Vol. ■ No. ■ Month 2016

# Expansile radiolucency of the mandible

Victoria L. Woo, DDS,[a] Mary J. McDonald, MD, MPH,[b] and Jeff E. Moxley, DDS[c]

(Oral Surg Oral Med Oral Pathol Oral Radiol 2016;■:1-6)

A 27-year-old female presented for evaluation of an expansile lesion of the left posterior mandible. She reported that the swelling began several months prior and had progressively enlarged, now causing her intermittent pain. On further questioning, she denied paresthesia or significant compromises in function, including disruptions in eating, speaking, or swallowing.

Extraoral examination revealed facial asymmetry attributed to a diffuse swelling of the left mandible. No compressibility or crepitus was noted on external palpation of the affected side, and the overlying skin was normal in appearance, without evidence of erythema. On intraoral examination, it was noted that the buccal and lingual cortices were expanded from tooth #18 to #23. The overlying gingiva and alveolar mucosa were pink, with no appreciable bruits or pulsations on auscultation. Examination of the associated teeth showed grade I mobility of tooth #18 and grades II and III mobility of teeth #24 and #23, respectively. All involved teeth tested vital with cold and electric pulp testing. The patient was edentulous in the regions of teeth #19, #20, #21, and #22 and teeth #28, #29, and #30. She stated that the left mandibular teeth had been extracted to treat an "abscess" 2 months prior. No history was provided regarding the missing right mandibular teeth.

Examination of a panoramic radiograph revealed a large, multilocular radiolucency with well-defined borders extending from the mesial aspect of tooth #18 to the distal aspect of tooth #25 and encroaching on the inferior border of the mandible (Figure 1). The lesion enveloped the roots of teeth #23 and #24 and caused mild displacement of the teeth mesially. Distinct scalloping of the inferior mandibular cortex was seen. Internally, the lesion was predominately radiolucent, with focal evidence of radiopaque trabeculations.

[a]Department of Biomedical Sciences, School of Dental Medicine, University of Nevada, Las Vegas, Las Vegas, NV, USA.
[b]LMC Pathology Services, Las Vegas, NV, USA.
[c]Department of Clinical Sciences, School of Dental Medicine University of Nevada, Las Vegas, NV, USA.

Received for publication Apr 29, 2016; returned for revision Sep 13, 2016; accepted for publication Oct 2, 2016.



2212-4403/$ - see front matter
http://dx.doi.org/10.1016/j.oooo.2016.10.002

## DIFFERENTIAL DIAGNOSIS

The differential diagnosis for an expansile, multilocular radiolucency in a young adult female is broad and encompasses a variety of developmental, neoplastic, inflammatory, and reactive pathoses. Although many of these entities warrant inclusion in the differential diagnosis for the current case, we herein highlight those of foremost diagnostic consideration.

Odontogenic pathology entered prominently in the differential, as many of the odontogenic cysts and tumors bear clinical and radiographic resemblance to this case. Notable among these are odontogenic keratocyst (OKC, keratocystic odontogenic tumor), conventional solid or multicystic ameloblastoma, and odontogenic myxoma. OKCs are developmental lesions that vary from small, asymptomatic lesions to larger, more aggressive swellings that may be associated with significant expansion, cortical destruction, and soft-tissue extension.[1] The posterior mandible is the most frequently involved site, and radiographic examination typically shows a well-defined radiolucency that may be unilocular or multilocular in configuration. Many of these features are also shared by ameloblastoma, odontogenic myxoma, and certain odontogenic cysts such as glandular odontogenic cyst. Features of the present case that may argue against these diagnoses include the presence of significant expansion, which is often not prominent in OKCs; the lack of distinct internal loculations and a "soap-bubble" or "honeycombed" radiographic appearance that is classically described in conventional ameloblastomas; and the absence of perpendicularly oriented internal trabeculations characteristic of odontogenic myxomas.[2] However, these characteristics are neither pathognomonic nor exclusive to these entities. Hence, histopathological examination is ultimately necessary to differentiate between the odontogenic lesions and other intraosseous pathology with a similar presentation.

Vascular anomalies represent a diverse group of lesions that comprise both neoplasms and malformations, according to the International Society for the Study of Vascular Anomalies classification system.[3] Intraosseous vascular anomalies (IVAs) are rare lesions that account for only 0.5%-1% of all bone pathology.[3] The majority of IVAs within the craniofacial region have been shown to be either low-flow venous malformations or high-flow arteriovenous malformations.[3] Gnathic IVAs can vary in presentation from a nonspecific swelling that neither

Fig. 1. The panoramic radiograph shows a well-defined, multilocular radiolucency of the left mandible that is causing mild mesial displacement of left lateral and central incisors. Scalloping resorption of the inferior cortex is seen.

displaces nor resorbs adjacent teeth and structures (venous malformations) to a longstanding mass accompanied by oral mucosal discoloration, tooth mobility, and bruits or pulsations on auscultation (arteriovenous malformations).[3] Radiographic examination will reveal a well-defined unilocular or multilocular radiolucency or, less commonly, a poorly delineated radiolucency associated with cortical breakthrough and periosteal reactivity.[3] The radiographic presentation of an IVA may be similar to the current case, although the presence of mucosal blushing and abnormal sounds on auscultation would be more suggestive of vascular etiology. Advanced imaging and angiographic studies can further aid in establishing a diagnosis of IVA.[3]

Central giant-cell granuloma (CGCG) is an intraosseous lesion of uncertain etiology that may present as a well-defined, often noncorticated unilocular or multilocular radiolucency of the mandible.[4] Lesions have a slight predilection for the anterior jaws and can pursue a relatively indolent clinical course characterized by slow growth with minimal symptoms or, conversely, demonstrate more aggressive behavior such as rapid growth and cortical perforation.[4] CGCGs may arise sporadically or occur in the setting of an underlying genetic disorder or systemic condition.[4] Moreover, coexistence with other intraosseous pathology such as aneurysmal bone cyst and central odontogenic fibroma has been described.[4] While the posterior localization of the present lesion is slightly less in favor of this diagnosis, the clinical and radiographic features of CGCGs are otherwise indistinguishable from the current case.

Simple bone cysts (SBCs) are osteolytic bone lesions most frequently encountered in children and adolescents.[5] The diversity of terms used to describe this entity—including traumatic, hemorrhage, solitary, and unicameral bone cyst—speak to its somewhat enigmatic nature. Extragnathic SBCs have a predilection for the proximal humerus and femur, while jaw lesions favor the posterior mandible and symphyseal region.[4] Gnathic SBCs are typically asymptomatic, with pain and paresthesia reported only in rare cases.[4] Radiographic examination will reveal a well-defined unilocular or, rarely, multilocular radiolucency that classically demonstrates scalloping projections between the roots of the adjacent teeth.[4] The diffuse clinical expansion and multilocular presentation of the present case are not typical of SBC. In addition, the intraoperative finding of scant tissue within an empty cavity or a clear fluid-filled crypt is highly suggestive of SBC[5] and may aid in distinguishing this entity from clinical and radiographic mimics.

Lastly, aneurysmal bone cysts (ABCs) are also osteolytic bone lesions that most often affect pediatric and young adult patients.[5] The clinical presentation of an ABC can vary, although rapid-onset swelling accompanied by pain is common.[5] Radiographic examination will reveal a unilocular or multilocular radiolucency that frequently expands the involved bone, causing bulging and thinning of the adjacent cortices.[5] The intraoperative finding of a hemorrhagic, sometimes briskly bleeding, lesion is characteristic of ABC[6,7] and may be helpful in differentiating this entity from SBC and other intrabony pathology.

## DIAGNOSIS AND MANAGEMENT

The patient was scheduled for intravenous general anesthesia and aspiration, followed by biopsy of the lesion via an open approach. Informed consent was obtained. Aspiration was performed, which yielded 2 mL of serosanguinous fluid, suggesting a nonvascular etiology. Next, a mucoperiosteal flap was elevated from the ascending ramus to the midline. The anterior and lateral walls of the mandible were exposed. The remaining cortical bone in the region of teeth #19 to #22 was thin and easily removed with a curette, revealing a mesh-like network of fibrous soft tissue and blood-filled spaces. The lesion separated easily from the remaining bony walls and was thoroughly excised. The inferior border of the mandible, though resorbed by the lesion, was completely intact after the surgery.

Gross examination of cut sections showed tan-brown soft tissue disrupted by focal calcifications and sharply circumscribed spaces filled with dark coagulated blood. On microscopic examination, the majority of the specimen consisted of moderately cellular fibrous connective tissue composed of uniform ovoid and spindle-shaped cells amongst osteoclast-like multinucleated giant cells and extravasated erythrocytes; the stroma was interrupted by numerous hemorrhagic spaces of varying caliber which were devoid of endothelial or epithelial lining (Figures 2A [eSlide VM02938] and B [eSlide VM02937]). Also observed in some areas were delicate trabeculae of viable