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Bug Smash, Bug Splash: A Case Report of an Unusual Transmission of American Trypanosomiasis with a Brief Review of the Literature

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Conflict of interest: None declared

Patient: Male, 44
Final Diagnosis: Acute phase Chagas disease
Symptoms: Fever • headache • periorbital oedema
Medication: —
Clinical Procedure: —
Specialty: Infectious Diseases

Objective: Rare disease

Background: Chagas disease is a chronic parasitosis transmitted by the inoculation of infected triatomine feces into wounds or conjunctival sac, transfusion, congenitally, organ transplantation, and ingestion of contaminated food. The disease is classified into an acute and chronic phase; the latter is a life-long infection that can be asymptomatic or progress to cardiac or digestive complications.

Case Report: We report a case of acute-phase Chagas disease, transmitted by the splash of gut content from an infected triatomine into the conjunctival mucosa.

Conclusions: The diagnosis of Chagas disease is made by the direct visualization of the parasite in blood smears during the acute phase of the disease; during the chronic phase of the disease the diagnosis is made by the detection of IgG antibodies. Parasitological cure can be achieved in up to 80% of the cases in acute phase of the disease, in contrast with less than 30% during the chronic phase.

MeSH Keywords: Acute Disease • Chagas Disease • Trypanosoma Cruzi

Full-text PDF: http://www.amjcaserep.com/abstract/index/idArt/900539

Background

Chagas disease is a chronic parasitosis caused by the kinetoplastid protozoon *Trypanosoma cruzi*. Due to the worldwide burden of the disease, the WHO recognized it as one of the most important neglected tropical diseases [1,2]. Currently, there are no screening programs in endemic countries [2]. According to a 2010 WHO report, more than 25 million people are at risk of infection globally and in México it could potentially affect 5.5 million [3]. The global economic burden of Chagas disease is estimated to be at $7.19 billion per year and $188.80 billion per lifetime, similar to or higher than other prominent diseases such as cervical cancer [4]. In an effort to reduce vector transmission, several programs have been implemented during the last decades, such as the Southern Zone Initiative to Control/Eliminate Chagas disease, the Andean Pact Initiative, and the Central America Initiative. However, recent studies report resistance to pyrethroids, the insecticide most commonly used to eliminate the vectors, and this finding might negatively affect the progress of these programs [5]. México hosts one of the most diverse triatomine populations, with at least 21 species able to be infected with *T. cruzi*. The rate of infection in prone species has been reported to be as high as 50%. *Meccus pallidipennis* is the most commonly captured triatomine in the state of Morelos [3]. More than 180 domestic, synanthropic, and wild species of mammals, especially nest-building rodents and opossums, are likely to be infected with *T. cruzi* and to be involved in the transmission cycle of the disease. Dogs, cats, and rodents are important domestic reservoirs. Moreover, dogs and cats are very often observed to have a very high prevalence of *T. cruzi* infection [3].

The transmission of the disease occurs mainly by the vector (80–90%) when the feces of the infected triatomine bug are inoculated into a bite wound or through intact mucous membranes. Less frequent modes of transmission include transfusion (5–20%) and congenitally (0.5%). Rarely, the disease can be acquired by organ transplantation, laboratory accidents, and ingestion of food or liquids contaminated by *T. cruzi* [1,2,6]. The disease is classified into an acute and a chronic phase. The acute phase occurs 1–2 weeks after the infected bug bites the host; most patients present a mild, self-limited, nonspecific febrile illness and a sign of portal of entry, such as chagoma or Romaña's sign. The latter occurs when the parasite is inoculated into the conjunctival sac. Severe, acute, life-threatening infection can present with myocarditis, meningoencephalitis, or both; death occurs occasionally in the acute phase (<5–10% of symptomatic cases) [1]. The chronic phase can be an indeterminate form with a life-long asymptomatic infection, or progress to a determinate form with myocardiopathy, megadigestive syndromes, or the combination of both. The annual mortality rate in these patients varies widely across different studies (0.2–19.2%), in part due to the heterogeneity of the patient populations [7]. The acute phase of the disease is characterized by highly active parasite replication, and only during this phase can the parasite be detected by microscopy in blood smears, which makes the diagnosis of acute-phase infection possible. Due to the host immune response, the parasite is undetectable by blood smear microscopy during the chronic phase, making the detection of IgG antibodies against the parasite the best diagnostic test during this phase [1,8,9].

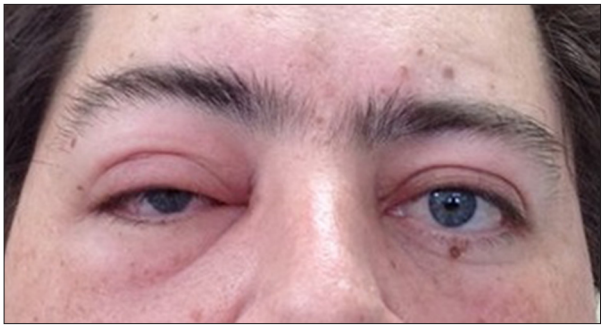

Figure 1. Romaña's sign.

Case Report

The patient was a 44-year-old man from Morelos, México, with medical records of systemic hypertension, obesity, and impaired fasting glucose. He had been well until approximately 1 month before admission, when he smashed a bug whose gut contents splashed onto his right eye. Twenty 20 days later he developed periorbital edema in his right eye, 39°C fever, and frontotemporal headache. He presented to his primary care physician, who diagnosed periorbital cellulitis on the basis of clinical presentation and prescribed antibiotic and anti-inflammatory treatment while the patient was ambulatory, without any clinical improvement. Upon admission to our hospital the patient looked ill, was diaphoretic, and had periorbital edema. The conjunctiva was hyperemic, photomotor and consensual reflexes were present, there were no palpable lymph nodes, and the other results of the physical examination were unremarkable. Because the patient lives in a place endemic for American trypanosomiasis, the periorbital edema was suspected to be a sign of portal of entry (Romaña's sign) (Figure 1). The patient was shown images of insects and recognized the insect he smashed as a triatomine bug, *M. pallidipennis* [10].

His laboratory studies reported leukocytes of 11 800 cel/μL, neutrophils 6372 cel/μL, lymphocytes 4720 cel/μL, Hb 12.7 g/dL, platelet count 218 000/μL, glucose 115 mg/dL, urea 40 mg/dL, creatinine 1.5 mg/dL, total bilirubin 0.7 mg/dL, aspartate aminotransferase 43 U/L, alanine aminotransferase 40 U/L, DHL 765 U/L, total proteins 6 mg/dL, and albumin 2.3 mg/dL. A 12-lead electrocardiogram showed sinus rhythm, frequency of 100', and QRS axis at 10°. Cardiac enzymes reported CPK 158 U/L

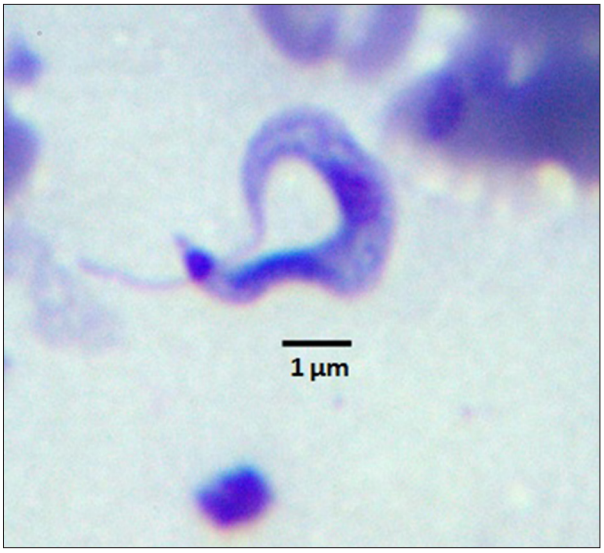

Figure 2. Wright-stained peripheral blood smear (1000×), showing *Trypanosoma cruzi* in trypomastigote stage. The __ is equivalent to 1 micrometer.

and CKMB 14.5 U/L. Due to the patient history, American trypanosomiasis was suspected and a blood smear was requested, which showed the presence of a flagellated parasite, *T. cruzi* (Figure 2). American trypanosomiasis in acute phase without myocarditis diagnosis was established. The patient began nifurtimox treatment at a 10 mg/kg/day dose for 60 days. He showed remarkable clinical improvement and was discharged home to complete treatment. Currently, the patient remains asymptomatic and is evaluated annually to monitor for any chronic complications of Chagas disease.

Discussion

Active parasite replication exists during the acute phase of the disease, which makes the detection of the parasite in blood smears possible. During the chronic phase, the immune system lowers the parasite burden to levels that are undetectable by blood-smear microscopy. However, *T. cruzi* amastigotes can be found in tissues, especially in cardiac and skeletal muscle [11]. The amplification of *T. cruzi* DNA using polymerase chain reaction is the leading test for assessing response to treatment in a short period of time, in contrast with microscopy, which lacks sensitivity, and blood cultures, which can take up to 30 days and have a higher rate of false-negative results [12,13]. Currently, there are no biomarkers to assess the therapeutic response in chronic Chagas disease [13]. The diagnosis of the acute phase of the disease is difficult due to the mild symptoms; it is diagnosed in less than 10% of patients who have this phase of Chagas disease [14].

It is recommended to treat all patients with acute, congenital, and reactivated infection, as well as infected children irrespective of the phase of the disease, patients younger than 18 years of age, and patients 19–50 years with indeterminate phase of the disease [1,8]. Nifurtimox and benznidazole are the only 2 therapeutic options that have been available in recent decades. Due to its safer profile and better tolerability, benznidazole is considered the first-line treatment and is given in doses of 5 mg/kg/day for 60 days. Nifurtimox is given in a dose of 8–10 mg/kg/day in 3 divided doses for 60–90 days [1,6,11]. In the acute phase of the disease, treatment reduces the duration and severity of the symptoms and achieves a parasitological cure in 60–85% of patients [11]. In the chronic phase of the disease, treatment can slow the development and progression of Chagas cardiomyopathy.

After about 20 years, 15–30% of patients will develop a chronic complication of Chagas disease; this prompts the search for cardiovascular and gastrointestinal symptoms and use of a resting 12-lead electrocardiogram (ECG) to define the clinical form of the disease. Patients with a normal ECG and with no gastrointestinal symptoms should be followed up every 12–24 months [1]. These patients have a better prognosis than patients with advanced heart disease, which indicates a poor prognosis [1].

Conclusions

Chagas disease continues to be an important public health problem worldwide. Efforts to reduce the rates of transmission have been unsuccessful due to insecticide resistance and the complexity of the distribution of the disease between wild and domestic reservoirs. Diagnosis during the acute phase of the disease is critical for patient prognosis, but the nonspecific clinical findings make this challenging. Chagas disease should be considered a differential diagnosis in patients living in endemic areas presenting with nonspecific febrile illness. We believe this is the first case report of Chagas disease transmitted by the splash of gut contents from an infected triatomine bug into the human conjunctival mucosa.

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Conflict of Interests

None.

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