

A 31-year-old female with diffuse pulmonary infiltrates and intracardiac thrombus

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Abstract

Intracardiac masses on echocardiography can represent thrombi, vegetations, or tumors. Cardiac Magnetic Resonance Imaging (MRI) is a sensitive tool that differentiates between the three. Intracardiac thrombi can develop during various pathological conditions that cause stasis of blood and predispose to the aggregation of thrombotic material. Atrial clots can occur in conditions like atrial fibrillation, structural heart diseases, throm-

bophilia, and chronic inflammations. Tuberculosis (TB) is a prothrombotic state and can predispose to venous and sometimes arterial clot formation. But intracardiac clots in TB are rare. We report a young patient presenting with bilateral intracardiac clots and diagnosed to have disseminated tuberculosis. Treatment with anti-tubercular therapy and therapeutic anticoagulation leads to complete resolution of the thrombi.

Case Report

A 31-year-old non-smoking female presented with a high-grade fever and fatigue persisting for 2 weeks. She denied experiencing cough, hemoptysis, palpitations, abdominal pain, headache, night sweats, or weight loss. Eight months prior, she delivered a healthy baby vaginally. Six months before the presentation, she was diagnosed with iron deficiency anemia and received appropriate treatment. Upon examination, her pulse rate was 130 beats/min, blood pressure was 110/83 mm Hg, respiratory rate was 19 breaths per minute, and oxygen saturation was 96% on room air. She had pallor and hepatosplenomegaly, with no jugular venous distension, appreciable lymphadenopathy, or pedal edema noted. The remainder of her examination was unremarkable. Laboratory investigations revealed a hemoglobin level of 5.8 gm/dL and a White Blood Cell (WBC) count of 4300/mL. Other liver, kidney, and procalcitonin tests were within normal ranges. Abdominal ultrasonography confirmed hepatosplenomegaly. Chest radiography revealed randomly distributed nodules, some with cavitations. Transthoracic Echocardiography (TTE) identified a mobile mass measuring 7x7 mm attached to the mitral valve (Figures 1 and 2). Subsequent Transesophageal Echocardiography (TEE) revealed mobile masses in both atria, completely filling the left and right atrial appendages. Cardiac Magnetic Resonance imaging (CMR) confirmed acute myocarditis, fibrinous pericarditis, and bilateral atrial clots. Ultrasonographic screening for deep vein thrombosis in the lower limbs was negative. Differential diagnoses considered included infectious, inflammatory, and malignant etiologies. In view of pancytopenia, the patient underwent a bone marrow aspiration and biopsy, which revealed epithelioid granulomas with necrosis (Figure 3). Cartridge-Based Nucleic Acid Amplification Test (CBNAAT) detected low *Mycobacterium tuberculosis* (MTB); Rif resistance was not detected. A diagnosis of disseminated tuberculosis with intracardiac thrombus was made, and the patient was initiated on drug-sensitive antitubercular therapy and systemic anticoagulation, along with low-dose steroids. Subsequent management involved transitioning to Novel Oral Anticoagulants (NOACs), and the patient was discharged home. Thrombolysis was not pursued due to the risk of embolization. The patient demonstrated clinical improvement after completing 5 months of antitubercular therapy, with the resolution of atrial clots observed on sequential TTE (Figure 4). Latent tuberculosis was ruled out in the patient's infant.

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Discussion

Intracardiac thrombi represent one of the three common differential diagnoses of intracardiac masses alongside vegetation and tumors.¹ TTE serves as the initial modality to identify and delineate intracardiac masses, while TEE can further enhance overall visualization, especially for masses located in posterior cardiac structures such as the atria and atrial appendages. CT and CMR imaging, along with Late Gadolinium Enhancement (LGE), offer additional insights into heart size, function, and better assessment of infiltrative myopathies. Fresh thrombi typically exhibit a higher signal than myocardium on T1-weighted CMR sequences, with contrast further accentuated on T2-weighted images.²

Atrial clots commonly occur as a complication of atrial fibrillation. Other etiologies of atrial clots include structural heart disease, inherited thrombophilia, antiphospholipid syndrome, and

neoplasms. Additionally, persistent inflammatory states such as autoimmune disorders or chronic infections can predispose to a prothrombotic state. Inflammation activates the coagulation cascade while concurrently reducing the activity of the anti-coagulant mechanism, thus promoting clot formation.³

Myocardial involvement and hemostatic complications, particularly intracardiac thrombosis in TB, are rare phenomena, documented in only a few case reports.⁴ The odds ratio of 1.62 for developing venous thrombosis due to TB has been reported.⁵ Patients with active TB exhibit thrombocytosis, elevated plasma fibrinogen, factor VIII, Plasminogen Activator Inhibitor 1 (PAI-1), along with decreased antithrombin III and protein C levels, resulting in activated coagulation and inhibited fibrinolysis. Other mechanisms described include direct endocardial tissue injury caused by the TB bacillus (endocardial miliary tubercles), resulting in alterations to normal blood flow, as well as rifampicin-induced direct endothelial injury.^{6,7}



Figure 1. Transthoracic Echocardiographic (TTE) image of four-chamber view showing Left Atrium (LA) clot.

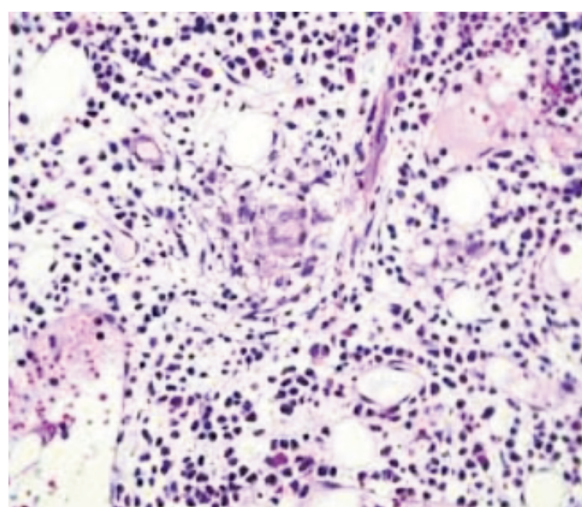


Figure 3. The pathological examination of bone marrow biopsy shows the presence of granulomas.



Figure 2. Transthoracic Echocardiographic (TTE) image of two-chamber view showing Right Atrium (RA) clot.



Figure 4. Transthoracic Echocardiographic (TTE) four-chamber view showing resolution of clot in Bilateral (B/L) atrial chambers.

Intracardiac clots can lead to depressed myocardial function, resulting in ventricular arrhythmias, congestive heart failure, and sudden cardiac arrest. Previous reports have suggested complete resolution of intracardiac thrombus with improvements in left ventricular function following Antitubercular Therapy (ATT) and anticoagulation, as was seen in our case. Hence, prompt diagnosis of TB and early initiation of treatment are crucial for optimal patient outcomes.

Conclusions

Cardiac MRI is a valuable tool for distinguishing intracardiac masses from thrombi. Mycobacterium tuberculosis can present in diverse ways, including intracardiac thrombosis. Anticoagulation alongside antitubercular therapy remains the mainstay of treatment, emphasizing the importance of patient counseling and adherence.

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