UCLA Health

Department of Medicine
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Abstract Form

Hospital Affiliation:	Harbor-UCLA Medical Center
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Project Title:	Recognition of Chagas Cardiomyopathy is vital given an increased risk of Ventricular Tachycardia and Sudden Cardiac Death

Research Category (please check one):

☐	**Original Research**	☒	**Clinical Vignette**	☐	**Quality Improvement**	☐	**Medical Education Innovation**

Abstract

Introduction: Chagas disease (CD) is due to an infection by the parasite *Trypanosoma cruzi*. Approximately 300,000 United States residents are infected with *T. Cruzi*, one-third of whom will develop Chronic Chagas Cardiomyopathy (CCC). Effects of CD on the heart include cardiac fibrosis, conduction abnormalities, cardiomyopathy, and sudden cardiac death (SCD). We present the case of a patient whose initial presentation of ventricular tachycardia (VT) led to the diagnosis of CCC.

Case Description: A 67-year-old Latin American woman with history of hypertension (HTN) and heart failure with reduced ejection fraction (HFrEF) with non-obstructive coronary artery disease presented to the ED with left-sided chest pressure and intermittent palpitations. Initially, the heart rate was 59 bpm and blood pressure was 123/78 mmHg. Troponin and basic metabolic panel were unremarkable. Electrocardiogram on presentation revealed normal sinus rhythm with a right bundle branch block and left anterior fascicular block (image 1). She subsequently developed sustained VT (image 2) with hypotension and was defibrillated twice prior to restore normal sinus rhythm. An amiodarone drip was started. Of note, she reported a recent admission at outside hospital for VT and was discharged without further medical management. Transthoracic echocardiography revealed an EF of 35%, inferolateral akinesis, severe central mitral regurgitation, and severe pulmonary hypertension with mildly reduced right ventricular function. Given the recurrence of VT in combination with non-ischemic cardiomyopathy and conduction disease, there was high clinical suspicion for CCC. Serology returned positive for CD. Inpatient management consisted of maintenance oral amiodarone and carvedilol and placement of secondary prevention implantable cardioverter-defibrillator (ICD). She remained without further episodes of sustained VT on amiodarone in the outpatient setting.

Discussion: This is a case of recurrent VT as the initial presentation for a diagnosis of CCC. The pathophysiology of Chagas-induced patchy myocardial fibrosis is still unclear but can ultimately lead to transmural scar, commonly over the inferior and apical walls, which can be complicated by difficult to treat ventricular arrhythmias. Other associated common conduction abnormalities include left anterior fascicular block and right bundle branch block. Given that ventricular arrythmias are the leading cause of SCD in patients with CCC, screening patients at risk for CD is vital. Management of VT in Chagas typically includes use of amiodarone for both its antiparasitic and antiarrhythmic properties. However, over half of these patients develop recurrent VT requiring treatment with ablation or sympathectomy. ICD implantation remains a cornerstone of primary prevention of SCD for individuals with an ejection fraction of less than 35% given the high rate of arrhythmias as well as for secondary prevention of further VT if the patient had prior hemodynamically unstable ventricular arrhythmias.

Conclusion: This case serves as a reminder to consider Chagas Disease in patients diagnosed with non-ischemic cardiomyopathy, especially with inferior or apical wall motion abnormalities with conduction disease. These individuals are at high risk of arrhythmias, including VT, which can lead to SCD.

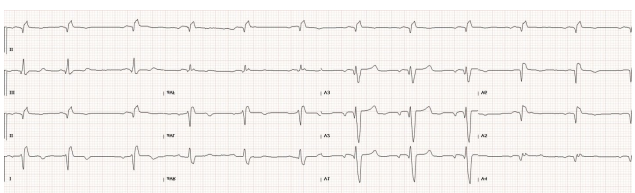

Image 1

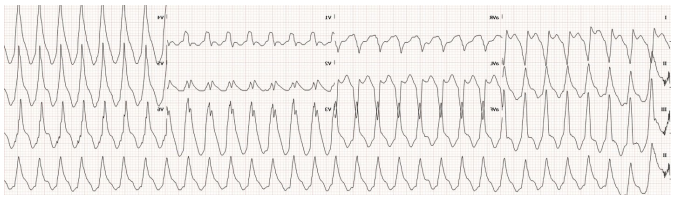

Image 2