Chagas Disease: Epidemiology, Diagnosis and Treatment in Mexico

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ABSTRACT

The article describes the etiology of Chagas disease, caused by the parasite *Trypanosoma cruzi*, and its transmission through vectors known as kissing bugs. The phases of the disease are detailed: acute (generally asymptomatic), indeterminate, and chronic. Serious complications such as Chagas cardiomyopathy, characterized by myocardial damage and fibrosis, and colonopathy are also analyzed. The article reviews direct, indirect, and molecular (PCR) diagnostic methods, as well as etiological treatment based on benznidazole and nifurtimox.

Chagas disease remains a significant public health challenge due to its high prevalence in vulnerable regions and its socioeconomic impact. The article concludes that strengthening epidemiological surveillance and ensuring access to timely diagnosis and treatment are essential to prevent fatal complications and improve the quality of life of those affected.

The objective of this research is to collect and analyze up-to-date information on Chagas disease, with a special focus on its epidemiology in Mexico and Latin America, the challenges of diagnosis and the management of its clinical complications.

Keywords: Chagas disease, *Trypanosoma cruzi*, public health.

INTRODUCTION

Chagas disease (CD), also called American trypanosomiasis, is a tropical disease caused by the flagellated protozoan *Trypanosoma cruzi* [1].

Chagas disease is the third most common parasitic disease worldwide, after malaria and schistosomiasis; it is also considered the most important parasitic disease in the Western world, with an estimated disease burden (disability-adjusted life years) 7.5 times greater than that of malaria [2].

It affects between 6 and 8 million people worldwide and causes approximately 50,000 deaths per year. An additional 65 to 100 million people live in areas at risk of infection worldwide.

Although more than a century has passed since its discovery, CD remains one of the main public health problems in most Latin American countries. In recent decades, celiac disease has also become a concern in non-endemic areas such as Canada, the United States, Europe, Australia, and Japan due to the constant migration of individuals from endemic regions (3).

As mentioned previously, celiac disease is a global problem that affects people daily in both endemic and non-endemic countries, primarily due to the high rate of migration to these territories. Therefore, it presents a challenge for global public health institutions to develop and implement timely diagnostic measures, since it not only affects individuals but also impacts socioeconomic levels, where the ability to generate income for subsistence becomes a greater challenge.

Therefore, according to the guidelines established by the Ministry of Health, it is necessary to make an accurate diagnosis, confirming the presence of *T. cruzi* through direct or indirect parasitological methods. Furthermore, patients should receive pre- and post-diagnosis counseling regarding the disease and their right to care [1].

The objective of this research is to gather information on Chagas disease, focusing on its epidemiology in Mexico and Latin America, the challenges and limitations involved in diagnosis, the disease's progression to Chagas cardiomyopathy, and its treatment.

Using the available literature, this study aims to emphasize the epidemiology of Chagas disease to highlight its relevance in the Potosí region, given its association with a poor quality of life for those affected.

BACKGROUND

Chagas disease (CD) was named after its discoverer, Carlos Ribeiro Justiniano Chagas, who was born on a coffee plantation in Oliveira, Minas Gerais state, Brazil, on July 9, 1878. Chagas graduated with a medical degree in 1903 and was invited by Oswaldo Cruz to work as a physician at the Brazilian Ministry of Public Health and Hygiene, where he first implemented indoor vector control against malaria.

On February 14, 1909, Chagas examined a patient who would become the first case of CD described in the literature: a two-year-old girl, Berenice, who presented

with high fever, hepatosplenomegaly, facial edema, and the presence of the parasite in her blood. Berenice remained asymptomatic throughout her life and died at age 73 from other causes. She was included in several clinical studies of CD from age 55 to 71.

The history of Chagas disease can be divided into three important phases:

- **1909 to 1934** - Identification and recognition of the disease.
- **1935 to 1960** - Clinical characterization and definition of its endemic nature.
- **1961 to the present** - Challenges for the implementation of public health policies for the control of Chagas disease.

EPIDEMIOLOGY

Chagas disease is included in the list of neglected tropical diseases published annually by the World Health Organization (WHO) due to its prevalence in low socioeconomic populations living in tropical and subtropical regions with poor sanitation and in close contact with infectious vectors.

Most people at high risk of infection often face multiple barriers to proper evaluation, diagnosis, and treatment due to limited access to healthcare. According to estimates from the WHO's 2010 epidemiological update on Chagas disease in Latin America, more than five million people are infected with *T. cruzi* in 21 Latin American countries. Argentina, Brazil, and Mexico had the highest prevalence, followed by Bolivia and Colombia.

It is estimated that between 20% and 25% of people infected with Chagas disease have Chagas cardiomyopathy, representing almost two million people [4].

In a systematic review with meta-analysis conducted in Mexico by Arnal A *et al.,* an analysis of epidemiological survey data from 2006 to 2017, found an estimated national seroprevalence of *T. cruzi* of 3.38%, with more than 4.06 million cases in Mexico [1]. The prevalence of *T. cruzi* infection in the adult population was 6.5% in San Luis Potosí [5].

In San Luis Potosí, according to data from the State Health Secretariat, a total of 261 infections were reported between 2003 and 2012, with Health Jurisdictions V and VI (corresponding to the Huasteca region) reporting the majority of cases [6]. Of 734 participants in the town of Zacatipan, Tamazunchale, SLP, the seroprevalence was 6.3% (n=46) [7].

ETIOLOGICAL AGENT

Also known as American trypanosomiasis, Chagas disease is caused by the protozoan parasite *T. cruzi*, an intracellular hemoflagellate parasite belonging to the order *Kinetoplastida*, family *Trypanosomatidae*. It has the ability to infect any cell, primarily macrophages, fibroblasts, and epithelial cells.

During its life cycle, the parasite develops through three main forms: **amastigotes** (the proliferative form found within the host cells of vertebrates), **epimastigotes** (the proliferative form found in the intestines of invertebrate hosts), and **trypomastigotes** (the infectious form present in the host's serum).

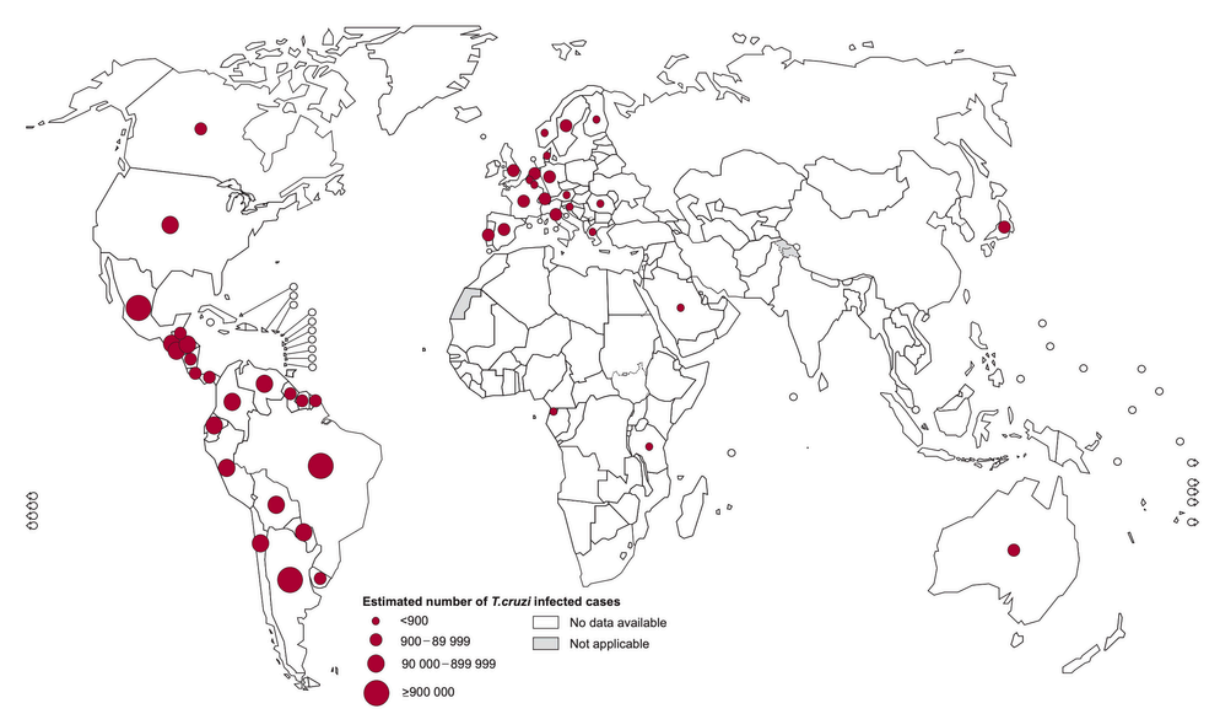


Figure 1. Global distribution of Chagas disease cases (2018). Taken from: World Health Organization. Global distribution of cases of Chagas disease, based on official estimates, 2018 [Internet]. Geneva: World Health Organization; 2018. Available from: https://cdn.who.int/media/docs/default-source/ntds/chagas-disease/chagas-2018-cases.pdf?sfvrsn=f4e94b3b_2.



Figure 2. *Trypanosoma cruzi* vector. Image from: https://www.cdc.gov/dpdx/trypanosomiasisamerican/index.html

Chagas disease is a vector-borne illness, with the parasite transmitted by hematophagous bugs, also known as kissing bugs (Figure 2) [3].

Trypomastigotes are found in feces near the bite wound or in intact mucosal tissues such as the conjunctiva. Within the host, trypomastigotes invade cells near the inoculation site, where they differentiate into intracellular amastigotes. These amastigotes multiply and differentiate again into trypomastigotes that are unable to replicate. Finally, these forms are released into the bloodstream and can infect cells in various tissues, transforming into intracellular amastigotes (Figure 3) [4].

PATHOGENESIS

The infection has two successive phases. The **acute phase** is characterized by high parasitemia, generally asymptomatic or oligosymptomatic, with fever, anorexia, and tachycardia. These manifestations resolve spontaneously in 90% of cases, and possibly between 60% and 70% of infected individuals will never develop signs or symptoms related to Chagas disease, which characterize the indeterminate form.

The remaining patients (30-40%) may progress to the **chronic phase** with neurological, cardiac, digestive (megacolon or megaesophagus), or cardiodigestive clinical symptoms. Chronic Chagas cardiomyopathy (CCC) is the most severe manifestation of the disease [3].

The complex host-parasite relationship that characterizes Chagas disease determines tissue damage and, therefore, myocardial involvement.

Local changes include the development of interstitial edema, lymphocytic infiltration, and reactive hyperplasia of the lymph nodes. The main sites of tissue involvement are muscle and ganglion cells, including the myocardium [4]. Infection of cardiac muscle cells, macrophages, neurons, and nervous support tissue (glial cells) can produce the most prominent lesions of this disease. Following transmission of the parasite by the vector, the incubation period is 1 to 2 weeks. After this period, an acute phase occurs, followed by a chronic phase [2].

From the time of infection by the parasite through inoculation by the triatomine bug, three phases in the development of the pathology are considered, according to authors and international organizations. Based on detections in patients under close monitoring, these phases are divided as follows:

- **Acute Phase**

Most cases in the acute phase are asymptomatic. At this stage, the invasive trypomastigote form can be detected in peripheral blood (parasitemia peaks 30 days after infection). Signs and symptoms of the vector-borne acute form appear after an incubation period of 7 to 30 days.

Occasionally, a specific edematous skin lesion (called a chagoma) may be observed at the entry site, in areas of thin skin. In 1934, Romaña and Mazza described their eponymous pathognomonic sign of unilateral, non-pitting eyelid edema with conjunctivitis and generalized facial edema (Figure 4).

The edema, secondary to lymphatic vessel obstruction, is the primary lesion, where the replicative form of the parasite, the amastigote, multiplies and progresses to invade the reticuloendothelial system. It may also present with hepatosplenomegaly, lymphadenopathy, and chagoma (a local inflammatory process) [2]. General and nonspecific symptoms of the acute phase include tachycardia, anorexia, malaise, vomiting, myalgia, and headache [8].

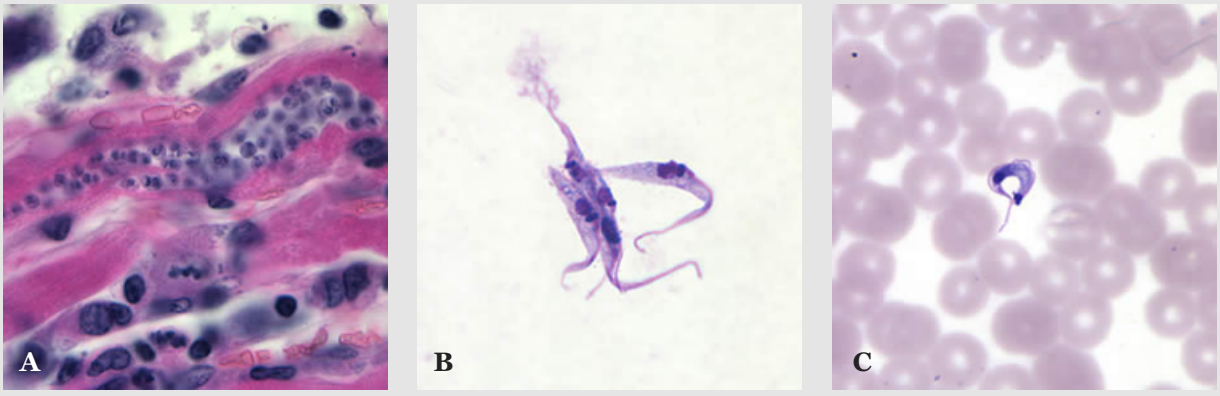


Figure 3. Main forms of *T. cruzi*. A) Amastigotes in cardiac tissue; hematoxylin and eosin (H&E) stain. B) Epimastigotes in culture. C) Trypomastigote in blood smear; Giemsa stain. Images from: https://www.cdc.gov/dpdx/trypanosomiasisamerican/index.html



Figure 4. Romaña's sign. Taken from: Beucler N, Torrico F, Hibbert D. A tribute to Cecilio Romaña: Romaña's sign in Chagas disease. PLoS Negl Trop Dis. 2020 Nov 12;14(11):e0008836. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7660534/

- **Indeterminate Phase**

After 4 to 8 weeks of the acute phase, the cell-mediated immune response controls replication, symptoms subside, and the disease enters the indeterminate phase. In this phase, there are no symptoms and no electrocardiographic abnormalities. 70-80% of the infected population remains in this phase for the rest of their lives [2].

- **Chronic Phase**

If left untreated, the acute phase is followed in 70% of cases by an indeterminate phase (asymptomatic infection) that can last a lifetime. The remaining 30% progress, within 10 to 30 years, to a chronic symptomatic form that affects the heart (20-30% of infections), the gastrointestinal tract (15-20%), and, more rarely, the peripheral nervous system (sensory polyneuropathy, less than 5%) [8].

COMPLICATIONS

- **Chagasic Cardiomyopathy**

Cardiac involvement is the most severe manifestation of chronic Chagas disease. It appears years or decades after the initial infection. It is a progressive disease characterized by a continuous inflammatory immune response that leads to myocardial damage and fibrosis [2].

Unlike other causes, Chagasic cardiomyopathy is characterized by its fibrosing nature, with alterations that begin in the posterobasal, posteroinferior, or apical region of the left ventricle, where a ventricular aneurysm typically appears. It affects the sinoatrial node and the atrioventricular node and can progress to a global condition. It has a high thrombogenic and arrhythmogenic predisposition, causing atrial fibrillation, extreme bradycardia, and ventricular tachycardia, which is the leading cause of death in this group of patients [9].

When infection and lysis of myocardial cells occur, an immune response against *T. cruzi* is triggered through the activation of phagocytic and cytotoxic cells, as well as the production of Th1-type cytokines. This response protects the host during the acute phase; however, due to chronicity, myocardial tissue damage is associated with an exacerbated inflammatory response, primarily mediated by T lymphocytes. Myocyte hypertrophy with abundant fibrosis occurs, leading to ventricular remodeling with bilateral ventricular dilation.

Thinning of the ventricular walls is common, as are apical aneurysms, aneurysms of the posterior wall of the left ventricle, and the development of mural thrombi. The combination of right bundle branch block and left anterior fascicular block is one of the earliest signs and is present in 25% of patients. Periganglionic inflammation of neurons of the parasympathetic nervous system occurs, leading to autonomic dysfunction. Chagas disease can present with various clinical manifestations in the heart, ranging from asymptomatic forms to severe cardiac disease, such as heart failure (the most frequent clinical presentation) and sudden death [2].

- **Chagasic colonopathy**

It presents as megacolon and, in later stages, as enlargement of the organ (megadolicocolon). Clinically, progressive constipation is characteristic, with periods of abdominal distension and flatulence. Diagnosis is based on plain abdominal radiography or a barium enema with contrast medium. Complications are generally due to intestinal obstruction, and treatment is surgical [9].

DIAGNOSIS

Diagnosing a patient with Chagas disease is a complex process that encompasses epidemiological criteria based on the prevalence and incidence of confirmed cases of the disease in the areas of residence of those seeking medical services. Clinical criteria, including the patient's symptoms, form the basis for suspecting the possible condition. Finally, to confirm the case, laboratory tests are used, resulting in a positive or negative diagnosis.

Laboratory diagnosis of *T. cruzi* infection [9] can be performed by:

- **Direct methods**, which verify the presence of the parasite in the sample.
  - Microscopic observation of fresh peripheral blood.
  - Thick blood smear.
  - Microstrout concentration method.

- **Molecular methods**, which verify the presence of the parasite's genetic material in the sample.
  - Qualitative PCR is the confirmatory test in immunocompromised individuals and children under 9 months of age.
  - Quantitative PCR allows for the measurement of parasite load and is especially useful in immunocompromised patients and organ donors. It also allows for post-treatment monitoring.

- **Indirect methods** detect the presence of specific antibodies against the parasite in the sample.
  - Indirect hemagglutination.
  - Enzyme-linked immunosorbent assay (ELISA).
  - Indirect immunofluorescence.
  - Western blot (Immunoelectrotransfer).

TREATMENT

Etiological treatment is divided into two established objectives [9] (Table 1).

- **Primary objective:** to eliminate the parasite and contribute to reducing the likelihood of developing clinical manifestations of the disease and its complications.
- **Secondary objective:** to contribute to interrupting the chain of transmission of *T. cruzi*.

If treatment has been interrupted for a period of less than 14 days, it will be continued for the same number of days until the 60 days of medication administration are completed. If the interruption is longer than 14 days, it will be necessary to restart treatment [9].

CONCLUSION

In conclusion, Chagas disease remains a significant public health challenge worldwide, especially in endemic regions of Latin America, but also in non-endemic areas due to migration. American trypanosomiasis, caused by *T. cruzi*, affects millions of people, with a considerably high disease burden that impacts not only individuals but also the socioeconomic sphere.

The disease, which can remain asymptomatic in most infected individuals, progresses in a significant percentage of patients to chronic stages with severe manifestations, such as Chagas cardiomyopathy, which is the leading cause of mortality in these cases.

This underscores the importance of timely diagnosis and appropriate treatment, which aims not only to eliminate the parasite but also to prevent severe complications and reduce transmission.

Despite advances in its understanding and control, Chagas disease remains a neglected disease, with significant barriers to its proper management, especially in vulnerable populations. The need for effective and accessible health policies, as well as the implementation of epidemiological surveillance, diagnosis and treatment programs, are essential to reduce the burden of this disease and improve the quality of life of those affected.

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Conflict of interest

The author declares no conflicts of interest.

Ethical considerations

This work did not involve the participation of human subjects or animals, nor the collection of primary data; therefore, it did not require approval from a research ethics committee. All data used came from previously published and publicly accessible secondary sources.

Table 1. Dosage Schedule.

Population	Benzonidazole	Nifurtimox	Comments
Pediatrics	10 mg/kg/day, orally, in two or three divided doses for 60 days. Do not exceed the maximum daily dose of 300 mg.		When the daily dose exceeds 300 mg, it is recommended to extend the treatment period until the total calculated dose for 60 days is reached.
Adults	5-10 mg/kg/day in two divided doses after meals for 60 days, not exceeding 300 mg as the maximum daily dose.	10 mg/kg/day in patients weighing less than 40 kg and 8 mg/kg/day in patients weighing more than 40 kg. Administer in three divided doses after meals for 60 days, not exceeding the maximum dose of 700 mg.	

Original design, with data obtained from Gob.mx. (2019). PROCEDURES MANUAL FOR CHAGAS DISEASE IN MEXICO.

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