Enlarging growth of the mental region in a 48-year-old man

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A 48-year-old man was referred to the Oral and Maxillofacial Surgery Clinic at the London Health Sciences Centre for a mass on the right chin (Figure 1). The mass had initially appeared 2 years before presentation, without preceding trauma. There was no reported pain, numbness, or discoloration of the overlying skin or oral mucosa associated with the lesion. Over the preceding 6 months, an interval increase in size was noted, with fluctuations in size reported to occur with anxiety and blood pressure elevation. The patient's past medical history was noncontributory, and he was a nonsmoker.

On physical examination, the patient appeared healthy and in no distress. His vitals were stable, and he was afebrile. A nonmobile mass, measuring approximately $3.2 \times 2.5 \times 2.0$ cm, protruded from the right chin. Examination of the oral cavity revealed a well-circumscribed mass in the right lower labial/vestibular mucosa, with recession of the attached gingiva surrounding the right lower anterior dentition (Figure 2). The mass was firm, with no tenderness on palpation and no audible bruit or palpable thrill. The tissue overlying the mass showed normal mucosal coloration. Dental examination was unremarkable.

The patient had previously undergone an ultrasound examination of the mass at a different institution, and the result had suggested a possible vascular etiology. To investigate further, computed tomographic angiography (CTA) and magnetic resonance imaging (MRI) of the head and neck were obtained. CTA showed a well-defined, oval-shaped, enhancing, soft tissue mass in the right parasymphyseal region, localized within the right mentalis and depressor muscles adjacent to, but not eroding, the mandibular body (Figure 3). The mass had multiple internal blood vessels (Figure 4) arising from the right facial and lingual arteries. MRI showed the mass to have low T1-weighted and high T2-weighted signals, flow-voids, and avid enhancement with contrast.

DIFFERENTIAL DIAGNOSIS

The presentation of the right mental mass was not clinically distinctive, and thus, correlation of both clinical and radiographic features was needed to develop a suitable differential diagnosis. The overall appearance of the mass, particularly its well-circumscribed periphery, lack of bone erosion, and reported slow growth, favored a benign process. Coupled with the imaging findings of internal vessels and avid enhancement with contrast, the overarching picture was that of a hypervascular lesion. Furthermore, the subepithelial location of the mass suggested mesenchymal origin. Therefore, the primary differential diagnostic considerations for the right mental mass included benign mesenchymal lesions with internal vasculature, namely, hemangioma, vascular malformation, and vascular soft tissue tumors, such as angioleiomyoma.

Currently, vascular anomalies are classified by the International Society for the Study of Vascular Anomalies into 2 distinct groups—vascular tumors and vascular malformations. This classification system is based on the work of Mulliken and Glowacki,[1] who first proposed the system of vascular anomalies on the basis of clinical features, behavior, and pathologic features.[2]

Hemangiomas are proliferating vascular tumors that can be categorized as infantile or congenital, on the basis of their clinical presentation and histology.[1,2] Infantile hemangiomas are the most common and present shortly after birth.[2] With respect to their clinical behavior, infantile hemangiomas generally exhibit early rapid growth, termed the *proliferative phase*, followed by involution over several years.[2] Although typically diagnosed in pediatric populations,

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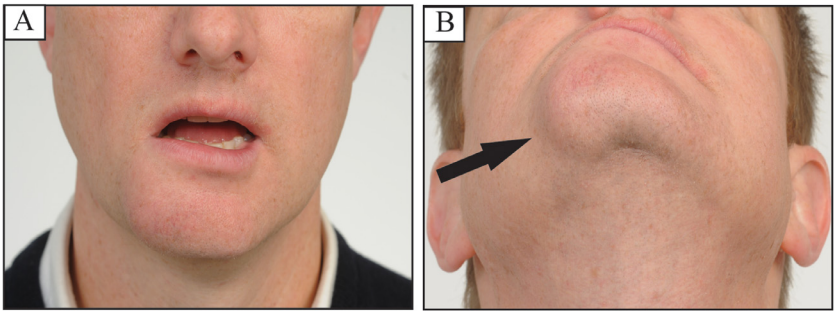

Fig. 1. **A, B,** Extraoral images of the right mental mass.

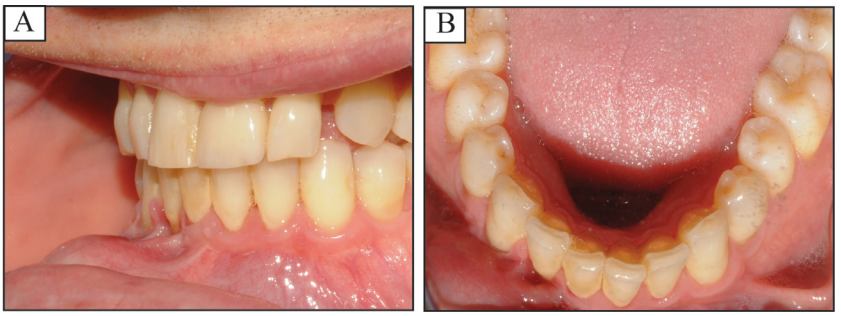

Fig. 2. **A, B,** Intraoral images showing a demarcated mass of the right lower labial/vestibular mucosa.

hemangiomas that involute may persist as clinically evident lesions in adulthood.[1,3] Hemangiomas can have clinical and histopathologic features in common with several other benign vascular lesions, most notably vascular malformations. Historically, it is probable that confusion regarding the nomenclature and categorization of benign vascular lesions contributed to the misclassification of many vascular lesions as hemangiomas, particularly the so-called cavernous hemangiomas, which likely represent venous malformations.[4] Human glucose transporter protein-1 (GLUT-1) has proven to be a useful marker in the classification of oral vascular lesions. Infantile hemangiomas, thought to develop as a result of endothelial cell proliferation, demonstrate consistent and reproducible GLUT-1 staining, whereas other vascular lesions, including other vascular tumors, vascular malformations, and reactive proliferations, do not.[2,5] Furthermore, 2 recent studies reported that 66% to 77% of oral lesions previously diagnosed as hemangiomas would be more appropriately classified as pyogenic granulomas or vascular malformations after examination of GLUT-1 expression.[3,6] Congenital hemangiomas are rare, and their pathogenesis is poorly understood. By definition, congenital hemangiomas are present at birth and may be categorized as rapidly involuting or noninvoluting.[5,7] In contrast to infantile hemangiomas, congenital hemangiomas are GLUT-1 negative.[2,5] Our patient reported that the lesion arose in adulthood and grew over a 2-year period. Therefore, the probability that our lesion represented a persistent infantile hemangioma was unlikely.

Vascular malformations are described by Mulliken and Glowacki[1] as low-flow or high-flow lesions, which present at birth but may not become apparent until later in life.[1,8] Unlike hemangiomas, vascular malformations do not show the same tendency for regression but may show continued growth and recurrence.[2] Individuals with vascular malformations can present with pain and/or swelling, which may be related alterations in normal blood flow or trauma-related thrombosis.[2] Our patient's lesion had no audible bruit or palpable thrill, and imaging findings did not support a high-flow vascular lesion. Therefore, arteriovenous malformation, was excluded from the differential diagnosis. The most common low-flow lesion, venous malformation, is composed of groups of dilated vascular channels.[2,7] The theoretical mechanism of continued growth in venous malformations is hypertrophy, rather than proliferation, primarily caused by increased blood flow across a collection of anomalous vessels.[2,8] Although often compressible, venous malformation may present clinically as a circumscribed soft tissue mass that can mimic other soft tissue lesions.[9]

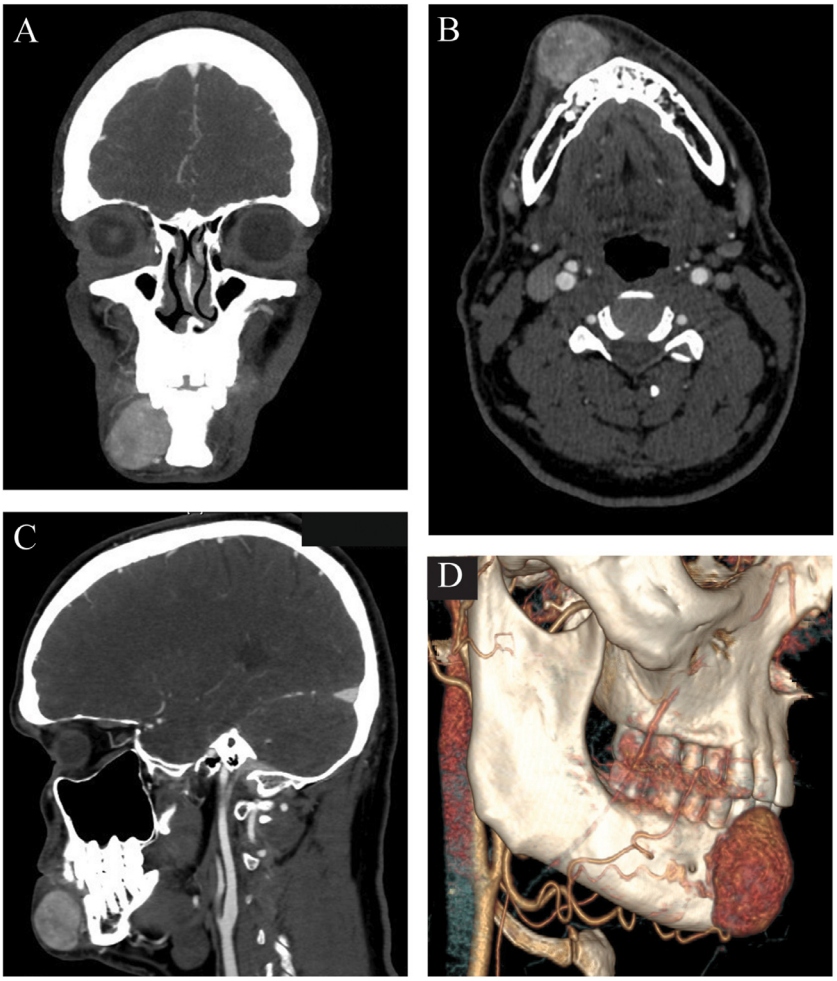

Fig. 3. Select coronal **(A)**, axial **(B)**, sagittal **(C)**, and 3-dimensional reconstruction **(D)** of the head and neck computed tomographic angiography (CTA) images show a well-defined, oval-shaped, enhancing mass within the right mentalis and depressor muscles, abutting on, but not eroding, the right mandibular parasymphyseal region. Blood vessels are seen rimming the periphery of the mass.

Given the well-demarcated, subepithelial nature of the lesion in this case, a vascular soft tissue tumor was also considered in the differential diagnosis. Several soft tissue tumors with a significant vascular component are known to occur in the oral cavity, including angioleiomyoma, solitary fibrous tumor, and angiolipoma. All of these entities are uncommon, and in many instances, their prominent vascularity appears to be based on histologic, rather than clinical or radiographic observations.[10-12] Intraoral angioleiomyomas are uncommon, benign tumors, thought to originate from vascular smooth muscle.[12] Angioleiomyomas are enhanced markedly with contrast and have flow voids on MRI,[13] similar to the imaging features of our case. Angioleiomyomas occur most commonly in the lip but can be seen in the palatal mucosa, buccal mucosa, tongue, gingiva, and, rarely, bone.[12,14] Angioleiomyomas may show bluish coloration and may be compressible,[12] characteristics shared with other benign vascular lesions. Clinically, angioleiomyomas are not distinctive and are commonly diagnosed as benign tumors, reactive lesions, or minor salivary gland tumors, depending on location.[14] Angioleiomyomas most commonly occur in males in their fifth to sixth decades.[14]

DIAGNOSIS AND MANAGEMENT

After consideration of the clinical and radiographic findings, complete surgical excision of the lesion under general anesthesia was planned. Because of the vascular nature of the lesion, the patient was scheduled for embolization 6 days before excision. Angiography revealed 2 dominant arterial pedicles arising from a common lingual facial arterial trunk, feeding the right mental mass (see Figure 4). Embolization was

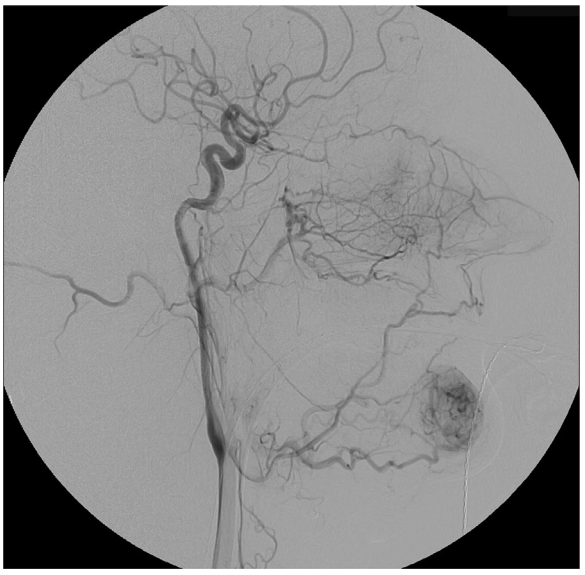

Fig. 4. Angiography shows the right facial and lingual arteries feeding into multiple internal blood vessels of the hypervascular mass.

performed by injection of 200 μm of polyvinyl alcohol particles held in a suspension of iodinated contrast, and 2 fibred coils. There was marked devascularization of the mass, with greater than 95% decrease in the extent of tumor blush.

Following embolization, the patient was brought to the operating room, where surgical resection of the right mental mass was completed. The mucosa overlying the lesion was incised, and dissection was carried out uneventfully. Multiple small vessels were cauterized, but a single, dominant vessel was clipped at the inferolateral aspect of the lesion; otherwise, no significant vasculature was encountered. The lesion was removed in its entirety. Grossly, the lesion was a white, firm-to-rubbery mass.

Histopathologic examination revealed well-demarcated, cellular proliferation of ovoid spindled cells in a predominantly patternless arrangement, admixed with dense collagen and a conspicuous branching vascular component (Figure 5). Occasional mitotic figures (3 per 10 high-power fields [HPF]) were seen throughout the stromal cells; however, no necrosis, fascicular growth, or pleomorphism was present. Immunohistochemistry (IHC) revealed diffuse nuclear positivity for STAT6 and membranous positivity for CD34. Stains for S-100 and smooth muscle actin were negative (Figure 6). The histopathologic and IHC findings supported a diagnosis of solitary fibrous tumor (SFT).

The patient was discharged home on postoperative day 1. At the 6-month follow-up, the patient was convalescing well, with no evidence of recurrence.

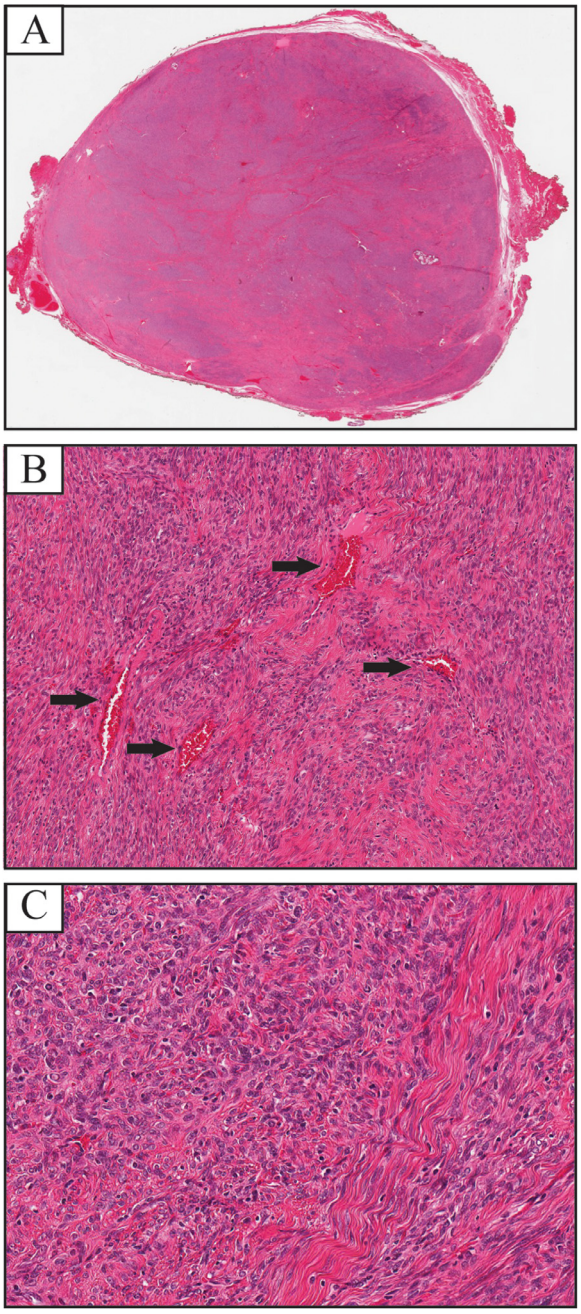

Fig. 5. Photomicrographs of hematoxylin and eosin−stained sections. **A,** Low-power image shows well-circumscribed to partially encapsulated cellular neoplasm (magnification × 0.4). *A high-resolution version of this slide for use with the Virtual Microscope is available as eSlide: VM05049.* **B, C,** Higher-power images (B, × 10; C, × 20) demonstrate a lesion composed of bland spindled cells with prominent stromal collagen and a conspicuous vasculature (arrows).

DISCUSSION

Since the time that SFTs were first described in the pleura,[15] their histogenesis has been a contentious issue.[16] Originally believed to arise from

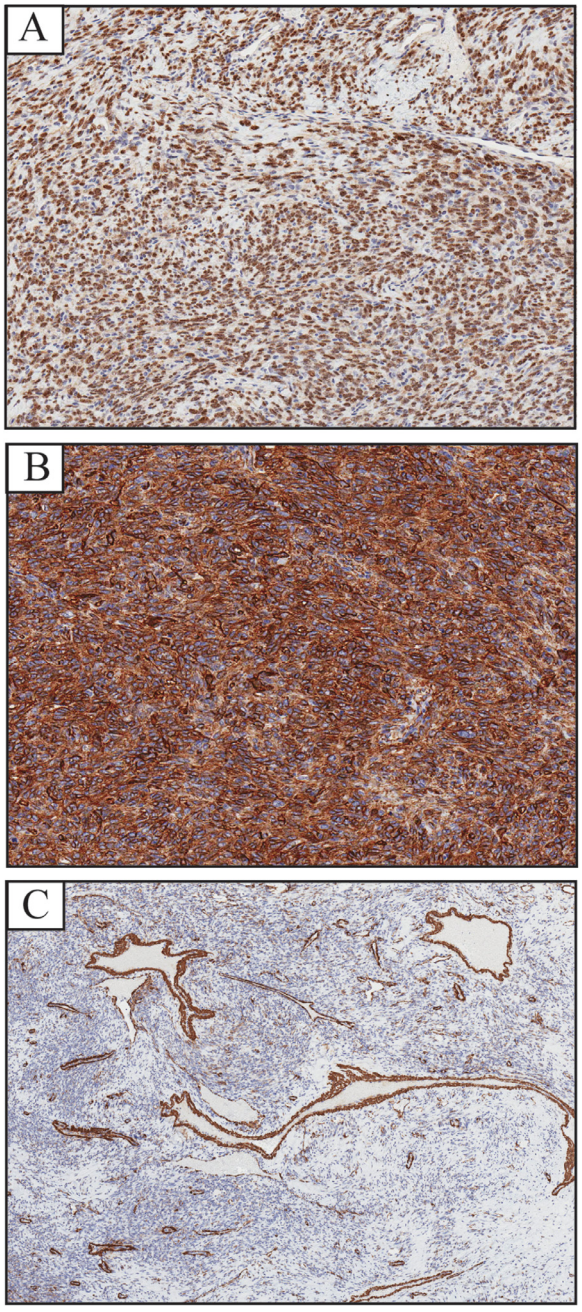

Fig. 6. **A,** Strong, diffuse nuclear positivity for STAT-6 (× 10). **B,** CD34 shows strong membranous positivity in stromal cells (× 10). **C,** SMA highlights vascularity of lesion (× 5).

mesothelial cells or submesothelial fibroblasts, SFTs were not thought to occur outside of the pleura and peritoneum.[17] However, advances in electron microscopy and IHC have led to the identification of SFTs in many extrapleural locations, establishing SFT as a ubiquitous mesenchymal tumor of fibroblastic/myofibroblastic origin.[16,18,19] Even after more widespread recognition in the mid-1990s, extrapleural SFTs were likely underreported, with many classified as hemangiopericytoma.[16,19] Outside of the sinonasal tract, where the term sinonasal-type hemangiopericytoma persists as a synonym for the myopericytic tumour, glomagiopericytoma, HPC is now considered an antiquated term and was eliminated in the 2002 World Health Organization soft tissue classification.[19,20]

In extrapleural locations, SFTs most commonly affect the abdominal cavity, orbit, upper respiratory tract, and soft tissue.[16] In a recent multicenter study, Smith et al.[21] reported on 88 cases of SFT in the head and neck region. The most commonly affected site was the sinonasal tract (30%), followed by the orbit (25%) and the oral cavity (15%). Of oral mesenchymal tumors, SFTs are decidedly uncommon, representing approximately 3% of cases.[10]

Clinically, oral SFTs typically present as asymptomatic, circumscribed, firm lesions.[17] SFTs have been reported to occur in a variety of intraoral sites, but the buccal mucosa, lip, and tongue are the most common.[22] Some authors have reported an association between SFT and previous trauma,[17] although no history of trauma was reported by our patient. Intraoral SFTs do not have a specific sex predilection and can occur over a wide age range, with the sixth decade being the most frequently reported.[22] The vascular nature of SFTs is typically not a prominent feature recognized clinically, radiographically, or preoperatively, although Carlos et al.[10] recently reported a case affecting the mucosa of the floor of mouth with a prominent superficial vasculature. The reported size of oral SFTs is variable, ranging from 0.7 to 7.5 cm.[17,22] At 3.2 cm in greatest dimension, the lesion in our patient was moderately sized.

Histologically, the classic type of SFT is a circumscribed spindle-cell lesion with a prominent branching or hemangiopericytoma-like vascular pattern. The spindled cells are bland with scant cytoplasm and arranged in a "patternless" pattern.[16,23] The stroma is typically collagenous but can show variability ranging from myxoid to sclerotic.[24] Several microscopic variants of SFTs have been described, including myxoid, epithelioid, lipomatous, and giant-cell rich types.[21] SFTs showing atypical or malignant features have also been described. In general, histologic features that may impart an aggressive or malignant clinical course include a mitotic rate greater than 4/10 HPF, increased cellularity, and cellular pleomorphism.[16]

Not surprisingly, the broad range of microscopic features in SFTs may lead to difficulty in diagnosis, particularly with small biopsy samples. Depending on the histomorphology of the lesions, SFTs can show overlap with several other benign and malignant mesenchymal lesions, including soft tissue perineurioma, spindle cell lipoma, myofibroma, desmoplastic fibroma, synovial

sarcoma, myxoid liposarcoma, and myxofibrosarcoma.[22] Therefore, IHC workup is necessary for definitive diagnosis. Several studies have demonstrated a *NAB2-STAT6* fusion in SFTs,[24-26] and nuclear expression of STAT6 is helpful in distinguishing SFTs from other spindle cell neoplasms.[24] Before the discovery of the *NAB2-STAT6* fusion and the subsequent development of the STAT6 antibody, CD34 expression was considered fundamental to accurately identifying SFTs, with strong expression present in 95% of cases.[16,24] Other markers, such as bcl-2, CD99, and EMA, are also expressed in SFTs, but are considered nonspecific because they can also be expressed by histologic mimics.[24] Currently, because of the limited expression of STAT6 in other soft tissue tumors, nuclear positivity for STAT6 is considered to be a highly sensitive and specific marker for SFTs.[24] The present case was found to be positive for both STAT6 and CD34, and therefore IHC staining for bcl-2, CD99, and EMA was not done.

The imaging features of SFTs, both pleural and extrapleural, are considered nonspecific. Typically described as well-circumscribed, avidly enhancing, hypervascular, soft tissue masses, SFTs show radiographic overlap with several vascular pathologies.[27-31] Differentiation between benign and malignant SFTs is difficult, but the presence of cystic degeneration, tumor necrosis, and bone invasion on imaging suggest malignant behavior.[27] SFTs can mimic vascular malformations, and Wignall et al.[28] found that 12 (35%) of their 34 cases of pleural and extrapleural SFTs had prominent, collateral, feeding vessels on CT, MRI, and CTA.[28,30] SFTs can also mimic angioleiomyomas because their MRI features are similar.[13,31] SFTs and angioleiomyomas both have an iso/hypointense signal to muscle on T1-weighted images and a variable signal on T2-weighted images, depending on the amount of collagen present in the tumor.[13,27-31] SFTs with high T2-weighted signals tend to have more myxoid stroma, whereas those with low T2-weighted signals tend to have more collagenous stroma[13,27-31]; however, Weon et al.[32] found that 4 of their 6 intracranial SFTs more characteristically showed a "black and white mixed" T2-weighted signal, in which round or linear, low-signal foci, as a result of variable amounts of tumor collagen, were scattered on a background of high signal. Later authors dubbed this heterogeneous T2-weighted signal the "chocolate chip cookie" pattern[29] and suggested that its presence, along with the finding of collateral feeding and internal tumor vessels, can signal the radiologist to include SFTs in the differential diagnosis.[29,30] Because our current case showed peripheral feeding and internal tumor vessels, but not a heterogeneous T2-weighted signal, only vascular malformation was considered in the interpretation of the imaging reports.

Complete surgical excision is the treatment of choice for SFTs, and in the oral cavity, in the vast majority of cases, SFTs are clinically benign and do not recur.[10,33] Cox et al.[33] reviewed 142 head and neck cases and found recurrence only in cases of incomplete excision, although not all cases with positive margins experienced recurrence. O'Regan et al.[22] reported no recurrence in their 21 oral cases and no metastases in the 17 cases with available follow-up information. Alawi et al.[17] had follow-up information available for 10 patients and did not report any recurrences. Smith et al.[21] reported margin positivity in 67% of cases but found no correlation with recurrence. Of the 13 oral tumors evaluated in their study, 4 cases had no evidence of disease at follow-up, 1 recurred, and 8 were lost to follow-up. Of note, the single case with recurrence showed increased cellularity, atypia, epithelioid morphology, and a mitotic count of 5/10 HPF.

Although rare, clinically aggressive and malignant SFTs have been reported in the head and neck region. In a series of 9 malignant SFTs, Yang et al.[34] reported that 3 patients died as a result of locoregional or distant metastasis, although none of the cases appeared to involve the oral cavity per se. Of note, all of the cases in their series were described as having poorly demarcated features on imaging. Cox et al.[33] described 10 cases with atypical or malignant histology; however, no metastases occurred, and recurrence was reported in only 1 case with known positive margins. Kao et al.[35] described 2 malignant cases and 1 atypical case of SFTs in the oral cavity. None of these cases had recurrences, and no metastases were reported.

Even when atypical or malignant histology is present, prediction of the clinical behavior of SFTs can be remarkably difficult. Demicco et al.[36,37] proposed a risk stratification model for metastases of SFTs, on the basis of findings from a large series, including 12 cases from the head and neck region. They reported that increased risk of metastasis and death resulting from disease was associated with large tumor size, patient age greater than 55 years, mitotic rate 4/10 HPF or greater, and necrosis. On the basis of these parameters, tumors with high risk features were seen to have a 73% risk of metastasis at 5 years, whereas low risk tumors had a 0% risk of metastasis at 10 years.[37] Smith et al.[21] described a series of 88 cases of SFTs in the head and neck region. In their series, metastases were reported in 3 patients, although in none of the reported cases SFTs occurred in the oral cavity. Interestingly, lesion size and mitotic rate appeared to be associated with recurrence, rather than with metastasis. Smith et al.[21] explained that clinicopathologic factors predicting locoregional recurrence may be more useful in evaluating head and neck lesions, where metastases are uncommon.

The current case did not show any clinical or radiographic features of malignancy. The histomorphology was in keeping with a benign lesion; however, because of measurable mitotic activity, it was decided that the patient would be monitored closely.

CONCLUSIONS

In summary, our case demonstrates the difficulty in the preoperative diagnosis of oral SFTs, and the potential overlapping of their features with those of other vascular lesions. A variety of imaging modalities may be required to narrow the clinical differential diagnosis, and even then, this lesion may mimic a vascular lesion. Although uncommon, SFTs may be considered in instances where there is clinical suspicion of a vascular soft tissue tumor. The vast majority of excised oral SFTs behave in a benign fashion; however, lesions showing atypical microscopic features or positive surgical margins can recur. Metastases from oral lesions, if they occur, appear to be exceptionally rare.

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