MASSES, METASTASES, THROMBUS, AND THROMBOLYSIS

Pulmonary Artery Mass in a Patient With Tuberculous Pericarditis

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INTRODUCTION

Infection of the heart by Mycobacterium tuberculosis is rare, seen in only 1% to 2% of people with pulmonary tuberculosis (TB). The bacilli can reach the heart via lymphatics from mediastinal nodes, hematogenous spread, or by direct invasion from the pericardium. Although the propensity of TB to cause pericarditis and myocarditis is well documented, here we present an extremely rare manifestation of cardiac TB: direct vascular invasion of the main pulmonary artery that was first detected by transthoracic echocardiography (TTE).

CASE PRESENTATION

A 40-year-old immunocompetent man presented to our urgent care clinic with 3 days of fevers, myalgias, diarrhea, and lightheadedness. The patient had a remote history of pericarditis in 1995, after attending a wedding in Haiti. Because of persistent tachycardia and hypotension, he was admitted to our hospital for further workup and management. Serial blood cultures were negative, and no leukocytosis was appreciated. However, his laboratory testing revealed elevated inflammatory markers, including a C-reactive protein level of 55.8 mg/L and an erythrocyte sedimentation rate of 33 mm/h, prompting TTE to evaluate for endocarditis. TTE did not reveal any vegetations but was notable for mildly impaired left ventricular systolic function, a loculated anterior pericardial effusion with extension into the anterior mediastinal space, and a septal bounce (Figure 1, Video 1). The effusion had a mild compressive effect on the adjacent right ventricular outflow tract but no overt signs of tamponade. The pulmonary artery appeared grossly normal (Figure 2).

Given the clinical suspicion for TB pericarditis in the setting of his clinical history, an interferon-γ release assay was performed, which came back positive. The mediastinal fluid was subsequently biopsied, and the patient was empirically started on rifampin, isoniazid, pyrazinamide, and ethambutol (RIPE) antimycobacterial treatment. Fluid cultures grew pan-sensitive M. tuberculosis, confirming a diagnosis of TB pericarditis.

One week before completing 6 months of RIPE therapy, follow-up TTE was performed. At this time, the patient’s C-reactive protein level had decreased to 12.6 mg/L, but his erythrocyte sedimentation rate remained elevated at 51 mm/h. Surprisingly, TTE revealed a fixed, well-circumscribed, spherical mass projecting into the lumen of the main pulmonary artery (Figure 3, Video 2). Although the mass was located in close proximity to the pulmonic valve, the pulmonic leaflets appeared unaffected, and there was only trace pulmonic insufficiency. Although color Doppler respected the borders of the mass, there was no significant flow acceleration to suggest obstruction (Figure 4, Video 3).

Cardiac magnetic resonance (CMR) imaging was used to further characterize the mass and similarly demonstrated an 8 × 5 mm immobile structure immediately superior to the medial pulmonic annulus, projecting 8 mm into the lumen of the main pulmonary artery (Figure 5, Video 4). Notably, the mass was contiguous with the thickened and calcified pericardium, consistent with direct extension from the adjacent tuberculous mediastinal collection (Figure 5). Importantly, the mass was steady-state free precession hypointense and T1 and T2 isointense and demonstrated subtle late enhancement, consistent with either tumor or an infectious, granulomatous etiology. Computed tomography additionally confirmed direct extension of the mass from the adjacent pericardium and anterior mediastinal TB collection (Figure 6).

Although the differential diagnosis for our patient’s mass was initially broad and included thrombus, tumor, and vegetation, several factors led us to the ultimate conclusion of a tuberculous granuloma. First, CMR imaging and computed tomography demonstrated continuity of the mass with the biopsy-proven TB pericarditis. Second, enhancement of the mass on CMR imaging effectively ruled out thrombus, which we also believed was unlikely given its presence in the high-flow area of the proximal pulmonary artery. Third, the spherical, fixed nature of the mass and its location were not characteristic of vegetation, and serial negative blood cultures further supported this judgment. Last, a tumor seemed very unlikely, as our patient had no history of metastatic disease, and primary tumors in this location are exceedingly rare and typically manifest with highly malignant systemic symptoms.

Given the patient’s clinical stability, the absence of hemodynamically significant obstruction, and the risks associated with invasively removing the pulmonary artery mass, the decision was made to complete the TB regimen and follow the granuloma with serial imaging. Follow-up TTE 6 months after completion of RIPE therapy showed complete resolution of the pulmonary artery mass and a reduction in size of the pericardial effusion (Figure 7). Ultimately, the resolution of the mass with targeted TB therapy alone—in the absence of empiric anticoagulation, intravenous antibiotics, or oncologic therapies—strongly confirmed our diagnosis of an intravascular tuberculous granuloma.

DISCUSSION

Although the prevalence of myocardial and pericardial involvement of M. tuberculosis infection is fairly well established, here we introduce an unusual manifestation of cardiac TB, involving the hallmark sign of TB infection: the granuloma. A granuloma is an organized structure of...
immune cells designed to wall off the *M. tuberculosis* infection. It may be centrally caseating or noncaseating. Although granulomas are commonly found in the lungs of patients with TB, there are very few cases in the literature describing intravascular invasion, as was seen in our patient. Previously described cases include the following: obliteration of the left coronary artery lumen by necrotizing granulomas, resulting in sudden cardiac death; pulmonary arteritis with centrally caseating epithelioid granulomas, causing hemodynamically
significant stenosis and the need for pulmonary artery reconstruction; and a mediastinal mass composed of noncaseating granulomas, invading and obstructing the main and branch pulmonary arteries.

Although histologic examination is the only way to definitively diagnose our patient’s mass, its fixed and spherical nature seen on all three imaging modalities (TTE, CMR imaging, and computed tomography), its late enhancement on CMR imaging, its direct continuity with the anterior fluid collection that grew TB, and its response to targeted antituberculous therapy make an intravascular tuberculous granuloma the most likely diagnosis. We suspect that the development of the pulmonary artery mass on therapy, and subsequent resolution without a change in treatment, reflects paradoxical worsening of TB. This is a well-described but poorly understood phenomenon in which tuberculous lesions may worsen or develop while on antimycobacterial therapy. Fortunately, it is usually self-limiting, as was the case in our patient.

The decision not to remove the mass in our patient proved appropriate, as the mass was nonobstructive and resolved; however, other case reports have shown that tuberculous granulomas have the potential to cause hemodynamically significant stenosis and do not always regress.

CONCLUSION

Our case demonstrates an extremely rare presentation of tuberculous pericarditis leading to local invasion and intravascular granuloma formation in the main pulmonary artery. In this case, TTE was vital not only in making the initial diagnosis of TB pericarditis but in discovering this rare complication and assessing its response to treatment.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found at https://doi.org/10.1016/j.case.2018.11.004.
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Figure 6  Axial contrast-enhanced computed tomography shows that the mass (arrow) is contiguous with the mediastinal fluid collection (asterisk), consistent with direct invasion.

Figure 7  Follow-up TTE after completion of RIPE therapy. Views of the main pulmonary artery bifurcation demonstrate complete resolution of the previously visualized mass and a reduction in size of the pericardial effusion.