CLINICAL VIGNETTE

Focal Left Ventricular Aneurysm as a Sign of Chronic Chagas Cardiomyopathy

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Case Presentation

A 56-year-old female was referred to cardiology for evaluation of abnormal electrocardiogram (ECG) in primary care clinic. She immigrated from El Salvador in her early twenties and was type II diabetes, hypertension and hyperlipidemia. She reported intermittent palpitations and dyspnea on exertion. Vitals showed a blood pressure of 128/70 with a heart rate of 59. Physical exam was unremarkable. Her ECG showed sinus bradycardia with q waves and nonspecific lateral T wave abnormalities. Patient's medications included metformin, atorvastatin, lisinopril and gabapentin. A transthoracic echocardiogram showed normal left ventricular systolic function with ejection fraction of 55-60%. A focal apical left ventricular aneurysm was also noted. Given risk factors for coronary artery disease, ECG changes, wall motion abnormalities and concerning symptoms, patient was referred for a cardiac catheterization. Cardiac catheterization showed normal coronary arteries. An extended Holter monitor showed non-sustained episodes of paroxysmal atrial tachycardia and few short runs of non-sustained ventricular tachycardia that were asymptomatic. A Chagas titer was sent to evaluate etiology of apical aneurysm which came back positive suggestive of cardiac involvement of Chagas disease in this patient.

Discussion

Chagas disease is caused by the protozoan parasite *Trypanosoma cruzi*. The major chronic manifestations involve the gastrointestinal and cardiovascular systems. Most of patients in acute phase are asymptomatic, and the chronic symptomatic phase appears years later. The protozoan can cause acute myopericarditis as well a chronic fibrosing cardiomyopathy. It is the most common cause of nonischemic cardiomyopathy in Latin America affecting about 30% of patients with chagas. Chagas disease has increasing prevalence in non-endemic areas outside Latin America due to human migration.[1]

Patients with chronic Chagas cardiomyopathy (CCC) may be asymptomatic or present with chest pain, shortness of breath, dizziness, palpitations, syncope, fatigue or edema. Clinical manifestations include chest pain syndrome, cardiac arrhythmias, heart failure, segmental wall-motion abnormalities, apical aneurysms, mural thrombi with embolic potential, stroke and sudden cardiac death. Heart failure evolves slowly and may start with systolic or diastolic left ventricular dysfunction which then progresses to biventricular failure. Atrial or ventricular arrhythmias include sinus node dysfunction, atrial fibrillation, complete atrioventricular block or ventricular arrhythmias and may cause sudden cardiac death.[2]

Physical findings may include mitral and/or tricuspid regurgitation, wide splitting of second heart sound due to right bundle branch block and a prominent diffuse apical thrust. CCC should be suspected in individuals who have lived in endemic countries of Latin America or are offspring of women from endemic countries. Initial diagnostic tests include ECG, chest x-ray, echocardiogram and Holter monitoring. Diagnosis is confirmed with serum immunoglobulin IgG antibody test for *T. cruzi*. Some patients may benefit from additional testing including: cardiac magnetic resonance imaging (CMR), radionuclide imaging, stress testing, cardiac catheterization, and endomyocardial biopsy. Epicardial coronary arteries are usually normal, in CCC and chest pain may be due to microvascular dysfunction.[3] If echocardiography is suboptimal or nondiagnostic, CMR may be helpful for to assess, ventricular size and function, and presence and extent of myocardial fibrosis. Myocardial fibrosis appears to correlate with disease severity.[4] CMR also may detect early right ventricular dysfunction, marker of poor prognosis.[5]

Management of patients with CCC includes monitoring for progression, selective use of antitrypanosomal therapy and supportive care for heart failure, arrhythmia, and thromboembolism. Heart failure in CCC is usually treated with standard goal directed medical therapy. Antiarrhythmic medications, anticoagulation therapy, implantable cardioverter-defibrillators, and pacemakers may be indicated for arrhythmia control and thromboembolic prophylaxis. Advanced heart failure therapies, including cardiac transplantation and mechanical circulatory support, are reserved for patients with refractory symptoms or end-stage disease. Patients should have annual history and physical examinations and electrocardiogram to monitor for new abnormalities and symptoms. Echocardiographic assessment every three to five years sis recommended given the importance of wall motion abnormalities. Mortality in CCC is usually due to cardiovascular involvement, with sudden death in 55 to 65 percent, due to progressive heart failure in 25 to 30 percent and stroke in 10 to 15 percent.[6] With the increasing prevalence of CCC in nonendemic regions including the United States, health care providers and health systems should be able to recognize, diagnose, and treat Chagas disease and prevent further disease transmission.

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