathyroidism with increased osteoclastic activity.

At imaging, early renal osteodystrophy is associated with replacement of normal trabeculation by areas of diffuse ground-glass attenuation and a loss of definition of the cortex, the lamina dura, and the wall of the inferior alveolar nerve canal, a pattern that mimics that of diffuse fibrous dysplasia. The more advanced disease pattern known as osteitis fibrosa is characterized by mixed osteolysis and sclerosis and a more heterogeneous appearance with bone resorption, osteoid production, and increased bone remodeling (Fig 15). In some patients, markedly expansile facial deformity is present, with macrognathia, cortical thickening, and protrusion and splaying of teeth, deformi-



a.

b.

Figure 16. Mandibular osteoradionecrosis in a patient with a history of radiation therapy. Axial (**a**) and coronal reformatted (**b**) CT images show sclerosis, loss of trabeculation, cortical interruptions, and areas of gas attenuation (white arrows) in the right mandible, findings consistent with osteoradionecrosis. More sclerotic necrotic bone (black arrow in **a**) is also seen.

ties that may not resolve after parathyroidectomy (14,21,22). Brown tumors (also known as osteitis fibrosa cystica) of hyperparathyroidism may be seen in patients with primary or secondary hyperparathyroidism and are often expansile and lytic, with proliferating osteoclasts and osteoblasts and multinucleated giant cells and reactive fibrous stroma, hypervascularity, and old hemorrhage.

Mixed Lytic and Sclerotic Jaw Lesions

Osteoradionecrosis

Mixed lytic and sclerotic jaw lesions, such as osteoradionecrosis, bisphosphonate-related osteonecrosis of the jaw, mandibular osteomyelitis, and primary chronic osteomyelitis, include components at the opposite extremes of attenuation and may contain gas (Table 3). As many as 37% of patients who undergo radiation therapy to the head and neck develop bone necrosis secondary to hypoxia, hypovascularity, hypocellularity, and fibrosis (23). Osteoradionecrosis is rare in patients who receive a dose of less than 60 Gy. The

mandible is involved more frequently than the maxilla, probably because of its less robust blood supply. The buccal cortex is more vulnerable than the lingual cortex, and the mandibular body is most commonly affected. The chin and angles of the mandible are spared, presumably because of its muscular insertions. At imaging, osteoradionecrosis appears as an area of marked sclerosis with a loss of trabeculation in spongiosa, cortical interruptions, bone fragmentation or sequestration, and areas of gas attenuation in bone with poorly marginated adjacent fluid collections or areas of soft-tissue attenuation (Fig 16) (24,25).

Bisphosphonate-related Osteonecrosis of the Jaw

Bisphosphonate-related osteonecrosis of the jaw (BRONJ) is associated with the use of oral or intravenous bisphosphonates to treat various bone conditions such as osteoporosis, multiple myeloma, metastasis, and Paget disease. Osteonecrosis may be spontaneous; it commonly occurs

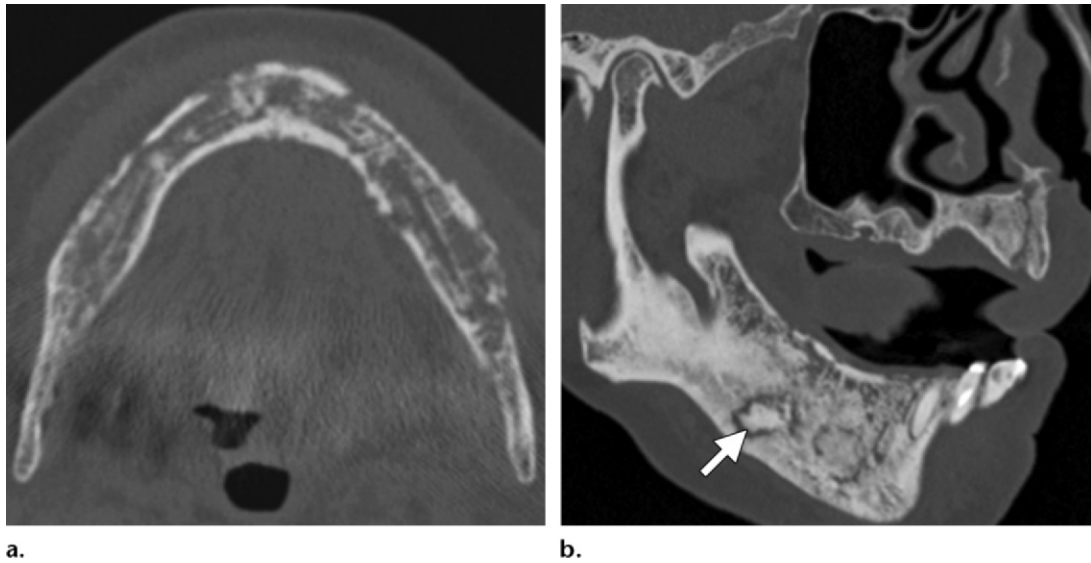


Figure 17. BRONJ in two patients. Axial (**a**) and sagittal reformatted (**b**) CT images show symmetric, poorly marginated, mixed sclerotic and lytic lesions throughout the mandible with multiple cortical interruptions. The lesions are bilateral. An area of evolving sclerotic necrotic bone (arrow in **b**) is also seen.

in the mylohyoid ridge or is precipitated by a traumatic procedure such as tooth extraction or dental surgery. BRONJ should be considered in patients undergoing bisphosphonate therapy with findings of bone necrosis and no history of radiation therapy. Bisphosphonates inhibit endothelial proliferation and interrupt intraosseous circulation. The risk for osteonecrosis is higher with concurrent steroid therapy. BRONJ is typically painful, but some patients may be asymptomatic. Maxillary osteoradionecrosis and osteomyelitis are unusual because of the rich blood supply of the maxilla. BRONJ may also be associated with infection by *Actinomyces* organisms. At imaging, BRONJ is seen as a poorly marginated diffuse area of low attenuation with bilateral symmetric sclerosis (Fig 17) (26–28).

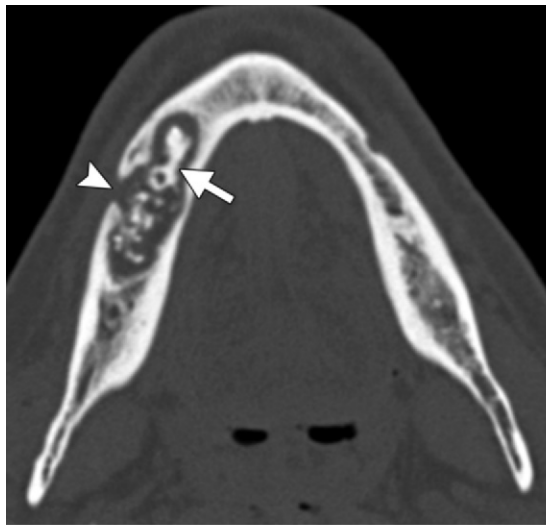
Mandibular Osteomyelitis

Osteomyelitis is much more common in the mandible than the maxilla, which is involved in only 1%–6% of cases because of its rich blood supply. Most patients with mandibular osteomyelitis have a history of antecedent dental caries

or dental extractions. Other causes of osteomyelitis include dental or mandibular fractures, osteoradionecrosis and, rarely, hematogenous spread of infection.

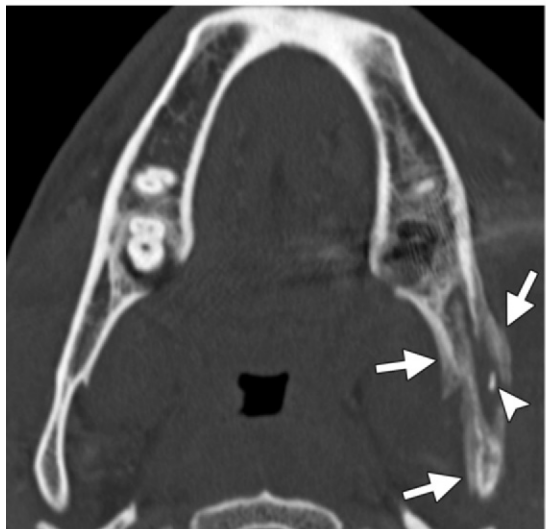
Chronic osteomyelitis, which is characterized by a duration longer than 1 month, may be complicated by sinuses, fistulae, osseous sequestra, or pathologic fractures. Risk factors for osteomyelitis include impaired immunity, as occurs in insulin-dependent diabetes mellitus, alcoholism, and malnutrition; conditions that affect mandibular blood supply, such as sickle cell disease and collagen vascular diseases and radiation therapy; and bone conditions, such as osteopetrosis. Imaging findings of mandibular osteomyelitis include cortical interruption, sclerotic sequestra in low-attenuation zones, periosteal new bone formation, and areas of gas attenuation (Fig 18) (29,30).

Proliferative periostitis is a lamellated pattern of periosteal new bone reaction that produces focal bone expansion (Fig 19). It most commonly occurs in children or young adults in the molar or premolar regions of the mandible. Underlying causes include dental caries with periapical inflammatory disease, periodontal infection, fracture, and nonodontogenic infection. Periosteal proliferation is usually maximal along the inferior mandibular and buccal cortices. Most cases are unifocal (31).



a.

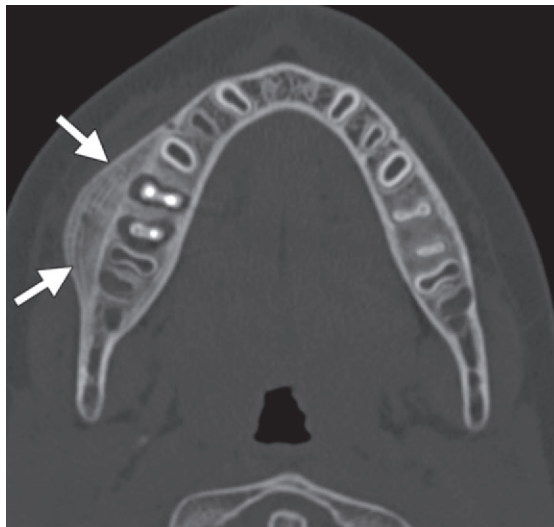
Figure 18. Mandibular osteomyelitis in two patients. (a) Axial CT image shows sclerotic sequestrum (arrow) with a surrounding low-attenuation zone and a sinus tract through the buccal lingual cortex (arrowhead). (b, c) Axial (b) and coronal reformatted (c) CT images show cloaking periosteal new bone formation (arrows), a finding referred to as “onion skinning,” and sequestrum (arrowhead).



b.



c.



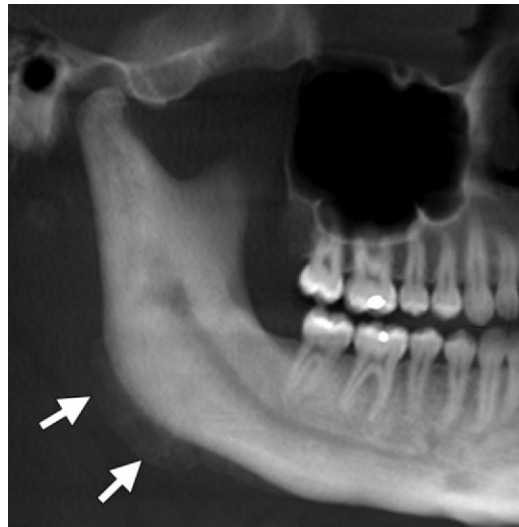
a.



b.

Figure 19. Proliferative periostitis in a child. Axial (a) and volume-rendered (b) CT images show focal mandibular buccal cortical expansion by lamellated periosteal new bone formation (arrows). Actinomycosis was seen at pathologic analysis of biopsy specimens.

Figure 20. Long-standing primary chronic osteomyelitis in an adult. Cropped and reconstructed CT panorex image shows diffuse mandibular sclerosis with fluffy periosteal new bone formation (arrows) at the mandibular angle.



Primary Chronic Osteomyelitis

Primary chronic osteomyelitis is a nonsuppurative inflammatory process; its cause is unknown (32). It has multiple names, including diffuse sclerosing osteomyelitis, chronic osteomyelitis with proliferative periostitis, periostitis ossificans, nonsuppurative osteomyelitis, and osteomyelitis sicca. Primary chronic osteomyelitis has no acute phase and manifests with insidious jaw swelling and normal mucosa. Its occurrence is unrelated to dentition status, and it peaks in childhood or early adolescence and after age 50. Absence of a fever and leukocytosis are characteristic, and associated teeth are typically vital. At imaging, it may initially appear as a poorly marginated lesion with progressive sclerosis, scattered osteolysis, bone expansion, and an “onion skin” periosteal reaction. In chronic cases, diffuse sclerosis predominates, a finding that may cause it to be confused with fibrous dysplasia or Paget disease (Fig 20) (32).

Summary

Diagnosis of radiopaque jaw lesions may be approached by categorizing the lesion according to its appearance, relation to the teeth, and exact location with reference to the teeth. Most radiopaque jaw lesions have a characteristic imaging appearance. Awareness of the demographic distribution of these lesions and their associated clinical features, as well as the radiologic approach, is important to explore the “terra incognita” of radiopaque jaw lesions.

References

1. Morgan PR. Odontogenic tumors: a review. *Periodontol* 2000;57(1):160–176.
2. Kaneda T, Minami M, Kurabayashi T. Benign odontogenic tumors of the mandible and maxilla. *Neuroimaging Clin N Am* 2003;13(3):495–507.
3. Zaitoun H, Kujan O, Sloan P. An unusual recurrent cementoblastoma associated with a developing lower second molar tooth: a case report. *J Oral Maxillofac Surg* 2007;65(10):2080–2082.
4. Slootweg PJ. Lesions of the jaws. *Histopathology* 2009;54(4):401–418.
5. Resnick CM, Novelline RA. Cemento-osseous dysplasia, a radiological mimic of periapical dental abscess. *Emerg Radiol* 2008;15(6):367–374.

6. Summerlin DJ, Tomich CE. Focal cemento-osseous dysplasia: a clinicopathologic study of 221 cases. *Oral Surg Oral Med Oral Pathol* 1994;78(5):611–620.
7. Abrahams JJ, Berger SB. Inflammatory disease of the jaw: appearance on reformatted CT scans. *AJR Am J Roentgenol* 1998;170(4):1085–1091.
8. Theodorou SJ, Theodorou DJ, Sartoris DJ. Imaging characteristics of neoplasms and other lesions of the jawbones: part 1—odontogenic tumors and tumor-like lesions. *Clin Imaging* 2007;31(2):114–119.
9. MacDonald-Jankowski DS. Idiopathic osteosclerosis in the jaws of Britons and of the Hong Kong Chinese: radiology and systematic review. *Dentomaxillofac Radiol* 1999;28(6):357–363.
10. Yonetsu K, Yuasa K, Kanda S. Idiopathic osteosclerosis of the jaws: panoramic radiographic and computed tomographic findings. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1997;83(4):517–521.
11. Fonseca LC, Kodama NK, Nunes FC, et al. Radiographic assessment of Gardner's syndrome. *Dentomaxillofac Radiol* 2007;36(2):121–124.
12. Kaplan I, Nicolaou Z, Hatuel D, Calderon S. Solitary central osteoma of the jaws: a diagnostic dilemma. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2008;106(3):e22–e29.
13. Jainkittivong A, Langlais RP. Buccal and palatal exostoses: prevalence and concurrence with tori. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2000;90(1):48–53.
14. Eversole R, Su L, ElMofty S. Benign fibro-osseous lesions of the craniofacial complex: a review. *Head Neck Pathol* 2008;2(3):177–202.
15. Regezi JA. Odontogenic cysts, odontogenic tumors, fibrous, and giant cell lesions of the jaws. *Mod Pathol* 2002;15(3):331–341.
16. Aldred MJ, Talacko AA, Savarirayan R, et al. Dental findings in a family with hyperparathyroidism-jaw tumor syndrome and a novel HRPT2 gene mutation. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2006;101(2):212–218.
17. Abdelkarim A, Green R, Startzell J, Preece J. Craniofacial polyostotic fibrous dysplasia: a case report and review of the literature. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2008;106(1):e49–e55.
18. Faivre L, Nivelon-Chevallier A, Kottler ML, et al. Mazabraud syndrome in two patients: clinical overlap with McCune-Albright syndrome. *Am J Med Genet* 2001;99(2):132–136.
19. MacDonald-Jankowski DS. Fibro-osseous lesions of the face and jaws. *Clin Radiol* 2004;59(1):11–25.
20. Fatahzadeh M. The spectrum of orofacial manifestations in renal osteodystrophy: diagnostic challenges of an uncommon presentation. *Quintessence Int* 2011;42(7):e78–e88.
21. Chang JI, Som PM, Lawson W. Unique imaging findings in the facial bones of renal osteodystrophy. *AJNR Am J Neuroradiol* 2007;28(4):608–609.
22. Kalyvas D, Tosios KI, Leventis MD, Tsiklakis K, Angelopoulos AP. Localized jaw enlargement in renal osteodystrophy: report of a case and review of the literature. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2004;97(1):68–74.
23. Lyons A, Ghazali N. Osteoradionecrosis of the jaws: current understanding of its pathophysiology and treatment. *Br J Oral Maxillofac Surg* 2008;46(8):653–660.
24. Hermans R. Imaging of mandibular osteoradionecrosis. *Neuroimaging Clin N Am* 2003;13(3):597–604.
25. Chong J, Hinckley LK, Ginsberg LE. Masticator space abnormalities associated with mandibular osteoradionecrosis: MR and CT findings in five patients. *AJNR Am J Neuroradiol* 2000;21(1):175–178.
26. Mortensen M, Lawson W, Montazem A. Osteonecrosis of the jaw associated with bisphosphonate use: presentation of seven cases and literature review. *Laryngoscope* 2007;117(1):30–34.
27. Bedogni A, Blandamura S, Lokmic Z, et al. Bisphosphonate-associated jawbone osteonecrosis: a correlation between imaging techniques and histopathology. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2008;105(3):358–364.
28. Phal PM, Myall RW, Assael LA, Weissman JL. Imaging findings of bisphosphonate-associated osteonecrosis of the jaws. *AJNR Am J Neuroradiol* 2007;28(6):1139–1145.
29. Hudson JW. Osteomyelitis of the jaws: a 50-year perspective. *J Oral Maxillofac Surg* 1993;51(12):1294–1301.
30. Schuknecht B, Valavanis A. Osteomyelitis of the mandible. *Neuroimaging Clin N Am* 2003;13(3):605–618.
31. Tong AC, Ng IO, Yeung KM. Osteomyelitis with proliferative periostitis: an unusual case. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2006;102(5):e14–e19.
32. Theologie-Lygidakis N, Schoinohoriti O, Iatrou I. Surgical management of primary chronic osteomyelitis of the jaws in children: a prospective analysis of five cases and review of the literature. *Oral Maxillofac Surg* 2011;15(1):41–50.

Radiopaque Jaw Lesions: An Approach to the Differential Diagnosis

Joel K. Curé, MD • Surjith Vattoth, MD, DNB, FRCR • Ritu Shah, MD

RadioGraphics 2012; 32:1909–1925 • Published online 10.1148/rg.327125003 • Content Codes: CT HN MK NR

Page 1910

The radiologic diagnosis of jaw lesions is informed by imaging features such as attenuation, margination (a narrow or wide transition zone), and the relationship of the lesion to adjacent teeth. Although cystic jaw lesions are not discussed in this article, these basic observations are essential when analyzing any type of jaw lesion.

Page 1912

Both cementoblastoma and cemento-osseous dysplasia are periapical, sclerotic, sharply marginated lesions with a low-attenuation halo. However, cementoblastoma occurs in children and young adults and fuses directly to the tooth root, whereas cemento-osseous dysplasia is more common among black women and women of Asian descent who are in the 4th or 5th decade of life and does not fuse to the tooth root.

Page 1916

No perilesional halo is seen, and it may not be possible to differentiate osteomas from idiopathic osteosclerosis if no bone expansion is present.

Page 1918

Some radiopaque jaw lesions may be entirely radiolucent early in their evolution. For example, early cemento-osseous dysplasia may be confused with a periapical inflammatory lesion; however, cemento-osseous dysplasia is associated with a vital tooth with an intact lamina dura and can demonstrate a central area of calcification. Likewise, early ossifying fibromas may be radiolucent and become more radiopaque with maturation.

Page 1920

Both ossifying fibroma and fibrous dysplasia demonstrate ground-glass attenuation at imaging. The presence of a well-defined narrow transition zone differentiates ossifying fibroma from fibrous dysplasia, which has an ill-defined wide zone of transition. Other features that indicate fibrous dysplasia are a longitudinal growth pattern, nondisplaced teeth, and crossing of sutures. At pathologic examination, osteoblastic rimming is seen in ossifying fibroma but not fibrous dysplasia.