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Maalouf et al. Solitary fibrous tumor multifaceted tumor

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Abstract

BACKGROUND

Solitary fibrous tumor (SFT) is a remarkably uncommon mesenchymal tumor. STAT 6 and a combination of clinical, pathological, and molecular features are required to arrive at a proper diagnosis.

CASE SUMMARY

In this report, we present an intriguing case involving a 43-year-old woman who initially exhibited symptoms of a bleeding retroperitoneal tumor, resembling a gastrointestinal stromal tumor (GIST), but was later confirmed as an SFT. However, a year later, what was initially believed to be a recurrence of her SFT was instead identified as a desmoid tumor.

CONCLUSION

Asymptomatic and slow-growing, SFTs are frequently incidentally discovered during imaging. Imaging studies also are nonspecific for accurate diagnosis. After confirming the diagnosis with histological and molecular features and excluding mimics, complete excision is the gold standard treatment for SFTs. Care should be offered at sarcoma reference centers with a specialized

multidisciplinary team. Radiotherapy can be useful in the case of non-R0 resection or in a neoadjuvant setting to shrink an unresectable tumor. SFTs still demonstrate poor sensitivity to conventional chemotherapy.
The key initial step in this process is differentiating SFT from its sister tumors. Biological and antiangiogenic therapies hold potential as adjuncts to surgery in treating SFT.

Key Words:
Solitary fibrous tumor; Retroperitoneal tumor; Soft tissue neoplasms; Sarcoma; NAB2-STAT6.

Core Tip: Solitary fibrous tumor (SFT) is a remarkably uncommon mesenchymal tumor. Due to the advancements in molecular tools, STAT 6 and a combination of histologic, immunohistochemical, and molecular features are required for a proper diagnosis. We present here an intriguing case involving a 43-year-old woman who initially exhibited symptoms of a bleeding retroperitoneal tumor, resembling a gastrointestinal stromal tumor (GIST), but was later confirmed as an SFT. However, a year later, what was initially believed to be a recurrence of her SFT was instead identified as a desmoid tumor. Asymptomatic and slow-growing, SFTs are frequently incidentally discovered during imaging. Accurate diagnosis plays an essential role in the effective treatment and management of SFT. The key initial step in this process is differentiating SFT from its sister

tumors. Biological and antiangiogenic therapies hold potential as adjuncts to surgery in treating SFT.

INTRODUCTION

With over 150 histological subtypes of soft tissue sarcomas (STS) and mesenchymal tumors, solitary fibrous tumors (SFT) are an exceptionally rare mesenchymal tumor, comprising 3.7% of cases [1]. Initially described by Klemperer and Rabin in 1931, they delineated the morphological characteristics of SFT in a series of five cases of pleural neoplasms [2]. SFT was exclusively observed in the pleura or lungs until 1990 [2]. Accurate classification of sister tumors is essential not only for diagnosis and prognosis but also for the appropriate management of patients [1]. With a rarely metastasizing aptitude, WHO recently classified SFT as a fibroblastic neoplasm with intermediate behavior [3].

SFT is referred to as the "great stimulator" among soft-tissue neoplasms because it presents with numerous potential differential diagnoses. [4]. Therefore, a combination of clinical, pathological, immunohistochemical, and molecular features is required to have an appropriate diagnosis [1]. A particular cytogenetic hallmark for SFT has emerged to help in this process, identified by the NAB2 (NGFI-A-binding protein 2)–STAT6 (signal transduction and activator of transcription 6) fusion oncogene [1, 2].

Here, we describe a compelling case of a 43-year-old woman who initially presented with a bleeding retroperitoneal tumor mimicking a gastrointestinal stromal tumor (GIST), which was subsequently diagnosed as SFT. However, a year later, what was initially suspected to be a recurrence of her SFT turned out to be another benign soft tissue tumor.

CASE PRESENTATION

Chief complaints

A 43-year-old woman with a history of B12 deficiency anaemia following Roux en Y gastric bypass; presented with diffuse vague abdominal pain of one-month duration.

History of the present illness

The patient had no associated symptoms of fever, chills, nausea, vomiting, change in bowel habits, weight loss, or night sweats.

Physical examination

Physical exam showed a soft non-distended abdomen with mild epigastric tenderness. Laboratory tests were unremarkable.

Imaging examinations

Computed tomography (CT) of the abdomen and pelvis revealed a lobulated mass-like lesion in the right mid abdomen, in close contact with the ascending colon, measuring 10x8x6.5 cm (Figure 1). It shows soft tissue attenuation density with heterogeneous enhancement. A small adjacent 7mm lymph node is seen. A small amount of free fluid is found in the abdomen, predominantly in the bilateral para-colic gutters, and to a lesser extent in the para-hepatic, para-splenic spaces, and pelvis.

FURTHER DIAGNOSTIC WORKUP

A gastroscopy and a colonoscopy were normal and excluded any intraluminal lesion. A CT-guided biopsy of the lesion showed a spindle cell tumor that can correspond to GIST or smooth muscle tumor.

TREATMENT

Ultimately, following deliberation in a multidisciplinary expert consultation and considering the risk of bleeding, a prompt decision was made to proceed with surgical removal. Open surgery revealed two hundred milliliters of hemoperitoneum, no peritoneal deposits, and no distal metastasis. A large tumor adherent to the ascending and transverse colon at the hepatic pedicle was identified. It was also encased by the omentum and in close contact with the duodenum. R0 was achieved after dissection of the tumor from the second part of the duodenum and the hepatic pedicle and surrounding bowels. Histopathology showed a completely excised tumor with spindle-shaped cells with slightly irregular nuclei with foci of necrosis. CD-34 positive on immunostaining along with STAT6 positivity, but DOG1 and EMA negative, compatible with solitary fibrous tumor. Numerous vessels run through the lesion. No abnormal mitotic activity was seen. The excised lymph node was reactive and showed no malignancy.

POSTOPERATIVE COURSE

Her postoperative stay was complicated by pneumonia and pulmonary embolism. On day 2 postoperatively, she had respiratory distress and desaturation. Chest x-ray and a spiral CT scan of the chest showed parenchymal consolidation of the right lower lobe and a loss of volume of the right lung with a shift of the mediastinum to the ipsilateral side along with left-sided pulmonary embolism. The accompanying abdomen scan showed superior mesenteric vein (SMV) thrombosis. After being treated with therapeutic anticoagulation for her pulmonary embolism and SMV thrombosis, the patient improved drastically and was discharged home on day 6 postoperatively.

FOLLOW UP

One year later, the patient had an abdomen pelvis CT that showed normal findings with no signs of recurrence. However, two years later, the CT scan

showed an interval appearance of a 3.8 x 5 cm slightly enhanced soft tissue mass in the mesentery (Figure 2). The tumor was shown to be in close contact with the small bowels. Mesenteric lymph nodes measuring up to 8 x 10 mm were also seen. A decision was made to perform tumor resection due to concerns about recurrence. No ascites or distant metastasis were found during laparotomy. A mesenteric lesion invading the small bowel was noted 80 cm from the ligament of Treitz for which en-bloc resection with partial enterectomy was done followed by end-to-end manual anastomosis. Histopathology showed a lesion made of spindle cells arranged in fascicles and sheets with many blood vessels. There was no evidence of necrosis or abnormal mitosis. Immunophenotyping was also done that showed DOG 1, CD117, SMA, S100, and CD34 negative but beta-catenin nuclear positive compatible with mesenteric desmoid tumor. The intestine was unremarkable and 12 lymph nodes were identified and shown to be reactive. Therefore, a spindle cell tumor was diagnosed.

DISCUSSION

SFTs can manifest across a broad age spectrum, with the highest occurrence observed in the fifth and sixth decades of life [1, 2]. They can appear anatomically anywhere, with the pleura representing the most common site,

accounting for 30% of cases. Other prevalent sites include the meninges (27%), abdominal cavity (20%), trunk (10%), extremities (8%), and head & neck (5%) [5]. SFTs localized in the retroperitoneum, peritoneum, or mediastinum often demonstrate a more aggressive course compared to tumors found in other anatomical locations [6]. No specific risk factors are known for the development of SFTs [7]. Typically asymptomatic, SFTs are slow-growing tumors often incidentally discovered during imaging studies [8]. Symptoms may occasionally arise due to pressure effects on adjacent organs [1,2]. In our case, vague abdominal pain prompted evaluation, likely due to mass effect, as no other symptoms indicated this diagnosis.

Furthermore, some patients may develop paraneoplastic syndromes that can aid in diagnosis. Pierre Marie–Bamberger syndrome or hypertrophic osteoarthropathy is an uncommon nonspecific condition occasionally linked to pleural SFT. Typical symptoms include distal digital clubbing, periostitis, and synovial effusions, believed to be associated with the upregulation of vascular endothelial growth factor (VEGF). Additionally, fewer than 5% of SFT patients may exhibit a refractory hypoglycemic syndrome due to the excessive production of insulin-like growth factor-2 by large peritoneal or pleural SFTs, known as Doege–Potter syndrome [9, 10].

No specific radiologic findings are associated with SFTs, typically revealing a well-defined isodense mass to skeletal muscle on CT scans, often exhibiting contrast enhancement in highly vascularized tumors (65%) [11]. SFTs commonly exhibit low-signal-intensity areas on both T1- and T2-weighted magnetic resonance imaging (MRI), indicative of collagen content. While 18F-FDG PET/CT may offer limited diagnostic value in suspected SFT cases, the detection of multiple high-grade lesions should prompt consideration of malignant SFT [1]. During both instances of our patient's presentation, CT

scans were utilized as the preferred imaging modality. While they aided in localizing the tumor and confirming its classification as an STS, they provided no additional information, and the diagnosis was not solely based on imaging. Given the tumor's nature and suspicion of bleeding, further testing or biopsy was not conducted before surgery to enhance tumor characterization. However, ideally, every STS should undergo a thorough biopsy and characterization for improved diagnosis and direct treatment.

Macroscopically, SFT exhibits well-defined, partially encapsulated features and a multinodular, firm, whitish-cut surface. In certain instances, myxoid change and hemorrhage may be observed. However, malignant and locally aggressive tumors might display irregular, infiltrative borders along with areas of necrosis [3]. Microscopically, SFTs represent tumors with variable cellularity, comprising ovoid to spindled cells that demonstrate either patternless growth or a storiform pattern set against a background stroma of varying collagen content, which contains thin-walled, large branching blood vessels resembling "staghorn" structures. Additionally, medium-sized blood vessels accompanied by perivascular fibrosis are frequently observed [5]. In cases of markedly cellular tumors, sheets of more primitive-appearing rounded cells are evident [2]. Clinical and histomorphological indicators indicative of malignancy include advanced age, larger tumor size, heightened cellularity, increased mitotic activity (≥4/10 HPFs or > 2 mitoses/2 mm^2), nuclear pleomorphism, tumor necrosis, and infiltrative margins [2]. In the presented case, the tumor cells displayed spindle-shaped histology with slightly irregular nuclei and areas of necrosis. The lesion contained numerous vessels, yet abnormal mitotic activity was absent. These histological characteristics serve as strong indicators for diagnosing SFTs when combined with other features.

The combination of CD34, CD99, and BCL-2 is commonly employed in the diagnosis of SFT. These immunohistochemical (IHC) markers are sensitive, showing strong expression in approximately 90% of cases. However, their utility is limited due to their expression in other neoplasms that closely resemble SFT histologically [12]. STAT6 IHC staining has emerged as a valuable surrogate marker for NAB2-STAT6 gene fusion, demonstrating excellent sensitivity and specificity, even in malignant cases [2]. Nevertheless, it can also be expressed in various other soft tissue neoplasms, including well-differentiated liposarcoma (WDL) or dedifferentiated liposarcoma (DDL), desmoid fibromatosis, unclassifiable sarcoma, neurofibroma, myxoid liposarcoma, undifferentiated pleomorphic sarcoma, low-grade fibro myxoid sarcoma, synovial sarcoma (SS), and ovarian fibroma [5, 13]. A combination of radiological, histological findings and markers was used in our case to diagnose SFT given the challenge of depending on a single factor for diagnosis.

The common histological presentation of SFT alongside other soft tissue tumors discussed previously contributes to a considerable challenge in differential diagnosis. Histological variants of solitary fibrous tumors include lipomatous variants (mimicking adipocytes), giant cell-rich variants (more common in the head and neck region), rhabdomyosarcomatous[2] tumors, and aggressive "de-differentiated" variants (usually with loss of CD34 and STAT6 expression) [1, 2].

For extrameningeal[3] solitary fibrous tumors, most pathologists rely on the DeMicco risk stratification scheme for metastatic potential. In the large series by DeMicco, tumors that rarely can exhibit strong expression of STAT6 include undifferentiated pleomorphic sarcoma, desmoid, well/dedifferentiated liposarcoma (particularly the latter), clear cell sarcoma, and "myxoid sarcoma". The NAB2:STAT6 rearrangement results from an intrachromosomal inversion in

chromosome 12. As the genes are in close proximity[4], the rearrangement may not be detected with conventional chromosome banding or by FISH but more reliably by RT-PCR or Next Generation Sequencing [3-4].

This challenge underscores the importance of discerning lesions based on subtle histological variations, as well as the utilization of novel IHC markers specifically studied for this purpose. For instance, the presence of positive MDM2 and CD4 marker expression, observed in WDL and DDL but not in SFT, aids in ruling out an SFT diagnosis. Moreover, desmoid fibromatosis shares remarkably similar histological features with SFT, alongside STAT6 expression [1, 2].

In our case, upon the new presentation, the diagnosis shifted to desmoid fibromatosis instead of recurrent SFT. Ultimately, the diagnosis of desmoid tumor can be confirmed through nuclear β-catenin positivity, along with positive staining for vimentin, smooth muscle actin, cyclooxygenase-2, and β-estrogen receptors, while showing negativity for desmin, CD34, S100, and KIT markers [14].

When treating these individuals, patients with SFTs should receive care at sarcoma reference centers, where a specialized multidisciplinary team consisting of a medical oncologist, surgical oncologist, radiation oncologist, pathologist, and radiologist, all well versed in the complexities of this condition, should oversee their management [1]. The gold-standard treatment for localized disease is considered to be complete en-bloc surgical resection with negative margins (R0) [15]. Typically, this resection exhibits a 10-year overall survival (OS) rate ranging from 54% to 89% [1]. Patients with negative margins (R0) and lacking high-risk histologic features are advised to undergo observation since there is no evident indication of an OS advantage with adjuvant radiotherapy (RT). Given the complete, en bloc resection in our

presented case, surgery proved adequate, prompting a follow-up at six months followed by yearly assessments thereafter for a prolonged period.

For intermediate- to high-risk SFT cases with positive margins (R1/R2), the consideration of re-resection should be raised for suitable patients if complete resection with minimal morbidity is attainable. Adjuvant RT stands as a reasonable choice when the patient is unsuitable for additional resection or achieving R0 surgery is technically unfeasible [16, 17]. Neoadjuvant RT may be considered in specific cases to enhance tumor resectability or when manageable wound complications are anticipated [1].

When SFTs are localized or deemed resectable, there is no substantiated evidence backing the utilization of systemic therapies in either the neoadjuvant or adjuvant contexts [18]. However, there is limited data on the response of SFT to conventional chemotherapy (CT) [1]. It is crucial to emphasize that adjuvant CT should never be employed as a means to compensate for inadequate surgery. Nonetheless, for certain patients with high-risk SFTs and/or extensive malignant tumors where achieving R0 surgery is unattainable, the consideration of neoadjuvant CT should be deliberated within a specialized multidisciplinary setting [1]. There is no evidence to suggest that multiagent CT enhances patient survival, and single-agent CT continues to be the standard approach [1].

Other treatment options include antiangiogenic agents such as sunitinib, sorafenib, and pazopanib [19, 20], reflecting the highly vascularized nature of these tumors. Inhibiting angiogenesis can impede tumor cell proliferation. Additionally, overexpression of IGF-1 is observed in SFT, with figitumumab, a fully human IgG2 anti-IGF-1 (IGF-1R) monoclonal antibody, establishing tumor responses in some patients with advanced SFTs [21]. Immunotherapy also

shows promise for SFTs; a translational study assessed the correlation of PD-1, PD-L1, and tumor-infiltrating lymphocyte (TIL) expression with prognosis [22]. The prognosis of SFT is promising following complete surgery. However, recurrence may occur in 10–25% of cases within 10 years [23]. In high-risk patients, the risk of metastatic recurrence within 5 years can reach 40% [24]. Late relapses, extending beyond 10 years and up to 20 years after the initial presentation, are common, highlighting the need for long-term monitoring [23,24]. Common sites for metastasis include the lungs, liver, and bones. SFT recurrence is more common in cases of incomplete resection (R1/R2), tumor seeding within serosal membranes (such as the pleura, and peritoneum), or distant hematogenous spread.

CONCLUSION

Sarcomas comprise a highly diverse group of tumors, exhibiting heterogeneity clinically and genomically. Accurate diagnosis is crucial for effectively treating and managing the rare STS tumor known as SFT. IHC serves as the most sensitive and specific method for diagnosing SFT, offering practicality and cost-effectiveness.

In our presented case, distinguishing SFT from its fellow tumors was pivotal. Initially, it was mistaken for GIST and later for recurrence but was ultimately identified as Desmoid. At each stage, the correct diagnosis led to appropriate treatment.

SFTs demonstrate poor sensitivity to conventional chemotherapy, with appropriate surgery remaining the optimal curative approach. However, biological and antiangiogenic agents show promise, with STAT6 and IGF-1 overexpression rendering SFT a targetable sarcoma. While data on the

effectiveness of immunotherapy are limited, ongoing research continues to uncover new insights into managing this multifaceted tumor.

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Footnotes

Informed consent: Written informed consent was obtained from the patient for publication and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Ethical approval: Case report approved for publishing by the ethical committee at Mount Lebanon Hospital University Medical Center, and Head of General Surgery division, Beirut, Lebanon, 2024.

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Figure Legends

Figure 1: Enhanced Ct scan transversal view showing a lobulated mass-like lesion in the right mid abdomen, in close contact with the ascending colon, measuring 10x8x6.5 cm. (Yellow arrow)

Figure 2: Enhanced Ct scan showing a 3.8 x 5 cm slightly enhanced soft tissue mass in the mesentery in close contact with the small bowels. (Yellow arrow)

1.	*enterectomy*	Unknown words	Correctness
2.	*rhabdomyosarcomatous*	Unknown words	Correctness
3.	~~extrameningeal~~ → extra meningeal	Misspelled words	Correctness
4.	~~in close proximity~~ → nearby, near, close	Wordy sentences	Clarity