DOI: 10.7860/NJLM/2026/88580.2998

Case Series

Pathology Section

Diagnostic Challenges and Management of Extrapulmonary Solitary Fibrous Tumours: A Series of Three Cases

SOUMITA GHOSH[1], MEGHADRI SEN[2], BISWANATH PAUL[3]

ABSTRACT

Solitary Fibrous Tumours (SFT)s are rare soft-tissue tumours of mesenchymal origin, which are even more rare in the head and neck region. They are typically benign in nature but in rare circumstances may show transformation into malignancy. Histologically, they show a well-encapsulated biphasic lesion, having hypo- and hyper-cellular with cells with bland spindle shaped nuclei, arranged in fascicles, in a stroma comprised of ropey collagen, having haemangiopericytoma like patulous vasculature. Occasionally, the lesion may show cystic spaces. Usually, these tumours do not show necrosis, atypia or abnormal mitoses. SFTs are typically positive for STAT6, CD34, BCL2 and CD99. Complete surgical excision is the treatment of choice, but often the tumour shows recurrence. This case series presents three cases. The first case is a 32-year-old male presenting with a painless firm mass, in right submandibular region since last one year. The second case is a 41-year-old male presenting with a firm swelling over the right-side of his nose, with tenderness in the area, associated with epistaxis from his right nostril and difficulty in breathing. He was previously operated for the same in 2018, but few months after every surgery the symptoms had recurred, and he had undergone a total of three operative procedures and one session of radiotherapy till 2024. The third case is a 62-year-old male presenting with a slow growing, non tender swelling over the calf of his left leg for one year. All the cases have been managed with total surgical resection, and microscopically showed the above-mentioned histologic features of SFTs. On immunohistochemistry, they showed positivity for STAT6, BCL2, CD34 and CD99. Based on the histological and Immunohistochemistry (IHC) findings, the tumours were diagnosed to be SFT. This series is focused on the extrapulmonary unusual sites of presentation of SFT, because early diagnosis and treatment is always helpful to the patients.

Keywords: Biphasic, Mesenchymal origin, Submandibular region, Well-encapsulated

INTRODUCTION

The SFT is a spindle cell neoplasm of mesenchymal origin showing fibroblastic differentiation and it can occur anywhere in the body [1,2]. Ernst Wagner's 1870 paper is historically recognised as the first published description of a solitary fibrous tumour (SFT), specifically of the pleura [3]. Klemperer P and Rabin CB, in 1931, divided pleural tumours into two classes [4]: (i) diffuse mesotheliomas; (ii) localised mesotheliomas or SFT. Although previously SFT was thought to have a mesothelial origin SFT is now considered as a mesenchymal neoplasm, occurring at any anatomical site [4-6], the pleura being the most common site, accounting for around >30% of the cases. Other sites include meninges (27%), abdominal cavity (20%), trunk (10%), extremities (8%), and the head and neck region (5%) [7]. SFTs in the head and neck region are usually uncommon, and their characteristics are unclear [8]. Among the SFTs in head neck regions, salivary gland origin specially the submandibular salivary gland and paranasal sinus, was very little documented. Most of the tumours are benign, some might have malignant potential [2] and most are presented as non specific mass effects or sometimes symptomless. Even the radiological finding does not help so much in diagnosis, for that reason SFTs could be misclassified. On the other hand, at the time on operation, it is very much essential to know the correct diagnosis, because SFTs have a chance of recurrence, so complete removal of the tumour is mandatory. For that reason, the knowledge about the different unusual sites of SFT is very essential.

In this context, the present study presents three cases of SFT of unusual site of occurrences.

Case 1

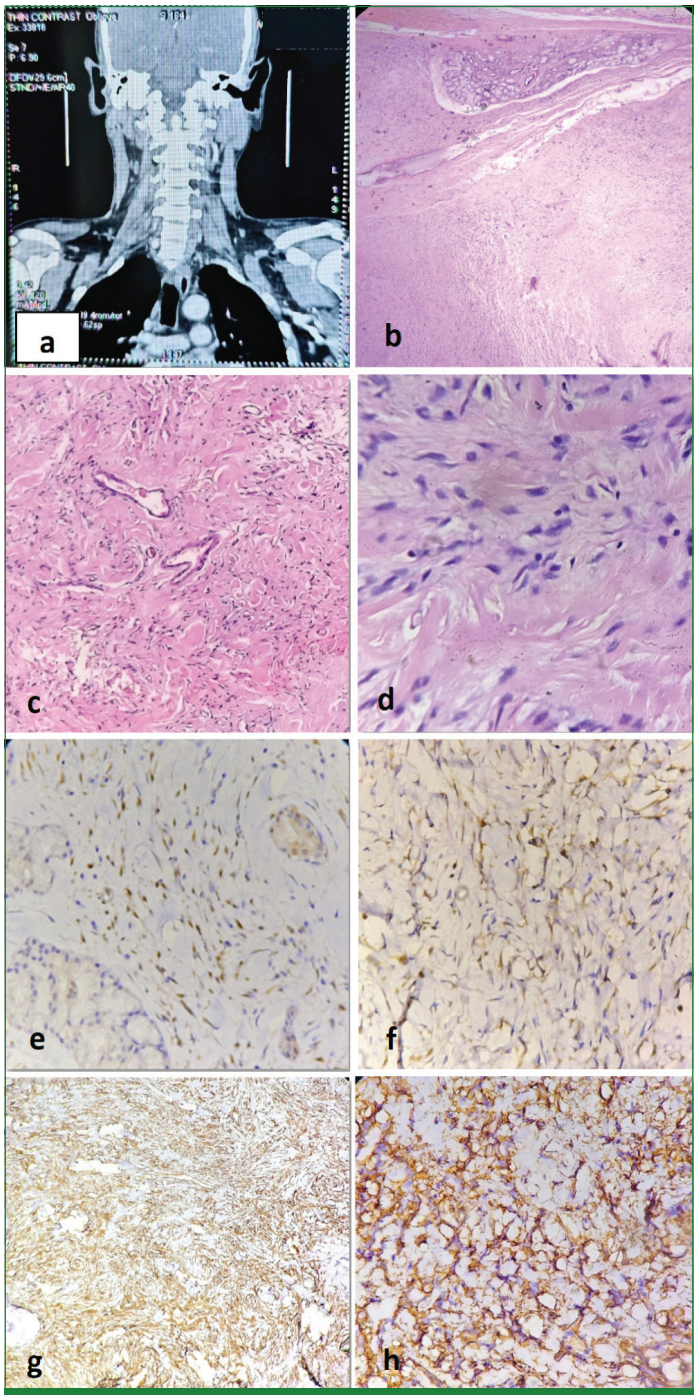
A 32-year-old male presented to the general surgery Outpatient Department (OPD) with a painless swelling in right submandibular region since last one year. The swelling was a firm mass, with no overlying skin changes. The facial nerve function was normal, and there were no other palpable lumps on examination of his neck.

Preoperative Computed Tomography (CT) scan of the neck showed a 5.5×4×2 cm fairly well marginated homogeneously enhancing lesion within the right submandibular gland, without any central necrosis or adjacent stranding, and based on the radiological features, it was provisionally diagnosed to be pleomorphic adenoma of the right submandibular gland [Table/Fig-1a].

After surgical excision of the gland, it was sent for histopathology. Grossly, the tumour was well circumscribed, but showed an irregular capsule, with pseudopodial extensions of the process into the adjacent submandibular gland parenchyma.

On microscopy, sections showed a tumour having hypocellular and hypercellular areas. The tumour composed of a variegated cellular mesenchymal proliferation of bland spindle-shaped cells, lacking any pattern of growth and associated with "ropey" keloidal collagen bundles and delicate, interlaced thin-walled vascular spaces. First, there was a population of densely packed, short fascicles of uniform spindled cell population with very ill-defined cell boundaries, giving a syncytial appearance, having ovoid, vesicular nuclei with small nucleoli. Mitoses were noted, with two per 10 high power fields, atypical mitoses were not found. The second population yielded a more heavily collagenised appearance with similar spindled cell population, but associated with a branching staghorn like vasculature, creating open patulous spaces. The two components were intimately blended with one another, in a background of wiry or ropy unevenly distributed stromal collagen. No destructive growth, necrosis or cellular pleomorphism was noted [Table/Fig-1b-d]. The surgical margins were free from tumour. On the basis of morphology a provisional diagnosis of benign SFT was done, but immunohistochemistry was done to

confirm the diagnosis and to ruled out other possibilities like nerve sheath tumours, myoepithelial tumours and salivary gland tumours. Immunohistochemistry was done and it was positive for CD34, BCL2, CD99 and diffuse nuclear STAT6 positivity which is most specific. The tumour was negative for S100, P63, SMA and had a Ki-67 index of 2% [Table/Fig-1e-h]. A diagnosis of benign SFT of submandibular gland was made based on histology and IHC.



[Table/Fig-1]: a) Preoperative CT scan image of the neck showing a 5.5×4×2 cm fairly well marginated homogeneously enhancing lesion within the right submandibular gland, without any central necrosis or adjacent stranding. b) Photomicrograph (10x) showing a well circumscribed biphasic lesion with an area of normal salivary gland tissue. c) Photomicrograph (100x) showing spindle shaped cells arranged in haphazard fascicles and staghorn blood vessels. d) Photomicrograph (400x) showing uniform spindled cell population with very ill-defined cell boundaries, giving a syncytial appearance, having ovoid, vesicular nuclei with small nucleoli. e) Photomicrograph (100x) showing STAT6 positivity. f) Photomicrograph (100x) showing BCL2 positivity. g) Photomicrograph (100x) showing CD34 positivity. h) Photomicrograph (100x) showing CD99 positivity.

After surgical excision the patient was followed up for four years and at the time of follow-up no recurrence was noticed till date.

Case 2

A 41-year-old male presented to the general surgery OPD with a firm swelling over the right-side of his nose, with tenderness in the area along with tenderness in his right orbit and forehead, associated with epistaxis from his right nostril and difficulty in breathing for eight months. Externally, there was no overlying skin changes. The facial nerve function was normal, and there were no other palpable lumps on examination of his neck. He had first had these symptoms in 2018, for which he was previously operated and a diagnosis of benign spindle cell tumour was made at that time and advised for immunohistochemistry, but patient could not afford the cost. Currently, he was visited to our general surgery OPD in 2024.

Preoperative CT scan of paranasal sinus showed features of post operative multicompartment recurrence of tumour. Magnetic Resonance Imaging (MRI) of face was grossly unremarkable without any enlarged submental or submandibular nodes. MRI of paranasal sinus revealed an enhancing mass lesion of 5×4×2 cm dimension within posterior nasal cavity arising from maxillary sinus, insinuating within right pterygomaxillary fissure and obliterating airway passages. Posterior nasal septum was deviated towards the left. MRI cerebral angiogram showed no obvious anomaly [Table/Fig-2a].

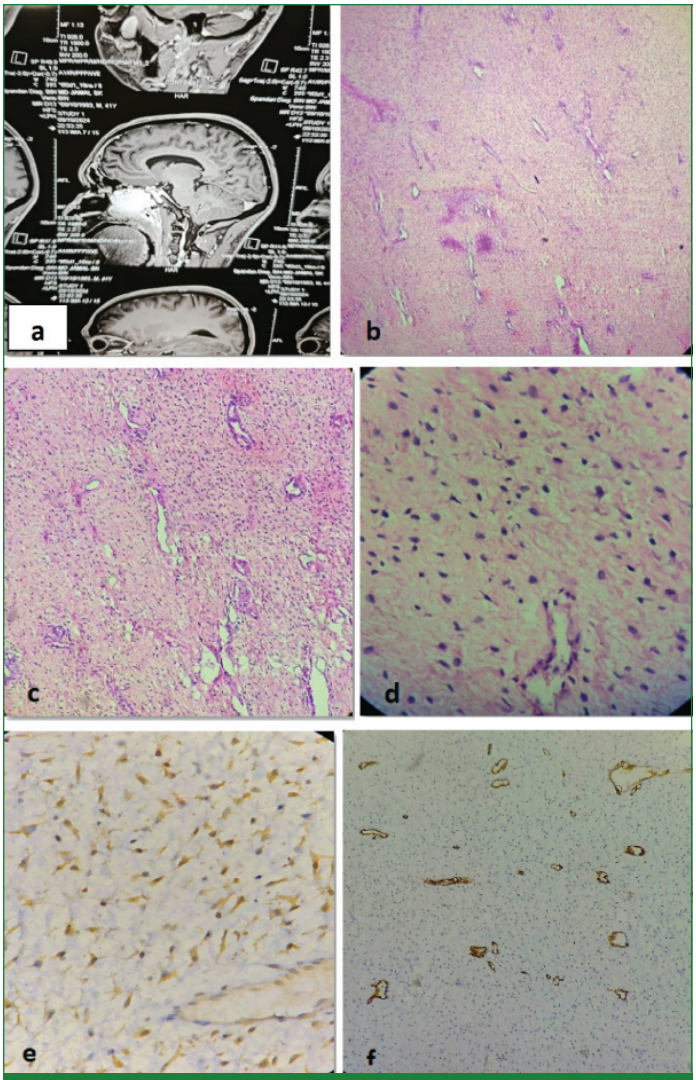
After a wide surgical excision, the excised mass was sent for histopathology. Grossly, three irregular well circumscribed tissue pieces were received, the largest one measured (3×2.5×2) cm and the other two pieces measured (3×2×1) cm each. On cut section, all the tissue pieces appeared to be homogeneously greyish with a few haemorrhagic areas.

Microscopically, sections showed a neoplasm, composed of bland spindle-shaped cells, without any pattern of growth amidst plenty of "ropey" keloidal collagen bundles and delicate, interlaced thin-walled blood vessels. The tumour was biphasic in appearance, consisting of a hypercellular and a hypocellular component, both blended into each other in a background of unevenly distributed collagen. There was a population of densely packed, short fascicles of uniform spindled cell population with very ill-defined cell boundaries with ovoid, vesicular nuclei and small nucleoli. Mitoses were noted, with 10 per 10 high power fields. The second population was more hypocellular, showing a more heavily collagenised appearance with similar spindled cell population, and branching staghorn like vasculature, creating open patulous spaces. Some rare tumour giant cells were noted. No atypical mitoses were noted [Table/Fig-2b-d]. No granuloma, destructive growth, necrosis or cellular pleomorphism were noted. Comment on surgical margins was not possible as fragmented tissues were received. But the marginal status is one the strongest predictors of recurrence in SFT. A provisional diagnosis of malignant SFT was done, but immunohistochemistry was done to confirm the diagnosis and to rule out other differential diagnosis like sinonasal glomangiopericytoma, nasopharyngeal angiofibroma, leiomyoma and angioleiomyoma, nerve sheath tumours, myoepithelial tumours and minor salivary gland tumours. IHC was done and it was positive for CD34, diffuse nuclear STAT6 positivity, BCL2, CD99, negative for S100, P63, SMA and had a Ki-67 index of 12% [Table/Fig-2e,f]. A diagnosis of malignant SFT of the sinonasal SFT was made based on histology and immunohistochemistry.

The patient was called for a follow-up of the case three months after his recent most surgery, as the marginal status of the tumour was not determined, and he presented with a swelling that has recurred in the same area associated with tenderness of the adjoining areas as before. However, he does not complain of epistaxis or difficulty in breathing.

Case 3

A 62-year-old male presented to the Department of General Surgery with a swelling over the calf of his left leg for one year. The swelling was slowly growing, and measured 4 cm in maximum diameter. It was non tender on palpation, not fixed to the underlying structures and no skin changes were noted over the swelling. The data was collected from hospital record.

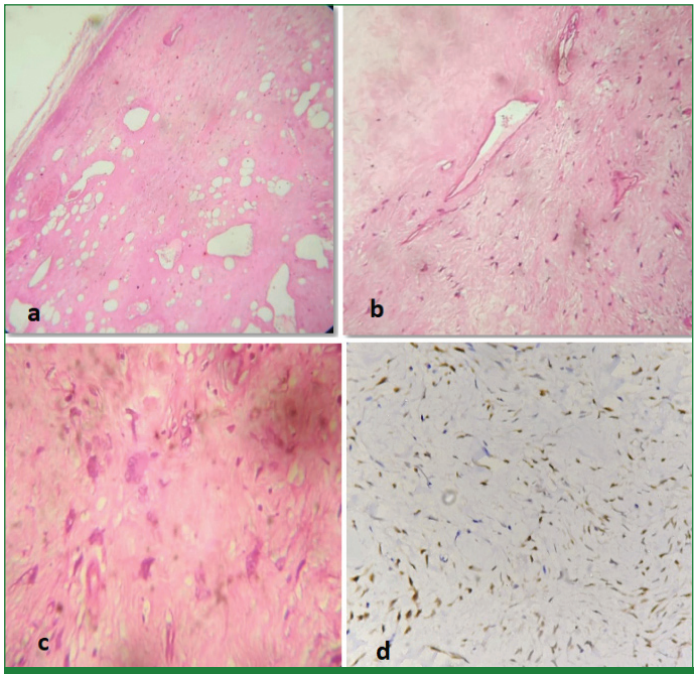


[Table/Fig-2]: a) MRI of paranasal sinus showing well marginated enhancing mass lesion within posterior nasal cavity insinuating within right pterygomaxillary fissure and obliterating airway passages. b) Photomicrograph (10x) showing hypocellular and hypercellular areas. c) Photomicrograph (100x) showing staghorn blood vessels and haphazardly arranged short fascicles of spindle shaped cells. d) Photomicrograph (400x) showing uniform spindled cells with bland eosinophilic cytoplasm and vesicular nuclei with small nucleoli. e) Photomicrograph (400x) showing STAT6 positivity. f) Photomicrograph (100x) showing CD34 positivity.

Preoperative USG imaging revealed 4×4×2 cm fairly well marginated homogeneously enhancing lesion without any central necrosis or adjacent stranding (the imaging plates have not been preserved by the patient). Fine needle aspiration cytology shows cellular smears. The smears were composed of spindle shaped cells in cohesive clusters and loose cells in background and it was provisionally diagnosed to be a spindle cell tumour.

Postsurgery, the specimen was sent for histopathology to the department of pathology, preserved in 10% buffered formalin. Grossly, an irregular well circumscribed tissue piece was received measuring 4×4×2 cm. On cut-section, the tissue piece appeared to be homogeneously greyish with a few haemorrhagic areas.

Microscopically, sections showed a neoplasm having hypocellular and hypercellular areas. The tumour consisting of a hypercellular and a hypocellular component, both blending into each other in a background of unevenly distributed ropey collagen. The tumour was composed of bland spindle-shaped cells, without any pattern of growth, branching staghorn like blood vessels. The hypercellular areas comprised of densely packed, short fascicles of uniform spindled cell population with very ill-defined cell boundaries with ovoid, vesicular nuclei and small nucleoli. The second population was more hypocellular, showing a more heavily collagenised appearance with similar spindled cell population, and branching staghorn like vasculature, creating open patulous spaces. No atypical mitoses were noted. No granuloma, destructive growth, necrosis or cellular pleomorphism was noted [Table/Fig-3a-c]. The surgical margins were free from tumour. On the basic of morphology, a provisional diagnosis of benign SFT was done, differential diagnosis of peripheral nerve sheath tumours, dermatofibrosarcoma protuberans also kept in mind, but immunohistochemistry was done to confirm the diagnosis and it was positive for CD34, BCL2, CD99 and diffuse nuclear STAT6 positivity which is most specific, along with negative for S100, P63, SMA and had a Ki-67 index of 2% [Table/Fig-3d]. A diagnosis of benign SFT was made based on histology and IHC. Patient was coming for postoperative follow-up for three years that was uneventful.



[Table/Fig-3]: a) Photomicrograph (10x) showing hypocellular and hypercellular areas. b) Photomicrograph (100x) showing cystically dilated blood vessels. c) Photomicrograph (400x) showing bland spindle-shaped cells and giant cells in a collagenous background. d) Photomicrograph (100x) showing STAT6 positivity.

DISCUSSION

The SFT is a rare tumour and has an uncertain biological character. Previously, it was thought that SFT is a pleural base tumour and originated from mesothelial cells, hence the old name was fibrous mesothelioma. But now it is evident that SFT can occur at various extra pleural sites, it is assumed that SFT has a mesenchymal origin rather than mesothelial. Although in recent time immunohistochemistry and molecular study re-categorised them as mesenchymal tumour.

Most of the SFTs are benign, only 10-15% show malignant features [9]. It typically occurs in adults with a wide age range between 19-79 years. Males and females are shown equal incidence rate [10].

In the literature, it was documented that the site of origins of extra pleural SFTs are variable, like meninges (27%), abdominal cavity (20%), trunk (10%), extremities (8%), and the head and neck region (5%) [7]. In the head neck region, it is documented in orbits, oral cavity, larynx, nasopharynx, salivary glands and paranasal sinuses [11]. SFTs of head neck region mostly affect the orbit, oral cavity [10] among the major salivary gland SFTs of parotid gland are well documented in the literatures [12], but there are few literatures described the SFT involving the submandibular salivary gland. A review of the literature identified six previously reported cases of submandibular gland SFT spanning 2002-2025. Shi W et al., and Garousi M et al., were documented SFTs in the submandibular region in a third decade of life, mimicking salivary neoplasms clinically and radiographically [11,13]. The article report by Hofmann T et al., described a submandibular gland SFT in a 45-year-old male, diagnosed using CD34, Bcl-2, CD99, and vimentin [14]. At that time, distinction from haemangiopericytoma was a recognised diagnostic dilemma, as highlighted by Garg D et al., in a 14-year-old

female with a submandibular mass [15]. Husain B and Gonzalez M highlighted that fine-needle aspiration of submandibular SFT often shows bland spindle cells with myxoid stroma, closely mimicking pleomorphic adenoma and leading to potential misdiagnosis [16]. Their case demonstrated that STAT6 immunohistochemistry on cell block material is essential to avoid surgical under-treatment. Hasyim NA et al., described a dedifferentiated submandibular SFT in a 62-year-old male, measuring 6.5 cm with necrosis, mitotic rate >4/10 HPF, and Ki-67 of 20% [17].

Among head neck SFTs paranasal sinus involvement also not common, according to Sing VK et al., it accounts for around, 0.1% of all the cases [18]. Thompson LDR et al. reported six cases of sinonasal solitary fibrous tumor and reviewed 86 previously reported cases from the literature [19]. Although the majority of sinonasal SFTs behave in a clinically indolent manner, the literature indicates that a subset may demonstrate aggressive behavior. Among 72 reported cases with available follow-up, five patients developed local recurrence at 3-69 months after resection, although no cases of metastasis or disease-related death were reported [19]. This spectrum is further exemplified by Wu H et al., who described a large sinonasal SFT with intracranial extension and reviewed 67 cases that local recurrences were observed in five patients [20]. Given this scenario the present study describes three cases of SFT affected submandibular gland, a recurrent sinonasal SFT another case involving leg.

The SFT is a benign tumour so most common clinical presentation is slow growing mass and causes pressure effects in surrounding structures [17]. In this series first and third cases show similar type of presentation. According to Alobid I et al., sinonasal SFTs most commonly presented with unilateral mass, nasal obstruction, rhinorrhoea, epistaxis, and exophthalmos [21]. In present study, it has history of recurrence.

Imaging examination like CT or MRI is the primary diagnostic process. On CT SFTs appears as a uniform, isointense or hypointense space occupying lesion, but does not give any specific clue for diagnosis. As our one case was diagnosed radiologically as pleomorphic adenoma.

Histopathological examination with immunohistochemistry is very much essential for diagnosis. Histologically pleural and extra pleural SFTs show similar type of microscopical features, the tumour composed of a variegated cellular mesenchymal proliferation of bland spindle-shaped cells, lacking any pattern of growth and associated with “ropey” keloidal collagen bundles and delicate, interlaced thin-walled vascular spaces. One population of densely packed, short fascicles of uniform spindled cell population with very ill-defined cell boundaries, giving a syncytial appearance, having ovoid, vesicular nuclei with small nucleoli. Mitotic count is variable, atypical mitoses were not found. The other population yielded a more heavily collagenised appearance with similar spindled cell population, but associated with a branching staghorn like vasculature, creating open patulous spaces. Some extravasated erythrocytes, mast cells and rare tumour giant cells were noted. The two components are intimately blended with one another, in a background of wiry or ropy unevenly distributed stromal collagen. No destructive growth, necrosis or cellular pleomorphism are noted. The submandibular SFT in current series, shows conventional histological features. Sometime SFTs may show multicystic degeneration. The present study describes one of such patterns in SFT involving the leg. The sinonasal SFT in current series shows microscopical similarity to malignant SFTs, as they exhibit increased mitotic count (10 per 10 high power fields). Roy S et al., described a recurrent sinonasal SFT with proliferating index of 8% [22].

The above-mentioned histopathological features may be confused with other tumours in variable locations, like in submandibular region the probable differential diagnosis are peripheral nerve sheath tumours, myoepithelial tumours and different salivary gland tumours, in paranasal sinuses differential diagnosis are sinonasal glomangiopericytoma, nasopharyngeal angiofibroma, leiomyoma and angioleiomyoma, peripheral nerve sheath tumours such as schwannoma and neurofibroma and in the leg it should be differentiated from peripheral nerve sheath tumours, fibrous histiocytoma, dermatofibrosarcoma protuberans, leiomyosarcoma and synovial sarcoma.

As the SFTs showing variable morphological features due to its wide differential diagnosis Immunohistochemical studies are very much essential to confirm the diagnosis. SFTs generally show positivity for CD34, CD99, and BCL2 [23]. However, a small proportion of SFTs may be negative for CD34 and also a recurrent sinonasal SFTs can show less CD34 immunoreactivity [24]. But the above markers are not specific for SFT. The diffuse nuclear STAT6 positivity is the most sensitive and specific, and it gets expressed even in malignant cases [25], this study follows the same pattern, all the cases show diffuse nuclear STAT6 staining and CD34, BCL2, CD99 positivity.

The diagnosis usually made by histopathological and immunohistochemical examination, but molecular genetic study is useful for final confirmation of the diagnosis. The NAB2-STAT6 fusion resulting from chromosome 12q13 inversion is the defining molecular alteration in SFT across all anatomic sites. This fusion generates a chimeric oncoprotein that converts NAB2 from a transcriptional repressor to a constitutive activator, driving tumour development. Importantly, detection of NAB2-STAT6 or nuclear STAT6 expression provides a reliable diagnostic marker. Therefore, molecular and immunohistochemical assessment for NAB2-STAT6 should be standard in the workup of suspected SFT [26].

Complete surgical removal with clear margins is the treatment of choice for the SFTs [27,28]. As this tumour has a 10-30% chance of recurrence. The local recurrence is common in tumours more than 10 cm in diameter, incomplete surgical removal, presence of necrosis, mitotic count more than 4 per high power field and Ki 67 index [29]. In this case series 2nd case presented with malignant sinonasal SFTs has history of recurrence, its mitotic count and Ki-67 index is also high.

Histologic criteria for malignancy in SFT remain imperfectly defined. The World Health Organisation classifies SFT as malignant based on hypercellularity, increased mitoses (>4 mitoses per 10 high-power fields), cytologic atypia, tumour necrosis, and/or infiltrative margins [30]. Many of these parameters are significantly associated with higher recurrence risk and decreased survival [19]. However, histology alone is an imperfect predictor of clinical behaviour: tumours classified as malignant by morphology may behave indolently, while recurrence and metastasis are well documented in SFTs lacking atypical histologic features. To address this discordance, several risk stratification models have been proposed. Beyond histology, patient age ≥55 years, tumour size >10 or ≥15 cm, and positive margin status have demonstrated utility in predicting outcome. Demicco EG et al., developed a 3-tier model incorporating age, tumour size, and mitotic index to stratify SFTs into low, intermediate, and high-risk for metastasis and death [31]. Margin status is critical in SFT management. The conventional submandibular and leg SFTs, in this series, were typically well-circumscribed with complete resection achieved, but according to Wu H et al., sinonasal SFT presented with large size making complete resection challenging [20]. This study follows the similar pattern in the 2nd case.

Therefore, regular follow-up with clinical examination and imaging is essential for all SFTs, at least for first 3-5 years postsurgery, and may be longer for high-risk lesions [32].

CONCLUSION(S)

Despite its rarity, SFT poses diagnostic challenges due to marked clinical and histopathological variations. In resource-limited settings, inadequate access to advanced imaging, immunohistochemistry, and molecular diagnostics contributes to delays that worsen prognosis. Given the risk of recurrence even after complete resection, standardised imaging surveillance protocols are essential. Integrating referral networks, long-term follow-up, and research collaboration

can reduce disparities and ensure equitable access to diagnosis, treatment, and long-term care. Prioritising these measures is a key to improving outcomes for patients with SFT.

Acknowledgement

The authors acknowledge the Department of Surgery, Institute of Post Graduate Medical Education and Research (IPGMER) and SSKM Hospital; Department of Pathology, IPGMER and SSKM Hospital for their support.

REFERENCES

[1] Briselli M, Mark EJ, Dickersin GR. Solitary fibrous tumours of the pleura: eight new cases and review of 360 cases in the literature. Cancer. 1981;47:2678-89.
[2] Olson NJ, Linos K. Dedifferentiated solitary fibrous tumour: a concise review. Arch Pathol Lab Med. 2018;142:761-66.
[3] Wagner E. Das tuberkelahnliche lymphadenom (der cytogene oder reticulirte tuberkel). Arch Heilk (Leipzig). 1870;11:497.
[4] Klemperer P, Rabin CB. Primary neoplasms of the pleura: A report of five cases. Arch Pathol. 1931;11:385-412.
[5] Rodriguez-Gil Y, Gonzalez MA, Carcavilla CB, Santamaria JS. Lines of cell differentiation in solitary fibrous tumour: an ultrastructural and immunohistochemical study of 10 cases. Ultrastruct Pathol. 2009;33:274-85.
[6] Fletcher CD. The evolving classification of soft-tissue tumours: an update based on the new WHO classification. Histopathology. 2006;48:3-12.
[7] Ronchi A, Cozzolino I, Zito Marino F, Accardo M, Montella M, Panarese I, et al. Extrapleural solitary fibrous tumour: A distinct entity from pleural solitary fibrous tumour. An update on clinical, molecular and diagnostic features. Ann Diagn Pathol. 2018;34:142-50.
[8] Yamada H, Hamada Y, Fujihara H, Fukami K, Mishima K, Nakaoka K, et al. Solitary fibrous tumour of the buccal space resected in combination with coronoidectomy. Oral Surg Oral Med Oral Pathol Oral Radiol. 2012;114(1):e9-e14.
[9] Wang X, Qian J, Bi Y, Ping B, Zhang R. Malignant transformation of orbital solitary fibrous tumour. Int Ophthalmol. 2013;33:299-303.
[10] AlQahtani SM, Alabdulaal MR, Aljubran HJ, Almomen A. Solitary fibrous tumour of the nasal septum: a case report and literature review. Journal of Surgical Case Reports. 2025;2025(7):rjaf556.
[11] Shi W, Wei Z. Solitary fibrous tumour of the submandibular region. Oncol Lett. 2015;9(2):984-86.
[12] Bauer JL, Miklos AZ, Thompson LD. Parotid gland solitary fibrous tumour: a case report and clinicopathologic review of 22 cases from the literature. Head Neck Pathol. 2012;6(1):21-31.
[13] Garousi M, Gandomkar H, Maleki M, Erteghaee F, Yazdani F, Ahmadian R. Solitary fibrous tumours of the submandibular gland: A case report. Clin Case Rep. 2025;13(12):e71665.
[14] Hofmann T, Braun H, Köle W, Beham A. Solitary fibrous tumour of the submandibular gland. Eur Arch Otorhinolaryngol. 2002;259:470-73.
[15] Garg D, Palaskar S, Shetty VP, Bhushan A, Bhatia P, Sarin J. Diagnostic dilemma: solitary fibrous tumour or hemangiopericytoma of the submandibular region in a patient with multiple odontogenic keratocysts. J Pediatr Oncol Nurs. 2009;26:136-41.
[16] Husain B, Gonzalez M. Solitary fibrous tumour of the submandibular gland: Novel insights from clinical practice on a close mimicker of pleomorphic adenoma and a diagnostic challenge for the cytopathologist. Cytopathology. 2021;32(2):261-65.
[17] Hasyim NA, Juneja M, Abraham MT, Kallarakkal TG. Dedifferentiated solitary fibrous tumour of submandibular gland. Indian J Pathol Microbiol. 2025;68(1):228-30. Doi: 10.4103/ijpm.ijpm_736_22.
[18] Singh VK, Mathew M, Kudva R, Pai K, Vijay A. Sinonasal solitary fibrous tumour: A borderline entity with diagnostic challenges. Int J Clin Diagn Pathol. 2019;2(1):316-20.
[19] Thompson LDR, Lau SK. Sinonasal tract solitary fibrous tumour: a clinicopathologic study of six cases with a comprehensive review of the literature. Head Neck Pathol. 2018;12(4):471-80. Doi: 10.1007/s12105-017-0878-y. Epub 2017 Dec 27. PMID: 29282671; PMCID: PMC6232205.
[20] Wu H, Hu X, Xiang W, Liu N, Fang S, Xu T, et al. A large solitary fibrous tumour of the nasal cavity and paranasal sinuses involving the anterior frontal fossa: a case report and review of the literature. Ear Nose Throat J. 2025;104(1_suppl):42S-50S.
[21] Alobid I, Alós L, Blanch JL, et al. Solitary fibrous tumour of the nasal cavity and paranasal sinuses. Acta Otolaryngol. 2003;123(1):71-74.
[22] Roy S, Mallick S, Kakkar A, Jana M, Julka PK. Recurrent malignant sino-nasal solitary fibrous tumour: Eliminate the enemy at the first instance. J Can Res Ther. 2015;11:650.
[23] Lee CK, Liu KL, Huang SK. A dedifferentiated solitary fibrous tumour of the parotid gland: a case report with Cytopathologic findings and review of the literature. Diagn Pathol. 2019;14(1):20.
[24] Kakkar A, Sakthivel P, Rajeshwari M, Kairo A, Sharma MC. Recurrent sinonasal CD34-negative malignant solitary fibrous tumour diagnosed on STAT6 immunohistochemistry and NAB2-STAT6 fusion. Head and Neck Pathol. 2020;14:250-56.
[25] Doyle LA, Vivero M, Fletcher CD, Mertens F, Hornick JL. Nuclear expression of STAT6 distinguishes solitary fibrous tumour from histologic mimics. Mod Pathol. 2014;27(3):390-95.
[26] Ren C, D'Amato G, Hornicek FJ, Tao H, Duan Z. Advances in the molecular biology of the solitary fibrous tumour and potential impact on clinical applications. Cancer Metastasis Rev. 2024;43(4):1337-52.
[27] Ridder GJ, Kayser G, Teszler CB, Pfeiffer J. Solitary fibrous tumours in the head and neck: new insights and implications for diagnosis and treatment. Ann Otol Rhinol Laryngol. 2007;116(4):265-70.
[28] Rais M, Kessab A, Sayad Z, El Mourabit S, Zrarqi R, Benazzou S, et al. Solitary fibrous tumour occurring in the parotid gland: a case report. BMC Clin Pathol. 2017;17:22.
[29] Georgiesh T, Boye K, Bjerkehagen B. A novel risk score to predict early and late recurrence in solitary fibrous tumour. Histopathology. 2020;77(1):123-32.
[30] Fletcher CDM, Bridge JA, Lee J-C. Extrapleural solitary fibrous tumour. In: Fletcher CDM, Bridge JA, Hogendoorn PCW, Mertens F, editors. WHO Classification of tumours of soft-tissue and bone. Lyon: IARC; 2013. pp. 80-82.
[31] Demicco EG, Park MS, Araujo DM, Fox PS, Bassett RL, Pollock RE, et al. Solitary fibrous tumour: a clinicopathological study of 110 cases and proposed risk assessment model. Mod Pathol. 2012;25(9):1298-306.
[32] Holley ZC, Halbach A, Steiniger J, Rivera M. Solitary fibrous tumour of the parotid gland: A case report highlighting challenges in management. ACS Case Reviews in Surgery. 2025;5(5):45-51.

PARTICULARS OF CONTRIBUTORS:
1. Third Year PGT, Department of Pathology, Institute of Post Graduate Medical Education and Research, Kolkata, West Bengal, India.
2. Second Year PGT, Department of Pathology, Institute of Post Graduate Medical Education and Research, Kolkata, West Bengal, India.
3. Associate Professor, Department of Pathology, Institute of Post Graduate Medical Education and Research, Kolkata, West Bengal, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Biswanath Paul,
22/2, Umesh Mukherjee Road, P.O- Belgharia-700056, Kolkata,
West Bengal, India.
E-mail: paul.biswanath1@gmail.com

PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Mar 06, 2026
- Manual Googling: Jul 02, 2026
- iThenticate Software: Jul 06, 2026 (1%)

ETYMOLOGY: Author Origin

EMENDATIONS: 7

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was informed consent obtained from the subjects involved in the study? NA (retrospective data from records was used)
- For any images presented appropriate consent has been obtained from the subjects. NA

Date of Submission: **Mar 05, 2025**
Date of Peer Review: **Apr 17, 2026**
Date of Acceptance: **Jul 07, 2026**
Date of Publishing: **Oct 01, 2026**