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A Review on American trypanosomiasis the "Chagas Disease"

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Abstract: Chagas disease, also known as American trypanosomiasis, is a tropical parasitic disease caused by Trypanosoma cruzi. T. cruzi is usually spread to humans and other mammals by the bite of a conenose . The disease was first described in 1909 by Brazilian physician Carlos Chagas, after whom it is named Chagas disease is classified as a neglected tropical disease. In Argentina, the disease is known as mal de Chagas-Mazza in the honor of Salvador Mazza. Over decades with chronic Chagas disease, 30–40% of people develop organ dysfunction (determinate chronic Chagas disease), which most often affects the heart or digestive system. Symptoms vary widely based on the size and location of brain abscesses, but typically include fever, headaches, seizures, loss of sensation, or other neurological issues that indicate particular sites of nervous system damage. Occasionally, these individuals also experience acute heart inflammation, skin lesions, and disease of the stomach, intestine, or peritoneum. Elimination of T. cruzi doesn't cure the cardiac and gastrointestinal damage caused by chronic Chagas disease, so these conditions must be treated separately. To manage irregular heartbeats, people could also be prescribed anti-arrhythmic drugs like amiodarone, or have a pacemaker implanted. It was estimated that 6.2 million people, mostly in Mexico, Central America and South America, have Chagas disease as of 2017, resulting in an estimated 7,900 deaths. As of 2019, a vaccine has not been developed.

Keywords: Trypanosoma cruzi , neglected tropical disease , mal de Chagas-Mazza , organ dysfunction , peritoneum , gastrointestinal damage , amiodarone

INTRODUCTION:

Chagas disease, also known as American trypanosomiasis, is a tropical parasitic disease caused by Trypanosoma cruzi[1]. It is spread mostly by insects referred to as Triatominae, or "kissing bugs"[1]. The symptoms change over the course of the infection. In the early stage, symptoms are typically either not present or mild, and may include fever, swollen lymph nodes, headaches, or swelling at the site of the bite[1]. After four to eight weeks, untreated individuals enter the chronic phase of disease, which in most cases does not result in further symptoms[2][4]. Up to 45% of people with chronic infection develop heart disease 10–30 years after the initial illness, which can lead to heart failure[2]. Digestive complications, including an enlarged esophagus or an enlarged colon, can also occur in up to 21% of individuals, and up to 10% of individuals may experience nerve damage[2].

T. cruzi is commonly spread to humans and other mammals by the bite of a kissing bug[3]. The disease can also be spread through transfusion, organ transplantation, eating food contaminated with the parasites, and vertical transmission (from a mother to her baby)[1]. Diagnosis of early disease is by finding the parasite within the blood employing a microscope or detecting its DNA by polymerase chain reaction[4]. Chronic disease is diagnosed by finding antibodies for T. cruzi in the blood[5]. It affects more than 150 types of animals[6].

Prevention focuses on eliminating kissing bugs and avoiding their bites [1]. This may involve the use of insecticides or bed-nets[7]. Other preventive efforts include screening blood used for transfusions[1]. As of 2019, a vaccine has not been developed[1]. Early infections are treatable with the medications benznidazole or nifurtimox, which usually cure the disease if given shortly after the person is infected, but subsided effective the longer a person has had Chagas disease[1]. When used in chronic disease, medication may delay or prevent the development of end–stage symptoms[1]. Benznidazole and nifurtimox often cause side effects, including skin disorders, gastrointestinal system irritation, and neurological symptoms, which may end in treatment being discontinued[1][2]. As of 2019, new drugs for Chagas disease are under development, and experimental vaccines have been studied in animal models.

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It is estimated that 6.2 million people, mostly in Mexico, Central America and South America, have Chagas disease as of 2017[1], resulting in an estimated 7,900 deaths. Most people with the disease are poor[6], and most do not realize they are infected[8]. Large-scale population migrations have carried Chagas disease to new regions, which now include the us and lots of European countries[1].

The disease was first described in 1909 by Brazilian physician Carlos Chagas, after whom it's named[1]. Chagas disease is classified as a neglected tropical disease[9].

HISTORY:

The formal description of Chagas disease was made by Carlos Chagas in 1909 after examining a two-year-old girl with fever, swollen lymph nodes, and an enlarged spleen and liver[10]. Upon examination of her blood, Chagas saw trypanosomes identical to those he had recently identified from the hindgut of triatomine bugs and named Trypanosoma cruzi in honor of his mentor, Brazilian physician Oswaldo Cruz[10]. He sent infected triatomine bugs to Cruz in Rio de Janeiro, who showed the bite of the infected triatomine could transmit T. cruzi to marmoset monkeys as well[10]. In just two years, 1908 and 1909, Chagas published descriptions of the disease, the organism that caused it, and the insect vector required for infection[11][12]. Almost immediately thereafter, at the suggestion of Miguel Couto, then professor of the Faculdade de Medicina do Rio de Janeiro , the disease was widely referred to as "Chagas disease"[11]. Chagas discovery brought him national and international renown, but in highlighting the inadequacies of the Brazilian government's response to the disease, Chagas attracted criticism to himself and to the disease that bore his name, stifling research on his discovery and likely frustrating his nomination for the Nobel Prize in 1921[11] [13].

In the 1930s, Salvador Mazza rekindled Chagas disease research, describing over a thousand cases in Argentina's Chaco Province[10]. In Argentina, the disease is known as mal de Chagas-Mazza in his honor[14]. Serological tests for Chagas disease were introduced within the 1940s, demonstrating that infection with T. cruzi was widespread across Latin America[10]. This, combined with successes eliminating the malaria vector through insecticide use, spurred the creation of public health campaigns focused on treating houses with insecticides to eradicate triatomine bugs[10]. The 1950s saw the discovery that treating blood with crystal violet could eradicate the parasite, leading to its widespread use in transfusion screening programs in Latin America[10]. Large-scale control programs began to take form in the 1960s, first in São Paulo, then various locations in Argentina, then national-level programs across Latin America[15]. These programs received a major boost in the 1980s with the introduction of pyrethroid insecticides, which did not leave stains or odors after application and were longer-lasting and more cost-effective[10][15]. Regional bodies dedicated to controlling Chagas disease arose through support of the Pan American Health Organization, with the Initiative of the Southern Cone for the Elimination of Chagas Diseases launching in 1991, followed by the Initiative of the Andean countries (1997), Initiative of the Central American countries (1997), and the Initiative of the Amazon countries (2004) [16].

SIGNS AND SYMPTOMS:

Figure 1: An acute Chagas disease infection with swelling of the right eye

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Chagas disease occurs in two stages: an acute stage, which develops one to 2 weeks after the sting , and a chronic stage, which develops over many years[17]. The acute stage is often symptom-free[2]. When present, the symptoms are typically minor and not specific to any particular disease[4]. Signs and symptoms include fever, malaise, headache, and enlargement of the liver, spleen, and lymph nodes[1][2][4]. Rarely, people develop a swollen nodule at the location of infection, which is named "Romaña's sign" if it's on the eyelid, or a "chagoma" if it's elsewhere on the skin[18]. In rare cases (less than 1–5%), infected individuals develop severe acute disease, which can cause life-threatening fluid accumulation around the heart, or inflammation of the heart or brain and surrounding tissues[2]. The acute phase typically lasts four to eight weeks and resolves without treatment[2].

Unless treated with antiparasitic drugs, individuals remain chronically infected with T. cruzi after recovering from the acute phase[2]. Most chronic infections are asymptomatic, which is mentioned as indeterminate chronic Chagas disease[2]. However, over decades with chronic Chagas disease, 30–40% of people develop organ dysfunction (determinate chronic Chagas disease), which most often affects the heart or digestive system[2][4].

The most common manifestation is heart disease, which occurs in 14–45% of people with chronic Chagas disease[2]. People with Chagas heart disease often experience heart palpitations and sometimes fainting due to irregular heart function[19]. By electrocardiogram, people with Chagas heart disease most frequently have arrhythmias[19]. As the disease progresses, the heart's ventricles become enlarged (dilated cardiomyopathy), which reduces its ability to pump blood[19]. In many cases the first sign of Chagas heart disease is heart failure, thromboembolism, or chest pain associated with abnormalities in the microvasculature[19].

Also common in chronic Chagas disease is damage to the digestive system, particularly enlargement of the esophagus or colon, which affects 10–21% of people[2]. Those with enlarged esophagus often experience pain (odynophagia) or trouble swallowing (dysphagia), acid reflux, cough, and weight loss[2]. Individuals with enlarged colon often experience constipation, which may cause severe blockage of the intestine or its blood supply[2]. Up to 10% of chronically infected individuals develop nerve damage that can result in numbness and altered reflexes or movement[2]. While chronic disease typically develops over decades, some individuals with Chagas disease (less than 10%) reach heart damage directly after acute disease[19].

Signs and symptoms differ for people infected with T. cruzi through less common routes. People infected through ingestion of parasites tend to develop severe disease within three weeks of consumption, with symptoms including fever, vomiting, shortness of breath, cough, and pain within the chest, abdomen, and muscles[2]. Those infected congenitally typically have few to no symptoms, but can have mild non-specific symptoms, or severe symptoms such as jaundice, respiratory distress, and heart problems[2]. People infected through transplant or transfusion tend to possess symptoms almost like those of vector-borne disease, but the symptoms might not manifest for anywhere from every week to five months[2]. Chronically infected individuals who become immunosuppressed due to HIV infection can suffer particularly severe and distinct disease, most commonly characterized by inflammation in the brain and surrounding tissue or brain abscesses[4]. Symptoms vary widely supported the dimensions and site of brain abscesses, but typically include fever, headaches, seizures, loss of sensation, or other neurological issues that indicate particular sites of nervous system damage[20].Occasionally, these individuals also experience acute heart inflammation, skin lesions, and disease of the stomach, intestine, or peritoneum[4].

CAUSE:

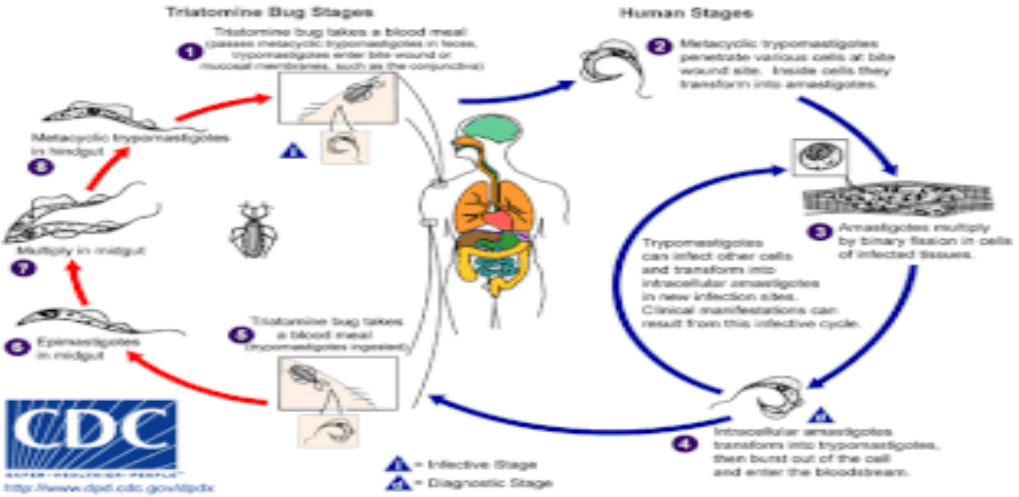

Figure 2: Life cycle and transmission of *T. cruzi*

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Chagas disease is caused by infection with the protozoan parasite T. cruzi, which is usually introduced into humans through the bite of triatomine bugs, also called "kissing bugs"[4]. At the bite site, motile T. cruzi forms called trypomastigotes invade various host cells[3]. Inside a host cell, the parasite transforms into a replicative form called an amastigote, which undergoes several rounds of replication[3]. The replicated amastigotes transform back to trypomastigotes, which burst the host cell and are released into the bloodstream[2]. Trypomastigotes then disseminate throughout the body to various tissues, where they invade cells and replicate[2]. Over a few years , cycles of parasite replication and immune reaction can severely damage these tissues, particularly the guts and digestive tract[2].

TRANSMISSION:

Figure 3: *Triatoma infestans*, a common vector of *T. cruzi*

T. cruzi are often transmitted by various triatomine bugs within the genera Triatoma, Panstrongylus, and Rhodnius[2]. The primary vectors for human infection are the species of triatomine bugs that inhibit human dwellings, namely Triatoma infestans , Rhodnius prolixus , Triatoma dimidiata and Panstrongylus megistus[21]. These insects are known by variety of local names, including vinchuca in Argentina, Bolivia, Chile and Paraguay, barbeiro (the barber) in Brazil, pito in Colombia, chinche in Central America, and chipo in Venezuela[22]. The bugs tend to feed at night, preferring moist surfaces near the eyes or mouth[17][21]. A triatomine bug can become infected with T. cruzi when it feeds on an infected host[17]. T. cruzi replicates in the insect's intestinal tract and is shed in the bug's feces[17]. When an infected triatomine feeds, it pierces the skin and takes in a blood meal, defecating at the same time to make room for the new meal[17]. The bite is typically painless, but causes itching[17]. Scratching at the bite introduces the T. cruzi-laden feces into the bite wound, initiating infection[17].

In addition to classical vector spread, Chagas disease can be transmitted through food or drink contaminated with triatomine insects or their feces[23]. Since heating or drying kills the parasites, drinks and especially fruit juices are the most frequent source of infection[23]. This route of transmission has been implicated in several outbreaks, where it led to unusually severe symptoms, likely thanks to infection with a better parasite load than from the bite of a triatomine bug[5][23].

T. cruzi also can be transmitted independent of the triatomine bug during transfusion , following organ transplantation, or across the placenta during pregnancy[2]. Transfusion with the blood of an infected donor infects the recipient 10–25% of the time[2]. To prevent this, blood donations are screened for T. cruzi in many countries with endemic Chagas disease, as well as the United States[5]. Similarly, transplantation of solid organs from an infected donor can transmit T. cruzi to the recipient[2]. This is very true for heart transplant, which transmits T.

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cruzi 75–100% of the time, and less so for transplantation of the liver (0–29%) or a kidney (0–19%)[2]. An infected mother can also pass T. cruzi to her child through the placenta; this occurs in up to 15% of births by infected mothers[24].

PATHOPHYSIOLOGY:

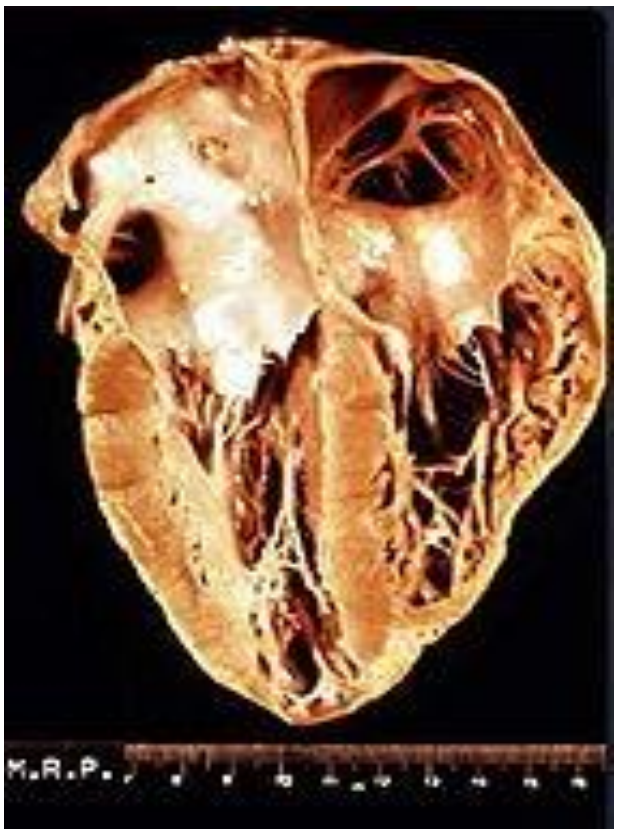

Figure 4: Large scale anatomy of a heart damaged by chronic Chagas disease

In the acute phase of the disease, signs and symptoms are caused directly by the replication of T. cruzi and the immune system's response to it[2]. During this phase, T. cruzi can be found in various tissues throughout the body and circulating in the blood[2]. During the initial weeks of infection, parasite replication is brought under control by production of antibodies and activation of the host's inflammatory response, particularly cells that target intracellular pathogens such as NK cells and macrophages, driven by inflammation-signaling molecules like TNF-α and IFN-γ[2].

During chronic Chagas disease, long-term organ damage develops over years thanks to continued replication of the parasite and damage from the system . Early in the course of the disease, T. cruzi is found frequently in the striated muscle fibers of the heart[26]. As disease progresses, the guts becomes generally enlarged, with substantial regions of heart muscle fiber replaced by connective tissue and fat[26]. Areas of active inflammation are scattered throughout the heart, with each housing inflammatory immune cells, typically macrophages and T cells[26]. Late in the disease, parasites are rarely detected in the heart, and may be present at only very low levels[26].

In the heart, colon, and esophagus, chronic disease also leads to a massive loss of nerve endings[19]. In the heart, this may contribute to arrythmias and other cardiac dysfunction[19]. In the colon and esophagus, loss of systema nervosum control is that the major driver of organ dysfunction. Loss of nerves impairs the movement of food through the digestive tract, which can lead to blockage of the esophagus or colon and restriction of their blood supply[19].

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DIAGNOSIS:

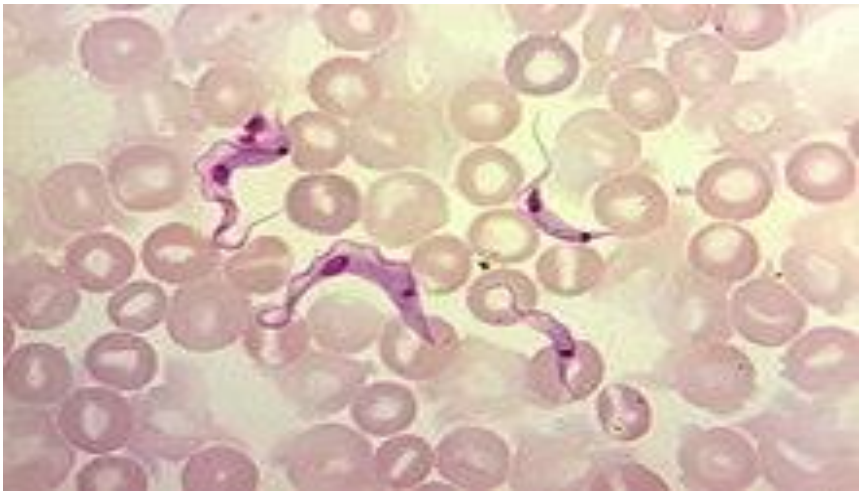

Figure 5: *T. cruzi* trypomastigotes seen in a blood smear

The presence of T. cruzi is diagnostic of Chagas disease. During the acute phase of infection, it can be detected by microscopic examination of fresh anti-coagulated blood, or its buffy coat, for motile parasites; or by preparation of thin and thick blood smears stained with Giemsa, for direct visualization of parasites[4][5]. Blood smear examination detects parasites in 34–85% of cases. Techniques such as microhematocrit centrifugation can be used to concentrate the blood, which makes the test more sensitive[2]. On microscopic examination, T. cruzi trypomastigotes have a slender body, often in the shape of an S or U, with a flagellum connected to the body by an undulating membrane[27].

Alternatively, T. cruzi DNA are often detected by polymerase chain reaction (PCR). In acute and congenital Chagas disease, PCR is more sensitive than microscopy[25], and it is more reliable than antibody-based tests for the diagnosis of congenital disease because it is not affected by transfer of antibodies against T. cruzi from a mother to her baby (passive immunity) [28]. PCR is also used to monitor T. cruzi levels in organ transplant recipients and immunosuppressed people, which allows infection or reactivation to be detected at an early stage[2][4][25].

During the chronic phase, microscopic diagnosis is unreliable and PCR is less sensitive because the level of parasites in the blood is low[2]. Chronic Chagas disease is typically diagnosed using serological tests, which detect immunoglobulin G antibodies against T. cruzi in the person's blood[5]. The most common test methodologies are ELISA, indirect immunofluorescence, and indirect hemagglutination[29]. Two positive serology results, using different test methods, are required to confirm the diagnosis[4]. If the test results are inconclusive, additional testing methods like Western blot are often used[2]. T. cruzi antigens may also be detected in tissue samples using immunohistochemistry techniques[5].

Various rapid diagnostic tests for Chagas disease are available. These tests are easily transported and can be performed by people without special training[30]. They are useful for screening large numbers of individuals and testing people that cannot access healthcare facilities, but their sensitivity is comparatively low[2], and it's recommended that a second method is used to confirm a positive result[30][31].

T. cruzi are often isolated from samples through blood culture or xenodiagnosis, or by inoculating animals with the person's blood. In the blood culture method, the person's red blood cells are separated from the plasma and added to a specialized growth medium to encourage multiplication of the parasite. It can take up to 6 months to get the result. Xenodiagnosis involves feeding the person's blood to triatomine insects, then examining their feces for the parasite 30 to 60 days later[30]. These methods are not routinely used, as they are slow and have low sensitivity[29] [30].

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PREVENTION:

Figure 6: Bed nets can be used in endemic areas to prevent bites from triatomine bugs

Efforts to stop Chagas disease have largely focused on vector control to limit exposure to triatomine bugs. Insecticide-spraying programs have been the mainstay of vector control, consisting of spraying homes and the surrounding areas with residual insecticides[16]. This was originally done with organochlorine, organophosphate, and carbamate insecticides, which were supplanted in the 1980s with pyrethroids[16]. These programs have drastically reduced transmission in Brazil and Chile[17], and eliminated major vectors from certain regions: Triatoma infestans from Brazil, Chile, Uruguay, and parts of Peru and Paraguay, as well as Rhodnius prolixus from Central America[19]. Vector control in some regions has been hindered by the development of insecticide resistance among triatomine bugs[16]. In response, vector control programs have implemented alternative insecticides (e.g. fenitrothion and bendiocarb in Argentina and Bolivia), treatment of domesticated animals (which also are ate up by triatomine bugs) with pesticides, pesticide-impregnated paints, and other experimental approaches. In areas with triatomine bugs, transmission of T. cruzi can be prevented by sleeping under bed nets and by housing improvements that prevent triatomine bugs from colonizing houses[17].

Blood transfusion was formerly the second-most common mode of transmission for Chagas disease[32]. T. cruzi can survive in refrigerated stored blood, and can survive freezing and thawing, allowing it to persist in whole blood, packed red blood cells, granulocytes, cryoprecipitate, and platelets[32]. The development and implementation of blood bank screening tests has dramatically reduced the risk of infection during blood transfusion[32]. Nearly all blood donations in Latin American countries undergo Chagas screening[32]. Widespread screening is additionally common in non-endemic nations with significant populations of immigrants from endemic areas including the uk (implemented in 1999), Spain (2005), the us (2007), France and Sweden (2009), Switzerland (2012), and Belgium (2013) [33]. Blood is tested using serological tests, typically ELISAs, to detect antibodies against T. cruzi proteins[32].

Other modes of transmission have also been targeted by Chagas disease prevention programs. Treating T. cruzi-infected mothers during pregnancy reduces the risk of congenital transmission of the infection[24]. To this end, many countries in Latin America have implemented routine screening of pregnant women and infants for T. cruzi infection, and the World Health Organization recommends screening all children born to infected mothers to prevent congenital infection from developing into chronic disease[1][34]. Similarly to blood transfusions, many countries with endemic Chagas disease screen organs for transplantation with serological tests[2].

There is no vaccine against Chagas disease[1]. Several experimental vaccines have been tested in animals infected with T. cruzi and were ready to reduce parasite numbers within the blood and heart[35], but no vaccine candidates had undergone clinical trials in humans as of 2016[36].

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MANAGEMENT:

Chagas disease is managed using antiparasitic drugs to eliminate T. cruzi from the body and symptomatic treatment to deal with the consequences of the infection[5]. As of 2018, benznidazole and nifurtimox were the antiparasitic drugs of choice for treating Chagas disease[2], though benznidazole is the only drug available in most of Latin America[37]. For either drug, treatment typically consists of two to three oral doses per day for 60 to 90 days[2]. Antiparasitic treatment is best early within the course of infection: it eliminates T. cruzi from 50 to 80% of people in the acute phase, but only 20–60% of those in the chronic phase[5]. Treatment of chronic disease is more effective in children than in adults, and the cure rate for congenital disease approaches 100% if treated in the first year of life[2]. Antiparasitic treatment also can slow the progression of the disease and reduce the likelihood of congenital transmission[1]. Elimination of T. cruzi doesn't cure the cardiac and gastrointestinal damage caused by chronic Chagas disease, so these conditions must be treated separately[5]. Antiparasitic treatment is not recommended for people who have already developed dilated cardiomyopathy[19].

Benznidazole is usually considered the first-line treatment because it has milder adverse effects than nifurtimox and its efficacy is better understood[2][25]. Both benznidazole and nifurtimox have common side effects that can result in treatment being discontinued. The most common side effects of benznidazole are rash , digestive problems, decreased appetite, weakness, headache, and sleeping problems. These side effects can sometimes be treated with antihistamines or corticosteroids, and are generally reversed when treatment is stopped[2]. However, benzidazole is discontinued in up to 29% of cases[2]. Nifurtimox has more frequent side effects, affecting up to 97.5% of individuals taking the drug. The most common side effects are loss of appetite, weight loss, nausea and vomiting, and various neurological disorders including mood changes, insomnia, paresthesia and peripheral neuropathy[2]. Treatment is discontinued in up to 75% of cases[2][25]. Both drugs are contraindicated for use in pregnant women and people with liver or kidney failure[1]. As of 2019, resistance to these drugs has been reported[37].

COMPLICATIONS:

In the chronic stage, treatment involves managing the clinical manifestations of the disease. The treatment of Chagas cardiomyopathy is similar to that of other forms of heart disease[2]. Beta blockers and ACE inhibitors may be prescribed, but some people with Chagas disease may not be able to take the standard dose of these drugs because they have low blood pressure or a low heart rate[2][19]. To manage irregular heartbeats, people may be prescribed anti-arrhythmic drugs such as amiodarone, or have a pacemaker implanted[4]. Blood thinners may be used to prevent thromboembolism and stroke[19]. Chronic heart disease caused by Chagas is a common reason for heart transplantation surgery[17]. Because transplant recipients take immunosuppressive drugs to stop organ rejection, they're monitored using PCR to detect reactivation of the disease. People with Chagas disease who undergo heart transplantation have higher survival rates than the average heart transplant recipient[19].

Mild gastrointestinal disease can be treated symptomatically, such as by using laxatives for constipation, or taking a prokinetic drug like metoclopramide before meals to relieve esophageal symptoms[4][38]. Surgery to sever the muscles of the lower esophageal sphincter (cardiomyotomy) is indicated in more severe cases of esophageal disease[38], and surgical removal of the affected part of the organ may be required for advanced megacolon and megaesophagus[4][29].

EPIDEMIOLOGY & RISK FACTORS:

Chagas disease, or American trypanosomiasis, is caused by the parasite Trypanosoma cruzi. Infection is most ordinarily spread through contact with the poop of an infected triatomine bug (or “kissing bug”), a blood-sucking insect that feeds on humans and animals.

More on: Triatomine Bugs

Infection can also occur from:

- Mother-to-baby (congenital),
- Contaminated blood products (transfusions),
- An organ transplanted from an infected donor,
- Laboratory accident (rare), or
- Contaminated food or drink (rare).

Chagas disease is common in parts of Mexico, Central America, and South America where an estimated 8 million people are infected. The triatomine bug thrives in poor housing conditions (for example, mud walls, thatched

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roofs), and in countries where the bug is present, people living in rural areas are at greatest risk for getting infected. Efforts by the general public health community to stop transmission of T. cruzi have resulted during a decrease within the number of latest T. cruzi infections and, in some areas, have completely stopped transmission of the parasite by triatomine bugs. Many countries where Chagas disease is common have also started screening donated blood for this disease. However, new cases of Chagas disease transmitted through infected organs, or by mother-to-child (congenital) transmission can still occur.

CDC estimates External that quite 300,000 persons with Trypanosoma cruzi infection sleep in the us . Most people with Chagas disease in the United States were infected in the parts of Latin America where Chagas disease is found. Although there are triatomine bugs within the us , only a couple of cases of Chagas disease from contact with the bugs are documented during this country.

Ref : https:www.cdc.gov

CONCLUSIONS:

As of 2018, standard diagnostic tests for Chagas disease were limited in their ability to measure the response to antiparasitic treatment. Serological tests, for example, may remain positive for years after T. cruzi is eliminated from the body, and PCR may give false-negative results when parasitemia is low. Various potential biomarkers of treatment response are under investigation, like immunoassays against specific T. cruzi antigens, flow cytometry testing to detect antibodies against different life stages of T. cruzi, and markers of physiological changes caused by the parasite, like alterations in coagulation and lipid metabolism.

Another research area is that the use of biomarkers to predict the progression of chronic Chagas disease. Serum levels of tumor necrosis factor alpha, brain and atrial natriuretic peptide, and angiotensin-converting enzyme 2, markers of heart damage and inflammation, are found to correlate with the severity of Chagas cardiomyopathy. Endothelin 1 has been studied as a prognostic marker in animal model.

T. cruzi shed acute-phase antigen (SAPA), which can be detected in blood using ELISA or Western blot has been used as an indicator of early acute and congenital infection. A novel assay for T. cruzi antigens in urine has been developed to diagnose congenital disease.

A number of experimental vaccines have been tested in animals. Some approaches have used inoculation with dead or attenuated T. cruzi parasites or non-pathogenic organisms that share antigens with T. cruzi, such as Trypanosoma rangeli or Phytomonas serpens. DNA vaccination has also been explored. As of 2019, vaccine research has mainly been limited to small animal models, and further testing in large animals is required.

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