# Periapical Lucency around the Tooth: Radiologic Evaluation and Differential Diagnosis[1]

*Margaret N. Chapman, MD • Rohini N. Nadgir, MD • Andrew S. Akman, MD, MBA • Naoko Saito, MD, PhD • Kotaro Sekiya, DDS • Takashi Kaneda, DDS, PhD • Osamu Sakai, MD, PhD*

**ONLINE-ONLY CME**

*See www.rsna.org/education/search/RG*

**LEARNING OBJECTIVES**

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

- Review the pathophysiology of apical periodontitis and how it can lead to the development of periapical lucency.
- Recognize the imaging features of complications due to periodontitis, including orbital, sinonasal, and intracranial sequelae.
- Discuss the characteristic imaging features of other odontogenic and non-odontogenic lesions that may cause lucent lesions around the tooth root and how these can be distinguished radiologically from periapical lucency caused by periodontitis.

**TEACHING POINTS**

*See last page*

Periapical lucencies are often seen incidentally at head and neck imaging studies performed for indications not related to the teeth. These lesions are, however, occasionally manifestations of diseases that have a wide range of effects and may at times represent the source of symptoms that prompted the study. The vast majority of periapical lucencies are the result of apical periodontal or pulpal disease. If found in an advanced state or left untreated, disease related to the tooth may spread to adjacent tissues, including the sinuses, orbits, deep fascial spaces of the neck, and intracranial structures, and result in a significant increase in patient morbidity and mortality. Although the majority of periapical lucencies seen on radiographs and computed tomographic images occur secondary to apical periodontal or pulpal disease, not all lucencies near the tooth root are due to infection. Lucency near the tooth root may be seen in the setting of other diseases of odontogenic and non-odontogenic origin, including neoplasms. Although imaging findings for these lesions can include periapical lucent components, awareness of the varied secondary imaging features can aid the radiologist in developing an accurate differential diagnosis. Familiarity with the imaging features and differential diagnoses of diseases or conditions that cause lucency around the tooth root results in appropriate referral and prompt diagnosis, management, and treatment, and can prevent unnecessary additional imaging or intervention. In addition, early recognition and appropriate treatment of infectious processes will result in improved clinical outcomes and a decrease in morbidity and mortality.

## Introduction

Periapical lucency is often seen incidentally at head and neck imaging studies performed for indications not related to the teeth but may represent the cause of a patient's symptoms. According to one study, 78% of periapical lesions are the result of an infectious or inflammatory process, usually due to apical periodontal or pulpal disease (1). Recognition of the typical radiologic features of apical periodontal disease results in early referral and proper treatment. Inadequate treatment of disease

**Abbreviation:** KCOT = keratocystic odontogenic tumor.

**RadioGraphics 2013;** 33:E15–E32 • **Published online** 10.1148/rg.331125172 • **Content Codes:** CT HN MK NR

[1]From the Department of Radiology, Boston Medical Center, Boston University School of Medicine, FGH Building, 3rd Floor, 820 Harrison Ave, Boston, MA 02118 (M.N.C., R.N.N., A.S.A., N.S., K.S., O.S.); Department of Radiology, Saitama International Medical Center, Saitama Medical University, Hidaka, Saitama, Japan (N.S.); and Department of Radiology, Nihon University School of Dentistry at Matsudo, Chiba, Japan (K.S., T.K.). Presented as an education exhibit at the 2008 RSNA Annual Meeting. Received November 9, 2011; revision requested November 22 and received March 31, 2012; final version accepted August 16. For this journal-based SA-CME activity the authors, editor, and reviewers have no relevant relationships to disclose. **Address correspondence to** O.S. (e-mail: *osamu.sakai@bmc.org*)..

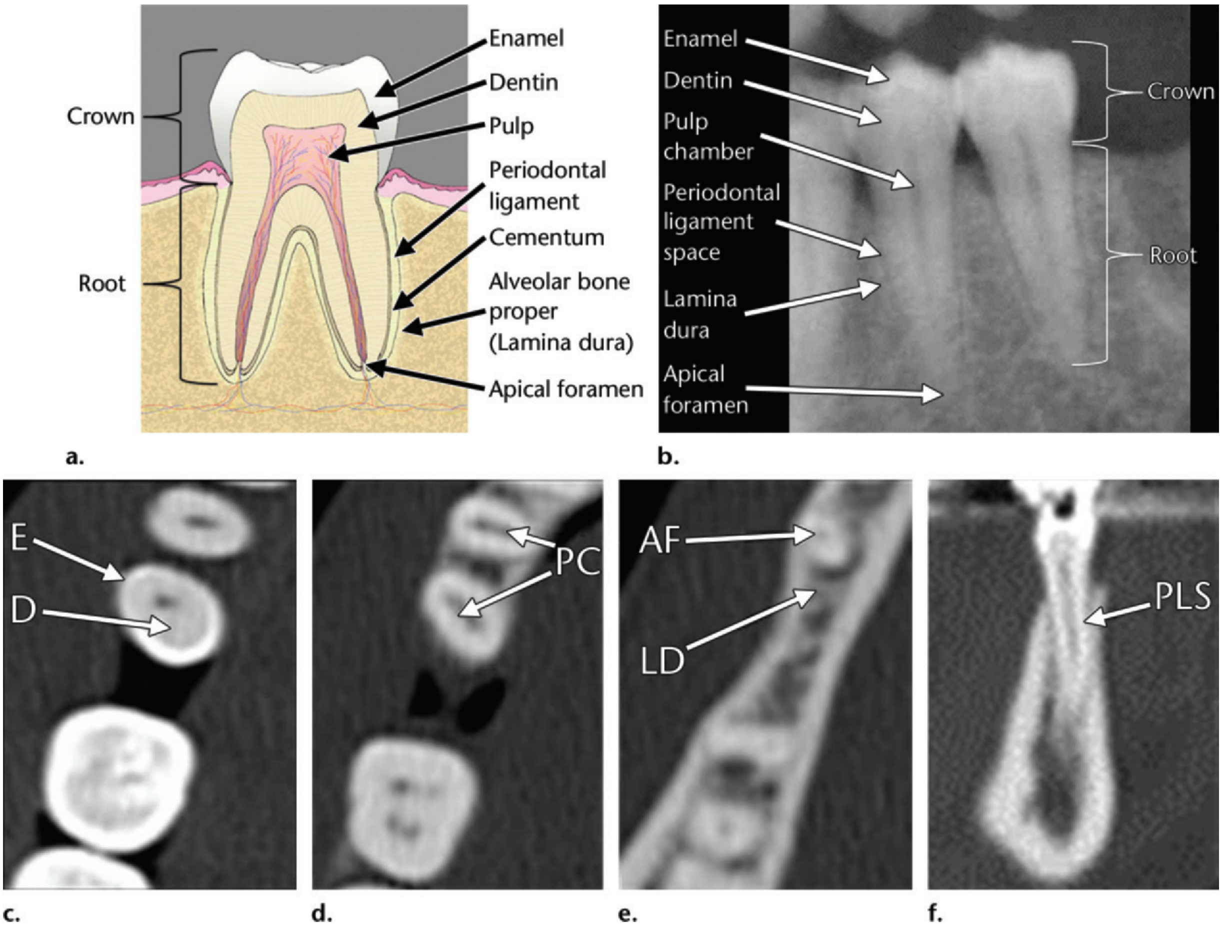



**Figure 1.** Schematic **(a)**, radiographic **(b)**, and CT (**c–e** [axial], **f** [sagittal reformation]) anatomy of the tooth. The extremely opaque enamel encases less opaque dentin in the crown. The more lucent pulp chamber and root canal are located within the center of the tooth. The apical foramen is the lucent central aspect at the tooth apex. The lamina dura is a layer of cortical bone that lines the tooth socket and separates it from adjacent cancellous bone. The periodontal ligament is the connective tissue located between the tooth apex and the lamina dura. *AF* = apical foramen, *D* = dentin, *E* = enamel, *LD* = lamina dura, *PC* = pulp chamber, *PLS* = periodontal ligament space.

related to the teeth may result in complications and contribute to increased patient morbidity and healthcare costs (2,3).

Our aim in this article is to discuss basic dental anatomy and review the pathogenesis, radiographic features, and complications of apical periodontitis. Differential considerations of other odontogenic and non-odontogenic lesions, both benign and malignant, that can manifest as a lucent lesion around the tooth root and mimic periapical lucency due to apical periodontal disease are also discussed.

## Radiologic Anatomy of the Tooth

The tooth sits in a socket of alveolar bone and is composed primarily of dentin, a bonelike substance of intermediate radiopacity. The crown—the portion of the tooth that projects out of the bone—is covered by enamel, the hardest substance in the human body and the most radiopaque. The root—the portion of the tooth embedded in the bone—is covered by cementum, which is less radiopaque than enamel and slightly less radiopaque than the underlying dentin. Cementum and dentin have similar opacities, and it is therefore difficult to differentiate them on radiographs or conventional computed tomographic (CT) images, unless a disease such as hypercementosis is present. Both enamel and cementum function as protective barriers (Fig 1) (4–8).

The pulp and root canal are located within the center of the tooth and consist of the neurovascular bundle and connective tissue, both of which are radiolucent on radiographs and CT images. The neurovascular bundle enters the tooth via the apical foramen, located at the apex of the tooth root (Fig 1a) (4,6,8). The lamina dura is a thin radiopacity that lines the tooth socket, usually 0.2–0.3 mm in its thickest dimension (4). The periodontal ligament is a thin layer of connective tissue between the

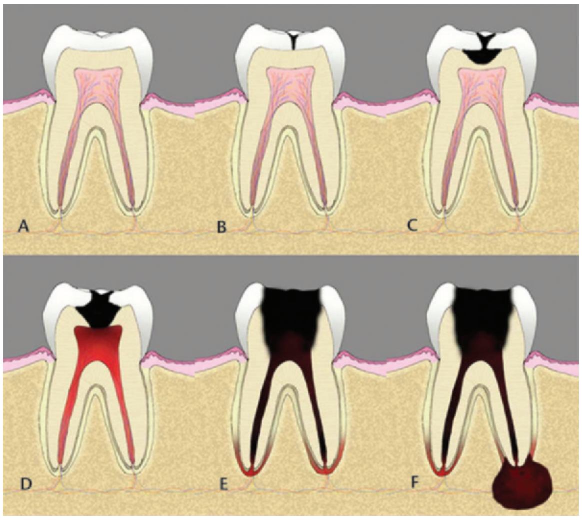



**Figure 2.** Drawing shows progression of apical periodontal disease: *A,* normal tooth; *B,* cavity formation; *C,* extension into dentin; *D,* pulpitis; *E,* apical periodontitis; *F,* advanced apical periodontitis.

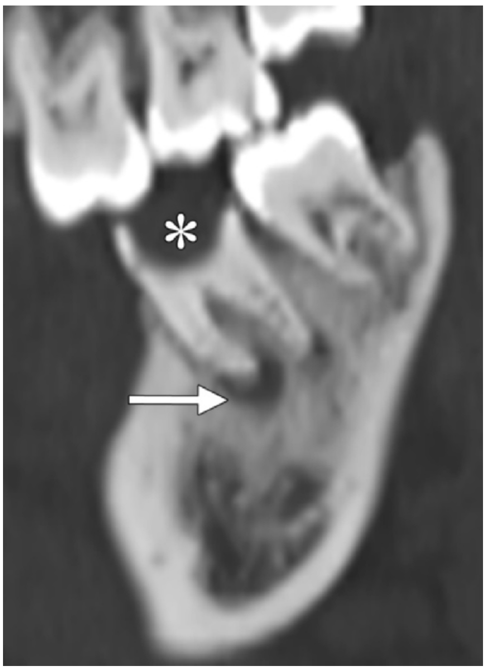


**Figure 3.** Cavity and periapical lucency (granuloma). Sagittal CT image demonstrates a large cavity (*) in the crown of a second mandibular molar, involving both enamel and dentin. Widening of the periodontal ligament (arrow) around the apex of the tooth is seen, consistent with apical periodontitis. Sclerosis of the bone surrounding the infected tooth suggests chronic inflammation.

lamina dura and cementum that holds the tooth in place (Fig 1). Expansion of the periodontal space is indicative of an underlying pathologic process (2,3,6,8).

Teaching Point

## Methods of Evaluation

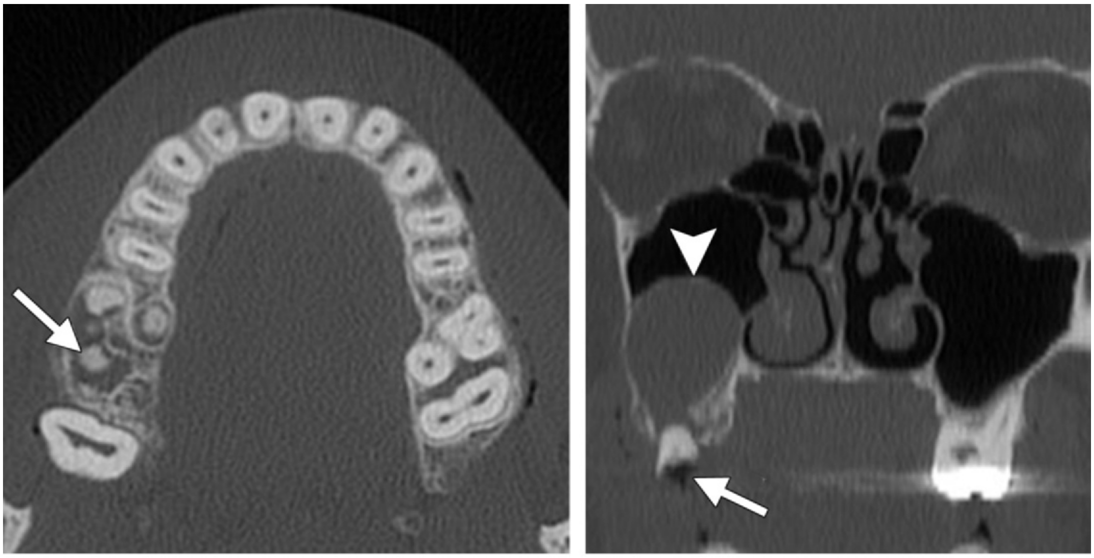
Intraoral radiography is the modality most frequently used to evaluate for carious and periapical disease in the dental clinic setting. It has high spatial resolution and provides detailed anatomy of the tooth and surrounding bone. Panoramic radiography can provide an overview of the maxilla and mandible and is frequently combined with intraoral radiography to increase the sensitivity and specificity of diagnosis of periapical lesions, because panoramic radiographs alone are often subject to variability in interpretation (9,10).

The extent of the lesion, the number of roots involved, the tooth type, and the relationship of the lesion to adjacent structures are often difficult to determine on intraoral or panoramic radiographs. Recently introduced cone-beam CT acquires high-spatial-resolution tomographic data with a small field of view and at a relatively low radiation dose compared with conventional CT (11,12). Cone-beam CT provides detailed anatomy of the teeth and bones and can be used to evaluate periapical lesions in multiple planes, thus allowing improved detection and anatomic delineation of disease (13–15). However, it has poor soft-tissue contrast resolution and is therefore not ideal for evaluation of extraosseous lesions (11,12).

Intraoral radiography, panoramic radiography, and cone-beam CT are usually performed in the outpatient dental setting and are coupled with a targeted physical examination, often with the benefit of specific symptoms and suspicion for dental disease. The study is usually interpreted by the treating dentist or oral maxillofacial radiologist and rarely interpreted by medical radiologists. In contrast, periapical lucencies identified in the hospital emergency department are often seen at conventional CT of the brain, face, and neck and can be incidental findings. Conventional CT has considerably higher contrast resolution than that obtained with the modalities already mentioned and is ideal for evaluating complications due to periapical disease, although its spatial resolution is slightly less than that of cone-beam CT (11).

## Periapical Infection or Inflammation

In the initiating steps of cavity formation, lactic acid–producing microbes form a film on the surface of the tooth and create an acidic microenvironment (16). Enamel, although dense, is acellular and will eventually be penetrated by the bacteria, thus forming caries (Fig 2b) (17). At CT, a cavity appears as a discontinuous hypoattenuating defect within the enamel along the surface of the crown (Fig 3).



a. b.

**Figure 4.** Periapical (radicular) cyst in a 48-year-old-man with left eye pain after an assault. **(a)** Axial CT image shows an incidental large cystic lesion involving the root of a carious right maxillary second molar (arrow) with adjacent sclerotic borders. **(b)** Coronal CT image shows the periapical lucency (arrow), with extension superiorly into the right maxillary sinus (arrowhead).

Acute pulpitis occurs when microbes gain entry into the pulp of the tooth by direct extension from a cavity that has caused breakdown of enamel and dentin (Fig 2b–2d) (18). Infected pulp can become necrotic as the inflammatory reaction progresses (Fig 2d) (18). An acute inflammatory reaction occurs at the apex of the tooth root if the infection spreads down the root and through the apical foramen (Fig 2e). In this situation, an influx of neutrophils, vascular congestion, and edema of the periodontal ligament result in apical periodontitis and widening of the periodontal ligament space. Although this inflammatory response typically causes pain and tenderness, it may be radiographically occult because tissue destruction has not yet occurred (4).

Teaching Point

Neutrophils cause localized tissue destruction and recruit osteoclasts and odontoclasts, which cause resorption of bone. At this stage, the periodontal space widens and a periapical lucency develops; these sequelae are radiographically visible (Fig 2f) (2). Neutrophils continue to destroy invading bacteria and are gradually replaced by delayed-phase adaptive immune cells such as lymphocytes, plasma cells, and macrophages (4,18). The persistent inflammatory reaction stimulates the formation of granulation tissue; when a fibrous capsule encases the periapical lesion, a periapical granuloma forms (18).

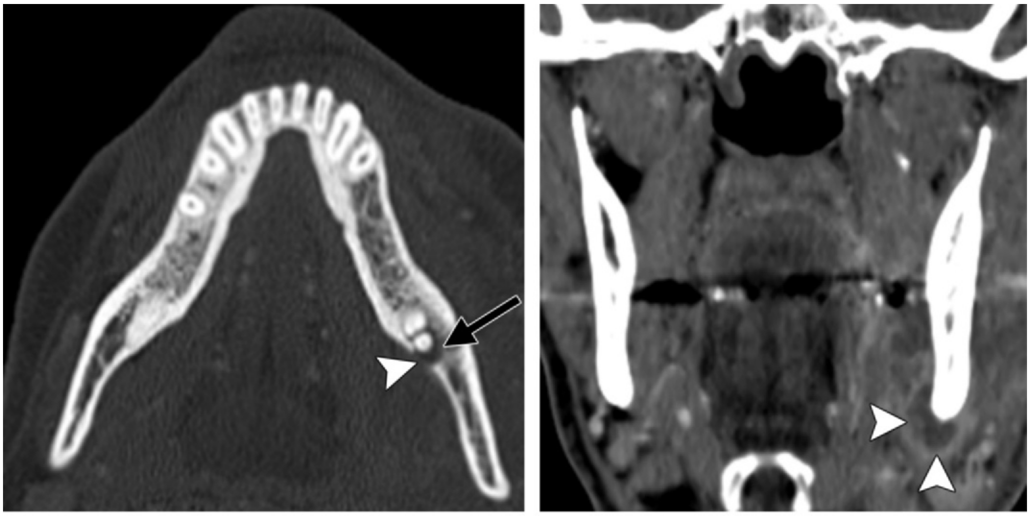
Periapical cysts occur when released cytokines stimulate proliferation of epithelial rests within the periodontal ligament (rests of Malassez), forming an epithelium-lined cavity and resulting in resorption of periapical bone (19). This commonly occurs in men 30–50 years of age (7).

The term *periapical abscess* is applied when a periapical granuloma or cyst becomes reinfected and microbes once again travel from the necrotic pulp and out of the apical foramen.

Prolonged apical periodontal inflammation results in a locally destructive environment caused by localized immune cells, and tooth loss occurs as a consequence. The increase in pressure surrounding the neurovascular bundle near the tooth root may create local ischemia and tooth death (20). Additionally, prolonged inflammation without treatment stimulates the resorption of bone in the affected tooth and results in loosening of the tooth within its socket (18).

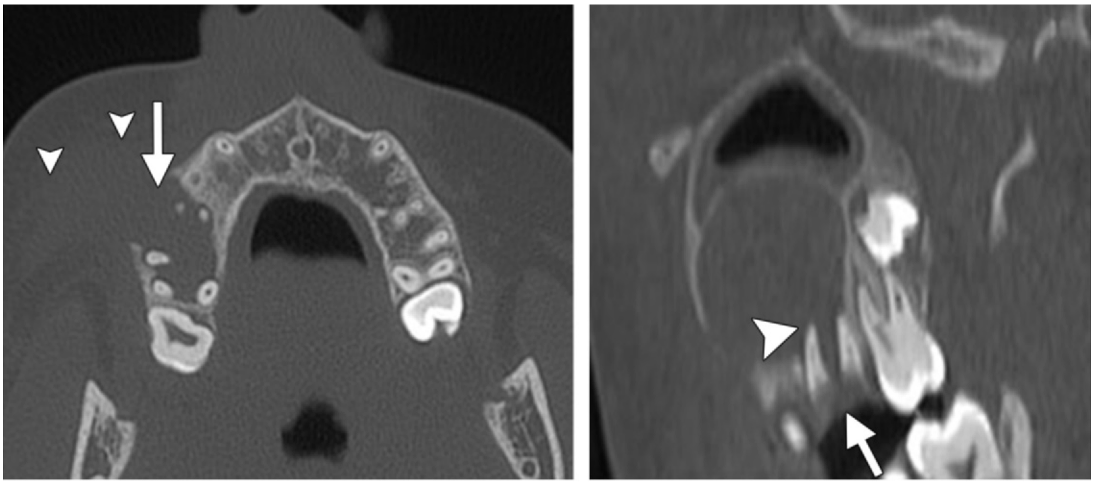
## Periapical Lucency Related to Apical Periodontitis

Periapical granuloma (Fig 3), cyst (Fig 4), and abscess (Fig 5) represent a spectrum of the same pathologic process and are difficult to distinguish from one another radiographically. The shared radiographic features include a lucent halo surrounding the tooth root, often with caries of the affected tooth. Ill-defined borders typically suggest an acute process (phlegmon or abscess), particularly when accompanied by pain (Fig 5), while sharply defined borders are indicative of a chronic process (cyst) (4,18). The presence of inflammatory changes in the bone marrow and adjacent



a. b.

**Figure 5.** Periapical abscess with extraosseous extension in a 44-year-old man with left-sided jaw pain and fever. **(a)** Axial CT image demonstrates a periapical lucency (arrow) involving the root of the left mandibular third molar. Note the associated cortical dehiscence (arrowhead). **(b)** Coronal contrast material–enhanced CT image demonstrates a peripherally enhancing extraosseous abscess (arrowheads) around the mandible.



a. b.

**Figure 6.** Ruptured periapical cyst in a 22-year-old-man who sustained trauma to the face while playing basketball. **(a)** Axial CT image demonstrates a ruptured periapical cyst (arrow) involving the maxilla. Note the expulsion of cyst contents *(*arrowheads*)* into the premaxillary soft tissues. **(b)** Oblique sagittal CT image shows the relationship of the cyst to the tooth root (arrowhead) and associated dental cavity (arrow).

soft tissues strongly suggests periapical abscess, particularly when associated with pain, fever, and swelling (Fig 5) (18,19). Fistulization of abscesses to adjacent soft tissues can occur and result in extraosseous abscess formation (Fig 5).

A periapical granuloma is a relatively small lucent lesion with ill-defined borders (Fig 3), whereas a periapical cyst is typically larger (19,21). A periapical lucency larger than 200 $mm^3$ on radiographs is characterized as a cyst (22,23). Periapical cysts appear as round or pear-shaped and well-defined radiolucent lesions with sclerotic borders around the tooth root, usually less than 1 cm in diameter (24). As the lesions grow in response to pressure, they may displace adjacent tooth roots and cause root and bone resorption (Fig 4). Dystrophic calcifications may be seen in long-standing cysts (24). Occasionally, the cyst will elevate the osseous floor of the sinus and protrude into the maxillary sinus, which is referred to as the “double floor” sign (Fig 4) (23). A periapical cyst may also rupture as the result of trauma (Fig 6).

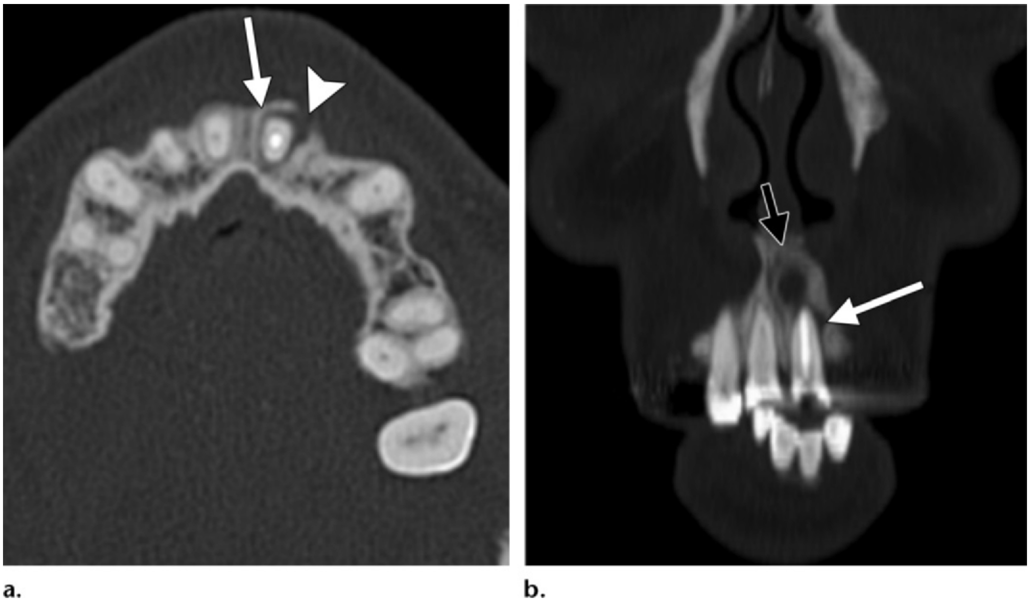


**Figure 7.** Failed root canal with periapical abscess in a 69-year-old woman with lip swelling. **(a)** Axial CT image demonstrates a periapical lucency (arrow) involving the left maxillary central incisor. Cortical breakthrough (arrowhead) occurred and led to recurrent cellulitis of the upper lip. **(b)** Coronal CT image shows the periapical lucency (white arrow) and additional ill-defined lucent lesion (black arrow) consistent with an abscess. Radiopaque material from prior root canal therapy fills the pulp cavity.

## Treatment of Periapical Lucency Related to Apical Periodontitis

Root canals are performed in the setting of periapical lucency due to apical periodontitis and, together with antibiotics, are aimed at eliminating infection. If treatment is successful, bone regeneration and healing of the periapical lesion will ensue and manifest as a gradual reduction in lesion size. Resolution may be seen on follow-up radiographs or CT images. Root canal failure is possible (Fig 7) and may be due to persistent infection; inadequate aseptic control; missed canal; or technical factors, including improper instrumentation, leaking fillings, and poor access to the tooth (25). Causes of persistent infection include inadequate treatment of infection in the apical root canal, extraradicular infection, extruded root canal filling causing foreign body reactions, and cyst formation. Because of surgically thinned bone, cortical breakthrough may occur and result in inflammation of adjacent soft tissues. Treatment of periapical cysts consists of extraction of the affected tooth, endodontic therapy, apical surgery, or marsupialization (3).

## Complications of Apical Periodontitis

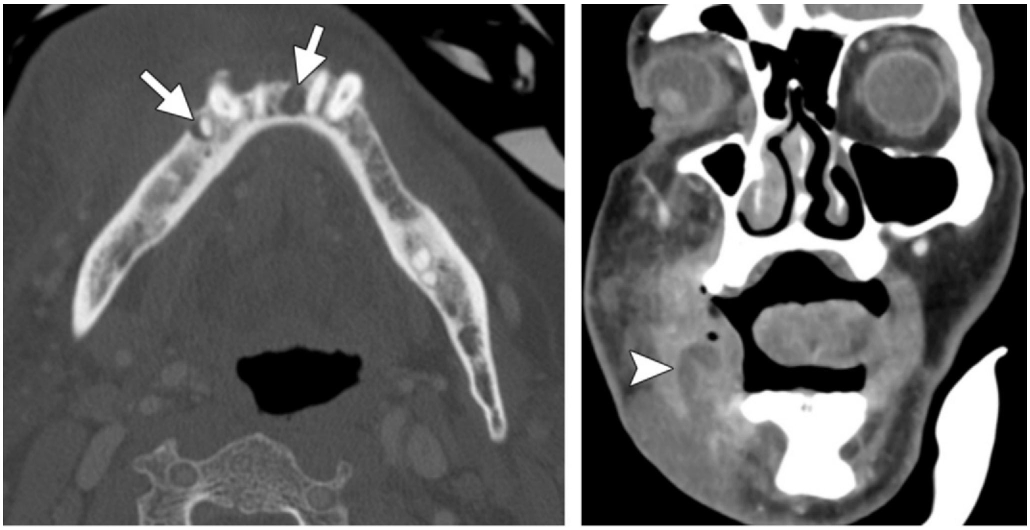
The long-term sequelae of apical periodontal disease depend on the duration of infection before treatment, specific tooth involvement, and the type of disease present.

### Subperiosteal and Extraosseous Abscess Formation

Untreated abscesses eventually compromise the integrity and strength of the subjacent lamina dura and cortical bone. Penetration through the bone will result in fistulization into adjacent soft tissues and may stimulate extraosseous abscess formation. This is characterized by soft-tissue swelling or edema and a peripherally enhancing, abnormal fluid collection, either along the cortex (subperiosteal) (Fig 5) or within adjacent soft tissues (Fig 8). It is important to carefully investigate the tooth roots when soft-tissue swelling is noted around the jaw. Intravenous administration of contrast material is useful for evaluating soft-tissue abscess formation.

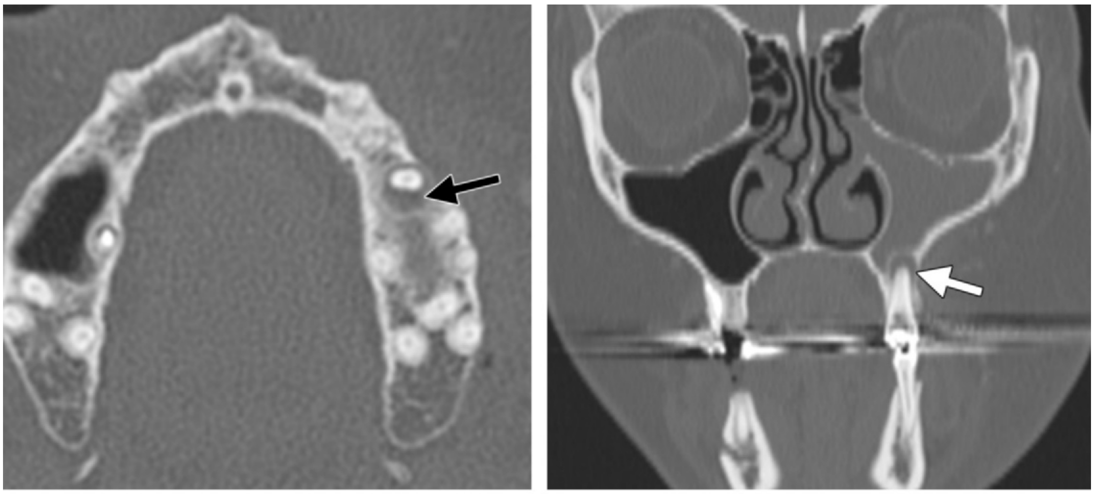
### Sinusitis

Apical periodontal disease of the maxillary teeth can extend into the maxillary sinuses, resulting in sinusitis. Odontogenic disease is thought to be responsible for 10%–12% of maxillary sinusitis cases (20). Patients with known apical periodontal disease have a twofold increased risk for developing maxillary sinusitis (26). Infection from an affected tooth can spread through the apical foramen into the periodontal ligament. Chronic



a. b.

**Figure 8.** Facial cellulitis and abscess of odontogenic origin in a 64-year-old man with unresolved facial swelling. **(a)** Axial CT image demonstrates multiple periapical lucencies (arrows) involving the right central incisor and right first premolar of the mandible. **(b)** Coronal contrast-enhanced CT image demonstrates an associated peripherally enhancing abscess (arrowhead), with diffuse cellulitis of the right side of the face.



a. b.

**Figure 9.** Odontogenic sinusitis in a 52-year-old-man with left-sided facial pain and recurrent episodes of sinusitis. **(a)** Axial CT image shows a periapical lucency (arrow) related to the left maxillary second premolar. **(b)** Coronal CT image shows the periapical lucency (arrow) and total opacification of the left maxillary sinus. Note the well-preserved aeration of other visualized sinuses.

infections and abscesses can result in disruption of the mucoperiosteum (Schneiderian membrane), creating a path of infection into the sinus. The apices of the second maxillary molars are the closest to the inferior maxillary sinus wall, with an average separation of only 1.7 mm (20).

Teaching Point

Identification of an odontogenic source of sinus infection is important because the pathophysiology, microbiology, and management differ from those for sinusitis originating within the sinus itself (20,26). CT findings in odontogenic sinusitis may be identical to those for "typical" sinusitis, but they are more often unilateral. Odontogenic sinusitis may mimic focal mucosal thickening or a mucus retention cyst. The combination of findings related to apical periodontal disease and periapical abscess and the presence of a defect in the sinus floor are highly suspicious for a causal relationship (Fig 9) (20,27,28). Careful evaluation of the ipsilateral maxillary teeth is important, particularly in patients with headache or sinus or facial pain. For evaluation of the integrity of the maxillary teeth, a dedicated sinus CT examination should cover the occlusal plane.

## Fascial Space (Deep Neck) Infections

Most apical periodontal infections result in a localized inflammatory reaction around the tooth root; however, with prolonged infection and inflammation, spreading into the fascial spaces of the neck may ensue (29). The spaces of the deep neck are defined by three layers of deep cervical fascia (superficial, middle, and deep) (30,31). These fascial layers can serve as a barrier to the spread of infection, although direct extension will eventually occur if the infection is left untreated (30).

Deep neck infections are associated with high morbidity and mortality. Until recently, mortality was reported to be as high as 50% (32) when descending infection involves the mediastinum and results in mediastinitis. Currently, mortality is thought to be as high as 25% (33), with the decrease due to earlier detection at cross-sectional imaging and the targeted use of antibiotics (34). A large proportion (47%) of deep neck infections arise from an odontogenic source (30). In the setting of deep neck infections of odontogenic origin, the submandibular space is involved most frequently (81%) (30).

Ludwig angina refers to a rapidly progressive swelling and edema that result in a mass effect on the airway. More than 90% of cases of Ludwig angina have an odontogenic origin (35). At imaging, an inflammatory reaction in the form of fat stranding, soft-tissue swelling, and edema (phlegmon) can be seen early on. In more advanced disease, a discrete mass with fluid or soft-tissue attenuation and peripheral enhancement can indicate an organized abscess (36). Reactive fluid may be seen in the deep neck spaces, including the retropharyngeal space. If clinically indicated, CT examinations of the neck should include the thoracic inlet to evaluate for descending infection along the retropharyngeal space into the chest. If infection is present, extension of the inferior field of view to cover the chest may be necessary.

## Orbital Inflammation

Teaching Point

Orbital inflammation represents a rare but important complication of odontogenic infection. Of utmost importance is preservation of vision, because untreated infection and inflammation may lead to irreversible blindness (37).

The development of orbital inflammation usually manifests via three typical routes of infection spread. Direct extension from underlying maxillary sinus disease (and less commonly from the other paranasal sinuses) is the most common pathway. Infection of the maxillary molars spreads from the tooth through the apical foramen and periodontal ligament, through the bone, and into the sinus. Similarly, orbital infection may arise from the spread of infection through the premaxillary soft tissues. This usually occurs in the setting of infected incisor teeth and is associated with orbitofacial cellulitis. Infection arising from the maxillary molars may travel through the masticator space and into the infratemporal fossa and inferior orbital fissure (38).

Symptoms of orbital inflammation include fever, orbital pain, tenderness, erythema, proptosis, loss of visual acuity, and decreased ocular motility. The presenting symptoms are the same as for orbital cellulitis of non-odontogenic origin; thus, a detailed search for an odontogenic source is necessary (37,38). CT findings include stranding, abnormal enhancement, a fluid collection in the pre- or postseptal orbit or both, edema or bulging of the soft tissues, proptosis, and enlargement of the extraocular muscles (Fig 10). An apical periodontal source can be suspected if inflammation is centered in or can be traced back to a tooth.

Prompt diagnosis of an odontogenic source is essential for proper coverage of microbial flora. If identification of an odontogenic source is not made in a timely manner, poor treatment response and increased risk for long-term sequelae that include loss of vision, either transient or permanent, may result (37).

## Intracranial Complications

Septic thrombosis of the venous sinuses is a rare but serious complication of infectious processes in the face, including orbital cellulitis and sinus disease, which may result from spread of apical periodontal infection. The ophthalmic vein drains into the cavernous sinus; thus, orbital infections may result in thrombosis in these regions. Symptoms of cavernous sinus thrombosis typically include headache in 50% of cases, meningismus in 40% of cases, and abnormal fundoscopic examination results that include papilledema in 60% of cases (39). In the case of cavernous sinus thrombosis, mortality approaches 30% (39). Prompt aggressive antibiotic therapy is the mainstay for thrombosis due to infection.

Imaging features of cavernous sinus thrombosis include expansion of the cavernous sinus due to mass effect from thrombus and dilata-

**Figure 10.** Periorbital cellulitis of odontogenic origin in a 27-year-old woman with right-sided facial swelling. **(a)** Axial contrast-enhanced CT image through the mandible shows a rim-enhancing right sublingual abscess. **(b)** Axial CT image (bone window) shows a periapical lucency surrounding the apex of the right third molar of the mandible, with cortical dehiscence of the lingual cortex (arrow), findings that confirm the odontogenic origin of the abscess. **(c)** Coronal CT image, obtained 1 day after surgical drainage of the abscess when new periorbital swelling was noted, shows development of abnormal circumferential soft-tissue attenuation around the walls of the right orbit (arrow). Note stranding within the retrobulbar fat, consistent with cellulitis and edema. **(d)** Axial CT image (soft-tissue window) from the same study shows stretching of the optic nerve, slight proptosis, and an abnormal contour of the posterior globe margin (arrow), findings that suggest increased intraorbital pressure.

tion of the superior ophthalmic vein. Lack of opacification of the affected venous structures is seen at contrast-enhanced CT. CT is also useful for assessing the cause of the process by careful evaluation of the teeth, sinuses, and orbits and by showing secondary signs of infection and inflammation.

In addition to venous sinus thrombosis, other intracranial sequelae of apical periodontitis may occur, including brain abscess, epidural abscess, subdural empyema, and meningitis (40). Intracranial sequelae are becoming increasingly rare, owing to early imaging coupled with the targeted use of antibiotics (41).

## Osteomyelitis

Tooth infection in the setting of inadequate treatment may result in spread of infection to the adjacent bone (5). The imaging appearance of osteomyelitis depends on the stage of disease. In the early stage of osteomyelitis, imaging is not sensitive, and CT findings are nonspecific. In the subacute to chronic phase, lytic, sclerotic, or mixed lytic and sclerotic lesions, sometimes associated with cortical changes such as thickening or periosteal reaction, may be seen (Fig 11) (42).

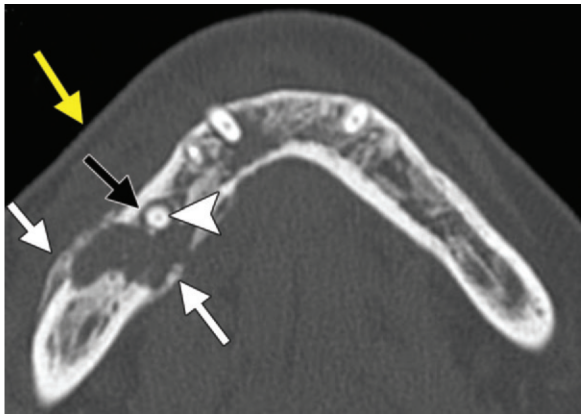


**Figure 11.** Osteomyelitis in a 66-year-old woman with mandibular pain. Axial CT image shows an irregularly marginated lucent lesion (black arrow) adjacent to the root of the right mandibular second premolar (arrowhead). Thinning and breakthrough of the surrounding lingual and buccal cortices and periosteal reaction (white arrows) have occurred. Adjacent soft-tissue stranding and thickening (yellow arrow) and enlarged lymph nodes (not shown) suggest acute infection.

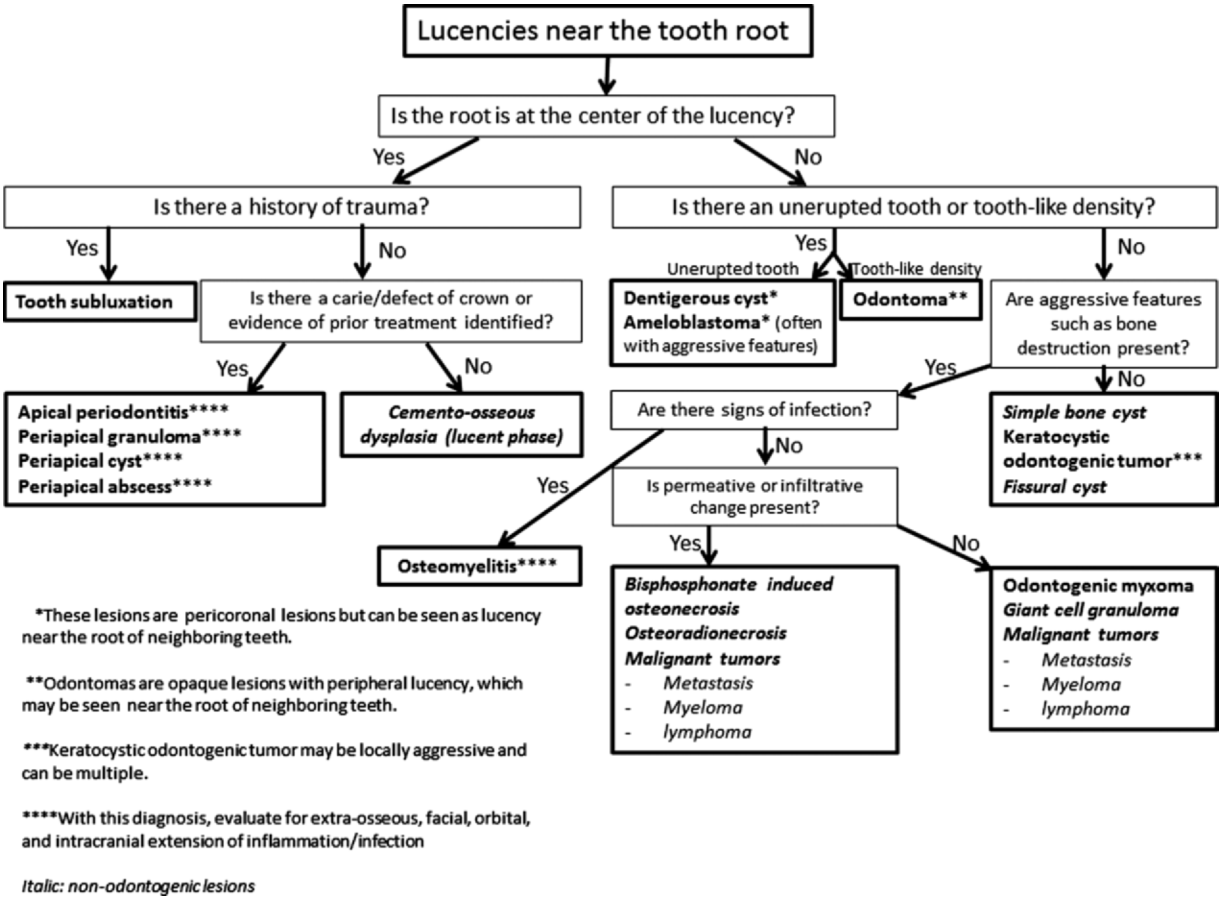



**Figure 12.** Framework for navigation through the differential diagnosis of a periapical lucency.

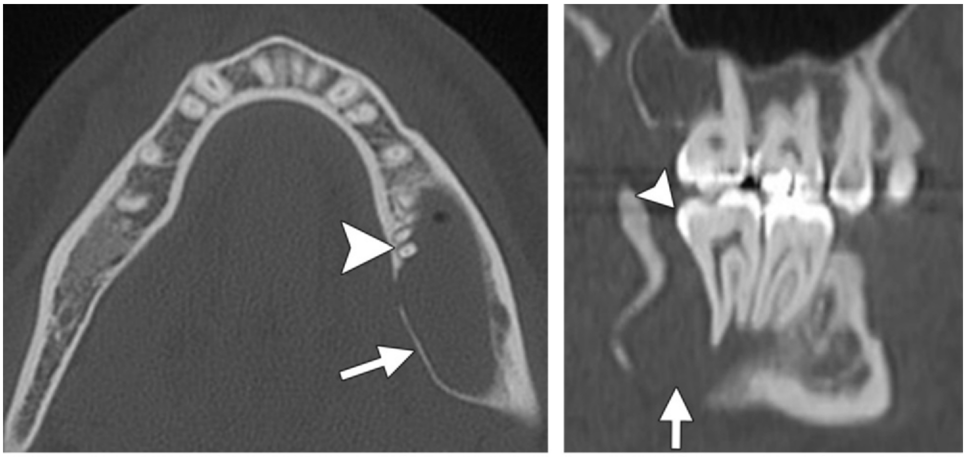
## Differential Considerations

Teaching Point

Although most periapical lucencies seen at radiography and CT are secondary to apical periodontitis, not all lucencies near the tooth root are due to infection. Some may also be secondary to other conditions, including odontogenic and non-odontogenic lesions, both benign and malignant, as well as trauma. A brief description of various representative lesions follows, but an in-depth discussion is beyond the scope of this article; these lesions have been reviewed in recent literature (42,43). Figure 12 provides a framework for navigating the differential considerations for a periapical lucency.

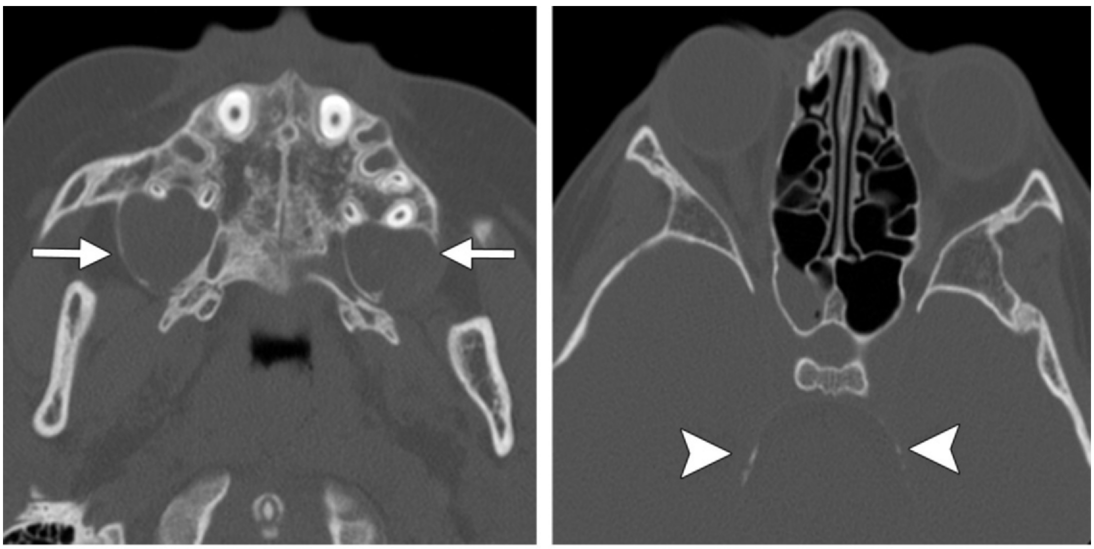
### Odontogenic Lesions

Nonapical periodontitis-related lucencies near the tooth root may arise from odontogenic cysts or tumors, including keratocystic odontogenic tumor (KCOT), dentigerous (follicular) cyst of the adjacent tooth, odontoma, and other odontogenic tumors such as ameloblastoma and odontogenic myxoma (5,42,43).



a. b.

**Figure 13.** KCOT in a 23-year-old man with a history of resection of KCOT, now with a new mandibular cystic lesion. **(a)** Axial CT image shows an expansile cystic lesion (arrow) involving the posterior body and ramus of the left mandible. Focal dehiscence along the buccal surface of the mandible at the level of the lesion is seen. The second molar is displaced anteriorly, and its distal root (arrowhead) is seen at the anteromedial aspect of the cystic lesion. **(b)** Oblique sagittal CT image shows associated focal dehiscence at the inferior margin of the mandible (arrow) and absence of a dental cavity in the second molar (arrowhead).



a. b.

**Figure 14.** Gorlin syndrome in a 7-year-old boy with a history of resection of multiple KCOTs. **(a)** Axial CT image demonstrates expansile cystic lesions (arrows), with cortical thinning and dehiscence of the posterior maxilla bilaterally, consistent with KCOTs. The roots of the molars are displaced and are seen at the anterior aspect of the cystic lesions. **(b)** Axial CT image through the orbits demonstrates dural-based calcification of the tentorium bilaterally (arrowheads), a common associated finding in Gorlin syndrome.

***Keratocystic Odontogenic Tumor.***—KCOTs are cystic lesions most commonly located in the mandibular ramus and body. These are benign lesions that may be associated with an impacted tooth and are most commonly seen in patients 20–40 years of age. KCOTs arise from the dental lamina and may be destructive (44) and expansile (42). They typically demonstrate smooth margins and may scallop the adjacent cortex (Fig 13) (42,43). The presence of multiple lesions and prominent dural calcifications suggest basal cell nevus syndrome (Gorlin syndrome) (Fig 14). The absence of caries or secondary findings of apical periodontal disease of the involved tooth or teeth can help differentiate this lesion from apical periodontitis.

***Dentigerous (Follicular) Cyst.***—Dentigerous cysts are the second most common odontogenic cysts after periapical cysts and are typically seen in individuals in the 3rd to 4th decades of life. In distinction to periapical cysts, dentigerous cysts form around the crown of an unerupted tooth. They may grow to be large and may displace adjacent teeth (43); an area of lucency may be seen near the root of an adjacent tooth (Fig 15). Resorption of the underlying bone or adjacent tooth root is not typically seen (42).

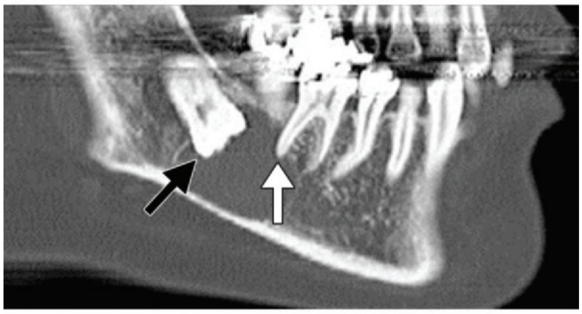
***Odontoma.***—Odontomas are hamartomas of odontogenic origin. They derive from differentiated epithelial and mesenchymal cells that give rise to ameloblasts and odontoblasts and are thus commonly composed of enamel and dentin (45). Compound odontomas consist of multiple small radiopaque structures similar to normal teeth with a thin radiolucent rim (46). Complex odontomas consist of more irregular amorphous radiopaque masses with little or no similarity to normal teeth. They are also surrounded by a thin radiolucent rim (46). The lucent rim may be seen near the adjacent tooth root.

***Ameloblastoma.***—Ameloblastomas represent 10% of odontogenic tumors (43) and occur predominantly in the mandible. They are thought to arise from surface epithelial cells and, although benign, are locally aggressive. The four forms of ameloblastoma are multicystic (solid), unicystic, extraosseous (peripheral), and desmoplastic. The multicystic form is the most common, representing approximately 85% of these lesions (43).

The radiologic features of a typical ameloblastoma include unilocular or multilocular expansile radiolucent lesions often associated with an unerupted tooth. There may be thinning or frank dehiscence of the adjacent cortical surfaces, possibly associated with erosion of the root of the adjacent tooth, reflecting the aggressiveness of the lesion. Variable resorption of the bone cortex and a soft-tissue component may be seen (Fig 16).

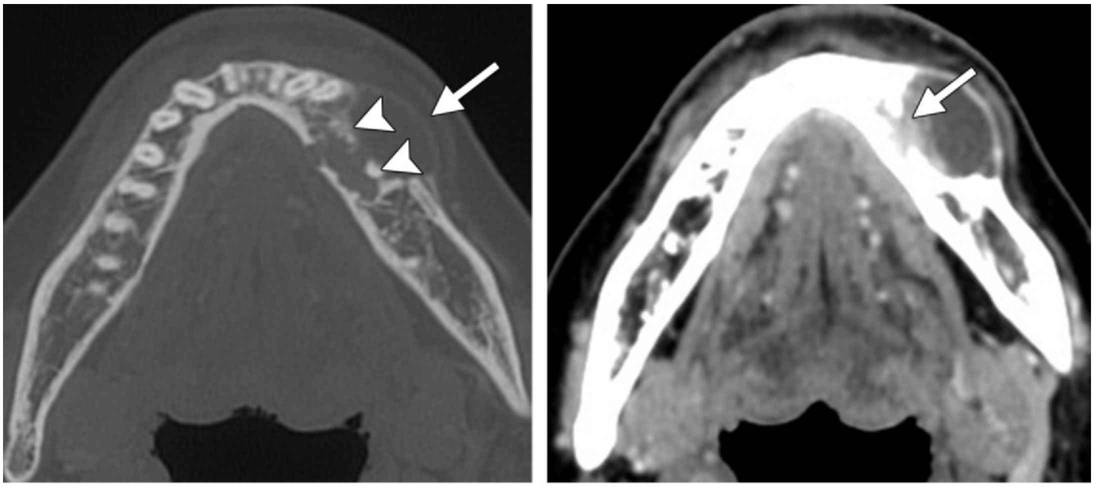
***Odontogenic Myxoma.***—Odontogenic myxomas are rare odontogenic tumors that arise from mesenchymal cells and represent as much as 17% of all odontogenic tumors (47). They are benign; however, they can be locally aggressive, with destruction of the osseous cortex and resorption of the root of the adjacent tooth (Fig 17) (19). Odontogenic myxomas vary in size and may demonstrate either distinct or ill-defined borders. Smaller lesions may be unilocular; larger lesions tend to be multiloculated. They exhibit internal trabeculae with a “honeycomb” or “tennis-racket” internal architecture (19,43,47,48) and may be associated with irregular calcifications.

a.



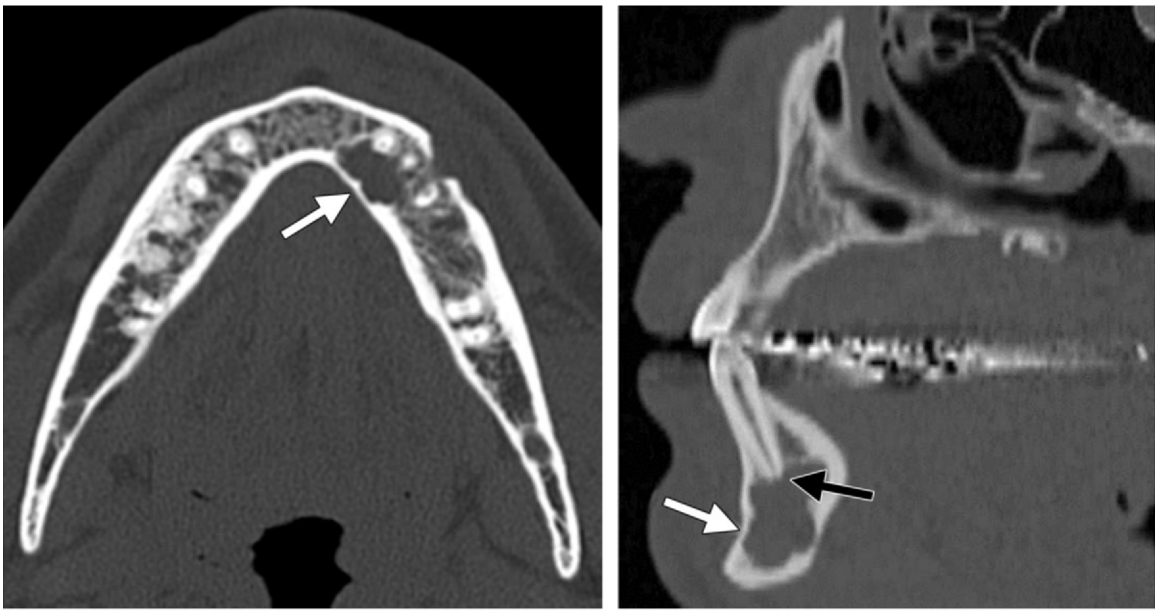
b.

**Figure 15.** Dentigerous cyst in a 54-year-old woman with an incidental finding of a lucent lesion within the mandible. **(a)** Axial CT image demonstrates an expansile lucent lesion (white arrow) within the posterior body of the right mandible. The distal root of the second molar (yellow arrow) is seen at the anteromedial aspect of the lesion. **(b)** Sagittal CT image shows the crown of an impacted third molar (black arrow) projecting into the lucent lesion. The lesion abuts the distal root of the second molar (white arrow), but there is no evidence of tooth displacement or root resorption.



a. b.

**Figure 16.** Ameloblastoma in a 54-year-old woman with left mandibular swelling. **(a)** Axial CT image demonstrates an expansile lucent lesion within the body of the left mandible; the roots of the premolars (arrowheads) are seen within the lesion. Note the expansion of the marrow space and thinning of the buccal cortex of the mandible (arrow). **(b)** Axial contrast-enhanced CT image shows enhancing soft tissue (arrow) within the lesion.



a. b.

**Figure 17.** Odontogenic myxoma in a 49-year-old man with an abnormality seen on a mandibular radiograph. **(a)** Axial CT image demonstrates a lucent lesion (arrow) within the anterior body of the left mandible. **(b)** On the sagittal CT image, root resorption (black arrow) is seen along with cortical thinning (white arrow), findings that indicate a locally aggressive process.

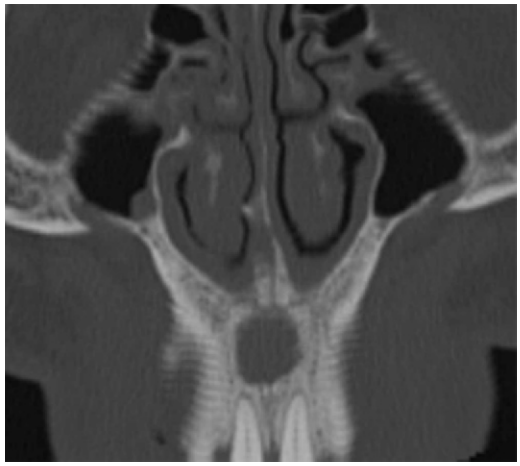
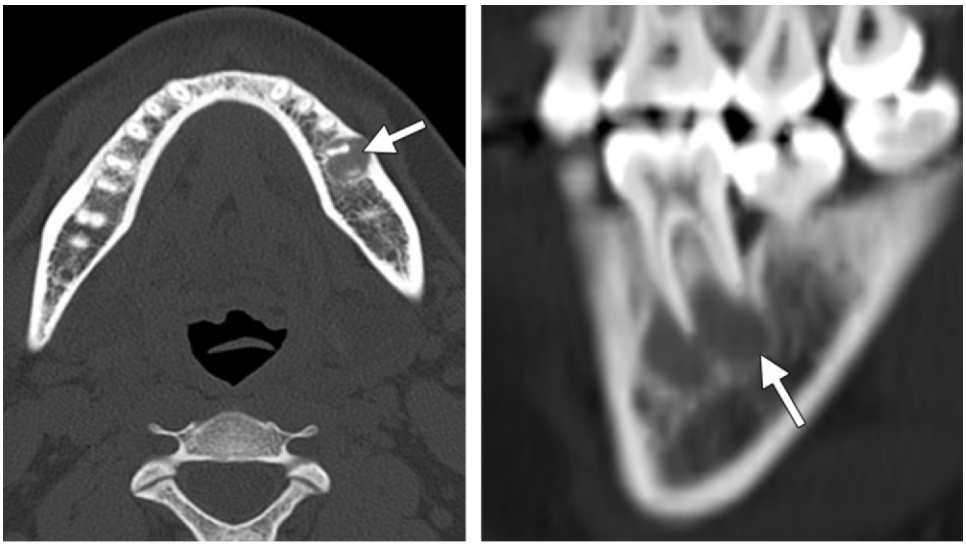


**Figure 18.** Nasopalatine duct cyst in a 53-year-old-man who underwent head CT after being struck by a car. Coronal CT image demonstrates a well-defined midline lucent lesion within the anterior palate. Note that the roots of the central incisors are close to but not involved with the lesion.



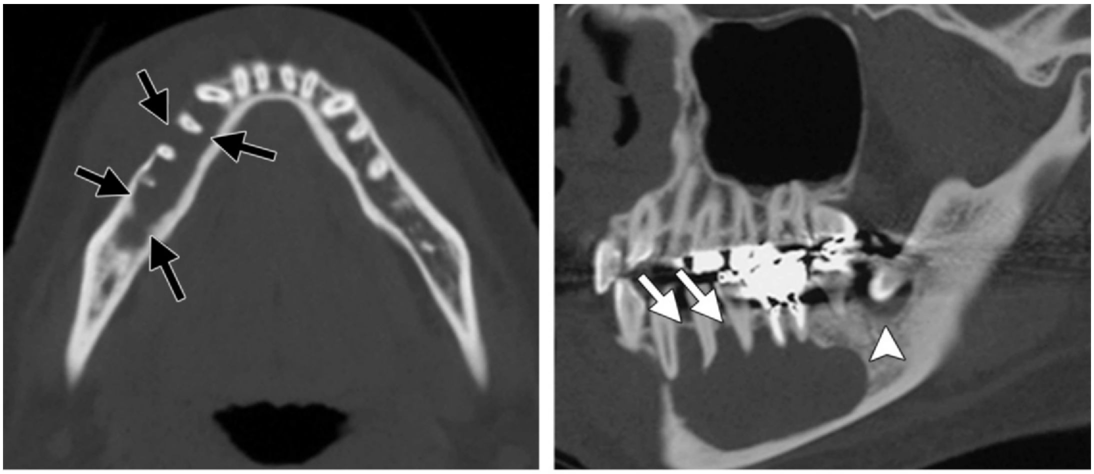
a. b.

**Figure 19.** Cemento-osseous dysplasia in a 31-year-old-woman with abnormal bone scan findings. **(a)** Axial CT image demonstrates a well-defined periapical lucent lesion (arrow) around the roots of the left mandibular first molar. **(b)** Sagittal CT image shows the same lesion (arrow) with a normal appearance of the associated tooth, including absence of dental caries and root resorption.

## Non-odontogenic Lesions

Myriad non-odontogenic lesions, both benign and malignant, may be seen as a lucency around the tooth root that mimics a periapical lucency due to apical periodontal or pulpal disease. These conditions include fissural cysts, cemento-osseous dysplasias, giant cell granulomata, and simple bone cysts.

***Fissural Cyst.***—Fissural cysts are named on the basis of their location; they include nasoalveolar (nasolabial), nasopalatine duct, and median palatal cysts. Nasopalatine duct cysts, also known as incisive canal cysts, are the most common fissural cysts and occur in the nasopalatine duct, which is located in the anterior hard palate at the midline and contains branches of the descending palatine and sphenopalatine arteries, the nasopalatine nerve, and mucus-secreting glands (5,7,23). A



a. b.

**Figure 20.** Simple bone cyst in a 42-year-old man who underwent CT for sinusitis. **(a)** Axial CT image shows an expansile lucent lesion (arrows) in the anterior body of the right mandible, with cortical thinning. **(b)** Oblique sagittal CT image shows scalloped margins of the lesion (arrows) without destruction of the adjacent tooth roots. Note the dentigerous cyst (arrowhead) associated with the third molar.

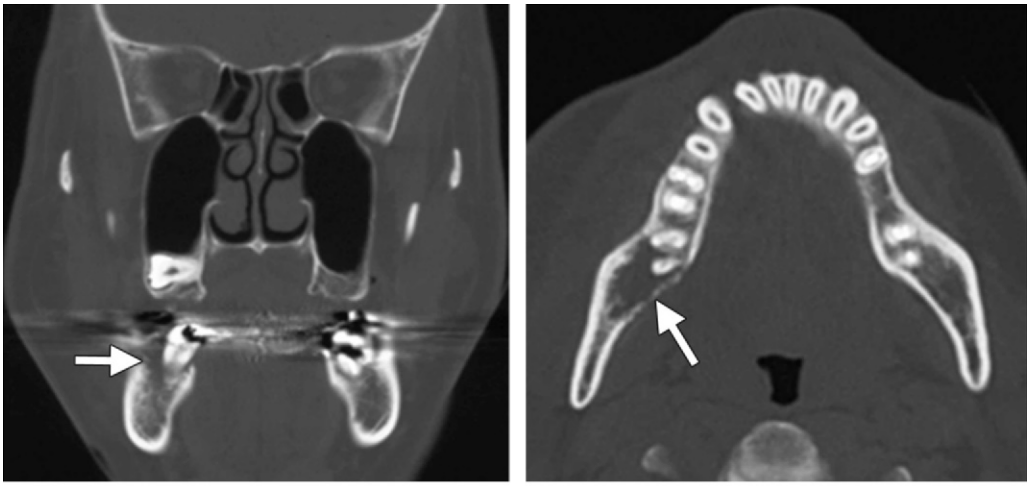
nasopalatine duct cyst may be seen close to the root of the maxillary central incisor; however, the lesion should be separate from the root (Fig 18).

***Cemento-osseous Dysplasia.***—Cemento-osseous dysplasia is due to proliferation of connective tissue within the periodontal membrane. These lesions occur predominantly in women, usually within the 3rd decade of life, with a higher predominance among blacks (64%) (46). Multiple lesions are referred to as "florid cemento-osseous dysplasia." They most frequently arise near the apices of mandibular incisors and are usually asymptomatic. Radiographically, cemento-osseous dysplasia begins as a well-defined radiolucency near the apex of a tooth (Fig 19). The lesion progresses over time to include both osseous and cementum components and is seen as a mixed radiolucent and opaque lesion with well-defined radiolucent rims (43). "Late stage" lesions are diffusely radiopaque, often with ill-defined borders (43,46).

***Simple Bone Cyst.***—The term *simple bone cyst* includes entities such as solitary bone cyst, hemorrhagic cyst, extravasation cyst, unicameral bone cyst, traumatic bone cyst, and idiopathic bone cavity. Despite the name, these lesions lack an epithelial lining and are not true cysts. Although the cause of simple bone cysts is unknown, these lesions are thought to originate from intramedullary hemorrhage caused by trauma. Simple bone cysts are typically unilocular, with well-defined borders that demonstrate a scalloped appearance between the tooth roots (Fig 20) (49,50).

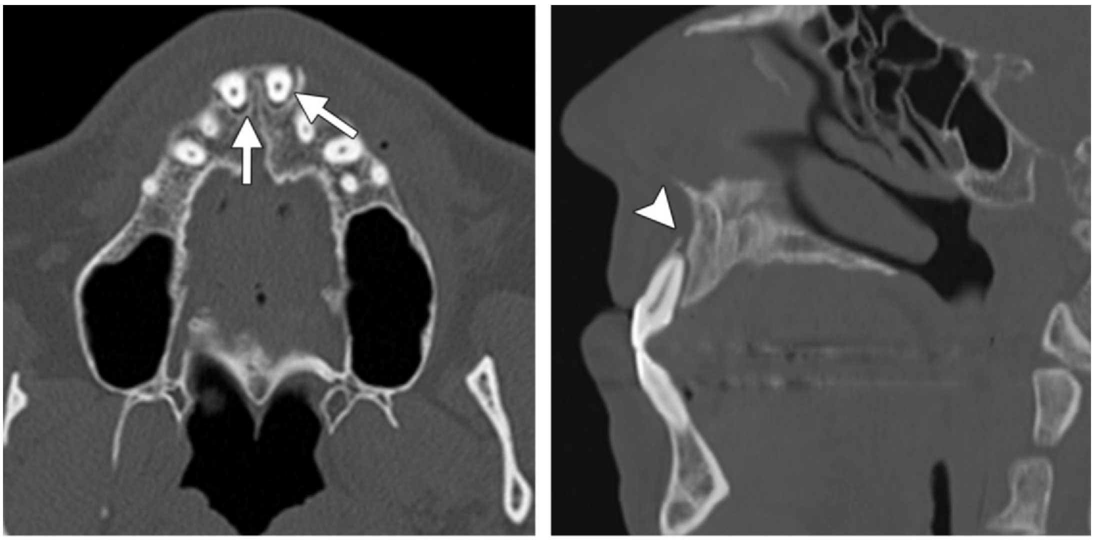
***Giant Cell Granuloma.***—Giant cell granuloma is a lucent lesion that occurs predominantly in younger individuals, usually before age 30 years (13,51). The mandible is the most common site of involvement (19). Small lesions are typically unilocular; as the lesions grow, they may become multiloculated and tend to expand the underlying bone. Lesions may displace teeth and cause resorption of the lamina dura and tooth roots (18). They may be seen as lucent lesions near the tooth root.

***Malignant Tumors.***—The maxilla and mandible are often involved by malignancies arising from adjacent structures such as the gingiva or oral cavity; therefore, squamous cell carcinoma (Fig 21) is the most common malignancy that involves the mandible or maxilla. Although primary malignant tumors are not common in the mandible or maxilla, the mandible is susceptible to hematologic disorders such as multiple myeloma, plasmacytoma, and lymphoma, along with metastatic tumors, because it contains abundant bone marrow space (5,23,42,43).



a. b.

**Figure 21.** Squamous cell carcinoma arising from the periodontal pocket in a 49-year-old man who underwent CT for sinusitis. **(a)** Coronal CT image demonstrates widening of the periodontal space of the right mandibular molar (arrow). **(b)** Axial CT image shows a lucent lesion in the posterior body of the right mandible, with permeative change in the lingual cortex (arrow).



a. b.

**Figure 22.** Tooth subluxation and fracture of the right maxillary alveolar ridge in a 49-year-old man injured in a bicycle accident, with profuse bleeding from the mouth. **(a)** Axial CT image demonstrates widening of the periodontal space of the bilateral central incisors, left more than right (arrows). **(b)** Sagittal CT image shows a fracture of the alveolar ridge (arrowhead) at the left central incisor.

## Trauma

Trauma can cause fracture or subluxation of teeth, as well as fracture of the mandible or maxilla. These conditions can create a gap between the affected tooth and bone and are seen as lucencies around the tooth root (Fig 22); however, the diagnosis is usually readily made if there is a history of recent trauma. It is essential to assess for other findings due to trauma. Periapical cysts may also develop secondary to remote trauma (21).

## Conclusion and Radiologist Guidelines

Periapical lucencies are often seen incidentally at CT examinations of the head and neck performed for nontooth-related indications, both in the outpatient and emergency department settings. Although a large proportion of these lesions are not associated with clinical symptoms, clinically unsuspected apical periodontal disease may be the inciting factor that prompts the imaging study. Untreated apical periodontal disease may result in a wide range of complications, some of which, although rare, are life threatening. Early diagnosis, prompt dental referral, and appropriate treatment are important to decrease morbidity and mortality. Familiarity with the various lucent lesions that occur around the tooth root, which may mimic the common periapical lucencies due to apical periodontal disease, is important to discern more serious conditions and to avoid unnecessary additional imaging.

## References


1. Stockdale CR, Chandler NP. The nature of the periapical lesion: a review of 1108 cases. J Dentistry 1988;16(3):123–129.
2. Ide R, Hoshuyama T, Takahashi K. The effect of periodontal disease on medical and dental costs in a middle-aged Japanese population: a longitudinal worksite study. J Periodontol 2007;78(11):2120–2126.
3. Pihlstrom BL, Michalowicz BS, Johnson NW. Periodontal diseases. Lancet 2005;366(9499):1809–1820.
4. Abrahams JJ. Dental CT imaging: a look at the jaw. Radiology 2001;219(2):334–345.
5. Kaneda T, Weber AL, Scrivani SJ, Bianchi J, Curtin HD. Cysts, tumors and nontumorous lesions of the jaw. In: Som PM, Curtin HD, eds. Head and neck imaging. 5th ed. St Louis, Mo: Mosby Elsevier, 2011;1469–1613.
6. Gahleitner A, Watzek G, Imhof H. Dental CT: imaging technique, anatomy, and pathologic conditions of the jaws. Eur Radiol 2003;13(2):366–376.
7. White SC, Pharoah MJ. Cysts and cystlike lesions of the jaws. In: White SC, Pharoah MJ, eds. Oral radiology: principles and interpretation. 6th ed. St Louis, Mo: Mosby Elsevier, 2009; 343–365.
8. Boeddinghaus R, Whyte A. Dental panoramic tomography: an approach for the general radiologist. Australas Radiol 2006;50(6):526–533.
9. Hedesiu M, Serbanescu A, Ciolan C, Niculae I, Fildan F, Marius M. Interobserver variability of the diagnosis of apical periodontitis on panoramic radiography assessment. Maedica 2007;2(4):289–293.
10. Molander B, Ahlqwist M, Gröndahl HG. Panoramic and restrictive intraoral radiography in comprehensive oral radiographic diagnosis. Eur J Oral Sci 1995;103(4):191–198.
11. Miracle AC, Mukherji SK. Conebeam CT of the head and neck, part 1: physical principles. AJNR Am J Neuroradiol 2009;30(6):1088–1095.
12. Miracle AC, Mukherji SK. Conebeam CT of the head and neck, part 2: clinical applications. AJNR Am J Neuroradiol 2009;30(7):1285–1292.
13. Tyndall DA, Rathore S. Cone-beam CT diagnostic applications: caries, periodontal bone assessment, and endodontic applications. Dent Clin North Am 2008;52(4):825–841.
14. Lofthag-Hansen S, Huumonen S, Gröndahl K, Gröndahl HG. Limited cone-beam CT and intraoral radiography for the diagnosis of periapical pathology. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;103(1):114–119.
15. Ludlow JB, Davies-Ludlow LE, Brooks SL, Howerton WB. Dosimetry of 3 CBCT devices for oral and maxillofacial radiology: CB Mercuray, New-Tom 3G and i-CAT. Dentomaxillofac Radiol 2006; 35(4):219–226.
16. Kidd EA, Fejerskov O. What constitutes dental caries? Histopathology of carious enamel and dentin related to the action of cariogenic biofilms. J Dent Res 2004;83(spec no C):C35–C38.
17. Darling AI. Resistance of the enamel to dnetal caries. J Dent Res 1963;42(1):488–496.
18. Nair PN. Pathogenesis of apical periodontitis and the causes of endodontic failures. Crit Rev Oral Biol Med 2004;15(6):348–381.
19. Scholl RJ, Kellett HM, Neumann DP, Lurie AG. Cysts and cystic lesions of the mandible: clinical and radiologic-histopathologic review. RadioGraphics 1999;19(5):1107–1124.
20. Mehra P, Murad H. Maxillary sinus disease of odontogenic origin. Otolaryngol Clin North Am 2004;37(2):347–364.
21. Akman A, Sakai O. Periapical lucency: underappreciated lesions on CT. Contemp Diagn Radiol 2006; 29(23):1–5.
22. Zain RB, Roswati N, Ismail K. Radiographic evaluation of lesion sizes of histologically diagnosed periapical cysts and granulomas. Ann Dent 1989; 48(2):3–5, 46.
23. Sakai O. Jaw. In: Sakai O, ed. Head and neck imaging cases. New York, NY: McGraw-Hill, 2011; 823–936.
24. Yoshiura K, Weber AL, Runnels S, Scrivani SJ. Cystic lesions of the mandible and maxilla. Neuroimaging Clin N Am 2003;13(3):485–494.
25. Nair PN, Sjögren U, Figdor D, Sundqvist G. Persistent periapical radiolucencies of root-filled human teeth, failed endodontic treatments, and periapical scars. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999;87(5):617–627.
26. Abrahams JJ, Glassberg RM. Dental disease: a frequently unrecognized cause of maxillary sinus abnormalities? AJR Am J Roentgenol 1996;166(5):1219–1223.
27. Rafetto L. Clinical examination of the maxillary sinus. Oral Maxillofac Surg Clin North Am 1999; 11:35–44.
28. Brook I. Sinusitis of odontogenic origin. Otolaryngol Head Neck Surg 2006;135(3):349–355.
29. Moncada R, Warpeha R, Pickleman J, et al. Mediastinitis from odontogenic and deep cervical infection. Anatomic pathways of propagation. Chest 1978;73(4):497–500.
30. Boscolo-Rizzo P, Da Mosto MC. Submandibular space infection: a potentially lethal infection. Int J Infect Dis 2009;13(3):327–333.
31. Mukherji SK, Castillo M. A simplified approach to the spaces of the suprahyoid neck. Radiol Clin North Am 1998;36(5):761–780, v.

32. Estrera AS, Landay MJ, Grisham JM, Sinn DP, Platt MR. Descending necrotizing mediastinitis. Surg Gynecol Obstet 1983;157(6):545–552.
33. Alsoub H, Chacko KC. Descending necrotising mediastinitis. Postgrad Med J 1995;71(832):98–101.
34. Furst IM, Ersil P, Caminiti M. A rare complication of tooth abscess: Ludwig's angina and mediastinitis. J Can Dent Assoc 2001;67(6):324–327.
35. Quinn FB Jr. Ludwig angina. Arch Otolaryngol Head Neck Surg 1999;125(5):599.
36. Freling N, Roele E, Schaefer-Prokop C, Fokkens W. Prediction of deep neck abscesses by contrast-enhanced computerized tomography in 76 clinically suspect consecutive patients. Laryngoscope 2009;119(9):1745–1752.
37. Caruso PA, Watkins LM, Suwansaard P, et al. Odontogenic orbital inflammation: clinical and CT findings—initial observations. Radiology 2006;239(1):187–194.
38. Mehra P, Caiazzo A, Bestgen S. Odontogenic sinusitis causing orbital cellulitis. J Am Dent Assoc 1999;130(7):1086–1092.
39. Mattle HP, Wentz KU, Edelman RR, et al. Cerebral venography with MR. Radiology 1991;178(2):453–458.
40. Dolan RW, Chowdhury K. Diagnosis and treatment of intracranial complications of paranasal sinus infections. J Oral Maxillofac Surg 1995;53(9):1080–1087.
41. Osborn MK, Steinberg JP. Subdural empyema and other suppurative complications of paranasal sinusitis. Lancet Infect Dis 2007;7(1):62–67.
42. Devenney-Cakir B, Subramaniam RM, Reddy SM, Imsande H, Gohel A, Sakai O. Cystic and cystic-appearing lesions of the mandible: review. AJR Am J Roentgenol 2011;196(6 Suppl):WS66–WS77.
43. Dunfee BL, Sakai O, Pistey R, Gohel A. Radiologic and pathologic characteristics of benign and malignant lesions of the mandible. RadioGraphics 2006;26(6):1751–1768.
44. Miles DA, Kaugars GE, Van Dis M, Lovas JG. Oral and maxillofacial radiology. Philadelphia, Pa: Saunders, 1991.
45. de Oliveira BH, Campos V, Marçal S. Compound odontoma—diagnosis and treatment: three case reports. Pediatr Dent 2001;23(2):151–157.
46. Eversole R, Su L, ElMofty S. Benign fibro-osseous lesions of the craniofacial complex. A review. Head Neck Pathol 2008;2(3):177–202.
47. Noffke CE, Raubenheimer EJ, Chabikuli NJ, Bouckaert MM. Odontogenic myxoma: review of the literature and report of 30 cases from South Africa. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;104(1):101–109.
48. Koseki T, Kobayashi K, Hashimoto K, et al. Computed tomography of odontogenic myxoma. Dentomaxillofac Radiol 2003;32(3):160–165.
49. Suomalainen A, Apajalahti S, Kuhlefelt M, Hagström J. Simple bone cyst: a radiological dilemma. Dentomaxillofac Radiol 2009;38(3):174–177.
50. Cortell-Ballester I, Figueiredo R, Berini-Aytés L, Gay-Escoda C. Traumatic bone cyst: a retrospective study of 21 cases. Med Oral Patol Oral Cir Bucal 2009;14(5):E239–E243.
51. Waldron CA, Shafer WG. The central giant cell reparative granuloma of the jaws: an analysis of 38 cases. Am J Clin Pathol 1966;45(4):437–447.

# Periapical Lucency around the Tooth: Radiologic Evaluation and Differential Diagnosis

*Margaret N. Chapman, MD • Rohini N. Nadgir, MD • Andrew S. Akman, MD, MBA • Naoko Saito, MD, PhD • Kotaro Sekiya, DDS • Takashi Kaneda, DDS, PhD • Osamu Sakai, MD, PhD*

**RadioGraphics 2013;** 33:E15–E32 • **Published online** 10.1148/rg.331125172 • **Content Codes:** CT HN MK NR

**Page E17**

Expansion of the periodontal space is indicative of an underlying pathologic process.

**Page E18**

Neutrophils cause localized tissue destruction and recruit osteoclasts and odontoclasts, which cause resorption of bone. At this stage, the periodontal space widens and a periapical lucency develops; these sequelae are radiographically visible.

**Page E21**

Identification of an odontogenic source of sinus infection is important because the pathophysiology, microbiology, and management differ from those for sinusitis originating within the sinus itself. The combination of findings related to apical periodontal disease and periapical abscess and the presence of a defect in the sinus floor are highly suspicious for a causal relationship.

**Page E22**

Orbital inflammation represents a rare but important complication of odontogenic infection. Of utmost importance is preservation of vision, because untreated infection and inflammation may lead to irreversible blindness.

**Page E24**

Although most periapical lucencies seen at radiography and CT are secondary to apical periodontitis, not all lucencies near the tooth root are due to infection. Some may also be secondary to other conditions, including odontogenic and non-odontogenic lesions, both benign and malignant, as well as trauma.