

Received: 2026.02.09

Accepted: 2026.06.25

Available online: 2026.09.29

Published: 2026.XX.XX

Primary Synovial Sarcoma of the Thyroid Gland Presenting With Early Pulmonary Metastasis: Diagnostic Pathway, SS18 Gene Fusion Confirmation, and Clinical Course

Authors' Contribution:

Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Corresponding Author: Andrew G. Edwards, 60 Prospect Avenue, Middletown, NY 10940, USAPhone: (845)608-4960, e-mail: aedwards2@student.touro.edu**Financial support:** None declared**Conflict of interest:** None declared**Patient:** Female, 47-year-old**Final Diagnosis:** Primary synovial sarcoma of thyroid**Symptoms:** Dysphagia**Clinical Procedure:** —**Specialty:** Otolaryngology**Objective:** Rare disease

Background: Primary synovial sarcoma of the thyroid gland (PSST) is an exceptionally rare and aggressive neoplasm. Twenty-one cases have been reported in the literature to date, making this the 22nd reported occurrence. This report presents the case of a 47-year-old woman with PSST confirmed by SS18 gene fusion on fluorescence in situ hybridization (FISH), who subsequently developed pulmonary metastasis and doxorubicin-induced congestive heart failure, illustrating the aggressive clinical course of this entity.

Case Report: We report the case of a 47-year-old woman presenting with dysphagia and a left-sided neck mass. Initial FNA was non-diagnostic; repeat FNA raised concern for a spindle cell neoplasm. Left hemithyroidectomy was performed with negative margins, and SS18 gene fusion was confirmed by FISH, establishing the diagnosis of PSST. Following multidisciplinary review, adjuvant chemotherapy was deferred and neck radiation was administered. Pulmonary metastasis was discovered 6 months postoperatively. The patient received eribulin mesylate and subsequent palliative doxorubicin, and her case was complicated by doxorubicin-induced congestive heart failure. She subsequently developed progressive systemic metastatic disease, including brain and bone involvement, and died of the disease 65 months after initial presentation.

Conclusions: This case demonstrates that SS18 FISH confirmation is essential when FNA is non-diagnostic for a thyroid spindle cell mass. Despite negative margins and adjuvant radiation, early pulmonary metastasis and ultimately fatal systemic metastatic disease to the brain and bone occurred, underscoring the aggressive clinical course of PSST. Doxorubicin-induced cardiac toxicity further illustrates the systemic therapy burden. BCL-2-directed strategies remain investigational. Complete surgical resection with negative margins is the cornerstone of management.

Keywords: BCL-2 Protein • Oncology • Otolaryngology • Synovial Sarcoma • Thyroid Neoplasms

Full-text PDF: <https://www.amjcaserep.com/abstract/index/idArt/953094>

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Introduction

Primary synovial sarcoma of the thyroid gland (PSST) is an exceptionally rare [1] and aggressive neoplasm arising from mesenchymal pluripotent cells, sharing histological features with synovial sarcomas at other anatomic sites. Synovial sarcoma (SS) is a high-grade soft tissue sarcoma that accounts for approximately 5-10% of all soft tissue sarcomas, most commonly arising in the para-articular regions of the lower extremities. Head and neck SS is uncommon, representing roughly 5% of cases, with presentation most frequently in the third decade of life. The thyroid gland is an exceptionally rare primary site: PSST was first described by Kikuchi et al in 2003, and only 21 cases have been previously documented worldwide. In the published PSST series, a slight male predominance has been observed (approximately 12: 8 male-to-female ratio), with a wide age range at presentation spanning from the second to the eighth decade of life. The typical presenting symptom is a rapidly enlarging cervical mass with compressive symptoms including dysphagia, dysphonia, and dyspnea, reflecting the locally aggressive behavior of these tumors. PSST is believed to originate from ectopic mesenchymal cells within the thyroid capsule or adjacent soft tissues, rather than from follicular thyroid epithelium. This unique origin creates a significant diagnostic challenge, as PSST presenting as a thyroid mass can closely resemble spindle cell variants of more common thyroid malignancies, including anaplastic thyroid carcinoma and medullary thyroid carcinoma. Definitive diagnosis requires an immunohistochemical profile negative for TTF-1 and thyroglobulin, combined with molecular confirmation of the SS18 gene fusion by FISH or next-generation sequencing. Awareness of this entity is essential for clinicians encountering atypical spindle cell thyroid masses, as cytologic evaluation alone may be insufficient to render a definitive diagnosis. Twenty-one cases have been reported globally to date, making this the 22nd reported occurrence [1,2]. Among the cases in the published literature with available survival data, most resulted in patient death, reflecting the aggressive biological behavior of this entity [3].

Case Report

A 47-year-old woman with a history of thyroid nodules presented to her primary ear nose and throat (ENT) physician with dysphagia and a visible lump on the left side of her neck for 4 months. An ultrasound-guided fine-needle aspiration (US-FNA) was performed at the primary ENT's institution, revealing a left thyroid nodule measuring 31 × 27 × 21 mm without suspicious features. At that time, the lesion was considered likely benign based on cytologic findings, and watchful surveillance was initiated. Two months later, the nodule was visibly larger and measured 31 × 30 × 31 mm. A repeat US-FNA suggested

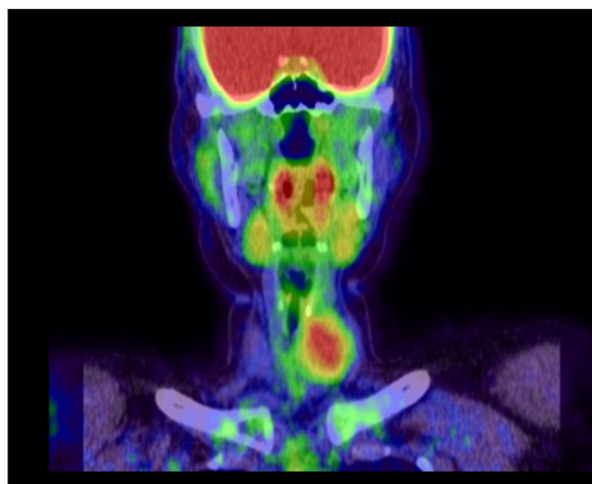


Figure 1. Coronal PET scan demonstrating fluorodeoxyglucose uptake in the left thyroid gland.

a spindle cell neoplasm, raising concern for malignancy. The patient was referred by her primary ENT physician to our clinic for a surgical second opinion. A computed tomography (CT) scan of the neck showed a 5-cm complex cyst in the left thyroid lobe with rightward deviation of the trachea. A preoperative PET scan revealed FDG uptake within the left thyroid gland (**Figure 1**) and axillary lymph nodes.

The patient underwent a left thyroid lobectomy. Intraoperatively, the tumor was found to be adherent to the lateral esophagus and the cricothyroid muscle. To ensure a negative margin, portions of both structures were resected. The tumor was extremely fragile and, despite careful dissection, fractured during the procedure. Following resection, the tracheoesophageal groove was meticulously skeletonized with successful preservation of the recurrent laryngeal nerve.

An SS18 gene fusion detected by fluorescence in situ hybridization (FISH) confirmed the diagnosis of synovial sarcoma. The specimen had a mitotic rate of 11 mitoses per 10 high-power fields and demonstrated lymphovascular invasion. It was separated from the thyroid parenchyma by a thick fibrous capsule and exhibited a biphasic growth pattern with both spindle and epithelial cell components (**Figure 2**). No significant necrosis or perineural invasion was identified. The tumor cells stained positive for vimentin, BCL-2, CD99, and EMA, focally positive for S100 and pan-keratin, and weakly focally positive for synaptophysin. They were negative for TTF-1, thyroglobulin, calcitonin, desmin, SMA, and chromogranin. Following pathologic confirmation of synovial sarcoma, the case was reviewed by a multidisciplinary tumor board. Given the histologically negative surgical margins and the absence of gross residual disease, initiation of adjuvant systemic chemotherapy was deferred. The consensus was to proceed with adjuvant neck radiation therapy with structured oncologic surveillance thereafter, consisting

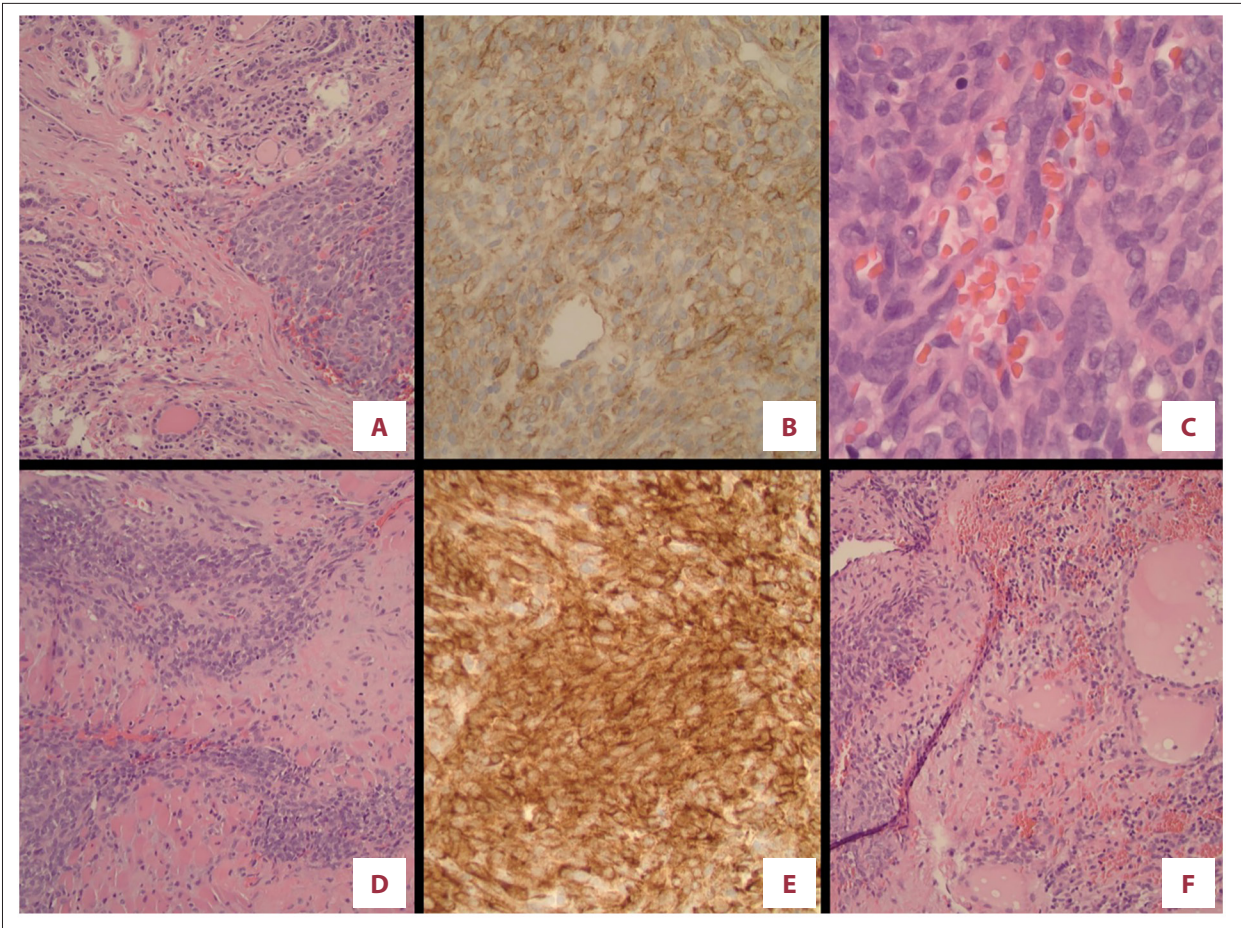


Figure 2. (A) Micrograph showing synovial sarcoma (left) with adjacent thyroid follicles (right), 100 ×. H&E stain. (B) Positive CD99 immunohistochemical stain. (C) Synovial sarcoma, high-power view, 400 × H&E. (D) Skeletal muscle invasion by synovial sarcoma, 100 × H&E. (E) Positive BCL-2 immunohistochemical stain. (F) H&E stain, 100 ×, alternate tissue section.

of contrast CT of the neck and chest every 3 to 6 months for the first 2 years, followed by annual imaging thereafter, with clinical examination at each oncology visit.

The patient completed postoperative neck radiation therapy (66 Gy in 33 fractions) 4 months after surgery. Despite no local or regional recurrence, the patient presented to the emergency department approximately 6 months postoperatively with shortness of breath and left-sided back pain. The patient disclosed she had recently undergone video-assisted thoracoscopic surgery due to pulmonary nodules that were being monitored during her oncologic treatment. Contrast CT chest showed multiple sub centimeter pulmonary nodules throughout the right hemithorax (measuring up to 0.6 cm) indicating the presence of pulmonary metastases. She was discharged and followed up with her oncologist. About 2 weeks later, repeat contrast CT chest, abdomen, and pelvis showed the nodules had grown to 1.1 × 0.9 cm (**Figure 3**). She began receiving eribulin mesylate, which was poorly tolerated, and subsequently received palliative lung radiation followed by doxorubicin. Her course was



Figure 3. Axial CTA chest at approximately 6 months postoperatively, demonstrating pulmonary metastases.

complicated by doxorubicin-induced congestive heart failure. She subsequently developed progressive systemic metastatic disease, including brain and bone involvement, and died of disease at 65 months of follow-up.

Discussion

The thyroid gland is an exceptionally rare primary site for synovial sarcoma. PSST is believed to arise from ectopic mesenchymal cells within the thyroid capsule rather than from follicular thyroid epithelium, distinguishing it biologically from all primary thyroid carcinomas. Clinically, PSST usually presents as a cervical mass with compressive symptoms—including dysphagia and dysphonia—that can closely mimic more common thyroid malignancies such as anaplastic thyroid carcinoma, spindle cell variants of papillary carcinoma, or medullary thyroid carcinoma. Fine-needle aspiration cytology may raise suspicion for a spindle cell process but is generally insufficient for definitive diagnosis of PSST, as demonstrated in the present case, in which the initial FNA was interpreted as benign and a definitive diagnosis required surgical resection and FISH-based molecular confirmation. The immunohistochemical profile is critical in narrowing the differential: characteristic positivity for BCL-2, CD99, and EMA combined with negativity for TTF-1 and thyroglobulin distinguishes PSST from thyroid-origin tumors. Molecular confirmation of the SS18 gene fusion via FISH or next-generation sequencing remains the diagnostic gold standard, and clinicians evaluating atypical spindle cell thyroid masses should incorporate SS18 FISH testing into their diagnostic algorithm when cytology is non-diagnostic or when IHC raises the possibility of a non-thyroid origin.

Review of the published PSST case series (Table 1) reveals several thyroid-specific epidemiologic and clinical features that distinguish this entity. Among the reported cases, males predominate at an approximate 12: 8 ratio to females, with patient age ranging from the second to the eighth decade of life (mean age approximately 38 years). The typical duration of symptoms prior to diagnosis is less than 6 months, although presentations as long as 5 years before diagnosis have been reported. Imaging findings are non-specific: ultrasound may reveal a cystic-solid heterogeneous mass that mimics a benign thyroid nodule, and CT imaging typically demonstrates a well-demarcated solid mass that may contain calcifications. FNA has a poor diagnostic sensitivity for PSST, with fewer than half of reported cases yielding even a suspicion of spindle cell malignancy preoperatively; in no reported case was a definitive diagnosis of SS established by FNA alone. This pattern was replicated in the present case, in which the initial FNA was non-diagnostic and definitive diagnosis required surgical resection with SS18 FISH confirmation. Regarding outcomes, of the cases with available survival data reported in the literature,

approximately one-third resulted in death from disease, with pulmonary metastasis being the predominant cause of death. This sobering outcome profile underscores the importance of early and aggressive multidisciplinary management, structured pulmonary surveillance, and timely referral to centers with sarcoma expertise.

Synovial sarcoma is defined by the genetic translocation t(X;18), which results in SS18/SSX fusion oncogenes. This fusion drives overexpression of BCL-2, an anti-apoptotic protein that blocks mitochondrial release of pro-apoptotic factors and neutralizes pro-apoptotic proteins such as BAX and BAK, thereby promoting cell survival [4]. BCL-2 overexpression has been shown to enhance synovial sarcomagenesis in animal models, and conditional genetic disruption of BCL-2 in mice reduces sarcomagenesis, confirming its contributory role in tumor development [5]. However, the relationship between BCL-2 expression and therapeutic vulnerability is more nuanced than expression alone would suggest.

Despite the prominent role of BCL-2 in sarcomagenesis, pharmacologic inhibition specific to BCL-2 has demonstrated limited direct efficacy against synovial sarcoma. Barrott et al found that BCL-2-specific inhibition had no demonstrable effect against human SS cell lines or mouse tumors, and identified BCL-xL—not BCL-2 itself—as a more tractable therapeutic target; targeting BCL-xL with the BH3 domain inhibitor BXI-72 achieved significant cytoreduction in preclinical models [5]. The limited efficacy of BCL-2-specific agents such as venetoclax in SS appears to be driven not by MCL-1 upregulation per se, but by constitutively low expression of NOXA, the endogenous inhibitor of MCL-1 [6]. In the absence of NOXA, MCL-1 remains unrestrained, conferring an MCL-1-mediated survival advantage that renders SS cells resistant to venetoclax [6]. Fairchild et al demonstrated that artificially restoring NOXA expression sensitized SS cells to venetoclax and that combined BCL-2 and MCL-1 inhibition induced tumor regression in SS patient-derived xenograft models [6].

Among the published cases of PSST with available immunohistochemical data, BCL-2 protein analysis was positive in all cases in which it was assessed [3]. In the present case, BCL-2 was likewise positive. This consistent BCL-2 positivity across reported PSST cases raises the hypothesis that BCL-2-directed combination strategies, particularly those also targeting MCL-1 or BCL-xL, may warrant investigation in this entity. However, no direct clinical evidence of therapeutic benefit from these agents was observed in the present case, and their application in PSST specifically remains entirely investigational. MCL-1 inhibitors are actively being studied across hematologic malignancies. Their development in solid tumors has been tempered by concerns over MCL-1 inhibitor cardiotoxicity [7], a risk that carries particular relevance in PSST given that patients may

Table 1. Summary of all 21 previously reported cases of primary thyroid synovial sarcoma.

Author (year) [Ref]	Age/sex	Subtype	Tumor size	Initialdiagnosis
Gupta 2025 [1]	25 M	Biphasic	~9.6 × 9.2 × 11 cm	Granulomatous thyroiditis / cystic colloid goiter (FNAC misdiagnosis)
Johnson 2025 [2]	29 F	Mono	6.5 × 4 × 3.5 cm	Malignant spindle cell neoplasm (FNA)
Ren 2023 [3]	59 F	Biphasic	5 × 4 cm	No confirmed pre-op diagnosis
Lekshmi 2023 [15]	14 M	Biphasic	NR	Spindle cell neoplasm/carcinoma (FNA)
Zahran 2022 [13]	47 M	Biphasic	NR	Oncogenic neoplasm with atypical cells (FNA)
Seyed-Alagheband 2021 [14]	43 F	Mono	NR	Undifferentiated cell carcinoma of thyroid (FNA)
Demirel 2020 [12]	26 M	Biphasic	4.5 cm	Papillary carcinoma (FNA)
Owen 2018 [11]	37 M	Mono	NR	NR
Owen 2018 [11]	47 M	Biphasic	NR	Core biopsy
Owen 2018 [11]	42 M	Mono	NR	Paraganglioma
Owen 2018 [11]	36 M	NR	NR	Synovial sarcoma
Owen 2018 [11]	30 F	NR	NR	Poorly diff. synovial sarcoma
Yadav 2018 [16]	12 M	Biphasic	NR	Malignant spindle cell tumor / MTC (FNA)
Park 2017 [17]	47 F	Mono	NR	Ovoid to spindle cells (FNA)
Bansal 2017 [18]	28 F	Biphasic	NR	Poorly diff. MTC (FNA)
Shi 2016 [10]	31 M	Biphasic	NR	Poorly differentiated tumor
Boudin 2014 [19]	55 M	Mono	7 cm	Anaplastic thyroid carcinoma (no cytology)
Ghafouri 2013 [20]	44 F	Mono	NR	NR
Ryu 2011 [9]	72 F	Mono	NR	Cystic change (FNA)
Jang 2007 [21]	15 M	Biphasic	6x5x5 cm	Benign follicular lesion (FNA)
Kikuchi 2003 [8]	60 M	Mono	6.8x6.5 cm	MTC (FNA)
Author (year) [Ref]	Surgery	SS18/SSX	Adjuvant therapy	Outcome/follow-up
Gupta 2025 [1]	Non-resectable; biopsy only; emergency tracheostomy	FISH negative (diagnosis by histomorphology + IHC)	Adriamycin x1 dose + 4 cycles chemo + 4 cycles RT (details NR)	AWD, 6 mo; pulmonary nodules + LN at presentation
Johnson 2025 [2]	TR + debulking + 11 LN resected	FISH+ (SYT-SSX1)	D&I + RT	No recurrence at 38 mo (lost to FU)
Ren 2023 [3]	TR + central cervical LN dissection	FISH+	E&I (RT refused by patient)	Alive, 9 mo
Lekshmi 2023 [15]	Lump excision	NR	RT + chemo referral	NR
Zahran 2022 [13]	Total thyroidectomy	NR	Chemoradiotherapy	Alive, 12 mo
Seyed-Alagheband 2021 [14]	TR	NR	D&I + RT	Alive at last FU

Table continued 1. Summary of all 21 previously reported cases of primary thyroid synovial sarcoma.

Author (year) [Ref]	Surgery	SS18/SSX	Adjuvant therapy	Outcome/follow-up
Demirel 2020 [12]	TR + LN dissection + partial laryngectomy (for recurrence)	FISH+	Radioiodine + chemo + targeted therapy	AWD, 41 mo; local recurrence 25 mo; PDM + bone 32 mo
Owen 2018 [11]	TR + debulking	FISH+	D&I + RT + trabectedin	DOD, 126 mo; local recurrence at 8 yr
Owen 2018 [11]	TR + bilateral LN dissection + thoracotomy	FISH+	D&I + RT	DOD, 55 mo; pulmonary metastasis 2 yr
Owen 2018 [11]	Tumor excision	NR	Lung mets excised + conventional chemo	Alive, 72 mo; pulmonary metastasis 1 yr
Owen 2018 [11]	Non-resectable	NR	D&I + RT + ATR inhibitor	NR
Owen 2018 [11]	NR	NR	NR	NR
Yadav 2018 [16]	TR	NR	NR	DOD, 7 mo
Park 2017 [17]	TR	NR	None	NED, 36 mo
Bansal 2017 [18]	TR + retrotracheal excision	NR	RT	NR
Shi 2016 [10]	TR	RT-PCR+	D&I + 50 Gy RT	NED, 34 mo; LN recurrence 2 yr
Boudin 2014 [19]	TR (intraoperative rupture; incomplete)	FISH+ (SYT rearrangement)	D&I (neoadjuvant)	AWD, 10 mo; local recurrence 15 days
Ghafouri 2013 [20]	Incomplete excision (capsule ruptured)	NR	None	NR
Ryu 2011 [9]	TR + tracheal fenestration	NR	Chemoradiation (planned; not completed)	DOD, 2 mo
Jang 2007 [21]	TR + cervical LN dissection	NR	None	NR
Kikuchi 2003 [8]	TR + LN dissection	RT-PCR+	None	DOD, 32 mo; pulmonary metastasis 18 mo

Abbreviations: M, male; F, female; Mono, monophasic; FNA, fine-needle aspiration; TR, total thyroidectomy; LN, lymph node; RT, radiotherapy; D&I, doxorubicin and ifosfamide; E&I, epirubicin and ifosfamide; NR, not reported; FISH, fluorescence in situ hybridization; mo, months; NED, no evidence of disease; DOD, died of disease; AWD, alive with disease; FU, follow-up.

have already been exposed to cardiotoxic chemotherapy regimens. In this case, the patient developed congestive heart failure during doxorubicin therapy, illustrating the compounding systemic toxicity risk that must be weighed when sequencing or combining agents in this population.

Importantly, the mainstay of treatment remains achieving negative resection margins to optimize outcomes. The present case illustrates that even with histologically negative margins and adjuvant radiation, pulmonary metastasis can occur,

and structured pulmonary surveillance should be incorporated into follow-up protocols for all PSST patients.

Conclusions

Primary synovial sarcoma of the thyroid gland is an extremely rare and aggressive neoplasm with a propensity for pulmonary metastasis. This case report demonstrates several directly observed clinical lessons: SS18 gene fusion confirmation by

FISH is essential to establish the diagnosis of PSST when cytology is inconclusive or non-diagnostic; aggressive clinical behavior—including early pulmonary metastasis and ultimately fatal systemic metastatic disease to the brain and bone—can occur despite histologically negative surgical margins and adjuvant radiation, underscoring the importance of structured pulmonary surveillance and early referral to sarcoma centers; and systemic chemotherapy carries a meaningful toxicity burden, as evidenced by doxorubicin-induced congestive heart failure in this patient. BCL-2 expression, present in this and the majority of reported PSST cases, suggests that BCL-2-directed combination strategies warrant future investigation. However, targeted BCL-2 inhibition has shown limited direct preclinical efficacy in synovial sarcoma models, and no direct evidence of therapeutic benefit was demonstrated in this case. Any such strategies remain entirely investigational. Complete surgical resection with negative margins is the cornerstone of management. Continued international case reporting is essential to accumulate sufficient outcome data for evidence-based diagnostic and treatment protocols in this rare malignancy.

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Acknowledgments

We thank the patient for providing consent to publish this report. We also thank Dr. Thomas Stockl and Ms. Amy Russell-Parlman for their assistance in obtaining the images used.

Institution Where Work Was Done

The patient was treated at Garnet Health Medical Center, 707 E Main Street, Middletown, NY 10940, USA.

Patient Consent

Consent was obtained from the patient.

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