Vascular Health and Risk Management

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CASE REPORT

Dual Left Atrial Masses Causing Inflow Obstruction: A Rare Presentation of Primary Cardiac Intimal Sarcoma

Xian Zhang[1,*], Yuejian Zhuo[2,*], Dongdong Zhang[1–3]

[1]Department of Hematology, Postgraduate Union Training Base of Xiangyang No. 1 People's Hospital, School of Medicine, Wuhan University of Science and Technology, Xiangyang, 441000, People's Republic of China; [2]Department of Oncology, Xiangyang No. 1 People's Hospital, Hubei University of Medicine, Xiangyang, 441000, People's Republic of China; [3]Department of Oncology, Xiangyang Central Hospital, Hubei University of Arts and Science, Xiangyang, People's Republic of China

*These authors contributed equally to this work

Correspondence: Dongdong Zhang, Department of Oncology, Xiangyang Central Hospital, Hubei University of Arts and Science, Xiangyang, People's Republic of China, Tel +8615072278600, Email zhangdongdong@whu.edu.cn

Aim: Primary cardiac intimal sarcoma is a rare entity with a poor prognosis, often requiring distinction from other undifferentiated cardiac sarcomas.

Case Presentation: A 55-year-old female presented with symptoms of heart failure. Imaging identified dual left atrial masses compromising mitral inflow and pulmonary venous return. Surgical excision was performed, and morphological analysis revealed a high-grade spindle cell sarcoma. Crucially, the diagnostic dilemma was resolved through molecular testing; immunohistochemistry showed diffuse overexpression of MDM2 and CDK4, and fluorescence in situ hybridization (FISH) confirmed the amplification of the *MDM2* and *CDK4* loci.

Conclusion: This case illustrates the diagnostic utility of MDM2 and CDK4 as specific biomarkers for cardiac intimal sarcoma. It reinforces the necessity of a multidisciplinary approach involving advanced molecular pathology to ensure accurate classification and appropriate management of rare cardiac malignancies.

Keywords: primary cardiac intimal sarcoma, left atrial mass, MDM2 amplification, CDK4, rare cardiac malignancies

Introduction

Primary cardiac tumors represent a rare pathological entity, with an autopsy incidence ranging from only 0.001% to 0.03%. While the majority (approximately 75%) are benign neoplasms such as myxomas, malignant tumors constitute the remaining 25%, with metastatic involvement being significantly more prevalent than primary malignancy.[1,2] Among these, primary cardiac intimal sarcoma (PCIS) stands out as an exceptionally rare and aggressive mesenchymal neoplasm. Although typically originating in the large vessels of the systemic or pulmonary circulation—such as the pulmonary artery and aorta—primary occurrences strictly arising within the cardiac chambers are exceedingly infrequent and sparsely reported in the literature.[3]

Clinically, PCIS present a diagnostic conundrum due to their non-specific symptomatology, which often mimics common cardiovascular conditions ranging from dyspnea and orthopnea to pulmonary edema and thromboembolic syndrome. Although multimodal imaging techniques such as echocardiography and cardiac magnetic resonance are pivotal in identifying space-occupying lesions and suggesting malignant features like poor mobility, heterogeneous echogenicity, and myocardial invasion, precise histological characterization remains beyond the scope of radiologic assessment. Consequently, definitive diagnosis relies heavily on postoperative histopathological and immunohistochemical analysis. Specifically, the amplification of the *MDM2* and *CDK4* genes has emerged as a distinct molecular hallmark observed in over 70% of cases, which is critical for distinguishing intimal sarcoma from other undifferentiated cardiac sarcomas.[4,5]

https://doi.org/10.2147/VHRM.S596709
Received: 19 January 2026
Accepted: 11 March 2026
Published: 17 March 2026

Therapeutic management remains a formidable challenge. Complete surgical resection with negative margins is the cornerstone for achieving extended survival, with evidence suggesting that patients undergoing radical surgery have a survival duration double that of non-surgical candidates.[6] However, the intricate anatomical involvement of myocardial structures, valves, and heart chambers frequently precludes complete excision. Furthermore, the disease is characterized by a dismal prognosis, typically involving a median survival of approximately one year driven by high rates of local recurrence and systemic metastasis to sites including the lungs, kidneys, and lymph nodes.[1,7–9]

Herein, we report a rare case of primary cardiac intimal sarcoma presenting with dual left atrial masses, confirmed by *MDM2* and *CDK4* amplification. Following surgical resection, the patient received adjuvant chemotherapy combined with targeted therapy and achieved a progression-free survival of nearly seven months. This case highlights the critical role of molecular profiling in distinguishing this entity from other cardiac masses and underscores the necessity for early identification and a multidisciplinary therapeutic approach to improve clinical outcomes in this devastating disease.

Case Presentation

A 55-year-old female presented to the Department of Cardiology on May 3, 2024, with an 8-day history of chest tightness and dyspnea accompanied by low-grade fever, chills, fatigue, and a productive cough. She had a significant medical history including a 20-year duration of hypertension and a 6-year history of diabetes mellitus. Upon admission, laboratory evaluation revealed an elevated B-type natriuretic peptide (BNP) level of 197.3 pg/mL, whereas myocardial enzyme profiles and cardiac troponin I (cTnI) remained within normal limits. Transthoracic echocardiography (TTE) revealed two distinct solid masses within the left atrium (Figure 1A and B). Mass A protruded into the mitral valve orifice, causing obstruction, while Mass B partially impeded pulmonary venous drainage. Subsequent contrast-enhanced chest computed tomography (CT) corroborated these findings, visualizing two hypodense masses; the larger lesion measured 5.2×6.6 cm^2 and demonstrated delayed enhancement (Figure 1C, D and Figure 2A, B).

Given the extensive tumor burden, the patient was transferred to the Department of Cardiothoracic Surgery. On May 11, 2024, she underwent tumor resection under cardiopulmonary bypass. Intraoperative exploration confirmed tumor infiltration within the left atrial cavity; specifically, Mass A protruded into and obstructed the mitral orifice, while Mass B extended into the ostia of the superior and inferior pulmonary veins. The procedure involved radical resection of the left atrial tumors, atrial septal defect repair, and valvuloplasty of the mitral and tricuspid valves. Histopathological examination demonstrated a high-grade malignant neoplasm characterized by poorly differentiated, spindle-shaped tumor cells arranged in a fascicular pattern with marked nuclear atypia (Figure 3A and B). Immunohistochemical analysis revealed strong, diffuse positivity for MDM2 and CDK4 (Figure 3C and D). Fluorescence in situ hybridization (FISH) confirmed the amplification of the *MDM2* and *CDK4* genes (Figure 3E and F), establishing a definitive diagnosis of primary cardiac intimal sarcoma.[10]

Postoperative CT imaging confirmed complete resection without residual disease (Figure 2C and D). From July 5 to December 23, 2024, the patient received eight cycles of adjuvant chemotherapy consisting of the AI regimen (Epirubicin and Ifosfamide). Upon completion of chemotherapy, maintenance therapy was initiated with the multi-targeting tyrosine kinase inhibitor anlotinib, administered orally at a dose of 12 mg once daily.[11] Follow-up evaluations indicated stable disease (Figure 2E and F). However, a surveillance CT scan on March 1, 2025, revealed round, hypodense shadows adjacent to the left side of the main pulmonary artery and the right side of the ascending aortic root (Figure 2G–J). Given the clinical history, these findings were suggestive of multiple metastases. Concurrently, the patient developed chest tightness and dyspnea, which were exacerbated by exertion and progressively worsened over three days. Emergency laboratory testing upon readmission revealed a significantly elevated BNP level of 2370.8 pg/mL, severe thrombocytopenia with a platelet count of 23 × 10^9/L, and a hemoglobin level of 103 g/L. Symptomatic treatment, including cardiotonics, diuretics, bronchodilators, and platelet transfusion, alleviated her respiratory symptoms; however, the platelet count showed no significant improvement. Considering the risks of hemorrhage and cardiotoxicity associated with anti-angiogenic therapy in the setting of severe thrombocytopenia and heart failure, anlotinib was permanently discontinued. Following rapid disease progression and intolerance to further surgical or cytotoxic interventions, the patient was transitioned to palliative care. She succumbed to refractory heart failure on March 18, 2025, with an overall survival of 9 months. Serial imaging surveillance is illustrated in Figure 2. The comprehensive timeline of diagnosis and management is presented in Figure 4.[12]

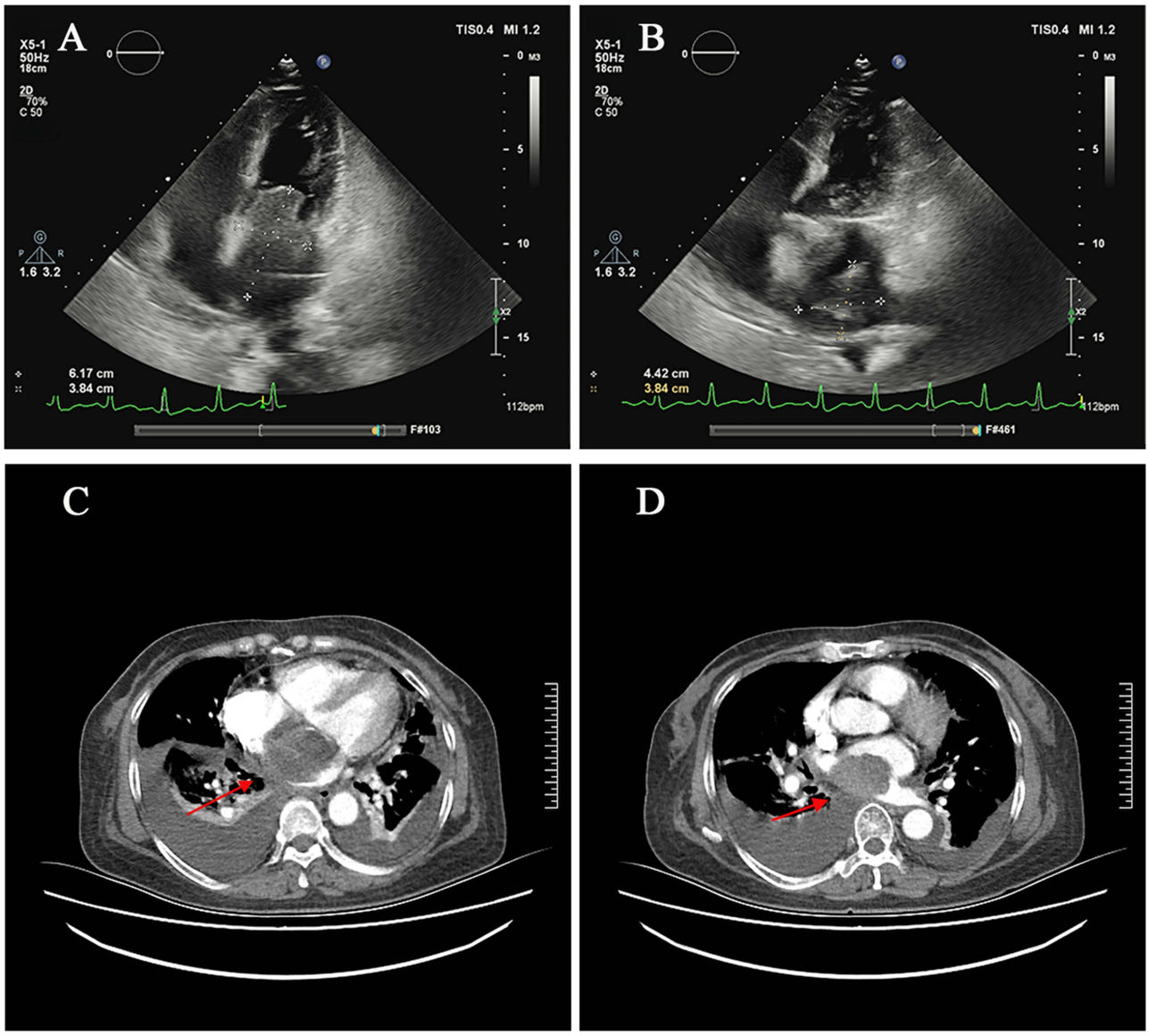

Figure 1 Preoperative multimodal imaging characteristics. (**A** and **B)** Transthoracic echocardiography reveals two slightly hypoechoic masses attached to the left atrial wall and the atrial roof. (**C** and **D**) Contrast-enhanced chest computed tomography (CT) demonstrates corresponding hypodense filling defects within the left atrium, exhibiting moderate, heterogeneous delayed enhancement.

Discussion

PCIS is an extremely rare and aggressive malignancy arising from the endocardium or the intimal layer of the great vessels. Among primary cardiac tumors, it represents a distinct subtype predominantly affecting the left atrium. Its clinical presentation is notoriously nonspecific, frequently mimicking heart failure, arrhythmias, embolic events, or constitutional symptoms. This lack of specificity often leads to misdiagnosis or delayed intervention, contributing to the perilous prognosis associated with the disease.[13] Consistent with existing literature, the case presented herein underscores the rapid progression and high invasiveness of this neoplasm, emphasizing that early detection followed by prompt surgical intervention remains the cornerstone of management.

Given the clinical presentation of a left atrial mass resulting in mitral valve obstruction, a rigorous differential diagnosis is imperative. The diagnostic spectrum encompasses infective endocarditis,[8] metastatic neoplasms,[14] cardiac lymphomas,[15] and primary cardiac tumors,[16] ranging from benign entities such as myxomas[17] and fibromas[18,19] to malignancies including angiosarcomas and intimal sarcomas. Since all these entities possess the potential to manifest

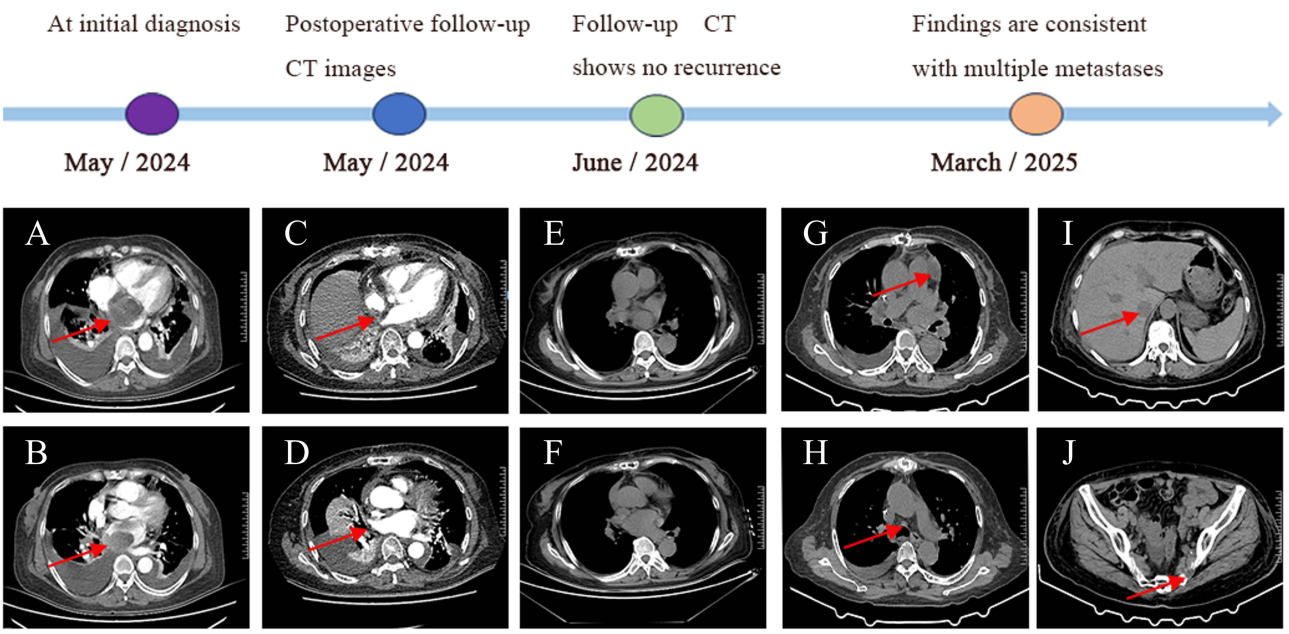

Figure 2 Postoperative radiological surveillance timeline. (**A** and **B)** Preoperative baseline imaging (for detailed characterization, refer to Figure 1). (**C** and **D**) Initial postoperative CT scan in May 2024 confirms complete tumor resection (red arrows) with no evidence of residual disease. (**E** and **F)** Follow-up CT scan in June 2024 shows no signs of local recurrence. **(G-J)** Surveillance imaging in March 2025 reveals widespread metastases (indicated by arrows), including hypodense nodules adjacent to the left pulmonary artery and the ascending aorta, an osteolytic lesion in the left iliac bone, and a new hepatic metastasis.

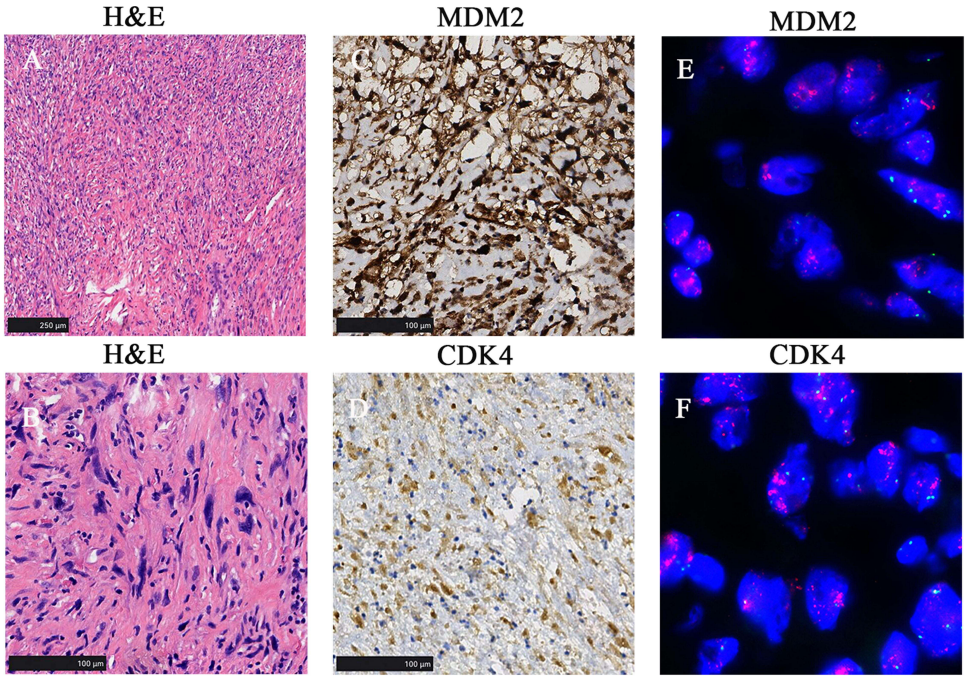

Figure 3 Histopathological and molecular characterization of the resected tumor. (**A** and **B**) Hematoxylin and eosin (H&E) staining reveals a high-grade malignant mesenchymal neoplasm composed of poorly differentiated spindle cells with marked nuclear atypia, arranged in a storiform pattern (original magnification ×100). (**C** and **D**) Immunohistochemical analysis demonstrates strong, diffuse nuclear positivity for MDM2 (**C**) and CDK4 (**D**) (original magnification ×200). (**E** and **F**) Fluorescence in situ hybridization (FISH) confirms the amplification of both *MDM2* **(E)** and *CDK4* **(F)** genes (original magnification ×200).

https://doi.org/10.2147/VHRM.S596709

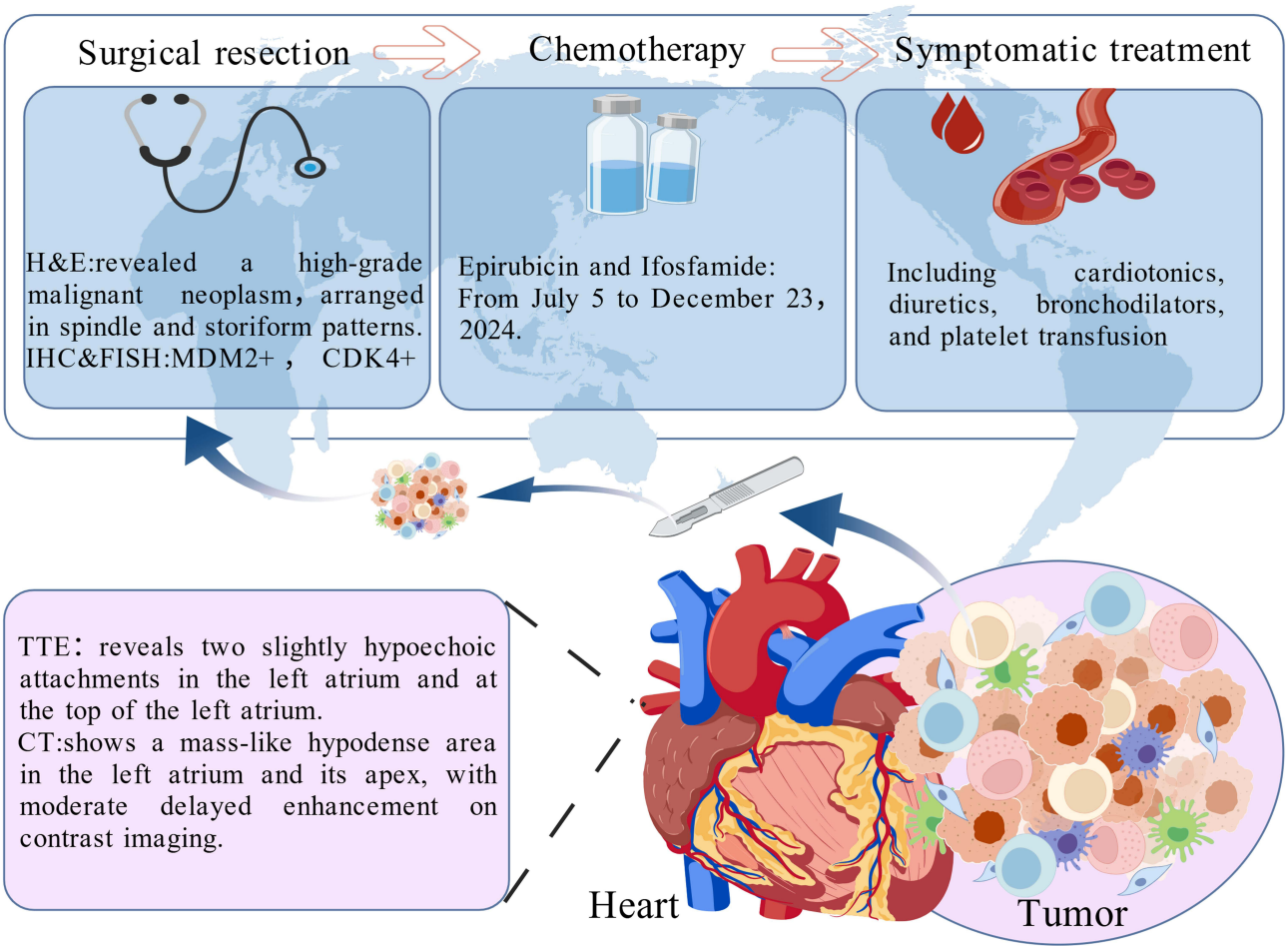

Figure 4 Clinical timeline. A Chronological overview of the patient's diagnosis, surgical treatment, adjuvant therapy, and subsequent disease progression. (Created with BioGDP.com).

within the left atrium and compromise valvular hemodynamics, a stepwise exclusion strategy was adopted in the present case. Clinically, infective endocarditis is characteristically associated with systemic manifestations, notably fever and elevated inflammatory markers;[20] the absence of these signs in our patient effectively precluded an infectious etiology. Furthermore, the absence of a prior oncological history significantly diminished the probability of metastatic disease. From a radiological perspective, benign lesions such as cardiac myxomas and fibromas typically present as well-circumscribed, pedunculated masses lacking delayed enhancement. Conversely, the tumor in this case demonstrated aggressive morphological features inconsistent with these benign pathologies. Ultimately, comprehensive histopathological and molecular evaluations definitively excluded alternative malignancies, including cardiac lymphomas and angiosarcomas, thereby substantiating the rare and complex diagnosis of primary cardiac intimal sarcoma.

Multimodal imaging and histopathological evaluation serve as the dual pillars of diagnosis. Advanced imaging modalities, particularly cardiac magnetic resonance, are indispensable for characterizing tissue properties and defining tumor boundaries.[21] However, definitive diagnosis relies on histopathology and, increasingly, molecular profiling. Recent genomic landscape analyses have elucidated that PCIS is characterized by distinct copy number variants, most notably the concomitant amplification of *MDM2* and *CDK4* on chromosome 12, often accompanied by *CDKN2A/B* losses.[22] This specific pattern of cell cycle dysregulation serves as a hallmark molecular signature, effectively distinguishing intimal sarcoma from other vascular malignancies such as angiosarcomas.[8] Crucially, these genetic alterations provide a mechanistic rationale for precision medicine. Beyond serving as diagnostic biomarkers, they identify the CDK4/6-MDM2 axis as an actionable therapeutic target,[23] representing a promising avenue for the development of novel targeted interventions.

https://doi.org/10.2147/VHRM.S596709

Regarding therapeutic management, radical surgical resection with clear margins remains the cornerstone of treatment and the only intervention offering the potential for long-term survival. In the present case, surgical intervention successfully relieved the hemodynamic obstruction and facilitated histopathological confirmation, thereby demonstrating both its palliative and diagnostic utility. However, the clinical benefit of adjuvant therapies remains constrained. While radiotherapy may contribute to local disease control, its utility is severely limited by the potential for dose-dependent myocardial toxicity.[24] Similarly, conventional chemotherapy regimens typically yield suboptimal response rates.[25] Anlotinib combined with chemotherapy may be effective for managing MDM2-amplified PCIS.[26,27] However, the long-term administration of anlotinib is associated with intrinsic cardiotoxicity, a risk that is significantly exacerbated when combined with anthracycline-based chemotherapy.[28,29] The rapid disease progression observed in this patient, despite adjuvant therapy, underscores the aggressive biological nature of cardiac intimal sarcoma and the inadequacy of current systemic treatments, highlighting an urgent need for novel therapeutic strategies. Emerging evidence indicates that immune checkpoint inhibitors, particularly those targeting the PD-1 axis, can elicit durable clinical responses in intimal sarcoma. Such therapeutic benefits appear to be driven by specific features of the tumor microenvironment, including PD-L1 overexpression and the presence of immune infiltrates such as tertiary lymphoid structures.[22,30] Furthermore, current research is increasingly focusing on synergistic strategies; the integration of immunotherapy with radiotherapy[31] or chemotherapy[32] holds significant potential to potentiate the anti-tumor immune response and overcome resistance. Consequently, these multimodal approaches warrant rigorous investigation as a future standard of care for this aggressive malignancy. Consequently, these multimodal approaches warrant rigorous investigation as a future standard of care for this aggressive malignancy.

Several limitations of this study must be acknowledged. First, as a single-center case report, the findings are inherently anecdotal and may not fully capture the heterogeneous clinical behavior of PCIS. Second, the rarity of the disease precludes the possibility of large-scale randomized controlled trials, forcing clinicians to rely on retrospective case series for treatment decisions. Consequently, a standardized treatment algorithm is currently lacking. Third, the rapid progression and high mortality rate associated with this malignancy often result in short follow-up periods, limiting the assessment of long-term complications or the delayed effects of adjuvant therapies. Future multi-center registries and molecularly driven clinical trials are essential to overcome these limitations and establish more robust management guidelines.

Conclusion

In conclusion, PCIS is a diagnostic chameleon characterized by high malignancy and a poor prognosis. Optimal management requires a multidisciplinary approach integrating clinical acumen, advanced imaging, and molecular pathology. While radical surgery remains the mainstay of treatment, it is rarely curative on its own. Improving patient outcomes will depend on a deeper understanding of the molecular mechanisms driving this disease, specifically targeting pathways such as *MDM2*, to develop more effective adjuvant immunotherapies and targeted agents.

Data Sharing Statement

The data is available from upon request from the two corresponding authors.

Ethics Committee Approval

This study was approved by the Ethics and Scientific Committee of Hubei University of Medicine (XYYYE20240074) and was performed according to the Good Clinical Practice Guidelines and the Helsinki Declaration. Institutional approval was obtained from Xiangyang No.1 hospital for the publication of the case detail.

Consent for Publication

Written informed consent was obtained from the patient for the publication of this case report and any accompanying images prior to her death.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Funding

This study was supported by Hubei Provincial Natural Science Foundation of China (Grants number: 2025AFD796).

Disclosure

The authors declare no competing interests in this work.

References

1. Yafasova A, Møller JE, Smerup MH, Dahl JS, Fosbøl EL. Case report of rare highly aggressive cardiac tumour: the intimal sarcoma. *Eur Heart J Case Rep*. 2023;7(1):ytad004. doi:10.1093/ehjcr/ytad004
2. Butany J, Nair V, Naseemuddin A, Nair GM, Catton C, Yau T. Cardiac tumours: diagnosis and management. *Lancet Oncol*. 2005;6(4):219–228. doi:10.1016/S1470-2045(05)70093-0
3. Andersen KF, Someh NS, Loft A, Brittain JM. Primary Cardiac Intimal Sarcoma Visualized on 2-[(18)F]FDG PET/CT. *Diagnostics*. 2020;10(9):718. doi:10.3390/diagnostics10090718
4. Jimbo N, Komatsu M, Itoh T, Hirose T. MDM2 dual-color in situ hybridization (DISH) aids the diagnosis of intimal sarcomas. *Cardiovas Pathol*. 2019;43:107142. doi:10.1016/j.carpath.2019.07.001
5. Van Dievel J, Sciot R, Delcroix M, et al. Single-Center Experience with Intimal Sarcoma, an Ultra-Orphan, Commonly Fatal Mesenchymal Malignancy. *Oncol Res Treat*. 2017;40(6):353–359. doi:10.1159/000476036
6. Ibrahim A, Luk A, Singhal P, et al. Primary intimal (spindle cell) sarcoma of the heart: a case report and review of the literature. *Case Rep Med*. 2013;2013:461815. doi:10.1155/2013/461815
7. Braams NJ, Kaffka Genaamd Dengler SE, Rutten E, de Boer K. Left atrial spindle cell sarcoma: a case report. *Eur Heart J Case Rep*. 2019;3(1):ytz005. doi:10.1093/ehjcr/ytz005
8. Koelsche C, Benhamida JK, Kommoss FKF, et al. Intimal sarcomas and undifferentiated cardiac sarcomas carry mutually exclusive MDM2, MDM4, and CDK6 amplifications and share a common DNA methylation signature. *Mod Pathol*. 2021;34(12):2122–2129. doi:10.1038/s41379-021-00874-y
9. Zhu X, Wang L, Zhu Y, et al. Extramedullary Plasmacytoma: long-Term Clinical Outcomes in a Single-Center in China and Literature Review. *Ear Nose Throat J*. 2021;100(4):227–232. doi:10.1177/0145561320950587
10. Nistor C, Carsote M, Cucu AP, et al. Primary Cardiac Intimal Sarcoma: multi-Layered Strategy and Core Role of MDM2 Amplification/Co-Amplification and MDM2 Immunostaining. *Diagnostics*. 2024;14(9):919. doi:10.3390/diagnostics14090919
11. Li T, Dong Y, Wei Y, et al. First-Line Anlotinib Treatment for Soft-Tissue Sarcoma in Chemotherapy-Ineligible Patients: an Open-Label, Single-Arm, Phase 2 Clinical Trial. *Clin Cancer Res*. 2024;30(19):4310–4317. doi:10.1158/1078-0432.CCR-23-3983
12. Jiang S, Li H, Zhang L, et al. Generic Diagramming Platform (GDP): a comprehensive database of high-quality biomedical graphics. *Nucleic Acids Res*. 2025;53(D1):D1670–d6. doi:10.1093/nar/gkae973
13. Ullah M, Waidyanatha S, Stamos K, Tsanaxidis N. Left ventricular intimal sarcoma: a case report. *Eur Heart J Case Rep*. 2024;8(5):ytae072. doi:10.1093/ehjcr/ytae072
14. Lin Z, Xiao H, Liu J, et al. Clinical features and prognosis of cardiac metastatic tumors. *BMC Cancer*. 2023;23(1):1235. doi:10.1186/s12885-023-11733-1
15. Aranda-Michel E, Sultan I. Commentary: primary cardiac lymphoma: chasing rarity. *J Thoracic Cardiovasc Surg*. 2022;164(2):583–584. doi:10.1016/j.jtcvs.2020.09.116
16. Kumari N, Bhandari S, Ishfaq A, et al. Primary Cardiac Angiosarcoma: a Review. *Cureus*. 2023;15(7):e41947. doi:10.7759/cureus.41947
17. Griborio-Guzman AG, Aseyev OI, Shah H, Sadreddini M. Cardiac myxomas: clinical presentation, diagnosis and management. *Heart*. 2022;108(11):827–833. doi:10.1136/heartjnl-2021-319479
18. Imai T, Shimoi T, Kawai A, Yonemori K. Diagnosis and treatment of cardiac tumors. *Med Oncol*. 2025;42(4):110. doi:10.1007/s12032-025-02661-0
19. Nakagawa-Kamiya T, Mori M, Ohira M, et al. Intimal Sarcoma: an Extremely Rare Case of a Left Atrial Tumor with Partial Obstruction of the Mitral Orifice. *CASE*. 2021;5(2):93–96. doi:10.1016/j.case.2020.12.009
20. Li M, Kim JB, Sastry BKS, Chen M. Infective endocarditis. *Lancet*. 2024;404(10450):377–392. doi:10.1016/S0140-6736(24)01098-5
21. Tüdös Z, Hutyra M. Cardiac magnetic resonance in cardiac angiosarcoma-Which contrast-enhancement pattern is typical? *Echocardiography*. 2018;35(2):287. doi:10.1111/echo.13802
22. Wang B, Chen R, Yin H, et al. Multi-omics analysis revealed potential use of immunotherapy and CDK4/6 inhibitors in intimal sarcoma. *Front Immunol*. 2025;16:1668537. doi:10.3389/fimmu.2025.1668537
23. Koyama T, Shimizu T, Kojima Y, et al. Clinical Activity and Exploratory Resistance Mechanism of Milademetan, an MDM2 Inhibitor, in Intimal Sarcoma with MDM2 Amplification: an Open-Label Phase Ib/II Study. *Cancer Discovery*. 2023;13(8):1814–1825. doi:10.1158/2159-8290.CD-23-0419
24. Isambert N, Ray-Coquard I, Italiano A, et al. Primary cardiac sarcomas: a retrospective study of the French Sarcoma Group. *Eur J Cancer*. 2014;50(1):128–136. doi:10.1016/j.ejca.2013.09.012
25. Randhawa JS, Budd GT, Randhawa M, et al. Primary Cardiac Sarcoma: 25-Year Cleveland Clinic Experience. *American Journal of Clinical Oncology*. 2016;39(6):593–599. doi:10.1097/COC.0000000000000106

26. Liang G, Zhang Q, Luo Y, Zhao Y, Luo B. Efficacy of anlotinib and chemotherapy combination as neoadjuvant therapy in the treatment of pulmonary artery intimal sarcoma: a case report. *Front Oncol*. 2025;15:1507281. doi:10.3389/fonc.2025.1507281
27. Zhong S, Luo Y, Fei Y, Zhang S. Primary cardiac endosarcoma: a case report. *J Med Case Rep*. 2025;19(1):447. doi:10.1186/s13256-025-05538-y
28. Wang ZM, Zhang SL, Yang H, et al. Efficacy and safety of anlotinib, a multikinase angiogenesis inhibitor, in combination with epirubicin in preclinical models of soft tissue sarcoma. *Cancer Med*. 2020;9(10):3344–3352. doi:10.1002/cam4.2941
29. Zhao S, Wang P, Yin F, et al. Cardiovascular toxicity associated with the multitargeted tyrosine kinase inhibitor anlotinib. *Tumori*. 2023;109(2):186–196. doi:10.1177/03008916221084362
30. Ribeiro MF, Demicco EG, Razak ARA. Clinical activity of pembrolizumab in refractory MDM2-amplified advanced intimal sarcomas. *Therapeut Adv Med Oncol*. 2024;16:17588359241250158. doi:10.1177/17588359241250158
31. Callaghan CM, Seyedin SN, Mohiuddin IH, et al. The Effect of Concurrent Stereotactic Body Radiation and Anti-PD-1 Therapy for Recurrent Metastatic Sarcoma. *Radiat Res*. 2020;194(2):124–132. doi:10.1667/RADE-20-00017
32. Liao H, Fang Y, Li D, et al. Tislelizumab combined with GT chemotherapy for intimal sarcoma of inferior vena cava: a case report. *Medicine*. 2024;103(21):e38056. doi:10.1097/MD.0000000000038056