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CASE REPORT

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A complex case of right heart masses in a leukemia patient: a case report

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Abstract

Background A patient with acute myeloid leukemia (AML) presented with a cardiac mass of unknown nature. This case underscores the importance of careful monitoring and a multidisciplinary approach in managing and differentiation of rare cardiac complications in leukemia patients. It aims to improve diagnostic accuracy and therapeutic outcomes in similar challenging scenarios. This case report discusses a 33-year-old male who was initially diagnosed with Acute Myeloid Leukemia (AML). During medical check-ups before allogeneic hematopoietic stem cell transplant (allo-HSCT), cardiac ultrasound revealed several mobile and homogenous masses of unidentified nature in his right atrium and right ventricle. The lesions presented gradually increasing calcification of the capsule, the nature of these masses remains unknown.

Case presentation The patient was diagnosed with Acute Myeloid Leukemia and achieved complete remission following multiple chemotherapy cycles. From a leukemia treatment perspective, an allo-HSCT was needed as soon as possible. However, several masses were found in his right heart before the transplant. A series of tests were performed to determine the nature of the cardiac mass. His echocardiograms and MRI revealed persistent mobile and nodular masses with a calcified capsule in the right atrium and right ventricular lateral wall, and no signals changes of the mass between MRI first-pass perfusion and delayed enhancement. Which complicated the differential diagnosis. Finally, considering the need for leukemia treatment, allo-HSCT was performed after extensive workup, including echocardiography, MRI, and PET/CT, which ruled out leukemic infiltration, typical infectious vegetation, and primary or metastatic cardiac tumors. The cardiac masses were first discovered during pre-transplant screening in April 2022, approximately 5 months after initial AML diagnosis in November 2021. At present, more than 2 years after transplantation, follow-up imaging examination of the masses revealed gradually increasing calcification, but of a still unknown nature.

Conclusions The case of this 33-year-old male with AML and concomitant cardiac masses highlights a complex challenge in his diagnosis and treatment. Despite extensive imaging and multidisciplinary consultations, including echocardiography, MRI, and PET/CT, the exact nature of these calcified, mobile nodular masses in the right atrium

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and right ventricular lateral wall remains elusive. Their persistence and atypical imaging characteristics underscore the need for continued research and collaboration to elucidate their underlying pathology.

Keywords Acute myeloid leukemia, Cardiac masses, Thrombus, Anticoagulation therapy, Differential diagnosis, Echocardiography, Cardiac MRI

Background

The differentiation of intracardiac masses remains one of the most important diagnostic dilemmas in clinical cardiology, especially in patients with hematologic malignancies. The correct identification of cardiac masses is significant because of their different etiologies-thrombotic, infectious, neoplastic, and inflammatory-which vary significantly in their treatment approaches. In AML patients, intracardiac masses create difficult management decisions; in particular, this may apply to any time-critical interventions such as allo-HSCT.

This case report presents a 33-year-old male diagnosed with Acute Myeloid Leukemia AML type M2a (based on FAB (French-American-British) classification). Initially presenting with bleeding points on his limbs and fatigue in November 2021, his condition was confirmed as AML through the accurate leukemia diagnosis using bone marrow MICM. MICM stands for Morphology, Immunology, Cytogenetics, and Molecular Biology. His disease had a poor response to induction chemotherapy, presenting the characteristics of refractory leukemia. Allogeneic HSCT is the only curative option for patients with primary refractory disease [1] and is advised to be performed as soon as possible after leukemia remission. However, upon completion of the pre-transplant examination, cardiac lesions of an unknown nature in the right atrium and right ventricle were found. These lesions exhibited heterogeneous characteristics on imaging, prompting further investigation to ascertain their etiology, necessitating a multidisciplinary approach to his treatment.

Case presentation

In November 2021, the patient presented to another hospital with complaints of fatigue and the appearance of petechiae with a diameter of less than 2 mm, scattered on the limbs. Laboratory investigations revealed a white blood cell count of 29.1 × 10^9/L, hemoglobin level of 57 g/L, and platelet count of 17 × 10^9/L.

Diagnostic workup

A bone marrow examination was conducted on November 24, 2021. Bone marrow smear showed 74.5% of blasts, indicative of acute non-lymphocytic leukemia M2a (based on FAB classification). Bone marrow biopsy confirmed highly diffuse proliferation of blasts. Flow cytometry immunophenotypic analysis revealed blasts representing 74.4% of nucleated cells, expressing markers CD34, CD117, and CD38, and partial expression of HLA-DR and CD13, while negative for a panel of other markers including CD15, CD7, CD19, and CD33. Cytogenetic analysis demonstrated a karyotype of 47, XY, +4, dup (17) (q23q25) [14]/ 46, XY [3]. Molecular testing for leukemia fusion genes was negative, while mutation analysis identified TP53, EZH2, GATA2, and KMT2A SNPs, and a low-frequency FLT3-TKD mutation (mutation frequency 2.06%). These findings confirmed a diagnosis of AML. According to European Leukemia Net (ELN) risk stratification [1], the group was identified as an intermediate-risk group.

Initial treatment and response

The patient underwent initial induction chemotherapy with an IA regimen (Idarubicin 20 mg on days 1–2, 15 mg on day 3, and Cytarabine 200 mg on days 1–7) starting on November 30, 2021. Bone marrow morphology assessment after the recovery of bone marrow hematopoietic function revealed a blast count of 87%. the second induction chemotherapy regimen DAC + CAG (decitabine in combined with aclacinomycin, cytarabine and G-CSF) [2] was administered on December 27, 2021, with Decitabine 25 mg on days 1–5, Aclarubicin Hydrochloride 20 mg on days 1–5, Cytarabine 18 mg twice daily on days 1–10, and G-CSF 300–400 μg on days 0–10, resulting in a reduced blast count of 48%. The third induction CLAG chemotherapy (Cladribine + Cytarabine + G-CSF) [3] commenced on February 1, 2022, with Cladribine 9 mg on days 1–5, Cytarabine 2 g on days 1–5, and G-CSF 300 μg on days 0–5. This regimen achieved a significant reduction in bone marrow blasts to 3.5%, with flow cytometry indicating minimal residual disease (MRD) at 5.71%. In view of the efficacy achieved by CLAG regimen, a subsequent fourth induction chemotherapy (CLAG) was given on March 14, 2022, resulting in complete remission with flow MRD < 0.001%. His disease was classified as refractory leukemia based on a poor response to induction therapy (refractory leukemia is defined as non-complete remission after two standard induction chemotherapy treatments [4]).

Complications and further management

In February 2022 after the third induction chemotherapy, the patient experienced right-sided chest pain which happened during breathing without fever or cough, and a chest CT on February 25, 2022, (Fig. 1) revealed a fungal infection in the right lung.

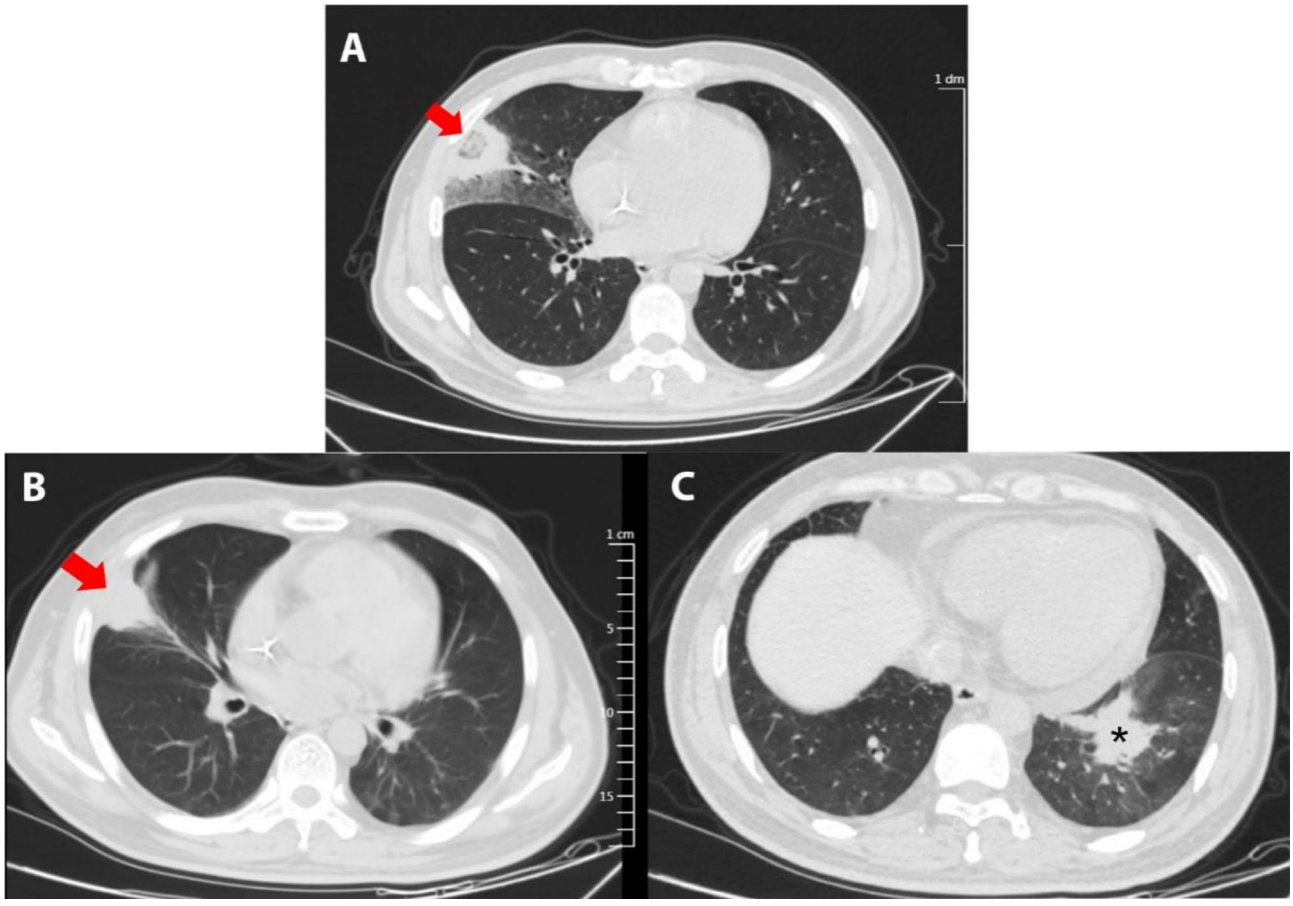

Fig. 1 Chest CT scan: **A**. February 25, 2022. **B**, **C**. April 1, 2022. A chest CT scan conducted on February 25 showed patchy consolidation with central and peripheral ground glass opacities in the lateral segment of the middle lobe of the right lung (indicated by the red arrow) and the anterior basal segment of the lower lobe of the left lung (indicated by the black asterisk)

After more than a month of antifungal based treatment(Amphotericin B lipid complex for a month, followed by Posaconazole tablets), chest pain relief and a chest CT scan on April 1, 2022, showed that the lesion in the middle and lower lobes of the right lung had shrunk compared to before, with central consolidation and absorption of ground glass opacities around it along with the emergence of a new left lung lesion. Tuberculosis-related tests of blood specimens and sputum (including TB-DNA, TB-lgG, T-SPORT, and sputum smear antacid staining) were conducted which did not support tuberculosis. According to imaging characteristics, negative serum inflammatory markers, and the patient being asymptomatic, it was still considered a fungal-based infection foci.

CT-guided left lung biopsy was performed to determine the nature of the lung lesions, (Fig. 2) also hoping to find clues about the intracardiac masses, and lung puncture pathology report was as follows:

Granulomatous lesions of lung tissue with focal alveolar epithelial hyperplasia. Histochemical staining: PAS (Periodic acid-Schiff) (-), TB (Toluidine blue) (-).

Further complexity arose with the discovery of multiple nodular masses in the right atrium and right ventricular lateral wall on echocardiography performed pre-transplantation on April 2, 2022, (Fig. 3) which was absent in a previous echocardiography performed by a local hospital (when the patient was diagnosed with leukemia). In order to clarify the nature of the cardiac mass, allo-HSCT was postponed and MRI, PET-CT, and other diagnostic evaluations were completed.

We communicated multiple times with cardiologists and surgeons in our hospital, and we concluded that interventional heart mass biopsy is difficult and has had a low success rate in our hospital. Because the mass sways with the heartbeat open heart surgery is traumatic and risky, and from the perspective of treating acute myeloid leukemia, there is not enough time to recover from heart surgery; if heart surgery is performed to clarify the nature of the heart mass, it would delay chemotherapy or hematopoietic stem cell transplantation and lead to a high risk of leukemia recurrence.

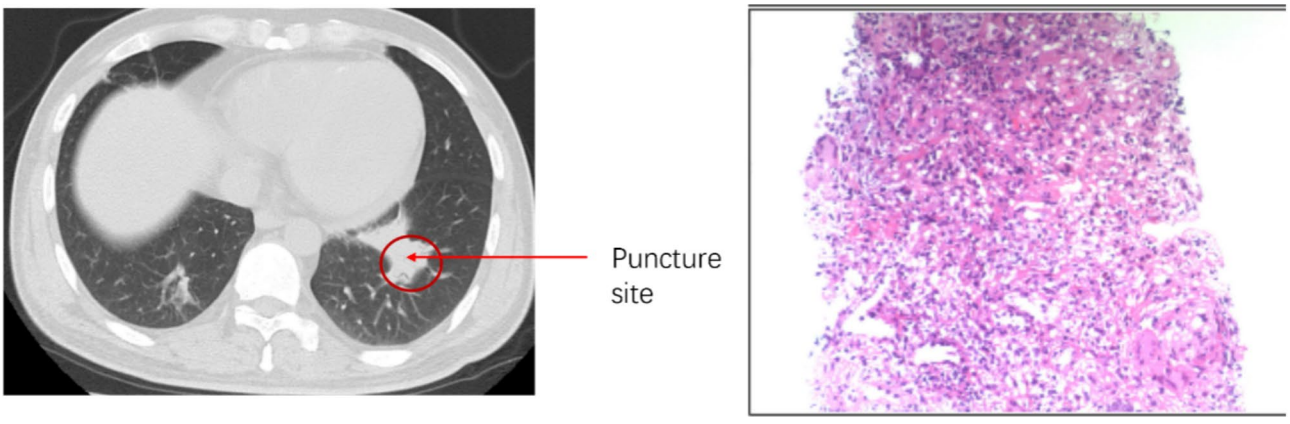

Fig. 2 Pathology of left lung puncture on April 24, 2022. Granulomatous lesions observed which could suggest sarcoidosis

An MRI on April 4, 2022, (Fig. 4) showed multiple nodular lesions in the right atrium and right ventricular lateral wall.

The nature of these masses was enigmatic, as they presented without delayed enhancement and displayed a capsule of calcification. A comprehensive workup, including a PET/CT was conducted on April 6, 2022 (Fig. 5) which didn't mention abnormalities in the heart (Because there is no blood vessel formation in the mass and it has no blood flow, the mass is not visible.) but only indicated increased glucose metabolism in pulmonary lesions, consistent with lung infection.

Following a multidisciplinary consultation involving cardiology, imaging, and hematology specialists, it was determined that a biopsy for pathological diagnosis is necessary to ascertain the nature of the cardiac mass. It was noted that performing a less invasive approach for pathological biopsy would be challenging. Based on the medical history, this cardiac mass appears to be newly developed (with no abnormal findings observed during the initial cardiac ultrasound at leukemia diagnosis), and there has been no impact on heart function. Given its imaging characteristics, there is a high likelihood of intracardiac thrombosis. Anticoagulation therapy with low-molecular-weight heparin therapy was initiated on April 9, 2022 for suspected cardiac thrombi.

Transplant and post-transplant course

Given the risk of recurrence of refractory leukemia and identification of an HLA-matched sibling donor, allo-HSCT was planned alongside continuous anticoagulation treatment.

The patient underwent allo-HSCT with peri-transplant management including broad-spectrum antibiotics, antifungal prophylaxis with Posaconazole, and low molecular heparin anticoagulation with dose adjustment according to platelet count. Echocardiograms continued to monitor the cardiac masses during and after transplant, with no significant changes observed.

Current status and follow-up

Low-molecular-weight heparin anticoagulant therapy began on April 9 2022, and continued until July 14, 2022, for a total of 14 weeks. Follow-up echocardiography showed no change in the size and number of cardiac lesions along with a gradual calcification pattern. (Fig. 6) It appears to refute the diagnosis of intracardiac thrombosis and the escalating calcification leads us to suspect the diagnosis of the cardiac masses to be a cardiac calcified amorphous tumor (CAT).

In addition, the patient was continued on a regimen of antifungal medication until 100 days after transplantation. The pulmonary lesion gradually dissipated during this time, as evidenced by repeated chest CT scans.

A sequential series of CT images in the mediastinal window shows the temporal progression of the lesion [see Additional file 8].

Discussion and conclusion

Cardiac masses: an enigmatic entity

This case presents an unusual clinical challenge in determining the nature of multiple masses identified within the right heart. Despite extensive diagnostic evaluations and anticoagulation treatment for 14 weeks, there was no significant change in the lesion. This may disprove the thrombus differential diagnosis. The etiology of these cardiac masses remains undetermined, prompting an open inquiry to the scientific and medical community for further insights.

Diagnostic uncertainties and differential considerations

Thrombosis

The leading hypothesis for the cardiac masses is thrombosis. Thrombotic events are not uncommon in hematological malignancies [5]. On the one hand, repeated

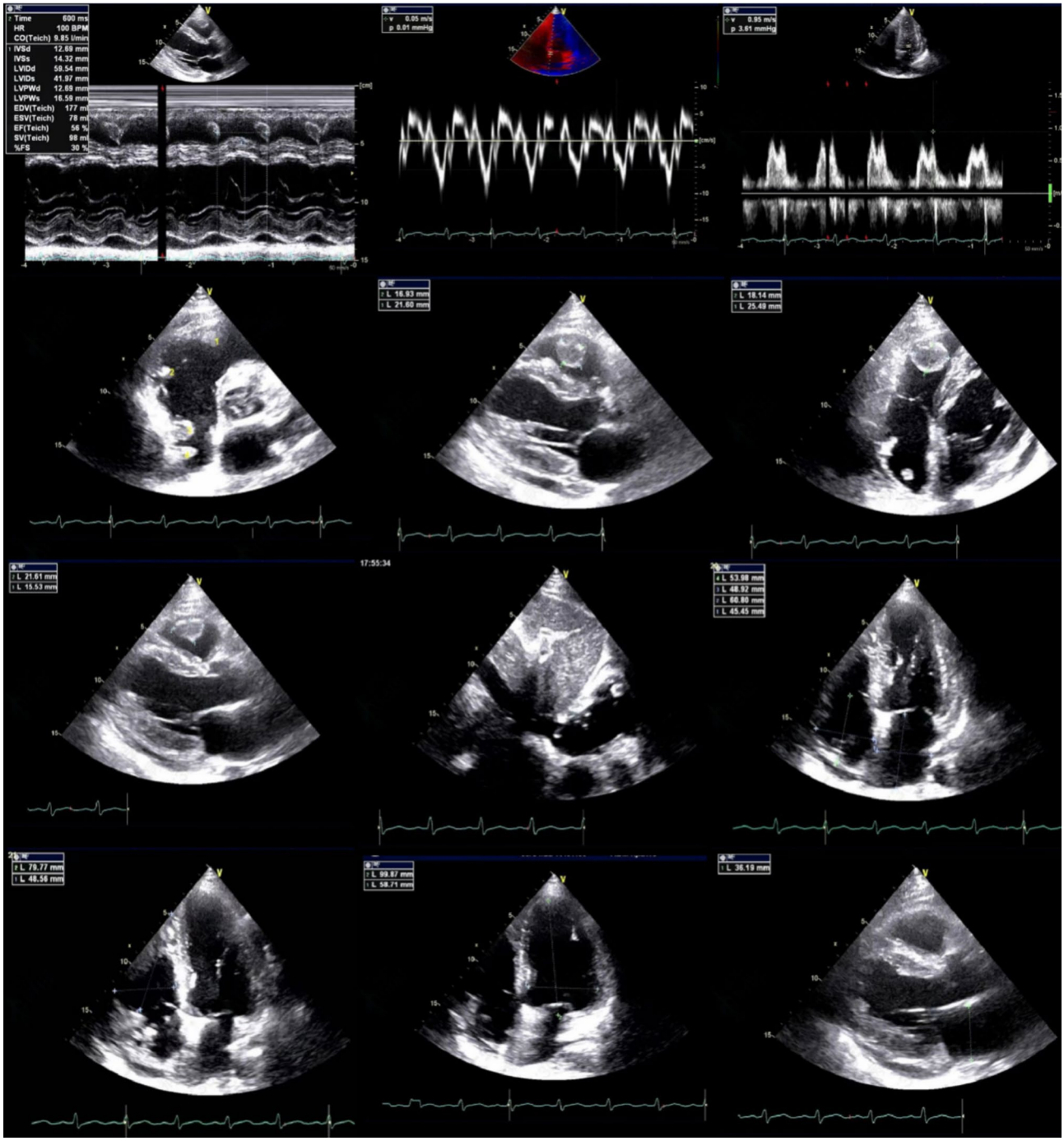

Fig. 3 Echocardiography: Multiple mass attachments can be seen on the inner membrane surface of the right ventricular wall, right roof wall, and right ventricular wall from the inferior vena cava orifice. The mass can be enveloped by a capsule and oscillates with the movement of the heart. The largest one is located on the anterior wall of the right ventricle, with a size of about 17 × 21 × 25 mm. Cardiac chambers were dilated (left atrium 45mmx61mm, left ventricle 59mmx100mm, right atrium 49mmx54mm, right ventricle 48mmx80mm). A small amount of pericardial effusion can be observed. There was no functional impairment because of the masses and the patient remained asymptomatic

chemotherapy can significantly damage the vascular endothelium, leading to the activation of the coagulation cascade and subsequent thrombosis formation [6]. On the other hand, blood flow vortices at the near heart end of the central venous catheter contribute to thrombus development [7]. Echocardiographic findings of mobile, homogenous masses within the right atrium and ventricle support this possibility. However, there were no obvious abnormalities on the coagulation examination and despite anticoagulant therapy, the size and characteristics

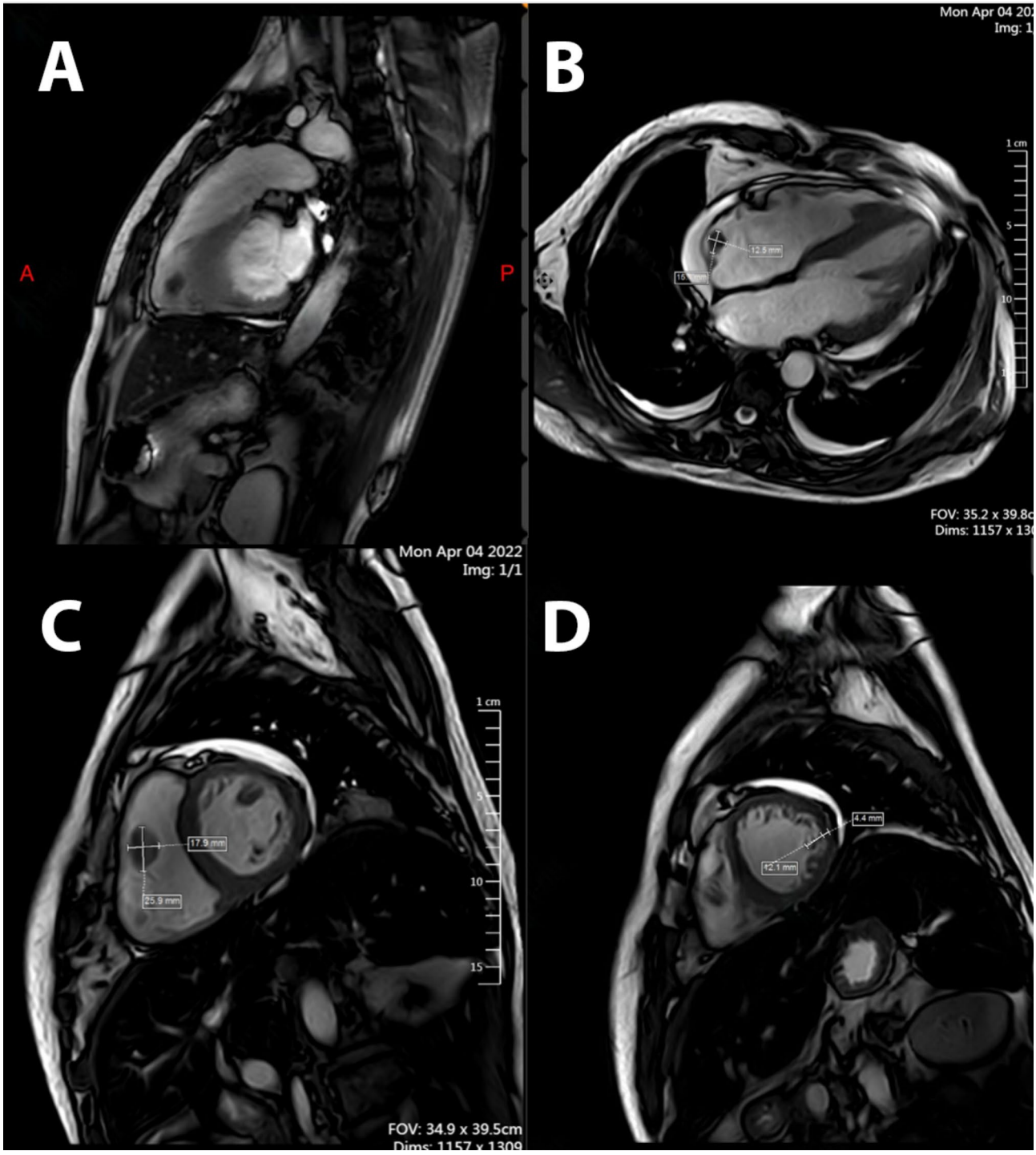

Fig. 4 2022-4-4 Enhanced Cardiac MRI: demonstrating multiple nodular lesions in right heart chambers. (**A**) Sagittal T1-weighted image showing heterogeneous nodular lesions in the right atrium and right ventricular wall. (**B**) Axial view with measurement of largest nodular mass. (**C**, **D**) Additional axial sequences showing left ventricular non-compaction in mid and distal segments. lesions showed mixed signals and no obvious signal changes between MRI first-pass perfusion and delayed enhancement. Two additional movie files and two additional pictures included show this in more detail [see Additional files 1, 2, 3 and 4]

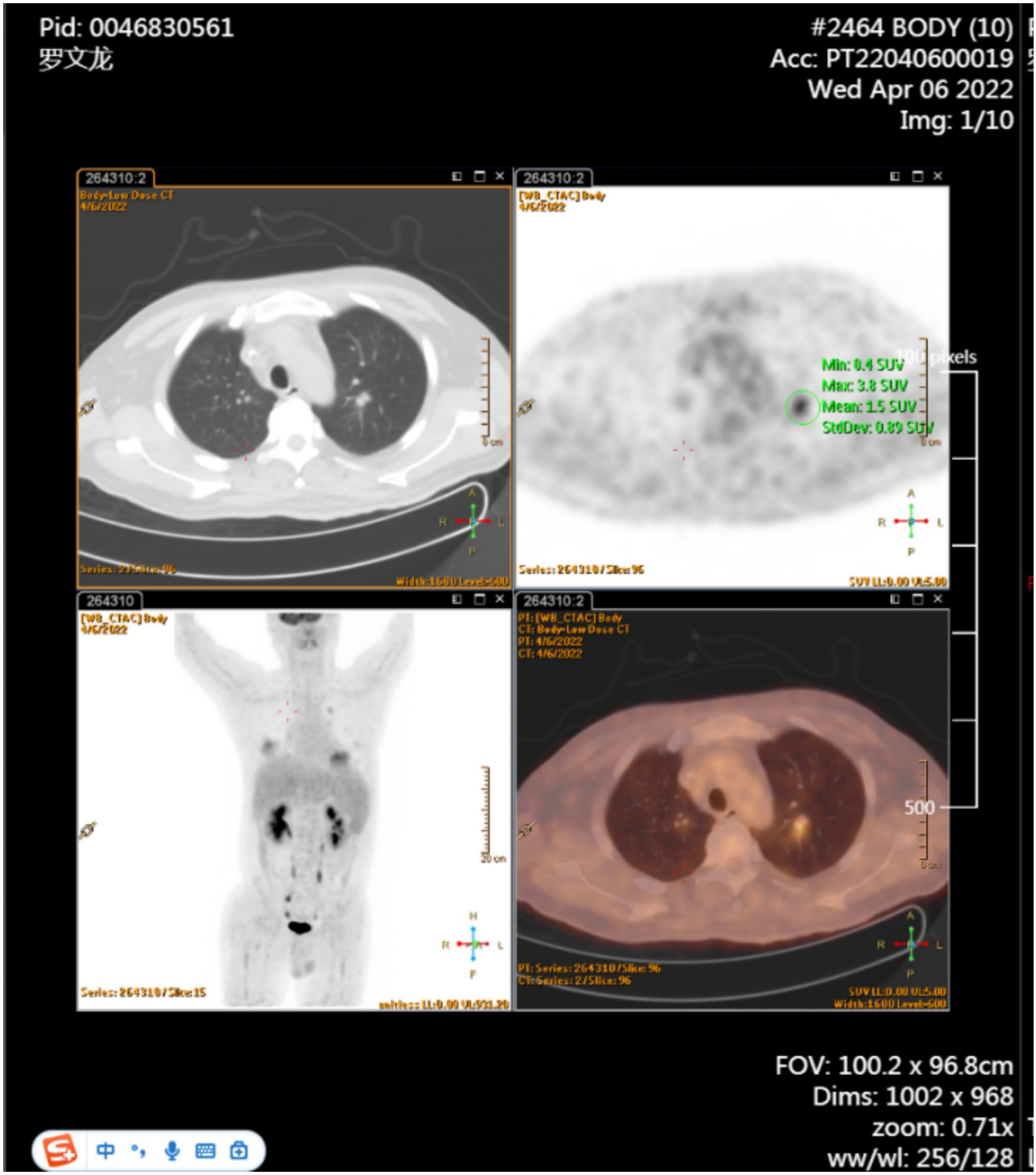

Fig. 5 PET-CT on April 6, 2022: No abnormalities were found in the mediastinum. High-density patchy shadows in the lower lobe of the right lung and the dorsal segment of the lower lobe of the left lung. The lung lesions are consistent with the chest CT scan on April 1, 2022

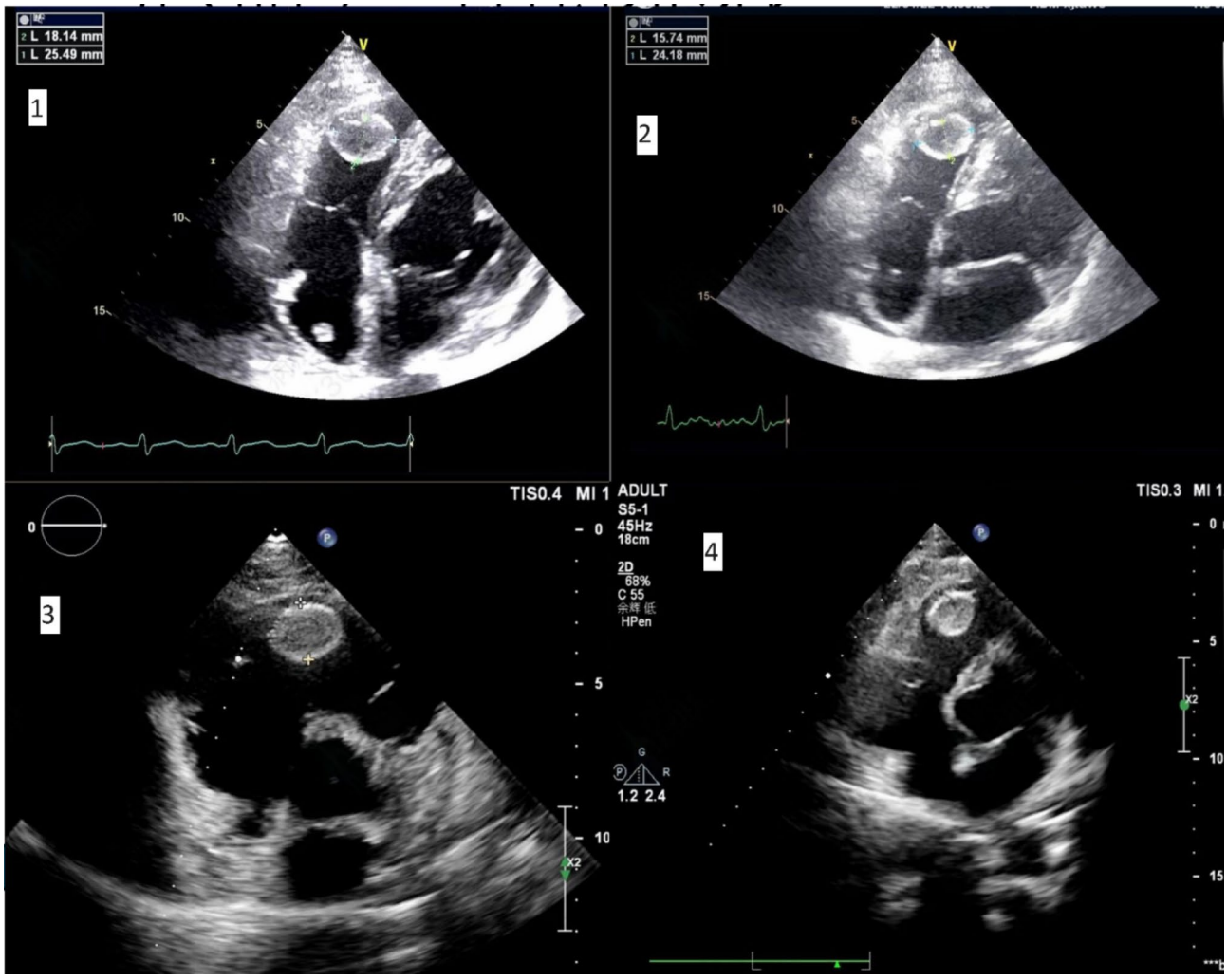

Fig. 6 Follow-up echocardiographic images of the mass: (1) When first discovered on April 2, 2022 (2) Before HSCT on April 22, 2022 (3) Before stopping antithrombotic therapy on July 10, 2022 (4) After stopping antithrombotic therapy for 2 months on September 14, 2022. Three additional movie files included show the echocardiography in more detail [see Additional files 5,6 and 7]

of the masses have not changed significantly, raising questions about the diagnosis.

Infective endocarditis

Given the patient's history of fungal infection in the right lung, infective endocarditis was also considered. Infective endocarditis in the context of AML and immunosuppression could involve atypical organisms, including fungi [8]. However, imaging studies, including MRI, chest CT, and PET-CT, did not show significant involvement of the cardiac valves, which is typically seen in bacterial endocarditis. Despite antifungal and antibacterial treatments, the masses did not resolve, making this diagnosis less likely. The fungal lung infection was diagnosed in February 2022, preceding the discovery of cardiac masses in April 2022. This temporal relationship raises the possibility that the fungal infection may have contributed to the formation of intracardiac structures.

Sarcoidosis

Sarcoidosis was considered due to the granulomatous lesions found in the lung biopsy. Cardiac sarcoidosis, although rare, can present with myocardial granulomas [9]. However, the clinical presentation and imaging characteristics of the cardiac masses do not align well with typical manifestations of cardiac sarcoidosis, such as myocardial thickening or systemic involvement. Moreover, sarcoidosis is usually a diagnosis of exclusion and requires histological confirmation, which was not pursued in this patient due to the invasive nature of obtaining cardiac tissue.

Neoplastic lesions

The possibility of Neoplastic masses, either primary cardiac tumors or metastatic lesions, was also evaluated. Cardiac tumors are rare, and secondary involvement from leukemia usually presents as infiltration rather than

discrete masses [10]. The masses were found in complete remission of leukemia and there have been no significant changes in the masses that would suggest a proliferative neoplastic process. Hence, Neoplasms are less favored.

Cardiac calcified amorphous tumor

In this case, the possibility of a cardiac calcified amorphous tumor (CAT) was considered because of the unusual presentation of multiple calcified, mobile masses in the right atrium and right ventricle. CAT is a rare, non-neoplastic, tumor-like cardiac lesion characterized by calcified nodules typically embedded within amorphous fibrous material [11]. While the etiology of CAT remains unclear, it has been associated with chronic inflammation, degenerative processes, and metabolic disturbances [11]. The diagnosis of CAT was suggested by: (1) Gradual increase in calcification over time (2) Resistance to anticoagulation therapy (3) Mobile, nodular appearance on imaging [12].

The diagnosis of CAT requires pathological diagnosis. Without pathology, we suspected CAT was because of the exclusion of thrombosis, infection, sarcoidosis, and primary cardiac tumors. Combined with the fact that the mass had a calcified capsule and the calcification pattern was gradual, cardiac MRI showed mixed signals in the mass, and no obvious changes were found in the signals between first-pass perfusion and delayed enhancement. Because the patient refused further pathological confirmation, CAT can only be a potential diagnosis.

Clinical course and management challenges

Throughout the patient's treatment for AML and subsequent complications, including fungal lung infection and persistent cardiac masses, the management has focused on addressing the most likely diagnoses. This is done while ensuring the patient remains in leukemia remission and has a smooth allo process. The anticoagulant therapy initiated for suspected thrombosis has been maintained, but the approach was shown to be ineffective and without significant changes in the cardiac masses.

Diagnosis and treatment of cardiac masses in AML patients should be done in a logical and, at the same time, flexible manner. Both the urgency of treating the leukemia and cardiac risk factors must play a role in formulating treatment decisions. As much as diagnosis by biopsy may be desirable, it is difficult to obtain the pathology of mobile cardiac masses by interventional means and cardiac surgery. and cardiac surgery is limited by the urgency of leukemia treatment. After recovery from allo-HSCT, we have repeatedly recommended the patient to go to hospitals that specialize in cardiac surgery for pathology of cardiac mass to make a clear diagnosis of its nature, but the patient refused because there were no clinical symptoms caused by the lesions.

Documentation of patient outcomes, response to interventions, and long-term follow-up is the basis for building up the evidence base for such cases. This approach allows for individualized care while contributing to our knowledge regarding cardiac masses in AML patients.

Multidisciplinary insights

Multidisciplinary consultation at the initial finding of heart masses involving cardiology and imaging specialists, along with a cardiothoracic surgeon provided a consensus leaning towards a thrombotic origin, albeit with significant uncertainty. The experts acknowledged the potential for fungal thrombi but noted the atypical presentation and imaging findings. Regular echocardiographic monitoring was recommended to observe for any changes in size or characteristics, which might offer further diagnostic clues.

Subsequently, after anticoagulation therapy, and the absorption of lung lesions after antifungal therapy, the mass of the heart has no morphological change except for increased calcification. In this case cardiac calcified amorphous tumor (CAT) is considered. It is worth mentioning that pathologic examination is the gold standard for definitive diagnosis for this lesion [13], but the patient refused.

Unresolved questions

Several critical questions remain unanswered:

Etiology Are these masses thrombotic, infectious, neoplastic, or a novel pathological entity?

Pathogenesis What mechanisms lead to the formation and calcification of these cardiac masses in AML patients?

Optimal management Beyond anticoagulation, what targeted therapies or interventions might effectively resolve these masses?

Limitations

A key limitation of our diagnostic workup was the inability to perform long inversion time (TI 600 ms) delayed gadolinium-enhanced imaging during cardiac MRI. This specialized sequence is particularly valuable in definitively differentiating thrombi from other cardiac masses, as thrombi often manifest as filling defects with characteristic enhancement patterns on long TI delayed imaging. The lack of this sequence in our imaging protocol somewhat limited our ability to definitively exclude thrombotic etiologies, particularly in the early stages of mass formation.

A significant limitation of this case report lies in its inability to definitively characterize the cardiac masses despite extensive diagnostic workup. The absence of a

definitive pathological diagnosis hampers the greater clinical applicability of this case. While the temporal progression and imaging characteristics were well-documented, the fact that no histopathological confirmation was obtained leaves many important questions regarding the exact nature of the masses unanswered. This lack of clarity in diagnosis limits the ability to clearly indicate treatment protocols or to state with authority how similar cases should be treated in the future. The case serves as a documentation of an unusual clinical presentation rather than offering concrete insights into the pathophysiology and optimal management of cardiac masses in leukemia patients.

Conclusion

In conclusion, the case of this 33-year-old male with Acute Myeloid Leukemia (AML-M2a) and concomitant cardiac masses highlights the diagnostic complexities and therapeutic challenges encountered in treating Acute Myeloid Leukemia (AML-M2a) patients. Despite extensive imaging and multidisciplinary consultations, including echocardiography, MRI, and PET/CT, the exact nature of these calcified, mobile nodular masses in the right atrium and right ventricular lateral wall remains **elusive**. While anticoagulation was initiated presumptively for thrombotic masses, their persistence and atypical imaging characteristics underscore the need for continued research and collaboration to elucidate their underlying pathology. This case underscores the importance of vigilant monitoring and a multidisciplinary approach to managing rare cardiac complications in leukemia patients, aiming for improved diagnostic accuracy and therapeutic outcomes in similar challenging scenarios.

Abbreviations

AML M2a	Acute Myeloid Leukemia type M2a
FAB	French-American-British Classification
MICM	Morphology, Immunology, Cytogenetics, Molecular biology
ELN	European Leukemia Net
Allo-HSCT	Allogeneic hematopoietic stem cell transplant
CAT	Cardiac calcified amorphous tumor
HLA	Human leukocyte antigens
HLA-DR	Human Leukocyte Antigen – DR isotype
IA	Idarubicin (IDA) 8 mg/m 2, 10 mg/m 2 or 12 mg/m 2 as induction chemotherapy
DAC + CAG	Decitabine in combined with aclacinomycin, cytarabine and G-CSF
CLAG	Cladribine + Cytarabine + G-CSF
MRD	Minimal residual disease
PAS	Periodic acid-Schiff
TB	Toluidine Blue

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s13019-024-03309-2.

Supplementary Material 1: Cardiac MR video file. Cardiac MRI cine sequence demonstrating the dynamic visualization of the lesions

Supplementary Material 2: Cardiac MR video file. Cardiac MRI cine sequence demonstrating the dynamic visualization of the lesions [.mp4 format]

Supplementary Material 3: Additional Cardiac MRI picture. Cardiac MRI sequence visualizing the lesion in optimal settings

Supplementary Material 4: Additional Cardiac MRI picture. Cardiac MRI sequence visualizing the lesion in optimal settings

Supplementary Material 5: Video file of cardiac ultrasonography. Echocardiographic video loop showing the dynamic characteristics and motility patterns of the intracardiac lesions

Supplementary Material 6: Video file of cardiac ultrasonography. Echocardiographic video loop showing the dynamic characteristics and motility patterns of the intracardiac lesions

Supplementary Material 7: Video file of cardiac ultrasonography. : Echocardiographic video loop showing the dynamic characteristics and motility patterns of the intracardiac lesions

Supplementary Material 8: CT mediastinal window timeline. Serial CT imaging in mediastinal window protocol demonstrating the temporal evolution of the lesion across multiple time points

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Author contributions
Xiaoning Wang and Mohammad Amin Ansarian and Mahsa Fatahichegeni and Juan Ren and Seifollah Ranjbarha wrote the main manuscript text and Juan Ren provided the case and figures. All authors reviewed the manuscript and contributed equally.

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Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate and publish
Verbal and written informed consent was obtained from the patient for publication of this case report and accompanying images.

Competing interests
The authors declare no competing interests.

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