Chagas Disease: A Case Report
Enfermedad de Chagas: Presentación de un caso

Summary
Chagas Disease is a parasitic infection, mainly transmitted by a vector insect, that occurs in the American continent. The infection has acute, indeterminate and chronic phases; the latter is defined by the presence of symptoms from system involvement, most often cardiac or gastrointestinal. Chagas cardiomyopathy has multiple mechanisms leading to fibrosis, myocardial remodelling and systolic dysfunction; it features heart failure, arrhythmias and embolism. Diagnostic studies have demonstrated alterations that are initially autonomic, subsequently electrocardiographic and finally echocardiographic and radiographic. Cardiac magnetic resonance imaging allows for the detection of alterations in contractility, remodelling and systolic dysfunction with the temporal and spatial resolution of video sequences, and also of fibrosis using late gadolinium enhancement; in some, from early stages of disease. We present the case of a patient with heart failure syndrome, showing radiographic and echocardiographic alterations, in whom cardiac MRI reflects structural, functional and tissue changes due to Chagas cardiomyopathy.

Resumen
La enfermedad de Chagas es una infección parasitaria, principalmente transmitida por un insecto y ocurre en el continente americano. La infección tiene fases aguda, indeterminada y crónica; esta última definida por síntomas de compromiso sistémico, principalmente cardíaco o gastrointestinal. La cardiopatía tiene múltiples mecanismos que llevan a fibrosis, remodelación miocárdica y disfunción sistólica; se manifiesta con falla, arritmias y cardioembolismo. Los estudios diagnósticos han demostrado alteraciones autonómicas inicialmente, subsecuentemente electrocardiográficas y finalmente ecocardiográficas y radiográficas. La resonancia magnética (RM) cardiaca permite ver alteraciones de la contractilidad, remodelación y disfunción sistólica con la resolución espacial y temporal de las secuencias de cine y la fibrosis mediante el realce tardío, en algunos incluso desde estadíos tempranos. Se presenta el caso de un paciente con síndrome de falla cardíaca, alteración radiográfica y ecocardiográfica, en quien la RM refleja cambios característicos estructurales, funcionales y tisulares de la cardiopatía chagásica.

Introduction
A 49-year-old man from Alto Baudó (Chocó) and resident of Medellín (Antioquia), who had previously worked as a farmer and gold miner and was displaced by the violence, was kidnapped for 22 months. He consulted for dyspnoea of great efforts of two months of evolution, with one week of progression to dyspnoea of small efforts, associated with nocturnal paroxysmal dyspnoea and pain in the right hypochondrium; he had no relevant personal medical or surgical history. Physical examination found blood pressure of 100/60 mm Hg, heart rate of 80 beats per minute, jugular engorgement at 30°, arrhythmic heart sounds, maximum left impulse point shift and hepatomegaly.

A chest x-ray was taken showing cardiomegaly by enlargement of the left chambers and no signs of decompensated heart failure (Figure 1). Echocardiography showed dilation of the left ventricle with generalized hypokinesia and severe systolic dysfunction, diastolic dysfunction and dilation of the left atrium (Figure 2).

In the absence of risk factors or echocardiographic findings of ischemic disease, he underwent cardiac magnetic resonance imaging (MRI), with the help of...
the The following findings: dilation of the left cavities (Figure 3), diffuse hypokinesia without regional contractility disorders (Figure 4), severe systolic dysfunction with an 11% ejection fraction and a non-ischemic late enhancement pattern, with diffuse involvement of epicardial and apical predominance (Figure 5). Chagas disease was proposed as the first possibility, a diagnosis that was supported by positive serology for *Trypanosoma cruzi*.

**Discussion**

Chagas disease occurs in America, between latitudes of 40° north and 45° south and at altitudes up to 1500 meters above sea level. The most affected geographical region is the Chaco, located in Bolivia, Paraguay and Argentina. The country where the maximum prevalence is reached is Bolivia with 7% (1, 2). Its etiologic agent, *Trypanosoma cruzi*, is a protozoan with a complex life cycle, capable of parasitizing any mammalian cell with a nucleus. Several species of hemato- phagous insects, called triatomines, act as vectors by frequently infesting houses in endemic rural areas and feeding on people. Other mammals, both domestic and wild, act as reservoirs. Other less frequent forms of spread include vertical transmission, ingestion of food contaminated by triatomines, blood transfusion, transplantation and puncture with contaminated needles (3).

In acute infection, symptomatic people develop fever, chills, chills, myalgia, tachycardia, and rash. An over raised skin lesion may appear at the site of entry of the parasite, called the chagoma and unilateral periorbital edema, known as the Romaña sign. Less frequently seen are conjunctivitis, lymphadenopathy, hepatosplenomegaly, and meningeal irritation. Laboratory study in this acute phase may show anemia, thrombocytopenia and elevation of liver enzymes; quantification of immunoglobulin M for *T. cruzi* is inaccurate and immunoglobulin G is frequently negative in this acute phase.

In 1 to 2 months there is resolution of the acute symptoms, and it gives way to the indeterminate phase that is clinically silent and can last from a few months to a lifetime. Up to 30% of patients develop chronic disease, with manifestations in which cardiac involvement predominates - which may be associated with cardioembolic cerebrovascular disease. Gastrointestinal disease, which is shown to coexist with megacolon, megaesophagus and other dysfunctions, can coexist (4).

In more than 90% of cases there is cardiac involvement, including those in the indeterminate phase. Up to 40% of patients develop symptoms. The incidence is 3% per year among people with infection, mainly men between 30 and 50 years of age. It manifests predominantly with right heart failure syndrome and also includes palpitations, syncope and thromboembolic events. There are many theories about the pathogenesis of heart disease, such as direct parasite aggression, parasympathetic derangement with a continuous increase in sympathetic tone, immune mechanisms triggered by the parasite, and autoimmune, microvascular, and coronary flow abnormalities resulting in perivascular inflammation, endothelial dysfunction, and platelet activation. The result of the continuous action of one or more of these mechanisms is myocardial structural modification associated with inflammation, necrosis, hypertrophy and ventricular dilatation, and bands of fibrous tissue can be observed in histology replacing the myocytes and accumulation of extracellular collagen that contains groups of muscle fibers. Inflammatory changes and fibrosis have been shown in people with indeterminate stage disease (5).

Patients with heart disease are asymptomatic in the indeterminate phase and during this phase, have serological positivity and alterations in the electrocardiogram. Non-invasive studies have shown alterations in this population, such as depression of the pressor and chronotropic response during exercise, ventricular arrhythmias induced by stress, shortening fraction and mean velocity of left ventricular shortening decreased, abnormalities of diastolic function and changes in segmental contractility (6).

Cardiac MRI has the ability to show structural and myocardial contractility alterations in film sequences, given its spatial and temporal resolution. In the initial stages, this sequence shows segmental alterations in contractility, which become more pronounced as the disease progresses. In advanced stages, cinema images show a marked decrease in contractility, a decrease in ejection fraction, diffuse parietal thinning and in some cases aneurysms and thrombi; of these, the apical aneurysm is characteristic, also called vortex injury, and is clearly demonstrated in MRI. Post-contrast intravenous gadolinium sequences acquired in late phase show myocardial enhancement in sites with fibrosis (6).

The study by Regueiro et al. showed changes in patients with only electrocardiographic manifestations, with tardive enhancement in 16% and dyskinesia in 3%. In patients with echocardiographic manifestations, this enhancement reached 52%, with heterogeneous patterns: subendocardial in 26.8%, intramural in 14%, subepicardial in 22.6% and transmural in 36%. Late enhancement was associated with low left ventricular ejection fraction, and is more common in the apex and inferolateral segment (7). Ventricular tachycardia is a common consequence of chagasic cardiomyopathy, leading to an annual mortality rate of 0.2% to 19% among patients. Mello et al (6) studied 41 patients with associated ventricular dysfunction or electrocardiographic changes and of these 63% had manifest ventricular tachycardia. MRI showed delayed enhancement, indicating myocardial fibrosis, in all patients. Two or more segments with transmural scar were the most frequent finding in the prior ventricular tachycardia group, which was a predictor of arrhythmia, regardless of ejection fraction, age, sex, and percentage of late enhancement. These patients were followed for a year and a half; those without prior ventricular tachycardia, transmural scarring, and those with less than 6% fibrosis showed no events. On the other hand, three patients suffered sudden death, all had one or more transmural scars and none had previous ventricular tachycardia. This is evidence of the relevance of cardiac MRI in assessing the risk of fatal arrhythmia in patients and helping to select candidates for therapies such as cardioembolitator (6).

In this case, a patient with symptoms of heart failure syndrome, who is in the group with chronic disease, is described, in addition to showing radiographic manifestations such as cardiomegaly and echocardiographic manifestations such as dilation and reduction of the ejection fraction; his MRI showed thinning of the myocardial wall, hypokinesia and akinesia with systolic dysfunction in the cinema sequence, consistent with the structural manifestations described for advanced heart disease and the pattern of heterogeneous late enhancement and apical predominance compatible with fibrosis, consistent
Figure 1. PA and lateral chest x-ray. Increase in the size of the heart, the retrosternal space is preserved by increasing the left cavities. Hilar vessels with no increase in calibre and no pulmonary opacities indicating decompensation.

Figure 2. Echocardiography. Long axis 4 cameras. a) End of systole images and b) end of diastole. Dilation and poor contractility of the left ventricle (LV).

Figure 3. Cardiac MRI. a) Anatomical coronal sequence in black blood, b) PA chest x-ray. In the comparison, the cardiac contours are characterized and the large dilation of the left ventricle (LV) is evident.
with the pattern of this in Chagas heart disease. The patient benefited from the cardiac MRI study, as their findings, coupled with the clinic, epidemiology and serology allowed diagnosis and classification to be made without resorting to invasive methods.

**Conclusion**

Cardiomyopathy occurs in the majority of patients with Chagas disease, causes fibrosis and myocardial remodeling, its manifestations are progressive, due to systolic dysfunction, arrhythmia and embolism, its echocardiographic and radiographic manifestations are late. The patient in this case has clinical and echocardiographic manifestations of advanced heart disease, in whom the use of cardiac MRI allowed the visualization of structural and functional disorders of the myocardium, as well as the detection and characterization of the distribution pattern of myocardial fibrosis. This method helped in the non-invasive diagnosis and staging of heart disease and its aetiology.
References


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