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(CASE REPORT)

Primary cardiac monophasic synovial sarcoma mimicking cardiac fibroma: Diagnostic challenges. A case report and brief literature review

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Abstract

Primary cardiac synovial sarcoma (PCSS) is an exceptionally rare malignant mesenchymal tumor, accounting for a small minority of primary cardiac sarcomas and often requiring histopathologic and molecular confirmation because clinical and imaging findings are usually nonspecific. We report a 47-year-old female with progressive exertional dyspnea, recurrent syncope, ventricular ectopy, and evolving heart failure who was initially considered to have hypertrophic cardiomyopathy and later a septal cardiac fibroma.

Imaging demonstrated a large interventricular septal mass extending into the left ventricular outflow tract, with fibrotic enhancement and limited metabolic activity, features favoring a benign fibrous tumor but not fully excluding sarcoma. Surgical exploration revealed infiltrative myocardial involvement, and pathology demonstrated a highly cellular spindle cell neoplasm with necrosis, brisk mitotic activity, diffuse TLE1 positivity, focal epithelial marker expression, high Ki-67, and SS18-SSX2 fusion, confirming primary monophasic cardiac synovial sarcoma.

Despite gross resection with narrow margins and adjuvant ifosfamide-doxorubicin chemotherapy, the patient developed early local recurrence, pulmonary and hepatic metastases, refractory biventricular failure, and died 8.5 months after surgery. This case highlights the capacity of cardiac synovial sarcoma to mimic cardiac fibroma (CF) radiologically, the importance of maintaining malignant spindle cell neoplasia in the differential diagnosis of adult fibroma-like septal masses, and the prognostic significance of early molecular diagnosis, margin status, and intensive surveillance.

Keywords: Primary cardiac synovial sarcoma; Synovial sarcoma; Mesenchymal tumor; Cardiac fibroma; Multidisciplinary; SS18-SSX2 gene fusion

1. Introduction

Synovial sarcoma (SS) is an aggressive soft-tissue malignancy that occurs in two primary histological subtypes, monophasic and biphasic, each with distinct morphological, immunohistochemical (IHC), and molecular characteristics. Monophasic synovial sarcoma consists primarily of uniform spindle cells arranged in a fascicular pattern. The biphasic variants combine spindle cells with epithelial glandular or nested components, creating a more complex tissue architecture. SS can also present in a poorly differentiated form composed of primitive round cells. [1,2]

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SS is characterized by the t(X;18) translocation, resulting in the SS18–SSX fusion gene. Overall, SS carries a poor prognosis, with a median survival of approximately 2 years following initial diagnosis. [3] Primary cardiac involvement is exceedingly rare and poses a significant diagnostic challenge due to nonspecific presenting symptoms, including dyspnea, exertional chest pain, and pericardial effusion.

Primary cardiac synovial sarcoma (PCSS) demonstrates a male predominance and typically affects individuals between 20 and 40 years of age. [1] Preoperative diagnosis is particularly challenging, as imaging findings may mimic those of benign cardiac masses, such as cardiac fibroma. [4] Complete surgical resection is often not feasible due to anatomic constraints, contributing to a high likelihood of recurrence. Advanced imaging modalities, including contrast-enhanced cardiac CT, FDG-PET/CT, and FDG-PET/MRI, play an important role in the evaluation and characterization of disease. [5] Definitive diagnosis requires identification of the SS18 gene rearrangement, typically confirmed by FISH or RT-PCR. [1]

Current treatment strategies include surgical resection and anthracycline-based chemotherapy. When complete resection is not achievable, chemotherapy has been shown to improve overall survival. [3]

We present this case to emphasize the diagnostic difficulty in distinguishing malignant from benign cardiac masses preoperatively and to illustrate the aggressive clinical course of primary cardiac synovial sarcoma presenting as a cardiac fibroma mimic.

2. Case Presentation

2.1. Clinical Presentation

A 47-year-old woman with a two-year history of progressive exertional dyspnea and recurrent near-syncopal episodes presented to the emergency department following a witnessed syncopal event at rest. She had initially been evaluated by her primary care physician approximately 18 months prior. At that time, an electrocardiogram revealed frequent premature ventricular contractions (PVCs) with a left bundle branch block. She was subsequently referred to a cardiologist and commenced on oral metoprolol succinate for presumed idiopathic ventricular arrhythmia, with modest symptomatic improvement.

Over the ensuing year, she experienced worsening palpitations, two additional syncopal episodes, and new-onset orthopnea requiring two to three pillows at night. A transthoracic echocardiogram performed six months prior had reportedly identified "prominent septal thickening," though the study was technically limited and attributed to hypertrophic cardiomyopathy. She had been prescribed furosemide and referred to for genetic counseling. Her current presentation followed a third syncopal episode accompanied by palpitations and diaphoresis, prompting immediate emergency evaluation and subsequent hospitalization for further diagnostic workup.

2.2. Physical Examination, History, Laboratory, Imaging & Differential Diagnosis

On physical examination, the patient appeared anxious and mildly respiratory distressed, with an oxygen saturation of 94% on room air, a heart rate of 92 beats per minute with frequent irregularity, a blood pressure of 108/74 mmHg, and a respiratory rate of 20 breaths per minute. Cardiac auscultation revealed a harsh III/VI systolic ejection murmur along the left sternal border without radiation, along with an S3 gallop. Mild bilateral pitting edema was noted at the ankles. Jugular venous distension was present. No hepatomegaly was appreciated on abdominal examination.

Personal history was notable for no prior cardiac surgery or a childhood diagnosis of congenital heart disease. However, she recalled being told as a young child that she had "something on her heart" that was never formally characterized. Family history was significant for a maternal uncle who died suddenly at age 38, the cause undetermined. A paternal cousin reportedly underwent heart surgery in childhood. There was no family history of known cardiomyopathy or malignancy.

Laboratory investigations revealed mildly elevated serum brain natriuretic peptide (BNP) at 388 pg/mL, troponin I within normal limits, and a comprehensive metabolic panel without significant abnormalities. Complete blood count demonstrated mild normocytic anemia. Inflammatory markers, including erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP), were within normal limits, arguing against an inflammatory etiology.

Transthoracic echocardiography (TTE) demonstrated a large, echogenic, non-mobile mass measuring approximately 5.8 × 4.3 cm arising from the interventricular septum and extending into the left ventricular outflow tract (LVOT).

Cardiac magnetic resonance imaging (MRI) with gadolinium contrast revealed a well-circumscribed, hypointense mass on T1- and T2-weighted sequences with central late gadolinium enhancement (LGE), consistent with fibrotic tissue. The mass did not demonstrate significant vascularity or necrosis.

The radiographic differential diagnosis encompassed the following entities, listed in order of likelihood: Cardiac fibroma (CF) was considered the leading diagnosis given the imaging characteristics. CF was favored being a solitary, intramyocardial, hypointense mass with a fibrotic gadolinium enhancement pattern in the interventricular septum of a middle-aged adult, without calcification on computed tomography (CT) chest. Cardiac CT with contrast confirmed a homogeneous, non-enhancing mass with a peripheral rim of enhancement and absence of calcification, somewhat atypical for fibroma but not excluding it. Cardiac lipoma was less likely given the absence of a fat signal on T1 sequences. Rhabdomyoma was considered given familial history, but is overwhelmingly a pediatric tumor, making it improbable in this age group. Cardiac sarcoma, particularly synovial sarcoma or undifferentiated pleomorphic sarcoma, remained a critical differential given the patient's age, ventricular involvement, and progressive hemodynamic compromise. Metastatic disease to the myocardium was included, though no primary malignancy was identified on CT of the chest, abdomen, and pelvis. Inflammatory myofibroblastic tumor was also listed, particularly given its potential to mimic fibroma on imaging.

2.3. Multidisciplinary Tumor Board Discussion

Given the diagnostic ambiguity between a primary benign cardiac fibroma and a malignant primary cardiac neoplasm, the case was presented at the multidisciplinary cardiac tumor board. The board included interventional cardiologists, cardiac surgeons, cardiothoracic radiologists, nuclear medicine physicians, medical and surgical oncologists, and cardiac pathologists.

Discussion centered on several critical concerns: the atypical age of presentation for a fibroma in the absence of a confirmed childhood diagnosis; the progressive ventricular dysfunction beyond what would be expected from mechanical obstruction alone; and the absence of calcification, which is present in up to 25% of fibromas. PET-CT with 18F-FDG was ordered, which demonstrated mild but heterogeneous metabolic activity within the mass (SUVmax 3.2), raising concern for low-grade malignancy, though not conclusive. The board debated whether hemodynamic instability precluded safe percutaneous or surgical biopsy. Ultimately, the consensus recommendation was to proceed with surgical resection with intent for complete excision, with intraoperative frozen section analysis to guide the extent of resection, including possible cardiac transplant listing should malignancy be confirmed intraoperatively and complete margins not be achievable.

2.4. Management, Pathology, and Final Diagnosis

Intraoperatively, the mass was found to be a firm, grayish-white, poorly circumscribed lesion deeply infiltrating the interventricular septum and extending into the anterolateral free wall of the left ventricle. Contrary to the imaging impression of well-circumscribed margins, operative findings revealed infiltrative borders. Frozen-section analysis demonstrated highly cellular spindle-cell proliferation arranged in fascicles, with focal nuclear pleomorphism and increased mitotic figures, raising immediate concern for malignancy. The surgical team achieved grossly negative but narrow margins.

Final histopathological examination of the permanent sections revealed a highly cellular spindle cell neoplasm with moderate nuclear atypia, brisk mitotic activity (>9 mitoses per 10 high-power fields), focal necrosis, and infiltrative growth into adjacent myocardium. (**Figure 1 A, B, C**) These features were incompatible with cardiac fibroma, which characteristically demonstrates a paucicellular, hypocellular stroma with dense collagen bundles, entrapped myocytes, and absent or rare mitotic figures.

IHC was performed on a comprehensive panel. The tumor cells demonstrated strong, diffuse positivity for vimentin and focal positivity for smooth muscle actin (SMA), consistent with a mesenchymal lineage. Desmin was focally positive. CD34 was negative. The absence of S-100 protein ruled out peripheral nerve sheath tumors. MUC4 staining was performed, given the differential for low-grade fibromyxoid sarcoma, which was negative. TLE1 (transducin-like enhancer of split 1) showed diffuse nuclear positivity, a highly sensitive and specific marker for synovial sarcoma. AE1/AE3 (pan-cytokeratin) and EMA showed focal positive staining, which is typical of both biphasic and monophasic synovial sarcoma. Proliferation index (Ki-67) was high, with>50% nuclear staining. (**Figure 1 D, E**) Molecular fluorescence in situ hybridization (FISH) testing was performed for the SS18 (SYT) gene rearrangement, confirming an SS18-SSX2 gene fusion, a pathognomonic feature of SS.

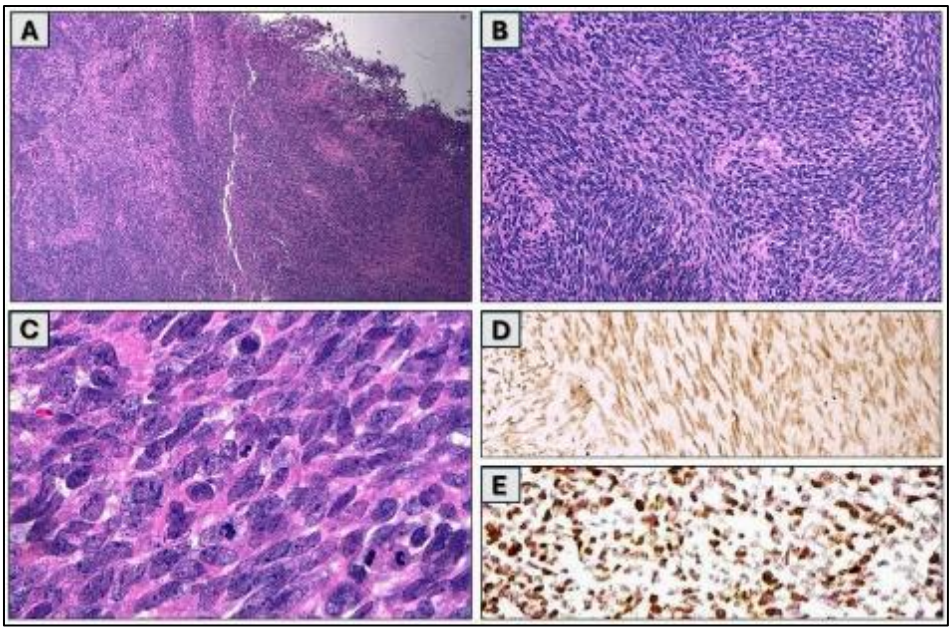
The final pathological diagnosis was: Primary Cardiac Synovial Sarcoma, Monophasic Type, with SS18-SSX2 gene fusion, intermediate-to-high grade, with narrow surgical margins and focal intramyocardial invasion.

2.5. Outcome, Follow-Up, Complications & Surveillance

The patient's postoperative course was complicated from the outset. She required a prolonged cardiopulmonary bypass time of 248 minutes. She experienced low cardiac output syndrome in the immediate postoperative period, necessitating temporary mechanical circulatory support with an intra-aortic balloon pump (IABP) for 72 hours. Complete heart block persisted, and a permanent dual-chamber pacemaker was implanted on postoperative day six. She was transferred to the cardiac intensive care unit with LVEF measured at 28% on postoperative echocardiography. She developed acute kidney injury requiring continuous renal replacement therapy (CRRT) for 11 days, from which she ultimately recovered to near-baseline creatinine.

Following a prolonged 34-day hospitalization, she was discharged on guideline-directed medical therapy for reduced EF, including sacubitril-valsartan, carvedilol, and spironolactone, in addition to anticoagulation for newly documented paroxysmal atrial fibrillation. A multidisciplinary oncology plan was formulated in consultation with sarcoma specialists. The patient received adjuvant ifosfamide-based chemotherapy in combination with doxorubicin, following the European Organization for Research and Treatment of Cancer (EORTC) 62012 trial regimen adapted for cardiac sarcoma (falling under the umbrella of soft-tissue sarcoma). She tolerated two cycles with significant toxicity, including grade 3 febrile neutropenia requiring hospitalization and dose reduction.

Surveillance cardiac MRI performed at three months post-resection identified a 1.8 cm nodular area of enhancement at the medial margin of the pericardial patch, concerning local recurrence. Repeat PET-CT confirmed FDG-avid local recurrence (SUVmax 5.6) along with two new hepatic lesions and bilateral pulmonary nodules consistent with hematogenous metastatic dissemination. Given her poor performance status, residual cardiac dysfunction, and metastatic disease, the multidisciplinary team and patient engaged in shared decision-making regarding goals of care.



1A: Low-power view showing a densely cellular, unencapsulated mass and scattered branching (staghorn) vascular pattern. (H&E stain X20); **1B:** Intermediate power view showing monotonous, uniform spindle cells growing in diffuse sheets and broad, interlacing sweeping fascicles. (H&E stain ×40).; **1C:** High-power view showing spindle cell neoplasm with distinct nuclear hyperchromasia, coarse chromatin clumping, and occasional nuclear moulding. Increased mitosis and focal presence of mast cells are seen (H&E stain X20); **1D**: Tumor cells positive for TLE1; **1E**: Tumor cells with high Ki-67 (>50%)

Figure 1 Histomorphologic features of the excised monophasic cardiac synovial sarcoma

She elected to pursue single-agent pazopanib as second-line therapy, which was administered for six weeks with minimal radiographic response and declining functional status. Serial echocardiography documented a progressive decline in LVEF to 18% with functional mitral regurgitation. The patient developed refractory heart failure with recurrent volume overload and was ultimately admitted to inpatient palliative care at seven months following her initial

cardiac surgery. She died 8.5 months following her index surgical resection, due to metastatic cardiac synovial sarcoma with refractory biventricular failure.

A formal autopsy was performed, confirming widespread metastatic synovial sarcoma involving the liver, bilateral lungs, and retroperitoneum, with local intracardiac recurrence infiltrating the right ventricular free wall and interventricular septum.

3. Discussion

3.1. Background (History, epidemiology, risk factors, and WHO classification)

Mesenchymal tumors, including SS, are notoriously difficult to classify due to their rarity of occurrence, the intrinsic complexity that does not always adhere to morphologic criteria, as well as the advanced technology required to diagnose them properly. [6] The World Health Organization (WHO) classifies synovial sarcomas as malignant soft tissue tumors of uncertain differentiation. [7] Early researchers believed that these tumors arose from joint synovium due to their proximity to joints, tendons, and bursae. However, modern histopathology has shown that these tumors typically arise from multipotent mesenchymal stem cells rather than the joint space itself. [8]

SSs account for approximately 5-10% of all soft-tissue sarcomas, underscoring their rarity. They are characterized almost exclusively by the t(X;18)(p11;q11) chromosomal translocation, which produces the pathognomonic SS18:SSX fusion gene. No inherited or environmental risk factors have been identified. Although synovial sarcoma can occur at any age, it most commonly affects adolescents and young adults, with peak incidence in the fourth decade of life. [6]

The vast majority of SS originates from the soft tissues of the extremities, near large joints such as the elbows and knees (80-95%). They can, however, arise from anywhere in the body, including the head and neck (5-10%), as well as the chest wall and viscera, including the lung, kidney, prostate, esophagus, pericardium, pleura, and heart. [4,9] Regarding metastasis, the most common site of metastasis is the lungs, accounting for 80% of distant metastases, with lymph node and bone metastasis occurring in 2-20% of cases. [4,6]

3.2. Pathogenesis, Pathophysiology

Synovial sarcoma (SS) pathogenesis is driven by a pathognomonic chromosomal translocation, t(X;18)(p11;q11). This fusion joins the SS18 gene on chromosome 18 with one of the SSX genes (usually SSX1 or SSX2) on chromosome X. The resulting SS18-SSX fusion protein disrupts transcription and chromatin remodeling by displacing the BAF47 tumor suppressor, leading to unchecked cellular proliferation and tumorigenesis. [10] While SS is common in the extremities (e.g., arms, legs), PCSS is extremely rare. However, when it does occur, the pathogenesis and cellular features remain identical to traditional SS. [9]

While the SS18-SSX fusion oncogene is the main driver of tumor initiation and growth, recent studies have shown that SS cells can survive when the SS18-SSX fusion protein is silenced or suppressed. [11] When SS18-SSX is knocked down, for example by targeted epigenetic therapies such as histone deacetylase (HDAC) inhibitors, the cancer cells undergo a compensatory survival response. The proto-oncogene FYN (an anti-apoptotic, or cell-death-preventing kinase) is identified as a direct target of SS18-SSX, which normally represses it. When fusion is suppressed, FYN is overexpressed, protecting tumor cells from apoptosis. [11,12] This explains why targeted monotherapies often fall short in SS. Because shutting down SS18-SSX triggers the FYN cell-survival pathway, the cancer cells become resistant to treatment. [13]

Because inhibiting SS18-SSX can activate compensatory survival networks (such as FYN), researchers recommend a synergistic approach. Preclinical studies show that combining SS18-SSX suppressors (such as HDAC inhibitors) with FYN inhibitors (such as PP2) significantly reduces tumor cell proliferation compared to using either therapy alone. [13] An early study suggested that PCSS may exhibit a higher rate of complex cytogenetic abnormalities, in addition to the classic t(X;18) translocation, potentially contributing to a higher metastatic tendency. [14]

While extremity SS presents as a palpable lump or limb pain, PCSS causes cardiopulmonary symptoms such as dyspnea, chest pain, arrhythmias, and cardiac tamponade due to tumor growth constricting heart function. [15]

3.3. Comparative Analysis of Our Case with Existing Literature.

The present case shared several features with reported primary cardiac synovial sarcomas, including nonspecific cardiopulmonary symptoms, arrhythmia-related manifestations, large tumor size at recognition, diagnostic reliance on TLE1 and SS18 rearrangement testing, and aggressive behavior despite multimodality therapy. [16,17] Reviews by

Wang and Li and by Coli et al. emphasized that primary cardiac synovial sarcoma remains poorly characterized because most evidence derives from isolated case reports. However, dyspnea, chest pain, palpitations, syncope, conduction abnormalities, and heart failure repeatedly appeared as presenting patterns. [3,17] Similarly, Zhou et al. described a monophasic left-sided cardiac synovial sarcoma in which atrial flutter preceded discovery of a large mass, supporting arrhythmia as a potential early clue rather than an incidental finding. [16]

This case differed from many reported tumors by its intramyocardial interventricular septal origin and close MRI mimicry of cardiac fibroma, including low T1/T2 signals and fibrotic late gadolinium enhancement. Prior series noted male predominance, atrial or pericardial involvement, and tumors often exceeding 5 cm. In contrast, our patient was a middle-aged woman with a septal lesion causing left ventricular outflow tract compromise. [1,16,18] The adverse outcome was nevertheless consistent with historical experience: Talukder et al. reported survival of less than nine months in this location, and Coli et al. found that resection and chemotherapy improved survival but did not eliminate relapse or metastatic risk. [3,19] Compared with recent cases achieving short-term control after aggressive therapy, our case suggested that fibroma-like imaging, narrow margins, myocardial invasion, CDKN2A deletion, and postoperative cardiac dysfunction may identify a particularly high-risk phenotype. [1,16,18]

4. What have we learned from This Case?

This case shows that an apparently benign, fibrotic cardiac mass in an adult should not be assumed to be a cardiac fibroma when the clinical course is progressive, arrhythmogenic, and hemodynamically significant. Key warning signs included adult onset, recurrent syncope, frequent ventricular ectopy, left ventricular outflow tract obstruction, worsening heart failure, lack of calcification, heterogeneous FDG uptake, and ventricular dysfunction out of proportion to the degree of mechanical obstruction. Together, these findings support early multidisciplinary evaluation and, when feasible, tissue diagnosis rather than reliance on imaging alone.

The case also reinforces the need to integrate morphology, IHC, and molecular confirmation for a definitive diagnosis of cardiac synovial sarcoma. Diffuse TLE1 expression, focal cytokeratin or EMA staining, high proliferative activity, and SS18-SSX fusion testing were decisive, particularly in the differential diagnosis of spindle cell cardiac tumors. [1,18] From a management perspective, narrow margins and intramyocardial invasion should trigger intensive postoperative surveillance, early referral to a specialized sarcoma program, and clear discussion of recurrence risk, systemic therapy tolerance, cardiac functional reserve, and goals of care.

Abbreviations

- Primary cardiac synovial sarcoma (PCSS)
- Synovial sarcoma (SS)
- Immunohistochemical (IHC)
- Left ventricular outflow tract (LVOT)
- Cardiac fibroma (CF)

5. Conclusion

We report a case of primary monophasic cardiac synovial sarcoma that closely mimicked cardiac fibroma, particularly because the septal mass showed fibrotic imaging features and low metabolic activity. This case adds to the limited literature by describing an interventricular septal tumor causing left ventricular outflow tract obstruction, harboring an SS18-SSX2 fusion and adverse molecular alterations, with early local recurrence, widespread metastases, and death 8.5 months after surgery despite systemic therapy. It underscores the need to consider malignancy in adult fibroma-like cardiac masses associated with progressive arrhythmia, syncope, obstruction, or heart failure, and to pursue early multidisciplinary evaluation.

Timely molecular confirmation, careful assessment of resectability, margin-conscious surgery, and close surveillance are essential in managing patients with suspected primary cardiac sarcoma.

Compliance with ethical standards

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Disclosure of conflict of interest

All authors make the following declarations:

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- Financial relationships: All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might be interested in the submitted work.

Statement of informed consent

The patient expired, and all attempts to reach the family members were unsuccessful. Therefore, the paper has been sufficiently anonymized to maintain patient confidentiality.

Data access statement

All relevant data are included in the paper.

Author contributions

All authors contributed equally to producing this manuscript.

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