Case Report

Uterine sarcoma with extensive cardiac metastatic involvement: A rare cardio oncology presentation

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ABSTRACT

A 67-year-old postmenopausal woman presented with chest pain and progressive exertional dyspnea. Multimodality cardiac imaging revealed large intracardiac masses involving the right ventricle (RV) and left ventricle with pericardial involvement, RV outflow tract obstruction, impaired RV systolic function, and moderate-gross pericardial effusion. Positron emission tomography/Computed tomography demonstrated widespread fluorodeoxyglucose-avid disease, including mediastinal and supraclavicular nodes, bilateral pulmonary nodules, and a bulky uterine subserosal mass. Ultrasound-guided uterine biopsy confirmed a high-grade spindle cell sarcoma (SMA positive, PanCK negative) with oncogenic alterations in PTEN and TP53 and CCND1 amplification. The patient received symptom-directed cardiac care and dose-adjusted palliative chemotherapy with clinical improvement in oxygen requirement and performance status. This case highlights diagnostic challenges and the need for coordinated cardio-oncology management when uterine sarcoma metastasizes to the heart.

Key words: Cardiac metastasis, Cardiac magnetic resonance imaging, Echocardiography, Positron emission tomography computed tomography, Uterine sarcoma

Cardiac metastases from uterine sarcoma are exceedingly rare, but can result in catastrophic clinical consequences when they occur. Although the heart is an unusual site for secondary involvement, the aggressive biology of high-grade uterine sarcomas permits hematogenous spread to the myocardium and pericardium. Reported cases describe involvement of the right atrium, right ventricle (RV), pulmonary arteries, and pericardium, often presenting as intracardiac masses or thrombus-like lesions [1-4]. Clinical manifestations may include right ventricular outflow tract (RVOT) obstruction, progressive right heart failure (HF), arrhythmias, or pericardial effusion with tamponade physiology, all of which significantly compromise cardiac function and limit therapeutic options, underscoring the importance of early recognition. Multimodality imaging plays a pivotal role in diagnosis and management. Transthoracic echocardiography enables rapid bedside assessment of chamber dilatation, valvular involvement, and pericardial effusion, while cardiac magnetic resonance (CMR) imaging provides superior tissue characterization, ventricular functional assessment, and precise delineation of myocardial infiltration. Positron emission tomography-computed tomography (PET-CT) further complements these modalities by defining systemic disease burden and metabolic activity, thereby guiding staging and treatment planning. Given the overlap of cardiac and oncologic considerations, management requires a multidisciplinary approach integrating cardiology, oncology, radiology, and pathology to stabilize hemodynamics and deliver individualized palliative therapy in the setting of compromised cardiac reserve [1,2].

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CASE REPORT

A 67-year-old postmenopausal woman with hypertension, hypothyroidism, and Type II diabetes presented to the cardiology department with chest pain and progressive exertional dyspnea. She denied cough or hemoptysis.

On examination, she was having an Eastern Cooperative Oncology Group (ECOG) Performance Status score of 3 and required continuous supplemental oxygen at 4 L/min; muffled heart sounds were noted on auscultation, and lung bases had bilateral crepitations.

Transthoracic echocardiography demonstrated a large mass involving the RV and pericardium, right atrial

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and RV dilatation, moderate tricuspid regurgitation, pulmonary arterial hypertension, grade I left ventricle (LV) diastolic dysfunction, preserved LV systolic function (LV ejection fraction 60%), moderate RV systolic dysfunction, and pericardial effusion. Cardiac magnetic resonance imaging (MRI) confirmed a non-dilated LV with preserved systolic function, a non-dilated RV with impaired systolic function (RV ejection fraction 30%), an RV mass infiltrating myocardium, pericardium, causing RVOT obstruction, an LV mass infiltrating adjacent myocardium and pericardium, and moderate to gross pericardial effusion with cardiac MRI score 6 (CMR score 6) (Fig. 1). PET/CT revealed multiple FDG-avid supraclavicular and mediastinal lymph nodes, bilateral pulmonary nodular opacities, a markedly FDG-avid heterodense mass involving the RV wall, interventricular septum, and LV myocardium extending into the pulmonary trunk, a bulky uterus with a large subserosal mass, and a low-grade FDG-avid umbilical subcutaneous lesion (Fig. 2). Ultrasound-guided biopsy of the uterine lesion showed fibromuscular stroma with highly atypical spindle cells, marked pleomorphism, intranuclear vacuoles, frequent mitoses and tumor giant cells, features of a high-grade sarcoma.

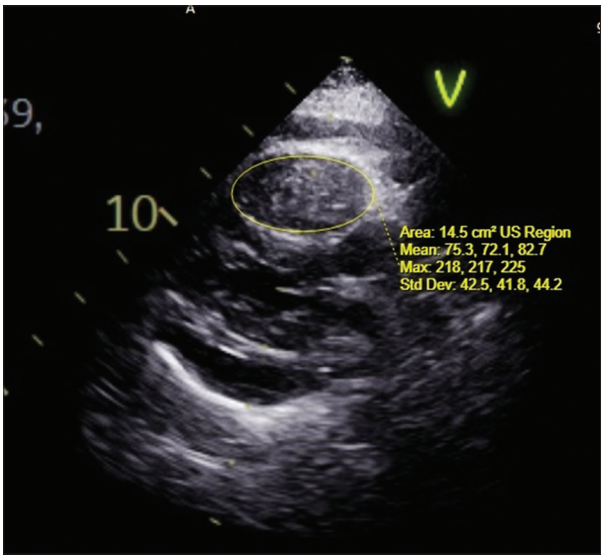

Figure 1: Transthoracic echocardiography showing a large mass involving the right ventricle and pericardium

Immunohistochemistry was PanCK negative and SMA positive, consistent with smooth muscle spindle cell origin. Next-generation sequencing identified oncogenic variants in PTEN and TP53 and CCND1 amplification. Integration of clinical, imaging, and pathologic data established the diagnosis of metastatic uterine sarcoma with extensive cardiac involvement causing RVOT obstruction.

The patient was admitted to the cardiac care unit and received diuretics and a mineralocorticoid receptor antagonist for symptomatic management of HF and pericardial effusion. Given the extent of metastatic disease and cardiac compromise, palliative systemic therapy was planned. Chemotherapy with epirubicin and ifosfamide was initiated with a 50% dose reduction of ifosfamide and strict fluid restriction (1–1.5 L/day) to mitigate cardiac risk; prophylactic low-dose intravenous heparin was administered. Concomitant cardiac therapy included diuretics, beta-blockers, and a sodium-glucose cotransporter 2 (SGLT2) inhibitor for HF with preserved ejection fraction secondary to myocardial metastases. After the first chemotherapy cycle, the patient's oxygen requirement resolved by day 10, and ECOG improved to 2; after three cycles, ECOG improved to 1. The plan was to complete six cycles followed by reassessment with PET-CT.

DISCUSSION

Cardiac metastasis from uterine sarcoma is exceedingly uncommon, with the true incidence difficult to determine due to its rarity and the frequent absence of antemortem diagnosis. Autopsy studies suggest that cardiac metastases from all malignancies occur in approximately 10–12% of cancer patients, but involvement from uterine sarcoma represents only a minute fraction of these cases [4]. Among uterine malignancies, leiomyosarcoma accounts for the majority of reported cardiac metastases, yet fewer than a few dozen cases have been documented in the literature [1-3]. Most cases are identified incidentally or during evaluation for unexplained cardiopulmonary symptoms, as clinical presentation is often non-specific. The scarcity of reported cases highlights both the rarity

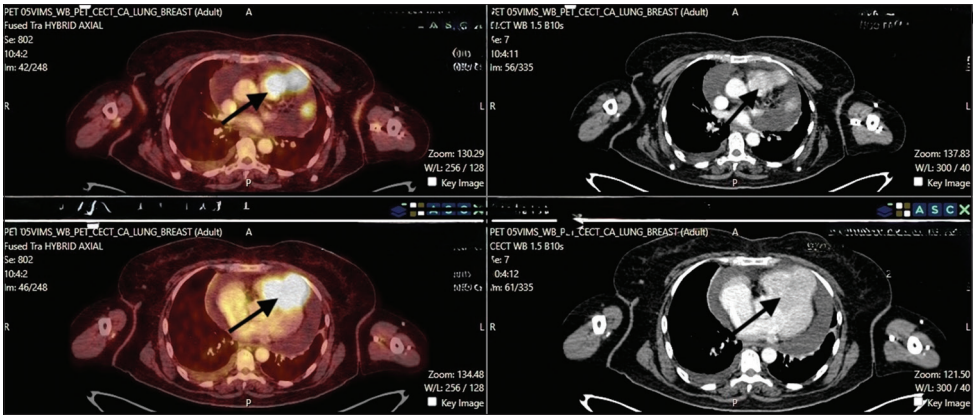

Figure 2: Positron emission tomography-computed tomography showing fluorodeoxyglucose avid heterodense mass (pointed by arrow) involving the right ventricle wall, interventricular septum, and left ventricle myocardium extending into the pulmonary trunk

of this metastatic pattern and the diagnostic challenges associated with detecting cardiac involvement during life.

Cardiac metastasis from uterine sarcoma is an exceptionally rare phenomenon, with only <1% sporadic case reports described in the literature [1-3]. The heart is generally an uncommon site for metastatic spread due to constant myocardial motion, high blood flow, and the relative resistance of the endocardial surface to tumor implantation. When cardiac involvement does occur, it typically reflects aggressive tumor biology and advanced systemic dissemination. Reported cases most often involve uterine leiomyosarcoma, with metastases identified in the right atrium, RV, pulmonary arteries, or pericardium [1,2]. Patients frequently present with non-specific symptoms such as dyspnea, chest pain, syncope, or signs mimicking pulmonary embolism, which contributes to diagnostic delay. In the present case, progressive exertional dyspnea, chest pain, hypoxia, and muffled heart sounds were early indicators of significant hemodynamic compromise due to extensive intracardiac tumor burden.

Multimodality imaging plays a central role in diagnosing and characterizing cardiac metastases. Transthoracic echocardiography remains the first-line tool for detecting intracardiac masses, assessing chamber dilatation, valvular involvement, and pericardial effusion. Cardiac MRI provides superior tissue characterization, delineates the extent of myocardial infiltration, and quantifies ventricular function, which is essential for risk stratification and therapeutic planning. PET-CT complements these modalities by identifying systemic disease burden and confirming the metabolic activity of intracardiac lesions. Similar imaging-driven diagnostic pathways have been emphasized in published cases, including the epicardial metastasis described by Krzelj *et al*. [1], where MRI and PET/CT were crucial in identifying isolated pericardial involvement before surgical management. In our case, the combination of echocardiography, CMR, and PETCT enabled precise identification of RV and LV masses, RVOT obstruction, and widespread metastatic disease, guiding the decision toward palliative systemic therapy rather than surgical intervention.

Histopathological confirmation remains essential, as imaging alone cannot reliably distinguish primary cardiac tumors from metastases. The biopsy in this case revealed a high-grade spindle cell sarcoma with SMA positivity and PanCK negativity, consistent with uterine leiomyosarcoma. Molecular profiling demonstrated PTEN and TP53 mutations and CCND1 amplification, alterations associated with aggressive tumor behavior and poor prognosis. These findings align with the extensive metastatic burden observed and are consistent with previously reported cases where cardiac metastasis typically occurs late in the disease course [2,3]. The literature suggests that survival after diagnosis of cardiac involvement is generally limited, with many patients succumbing to progressive HF or widespread metastases despite intervention, as illustrated in the case reported by Lahkim Bennani *et al*. [2]. This underscores the importance of early recognition and timely initiation of palliative therapy (Table 1).

Cardiac biopsy was not pursued in this patient, as the diagnosis of cardiac metastasis was established through a combination of multimodality imaging and histopathological confirmation from the uterine mass. In cases of suspected metastatic cardiac involvement, direct biopsy of intracardiac lesions is generally avoided due

Table 1: Literature review of cardiac metastasis from uterine sarcoma

Author, Year	Primary tumor	Cardiac site	Extracardiac metastases	Chemotherapy	Outcome	Survival or follow-up
Krzelj *et al*., 2024 [1]	Uterine leiomyosarcoma	Epicardium/ pericardium with apical LV myocardial invasion	None at detection (isolated cardiac metastasis on surveillance imaging)	Doxorubicin + dacarbazine (adjuvant; started 3 months post-operative)	No evidence of new metastases	12 months after surgery
Lahkim Bennani *et al*., 2025 [2]	Uterine leiomyosarcoma	Massive right atrial intracavitary lesion extending into IVC	Not clearly quantified in report (locally advanced disease; cardiac lesion found post-treatment)	Received chemotherapy + radiotherapy pre-op for primary; post-cardiac regimen not clearly specified	Death due to progressive HF after incomplete resection	Not stated (death reported after cardiac surgery attempt)
Present case, 2025	High-grade uterine spindle cell sarcoma consistent with leiomyosarcoma (SMA+, PanCK−); PTEN/TP53 variants; CCND1 amplification	RV + LV myocardial masses with pericardial infiltration; RVOT obstruction; moderate-gross effusion	Mediastinal + supraclavicular nodes; bilateral pulmonary nodules; umbilical subcutaneous lesion	Dose-adjusted epirubicin + reduced-dose ifosfamide (50%) with strict fluid restriction; HF therapy	Marked symptomatic improvement; oxygen independence; ECOG 3 → 1	Ongoing treatment (early response after cycle 1; functional improvement after 3 cycles)

LV: Left ventricle, RV: Right ventricle, IVC: Inferior vena cava, HF: Heart failure, ECOG: Eastern Cooperative Oncology Group, RVOT: Right ventricular outflow tract

to significant procedural risks, including arrhythmias, myocardial perforation, tamponade, and sampling error. These risks are particularly high when tumors infiltrate the right ventricular free wall or interventricular septum, as seen in this case [5,6]. Published reports indicate that cardiac metastases from uterine sarcoma can be reliably diagnosed using echocardiography, cardiac MRI, PET-CT, and biopsy of the primary or extracardiac metastatic site, without the need for invasive cardiac tissue sampling [1-3]. Given the patient's extensive systemic disease, characteristic imaging findings, and confirmed high-grade uterine sarcoma on biopsy, cardiac biopsy was deemed unnecessary and unlikely to alter clinical management.

Management of cardiac metastases is challenging due to the overlap between oncologic treatment requirements and cardiovascular vulnerability. While surgical resection or debulking has been attempted in selected cases with isolated obstructive lesions, as in the epicardial metastasis successfully resected by Krzelj *et al*. [1], most patients with widespread disease are managed with palliative chemotherapy. Anthracycline-based regimens remain the backbone of treatment for high-grade uterine sarcoma, with epirubicin and doxorubicin being the most commonly used agents [7-9]. However, anthracyclines carry well-established risks of cardiotoxicity, including dose-dependent LV dysfunction, arrhythmias, and exacerbation of preexisting HF. In patients with intracardiac metastases and impaired RV function, these risks are magnified.

In this case, the decision to administer epirubicin with a reduced dose of ifosfamide was guided by the patient's compromised cardiac reserve, RVOT obstruction, and the need to avoid fluid overload. Ifosfamide typically requires aggressive hydration to prevent hemorrhagic cystitis, but such hydration can precipitate decompensated HF in patients with elevated right-sided pressures. Therefore, strict fluid restriction (1–1.5 L/day) and a 50% dose reduction of ifosfamide were implemented to balance oncologic efficacy with cardiac safety. Mesna uroprotection was provided while minimizing fluid volume, an approach supported by limited case-based evidence in cardio-oncology practice. The patient also received beta-blockers, diuretics, and SGLT2 inhibitors to optimize HF management during chemotherapy. Prophylactic anticoagulation with low-dose heparin was appropriate, given the hypercoagulable state associated with sarcoma and the presence of an intracardiac tumor mass. Remarkably, after the first chemotherapy cycle, the patient demonstrated significant symptomatic improvement, including discontinuation of supplemental oxygen and improvement in ECOG performance status. This favorable response mirrors the limited literature suggesting that carefully individualized chemotherapy can produce meaningful palliation even in the setting of extensive cardiac involvement [1-3]. Survival in patients with cardiac metastasis from uterine sarcoma is generally poor, with most published reports describing outcomes ranging from a few weeks to several months after diagnosis, depending on tumor burden, cardiac involvement, and response to systemic therapy. Median survival after diagnosis of cardiac metastasis is 3–12 months, and 1 year survival is <30–40% in most reported cases. Close follow-up is essential, as these patients are at risk for rapid clinical deterioration due to progressive RV dysfunction, arrhythmias, pericardial effusion, or worsening obstruction of cardiac inflow or outflow tracts. In contemporary practice, management is best delivered within a dedicated cardio-oncology framework, which facilitates coordinated monitoring of cardiac function, optimization of HF therapy, and safe administration of potentially cardiotoxic chemotherapy. Serial echocardiography, periodic cardiac MRI when feasible, and functional assessment using ECOG or New York Heart Association classification form the cornerstone of follow-up. In this patient, improvement in performance status and oxygen requirement after chemotherapy highlights the potential for meaningful palliation when cardiac and oncologic care are closely integrated [10]. Continued follow-up with both oncology and cardio-oncology teams is planned to monitor treatment response, detect early signs of cardiac decompensation, and guide decisions regarding further systemic therapy or supportive care.

CONCLUSION

Cardiac metastasis from uterine sarcoma is an exceptionally rare, late-stage manifestation that poses major diagnostic and therapeutic challenges. This case highlights the indispensable role of multimodality imaging in defining intracardiac disease and guiding safe, individualized management without the need for high-risk cardiac biopsy. Despite extensive biventricular involvement and RVOT obstruction, tailored cardio-oncology-directed chemotherapy achieved meaningful palliation and functional improvement. Early recognition, multidisciplinary collaboration, and vigilant follow-up remain crucial to optimize care and preserve quality of life in patients with this uncommon but devastating presentation.

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