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Case Presentation

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Solitary Fibrous Tumor of the Nasal Cavity: A Case Report and Review of the Literature

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Abstract

Background: Solitary fibrous tumors (SFTs) are rare mesenchymal neoplasms that infrequently involve the sinonasal tract. Their diagnosis is challenging owing to nonspecific clinical and radiological features, and definitive identification relies on histopathological examination combined with nuclear STAT6 immunohistochemistry, the molecular marker arising from the pathognomonic NAB2–STAT6 gene fusion.

Case report: We report the case of a 51-year-old man presenting with long-standing left-sided nasal obstruction. Endoscopic examination and computed tomography revealed a well-circumscribed 42-mm soft-tissue mass confined to the left nasal cavity without bone destruction or sinus invasion. Complete endoscopic endonasal resection was performed. Histopathological analysis demonstrated a spindle-cell proliferation with staghorn-type vessels and collagenous stroma. Immunohistochemistry confirmed diffuse nuclear STAT6 positivity and CD34 expression, with negativity for S-100, SOX10, desmin, and cytokeratins. Risk stratification according to the Demicco model yielded a score of 1 (low risk). The postoperative course was uneventful, and the patient remains under regular surveillance.

Conclusion: This case illustrates the diagnostic approach, histopathological features, and surgical management of sinonasal SFT. Nuclear STAT6 expression is the cornerstone of diagnosis, enabling reliable distinction from histological mimickers. Complete surgical excision is the treatment of choice, and prolonged follow-up is warranted even in low-risk cases given the propensity for late recurrence.

Keywords: Solitary fibrous tumor; Nasal cavity; Sinonasal; STAT6; NAB2–STAT6 fusion; Endoscopic resection; Demicco risk model

Introduction

Solitary Fibrous Tumors (SFTs) are rare mesenchymal neoplasms of fibroblastic origin, first described as primary tumors of the pleura by Klemperer and Rabin in 1931 [1]. Originally conceptualized as exclusively pleural lesions, SFTs are now recognized as ubiquitous tumors capable of arising at virtually any anatomical site throughout the body [2]. Their molecular hallmark is an intrachromosomal inversion on chromosome 12q13 resulting in a NAB2–STAT6 gene fusion, which was identified as the defining driver mutation of SFT in 2013 [3,4]. This fusion converts the transcriptional repressor NAB2 into a transcriptional activator that deregulates EGR1-responsive genes, driving tumor proliferation [4].

Head and neck localizations account for approximately 5–27% of all extrapleural SFTs, with the orbit and oral cavity representing preferred sites within this region [5]. Involvement of the sinonasal tract is considerably rarer, representing fewer than 0.1% of sinonasal tumors [5]. Fewer than 100 cases of nasal cavity SFTs have been reported in the English-language literature to date [6]. The paucity of cases means that the clinicopathological characteristics, differential diagnosis, and optimal management of sinonasal SFTs remain incompletely defined, resting primarily on isolated case reports and small series [5-7]. Clinical presentation is typically nonspecific, most commonly manifesting as progressive unilateral nasal obstruction and, less frequently, epistaxis, rhinorrhea, or facial pressure [6,8]. Sinonasal SFTs generally present as slow-growing, painless masses; the size reported in the literature ranges from 2.8 to 8 cm in greatest dimension [8]. Radiological findings typically demonstrate a well-circumscribed, homogenously enhancing soft-tissue mass, but lack pathognomonic characteristics sufficient to distinguish SFT from other sinonasal neoplasms [9]. Definitive diagnosis therefore relies on histopathological examination and immunohistochemical confirmation [5,10]. We report a case of solitary fibrous tumor of the nasal cavity and provide a comprehensive review of the current literature with emphasis on diagnosis — including detailed differential diagnosis — management, and prognosis.

Case Presentation

A 51-year-old man with no significant past medical history was referred to our department for evaluation of long-standing, progressive left-sided nasal obstruction. He denied epistaxis, facial pain, headache, anosmia, or visual disturbance. There was no history of prior sinonasal surgery or malignancy. Anterior rhinoscopy and nasal endoscopy revealed a non-necrotic, smooth-surfaced polypoid mass occupying the left nasal cavity, arising from the left olfactory cleft, without surface ulceration or hemorrhage (for a representative endoscopic image from a comparable case, see **Figure 6** in the Discussion section). Contrast-enhanced Computed Tomography (CT) of the paranasal sinuses — performed as a single post-contrast acquisition — demonstrated an expansile soft-tissue process measuring 42 mm in its greatest dimension, occupying the anterior portion of the left nasal cavity **(Figure 1 and 2)** [9]. No associated bony destruction or remodeling was identified. Incidental findings included bilateral chronic maxillary sinusitis, a mild leftward deviation of the nasal septal base, and aplasia of the right frontal sinus. No cervical lymphadenopathy was identified. Magnetic Resonance Imaging (MRI) was not

performed preoperatively, as CT findings indicated a well-circumscribed, localized lesion without aggressive osseous or soft-tissue features.

The patient underwent complete endoscopic endonasal resection of the mass under general anesthesia. Endoscopic approaches are increasingly favored for localized sinonasal SFTs owing to superior visualization, reduced morbidity, and equivalent oncological outcomes compared with open surgery [11,12]. Intraoperative findings confirmed a well-demarcated polypoid mass pedunculated on the left olfactory cleft.

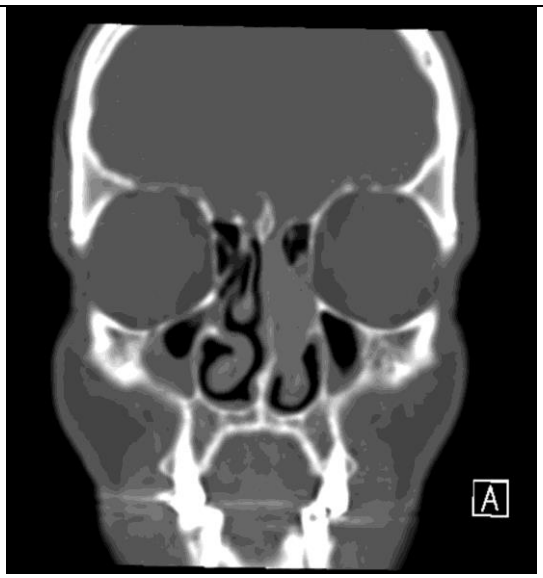

Figure 1: Contrast-enhanced CT scan of the paranasal sinuses, coronal reconstruction. An expansile, homogeneously enhancing soft-tissue mass measuring 42 mm in greatest dimension is demonstrated within the left nasal cavity, without associated bony erosion or sinus invasion. Note the bilateral chronic maxillary sinusitis and the absence of the right frontal sinus.

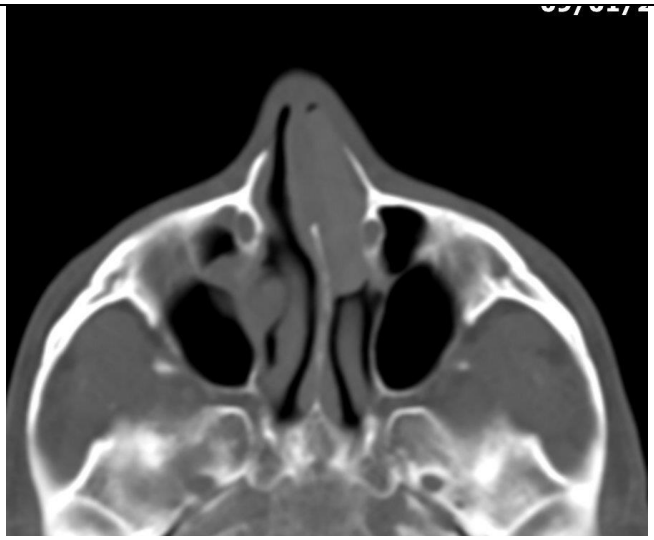

Figure 2: Contrast-enhanced CT scan, axial reconstruction at the level of the nasal cavity. The well-circumscribed, enhancing soft-tissue mass occupies the anterior left nasal fossa without extension across the nasal septum or into the adjacent ethmoid sinuses. No cortical bone disruption is identified.

Gross pathological examination revealed a large polypoidal lesion. Five small additional tissue fragments were also submitted. The specimen was embedded in its entirety (blocks A1–A7). Histopathological examination demonstrated a polypoid lesion surfaced by respiratory mucosa with focal squamous metaplasia and no epithelial atypia. The lamina propria appeared edematous with mild chronic inflammation and scattered mucosecretory glandular structures. The central portion of the specimen harbored a relatively well-demarcated mesenchymal neoplasm composed of multiple intersecting fascicles of monotonous, cytologically bland spindle cells with eosinophilic cytoplasm and ovoid nuclei bearing small nucleoli within fine chromatin **(Figure 3 and 4)**. Blood vessels were dilated, branching, and focally thick-walled or hyalinized — consistent with the characteristic hemangiopericytoma-like ("staghorn") vascular pattern **[5,10-12]**. Collagenous stroma was present between cellular fascicles. No necrosis or mitotic figures were identified in initial sections. The resection margin was in close proximity to the lesion, consistent with an enucleation-type resection.

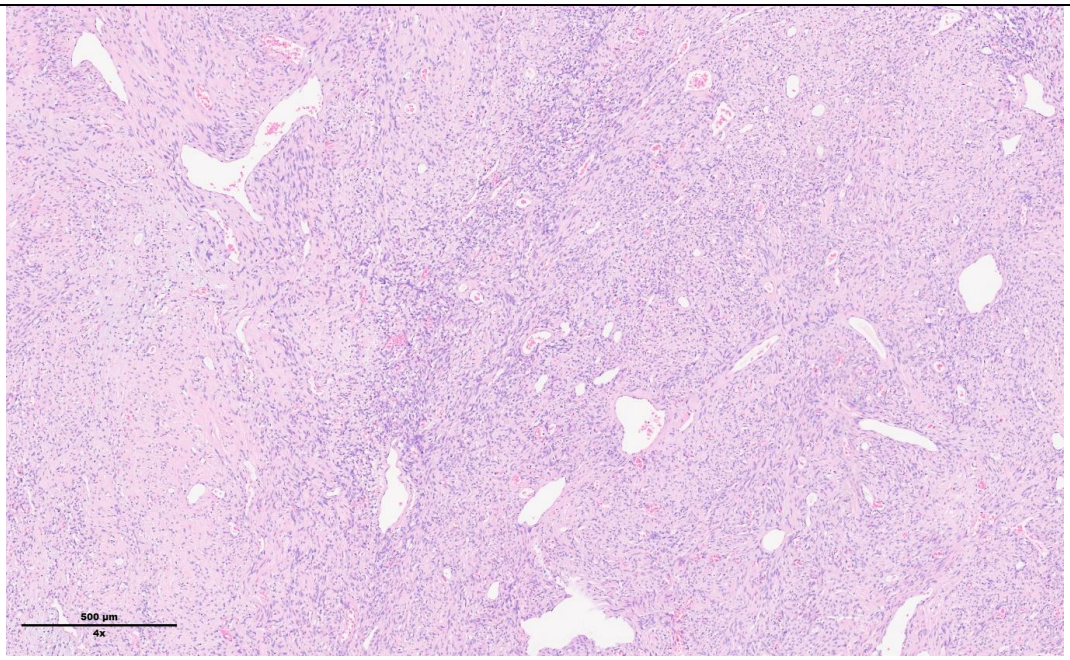

Figure 3: Hematoxylin and eosin (H&E), original magnification ×4. Low-power view demonstrating the characteristic "patternless pattern" of SFT: intersecting fascicles of spindle cells within a collagenous stroma, interspersed with multiple branching, thin-walled vascular channels of varying caliber. Scale bar = 500 µm.

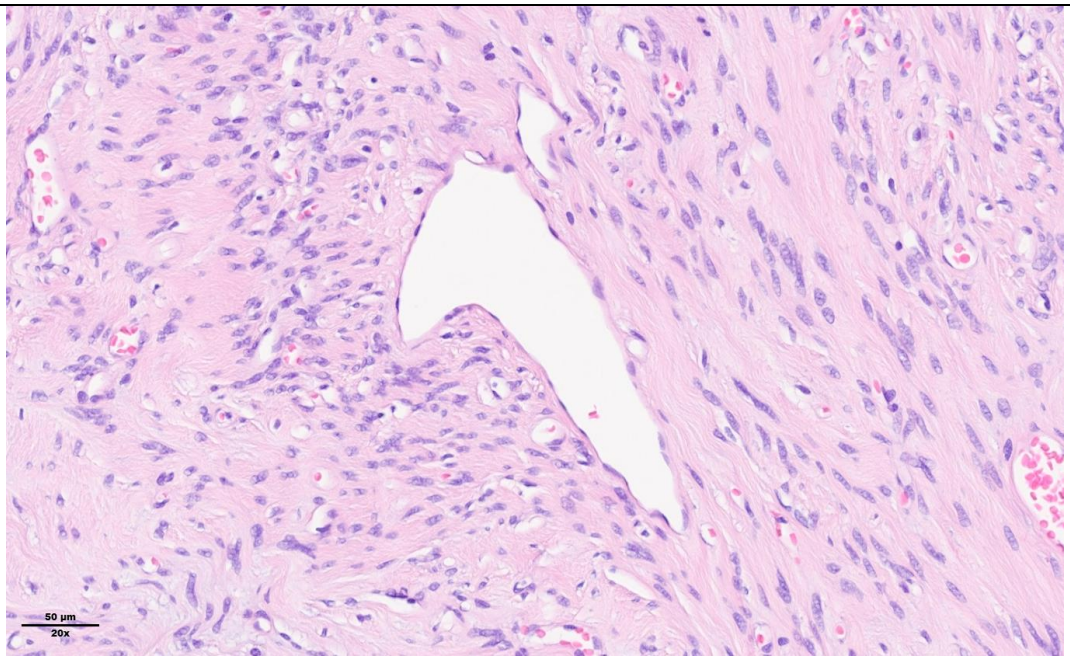

Figure 4: Hematoxylin and eosin (H&E), original magnification ×20. Higher-power view showing cytologically bland, monotonous spindle cells with ovoid nuclei, inconspicuous nucleoli, and finely dispersed chromatin, embedded in collagenous stroma. A prominent, branching, thin-walled hemangiopericytoma-like ("staghorn") vascular channel is evident centrally. No significant nuclear atypia or mitotic figures are identified in this field. Scale bar = 50 µm.

Immunohistochemical analysis demonstrated diffuse, strong nuclear STAT6 expression **(Figure 5)**. CD34 showed cytoplasmic positivity in spindle cells in addition to highlighting vascular structures. Actin (smooth muscle actin) highlighted vascular walls only. Tumor cells were negative for S-100 protein, SOX10, desmin, cytokeratins (AE1/AE3), Androgen Receptor (AR), and beta-catenin. Nuclear STAT6 expression, arising from the NAB2–STAT6 gene fusion, is currently regarded as the most sensitive and specific immunohistochemical marker for SFT, allowing reliable distinction from histological mimickers **[13-16]**. CD34 focal spindle-cell positivity is observed in a majority of SFTs **[5]**. The negativity for beta-catenin and actin effectively excluded sinonasal glomangiopericytoma, while negativity for S-100 and SOX10 excluded schwannoma and biphenotypic sinonasal sarcoma **[17-19]**.

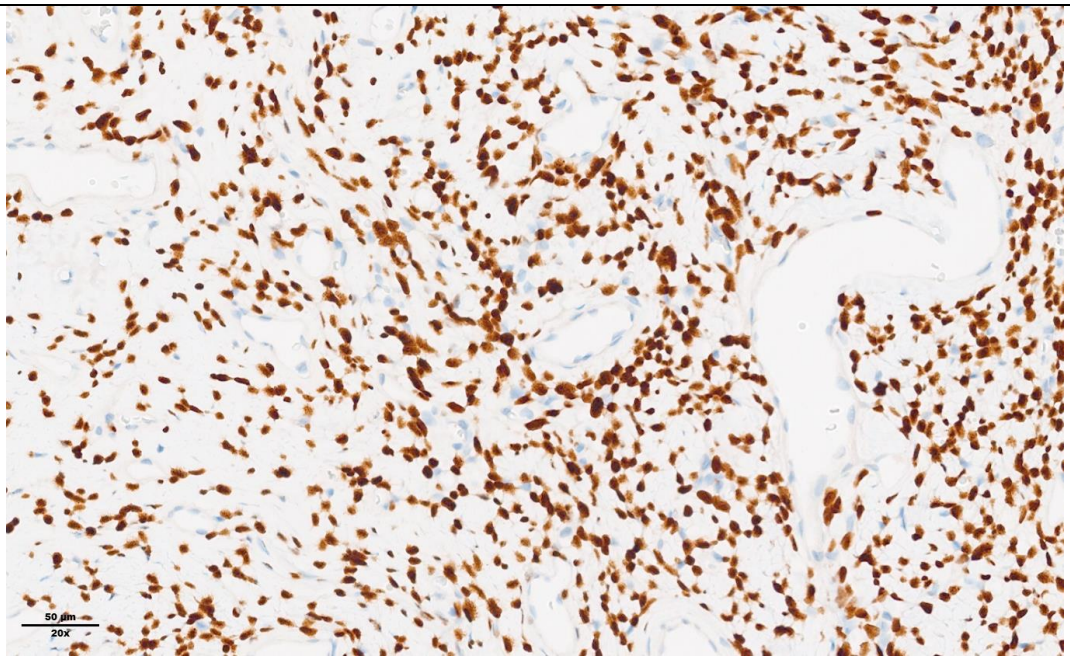

Figure 5: Immunohistochemistry for STAT6 (anti-STAT6 C-terminal antibody), original magnification ×20. Diffuse, intense nuclear brown (DAB) staining is demonstrated in virtually all neoplastic spindle cells, consistent with nuclear accumulation of the NAB2–STAT6 fusion protein resulting from the pathognomonic chromosome 12q13 inversion. Vascular endothelial cells serve as an internal negative control (unstained nuclei). Scale bar = 50 µm.

Risk stratification according to the four-variable Demicco model (2017) [15,16] yielded the following scores: age 51 years (<55 years: 0 points); tumor size 4.2 cm (<5 cm: 0 points); mitotic activity 2 mitotic figures per 10 HPF (1–3/10 HPF: 1 point); tumor necrosis 5–10% (<10%: 0 points). The total score was 1, placing this tumor in the low-risk category (score 0–3) for metastatic potential.[16] It is noteworthy that although the initial macroscopic report described absent necrosis and mitoses, formal quantitative assessment on complete embedding revealed 2 mitotic figures per 10 HPF and focal necrosis estimated at 5–10%, underscoring the importance of complete tumor embedding and thorough histological sampling in SFT risk scoring.

The postoperative course was uneventful. The patient was discharged on postoperative day one. He remains under regular clinical and endoscopic follow-up, with no evidence of local recurrence at the most recent review.

Discussion

Epidemiology and Clinical Features

Sinonasal SFTs represent a particularly rare extrapleural manifestation of this neoplasm [5,6]. Unlike their pleural counterparts, which show a slight female predominance, sinonasal SFTs appear to affect males more frequently, based on the limited available case series [9]. Most patients are adults presenting after the fourth decade of life, consistent with the broad age range reported in the literature (18–79 years) [8]. The clinical presentation is invariably nonspecific: unilateral nasal obstruction is the most frequent symptom, followed by rhinorrhea, epistaxis, and, in cases with extensive local extension, proptosis, epiphora, or facial pain [6,8,12]. Our patient presented with the most common symptom — isolated,

progressive unilateral nasal obstruction — without any features suggesting aggressive behavior.

The characteristic endoscopic appearance of a sinonasal SFT is illustrated in **Figure 6**, which shows a representative case courtesy of Dr. S. Akhtar **[8]**. As observed in our patient, the lesion typically presents as a smooth-surfaced, non-ulcerated polypoid mass occupying the nasal lumen, without surface necrosis or hemorrhage. This benign macroscopic appearance reflects the indolent growth pattern of low-risk tumors but provides no reliable criterion for distinguishing SFT from other unilateral sinonasal masses, underscoring the indispensable role of histopathological examination.

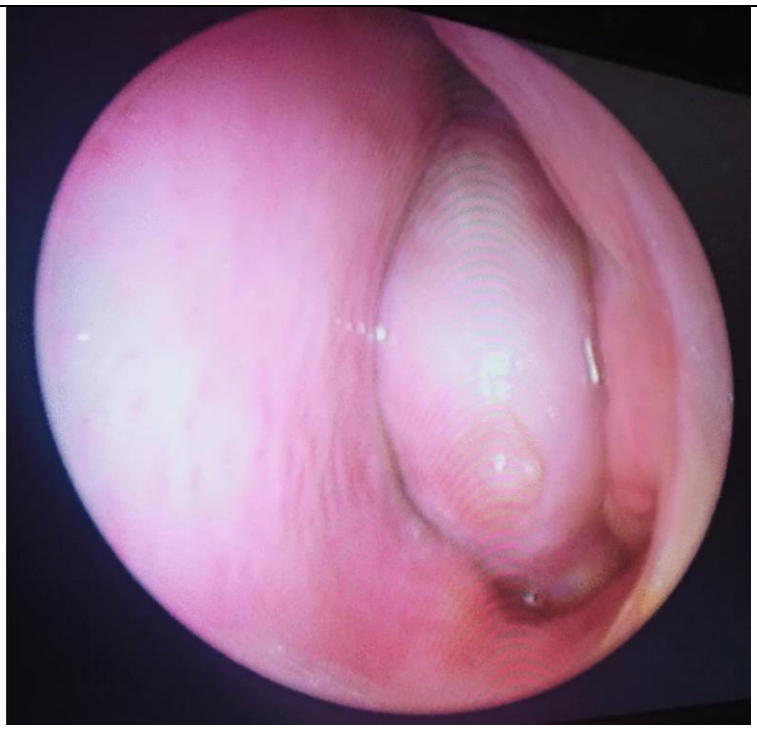

Figure 6: Representative nasal endoscopic view of a solitary fibrous tumor of the left nasal cavity (courtesy of Dr. S. Akhtar, Department of Otorhinolaryngology, Dr. Ziauddin University Hospital, Karachi, Pakistan **[8]**; image obtained from a comparable case, as intraoperative endoscopic recording was not performed in the present case). A smooth-surfaced, non-ulcerated polypoid mass is visible occupying the nasal lumen, without evidence of surface necrosis or hemorrhage — appearance consistent with that observed in our patient.

Radiological Features

On CT, sinonasal SFTs characteristically appear as well-defined soft-tissue masses showing homogeneous isoattenuation relative to gray matter on non-contrast sequences, with marked enhancement following contrast administration, reflecting their hypervascular nature **[9]**. Bony remodeling and thinning — rather than erosion — represent the most common form of osseous involvement **[9]**. On MRI, tumors are typically isointense to gray matter on both T1- and T2-weighted sequences, with heterogeneous marked enhancement on post-gadolinium images; a washout pattern on dynamic contrast-enhanced MRI is characteristic and unusual among nasal lesions, shared otherwise only with juvenile angiofibroma **[9]**. Nevertheless, none of these

features are pathognomonic, and the imaging differential remains broad.

Differential Diagnosis

The differential diagnosis of sinonasal SFT may be approached at two complementary levels: (1) the clinical differential diagnosis of unilateral sinonasal masses, which is the first question faced by the clinician, and (2) the histopathological differential diagnosis of spindle-cell tumors sharing morphological features with SFT at the sinonasal level specifically. Systematic immunohistochemical workup is essential to avoid misclassification [17-26].

Clinical Differential Diagnosis of Unilateral Sinonasal Masses

From a clinical and radiological standpoint, SFT must be distinguished from the broader spectrum of unilateral nasal masses encountered in otorhinolaryngological practice. Although definitive diagnosis ultimately rests on histopathological examination, the following entities are relevant to the preoperative clinical differential diagnosis.

Inflammatory (antrochoanal) polyp is the most common unilateral nasal mass in clinical practice, typically originating from the maxillary sinus and presenting with a characteristic bilobed appearance on CT. It lacks the solid enhancement pattern of SFT and is readily distinguished histologically by its edematous, inflammatory stroma without mesenchymal spindle-cell proliferation [26].

Inverted papilloma is a benign but locally aggressive Schneiderian neoplasm, predominantly arising from the lateral nasal wall or maxillary sinus. CT characteristically shows a convoluted cerebriform pattern. Unlike SFT, inverted papilloma carries a significant risk of malignant transformation and mandates a more radical surgical approach, including subperiosteal dissection of the attachment site and drilling of the base of implantation [26].

Juvenile angiofibroma occurs exclusively in adolescent males and arises from the sphenopalatine foramen region. It is highly vascular and may be locally destructive. On dynamic contrast-enhanced MRI, a washout pattern is characteristic — a feature shared with SFT — which may generate diagnostic confusion on imaging alone [9].

Schwannoma of the nasal septum or turbinates is a rare but well-documented cause of unilateral sinonasal mass. Imaging shows a well-circumscribed, homogeneously enhancing mass, indistinguishable from SFT on CT alone. Histological distinction relies on biphasic Antoni A/B architecture and diffuse S-100/SOX10 positivity [23].

Respiratory epithelial adenomatoid hamartoma (REAH) and related hamartomas are benign lesions arising from the olfactory cleft that may present as a unilateral fleshy polypoid mass, mimicking an inflammatory polyp or, less commonly, a solid neoplasm. CT characteristically shows widening of the olfactory cleft without bone erosion. Associated subtypes include chondro-osseous REAH (COREAH) and seromucinous hamartoma, the latter requiring careful distinction from low-grade sinonasal adenocarcinoma [24-27].

Sinonasal glomangiopericytoma is also an important histological mimicker of SFT (see section 3.3.2) and presents clinically as a unilateral nasal mass, often accompanied by epistaxis. Its characteristic immunophenotype (nuclear beta-catenin and SMA positivity, STAT6 negativity) enables reliable distinction [17,18].

Sinonasal malignancies including squamous cell carcinoma, sinonasal undifferentiated carcinoma, olfactory neuroblastoma, and intestinal-type adenocarcinoma should be considered whenever

bony destruction, surface ulceration, rapid growth, or cervical lymphadenopathy is identified on clinical examination or imaging.

In our patient, the absence of bony erosion, the well-circumscribed nature of the mass, the smooth endoscopic surface, and the slow clinical course oriented the preoperative evaluation toward a benign or low-grade neoplasm. Definitive classification, however, was only achieved after histopathological examination and immunohistochemistry.

Histopathological Differential Diagnosis of Spindle-Cell Tumors in the Sinonasal Tract

Sinonasal Glomangiopericytoma (Sinonasal-Type Hemangiopericytoma): This is one of the most important mimickers. Glomangiopericytoma (SNGP) is a benign, sinonasal-specific neoplasm with a perivascular myoid phenotype [18]. It shares the staghorn vasculature with SFT but is distinguished by uniform, closely packed round-to-ovoid rather than elongated spindle cells, a characteristic subepithelial growth pattern with a "Grenz zone," and diffuse nuclear beta-catenin and smooth muscle actin (SMA) positivity [17,18]. Crucially, SNGP is consistently negative for STAT6, although rare cases with focal CD34 positivity have been reported [17].

Schwannoma: Schwannomas of the sinonasal tract display a biphasic architecture with alternating hypercellular (Antoni A) and hypocellular (Antoni B) areas, nuclear palisading (Verocay bodies), and perivascular hyalinization — features absent in SFT [17]. Immunohistochemically, schwannomas show diffuse S-100 and SOX10 positivity, are consistently negative for STAT6 and CD34, and lack the CD34/STAT6 co-expression characteristic of SFT. In our case, the negativity of S-100 and SOX10 effectively excluded this entity.

Biphenotypic Sinonasal Sarcoma (BSNS): BSNS is a low-grade spindle cell sarcoma exclusive to the sinonasal tract characterized by dual neural and myogenic differentiation [19]. Its key distinguishing feature is concurrent S-100 and SMA (or MSA) positivity, along with nuclear beta-catenin expression in most cases [19]. BSNS harbors PAX3 gene rearrangements, most commonly involving MAML3 [19]. Unlike SFT, BSNS is negative for STAT6, and the presence of thick bands of stromal collagen and staghorn vessels in SFT, absent in BSNS, further facilitates distinction [17].

Malignant Peripheral Nerve Sheath Tumor (MPNST) and Fibrosarcoma: High-grade malignant spindle-cell neoplasms, including MPNST and fibrosarcoma, have been described in the sinonasal tract, albeit rarely. They are distinguished from SFT by the presence of significant nuclear atypia, high mitotic activity, necrosis, and, in fibrosarcoma, a herringbone growth pattern — features absent in low-risk SFT [17]. MPNST demonstrates at least focal S-100 and SOX10 expression, while fibrosarcoma is consistently negative for neural and smooth muscle markers. Both entities are STAT6-negative.

Synovial Sarcoma: Monophasic synovial sarcoma of the sinonasal tract, though rare, has been documented in the literature and may closely simulate SFT morphologically. TLE1 is frequently expressed in synovial sarcoma but may also occur in SFT and other sarcomas, limiting its specificity [17]. SS18::SSX fusion-specific antibodies offer superior sensitivity (87–95%) and near-complete specificity for synovial sarcoma and constitute a reliable discriminator when molecular studies are unavailable [17]. Synovial sarcomas are STAT6-negative.

Leiomyoma / Leiomyosarcoma: Smooth muscle tumors of the sinonasal tract are exceptional but have been reported. They are distinguished from SFT by the presence of cells with blunt-ended,

cigar-shaped nuclei and perinuclear halos, diffuse SMA and desmin positivity, and consistent STAT6 negativity [17]. The absence of desmin and SMA in our case, combined with positive STAT6 and CD34, firmly excluded this lineage.

A practical immunohistochemical panel for the differential diagnosis of sinonasal SFT should include **STAT6** (nuclear positivity: high sensitivity and specificity for SFT [13,14]), **CD34**, **S-100**, **SOX10**, **SMA**, **desmin**, and **beta-catenin**. Molecular studies (RT-PCR or FISH for NAB2–STAT6 fusion; PAX3 rearrangement by FISH for BSNS; SS18::SSX for synovial sarcoma) are decisive in diagnostically challenging cases [17].

Molecular Pathogenesis

The defining molecular event in SFT is an intrachromosomal inversion at chromosome 12q13 generating a fusion between the *NAB2* and *STAT6* genes [3,4]. The resulting chimeric protein, NAB2–STAT6, redirects the transcriptional repressor NAB2 to act as a transcriptional activator of EGR1-responsive target genes (including IGF2, FGFR1, and Cyclin D1), driving tumor cell proliferation [4,20]. Multiple fusion variants have been characterized, with NAB2ex4–STAT6ex2 and NAB2ex6–STAT6ex16/17 being the most prevalent. NAB2ex4–STAT6ex2 is preferentially associated with older patients, pleuropulmonary location, and less aggressive behavior, whereas NAB2ex6–STAT6ex16/17 correlates with younger age, extrathoracic location, and higher recurrence rates [20,21]. Secondary molecular alterations — including *TERT* promoter mutations and *TP53* mutations — have been associated with malignant progression and poor prognosis [22]. NAB2–STAT6 fusion causes nuclear accumulation of the fusion protein, which is detectable by immunohistochemistry using C-terminus anti-STAT6 antibodies — the diagnostic basis of STAT6 IHC [13].

Histopathology and Immunohistochemistry

The classical histopathological appearance of SFT is a variably cellular proliferation of bland spindle cells embedded within a collagenous stroma, associated with branching hemangiopericytoma-like ("staghorn") vessels — the so-called "patternless pattern" [5,10]. Mitotic activity is typically low in benign tumors (0–2/10 HPF in the Thompson series [5]), and necrosis is absent. Histological features associated with aggressive or malignant behavior include hypercellularity, high mitotic activity (≥4/10 HPF), nuclear pleomorphism, necrosis, and infiltrative margins [5,10].

Nuclear STAT6 expression is the most sensitive (approximately 86% of SFTs [13]) and specific immunohistochemical marker for SFT and reliably distinguishes it from histological mimickers encountered in the sinonasal differential diagnosis, including all entities described in sections 3.3.1 and 3.3.2 [13,14]. It should be noted that strong nuclear STAT6 is also rarely observed in a subset of well-differentiated/dedifferentiated liposarcomas and desmoid tumors; in the sinonasal context, however, STAT6 nuclear positivity in combination with compatible morphology and CD34 expression is highly diagnostic [13]. CD34, while less specific, is expressed in the majority of SFTs and is helpful when positive [5]. Additional markers such as Bcl-2 and CD99 may be positive but are of limited diagnostic utility given their nonspecificity [8].

Risk Stratification

The Demicco risk stratification model, accepted by the current WHO Classification of Soft Tissue and Bone Tumours (5th edition, 2020), incorporates patient age, tumor size, mitotic count, and tumor necrosis to assign a risk score stratifying patients into low-risk (score 0–3), intermediate-risk (score 4–5), and high-risk (score 6–7) groups for metastatic potential [15,16]. In the validation

cohort of Demicco et al. [16], no metastases developed in low-risk patients (n=23), a 7% 10-year metastatic risk was observed in the intermediate-risk group, and a 49% 5-year metastatic risk was recorded in the high-risk group. In the present case, the score was calculated as follows: age 51 years (<55 years: 0 points); tumor size 4.8 cm (<5 cm: 0 points); mitotic activity 2/10 HPF (1–3/10 HPF: 1 point); tumor necrosis estimated at 5–10% (<10%: 0 points); total score: 1 — low-risk category. Despite this favorable risk profile, long-term surveillance remains mandatory, as late recurrences and rare metastatic events have been documented even in low-risk sinonasal SFTs [5-7].

Treatment

Complete surgical excision with negative margins remains the cornerstone of treatment for sinonasal SFTs [11,12]. Endoscopic endonasal surgery is the preferred approach for anatomically accessible lesions, offering superior visualization, lower morbidity, shorter hospital stay, and equivalent or superior rates of complete resection compared with open surgery [11,12]. For tumors extending to the skull base, orbit, or intracranial cavity, combined endoscopic and open (transcranial or lateral rhinotomy) approaches may be necessary [6,12]. The role of adjuvant radiotherapy in sinonasal SFT with close or positive margins remains poorly defined due to the paucity of data; it has been employed in selected cases of recurrent or incompletely resected extrapleural SFTs [10]. Systemic therapies — including doxorubicin-based chemotherapy and antiangiogenic agents — are reserved for unresectable or metastatic disease [16]. In our case, surgical margins were close (enucleation-type resection), reflecting the challenge of achieving a macroscopically clear plane in this anatomical location. This finding underscores the importance of long-term endoscopic and imaging surveillance, regardless of the low-risk Demicco score.

Conclusion

Sinonasal SFTs are rare mesenchymal neoplasms with a nonspecific clinical and radiological presentation that renders them diagnostically challenging. Diagnosis is established by histopathological examination demonstrating the characteristic spindle-cell morphology with staghorn vasculature and collagenous stroma, confirmed by nuclear STAT6 immunohistochemistry, which confers high sensitivity and specificity owing to the pathognomonic NAB2–STAT6 gene fusion. A comprehensive immunohistochemical panel is essential to exclude the numerous histological mimickers within the sinonasal tract. Complete endoscopic endonasal excision is the treatment of choice when feasible. Risk stratification according to the Demicco model provides validated prognostic information to guide follow-up intensity. Long-term surveillance is mandatory for all patients, including those with low-risk tumors, given the documented potential for late local recurrence and distant metastasis.

Conflict of Interest

The authors declare no conflict of interest.

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Ethical Approval

Written informed consent was obtained from the patient for publication of this case report. This study complied with the ethical standards of the Declaration of Helsinki.

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