Primary thoracic synovial sarcoma in a 45-year-old male: A case report and literature review

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Abstract. Synovial sarcoma (SS), a rare soft tissue cancer, develops at various body sites, with a predominant occurrence in the limbs. This report presents a rare case of primary thoracic SS (PTSS) in a 45-year-old male patient, characterized by chronic cough and occasional shortness of breath. Imaging examination revealed a substantial tumor mass in the left thoracic cavity. Although preoperative biopsy misdiagnosed the tumor as a solitary fibrous tumor, postoperative molecular analysis revealed that the tumor was SS based on the characteristic t(X;18)(p11.2;q11.2) translocation. The precise origin of the tumor within the thoracic cavity remained unclear, and the involvement of the pleura, chest wall and mediastinum was considered. The patient underwent surgical resection of the tumor followed by adjuvant chemotherapy with doxorubicin and nedaplatin. Regrettably, the patient showed widespread metastatic progression 8 months post-surgery and died 18 months thereafter because of tumor dissemination and associated complications. This case highlights the diagnostic challenges of PTSS and underscores the need for increased clinical awareness of rare thoracic tumors.

Introduction

Synovial sarcoma (SS), a relatively rare malignant soft tissue tumor, has been progressively understood since its first description in 1936 (1,2). Although it most frequently develops in the extremities, cases with a primary thoracic origin, referred to as primary thoracic SS (PTSS), are exceedingly rare, with only a few incidences reported in the literature (3-6). PTSS often shows nonspecific clinical features, with common symptoms such as cough, chest pain and shortness of breath, which could be easily misdiagnosed as frequently reported thoracic diseases, such as lung cancer or benign lung conditions.

PTSS poses major diagnostic challenges because of its rarity and its ability to mimic other malignancies. Its diagnosis often relies on the combination of imaging modalities, histopathological examination and molecular analysis. A key diagnostic feature of SS is the chromosomal translocation t(X;18)(p11.2;q11.2), detectable by techniques such as fluorescence *in situ* hybridization (FISH) and reverse transcription-polymerase chain reaction. However, in certain cases, the preoperative diagnosis can be difficult, as noted in the present case, where the tumor was initially misdiagnosed as a solitary fibrous tumor (SFT) based on histological and immunohistochemical (IHC) findings.

The origin of PTSS within the thoracic cavity remains controversial, with certain reports suggesting the involvement of the pleura, chest wall, mediastinum or a combination of these structures (7). Accurate localization of the tumor's primary site is crucial for appropriate staging and treatment planning; however, in numerous cases, including the present case, the precise origin cannot be definitively determined (8).

Given the limited number of PTSS cases and the lack of high-level evidence for standardized treatment protocols, therapeutic strategies often involve a combination of surgery, chemotherapy and radiation therapy customized according to the patient's health condition. However, the prognosis for PTSS remains poor, with a high risk of recurrence and metastasis (9).

The current study presents the case of a patient diagnosed with PTSS, who, despite receiving surgical tumor resection and subsequent adjuvant chemotherapy, died due to widespread metastasis and associated complications.

Case report

A 45-year-old male presented to The Second Forestry Hospital of Heilongjiang Province (Yichun, China) in April 2021 with

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persistent chest pain and dyspnea. The patient's medical history was notable for chronic smoking, with no other significant comorbidities. On initial examination, the patient appeared normal but exhibited mild respiratory distress (10). Chest X-ray revealed a large mass in the left hemithorax. Additional imaging with a computed tomography (CT) scan confirmed the presence of a tumor mass measuring 18.5x16.4x9.3 cm, localized to the left thoracic cavity, with broad contact to the parietal pleura but no clear chest wall invasion (Fig. 1A). The preoperative staging work-up included contrast-enhanced chest CT, non-contrast head CT and abdominal ultrasonography. Based on these examinations, no evidence of distant metastasis was detected. A positron emission tomography-CT scan, which could have provided a more comprehensive metastatic survey, was not performed due to the patient's financial constraints. All laboratory tests, including complete blood count; renal, liver and coagulation profiles; and tumor markers (cancer antigen 125, cancer antigen 153 and carcinoembryonic antigen), were within their normal ranges.

CT-guided percutaneous biopsy was performed. The specimens were fixed in 10% neutral buffered formalin, embedded in paraffin and cut at 4-μm thickness. Histological examination (H&E staining; Fuzhou Maixin Biotechnology Development Co., Ltd.) was performed according to standard clinical protocols and revealed randomly arranged spindle-like cells with indistinct cell borders. All immunohistochemical staining in this case was performed on an automated staining system (Benchmark XT; Roche Diagnostics) strictly according to the manufacturer's protocols. Primary antibodies were obtained from Fuzhou Maixin Biotechnology Development Co., Ltd. as ready-to-use reagents (prediluted 1:100). The initial panel focused on the most common differential diagnosis and showed positive staining for vimentin (cat. no. MAB-0735), cluster of differentiation 34 (CD34; cat. no. MAR-1076) and B-cell lymphoma 2 (BCL-2; cat. no. MAB-0711) (Fig. 2A and B). Because this profile typically represents SFT, additional staining was not initially performed, as the findings were deemed sufficient for a preoperative diagnosis. Following a multidisciplinary discussion, the team decided to proceed with direct surgical intervention on the basis of the pathological findings (11,12).

Because of the large tumor size, the patient underwent open thoracotomy. The procedure, which lasted for 4 h with an estimated blood loss of 600 ml, revealed a large soft tissue mass occupying the thoracic cavity. The tumor's epicenter was located on the parietal pleura, with firm adhesions and direct invasion into the adjacent mediastinum and chest wall. Scattered satellite lesions were also noted on the lung surface. Because the preoperative diagnosis of SFT suggested a benign nature of the mass, macroscopic complete resection of the tumor mass was planned. Therefore, intraoperative frozen section analysis was not performed, and an *en bloc* resection of the main tumor mass, along with the involved mediastinal pleura, chest wall and all identifiable satellite nodules, was conducted. Macroscopically, the resection appeared complete. However, postoperative pathological examination of the entire specimen revealed deep tumor infiltration and the surgical margins were microscopically positive (R1 resection) at the mediastinal involvement site. The R1 resection, with positive surgical margins, was a significant factor influencing the high risk of recurrence and metastasis in this case.

Gross examination of the resected specimen (Fig. 1C) revealed a well-circumscribed, but unencapsulated, greyish-white soft tissue mass measuring 18.5x16.4x9.3 cm in the greatest dimension. The cut surface was solid and tan-white, with focal hemorrhagic areas and ~20% necrosis. Histological examination (Fig. 1D) demonstrated a hypercellular tumor composed of relatively uniform, spindle-shaped cells arranged in intersecting fascicles. The tumor cells exhibited oval nuclei, scant cytoplasm and predominantly monophasic fibrous morphology. Mitotic activity, assessed on H&E-stained sections, was elevated, with an average of 12 mitoses per 10 high-power fields. An extensive IHC panel was performed as described above, using ready-to-use primary antibodies (prediluted 1:100; Fuzhou Maixin Biotechnology Development Co., Ltd.), which revealed diffuse nuclear positivity for transducin-like enhancer of split 1 (TLE1; cat. no. MAB-0686) (Fig. 2C) and membranous positivity for CD117 (c-kit; cat. no. Kit-0029) as well as focal immunoreactivity for epithelial membrane antigen (EMA; cat. no. MAB-1101) and low-molecular-weight cytokeratins [CK (cat. no. RAB-0050) and CK19 (cat. no. MAB-0829)] (Fig. 3A-D). The tumor was entirely negative for STAT6 (nuclear; cat. no. RMA-1066) (Fig. 2D), which effectively ruled out the diagnosis of SFT. A mesothelial marker panel [Calretinin (cat. no. MAB-0716), Wilms tumor-1 (cat. no. RMA-1151), D2-40 (cat. no. MAB-0567) and CK5/6 (cat. no. RMA-1144)] also yielded a negative result, excluding the diagnosis of sarcomatoid mesothelioma (Fig. 4A-D). The Ki-67 (cat. no. RMA-0542) proliferative index was ~30%, indicating a moderately high rate of cell proliferation (Fig. 2E) (13,14). The surgical margins were positive for tumor cells (R1 resection), with the closest margin measuring <1 mm at the mediastinal aspect. The diagnosis was confirmed cytogenetically by fluorescence *in situ* hybridization (FISH) using the Vysis SS18 Break Apart FISH Probe Kit (Abbott Molecular), performed strictly according to the manufacturer's protocols. The analysis revealed a rearrangement of the SS18 (SYT) gene locus in 80% of the nuclei assessed, and thus, fusion-specific SS18-SSX IHC was not performed (Fig. 2F). Pathological staging of the tumor was T4G3NxM0, which is classified as stage III according to the American Joint Committee on Cancer staging system (15,16).

The patient's postoperative recovery was uneventful. A follow-up chest CT scan demonstrated improved expansion of the left lung (Fig. 1B), and the chest tube was removed on postoperative day 5 once drainage decreased to <100 ml per day. In light of the high-risk pathological features (M0 by preoperative imaging, T4G3NxM0 and stage III disease with R1 resection), the multidisciplinary tumor board recommended a comprehensive adjuvant strategy consisting of radiotherapy to the tumor bed followed by systemic chemotherapy (17). Chemotherapy was commenced on postoperative day 21 with a 6-cycle regimen of doxorubicin (70 mg/m^2) and nedaplatin (50 mg/m^2). However, the patient discontinued treatment after 3 cycles due to grade 2 nausea and fatigue, exacerbated by personal and financial constraints. The total cost of the initial surgical resection was contained under $3,000, reflecting these substantial financial limitations. The rationale for adjuvant radiotherapy was thoroughly explained to the patient to mitigate the high risk of local recurrence; however, despite counseling, the patient declined this intervention. At 8 months

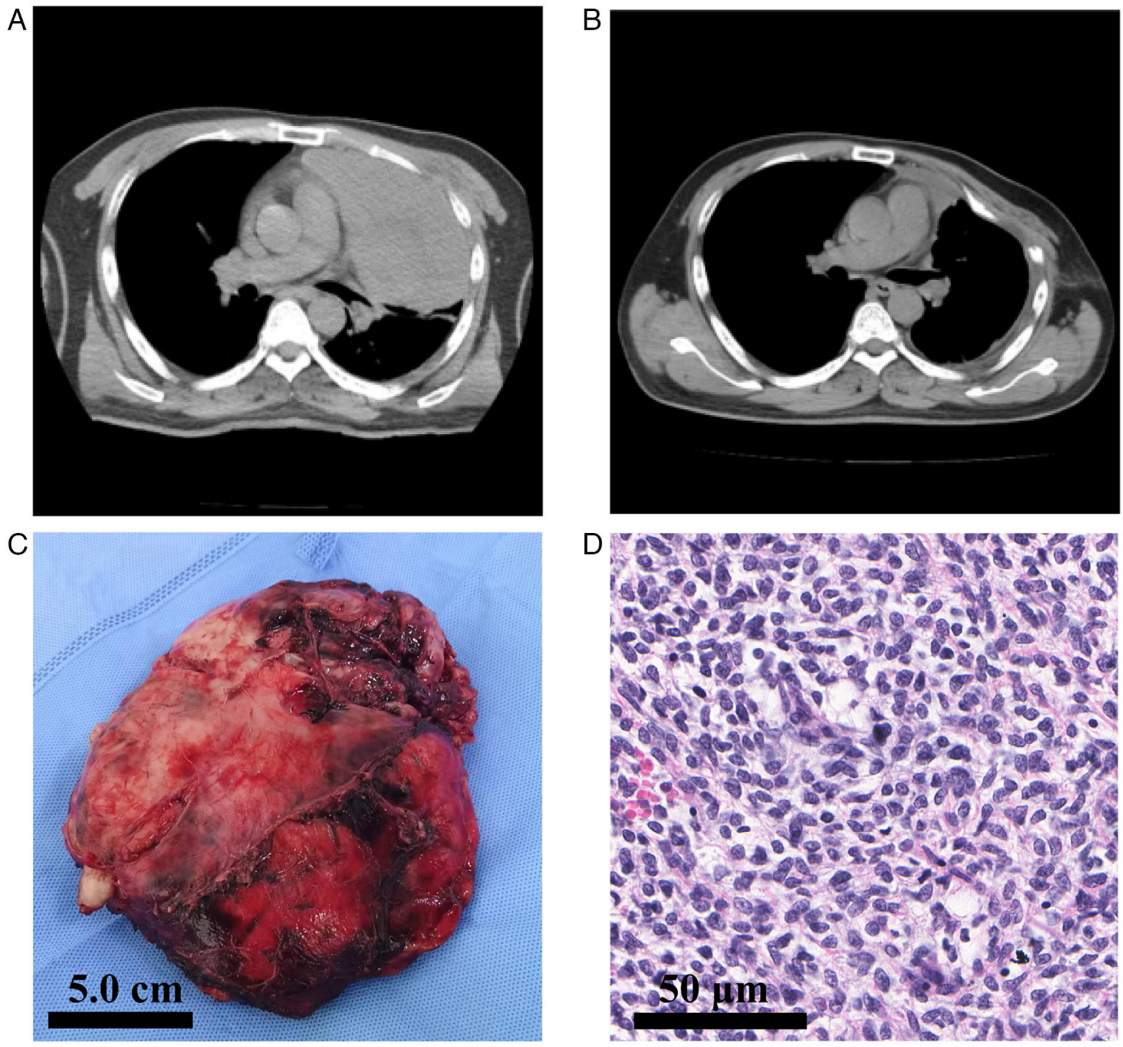

Figure 1. Radiological, gross and histopathological findings of the primary thoracic synovial sarcoma. (A) Axial contrast-enhanced chest CT scan (mediastinal window) reveals a large, heterogeneous mass (18.5x16.4x9.3 cm) occupying the anterior left hemithorax. The mass causes a substantial mass effect, leading to compressive atelectasis of the left lung. No definite mediastinal lymphadenopathy was identified. (B) A follow-up chest CT scan (mediastinal window) on postoperative day 5 shows successful re-expansion of the left lung, with no residual pneumothorax or significant pleural effusion. (C) Gross specimen of the resected tumor following left pneumonectomy. The tumor measured 18.5 cm in diameter and appeared solid and tan in color, with visible areas of hemorrhage. (D) Histopathological examination (hematoxylin and eosin staining; original magnification, x400; scale bar, 50 μm) demonstrates hypercellular proliferation of uniform spindle-shaped cells with indistinct cytoplasm arranged in intersecting fascicles, consistent with the monophasic variant of synovial sarcoma.

after surgery, the patient was re-admitted with persistent cough and cachexia; repeat CT imaging revealed multiple bilateral pulmonary nodules, indicative of widespread metastatic progression (data not shown). The accumulated financial burden was a major factor contributing to the patient's reluctance towards further therapies. The multidisciplinary team discussed evidence-based salvage options, including palliative radiotherapy, second-line systemic agents (such as ifosfamide, trabectedin or pazopanib) and best supportive care. After considering the limited prognosis and potential treatment-related burdens, the patient elected to forgo all further anticancer therapy. The patient died 18 months postoperation. The overall clinical course is summarized in Fig. 5.

Discussion

PTSS is a rare and diagnostically challenging malignancy (10), typically presenting with nonspecific symptoms that complicate and often delay accurate diagnosis. To summarize recent findings, a comprehensive literature search of the PubMed (https://pubmed.ncbi.nlm.nih.gov/), Embase (https://www.embase.com/) and Web of Science (https://www.webofscience.com/) databases from 2020 to the present was conducted. The search strategy utilized the following keywords: 'mediastinal synovial sarcoma', 'chest wall synovial sarcoma', 'thoracic synovial sarcoma', and 'pulmonary synovial sarcoma'. As summarized in Table I (11,18-37), which shows a comprehensive literature review from 2020 to the present, this entity primarily affects young to middle-aged adults and often appears as sizable tumors at the time of identification. A key issue related to its characterization is the controversy over its primary anatomic site-whether it originates from the pleura, chest wall or mediastinum. This diagnostic uncertainty is compounded by the frequent finding of extensive tumor infiltration, which can obscure the true anatomic epicenter. Consequently, attempting to define the tumor origin based

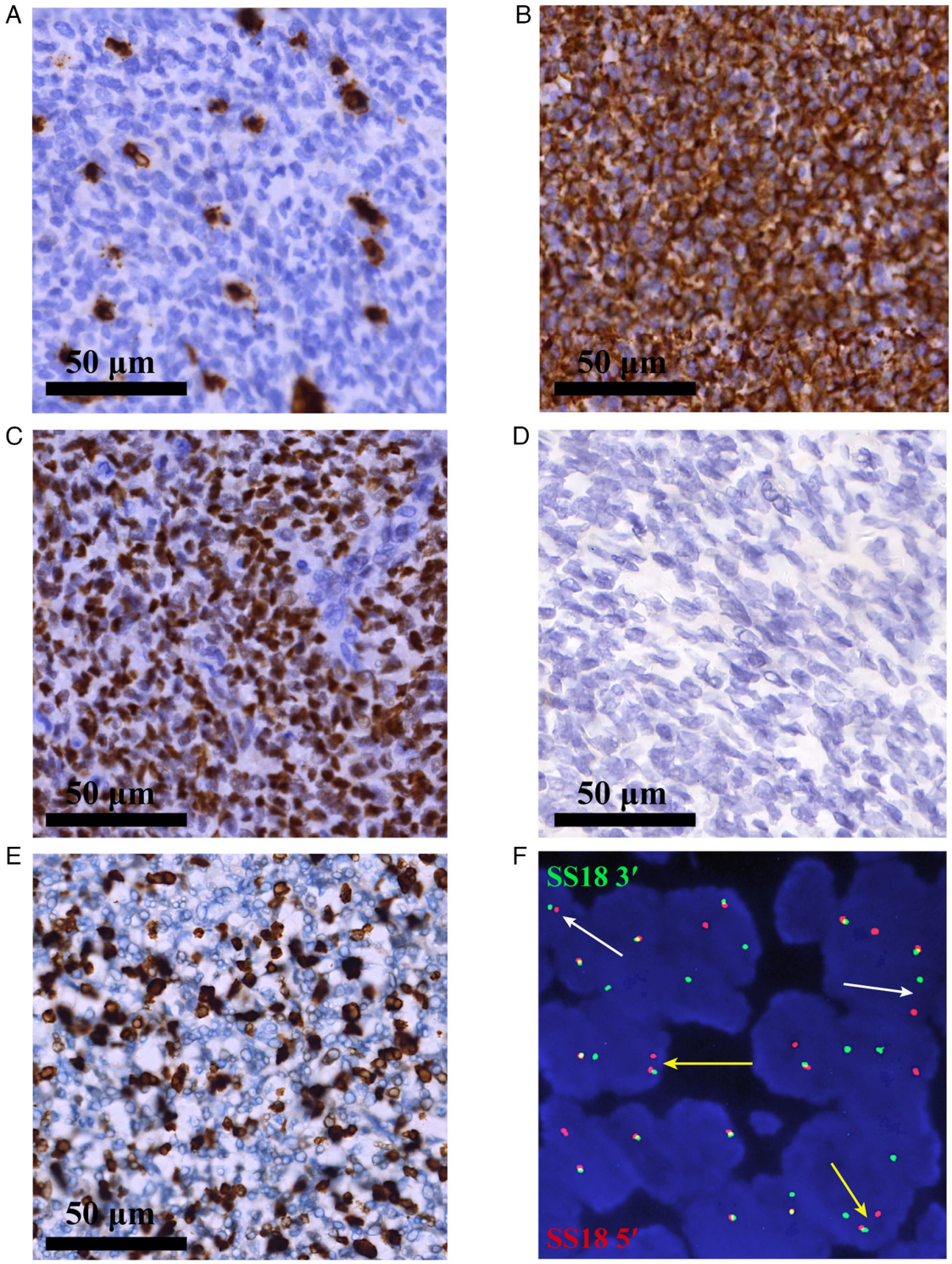

Figure 2. IHC findings and confirmatory molecular cytogenetic profiling. (A and B) Initial IHC staining (original magnification, x400; scale bar, 50 μm) of the biopsy specimen shows positive staining for (A) CD34 and (B) BCL-2, a profile that initially raised consideration for solitary fibrous tumor. IHC and molecular cytogenetic profiling confirming the diagnosis of monophasic synovial sarcoma. (C-E) IHC staining (original magnification, x400; scale bar, 50 μm) shows (C) strong and diffuse nuclear positivity for transducin-like enhancer of split 1, supporting the diagnosis; (D) negative nuclear staining forSTAT6, effectively excluding solitary fibrous tumor; and (E) a high Ki-67 proliferation index of ~30% (scale bar, 50 μm). (F) SS18 gene fluorescence *in situ* hybridization analysis using break-apart probes (original magnification, x1,000) reveals multiple positive signal patterns indicative of SS18 gene rearrangement. The 5' and 3'ends of the *SS18* gene are labeled with red and green fluorescence, respectively. One pattern shows an abnormal signal (a single red SS18 5' signal accompanied by normal red-green fusion signals, indicated by yellow arrowheads), while another shows a typical break-apart pattern (separated green SS18 3' and red SS18 5'signals, indicated by white arrowheads). IHC, immunohistochemical.

solely on proximity to surrounding structures is an oversimplification that can be misleading (10). In the present case, preoperative imaging revealed the mass to have its broadest and most intimate contact with the parietal pleura. Intraoperatively, while the tumor showed adhesions to both the parietal pleura and mediastinum, the most extensive and firmest attachment

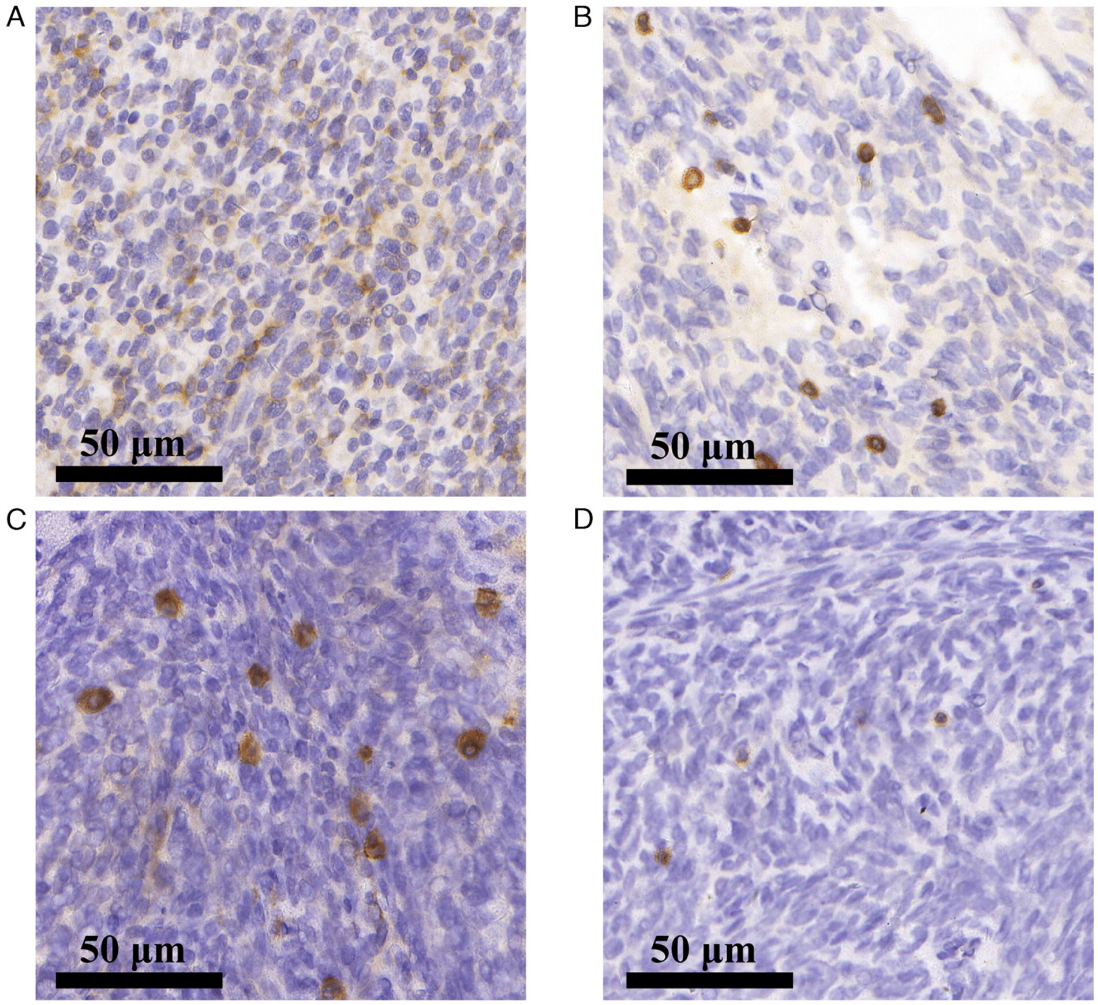

Figure 3. Immunohistochemical profile supportive of monophasic synovial sarcoma. (A-D) IHC staining showing (A) focal immunoreactivity for epithelial membrane antigen, (B) strong and diffuse membranous positivity for CD117 (c-kit), and (C and D) focal positivity for low-molecular-weight (C) CK and (D) CK19 (original magnification, x400; scale bar, 50 μm). CK, cytokeratin.

was observed to the parietal pleura. Therefore, synthesizing imaging and surgical findings, the parietal pleura is considered the most probable site of origin. However, this assessment is made with caution due to notable inconsistencies: A primary tumor of the parietal pleura would typically be expected to show direct infiltration into the adjacent chest wall musculature, which was not observed. Similarly, a primary mediastinal origin fails to adequately explain the overall growth pattern and adhesion findings. Given these unresolved questions and in the interest of utmost pathological and clinical rigor, it may be concluded that, while the parietal pleura is the most likely origin, a definitive anatomic epicenter cannot be assigned. Consequently, the tumor is pragmatically classified as a PTSS arising within the thoracic cavity, consistent with the literature describing cases with widespread involvement where the precise origin remains indeterminate (10,11,23,25,33,38-41).

In the present case, the diagnostic challenge of PTSS was further complicated by the discrepancy between the findings of preoperative biopsy and postoperative pathological examination. Initially, the tumor was misdiagnosed as an SFT, leading to the missed opportunity for initiating neoadjuvant chemotherapy. The definitive diagnosis of SS was made postoperatively after identifying the characteristic t(X;18) (p11.2;q11.2) translocation (15,16). This underscores the importance of preoperative molecular analysis. The initial biopsy findings, based on cellular morphology and an IHC profile of CD34- and BCL-2-positive spindle cells, were highly suggestive of SFT. At this stage, these findings were considered sufficient for a preoperative diagnosis, and the rarity of PTSS lowered the level of clinical suspicion. Consequently, neither additional IHC staining (such as STAT-6) nor molecular analysis was performed preoperatively. The clinical treatment approach primarily focused on obtaining a definitive diagnosis and achieving local control through surgical resection. This case highlights a critical diagnostic pitfall: For large or atypical thoracic spindle cell tumors, a higher index of suspicion for rare sarcomas and broader preoperative diagnostic panels can help avoid misdiagnosis and facilitate neoadjuvant treatment planning (42,43).

Complete surgical resection (R0) remains the cornerstone of PTSS treatment, strongly correlating with improved survival outcomes (11). However, in the present case, achieving clear surgical margins was particularly challenging because of the tumor's extensive involvement of the mediastinum, chest wall and lung surface. The tumor had to be bluntly dissected from these structures during surgery, making it impossible to

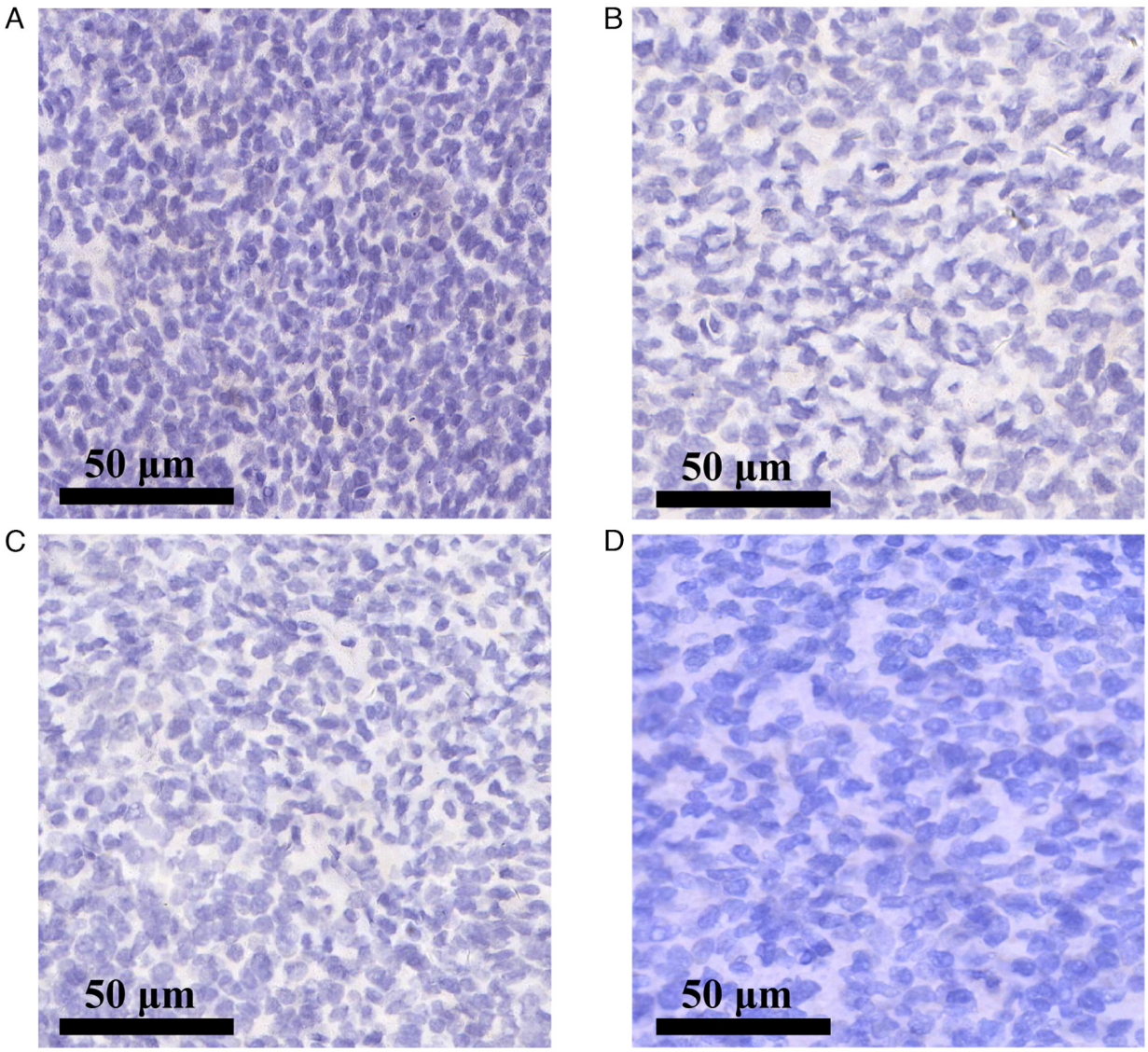

Figure 4. Immunohistochemical staining excluding major differential diagnoses. (A-D) IHC staining (original magnification, x400) shows negative results for key markers used to rule out other entities: (A) Calretinin, (B) Wilms tumor-1, (C) D2-40 and (D) cytokeratin 5/6 (scale bar, 50 μm).

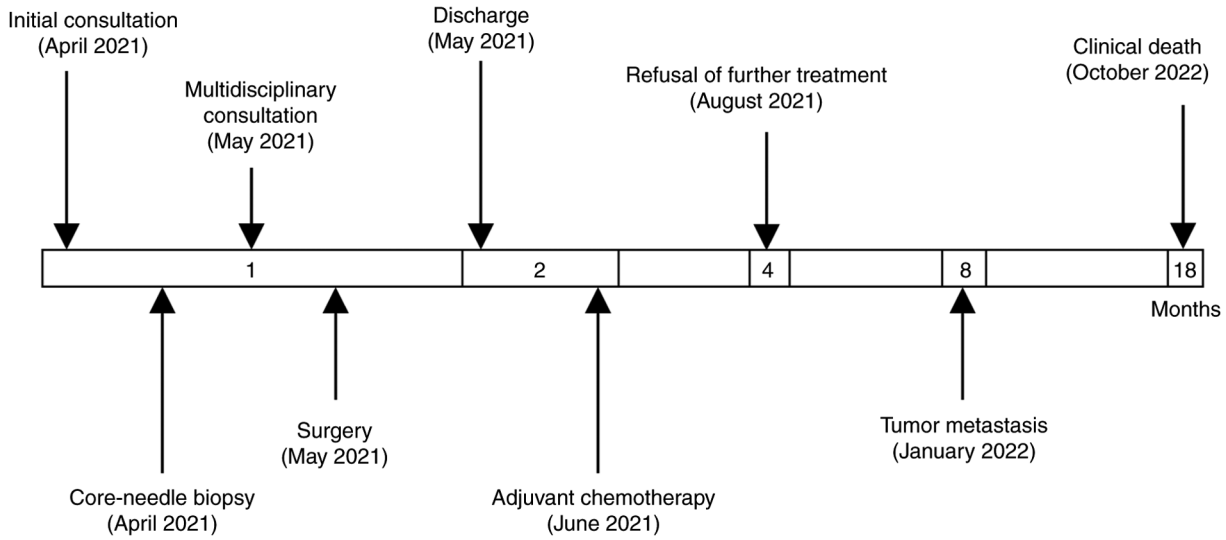

Figure 5. Timeline showing the clinical course, management and outcome of the patient with primary thoracic synovial sarcoma. This schematic outlines key events from initial presentation to death over an 18-month period, highlighting the rapid disease progression despite multimodal therapy. The interval from surgery to metastasis detection was ~8 months, and the overall survival was 18 months post-diagnosis.

Table I. Summary of the clinical characteristics of primary thoracic synovial sarcoma from selected literature.

First author, year	Number of cases	Median age (range), years	Gender (M/F)	Common symptoms	Origin	Median tumor size, cm	Primary treatment	5-year survival	(Refs.)
Pieropan, 2022	20	41 (28-54)	13/7	Chest pain: 11 (55%), Dyspnea: 5 (25%), Hemoptysis: 3 (15%), Cough: 3 (15%), Dysphagia: 2 (10%), Chest bulge: 1 (5%), Arm swelling: 1 (5%); Incidental finding: 2 (10%)	Thoracic Cavity	11.0 (IQR: 8-15)	Surgery: 20 (100%), Chemotherapy: 2 (10%), Radiotherapy: 11 (55%)	22% (OS) 1	(11)
Abdulghaffar, 2020	1	39	M	Chest pain	Chest wall	17.5	Surgery + chemotherapy	-	(18)
Wan, 2020	1	67	M	Incidental finding	Pleura	3.8	Surgery + chemotherapy	-	(19)
Choudhary, 2020	1	71	M	Dry cough, right-sided chest pain, dyspnea	Pleura	-	Chemotherapy	-	(20)
Kagawa, 2020	1	61	F	Right-sided chest pain	Lung	13	Surgery + chemoradiotherapy	Death	(21)
Kim, 2020	1	26	F	Chest pain	Chest wall	3	Surgery	-	(22)
Khalili, 2021	2	24, 29	F	Coughing, shortness of breath	Pleura	9.5/-	Surgery ± chemotherapy	-	(23)
Kumar, 2021	1	26	M	Dyspnea, cough, fever, left-sided chest pain	Pleura	-	Palliative chemotherapy and radiotherapy	-	(24)
Saad, 2021	1	14	F	Dyspnea	Mediastinum	1.4	Not specified	Death	(25)
He, 2021	13	48 (IQR: 27-58)	7/6	Chest pain: 5 (38.46%), Coughing: 8 (61.54%), Dyspnea: 6 (46.15%), Blood in sputum: 3 (23.08%), Yellow sputum: 2 (15.38%), Body examination: 1 (7.69%)	Thoracic cavity	6.0 (IQR: 5.5-9.0)	Surgery: 10 (76.92%), Chemotherapy: 10 (76.92%), Radiotherapy: 4 (30.77%), Immunotherapy: 2 (15.38%), Targeted therapy: 1 (7.69%), Knife therapy: 1 (7.69%)	Death: 7 (53.85%), Survival: 6 (46.15%)	(26)
Vishnoi, 2021	1	39	M	Chest pain	Chest wall	6	Surgery + chemoradiotherapy	Death	(27)
Patel, 2022	1	9	M	Pneumothorax	Pleura	1.5	Surgery	-	(28)
Oneglia, 2022	1	12	M	Dyspnea, cough, chest pain, pneumothorax	Lung	<5	Surgery + chemoradiotherapy	-	(29)
Shilo, 2023	1	29	F	Pneumothorax, cough	Lung	1.3	Surgery	-	(30)
Wu, 2023	1	38	F	Hemoptysis	Pleura	13.0	Surgery + anlotinib	Death	(31)
Hozain, 2023	1	36	F	Chest pain, syncope	Mediastinum	12.5	Surgery + chemoradiotherapy	-	(32)
Zang, 2023	1	18	M	Dry cough, wheezing	Pleura	7.8	Surgery + chemotherapy	-	(33)

Table I. Continued.

First author, year	Number of cases	Median age (range), years	Gender (M/F)	Common symptoms	Origin	Median tumor size, cm	Primary treatment	5-year survival	(Refs.)
Do, 2023	1	60	F	Pneumothorax	Pleura	-	Surgery	-	(34)
Idrees, 2024	1	23	F	Pain, chest wall mass	Chest wall	12.0	Surgery + chemotherapy	-	(35)
Imen, 2024	1	54	M	Left-sided chest pain, dyspnea	Pleura	6.8	Surgery	Death	(36)
Vega-Gonzalez, 2025	1	58	F	Rapid weight loss, dyspnea	Pleura	-	Chemotherapy	-	(37)
Present case, 2025	1	45	M	Cough, dyspnea	Thoracic cavity	18.5	Surgery + chemotherapy	Death	-

M, male; F, female; IQR, interquartile range; '-', data were not reported in the original study.

confirm whether residual tumor tissue remained. Given the uncertainty regarding margin clearance, adjuvant therapies such as radiation and chemotherapy are essential to reduce the risk of recurrence, particularly in cases with a high likelihood of residual disease (9).

In the broader context of sarcoma management, ongoing efforts aim to optimize systemic therapy, particularly for rare subtypes where high-level evidence is scarce (44). For the present patient with PTSS, the established first-line adjuvant regimen is doxorubicin combined with ifosfamide (42,43). However, following a multidisciplinary tumor board (MDT) discussion, a modified regimen of doxorubicin plus nedaplatin was opted for. This decision was primarily driven by concerns regarding the potential nephrotoxicity and neurotoxicity of ifosfamide, with the aim of preserving the patient's quality of life during recovery. It must be acknowledged that high-level evidence specifically supporting the efficacy of nedaplatin in SS is lacking. The consideration of a platinum-based agent in this case was grounded in evidence suggesting class activity of platinum drugs in SS. This is supported by studies reporting the treatment activity of cisplatin in SS (45), a finding corroborated by another report (46). Furthermore, cisplatin-containing regimens have demonstrated activity in advanced thoracic sarcomas (45,46). Based on this pharmacological rationale and its potentially more favorable toxicity profile, nedaplatin was selected as an alternative. This case highlights that the management of complex thoracic sarcomas often requires individualized decision-making within an MDT framework, where efficacy, toxicity and patient-specific factors must be carefully balanced (47).

Despite the benefits of conventional chemotherapy, there is a critical need to develop novel therapeutic strategies for treating advanced SS. Immunotherapy, particularly with programmed cell death-1/programmed death ligand 1 inhibitors, has shown modest efficacy as a monotherapy, and combination strategies are currently under investigation (48). Adoptive T-cell therapy, particularly targeting antigens such as NY-ESO-1 and MAGE-A4, shows promising potential for treating SS. Clinical trials have demonstrated positive results, and Food and Drug Administration-approved therapies have shown remarkable progress in treating advanced cases. Despite challenges such as tumor heterogeneity, these therapies are becoming more effective, and future treatments may improve with more potent strategies, including dual antigen targeting and checkpoint inhibitors (49). Additionally, targeted therapies such as ATR inhibitors, which target key kinases in the DNA damage response pathway, as well as BCL-2 inhibitors, are being actively explored (46,50). These emerging therapies represent the priority areas of SS research and provide hope for improving future treatment outcomes.

In the present case, the pathological staging of the tumor was T4NxM0G3 (stage III), which typically necessitates aggressive adjuvant therapy to improve both local control and overall survival (12,51-53). Regrettably, because of incomplete adherence to adjuvant therapy, primarily driven by treatment-related toxicities and, crucially, by the patient's significant financial constraints, the prognosis was adversely affected. This case illustrates the gap between optimal multimodal treatment plans and their feasible execution, highlighting that socioeconomic factors are critical determinants of outcome alongside

medical strategies. Several key insights can be drawn from this case: Improving preoperative diagnostic accuracy is crucial for considering neoadjuvant chemotherapy; early intervention, ideally at the localized disease stage, may increase the likelihood of achieving complete resection; and adherence to a full course of adjuvant therapies, which may require addressing underlying socioeconomic barriers, is essential to enhance survival outcomes.

In conclusion, PTSS is a rare and aggressive malignancy with significant diagnostic challenges. Accurate preoperative diagnosis, timely surgical intervention and comprehensive multimodal adjuvant therapy are essential for improving patient outcomes. The present case report highlights the importance of maintaining a high index of suspicion for rare sarcomas and the need for a broader diagnostic approach, including molecular analysis, to avoid misdiagnosis. Given the limited evidence and rarity of PTSS, further research is necessary to refine treatment strategies and improve patient survival. Additional multicenter studies, particularly involving neoadjuvant and targeted therapies, are required to establish more effective treatment protocols and improve survival outcomes for patients with PTSS.

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Availability of data and materials

The data generated in the present study may be requested from the corresponding authors.

Authors' contributions

BL designed the study, analyzed the data and drafted the original manuscript. JZ, MS and CL were responsible for data acquisition and the literature search. YL, MH and QC contributed to the analysis and interpretation of the data and critically revised the manuscript for important intellectual content. BL and QC confirmed the authenticity of all the raw data. All authors have read and agreed to the published version of the manuscript.

Ethics approval and consent to participate

Not applicable.

Patient consent for publication

As the patient is deceased, written informed consent was obtained from the patient's next of kin for the publication of this case report and any accompanying clinical information and images.

Competing interests

The authors declare that they have no competing interests.

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