Journal of Karnali Academy of Health Sciences
An Official Journal of Karnali Academy of Health Sciences

Solitary fibrous tumor of the nasal cavity- a rare case report

Sudeep KC[1], **Manisha Paneru**[2]

[1]Department of Radiology, Patan Academy of Health Sciences, Lalitpur, Nepal
[2]Department of ENT, Patan Academy of Health Sciences, Lalitpur, Nepal

ABSTRACT

Introduction: Solitary fibrous tumor (SFT) of the nasal cavity is a rare vascular tumor arising from adult mesenchymal stem cells. It occurs most commonly in the pleura, and in the nasal cavity, it is rare. Solitary fibrous tumors (SFTs) of the nose and paranasal sinuses are extremely rare.

Case presentation: This article reports a case of a 59-year-old lady presenting with complaints of progressive nasal blockage and recurrent epistaxis. Imaging showed a mass lesion in the left nasal cavity with bone-eroding properties mimicking a malignant lesion. The patient underwent surgery, during which the mass was excised. Histopathological analysis of the specimen was consistent with SFT.

Conclusion: This case highlights the importance of diagnosing SFT of the nasal cavity and paranasal sinuses, as their imaging findings and management differ from those of other tumors.

Keywords: DCE, DWI, Immunohistochemistry, Solitary fibrous tumor

INTRODUCTION

Solitary fibrous tumor (SFT) is a rare vascular tumor formerly called hemangiopericytoma. [1] Its occurrence in the nasal cavity is extremely rare, representing less than 0.1% of all types of sinonasal malignancy and approximately 3% of head and neck tumors.[2] Clinical SFT presentation varies, ranging from asymptomatic to massive epistaxis.[3] Therefore, they should also be considered in the differential diagnosis of vascular lesions of the nasal cavity. We present the CT and MR imaging features of a patient with sinonasal SFT confirmed by histopathology. No definite treatment protocol exists for this unique entity, but the tumor should primarily be treated with aggressive, wide local excision.

CASE PRESENTATION

A 59-year-old female presented to the Outpatient Department of Ear, Nose, and Throat (ENT) complaining of nasal obstruction (left side) for 6years. Nasal obstruction was progressive, bilateral, and more severe in the left nostril than in the right. Associated with unprovoked epistaxis, which was intermittent, scanty in quantity, 3-5 episodes in a month, controlled by nasal pinching or ice pack. Nasal obstruction was associated with a decreased sense of smell. On anterior rhinoscopy, there was a pale fleshy mass in the left nasal cavity pushing the septum towards the right. The patient was sent for a contrast-enhanced MRI (CE MRI) of the paranasal sinuses (PNS) for further evaluation.

CE MRI (Figure 1) showed a 4.8 x 4.3 × 3.7 cm-sized well-defined expansile multi-lobulated T1 heterogeneously hypointense lesion with corresponding T2/STIR hyperintensity noted within the nasal cavity; the epicenter of the lesion was in the left middle turbinate. Multiple T2 hypointense septae are pointed out within the lesion. Mild diffusion restriction was noted in its soft-tissue component, with an Apparent Diffusion Coefficient (ADC) value of 1.3x10-3 $^{mm-2/s}$. Post-contrast dynamic study showed avid and progressive enhancement of the lesion, with peak contrast uptake at 25 seconds and delayed washout. Contralateral extension noted involving the right middle turbinate via a midline septal defect of 18mm. Laterally, indentation of the medial wall of the bilateral orbit without intraorbital extension/erosion was observed. Superiorly, the lesion is abutting the bilateral cribriform plate and shows no intracranial extension. No abnormal enhancement or thickening of the adjacent dural lining was seen. Posteriorly, the lesion extended up to the posterior margin of the choana without extension into the nasopharynx. Extrinsic compression of the bilateral frontal recess and bilateral osteomeatal unit, causing retention of secretions in the bilateral frontal sinus and left maxillary antrum.

The patient also underwent CT angiography (Figure 2), which showed a hypervascular lesion (Figure 2a) with avid enhancement in the arterial phase, with progressive enhancement in subsequent phases (Figure 2c). Multiple enhancing septae are noted within the lesion. Focal erosion of the crista galli and bilateral cribriform plate was noted without obvious intracranial extension. Fovea ethmoidalis appears intact on both sides. Prominent bilateral terminal branches of the superior ophthalmic artery and the terminal branches of the facial artery at the nasolabial fold were noted to supply the lesion. The sphenopalatine artery on the bilateral side appears unremarkable and does not supply the above-mentioned lesion. No obvious vascular supply from the meninges was seen. Provisional diagnosis of vascular tumor, possibly solitary fibrous tumor, based on MRI and CT imaging was made, with other differentials being hemangioma and angiomatous polyps.

She underwent endonasal excision via the endoscopic modified Denkers approach with CSF leak repair via the bath plug technique. The operative findings showed a fleshy, highly vascular mass originating from the nasal septum, occluding the left nasal cavity and extending to the right nasal cavity, with doubtful attachment to the cribriform plate. The postoperative course was uneventful. On following up with the patient, the histopathological findings were consistent with a solitary fibrous tumor. Unfortunately, the IHC test was not available.

The histopathology report showed nasal mucosal tissue lined by ciliated pseudostratified columnar epithelium. Underlying stroma showed a well-encapsulated, moderately cellular nodular mass, composed of spindle cells in a collagenous stroma with prominent dilated staghorn-type vasculature. Mitotic rate was approximately 1-2 per 10 high-power fields. Overt cytologic atypia and tumor necrosis were not identified. The section showed fibrocollagenous tissue with sparse blood vessels. Stroma showed few mucous glands. Atypia was not identified.

Corresponding Author:
Dr. Sudeep KC
Email: drsudeepkc@gmail.com

Date of Submission: July 6, 2025
Date of acceptance: Nov 16, 2025
Date of Publication: Jan 10, 2026

DOI: https://doi.org/10.61814/jkahs.v8i3.1001

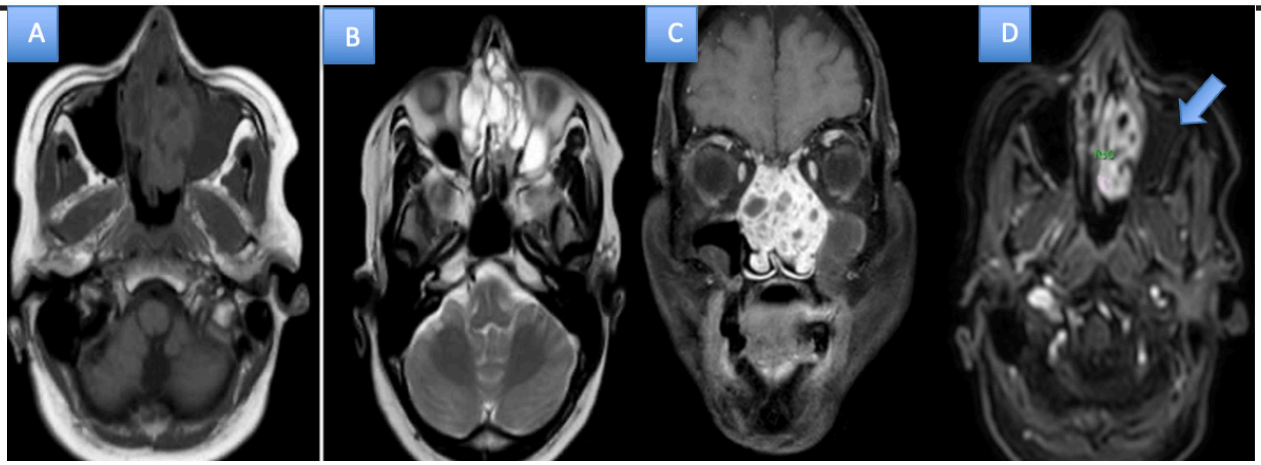

Figure 1. Axial T1-weighted MR image(A) shows well-defined homogeneous iso-intense signal mass in the left nasal cavity with heterogeneous hyperintense signal on T2 weighted axial image with multiple septations(B). Coronal(C) and Axial(D) contrast-enhanced MR image with fat saturation shows heterogeneous marked tumor enhancement located within nasal cavity only. No enhancement of the lesion of the left maxillary sinus(arrow) is noted, indicating obstructive maxillary sinusitis.

The postoperative period was uneventful. The patient was advised to follow up. No post-operative adjuvant therapy was given. At the time of reporting this study, there has been no evidence of recurrence in 2 years since the primary treatment.

DISCUSSION

Lietaud initially reported SFT in the pleura in 1767, and Wagner followed suit in 1870. Pleural tumors were divided into two categories by Klemperer and Rabin in 1931: diffuse mesotheliomas and localized mesotheliomas, often known as SFT.[4] Witkin and Rosai were the first to document a head-and-neck instance in 1991. Based on histological characteristics, SFTs were classified as benign or malignant. Since immunohistochemical and ultrastructural studies have shown that SFT are most likely generated from adult mesenchymal stem cells rather than mesothelium, the term SFT is currently preferred.[5]

Although SFT typically originates in the pleura, it has also been found in meninges (27%), abdomen (20%), trunk (10%), extremities (8%), and neck (5%). The head and neck region is a rare site for SFT, with very few isolated case reports available worldwide. Previous case series indicate that SFT in this region has no gender preference and tends to appear during the fourth decade of life. The majority of the lesions are unilateral, followed by combined involvement of the nasal cavity and paranasal sinuses.[6]

Clinical presentation is widely variable, from nasal obstruction to massive epistaxis.[3] Rarely, these lesions are associated with paraneoplastic hypoglycemia (Doege-Potter syndrome), arthralgia, osteoarthropathy, and finger clubbing. These symptoms usually subside upon the tumor's resection.[7]

The sonographic and X-ray appearance of SFT is not pathognomonic. On CT, these lesions appear iso-dense relative to muscle/grey matter and show marked enhancement. Internal calcifications can be identified on occasion. Bone remodeling, weakening, local absorption, and possibly reactive sclerosis can occur in large sinonasal SFT. However, frank bone destruction is uncommon.[3] According to Rosado-de-Christenson et al., isodensity on pre-contrast CT scans correlates with hypercellular areas and capillary networks. At the same time, markedly enhanced regions represent hypervascular areas, intermediate enhanced areas correlate with hypocellularity, and patchy hypodensities correspond to necrotic, myxoid, or cystic changes.[8] Preoperative angiography helps in diagnosis and defines the blood supply to the tumor, and also permits selective embolization of the feeder vessels.[9]

Currently, Gadolinium-enhanced MRI is also the best imaging modality for confirming the location and extent of nasal SFT, assessing its relationship to adjacent structures, and following SFT in the nasal cavity. Compared with CT, MR imaging can demonstrate characteristic features: these lesions appear as homogeneously T1 isointense relative to cerebral gray matter and heterogeneous hypointense on T2-weighted imaging (T2WI).[10] Signal intensity on T2-weighted images depends upon the amount of collagen, cellularity, and degeneration. A predominant low signal on T2-WI images is unusual in other nasal lesions; this feature is an important diagnostic clue for SFT. Marked heterogeneous enhancement of sinonasal SFT is generally noted due to their high vascularity and multiple flow voids within the tumors, which is also a critical MR imaging feature.[10,11] Similarly, to the Tateishi et al. study, the enhancing portions corresponded histologically to areas of abundant cellularity combined with microvessels.[12]

Dynamic contrast-enhanced MRI (DCE-MRI) can provide information on lesion perfusion and microvascular permeability and can predict their biological behavior. The time intensity curve (TICs) of this patient showed a washout pattern (type 3), which is similar to those of the internal carotid artery, suggesting SFT is a markedly hypervascular

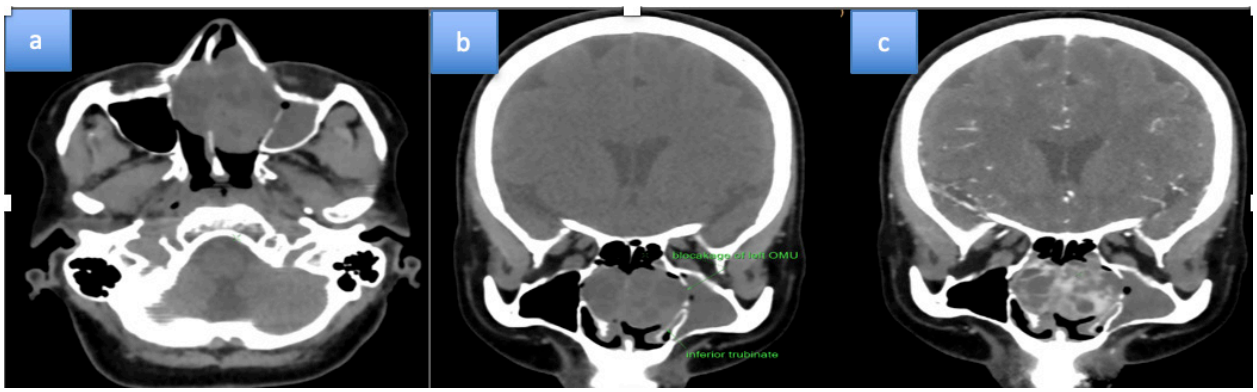

Figure 2: Axial and Coronal CT scan(a,b) shows a well-defined soft tissue density mass in the bilateral nasal cavity with subtle hyperdensity. Associated remodeling, thinning, and local absorption of the adjacent bony walls(b). Post-contrast study showing avid enhancement of the lesion with non-enhancing areas(c)

lesion, and this TIC pattern is unusual for nasal lesions except for juvenile angiofibroma.[13]

Diffusion weighted imaging (DWI) is sensitive in evaluating the histologic and physiologic status of various lesions and providing quantitative information distinguishing malignant tumors from benign lesion.[11,12] To the best of our knowledge, no prior literature reports of DWI of nasal SFT have been published till now. Our case showed relatively high ADC values, consistent with benign lesions based on the ADC threshold of 1.0 $\times 10^{-3}$ mm2/s for predicting lesion nature. Further studies to suggest the diagnostic value of DWI in the discrimination of tumors in this region is still under study.[10]

The role of preoperative biopsy is controversial given the lesion's highly vascular nature.[14] Preoperative embolization might reduce the intraoperative bleeding. The histopathologic appearances of SFT exhibited proliferation of spindle-shaped cells in various architectural patterns, including a "patternless pattern". Positivity for Cluster of Differentiation (CD)34, vimentin, CD99, and B-cell lymphoma 2 (Bcl-2) is non-specific for SFT. In contrast, SFTs are generally negative for cytokeratins, actins, desmin, and S-100 protein.

Recently, Signal Transducer and Activator of Transcription 6 (STAT6) has emerged as a more reliable marker for diagnosing SFT.15 Pathological criteria for aggressiveness are not well defined. However, WHO categorizes SFT as malignant based on tumor size, cellularity, nuclear pleomorphism, mitosis, and necrosis. Sometimes recurrence and metastasis are seen in SFT without atypical histological features; thus, histology alone cannot predict the aggressiveness of these lesions.16

Differential diagnosis for sinonasal SFT includes inverted papilloma, hemangioma, juvenile angiofibroma, angiomatous polyps, and hemangiopericytoma. A convoluted cerebriform pattern is typical for inverted papilloma. Hemangioma shows high signal intensity on T2-weighted MRI images, with a marked "progressive" enhancement pattern on DCE-MRI. Schwannoma is more common in the extraconal space of the orbit, is heterogeneously isointense or mildly hyperintense on T2WI, and has a persistent TIC pattern on DCE-MRI. Hemangiopericytoma is a rare vascular tumor that appears as a well-defined, enhancing soft-tissue mass that is isointense to gray matter on T1WI and hyperintense on T2WI, and may be associated with infiltration into adjacent structures or bony erosion. Angiomatous polyps often arise in the choanal region, showing a hyperintense signal on T2-weighted images and marked enhancement. Male adolescents make up the majority of patients with juvenile angiofibroma arising at or near the sphenopalatine foramen. The typical imaging findings include bony remodeling and destruction, as well as marked enhancement. [17]

The role of PET/CT is limited for assessing multifocal disease, distant metastases, and disease recurrence after resection.[18]

Surgical resection with optimal margins is the treatment of choice as SFT can have a potentially malignant behavior, and to avoid local recurrence. 10-15% cases of SFT are malignant. No definitive role for radiotherapy or chemotherapy in the management of sinonasal SFT is suggested in the literature. However, they are used for locally advanced or recurrent disease in individual cases.[19] Careful follow-up, including imaging evaluation, is necessary because the recurrence rate of SFT in this region after primary resection is approximately 20%. Recurrent SFT tends to extend locally and invade the adjacent bone or soft tissue, making complete resection more difficult.[10]

CONCLUSION

Although sinonasal SFT is an uncommon entity, its typical imaging characteristics, including bony remodeling and thinning, marked contrast enhancement, and a washout TIC pattern, may help suggest this diagnosis preoperatively. Head and neck SFTs have a good prognosis and are more likely to be benign than elsewhere. There is low potential for disease recurrence, requiring close clinical surveillance.

DECLARATION

Acknowledgements

None

Author Contributions

SK and MP conceptualized and designed the review of the literature; SK and MP conducted data collection; SK and MP drafted the manuscript and conducted the critical analysis; all authors reviewed the manuscript and approved the final version. All authors agreed to be accountable for all aspects of the research work.

Ethical Approval

Not applicable

Consent/Assent

Written informed consent was obtained from the patients for publication along with relevant images. Patients' identities are not disclosed.

Data Availability Statement

Any required information is available upon request from the corresponding author.

Conflicts of Interest

None

Source of Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors

REFERENCES

1. Nicolas Penel, Eric Yaovi Amela, Gauthier Decanter, Yves-Marie Robin, Perrine Marec-Berard, "Solitary Fibrous Tumors and So-Called Hemangiopericytoma", Sarcoma, vol. 2012, Article ID 690251, 6 pages, 2012. | DOI |
2. Di Bartolomeo M, Negrello S, Pellacani A, Cesinaro AM, Vallone S, Presutti L, Chiarini L, Anesi A. A Case Report of a Solitary Fibrous Tumor of the Maxillary Sinus. Reports. 2021; 4(4):33. | DOI |
3. Mathew GA, Ashish G, Tyagi AK, Chandrashekharan R, Paul RR. Solitary Fibrous Tumor of Nasal Cavity: A Case Report. Iran J Otorhinolaryngol. 2015 Jul;27(81):307-12. | PubMed |
4. Jadczak P, Guz W, Kaznowska E, Ramotowski R, Szalacha-Tarała E, Górecki A, Samojedny A. Solitary fibrous tumor of the pleura - cases analysis. Pol J Radiol. 2014 Oct 21; 79:368-73. | DOI |
5. Rodríguez-Gil Y, González MA, Carcavilla CB, Santamaría JS. Lines of cell differentiation in solitary fibrous tumor: an ultrastructural and immunohistochemical study of 10 cases. Ultrastructural Pathology. 2009 Jan 1;33(6):274-85. | DOI |
6. Tariq MU, Din NU, Abdul-Ghafar J, Park YK. The many faces of solitary fibrous tumor: diversity of histological features, differential diagnosis, and role of molecular studies and surrogate markers in avoiding misdiagnosis and predicting the behavior. Diagn Pathol. 2021 Apr 20;16(1):32. | DOI |
7. Solsi A, Pho K, Shojaie S, Findakly D, Noori T. Doege-Potter Syndrome and Pierre-Marie-Bamberger Syndrome in a Patient with Pleural Solitary Fibrous Tumor: A Rare Case with Literature Review. Cureus. 2020 May 1;12(5):e7919. | DOI |
8. Rosado-de-Christenson ML, Abbott GF, McAdams HP, Franks TJ, Galvin JR. From the archives of the AFIP: localized fibrous tumor of the pleura. Radiographics. 2003;23(3):759–783. | DOI |
9. Ciochon UM, Schmidt G, Jensen RJ, Loya AC, Lönn LB, Shekhrajka N. Preoperative Embolization of a Solitary Fibrous Tumor Originating from External Auditory Meatus: A Case Report with Literature Review. Diagnostics(Basel). 2021 Jan 2;11(1):62. | DOI |
10. Yang BT, Song ZL, Wang YZ, Dong JY, Wang ZC. Solitary fibrous tumor of the sinonasal cavity: CT and MR imaging findings. AJNR

Am J Neuroradiol. 2013 Jun-Jul;34(6):1248-51. | DOI |

11. Li XM, Reng J, Zhou P, Cao Y, Cheng ZZ, Xiao Y, Xu GH. Solitary fibrous tumors in abdomen and pelvis: imaging characteristics and radiologic-pathologic correlation. World J Gastroenterol. 2014 May 7;20(17):5066-73. | DOI |

12. Tateishi U, Nishihara H, Morikawa T, Miyasaka K. Solitary fibrous tumor of the pleura: MR appearance and enhancement pattern. J Comput Assist Tomogr. 2002, 26 (2): 174-179. | DOI |

13. Liu Y, Tao X, Shi H, Li K. MRI findings of solitary fibrous tumors in the head and neck region. Dento Maxillofac Radiol. 2014;43(3):20130415. | DOI |

14. Shu Q, Liu X, Yang X, Guo B, Huang T, Lei H, et al. Malignant solitary fibrous tumor of the liver: a case report. Int J Clin Exp Pathol. 2019 Jun 1;12(6):2305-2310. | PubMed |

15. Geramizadeh B, Marzban M, Churg A. Role of immunohistochemistry in the diagnosis of solitary fibrous tumor, a review. Iran J Pathol. 2016;11(3):195-203. | PubMed |

16. Thompson LDR, Lau SK. Sinonasal Tract Solitary Fibrous Tumor: A Clinicopathologic Study of Six Cases with a Comprehensive Review of the Literature. Head Neck Pathol. 2018 Dec;12(4):471-480. | DOI |

17. Yang BT, Song ZL, Wang YZ, Dong JY, Wang ZC. Solitary fibrous tumor of the sinonasal cavity: CT and MR imaging findings. AJNR Am J Neuroradiol. 2013 Jun-Jul;34(6):1248-51. | DOI |

18. Kruse M, Sherry SJ, Paidpally V, Mercier G, Subramaniam RM. FDG PET/CT in the management of primary pleural tumors and pleural metastases. AJR Am J Roentgenol. 2013 Aug;201(2): W215-26. | DOI |

19. Kaoutar C, Ahmedou AB, Omar B, Anas B, Oukessou Y, Abada R, Sami R, Mohamed R, Mohamed M, Meriem R, Mehdi K. Endoscopic resection of solitary fibrous tumor of the ethmoid: Case report review of the literature. Ann Med Surg (Lond). 2020 Dec 7; 60:714-718. | DOI |