grade, and the patient was referred for a wider excision. The excision was free of residual tumor and the patient has no signs of recurrence 12 months after surgery.

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CLINICAL PATHOLOGIC CONFERENCE CASE 3: AN EXPANSILE SINONASAL MASS WITH OCCULAR AND NEUROLOGIC SYMPTOMS

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Clinical Presentation: A 19-year-old African American female presented with facial asymmetry and prominent left facial swelling for 2 years. The patient reported loss in her sense of smell and blurred vision as well as 2 episodes of severe headaches shortly before presentation. She also indicated little to no improvement when she used medications for headaches. On physical examination, facial asymmetry with bulging left maxillary bone and left upward proptosis was noted. Extraocular muscle movement and cranial nerves II-XII were grossly intact and symmetrical. Rhinoscopy showed a markedly deviated nasal septum to the right without turbinate hypertrophy. Oropharyngeal examination showed good dentition, no palatopharyngeal mass, and fully mobile and symmetric tongue and palate. Computed tomography (CT) images showed an extradural mass, measuring $81.2 \times 44 \times 39$ mm, involving the left nasal cavity and paranasal sinuses with mass effect on the left orbit and skull base. Magnetic resonance imaging was subsequently performed and revealed a lobulated, well-defined heterogeneously enhanced expansile lesion with calcified matrix and soft tissue component involving the left maxillary sinus with extension into the left nasal cavity, frontal and ethmoid sinuses, and medial wall of the left orbit (Figures 1A and 1B). The patient received debulking of the lesion via endoscopic surgery, and a tissue specimen was submitted for microscopic examination.

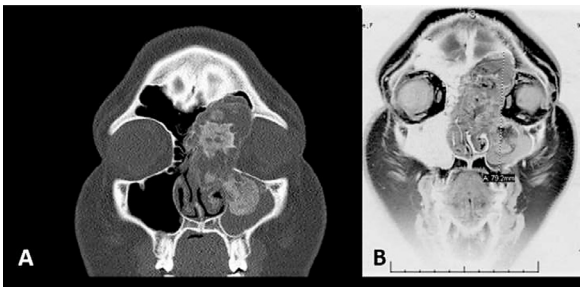

Fig. 1. (A) Coronal computed tomography showing expansile mixed lesion with calcified matrix involving the left side of maxilla, maxillary sinus, nasal cavity, paranasal sinuses and intracranial cavity. (B) Coronal magnetic resonance with enhancement showing heterogeneously enhanced expansile mass.

Differential Diagnosis: Given the clinical presentation above, 4 factors should be considered when determining possible diagnoses. These factors are (1) the young age of the patient, (2) the sinonasal location, (3) the extensive involvement of the tumor, and (4) the presence of calcifications within the soft tissue mass. Taking these into account, the differential diagnosis includes rhabdomyosarcoma, Ewing sarcoma, NUT midline carcinoma, osteosarcoma, and chondrosarcoma.

Rhabdomyosarcoma, a malignancy of skeletal muscle, is the most common malignancy of the nasal cavity and paranasal sinuses in children. Most cases are diagnosed before the age of 12 years. It can present with nasal obstruction, rhinorrhea, cheek numbness, headache, and otitis media.[1] These tumors cause bone destruction and extension into surrounding spaces. CT imaging shows an ill-defined soft tissue mass without hemorrhage or calcification.[2]

Ewing sarcoma typically arises in long bones. Cases in the head and neck most commonly occur in the mandible and maxilla, but sinonasal cases have been reported. Patients diagnosed with head and neck Ewing sarcoma are usually younger than 20 years old. Sinonasal Ewing sarcoma presents with nasal obstruction, epistaxis, proptosis, and cheek swelling.[3] Imaging typically shows a soft tissue mass causing erosion or remodeling of bone, extension into adjacent structures, and occasional calcifications.[4]

NUT midline carcinoma is a rare, aggressive form of squamous cell carcinoma that has a predilection for midline structures of the upper aerodigestive tract and mediastinum in patients with a broad age range. In the head and neck, nasopharyngeal involvement is most common. NUT midline carcinoma presents with nasal congestion, rhinorrhea, sinus pain, epistaxis, and facial numbness. CT images show a soft tissue tumor causing expansion or destruction of bone, opacification of paranasal sinuses, and intralesional calcifications.[5]

Osteosarcoma, a malignant neoplasm of bone, most commonly arises in long bones. Craniofacial osteosarcomas most commonly occur in the jaws in patients younger than 40 years old. Rare paranasal osteosarcomas tend to occur in patients younger than 30. These tumors present with pain, epistaxis, paresthesia, swelling, lacrimation, nasal obstruction, and displacement of the eye. Imaging shows an aggressive tumor with bone destruction and tumoral calcification, sometimes in a sunburst pattern.[6]

Chondrosarcoma is a malignant tumor of cartilage origin that most commonly presents in patients between 40 and 50 years old. Ten percent of all chondrosarcomas arise in the head and neck, of which half occur in the sinonasal tract. They present with nasal obstruction, nasal discharge, epistaxis, headache, pain, visual impairment, diplopia, and proptosis. CT imaging shows a partially calcified tumor causing bone destruction.[7,8]

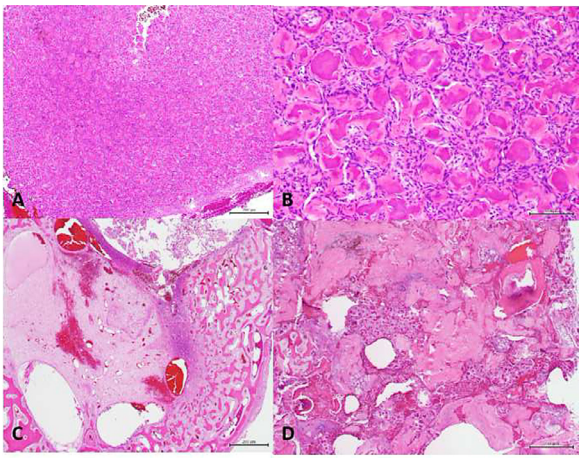

Fig. 2. (A and B) Tissue sections showing cellular fibroblastic tissue proliferation supporting numerous closely packed psammomatous calcifications (hematoxylin and eosin stain, original magnification × 40 and × 200, respectively). (C and D) Tissue sections showing multiple variably sized cystic spaces containing amorphous fluid without peripheral giant cells and focal necrotic zones intermixed with hemorrhage and hemosiderin pigment deposits (hematoxylin and eosin stain, original magnification × 40 and × 100, respectively).

Mesenchymal chondrosarcoma is a subtype of chondrosarcoma that has a predilection for the ribs and facial bones. It is most commonly diagnosed during the second decade of life. The presenting symptoms are the same as conventional chondrosarcoma. Imaging shows a partially calcified, invasive tumor that erodes adjacent bone. It may appear to be encapsulated but does extend into adjacent soft tissue.[9]

Diagnosis and Management: Because of the progression of the patient's symptoms and extensive nature of the tumor on imaging, the patient received left medial maxillectomy, left total ethmoidectomy, left sphenoidotomy, left frontal sinusotomy, left orbital decompression, and resection of the left extradural anterior skull base. Intraoperatively, the entire left maxillary sinus was blocked by tumor, which caused right-sided septal deviation. The medial and inferior walls of the orbit and the anterior portion of the lamina papyracea were involved with tumor and carefully debulked. Exposure of the orbital fat through the floor of the orbit allowed for ocular pressure release. The Neurosurgery team then assisted in the removal of the tumor at the anterior skull base and cribriform plate. Finally, the tumor was removed from the frontal recess and frontal sinus anteriorly. No cerebral spinal fluid leak was observed at the conclusion of the procedure.

Microscopic examination revealed sections consisting of sinonasal mucosa infiltrated in areas by a lobulated benign fibro-osseous proliferation. The tumor was composed of numerous closely packed, round uniform basophilic psammoma bodies with eosinophilic rings interspersed by cellular fibroblastic proliferation (Figures 2A and 2B). The intervening cellular proliferation is composed of spindle-shaped fibroblasts exhibiting uniform elongated, deeply basophilic nuclei without atypia or

mitosis. In some areas, myxoid zones and variably sized cystic spaces containing amorphous fluid, without peripheral giant cells, were noted (Figure 2C). Foci of pale eosinophilic necrosis intermixed with blood and hemosiderin pigment were observed (Figure 2D). The final diagnosis was juvenile aggressive ossifying fibroma, psammomatoid variant with cystic changes and foci of necrosis.

Discussion: Ossifying fibromas are benign neoplasms further subdivided into cementifying-ossifying fibroma (COF) and juvenile aggressive ossifying fibroma (JAOF) dependent on the presence of specific histologic features. Unfortunately, COF, JAOF, and other fibro-osseous lesions such as fibrous dysplasia may show significant histological overlap particularly on small biopsies. The clinical and surgical management of these entities are markedly different; hence, it is imperative that these lesions are correctly diagnosed.

JAOF is a rare benign but potentially aggressive tumor, seen considerably less frequently than its conventional COF counterpart. It exhibits distinct clinical, radiological, and histological features, which allow separation from the conventional COF of the jaws in majority of cases.[10-12] Molecular findings have been recently identified that characterize JAOF.[13-15] Of note, multiple fibro-osseous tumors are associated with hyperparathyroidism-jaw tumor syndrome, a hereditary disorder presenting with hyperparathyroidism by way of parathyroid adenoma(s). Identification of this lesion is important, because hyperparathyroidism-jaw tumor is associated with development of other epithelial tumors, including pancreatic adenocarcinoma, papillary renal cell carcinoma, and testicular mixed germ cell tumor.[16]

JAOF usually demonstrates a rapid growth involving the craniofacial and orbital bones in 85% of patients with a minority of cases affecting the calvarial bones.[11,12,17-19] JAOF generally has equal gender distribution but slight male predilection is reported.[20-24] El-Mofty reported a detailed description of 2 pathological variants, trabecular and psammomatoid, and indicated that their classification as 2 distinct clinicopathologic entities is warranted.[12] The trabecular variant of JAOF frequently involves the mandible and occurs most commonly in children younger than 15 years of age.[20-26] The psammomatoid variant of JAOF most frequently involves the paranasal sinuses with the ethmoid, frontal, maxillary, sphenoid, and temporal bones being affected and usually is seen in people around 20 years of age.[11,12,15,18] Overall, cases of JAOF have been reported in patients from 3 months old to 72 years old with the average ranging from 16 to 33 years old.[11,12,27-29] Clinically, JAOF may present with facial asymmetry, headaches, facial pain, and chronic sinusitis due to rapid growth and obstruction. Radiological findings include a well-circumscribed, radiolucent, uni- or multilobulated lesion with varying opacification depending on calcification and cystic changes. CT scans usually reveal mixed soft tissue and bone densities with peripheral thinning of host bones.[17,20] Microscopically, JAOF usually demonstrates well-demarcated but nonencapsulated proliferative growths that allow infiltration and destruction of adjacent bones. Distinctive histopathologic features characteristic of trabecular and psammomatoid variants of JAOF have been described.[12] The trabecular variant shows hypercellular proliferation consisting of fibroblastic spindle cells with long and slender cellular osteoid forming a "paint brush strokes" pattern. By contrast, the psammomatoid variant shows numerous closely packed spherical basophilic calcified psammoma bodies embedded in hypercellular fibroblastic stroma. Myxoid changes, cystic degeneration, and sometimes secondary aneurysmal bone cysts have been reported in both variants.[12,18,27] Molecular studies identified chromosomal breakpoints that characterize JAOFs and separate them from other fibro-osseous lesions affecting the maxillofacial complex.[12-14] A recent study performed by Tabareau-Delalande et al. revealed that JAOFs demonstrated long arm rearrangements covering the MDM2 and RASAL1 genes on chromosome 12, which caused simultaneous amplification of MDM2 and RASAL1 significantly more frequent in JAOF when compared with fibrous dysplasia and non-juvenile ossifying fibroma (69% vs 9% and 6%, respectively).[13] This abnormality is not only a potential diagnostic molecular marker but may also be indicative of aggressive and more extensive forms of this entity with a higher risk of recurrence.[13] In addition, MDM2 amplification using fluorescence in situ hybridization was found (in those 69% of cases) without concordant immunohistochemistry overexpression.[13] Sawyer et al. reported nonrandom chromosome breakpoints at Xq33 & 2q33, resulting in (X;2) translocation in a subset of orbital cases of psammomatoid variant of JAOF.[14] Treatment for JAOF ranges from enucleation and curettage to resection of the tumor with clear margins, when possible.[15,24] Small lesions are usually treated with curettage or ostectomy, whereas extensive and deeply infiltrating lesions extending into the base of the skull may be treated with debulking or complete resection via endoscopic surgery and craniotomy. Authors reported a high recurrence rate after conservative or minimally invasive treatment (30%-58% of cases); thus, complete surgical resection remains the preferred line of treatment and portends a good prognosis.[10,20,25,26]

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CLINICAL PATHOLOGIC CONFERENCE CASE 4: MALIGNANCY MIMICKING A REACTIVE PROCESS: MULTIFOCAL LEUKOPLAKIAS AND PIGMENTED LESIONS ON THE BUCCAL MUCOSA

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Clinical Presentation: The patient was a 50-year-old male with a social history significant for heavy smoking of many years' duration. On intraoral exam, the right buccal mucosa displayed multiple scattered areas of pigmentation that vary in size, shape, and intensity of color with 2 areas of overlying leukoplakia that were relatively well defined (Figure 1). There was also a solitary oval pigmented lesion that was heterogeneous in color on the left soft palate (Figure 2). Because of the patient's social history, the clinician was concerned about the leukoplakias on the right buccal mucosa, which prompted an incisional biopsy of the anterior and posterior leukoplakic areas. The patient was relatively light-skinned with dark hair and medical history was stated to be unremarkable.

Differential Diagnosis: Because the patient presented with leukoplakias overlying areas of pigmentation, the differential diagnosis is broad. Leukoplakias in the oral cavity can represent many histologic diagnoses, including hyperkeratosis, epithelial dysplasia, and squamous cell carcinoma, among others.[1] The differential diagnosis for pigmented lesions in the oral cavity is also extensive and can include physiologic pigmentation, exogenous sources of pigmentation, melanocytic pigmentation, and systemic diseases that cause oral pigmentation.[2,3]

Hyperkeratosis is a benign diagnosis in the oral cavity with the characteristic histologic feature being a thickened surface keratin layer. Epithelial dysplasia can appear clinically similar to hyperkeratosis but with atypical changes to the keratinocytes that

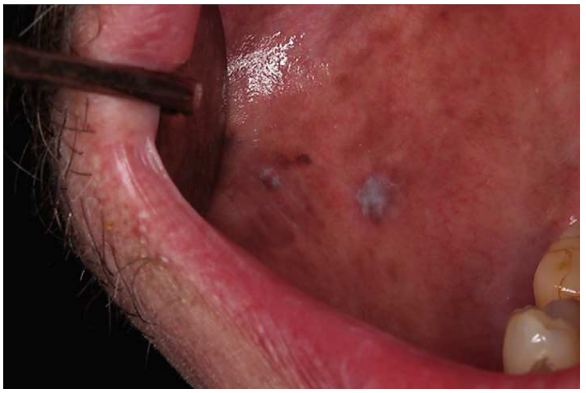

Fig. 1. Irregular scattered areas of pigmentation are seen on the left buccal mucosa. Two areas of pigmentation also have associated overlying relatively well-defined leukoplakias.