

An Unusual Atrial Mass: A Case Study

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BACKGROUND

This case highlights the evolution of a broad differential diagnosis when presented with a rare and diagnostically enigmatic clinical finding.

CASE PRESENTATION

A 43-year-old Puerto Rican male with a history of homelessness, intravenous drug abuse, and incarceration presented following an episode of syncope. His past medical history was notable for a recent diagnosis of granulomatous nephritis, thought to be secondary to isolated renal sarcoidosis. He had been treated with high dose prednisone and subsequently initiated on hemodialysis. On presentation, he complained of rigors, shortness of breath, cough, abdominal pain and coffee-ground emesis. On initial examination, the patient was ill-appearing, febrile, tachypneic and tachycardic. He was awake and oriented with no focal neurological deficits.

His sepsis evaluation included computed axial tomography (CT) scans of his chest, abdomen and pelvis. Imaging was notable for para-aortic lymphadenopathy, diffuse micro-nodular densities throughout the lungs, hilar and mediastinal lymphadenopathy, and advanced xanthogranulomatous pyelonephritis with extensive calcification of the right kidney. His blood and urine cultures grew a resistant strain of *Escherichia coli* and he was initiated on broad antibiotic coverage, delivered via a central venous catheter. Further evaluation for endocarditis included both transthoracic (TTE) and transesophageal (TEE) echocardiograms, which identified a well-demarcated, heterogeneous right atrial (RA) density measuring 1.7 x 1.6 cm. He was initiated on full anticoagulation for this presumed catheter-associated right atrial thrombus. A head CT demonstrated an abnormal 1.2 x 3.6 cm lobular hyper-density in the right frontal lobe with surrounding edema, and multiple dispersed punctate calcified hyper-densities.

Soon after presentation, the patient developed seizures and acute respiratory failure, necessitating intubation. His chest x-ray demonstrated progression of innumerable bilateral pulmonary micro-nodular densities. He underwent bronchoscopy, and pathology was notable for acute fibrinous and organizing pneumonia. Initial concentrated sputum smears were negative for acid-fast bacilli (AFB). He was, however, initiated on empiric anti-tuberculosis quadruple pharmacotherapy (RIPE) given his exposure risk, chronic immunocompromised state, and concern for miliary tuberculosis. He was eventually weaned from the ventilator and was extubated. Subsequently, a right-sided percutaneous nephrostomy tube was placed to relieve his obstructing xanthogranulomatous pyelonephritis. Cultures from the nephrostomy tube were notable for the growth of Mycobacterium Tuberculosis Complex (MTB), as were cultures from his bronchoscopy, confirming disseminated MTB. His original sputum cultures later grew MTB 19 days after collection.

Several weeks into his hospital stay, a lower gastrointestinal bleed prompted the discontinuation of his anticoagulation, necessitating re-evaluation of the right atrial mass. TTE revealed an expanded (4.2 x 3.3 cm) multi-lobulated, heterogeneous echo-dense RA mass extending towards the inferior vena cava/RA junction with new focal areas of brightness suggesting calcification. Hemodynamic assessment including orthostatics and central venous pressure estimation were normal. Although airborne precautions precluded endoscopic evaluation of the gastrointestinal hemorrhage, the imminent risk of the growing atrial mass prompted reinstatement of anticoagulation, which he was able to tolerate.

In the setting of disseminated MTB infection, the patient's chronic steroids were slowly tapered. However, he quickly developed a new facial droop, unilateral weakness and altered mental status. It was

thought that his neurological change was secondary to tuberculous cerebritis. He was quickly re-initiated on high dose prednisone, and his mental status improved. He later developed recurrent polymicrobial bacteremia from his pyelonephritis and required a prolonged course of antibiotics. Nephrectomy was deemed to be prohibitively high risk in the setting of his multiple comorbid conditions.

As his infections were appropriately managed, a multidisciplinary approach in concert with cardiology, infectious diseases, radiology and interventional radiology was undertaken to further elucidate the etiology of his undifferentiated atrial mass. Given his multiple co-morbidities, poor functional status and chronic immunosuppression, invasive diagnostics were deferred, and it was decided that he would be continued on empiric anticoagulation in combination with RIPE therapy for both thrombus and atrial tuberculoma.

DIFFERENTIAL DIAGNOSIS

The patient presented with a previous pathological diagnosis of renal-limited sarcoidosis and had been treated with several months of high-dose corticosteroids. He then developed persistent fevers, neurological deficits, miliary pulmonary infiltrates, calcified cerebral hyper-densities, presumed enteritis, xanthogranulomatous pyelonephritis, and an atrial mass. Initially, a diagnosis of systemic sarcoidosis remained high on the differential. Given the patient's immunocompromised state, polymicrobial infections, and progression of systemic manifestations on corticosteroids, also included in the initial differential diagnosis were lymphoproliferative disorders, neoplasms, and infections (including disseminated tuberculosis).

An intra-cardiac mass is an uncommon finding that carries a broad differential diagnosis, including thrombus, vegetative endocarditis, myxoma, myosarcoma, rhabdomyosarcoma, infiltrative lymphoma, secondary deposits and cardiac tuberculoma.

OUTCOME AND FOLLOW-UP

Following institution of RIPE therapy for disseminated tuberculosis, targeted antimicrobials for bacteremia, steroids for tuberculous cerebritis and empiric anticoagulation for his atrial mass, the patient's clinical status

improved. He has remained hemodynamically stable, precluding the need for surgical intervention of his atrial mass. He will require monthly monitoring with TTE, a prolonged course of RIPE, and long-term anticoagulation for management of his disseminated tuberculosis and undifferentiated right atrial mass.

DISCUSSION

The constellation of findings presented in this case represent a broad differential diagnosis, including systemic sarcoidosis, lymphoproliferative disorders, and infections.¹ Sarcoidosis and lymphoproliferative disorders are known to involve the lungs, kidneys, central nervous system, gastrointestinal tract and reticuloendothelial system. However, the isolation of MTB from two separate sources in an immunocompromised patient at high risk for previous MTB exposure makes the diagnosis of primary or reactivation disseminated tuberculosis most likely.¹⁻³ With strong clinical, microbiological and radiographic evidence supporting a diagnosis of disseminated tuberculosis, the immediate institution of RIPE and rapid identification and management of potential end-organ complications is paramount.³

Less than 1.5% of all MTB infections disseminate lymphohematogenously. Most often the lungs are affected, but, after lymphadenopathy, the next most common manifestations of non-pulmonary tuberculosis include gastrointestinal and hepatic (80%), genitourinary (27%), and meningeal (20%).^{1,4,5} Infiltration of the myocardium is uncommon, and the documented incidence of intra-cardiac tuberculoma is rare, with a majority of cases diagnosed at autopsy.⁶

The few reported cases of cardiac tuberculoma diagnosed *in vivo* describe single or multiple well-circumscribed, heterogeneous masses often occupying the right atrial free wall.⁶⁻⁹ Echocardiography is often used to characterize size, location and echogenicity. Magnetic Resonance Imaging is able to provide more detail, but often fails to distinguish tuberculoma from thrombus and myxoma.⁷ Percutaneous biopsy under echocardiographic guidance provides a means of sampling the mass. However, identification of characteristic histopathological findings lacks sensitivity, and most reports indicate unacceptable yields for identification of MTB following Ziehl-Neelsen staining.^{6,7,9} A confirmatory diagnosis requires invasive removal of the mass for

gross histopathological diagnosis.⁷ Few studies have demonstrated successful reduction in size, and even complete resolution of cardiac tuberculomas following appropriate anti-tuberculosis pharmacotherapy alone.^{6,8} Surgical intervention is indicated if the mass results in clinically significant hemodynamic compromise, or if diagnosis remains unclear after pharmacotherapy has failed to demonstrate improvement by imaging.^{6,8,9}

Given the patient's encouraging response to anti-tuberculosis pharmacotherapy and anticoagulation, as well as persistently stable hemodynamics, further diagnostics of the right atrial mass were deferred. The risk of interventional diagnostics were thought to outweigh any benefits, given that the treatment plan of combining anticoagulation with RIPE therapy would remain unchanged.

KEY POINTS

Cardiac tuberculomas are a rare complication of disseminated MTB infection and should be considered in the differential diagnosis of an intra-cardiac mass in a patient with proven MTB. With strong clinical and radiographic evidence, and without hemodynamic compromise, invasive diagnostics may be deferred in favor of empiric medical management.

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"Sunset over Naragansett Bay, Rhode Island"
photograph by Andrew Zabolotsky, MD

