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Cardiac involvement in chagas disease

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Abstract

Chagas disease is an infectious disease caused by Trypanosoma cruzy, this is a highly frequent disease in rural areas of Latin America, it is characterized by presenting two phases, an acute one which is often asymptomatic and with low mortality which is mostly conferred to acute myocarditis due to Chagas, and a chronic phase which consists of three presentations, one indeterminate, one digestive and one cardiac, the latter being the most serious and the cause of greater complications and higher mortality in patients that occur with this, it should be noted that the alterations presented at the cardiac level that affect the heart in its structural, autonomic, functional and electrical conduction aspects, which require specialized treatment and diagnostic aids to determine characteristic myocardial involvement, degree of involvement and patient prognosis due to Chagas disease.

Keywords: Chagas; Chagas heart disease; *Trypanosoma cruzi*; Chagas complications

1. Introduction

Chagas disease is an infectious disease that mainly affects rural populations, it is caused by a parasitic agent called *Trypanosoma cruzi*, subphylum Mastigophora, order Kinetoplastida, family Trypanosomatidae, this protozoan is the cause of a tissue and blood disease which occurs mostly in Latin America and according to the world health organization it is estimated that there are between 6 and 7 million people infected by *Trypanosoma cruzi* in the world, most of them in Latin America. This disease was initially reported by the Brazilian physician Carlos Ribeiro Justiniano Chagas, although paleontological studies from 1909 report that *Trypanosoma cruzi* (T. Cruzi) was found in mummies from 4000 BC to 1400 AD in regions north of the coast of Chile. This infection is caused by flagellate protozoa T. cruzi and is often transmitted by hematophagous insects (1,2).

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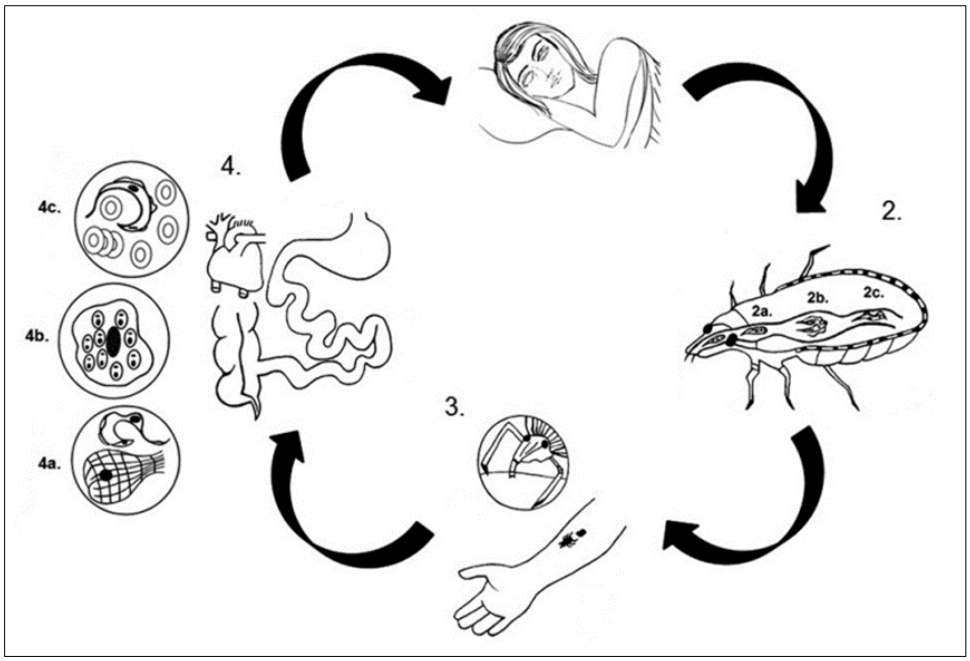


Taken from: Palmezano JM, Plazas LK, Rivera KE, Rueda VP. Chagas disease: reality of a frequent pathology in Santander, Colombia. MEDICAL UIS. 2015; 28(1): 81-90.

Figure 1 Life cycle of T. cruzi

- Final hosts are wild, domestic, vertebrate and human.
- Vectors are insects called "whistles'' that infect cones that circulate in the blood by biting a host infected with Chagas disease. Conical mastigotes (2a) and epimastigotes (2b) are present in the digestive tract of insects, while subcircular conical mastigotes are present in the rectum and feces, which are the infective forms of vertebrates (2c).
- The human being is infected with the feces of the vector, which remains on the skin or mucous membranes at the time of the bite.
- Intracellular parasites affect different tissues such as the heart and the gastrointestinal tract. Infection begins with subcircular pyramidal flagellates (4a), which multiply intracellularly to become amastigotes (4b), which then appear in the peripheral blood and circulate in the infection vector (4c).

There are various mechanisms of oral transmission, contact with the skin through trypomastigotes found in the fecal matter of the vector, contagion can also occur through transfusion of blood components, organ transplants and maternal-fetal transmission (2).

Currently, the disease caused by T. cruzi has become more important because, in addition to the increase in reported cases, there has also been a significant increase in heart disease secondary to infection by this agent, a highly serious complication that has a significant impact in the life expectancy of the sufferer (3).

In this parasitosis, important stages are highlighted: an acute phase, a chronic phase, initially in the acute phase it is usually a mild condition which in many cases can go unnoticed due to the absence of symptoms, or the presence of them with imprecise characteristics in this phrase. , this is due to the fact that at this moment is where high amounts of the parasite are found circulating in the blood, instead the chronic phase is characterized mainly by the presence of alterations at the cardiac level considered as "cardiomyopathy due to Chagas" and alterations in the cardiac apparatus. Gastrointestinal (3).

The clinical manifestations present in these patients will be determined by the phase in which the patients are and in the same way they will have different forms of diagnosis and treatment, so it is necessary that the treating personnel be clear about each of the characteristics present. in each phase and forms of presentation of each of them; The disease begins in the acute phase with a feverish picture due to infection by parasites, since it is not found in time, the disease will progress to the chronic phase where the frequent forms of the chronic phase will be seen, such as the form indeterminate, the cardiac form and the digestive form (4)

Table 1 Chagas disease transmission mechanisms

vector transmission Incubation period: 5-15 days	It occurs through contamination of the skin and mucous membranes contaminated with feces from infected triatomines which, when bitten, defecate on the host, leaving dead cells under the skin. Trypomastigotes are transmitted by sores on the skin (secondary to scratching), by contact with the conjunctiva of the eye or through the proboscis of an insect when bitten.
oral transmission Incubation period: 3-22 days, the period between Ingestion of blood contaminated with the parasite and excretion of infectious forms through feces is 10 to 20 days.	Given through the consumption of food or drinks contaminated with feces of infected triceratops, contamination by utensils used to prepare food or handling carcasses of infected animals. Be suspicious of symptoms: Simultaneous presence of two or more confirmed acute cases, with an epidemiological link between them. Presentation of severe clinical picture. Absence of domiciled triatomines or around the domicile in the area of occurrence of the cases.
Transmission by blood transfusion/ transplantation Incubation period: 30 - 40 days.	occurs due to the presence of live, infectious chromosomes in the blood of donors in endemic areas or transplantation of contaminated organs
vertical transmission Incubation period: Congenital: 4 to 9 months. During childbirth: variable.	Passage of the parasite through the placenta to the fetus.
accidental transmission Incubation period: variable	by puncture or other contact with material contaminated with T. cruzi or transmission through breast milk

Table 2. Most important clinical phases of Chagas disease and clinical picture

		Clinical picture
chronic phase	Between 7 days and 84 days: Incubation easy detection febrile period	Romana sign (45.8%): acute infection and swelling of the right eye, Chagoma (1.7%): If the penetration was through the skin, the so-called inoculation chagoma appears. It is a subcutaneous, rounded, erythematous, hard and painless nodule accompanied by lymphadenopathy and fever. Edema (3.4%) Headaches (47.5%) Febrile syndrome (44.7%) Hepatomegaly (8.5%) Signs of heart failure (17%) with myocarditis. Myalgias (50%)
chronic phase	Between 3650 days and 10950 days: Difficult detection Indeterminate form (70%) cardiac shape digestive form	indeterminate form digestive form cardiac shape

Taken from: Ministry of Health and Social Protection - Colombian Medical Federation, Chagas disease - memories, 2013.

Chagas heart disease occurs after the acute infectious phase, untreated Chagas disease progresses to the chronic phase, initially asymptomatic or indeterminate. Subsequently, 20-30% of patients develop heart disease (cardiac type), 10% develop gastrointestinal disease (digestive type) or both (mixed type), and less than 5% develop neurological disease. The rest will remain indeterminate without any clinical symptoms throughout life. Due to the potential severity and frequency of cardiac complications, the Second Symposium on Imported Chagas Disease focuses on the clinical features and requirements for proper diagnosis, management, and treatment of chronic Chagas disease. The pathogenesis of the disease is controversial, although current knowledge indicates a mixed aetiology, in which the parasite is directly involved, causing myocardial damage and related autoimmune phenomena. Other genetic mechanisms of the disease described include microvascular alterations and autonomic denervation (4)

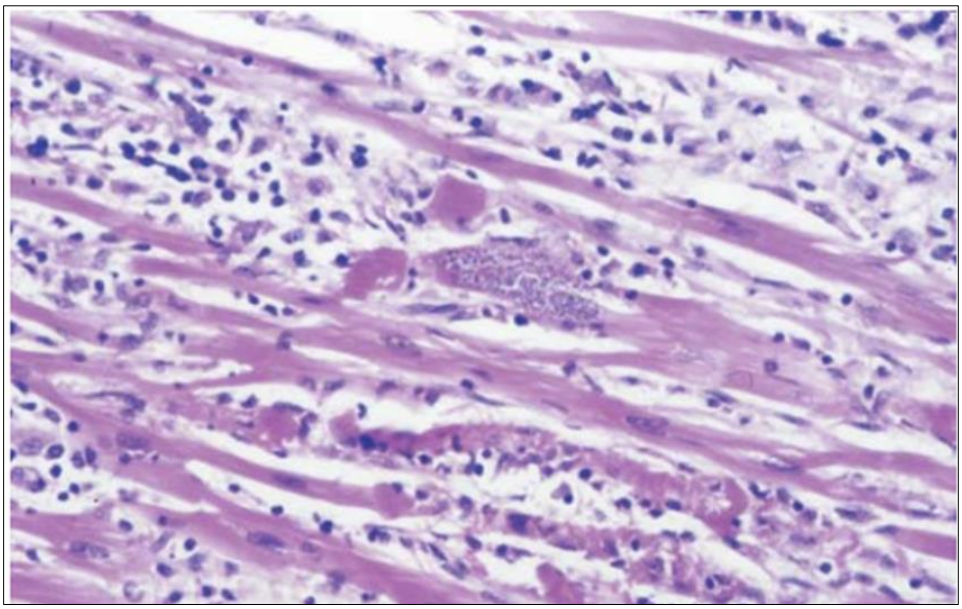
2. Methodology

To carry out this document, a systematic search of original articles, case reports and bibliographic reviews was carried out in databases including pubmed, ScienceDirect, Elsevier, Scielo, Ovid and Medline, using articles related to Cardiac conditions in Chagas disease, since 2005 - 2022, also using MeSH terms such as Chagas, Chagas heart disease, *Trypanosoma cruzi*, Chagas complications and Boolean operators and and or, obtaining approximately 10-35 relevant results in the English and Spanish languages. 13 articles that met these characteristics and that spoke in a relevant way of the topic addressed.

3. Results

3.1. Chagas disease has two phases, an acute phase and a chronic phase

3.1.1. The acute phase



Taken from: Pérez C, Orjuela A, Vanegas D, Montenegro J, Etiology, pathophysiology and immunology of Chagas disease, Colombian Society of Cardiology and Cardiovascular Surgery, 2007.

Figure 2 Acute chagoic cardiomyopathy (histological cut)

It is the first stage, in which the amastigotes of *Trypanosoma cruzi* reproduce and destroy the cells. The parasites freely attack other cells, they also alter and trigger an inflammatory response and invade different types of white blood cells. Inflammatory lesions located at the entrance are visible as inoculation vesicles and are called chagomas. As the swelling spreads to the lymph nodes in the area, the lymphatic channels become blocked and local swelling develops. When the eyelids are involved, it is called a Roman sign. Later, intracellular parasites are found in the lymph nodes and other organs such as the spleen, bone marrow, heart, gastrointestinal tract, adrenal glands, brain, and sometimes the ovaries, testes, and thyroid. Repaired tissue cells, muscle cells, adipocytes, glial cells and, in general, the cells of the reticuloendothelial system are destroyed by the growth and reproduction of the parasite. In this phase the circulating parasite is in the blood in detectable quantities, the symptoms can last up to 12 weeks or disappear spontaneously, therefore this phase can go unnoticed since, on many occasions, it becomes asymptomatic. Among the clinical

symptoms, 50% had myalgia, 47.5% had headache, and 45.8% had symptoms of Rome. If the conjunctiva has penetrated, the manifestation is unilateral, flexible, painless swelling, often accompanied by purple-red eyelids, conjunctiva. There is increased congestion and swelling in the preventricular lymph nodes, but parotid and subnodal lymph nodes may also be involved. This sign may go away on its own after two to three weeks. 44.7% had febrile syndrome, 17% symptoms of heart failure accompanied by myocarditis, 8.5% hepatomegaly, 3.4% edema, and 1.7% round, red, hard, painless subcutaneous nodules, diarrhoea, swollen glands and fever that can occur if the agent penetrated through the skin, the acute mortality rate is low, around 10%, and only 1% to 2% of patients can be diagnosed in this phase. The main cause of death is myocarditis, which can be evidenced in electrocardiographic changes, it presents with signs of cardiac compromise, where changes in the t wave, QT and PR prolongation, right bundle branch block, among others, can also be seen. We can find complications such as meningitis and bronchopneumonia, which are more serious complications of the acute phase that can lead to death (5)

Severe diffuse myocarditis with infiltration of lymphocytes and plasma cells, edema, and severe fibrous destruction. There are myocardial fibers in the center with abundant amastigotes. Hematoxylin-eosin stain, 400X

3.1.2. Chronic phase

The chronic phase is characterized by beginning when the infection becomes persistent and the acute episode resolves and develops clinically 10 to 30 years after the initial infection. Target organ damage, which does not occur in about 70% of cases and is asymptomatic for life, is known to be unspecified. In other cases, it can damage the heart or hollow internal organs, especially the esophagus and colon, although this form of digestion is rare in Colombia. People with chronic Chagas disease who have a weakened immune system may experience relapses with worse symptoms, in this case known as reactivation. The clinical picture of reactivation may be similar to that of the acute phase, but atypical patterns may also be observed, such as cerebral reactivation, which, although less frequently, is a mass, tumor, pseudotumor, or chagoma, followed by large cone meningitis. Cerebrospinal fluid, which can be fatal if neurologically damaged The most important clinical signs and symptoms related to Chagas disease and immunosuppression are panniculitis, meningoencephalitis, and myocarditis; Likewise, this phase is characterized by forms of presentation, whether indeterminate, digestive form or cardiac form (6)

3.1.3. Indeterminate form

This occurs in patients with positive serology for Chagas but who have not presented signs, symptoms, electrocardiographic alterations or chest X-rays, approximately 70% to 80% of these patients progress favorably without signs of cardiac damage or digestive, however, 2 to 5% of these patients progress to the cardiac form of the disease (6)

3.1.4. Digestive form

This form of presentation affects hollow viscera such as the colon, representing a megacolon with characteristic symptoms such as slow-onset constipation of a progressive nature that may also be accompanied by meteorism, abdominal distension, tympanism and dyschetia; and also affecting the esophagus presenting as megaesophagus in the latter, characteristic symptoms such as slowly progressive dysphagia with exacerbation of food intake are presented, the reason for regular consultation of this presentation is regurgitation esophageal pain, heartburn, hiccups, sialorrhoea in the company of glands hypertrophic salivary glands, cough and malnutrition, in this form inflammation and tissue injury predominate (7)

3.2. Cardiac form

In this phase, cardiac involvement is revealed, which progresses to dilated cardiomyopathy and congestive heart failure in approximately 30% of cases. This is considered the cause of more deaths in patients with Chagas disease. It also evidences other alterations such as arrhythmias such as ventricular extrasystoles, ventricular tachycardia, ventricular and atrial fibrillation, bradyarrhythmias and AV blocks, these alterations are accompanied by symptoms such as palpitations, syncope, fainting, as well as a high incidence of thromboembolic processes and ventricular aneurysms. That suggests a high mortality in patients who suffer from it. These alterations at the cardiac level due to Chagas disease are responsible for approximately 21,000 deaths per year in Latin America, with sudden death occurring more frequently in 48% of patients and followed by heart failure with a fatality rate of 37% (two). In these cardiac abnormalities, massive overgrowth of filamentous cardiomyocytes occurs, leading to ventricular remodeling, bilateral ventricular dilatation, and, in most cases, greater enlargement of the right side of the heart than the left. Thin ventricular walls are common, as are apical aneurysms, posterior left ventricular aneurysms, and wall thrombus formation. Diastolic function begins to deteriorate, contractions are delayed (7)

Table 3. Signs and symptoms most frequently related to cardiac involvement by T. Cruzi

Symptoms secondary to bradyarrhythmias and tachyarrhythmias: Palpitations, Syncope, presyncope, fainting Sudden death.
Symptoms of heart failure: Dyspnea on exertion Pain in the right hypochondrium (due to liver congestion) Symptoms of pulmonary congestion such as orthopnea and paroxysmal nocturnal dyspnea.
Symptoms secondary to venous and systemic thromboembolic phenomena: Pulmonary or systemic embolisms Cerebral vascular accident (generally ischemic).
Microvascular (or esophageal) alterations: Precordial or retrosternal pain.

Taken from: Gascón J, Albajar P, Cañas E, Flores M, Gómez J, Herrera E, Lafuente C, Luciardi H, Moncayo A, Molina L, Muñoz J, Puente S, Sanz G, Treviño B, Salles X, Diagnosis, management and treatment of chronic chagasic heart disease in areas where Trypanosoma cruzi infection is not endemic, Rev Esp Cardiol. 2007; 60(3): 285-93

Chagas disease has several characteristics that distinguish it from other more common heart diseases:

- It is a fibrous heart disease, generally located in the posteroinferior and apical regions of the left ventricle, in the sinus node, in the conduction system below the node and bundle of His, mainly affecting its contractile segment.
- This is a dilated cardiomyopathy that tends to bulge, especially at the top of the heart.
- There is a high potential for arrhythmias, ventricular arrhythmias are common and are often accompanied by arrhythmias (of sinus and/or atrioventricular origin).
- Shows a high frequency of thromboembolism.

3.2.1. Congestive heart failure

Manifested by increased dilatation of the heart, gradual loss of the effective volume of the left ventricle, myocardial fibrosis and predisposition to the development of thromboembolism. Symptoms appear between the ages of 20 and 50, mainly systemic congestion due to biventricular failure, rare episodes of paroxysmal nocturnal dyspnea, and acute pulmonary edema.

3.2.2. Thromboembolism

Sometimes this is the first symptom of Chagas disease. Cardiac embolism can reach the lungs and systemic circulation, with the brain being its most obvious target organ. For severe Chagasic heart disease, if the patient is a candidate, the treatment options would be resynchronization and heart transplantation (8).

These alterations are accompanied by precordial pain, often atypical, although it may eventually resemble ischemic heart disease. In the natural course of the disease, changes in the heart appear gradually 20 to 30 years after infection. Regarding the secondary diagnostic methods, it can be concluded that a normal ECG excludes the presence of moderate or severe left ventricular dysfunction with a negative predictive value close to 100%. On the other hand, the more the electrocardiogram changes, the worse the ventricular function (8)

Table 4 Most frequent electrocardiographic alterations in chagasic heart disease

Complete right bundle branch block isolated or associated with left anterior hemi block
Ventricular extrasystoles, isolated or repetitive
Primary alteration of ventricular repolarization, which may mimic ischemic heart disease
Electrically inactive zones (q waves)
Atrioventricular blocks
Other less frequent alterations: left bundle branch block, sinus dysfunction, supraventricular tachyarrhythmia (particularly atrial fibrillation)

Taken from: Arrúa Torreani, Chagasic Heart Disease, and Mem. Inst. Investig. Science. Health, Vol. 11(2) December 2013: 65-77.

Table 5 Most Frequent Echocardiographic Findings in Chagasic Heart Disease

Segmental alterations of myocardial contractility (75% of cases)
The most frequently affected regions are the posteroinferior wall and the apex of the left ventricle.
Aneurysms in the apex of the left ventricle (of variable morphology and size, but with a narrow neck and frequently with mural thrombi)
Hypokinesis or akinesia of the posterior inferior wall
Involvement of the basal portion of the anterior septum, with akinesia and even
formation of subaortic aneurysms
Dilated cardiomyopathy
Right ventricular dilatation and dysfunction

Taken from: Arrúa Torreani, Chagasic Heart Disease, and Mem. Inst. Investig. Science. Health, Vol. 11(2) December 2013: 65-77

Diagnosis is based on epidemiological, clinical and serological syndromes. In the acute form, IgM serology is positive for T. cruzi. Other techniques can detect parasites in blood smears from immunocompromised patients and in cerebrospinal fluid, bone marrow, or lymph node aspiration from immunosuppressed patients. Blood cultures and xenogeneic diagnoses take a long time to obtain results and have low sensitivity. The chronic form shows IgG seropositive, caution should be taken in case of false positives for leishmaniasis, malaria, syphilis and its low specificity, requiring multiple samples before diagnosis. PCR positivity for parasites at any time during the disease is diagnostic as long as the symptoms and epidemiological support are met (8)

Table 6 Summary of the main indications for diagnostic tests in chagasic heart disease

Electrocardiogram	**In all patients with evidence of T. cruzi infection**
Echocardiography	In all patients with evidence of T. cruzi infection
24-hour Holter	Low output symptoms (syncope, near syncope, palpitations...) and/or electrocardiographic changes compatible with tachyarrhythmia or bradyarrhythmia
Electrophysiological study	Unclear syncope, ventricular tachycardia (VT) with symptomatic or ventricular dysfunction
Stress test	Valoración de la respuesta funcional y/o de la capacidad cronotrópica
Cardiac catheterization	Chest pain with anginal characteristics

Taken from: Arrúa Torreani, Chagasic Heart Disease, and Mem. Inst. Investig. Science. Health, Vol. 11(2) December 2013: 65-77

This treatment is indicated in cases of acute infection, immunocompromised patients of any cause and the professional risks of handling samples. There is currently no consensus on the value of treatment in the chronic or indeterminate phase. The treatment is based on two antiparasitic drugs: nifurtimox - reduces the duration, severity and mortality with multiple side effects (hematological, hepatic, dermatological, neurological...) with a variable area of efficacy (8-10 mg/kg/day/90 -120 days) and bennidazole (5 mg/kg/day/60 days). Neither one turned out to be better than the other. In each case, supportive measures for cardiomyopathy (ACE inhibitors, diuretics, anticoagulants) are required. In the treatment of electrical disorders, class I antiarrhythmic drugs are contraindicated because of their increased mortality from torsades de pointes. Treatment with amiodarone and limited beta-blockers is recommended due to their inhibitory effect on conduction and constriction. In selected cases, defibrillators and pacemakers were used. Heart transplantation is in NYHA class III-IV functional class and carries a risk of disease recurrence after pharmacological immunosuppression, which is often more toxic than natural disease. For this reason, nifurolimus prophylaxis should be started before surgery. The acute phase of southern trypanosomiasis has a good prognosis, unless it is visceral. The problem is in the chronic phase, where 10-year survival is 98% in asymptomatic patients, dropping to 65% if they have some type of ECG involved and 9% if they have heart failure. 50% of heart failure patients die within 2 years of diagnosis. There is now a very short series of transplant patients with survival rates ranging from 46% to 71% (8)

	Benznidazole
Description	First line of treatment. Better adverse effect profile than nifurtimox, and greater evidence of efficacy.
Dose	age <12: 5-7.5 mg/kg w/ d.o.v. divided in two doses per 60 days >12: 5-7 mg/kg w/ d.o.v. in two divided doses for 60 days
Adverse effects	• Mild dermatologic flares τηατ Anorexia/weight loss usually respond to antihistamines. Nausea and vomiting • Paresthesi α Leukopenia • Peripheral neuropathy Thrombocytopenia
	Nifurtimox
Description	Nitrofuran inhibits pyruvic acid synthesis and alters carbohydrate metabolism of *T. cruzi*.
Dose	age <10: 15-20 mg/kg w/d divided in 3-4 doses for 90 days 11 - 16: 12.5-15 mg/kg w/d divided in 3-4 doses for 90 days >17: 8-10 mg/kg w/d divided in 3-4 doses for 90 days
Effects adverse	• Anorexia/weight loss • Nausea • Vomiting • Abdominal discomfort • Headache • Dizziness or vertigo • Mood swings • Insomnia • Myalgia • Peripheral neuropathy • Impaired short-term memory • Leukopenia

Taken from: Castro V, Zumbado R, Hines K, Chagas disease: Cardiac affection, Revista Médica Sinergia, 2019 Vol. 4 (5), ISSN: 2215-4523 / e-ISSN: 2215-5279 http://revistamedicasinergia. Com

Figure 3 Medications against *T. Cruzi* infection

3.3. Management of ventricular dysfunction and heart failure

In general, the management of these patients is closely related to the management of other cardiac conditions. Note also that, in many cases, unapproved general strategies specific to this disease are applicable to heart disease in Chagas disease. Beta blockers and ACE inhibitors are recommended in patients with moderate to severe ventricular dysfunction (EF < 40%). Amiodarone for complex arrhythmias, when considering the therapeutic management of a patient, several characteristics of Chagas' heart disease must be taken into account.

- Chronic Chagasic heart disease is manifested by a high frequency of arrhythmias (atrioventricular block, sinus dysfunction). For this reason, medications such as digitalis, beta-blockers, some calcium channel blockers, and amiodarone should be used with caution, starting at a low dose and closely monitoring for complications.
- Patients with heart failure can be evaluated as candidates for heart transplantation. Although some cases of recurrent Chagas disease have been reported, the series of published studies have shown positive results and better survival for these patients compared to patients with myocardial ischemia or idiopathic dilated cardiomyopathy.
- In this pathology there is no indication for cardiomyoplasty or reconstructive surgery.

The efficacy of cardiac resynchronization therapy in these patients is still under discussion, and echocardiographic and electrocardiographic criteria should be used to assess the indications.

Unpublished initial experience that stem cells improve myocardial function in patients with Chagas disease.

Treatment of bradycardia: Symptomatic bradycardia is sometimes a candidate for pacemaker implantation. International guidelines for the useful implantation of pacemakers in patients with Chagas' heart disease.

Management of ventricular arrhythmias: Premature beats and ventricular tachycardia are common in patients with Chagas disease, most commonly myocardial damage leading to reentry. Most of the persistent ventricular tachyarrhythmias in patients with Chagas disease do not originate in the left parietal aneurysm, but in the inferior lateral part. As this is a progressive disease with multiple arrhythmia sites, radiofrequency ablation should not be considered the technique of choice. Instead, despite the lack of specific studies on chronic heart disease in South America, the implantation of an implantable defibrillator (ICD) is recommended to reduce the probability of sudden death in the following situations:

- Sustained ventricular tachycardia (SVT). In these patients, based on experience, amiodarone is recommended as an adjunct to the ICD to reduce the frequency of lightning strikes and reduce the likelihood of thunderstorms. If the patient is in excessive shock despite these measures, radiofrequency ablation should be considered. Amiodarone can be used in patients with non sustained ventricular tachycardia (NSVT) and normal (non-induced) electrophysiological tests (9).

4. Discussion

Chagas disease is an infection with particular characteristics depending on the phase where the patient is and the level of severity that is present, Gonzales et al, establish that those patients with Chagas disease and who are asymptomatic require an ECG study, chest x-ray, echocardiogram and eventually, 24-hour Holter, this in order to prevent future pathologies at the cardiac level or any of the other forms of presentation of the acute phase, which can lead to complications for the patient and after that take him to death (10,11).

Perez et al. On the other hand, they establish that it is necessary to carry out a diagnosis supported by microbiology, clinics and epidemiology in those patients who have a high suspicion of Chagas disease, likewise it is necessary to rely on some other diagnostic aids to be able to determine the phase in which is found and the management to follow with these patients, likewise it should be noted that everything is directed to carry out a timely management of the present alterations and avoid possible complications that affect the quality of life of the patient and impact on the current mortality due to this disease and its complications (12,13).

Likewise, Salazar et al, determined recommendations for the surveillance and control of patients exposed to vectors, for the prevention of this infection in endemic areas and mainly those patients who have associated risk factors. Therefore, it is of the utmost importance to take measures to early detection of cases, focusing on the age of the child, with two purposes: to identify the active area of infection and to intervene to prevent the transmission vector of the disease, on the other hand, it reveals incurable diseases. Changes before the appearance, opportune treatment against parasites (14).

Rosas, in his article, indicates that the etiology of Chagas disease treated with bennidazole or nifurtimox in the acute phase, congenital infection, in cases of reactivation, and more recently in the chronic phase, has now been identified. Rapid diagnosis and etiological treatment of acute or reactivated cases can alter the course of the disease, likewise The Benznidazole Interventional Trypanosomiasis Evaluation Project (BENEFIT) is currently conducting a scientific evaluation of the established etiological treatment of Chagas disease in the chronic stage. The study included patients with chronic cardiomyopathy due to Chagas disease from Argentina, Bolivia, Brazil, El Salvador, and Colombia, who were randomized and double-blinded to receive benznidazole or placebo. The recruitment period for the study ended in September 2011, so patients are currently being followed, and it should be noted that in the near future there is the potential for an alternative treatment for Chagas disease. Ergosterol biosynthesis inhibitors, such as posaconazole, are currently used clinically for systemic mycoses, as they also show high activity against T. cruz (15).

5. Conclusion

Chagas disease should be suspected in patients with characteristic symptoms in those who are in endemic areas and with associated risk factor, likewise in its acute phase it has lower mortality, however in this phase it is necessary to suspect future complications, since It has high mortality in its chronic phase, especially in the cardiac form, where serious alterations suggestive of damage and myocardial injuries are shown that can affect the quality of life of patients and also lead to death. This is why, in addition to having suspicion in the cases described above, it is necessary to have diagnostic aids for a timely diagnosis. It should be noted that active intervention is especially important in endemic areas, such as rural areas with the aforementioned characteristics, without however, this should not be underestimated in other regions due to migration between countries and from urban to rural areas. The difficulty in controlling this pathology is due in part to the wide variety of clinical manifestations, which can be asymptomatic and promote disease

transmission, especially in populations with repeated exposure to infectious diseases. Similarly, early detection of infection is crucial in the treatment of Alzheimer's disease to ensure that the disease is adequately controlled and to prevent progression to severe disease.

Active intervention is especially important in endemic areas such as rural areas and host habitats with these characteristics. However, this should not be underestimated in other regions due to migration between countries and from urban to rural areas. The difficulty in controlling this pathology is due in part to the great variety of clinical manifestations that can be asymptomatic and facilitate transmission, especially in populations with high exposure to chloroquine from infectious diseases. Similarly, early detection of infection is crucial in treating the disease to ensure that the disease is properly controlled and to prevent the disease from progressing to a severe form, treatment of the disease is aimed at treating the various complications. accompanied with benzonidazole and nifurtimox, however, taking into account the possible side effects, they are not usually as beneficial for the patient, which is why maintaining prevention and expansion programs in sensitive populations can improve quality of life and prevent future deaths and Chagas complications.

Compliance with ethical standards

Disclosure of conflict of interest

The authors declare no conflict of interest.

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