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CASE REPORTS

Osteoblastoma in the Anterior Maxilla Mimicking Periapical Pathosis of Odontogenic Origin

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A patient with a 6-yr history of chronic orofacial pain and periapical pathosis in the anterior maxilla presented for evaluation and treatment. Previous root canal therapy had failed to resolve the persistent pain. Further evaluation suggested a nonodontogenic etiology of the patient's symptoms. Exploratory surgery revealed an osseous cavity across the maxillary anterior palatal midline filled with osteoid and early mineralized bone. The tumor was surgically removed in toto. A diagnosis of benign osteoblastoma was made.

Osteoblastoma is a benign nonodontogenic fibro-oseous lesion of bone that accounts for <1% of all bone tumors (1, 2). Thirty cases of osteoblastoma of the jaws have been documented, 11 of which have occurred in the maxilla (3). The posterior tooth bearing regions are the usual sites of involvement. The midline areas of the jaws are rarely affected (1). Etiology of osteoblastoma is unknown; however, it is considered by most authorities to be a true neoplasm of bone (4, 5).

Osteoblastomas may present with a variety of signs and symptoms. Pain, often quite severe, is the most consistent symptom. The well-circumscribed radiographic features are variable, consisting of combinations of radiolucent and radiopaque patterns. A thin radiolucency may be noted; however, sclerosis of perilesional bone is usually absent. The histological appearance consists of irregular trabeculae of osteoid and immature bone present within a highly vascular connective tissue matrix (1).

Surgical excision of osteoblastoma is the preferred method of treatment. A conservative approach, curettage or local excision, is curative in virtually all cases (1). Recurrence following surgical intervention is rare; only two cases have been documented to show recurrence (6, 7).

This is a study of an unusual case of osteoblastoma in the anterior maxilla. Its radiographic appearance resembled periapical pathosis of odontogenic origin. The tumor, which measured 3 cm in diameter, was surgically removed in toto.

CASE REPORT

A 69-yr-old male presented to the dental school with a 6-yr history of spontaneous, throbbing pain that consistently originated from tooth 7 and radiated to the right temporal and frontal areas, and across the maxillary anterior midline to the canine area. The pain was not relieved by aspirin or acetaminophen. His medical history was significant for gastroesophageal reflux, for which he was taking ranitidine (150 mg twice a day).

Because of the origin of the pain from tooth 7, its sensitivity to percussion, and the presence of a radiolucency, endodontic treatment with intracanal calcium hydroxide was performed by the patient's general dentist, despite a vital pulpal diagnosis. Because the pulp tested vital and a second X-ray could have revealed intact apical periodontal ligaments on all teeth, this treatment probably showed poor judgment. The treatment predictably provided no resolution at all of the patient's symptoms; so, he was referred at once to the division of graduate endodontics for further evaluation and treatment.

Extraoral examination was unremarkable. Intraorally, the buccal soft tissue was normal. However, the palatal mucosa was tender to palpation anteriorly with no expansion of cortical bone. Tooth 7 was sensitive to percussion, whereas teeth 6 and 8 tested vital. Periapical radiographs revealed a 1.2 cm in diameter area of rarefying osteitis superior to the apices of both teeth 7 and 8. No calcified areas within the lesion were evident, and there was no sclerosis of perilesional bone. A vertically angulated periapical radiograph moved the lesion apically on the radiograph, and it did not seem to affect the apices of either tooth 7 or 8, as evidenced by intact apical periodontal membrane spaces (Fig. 1).

At that time, the canal in tooth 7 was cleansed and shaped using 2.5% NaOCl irrigation and obturated with gutta-percha (Kerr Co., Romulus, MI) and sealer (Roth 801, Roth Intl., Chicago, IL) using lateral condensation.

The patient returned 2 wk later for exploratory surgery. The area was anesthetized with 2% xylocaine HCl with 1:50,000 epinephrine (Astra), and a full mucoperiosteal palatal flap was elevated. To aid in visualization, a suture was placed anteriorly to retract the flap. Upon reflection, a loculated osseous cavity filled with a solid material and partly divided by a midline septum was encountered. The diameter of the lesion was 3 cm. After the nasopalatine nerve had been isolated and protected, the cavity was thoroughly curetted

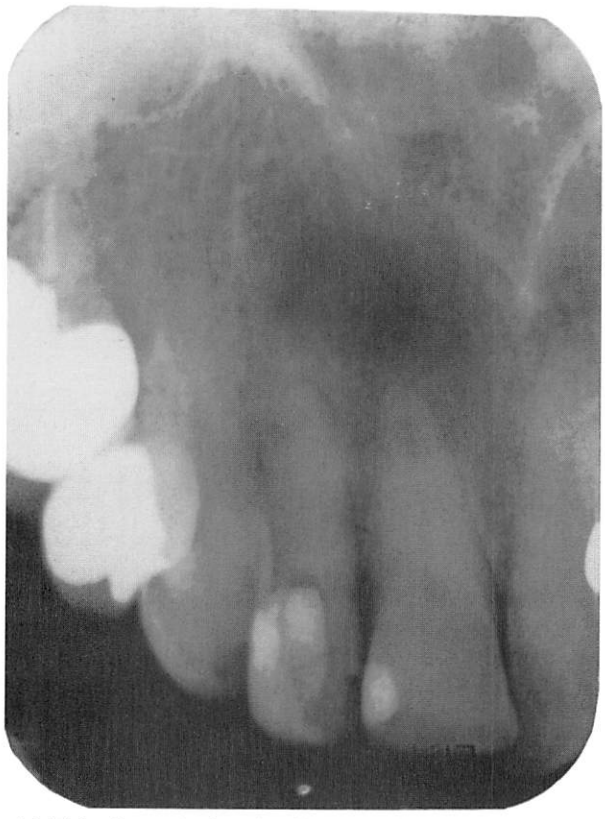

FIG 1. Initial radiograph of tooth 7 (June 1991), status post-root canal therapy and post-calcium hydroxide treatment. This vertically angulated radiograph moved the area of rarefaction away from the apices of the teeth.

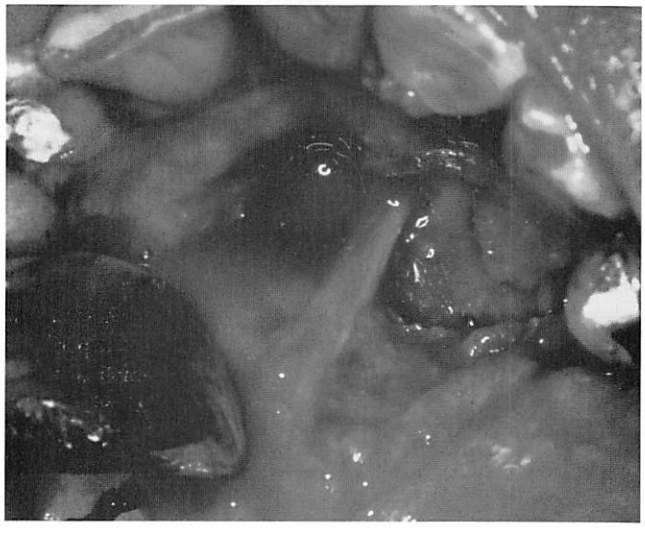

FIG 2. Surgical site, status post-curettage. The lesion is seen to cross the palatal midline.

for biopsy (Fig. 2). The flap was reapproximated, and a palatal stent lined with resilient denture liner (Coe-soft, Coe Laboratories, Chicago, IL) was positioned and secured using 25-gauge orthodontic wires. The patient was placed, prophylactically, on penicillin VK and given a prescription for Tylenol #3 as needed for pain, along with postoperative instructions. The postoperative course was uneventful.

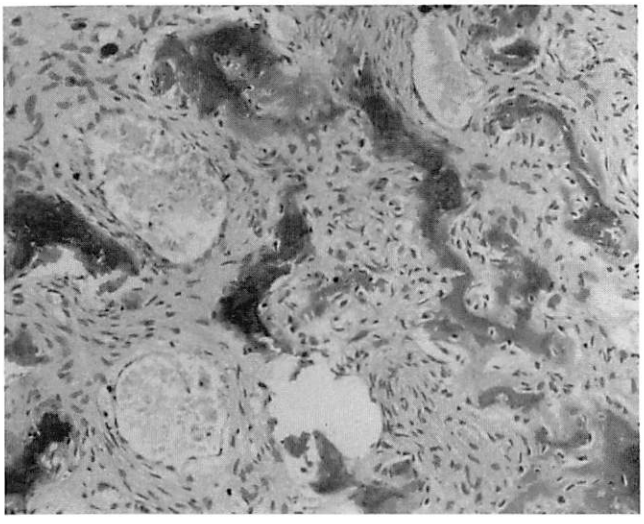

FIG 3. Photomicrograph of biopsy section (hematoxylin-eosin; original magnification ×400).

PATHOLOGICAL EXAMINATION

At gross inspection, the surgical specimen consisted of multiple irregular fragments of soft, rubbery, brown-yellow tissue measuring 1.5 × 0.5 × 0.5 cm in aggregate. The largest portion of the specimen was bisected to reveal a mottled, brown-yellow, tan-cut surface. Microscopically, the specimen consisted of fragments of cellular fibrous connective tissue that contained numerous angular and spherical spicules of osteoid and early mineralized bone. A rich vascular network was evident throughout the proliferating stroma. Multiple hyperchromatic osteoblasts lining the bony trabeculae were also evident. Cytological atypia and abnormal osteoblastic mitotic activity were not observed. A diagnosis of osteoblastoma was made (Fig. 3).

DISCUSSION

This case represents classic benign osteoblastoma of the maxilla. It occurred anteriorly and affected the midline. This presentation is extremely uncommon. In addition, 90% of all documented osteoblastomas have presented before the age of 30 (1); this patient was 69 yr old at the time of diagnosis.

Radiographic interpretation of the lesion was crucial in ruling out periapical pathosis of odontogenic origin. A vertically angulated periapical radiograph moved the radiolucency away from the periapices of both teeth 7 and 8. In addition, this observation indicated that the area of rarefaction was located palatally.

The persistent sensitivity to percussion of tooth 7 complicated the diagnostic process. The most plausible explanation for this finding lies in the fact that in <50% of the lateral incisors, the root apex is closer to the palatal alveolar process when compared with the apices of the central incisor and canine that generally lie closer to the labial aspect of the alveolar process (8). The lateral incisor's root could not be observed during curettage, indicating that the lesion was not intimately associated with the radicular structures of the tooth.

Osteoblastoma must be differentiated from a number of bone-producing lesions, including osteoid osteoma, cementoblastoma, ossifying fibroma, fibrous dysplasia, and osteosarcoma (1). Os-

teoid osteoma bears clinical, radiographic, and histological similarities to osteoblastoma. Classically, the distinction rests primarily in the size of the lesion, with osteoid osteoma being under 2 cm and osteoblastoma being larger than 2 cm. Peripheral sclerotic bone is a significant feature of osteoid osteoma. Cementoblastoma is differentiated from osteoblastoma, because the former lesion arises from the surface of a tooth root and is fused to it, and the latter is normally not. Plump osteoblasts lining bony trabeculae serve to distinguish osteoblastoma from ossifying fibroma. The clinical and radiographic features of these lesions may be similar, although pain is not a usual feature of ossifying fibroma. Fibrous dysplasia exhibits a poorly defined margin in contrast to the well-circumscribed appearance of osteoblastoma. The relatively rapid onset and the pain associated with some osteoblastomas necessitate differentiation from osteosarcoma. The hyperchromatic, large osteoblasts noted in osteoblastoma must be distinguished from the malignant tumor cells of osteosarcoma. Cytological atypia, abnormal mitotic figures, and delicate osteoid adjacent to tumor cells are features of osteosarcoma.

Rarely will osteoblastomas show aggressive behavior or sarcomatous changes (9–11). Malignant transformation of benign osteoblastoma of the jaw bones has never been reported (3); however, such transformation has been documented in extragnathic sites (12–14).

Documented studies indicate that the probability of osteoblastoma in the maxilla is very low. For this reason, during the process of developing a differential diagnosis, which was based not only on the signs and symptoms produced by several suspected pathological entities, but also on the statistical knowledge relative to the incidence of each disease entity as well, osteoblastoma was ranked at the bottom of the list. Nevertheless, based on this study, a differential diagnosis of orofacial pain of nonodontogenic origin should include the possibility of benign osteoblastoma as the source of pain.

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