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LETTER TO THE EDITOR

## Orally transmitted acute Chagas disease in domestic travelers in Colombia

Chagas disease still represents a significant threat in endemic, but also in non-endemic areas due to travel and migration [1,2]. In this setting, during last decade, oral transmission and its associated acute forms, have been recognized as significant concerns in endemic areas, where this would lead to severe life-threatening and even fatal consequences [3,4].

Colombia has been one of the Latin American countries with a considerable number of acute Chagas disease outbreaks where oral transmission of *Trypanosoma cruzi* has been evidenced [5]. Nevertheless, its diagnosis, in the setting of multiple endemic tropical infectious diseases, such as tuberculosis, malaria, dengue, chikungunya, Zika, among many other, even in coinfections [6,7], represents a real challenge. In those patients with history of travel to endemic areas of Chagas disease, there should be a high suspicion of this disease, which should be included in the differential diagnosis. Here, we report two cases, in which patients traveled to the department of Casanare (Fig. 1A), where between March and April 2014, an outbreak of oral Chagas disease was reported locally.

*Case 1*. A 33-year-old male, coming from Paz de Ariporo and Gaitán, Casanare, Colombia (Fig. 1), presenting fever, headache, arthralgia, myalgia, bilateral palpebral edema and exanthema with 20 days of evolution was received at our institution in Sincelejo, Sucre (Fig. 1A) (Clínica Santa María). At physical examination patient presented cervical lymphadenopathy, splitted first heart sound, with pericardial rub, the liver was enlarged, being palpable at 2 cm below the subcostal margin. Also presented a distended abdomen with skin rash. An X-ray and CT-scan revealed cardiomegaly and pericarditis. At the ECG an AV first degree block was evidenced. A blood smear revealed the presence of circulating trypomastigotes of *Trypanosoma cruzi*. In addition, an ELISA and indirect immunofluorescence were positive for anti-*T. cruzi* antibodies. A CBC showed a leukocyte count of 4000 cells/mL, with a C-reactive protein in 92 mg/L, an ESR 4 mm, platelets in 196,000 cells/mL. At the echocardiogram a pleural effusion of 1000 mL was evidenced, with an ejection fraction of 60% (Fig. 1B). Patient was treated with benznidazole plus spironolactone, enalapril and carvedidol. After 15 days, this patient progressively decreased its pericardial effusion until fully subsided at 30 days. Patient was discharged after 30 days.

*Case 2*. A 47-year-old male, coming from Paz de Ariporo and Gaitán, Casanare, Colombia (Fig. 1A), presenting fever, chills, sweating, headaches, myalgia, arthralgia, palpebral edema, chest pain, dyspnea, edema in lower members and skin rash. At physical examination was evidenced cervical lymphadenopathy, splitted second heart sound with pericardial rub. Lung basis crackles, with a palpable liver at 1 cm below the right subcostal border. Also, edema in lower members was found. An echocardiogram showed a left ventricular dilatation with an ejection fraction of 50%. At the chest X-ray cardiomegaly was evidenced. The ECG showed a prolonged PR in V1. A blood smear revealed the presence of circulating trypomastigotes of *T. cruzi*. In addition, an ELISA and indirect immunofluorescence were positive for anti-*T. cruzi* antibodies. A CBC showed a leukocyte count of 5000 cells/mL, with a C-reactive protein in 60 mg/L, an ESR 35 mm, platelets in 200,000 cells/mL. Patient was treated with benznidazole, 100 mg, q6 h, plus beta blockers, ACE inhibitors and aldosterone inhibitors. After 2 weeks, his clinical findings subsided. Patient was discharged after 3 weeks.

Acute Chagas disease secondary to oral transmission is associated with acute myocarditis and a high case fatality rate [1,3,4]. Oral transmission has been associated with consumption of contaminated food and beverages [8]. Inflammatory processes leading to acute myocarditis are related to multiple factors, including sustained production of IFN-γ-inducible cytokines that establishes a

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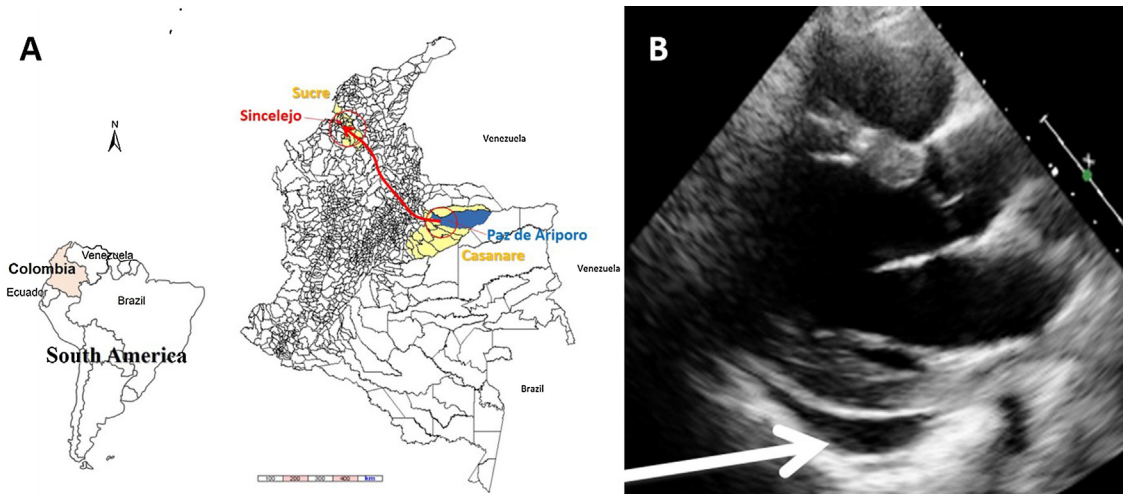


**Fig. 1** (A) Travel history of the patients. Place of exposure was in the department of Casanare, eastern lowlands of Colombia. Place of diagnosis and attention was in the department of Sucre, Caribbean coast, north of Colombia. (Distance between Paz de Ariporo and Sincelejo is 549.05 km). (B) Left ventricle with a normal size, with pericardial effusion without signs of tamponade (arrow).

facilitative environment for continued inflammation [1]. During the acute phase, parasitization of cardiac muscle and brain may cause acute myocarditis and meningoencephalitis, respectively [1,4]. If left untreated, mortality ranges from 5 to 10% [1,3]. The acute phase is then followed by a period of clinical latency referred to as the indeterminate phase that may last for years (or even a lifetime) although patients continue to carry antibodies and possess a low level of parasitemia [1]. There is question of a discrete subclinical intermediate phase because of studies that suggest that chronic tissue damage is a continuous progressive process despite lack of overt clinical manifestations and most patients in this phase have subclinical functional cardiac involvement [1]. Then, these patients require periodical follow-up, as has been determined for our reported cases. These findings have significant implications for the diagnostic and therapeutical approach of those patients returning from endemic areas (Fig. 1A), even being domestic travelers as is our case in Colombia.

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## Competing interests

None declared.

## Ethical approval

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