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CLINICAL VIGNETTE

A Small Sterile Vegetation Noted in a Recovering Critically Ill Patient with Hypercoagulability

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Case Report

Our patient is a 40-year-old female with a complex past medical history who was admitted to rehabilitation with critical illness myopathy after hospitalization for congestive heart failure. Her medical history includes type 1 diabetes, recurrent STEMI with cardiac arrest and AICD placement, AKI with temporary dialysis, recurrent DVT's on chronic warfarin, drug induced liver disease, urinary retention, and C. difficile diarrhea. She had recurrent admissions for decompensated CHF.

During her stay at rehab, a routine transthoracic echocardiogram showed a vegetation on the right ventricular pacemaker wire. Transesophageal echocardiogram confirmed a mass on the right ventricular pacemaker lead, presumed to be a thrombus/vegetation. Cardiology and infectious disease felt the vegetation was a sterile thrombus, or a combination of thrombus plus sterile remnants of an old infection, as the patient did not appear infected. She had no fever, normal wbc (7.7), normal erythrocyte sedimentation rate, and normal C reactive protein. The patient was monitored off antibiotics and blood cultures remained negative. During this time, she was managed by nephrology for AKI and volume control in the setting of congestive heart failure. She responded to physical therapy for reconditioning and eventually was discharged home.

Discussion

This 40-year-old woman with hypercoaguability had a sterile thrombin and fibrin vegetation. The extensive workup excluded rheumatological conditions and infective endocarditis (bacterial or otherwise). This case illustrates that some vegetation in hypercoagulable patients due to clot formation at the ends of otherwise healthy native valves. The lack of atrial fibrillation makes this case more unusual, though the clots that form in atrial fibrillation tend to be in the atrial appendage.[1] Treatment with oral anticoagulation was continued after diagnostic certainty that there was no active infection. Non-bacterial thrombotic endocarditis is an unusual disorder of varying clinical severity that results from the formation of fibrin and thrombin vegetations on the tips of cardiac valves.[2] The incidence ranges from 0.3 to 9.3%.[2] It is much rarer than bacterial colonization of cardiac valves.[3]

Etiology and pathogenesis of NTBE is thought to be multifactorial, with common factors of endothelial damage and subsequent exposure of the subendothelial connective tissue to circulating platelets.[2] Four factors implicated in initiation of verrucae are: immune complexes,[4] hypoxia,[5] hypercoagulability, and carcinomatosis.[2] Carcinomatosis and generation of NBTE is associated with mucin producing adenocarcinomas.[6] DIC has been associated with NBTE Factor, also implicated in hypoxia pathogenesis.[7]

No pathognomonic clinical features exist for NBTE.[2] Along with high clinical suspicion, three clinical signs have been proposed by Mckay and Walher.[8] They include presence of a disease known to be associated with NBTE, absence of a heart murmur and multiple episodes of endocarditis. Features that are often present in infective endocarditis and notably absent in NBTE include fever, cardiac murmur, leukocytosis, elevated CRP, positive blood cultures, and negative antiphospholipid antibodies.[2]

On echocardiogram, the vegetations seen in NBTE differ in appearance, location, and lack propensity for valve and rupture when compared to those seen in infective endocarditis.[9] Diffusion weighted MRI brain images typically show a stroke pattern of multiple, small, and medium or large disseminated lesions in NBTE compared to IE, which is usually a single lesion, territorial infarction or multiple punctuate disseminated.[10]

If clinical suspicion is high, echocardiography is recommended to establish the diagnosis. TEE has a higher sensitivity for vegetations compared with TTE, especially for vegetations less than 5mm.[2]

Given that the etiology of NBTE is broad, management involves the treatment of the underlying cause. For prevention of systemic embolization, indefinite systemic anticoagulation remains the mainstay of treatment, provided there are no contraindictations.[11-14] Similar to IE, surgery may be indicated in cases of heart failure and acute valve rupture, but also for prevention of recurrent embolization in select cases.[15]

Atheroembolism to viscera is common,[2] with rates widely varying between 14 and 90%.[2] One variant of this vegetation is Libman-Sacks endocarditis, usually seen in inflammatory conditions like systemic lupus erythematosus (SLE).[16] Non-bacterial endocarditis, including fungal endocarditis usually results in significant morbidity.[17]

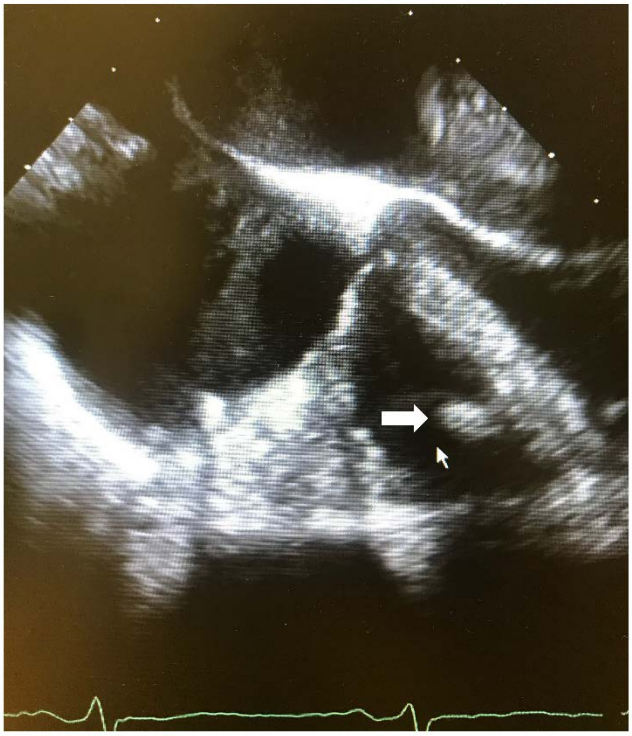

Figure 1. Echocardiogram showing a small sterile vegetation.

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