Cysts and Cystic Lesions of the Mandible: Clinical and Radiologic-Histopathologic Review

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Many lesions that occur in the mandible have a cystlike radiographic appearance. These lesions are often difficult to differentiate on the basis of their radiographic features alone. Mandibular lesions may be odontogenic or nonodontogenic. Among odontogenic lesions without mineralization, ameloblastomas, odontogenic keratocysts, and dentigerous cysts can all appear as well-defined, unilocular, well-corticated, lucent lesions that are often associated with the crowns of impacted or unerupted teeth. Most radicular cysts appear as round or pear-shaped, unilocular, lucent lesions in the periapical region. Among odontogenic lesions with mineralization, complex odontomas contain multiple masses of dental tissue and compound odontomas contain multiple teeth or toothlike structures. Odontogenic myxomas are characterized by lytic osseous changes of varying size, which may be demarcated and expansile or exhibit ill-defined borders. Nonodontogenic lesions that mimic odontogenic lesions include benign fibro-osseous lesions (conventional or juvenile ossifying fibroma, focal or periapical cemento-osseous dysplasia, florid osseous dysplasia), traumatic bone cyst, lingual salivary gland inclusion defect, central giant cell granuloma, brown tumor of hyperparathyroidism, arteriovenous malformation, and mucoepidermoid carcinoma. The clinical and radiographic features of these mandibular lesions help establish a differential diagnosis, although microscopic tissue evaluation is generally necessary to accurately identify the lesion.

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INTRODUCTION
Cystic and cystlike lesions of the mandible are primarily ellipsoid, radiolucent, and clearly demarcated and may be odontogenic or nonodontogenic. Odontogenic cysts and tumors develop during or after the formation of teeth (1). Odontogenic lesions may be grouped into lesions without mineralization and lesions with mineralization. The term mineralization refers to the elaboration of mineralized products by the lesion itself; such products include enamel, dentin, and cementum or cementumlike calcified tissue. Nonmineralized odontogenic lesions fail to demonstrate internal mineralization and are classically described as radiolucent. Such lesions may partially or completely surround a normal mineralized structure such as a tooth. Mineralization produces lesions with varying degrees of opacity. Although these lesions are classically described as radiopaque or of mixed opacity, we prefer the more precise, biologic process-oriented term lesions with mineralization. In addition, some nonodontogenic lesions mimic odontogenic lesions at radiography.

Most odontogenic mandibular lesions are benign, but some may exhibit aggressive and destructive behavior locally (2). In addition, many of these processes are asymptomatic, particularly in their early stages, and are discovered incidentally at routine dental radiography. The most common symptom is pain, which may or may not be accompanied by swelling. Other reported symptoms and signs include paresthesias and tooth displacement or mobility. The presence or absence of signs and symptoms does not always aid in differentiating benign from malignant processes (1).

It is often difficult to distinguish cystic-appearing mandibular lesions from one another with radiography. In this article, the clinical and radiographic appearances of odontogenic lesions without mineralization, odontogenic lesions with mineralization, and nonodontogenic lesions that mimic odontogenic lesions are presented. Careful consideration of the patient history and the location of the lesion within the mandible, its borders, its internal architecture, and its effects on adjacent structures generally makes it possible to narrow the differential diagnosis.

ODONTOGENIC LESIONS WITHOUT MINERALIZATION

Ameloblastoma
Ameloblastomas are benign epithelial neoplasms and represent about 10% of odontogenic tumors. These neoplasms develop from various sources of odontogenic epithelium, including dental follicular lining epithelium, and exhibit locally aggressive behavior (1). Ameloblastomas typically manifest in the 3rd to 5th decades of life but have also been reported in younger or older individuals. Patients usually present with a slow-growing, painless mass. Most ameloblastomas occur in the ramus and posterior body of the mandible (80% of cases) (2). Large tumors may infiltrate adjacent soft tissues, usually secondary to perforation of the lingual cortex.

Ameloblastomas can vary in their radiographic appearance. Some appear as well-defined, unilocular, well-corticated, lucent lesions (Fig 1) that are often associated with the crowns of impacted or unerupted teeth; as a result, such ameloblastomas are indistinguishable from odontogenic keratocysts and dentigerous cysts at radiography. Other ameloblastomas are multilocular with internal septa and a honeycomb or soap bubble appearance and are often similar in appearance to large odontogenic keratocysts (Fig 2). Ameloblastomas are typi-
Figure 1. Ameloblastoma in a 67-year-old man. An abnormality was seen incidentally on a full-mouth radiographic series obtained for routine dental care; additional radiographic views and a computed tomographic (CT) scan were then obtained. (a) Panoramic radiograph shows a well-defined, noncorticated, lucent lesion between the roots of a canine and the first premolar (arrows). There is loss of the lamina dura with some minimal tooth displacement. (b) Axial CT scan shows a focal area of decreased attenuation in the right anterior aspect of the mandible with perforation of the buccal plate. Enucleation of the lesion (removal of contents and curettage of margins) was performed. (c) Photomicrograph (hematoxylin-eosin stain) shows islands of odontogenic epithelium in a fibrous connective-tissue stroma. This lesion was small and at an early stage; it is unusual to incidentally discover an ameloblastoma at radiography. The differential diagnosis for this case includes ameloblastoma, traumatic bone cyst, lateral periodontal cyst, radicular cyst, and other odontogenic tumors.
cally expansile with an osseous shell that represents the involved bone (Fig 3). They can perforate the lingual cortex of the mandible and extend into the adjacent soft tissues. CT findings include cystic areas of low attenuation with isoattenuating solid regions. Approximately 50% of ameloblastomas arise from the epithelial lining of dentigerous cysts and are called mural ameloblastomas (Fig 4).

Treatment of ameloblastoma depends on the extent of tumor infiltration through the cyst wall and into surrounding bone. Excision of a relatively contained ameloblastoma could involve localized removal of the lesion with wide margins; if the lesion is highly infiltrative and extensive, en bloc resection would be performed. There is controversy about the prognostic significance of an ameloblastoma with a unilocular (ie, cystic) radiographic appearance versus a multilocular radiographic appearance. The invasiveness of the tumor beyond the bone wall is determined with histopathologic inspection of the specimen. CT or magnetic resonance (MR)
Figure 5. Odontogenic keratocyst in a 13-year-old boy. An abnormality was seen incidentally on conventional radiographs obtained for planning of orthodontic treatment. (a) Lateral oblique radiograph shows an ellipsoid, expansile, multilocular, corticated, lucent lesion occupying the anterior two-thirds of the left ramus with an impacted third molar crown displaced inferiorly within the lesion. The mandibular canal appears to be displaced inferiorly as well. (b) Posteroanterior radiograph shows buccal (lateral) displacement of the third molar by the lesion. The patient underwent en bloc resection. The differential diagnosis includes dentigerous cyst, odontogenic keratocyst, and ameloblastoma.

Odontogenic keratocysts are believed to arise from the dental lamina and other sources of odontogenic epithelium. They represent 5%–15% of all jaw cysts. Most odontogenic keratocysts are found during the 2nd to 4th decades of life, although they can occur at any age. The lumen of the cyst often contains "cheesy" material and has a parakeratinized lining epithelium (1). Daughter cysts and nests of cystic epithelia are found outside the primary lesion; as a result, odontogenic keratocysts have the highest recurrence rate of any odontogenic cyst (50%) when treated conservatively with curettage (3).

At radiography, an odontogenic keratocyst usually appears as a unilocular, lucent lesion with smooth, corticated borders that is often associated with an impacted tooth (Fig 5). Although odontogenic keratocysts are most commonly located in the body and ramus of the mandible, they may also occur in the anterior mandible or anywhere in the maxilla. In some
cases, they arise in the lining of primordial cysts without a tooth (Fig 6). Such lesions are indistinguishable from dentigerous cysts at radiography. Odontogenic keratocysts are more likely to show aggressive growth than other odontogenic cysts and may have undulating borders and a multilocular appearance; these characteristics make odontogenic keratocysts indistinguishable from ameloblastomas (1). Odontogenic keratocysts may cause cortical thinning, tooth displacement, and root resorption (2).

The preferred treatment for an odontogenic keratocyst is surgical enucleation with wide bone margins or marsupialization. Most recurrent lesions are found in the first 5 years after enucleation, but recurrence may be delayed for up to 10 years. Recurrent odontogenic keratocysts have been found in bone grafts. Multiple odontogenic keratocysts, along with nevoid basal cell carcinomas and bifid ribs, are components of the basal cell nevus syndrome (Gorlin-Goltz syndrome) (1).

- Dentigerous Cyst
The dentigerous (follicular) cyst is the most common type of noninflammatory odontogenic cyst (4) and the most common cause of a peri-coronal area of lucency associated with an impacted tooth. A dentigerous cyst forms within the lining of the dental follicle when fluid accumulates between the follicular epithelium and the crown of the developing or unerupted tooth. Most dentigerous cysts manifest in adolescents and young adults and often form around the crown of an unerupted mandibular third molar. Patients are typically pain free. The most important features of this cyst are its ability to expand asymptotically and its potential to displace or resorb adjacent teeth or bone.

At radiography, dentigerous cysts appear as well-defined, round or ovoid, corticated, lucent lesions around the crowns of unerupted teeth, usually third molars (Fig 7). The roots of the involved tooth are often outside the lesion and in mandibular bone (1). Dentigerous cysts can vary in size; cysts 2 cm in diameter or larger may cause mandibular expansion (2). The radiographic appearance of such dentigerous cysts is comparable with that of cystic, unilocular odontogenic keratocysts. Extremely large dentigerous cysts often develop undulating borders due to uneven rates of expansion through areas of varying bone density; the resulting radiographic appearance is comparable with that of a larger odontogenic keratocyst or ameloblastoma. In rare instances, an untreated cyst may develop an ameloblastoma within its lining (ie, a mural ameloblastoma [Fig 4]); however, malignant transformation is exceedingly rare (1).
Treatment includes extraction of the associated tooth and removal of the entire cyst. Removal of extremely large cysts may require stabilization of the bone with metal plates and bone grafting into the surgical site.

- **Radicular Cyst**

The radicular (periapical) cyst is the most common cyst of the jaw (1). Radicular cysts are most often seen in patients between 30 and 50 years old (2) and usually do not cause pain. A radicular cyst is the last step in a progression of inflammatory events following the formation of a periapical inflammatory lesion secondary to pulpal necrosis in a tooth. Over time, an inflammatory cyst can develop in the bone at the root apex of a carious tooth due to inflammatory stimulation and proliferation of the epithelial rests of Malassez (residual epithelial cells in the periodontal ligament).

At radiography, most radicular cysts appear as round or pear-shaped, unilocular, lucent lesions in the periapical region (1). They are usually less than 1 cm in diameter and are bordered by a thin rim of cortical bone (2). The associated tooth usually has a deep restoration or large carious lesion (Fig 8). The cyst may displace adjacent teeth or cause mild root resorption.
Radicular cysts and periapical granulomas have a similar radiographic appearance, although radicular cysts are less common and often larger.

Treatment consists of enucleation of the cyst lining coupled with endodontic obturation (root canal) and, in some instances, retrograde surgical endodontic treatment (surgical exposure of the root apex followed by amputation of the root apex and obturation of the opening of the pulp canal) (1). Extraction of the tooth or root fragment may be required.

**ODONTOGENIC LESIONS WITH MINERALIZATION**

- **Odontoma**
  An odontoma is an odontogenic hamartomatous malformation, often referred to as a tumor, that is composed of any or all odontogenic tissues in various states of morpho- and histodifferentiation (1). It is the most common odontogenic “tumor” (67% of cases) (5). There are two types of odontomas: complex and compound. Complex odontomas contain multiple masses of dental tissue and are seen as well-defined lesions with amorphous calcifications on radiographs (2) (Fig 9). The more common compound odontoma contains multiple teeth or toothlike structures. Odontomas are typically discovered in the 2nd decade of life, often during the search for a nonerupted permanent tooth. Most are 1-3 cm in diameter and can cause impaction, malpositioning, or resorption of adjacent teeth. Odontomas may be surrounded by a lucent follicle, which can become cystic (Fig 10) or rarely neoplastic. The follicle is in turn surrounded by a follicular cortex of uniform density and width. The differential diagnosis of more amorphous odontomas includes focal cemento-osseous dysplasia, ameloblastic fibro-odontoma, and adenomatoid odontogenic tumor.

  Simple surgical excision is generally the treatment of choice. Odontomas do not tend to recur.

- **Odontogenic Myxoma**
  An uncommon benign neoplasm (3%–6% of odontogenic tumors), odontogenic myxoma originates from mesenchymal odontogenic tissue. This tumor can be locally aggressive and cause considerable destruction of adjacent bone and soft-tissue infiltration (1). Odontogenic myxomas develop only in the bones of the jaws (2) and have a slight predilection for the maxilla. They are typically found in 10–30-year-old patients, slightly more often in female patients (1). Congenitally missing or unerupted teeth may be associated with this neoplasm. Odontogenic myxomas are usually painless.

  At radiography, odontogenic myxomas are characterized by lytic osseous changes of varying size, which may be demarcated and expansile or...
Figure 10. Cystic odontoma in a 17-year-old boy with painful third molars. An abnormality was discovered incidentally on a panoramic radiograph obtained for planning of tooth extraction; a CT scan was then obtained. (a) Panoramic radiograph shows a large, focal area of heterogeneous calcification with displaced second and third molars. The opaque portions are surrounded by a lucent follicular space (arrowhead). There is marked cystic expansion of all bone margins (arrows). (b) Coronal CT scan shows a heterogeneously calcified mass with expansion and cyst formation superiorly. A toothlike structure is seen internally (arrow). (c) Axial CT scan also shows the lesion. (d) Photomicrograph (hematoxylin-eosin stain) shows cyst formation in bone (B) with an epithelial lining (arrow) and an internal mass of mature dental hard tissue (D). The patient underwent enucleation and curettage. The differential diagnosis is limited and includes cystic odontoma and ameloblastic fibro-odontoma. An odontogenic keratocyst or mural ameloblastoma could develop in the cystic portion of this lesion.
exhibit ill-defined borders. They are often multilocular with internal osseous trabeculae (2) and a honeycomblike internal structure. Foci of irregular calcification are frequently seen (Fig 11). Smaller lesions may be unilocular. Occasionally, myxomas scallop between the roots of adjacent teeth, although teeth may be displaced and roots may be resorbed. The differential diagnosis may be extensive and can include malignancy, traumatic bone cyst, central giant cell granuloma, and other odontogenic tumors. Calcifying epithelial odontogenic tumor, another locally aggressive, expansile tumor that may be heavily calcified, can strongly resemble an odontogenic myxoma at imaging. Treatment is surgical resection with a wide margin of bone because of the tendency of the tumor to infiltrate and recur (1).

**NONODONTOGENIC LESIONS THAT MIMIC ODONTOGENIC LESIONS**

- **Benign Fibro-osseous Lesions**

  Benign fibro-osseous lesions occur commonly in the jaw. They represent a group of diverse diseases characterized by similar histopathologic features (6). Ossifying fibromas are typically encapsulated, circumscribed, benign neoplasms made up of highly cellular fibrous connective tissue that contains varying amounts of osteoid, bone, cementum, and cementumlike calcified tissue. Most ossifying fibromas of the craniofacial skeleton grow slowly and symmetrically and can result in bone expansion with facial asymmetry. In the craniofacial region, there are two apparent clinicopathologic variants of ossifying fibroma: the conventional slow-growing ossifying fibroma and the so-called juvenile active (aggressive) ossifying fibroma. Compared with conventional ossifying fibromas, juvenile ossifying fibromas occur more often in younger patients.
and have a greater tendency to recur; they can be locally destructive, primarily due to a combination of anatomic inaccessibility and incomplete removal. At the microscopic level, juvenile ossifying fibromas also demonstrate encapsulation; however, their stroma tends to be more cellular and vascular than that of conventional ossifying fibromas, and their calcified elements proliferate in what is described as a characteristic garlandlike pattern. Still, juvenile ossifying fibromas are benign neoplasms and do not metastasize. Like most benign tumors, both conventional ossifying fibromas and juvenile ossifying fibromas are asymptomatic but can cause considerable facial asymmetry and tooth displacement. Compared with juvenile ossifying fibromas of the mandible, juvenile ossifying fibromas of the maxilla tend to be more difficult to remove adequately without surgical morbidity and eventual recurrence.

At radiography, either type of ossifying fibroma can appear lucent, opaque, or of mixed opacity depending on the degree of calcification within the mass. Incipient lesions may appear lucent initially (Fig 12); however, if untreated, over time they will usually exhibit increasing opaque foci that appear to coalesce. These neoplasms may be demarcated from their surrounding osseous...
bed by a thin line of lucency, which represents the fibrous capsule; the latter may, in turn, be surrounded by a sclerotic rim of reactive bone elaborated in response to the slowly expansile growth of the central mass. On bone scans, ossifying fibromas classically demonstrate intense focally increased uptake. The differential diagnosis depends on the size, location, and calcification of the lesion. It includes odontoma and sequestrum for smaller lesions and fibrous dysplasia, vascular lesions, and benign tumors for larger lesions, particularly those with minimal or absent mineralization.

Treatment for conventional ossifying fibroma is simple enucleation, and recurrence is uncommon. However, juvenile ossifying fibroma may require surgical excision with an adequate margin of uninvolved bone.

Focal cemento-osseous dysplasia represents one clinicopathologic variant in a spectrum of related and common nonneoplastic benign fibroosseous lesions that arise exclusively in the toothbearing regions of the jaw bones (Fig 13). This subgroup of fibro-osseous diseases also includes periapical cemento-osseous dysplasia (periapical cementoma) (Fig 14) and florid osseous dysplasia (Fig 15). All of these lesions are discovered during adult life, often as incidental radiographic findings without any attendant symptoms or obvious clinical abnormalities. Adjacent teeth are characteristically vital at pulpal testing. Most variants of cemento-osseous dysplasia tend to be localized, limited, discrete lesions with a decided preference for the mandible. However, florid osseous dysplasia, the most dramatic and extensive expression of cemento-osseous dysplasia, manifests as diffuse, multiquadrant distribution of mixed lucent-opaque osseous changes in both the mandible and the maxilla. The potential for symptomatic activation secondary to superimposed osteomyelitis exists.

Except for its signature location in the periapical region of lower anterior teeth, the radiographic features of periapical cemento-osseous dysplasia are virtually identical to those of focal cemento-osseous dysplasia. Both entities are characterized by a relatively distinct demarcation from surrounding normal bone and manifest as one or more closely apposed or confluent, round or ovoid, lucent lesions with varying amounts of opacity. At radiographic follow-up, the lesions begin as cystic areas of lucency and tend to become progressively more opaque internally but usually do not exhibit extension into adjacent bone or cause cortical expansion. The differential diagnosis includes apical periodontitis for small, discrete lesions and is comparable with that of ossifying fibroma for larger lesions.

Instead of “treatment,” appropriate management of focal cemento-osseous dysplasia and periapical cemento-osseous dysplasia involves radiographic follow-up for 18–24 months. Surgi-
cal exploration and biopsy are indicated only if lesions become symptomatic or demonstrate significant clinical or radiographic change (6).

- **Traumatic Bone Cyst**
  A traumatic (simple) bone cyst is not a true cyst because it lacks an epithelial lining. The cause of traumatic bone cyst is unknown, although some believe that it develops in response to trauma. These lesions are usually discovered in the 2nd decade of life (2). Their most common site of occurrence is the mandible (1). Traumatic bone cysts are usually asymptomatic and are incidental radiographic findings.

  The lesions are typically unilocular, lucent defects that often have a characteristic scalloped superior margin extending between the roots of teeth (Fig 16). There may be attendant thinning...
of the mandibular cortex with osseous expansion. Multiple lesions occur in some unusual cases. The differential diagnosis includes vascular lesions, central giant cell granuloma, and ossifying fibroma.

The lesion is explored surgically to rule out more serious conditions and typically consists of an empty cavity in the bone with organizing granulation tissue and hemorrhage. Once curettage has been performed and the diagnosis is established, osseous regeneration ensues and the lesion resolves.

**Lingual Salivary Gland Inclusion Defect**

Lingual salivary gland inclusion defect (Stafne cyst) is a well-defined depression in the lingual surface of the posterior body of the mandible (1), usually near the mandibular angle (2). At surgical exploration, an aberrant lobe of the submandibular gland, or occasionally fat, is found to extend into the depression. The defect can penetrate the mandible to depths extending from the lingual to the buccal cortex. These lesions are typically asymptomatic and are incidental radiographic findings.

On radiographs, the defect appears as an ovoid or rectangular, well-defined area of lucency that classically arises just above the inferior border of the mandible, anterior to the angle...
of the jaw, inferior to the mandibular canal, and posterior to the third molar (Fig 17). The border of the defect is often surrounded by an opaque line because of passage of the x-ray beam through the thick buccolingual wall of the defect. Because this radiographic finding is virtually pathognomonic of a lingual salivary gland inclusion defect, no treatment was given. The differential diagnosis for larger, less characteristic variants includes arteriovenous malformation and hemangioma.

**Central Giant Cell Granuloma**

Central giant cell granuloma is a relatively common lesion of the jaw and typically occurs in adolescents and young adults (75% of patients are less than 30 years old) (7). The mandible is affected twice as frequently as the maxilla with most lesions developing anterior to the first molars, where deciduous teeth are found. Patients typically experience painless swelling (8); palpation may elicit tenderness, and there is frequently expansion of bone and displacement of teeth (1).

The radiographic features of central giant cell granuloma vary. Early lesions are usually small, unilocular areas of lucency that can mimic an odontogenic cyst. As it develops, the lesion may appear multilocular with wispy internal septa and osseous expansion (Fig 18). As a result of slow growth, these lesions tend to be well defined, although poorly defined borders may be seen in lesions that grow more rapidly (9). Central giant cell granuloma has a propensity for crossing the midline, especially in the maxilla. Divergence of tooth roots, resorption of the lamina dura and roots, and more aggressive bone destruction may occur. Brown tumors of hyperparathyroidism appear identical to giant cell granulomas at radiography and are similar histologically, but the age of the patient and the serum parathyroid hormone and calcium levels should help distinguish these entities (1).

Enucleation and curettage is the indicated treatment for central giant cell granuloma, and recurrence is uncommon.

**Brown Tumor of Hyperparathyroidism**

Brown tumors (osteoclastomas) are central giant cell lesions that occur in patients with long-standing hyperparathyroidism. These lesions can arise in any bone, including those of the facial skeleton. Brown tumors can occur in multiple areas within one bone or as a polyostotic process.
At radiography, the lesions have variably well-or ill-defined margins and may cause cortical expansion (Fig 19). Concurrent bone changes associated with hyperparathyroidism, such as generalized demineralization of the medullary bones of the jaw and loss of lamina dura around the roots of teeth, can help differentiate brown tumors from other processes. Serum abnormalities in hyperparathyroidism include hypercalcemia, hypophosphatemia, and elevated levels of parathyroid hormone (10).

After appropriate medical or surgical treatment of the underlying endocrine abnormality, almost all radiographic changes tend to return to normal (1).

- **Arteriovenous Malformation**

  Arteriovenous malformations are abnormal, direct communications between arteries and veins that bypass the capillary bed. They are uncommon lesions in the head and neck. Most lesions in the jaws occur in the ramus and posterior body of the mandible. It is important to recognize the hemorrhagic potential of these lesions because extraction of a tooth adjacent to an arteriovenous malformation may result in lethal exsanguination (11). The clinical features of arteriovenous malformations are variable. The lesion can cause soft-tissue swelling and can expand bone; the swelling is occasionally pulsatile. Aspiration will produce bright red blood.

  On radiographs, arteriovenous malformations may appear cystlike because of resorption in adjacent bone. The lesion may be multilocular, and there may be evident calcification in the wall. The margins of these anomalies may appear erosive and thus simulate a malignant lesion. The differential diagnosis includes traumatic bone cyst, central giant cell granuloma, and ossifying fibroma.

  Angiography is necessary to confirm the diagnosis and determine the size and extent of the lesion (Fig 20). These lesions are managed surgically in a hospital setting with blood transfusion services available (1).

- **Mucoepidermoid Carcinoma**

  Mucoepidermoid carcinomas infrequently arise centrally within the mandible; they usually arise in the salivary glands or sinonasal cavities. On
Figure 20. Arteriovenous malformation in a 23-year-old woman with painless, progressive swelling in the right mandibular angle and submandibular region. The mass was faintly pulsatile. (a) Posteroanterior radiograph shows a laterally expansile, lucent lesion in the right aspect of the mandible with smooth, thin, corticated borders and no internal structure. (b) Lateral oblique radiograph shows the lesion to be at the mandibular angle. The margins within the mandible are poorly defined (arrows), whereas the external margins are well defined (arrowheads). Aspiration produced bright red blood, and angiography was performed. (c) External carotid arteriogram shows a vascular mass supplied by the lingual artery overlying the posterior body and angle of the mandible. The lesion was resected. The differential diagnosis before angiography included arteriovenous malformation, hemangioma, and soft-tissue malignancy invading the mandible.

Radiographs, mucoepidermoid carcinomas in the jaws typically appear more infiltrative than the case shown in Figure 21 (12). These lesions tend to destroy cortical borders such as the walls of the mandibular canals and the cortical covering and ridges of the mandible.
CONCLUSIONS

Cystic-appearing lesions that occur in the mandible are often difficult to distinguish from one another with radiography. They are all usually benign, but some can be locally aggressive and destructive. The patient history and careful consideration of the location of the lesion within the mandible, its borders, its internal architecture, and its effects on adjacent structures generally make it possible to narrow the differential diagnosis. In most cases, these lesions must be surgically removed and examined microscopically to accurately establish the diagnosis.

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