Case report

Ventricular tachycardia. Do not forget Chagas heart disease

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ABSTRACT

A 58 year old man from Ecuador was admitted to the emergency department with palpitations and ongoing ventricular tachycardia. Diagnostic tests: analysis, chest-x-ray, echocardiogram and cardiac magnetic resonance (CMR) were consistent with myocarditis. Due to the patient’s nationality, serological tests were performed and Chagas’ heart disease was confirmed.

We should never forget Chagas’ heart disease when we make the differential diagnosis, not only because the disease has become more frequent in non endemic areas, but also because sudden cardiac death may be the first clinical manifestation.

BACKGROUND:

In Latin America, Chagas’ heart disease is the most common form of cardiomyopathy, nevertheless, due to immigration, the disease has become more frequent in non endemic areas and should be included in the differential diagnosis.

There are two steps in the diagnosis of Chagas heart disease, firstly we must set up cardiac involvement and secondly, Trypanosoma Cruzi infection must be confirmed by serologic tests, polymerase chain reaction, or culture.

CASE PRESENTATION:

A 58 year old man with a history of palpitations, was admitted to the emergency department with ongoing symptoms of dizziness and palpitations, associated with chest pain. On admission, physical exam revealed that patient was haemodinamically stable, despite a 236 beats/min tachycardia.

A routine electrocardiogram showed a ventricular tachycardia with right bundle branch block morphology, and superior left axis.
Figure 1: Electrocardiogram on admission showing ventricular tachycardia with right bundle branch block morphology, and superior left axis.

There was no response to vagal maneuvers, nor adenosine. Finally tachycardia was controlled with amiodarone perfusion. A subsequent electrocardiogram showed sinus rhythm with right bundle branch pattern.

Figure 2: Electrocardiogram after pharmacological cardioversion, showing sinusual rhythm with right bundle branch pattern.
Secondly a chest x ray showed cardiomegaly, and troponine T levels were 0.05 ng/mL, with no other alterations.

![Figure 3: Chest x ray showing cardiomegaly](image)

The episode lasted 2 hours. Patient was admitted to cardiology Service. Firstly, an echocardiogram demonstrated a non dilated left ventricle, with mild to moderate systolic dysfunction due to global hypokinesis. There was no right ventricular dilatation, and the study did not suggest arrhythmogenic right ventricular cardiomyopathy. Valve disease was also excluded.

![Figure 4: CRM short axis view: subepicardial Late enhancement in lateral basal segments](image)

Secondly, with CRM arrhythmogenic right ventricular cardiomyopathy was ruled out. In cine imaging, there was no left ventricle dilatation, however mild systolic global dysfunction and segmental left ventricular dysfunction was detected, with hypokinesis of lateral basal and medial segments.

In myocardial delayed enhancement basal short axis views, we recognized subepicardial enhancement in lateral basal segments.
Myocardial late enhancement four chamber view showed there was mild-wall and subepicardial enhancement in lateral basal segments and in septal basal and medial segments.

Finally in myocardial delayed enhancement three chamber view, there was also subepicardial posterior basal enhancement.

![Figure 5: A: Four chamber view showing mild-wall and subepicardial enhancement in lateral basal segments and in septal basal and medial segments. B: Three chamber view with subepicardial enhancement in posterior basal segment.](image)

Coronary angiography was performed and coronary lesions were absent.

Myocardial fibrosis areas were non specific and compatible with myocarditis. Given the patients nationality a Chagas’ disease serology was performed and resulted positive, so Chagas heart disease was diagnosed and an implantable cardioverter-desfibrillator (ICD) was implanted in order to prevent sudden cardiac death.

**DISCUSSION:**

Chagas' disease (CD) is a protozoan infection due to Trypanosoma cruzi which is a major public health problem in endemic areas in many South American countries.\(^1\) The World Health Organization (WHO) has estimated that 10 to 12 million people are infected with T. cruzi, of which 20 to 30% will develop cardiac affection.\(^1\)

In Latin America, Chagas’ heart disease is the most frequent form of cardiomyopathy, however, we should not forget that immigration from endemic areas to Europe makes this disease more frequent than some years ago. Moreover, sudden cardiac death is sometimes the first manifestation, so it is necessary to think about this disease when making our differential diagnosis.\(^1-2\)
FORMS OF TRANSMISSION: The main form of transmission is vectorial transmission. There are also many other ways of transmission such as blood transfusion, organ transplantation and vertical transmission of T. cruzi from mother to child. Oral transmission secondary to the ingestion of contaminated products has also been reported.

ACUTE AND CHRONIC PHASES: The disease has an acute phase and a chronic phase. During the acute phase, many subjects remain asymptomatic. Only 10% of the patients develop symptoms; fever, myalgias, sweating, hepatosplenomegaly, and variable signs of cardiac failure secondary to myocarditis, and, less frequently, meningoencephalitis. During the acute phase, the electrocardiogram may show alterations, like changes in ST-T segments or first degree atrioventricular blockade, pericardial effusion can also take place. Cardiac involvement is responsible for 10% of deaths. Spontaneous recovery during the acute phase occurs over a period of a few months in about 95 percent of patients.

Initial infection occurs usually unnoticed, passing to the chronic stage one to several decades later. Natural history of chronic phase of Chagas heart disease is characterized by the gradual appearance of clinical and electrocardiographic markers of cardiac involvement, which signals the onset of clinical form. Progression from the indeterminate stage, in which patients do have a positive serology but no symptoms nor physical signs or basic (EKG and X rays) evidence of involvement, to the full-blown clinical form in the chronic phase takes place at a rate of approximately 2 percent per year, however some patients remain asymptomatic all their life.

During chronic form three basic syndromes often coexist in the same patient: cardiac dysrhythmia, heart failure and thromboembolism (systemic and pulmonary).

Firstly, cardiac dysrhythmia is responsible for 55-65% of mortality, and even though sudden cardiac death can occur in previously asymptomatic patients, it typically affects patients with severe cardiac affection, that is, patients with very low left ventricular function and frequently with cardiac aneurysms.

Secondly, heart failure causes 25-35% of mortality, there is often biventricular involvement, and signs of right-sided failure are usually more pronounced. Diastolic dysfunction appears generally before systolic dysfunction.
Lastly, thromboembolism is secondary to intracardiac thrombus formation and this syndrome causes 10-15% of mortality, specially due to pulmonary and cerebral embolism.  

**DIAGNOSIS:**  
To set up Chagas’ heart disease diagnosis, cardiac affection must be established and after that, Trypanosoma Cruzi infection must be confirmed by detection of T.Cruzi by either blood culture or PCR chain reaction, or by serologic tests.  
Apart from clinical signs and symptoms, many proves may help us to establish cardiac affection and prognosis: electrocardiogram, chest x ray, echocardiography and CMR.  
Firstly electrocardiogram, in which it is possible to find different alterations, the most common of which is right bundle-brunch block associated or not with left anterior fascicular block.  
Secondly, chest X-ray is also important because cardiomegaly has been described as a mortality predictor factor.  
Echocardiography provides important diagnostic and prognostic information. During the acute phase, pericardial effusion is frequent, left ventricular systolic function is usually preserved and it is possible to see apical aneurysms. However, during chronic phase ventricular systolic function ranges from normal systolic function (frequently patients in the indeterminate form) to severely depressed systolic function. Segmental left ventricular contractile abnormalities may also be detected, the most common is at the posteroinferior wall.  
Left ventricular aneurysms appear in more than 50% of patients, and are mainly located (82%) in the left ventricular apex. Left ventricular aneurysms have been described as an independent predictor of mural thrombus and have a significant association with stroke during a mean follow-up of 2 years.  
Left ventricular end systolic and end diastolic dimensions and also left ventricular ejection fraction have been reported as mortality predictors.  
Concerning CMR findings, during the first stage, systolic function is usually preserved or lightly decreased and myocardial delayed enhancement pattern is not specific, similar to other types of myocarditis. In more advanced stages, end-diastolic and end-systolic volumes increase, systolic function decreases and myocardial delayed enhancement, that is myocardial fibrosis, increases.
Carlos E.Rochitte et al, enrolled 51 seropositive patients for Chagas disease without history of myocardial infarction and at low risk for coronary artery disease (CAD): 15 patients in an indeterminate group (asymptomatic patients without signs of cardiac involvement and with normal echocardiography); 26 patients with known heart involvement defined as abnormal electrocardiogram and/or LV dysfunction and a third ventricular tachycardia group, comprising 10 patients with previously documented episode of ventricular tachycardia (VT).  

Myocardial fibrosis (MF) was detected in all groups, and with a progressively higher proportion of patients with MF from indeterminate group to VT group. Similarly, the magnitude of MF increased progressively. MF also increased progressively from NYHA functional class I to II and III patients.  

Patients with small areas of MF showed preserved ejection function, whereas patients with large areas of MF had severe LV dysfunction. Segmental MF and dysfunction involved preferably apex, inferior and inferolateral segments.  

MF parallels with other well-established prognostic factors, and provides unique information for clinical disease staging, as in other cardiomyopathies.  

In a systematic review of Publisher studies, several predictors of mortality have been described in chronic Chagas’ Heart disease: NYHA III/IV, cardiomegaly and non sustained ventricular tachycardia.

**TREATMENT:**  
Concerning management several points must be taken in consideration. Firstly, ventricular dysfunction and heart failure treatment is similar to that of other cardiomiopathies. However, B- blockers, some calcium channel blockers and amiodarone should be employed carefully because of risk of bradycardia. Cardiac transplant is also a good option for refractory heart failure. Despite some cases in which Chagas reactivation has been described, cardiac transplant results in Chagas’ Heart disease are even better than those of ischemic cardiomiopathy or non ischemic dilated cardiomyopathy.  

In order to prevent embolisms, anticoagulation should be considered in patients with atrial fibrillation, previous embolic events and with intracardiac thrombus. Conventional indications for cardiac pacing concerning bradiarrythmia published in guidelines for this objective also apply to Chagas heart disease.
ICD is indicated in non sustained ventricular tachycardia (NSVT) documented patients, in whom amiodarone could also be employed so as to decrease ICD’s discharges, and also in patients with sustained ventricular tachycardia (SVT) induced during electrophysiological study.

Due to progressive natural history of this disease, radiofrequency ablation is not indicated, so as to prevent sudden cardiac death, but could be helpful to decrease ICD’s discharges.

Finally, following the 2005 assent paper, etiologic treatment with benznidazol should be administrated to patients in chronic phase with positive serologic tests, and should be considered for other patients. Potential adverse effects have to be always taken into account.13

BIBLIOGRAFÍA:


