ABSTRACT

Chronic Chagas cardiomyopathy (CCC) is a late complication of Chagas disease with various manifestations including arrhythmia, heart failure, thromboembolism, and stroke. In a patient with symptoms of heart failure and left ventricular apical aneurysm unexplained by structural heart or coronary vascular abnormalities, CCC should be strongly considered and inquiry made about exposure status. Typical electrocardiographic findings of bundle branch block, complete heart blocks, and ventricular arrhythmia are helpful clues. A positive trypanosomal immunoglobulin G antibody is supportive. Initiation of stage appropriate guideline-recommended heart failure regimen is the goal with careful attention paid to prevention of sudden cardiac death from ventricular arrhythmias.

Key words: Apical ventricular aneurysm, chagas cardiomyopathy, echocardiography, heart block, nonischemic cardiomyopathy

INTRODUCTION

Chagas disease (CD) results from infection with a hemoflagellate Trypanosoma cruzi. Chronic Chagas cardiomyopathy (CCC) is one of the late manifestations of heart involvement in CDs with a latent period of 5–30 years.[1] In nonendemic setting like the United States, recognition of cardiac involvement in CD can be quite challenging. With new onset heart failure, the initial evaluation is focused on establishing the etiology. CCC is an example of nonischemic cause of cardiomyopathy.

A transthoracic echocardiogram is one of the initial tests indicated. A left ventricular apical aneurysm is the most common echocardiographic abnormality seen in >50% of patients with CCC.[2] A high index of suspicion is required if this abnormality is detected on echocardiogram. History of exposure to endemic areas and typical electrocardiographic finding of various heart blocks (right bundle branch block, fascicular block, and complete heart block) are often important clues to the possibility of Chagas-related heart disease. Diagnosis is further supported by serological evidence of infection or tissue isolation of T. cruzi.

This is a report of the first diagnosis of Chagas-induced cardiomyopathy in a 37-year-old South American immigrant who presented with symptoms and signs of congestive heart failure to our institution.

CASE PRESENTATION

A 37-year-old male immigrant from Bolivia presented to our facility with gradual onset and progressive exertion-related chest discomfort and shortness of breath. His symptoms were associated with palpitation. Review of systems was unremarkable. He recalled having a farm house in Bolivia although he spent most of his time in the city. Past medical history was not significant. Family history revealed early myocardial
infarction in his father and CD in his sister. He was not a blood, tissue, or organ recipient.

Vital signs and laboratory studies were normal. EKG [Figure 1] showed right bundle branch block (RBBB). Transthoracic echocardiogram showed left ventricular ejection fraction (EF) of 40% with diffuse hypokinesis and possible apical aneurysm [Figure 2]. Cardiac catheterization with coronary angiography showed EF of 35%–40%, a dyskinetic apical wall and normal coronaries. Serum T. cruzi Immunoglobulin G (IgG) antibody was positive at 15 units consistent with current or past infection.

The final assessment was non-ischemic cardiomyopathy (NICMP) due to CD with the New York Heart Association Stage II (NYHA) heart failure. The patient was started on a low-dose Lisinopril. Beta blocker could not be started immediately because of low blood pressure.

**DISCUSSION**

CD commonly called as “American trypanosomiasis” is a zoonosis caused by hemoflagellate protozoan T. cruzi. Infection is spread to humans by infected
feces of *Triatominae bug* when it comes in contact with the oral mucus membranes, nasal mucosa, and the conjunctiva. Infection may also occur through transfusion of blood, transplantation of organs, through contaminated food, drinks, or by vertical transmission.\(^2\)\(^-\)\(^4\)

NICMP due to CD is one of the well-recognized chronic complications of CD with a latent period of 5–30 years.\(^1\) There are estimated 30,000–45,000 cases of CCC in the United States.\(^5\) The majority of cases involve immigrants from South America, where the disease is endemic. The etiology of cardiomyopathy in chronic CD is not completely understood. Four different mechanisms have been proposed to explain the cardiac findings in chronic CD including cardiac dysautonomia, microvascular disturbances, parasite-dependent myocardial damage, and immune-mediated myocardial injury.\(^6\)\(^-\)\(^8\) The histological consequence appears to be a chronic myocardial inflammation and fibrosis, which creates island of normal myocardial cells surrounded by dense fibrous tissue.\(^8\)

Presentation with exertional symptoms as in our patient often indicates overt heart failure which carries a poor prognosis.\(^10\) Both diastolic and systolic dysfunction may occur.\(^11\) On echocardiogram, the finding of reduced EF, apical wall dyskinesia, and or apical ventricular aneurysm in a patient with symptoms of heart failure should prompt considerations for CCC. Apical ventricular aneurysm particularly is seen fairly consistently in >50% of patients with cardiomyopathy due to chronic CD.\(^12\) Effort should be made to exclude ischemic etiology with coronary imaging by cardiac catheterization or any other modalities. Our patient had normal coronaries on diagnostic heart catheterization.

A positive serology typically an antibody to IgG subclass supports the diagnosis as it confirms exposure to *T. cruzi*.\(^13\)

Cardiomyopathy in CD may be accompanied by symptoms that suggest the presence of other cardiac manifestations of chronic CD such as syncope due to cardiac conduction disease manifesting as sinus

![Figure 2: Transthoracic echocardiogram showing apical ventricular aneurysm](image)
node disease, complete heart block, or bundle branch block. Our patient had evidence of RBBB on EKG [Figure 2]. Incessant ventricular arrhythmias leading to SCD and regional or global ventricular dyskinesia predisposing to thromboembolic phenomena are also major cardiac causes of morbidity and mortality in chronic CD. [10]

CCC with heart failure is treated in a similar manner to other causes of heart failure. There is little evidence for the benefit of antimicrobial treatment for CCC. [13] A combination of angiotensin-converting enzyme inhibitor and beta blockers improve symptoms and is considered an adequate regimen. [14] Careful considerations should be given to the need for the primary or secondary prophylaxis of ventricular arrhythmias. Despite appropriate treatment, CCC has a poorer outcome compared to other causes of NICMP with 4 years mortality of approximately 50%. [15] Ventricular dysrhythmias are a common cause of SCD, and most patients will require implanted cardiac defibrillator.

**CONCLUSION**

Although a rare cause of cardiomyopathy in the United States, the finding of apical wall dyskinesia or apical ventricular aneurysm in a patient with symptoms of heart failure and no evidence of myocardial ischemia should prompt the consideration for CCC. Historical evidence of any exposure to endemic areas should be sought, and appropriate serology test should be obtained to confirm diagnosis.

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**Conflicts of interest**

There are no conflicts of interest.

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