Introduction

Chagas disease (CD) is classically reported as a neglected disease of vectorial transmission and is endemic in 21 Latin America countries. While it has long been stigmatized as a mere tropical pathology, CD has recently gained prominence, since new means of infection have become more common, including blood transmission and consumption of food contaminated with feces from a given vector. Taking immigration into account, the disease is currently spreading beyond the limits of endemic areas and has become a global issue.1

Many acute CD (ACD) outbreaks have been reported since 1965, by oral accidental routes, in Brazil and in other Latin American countries.2,3 That phase of the disease is, in general, asymptomatic or presents unspecific symptoms, such as fever, fatigue, myalgia, facial and lower limb edema, abdominal pain, diarrhea, vomiting, and dyspnea. One to five percent of the patients develop severe disease, most commonly perimyocarditis, acute heart failure, and/or meningoencephalitis, with a mortality rate of around 5%.1,4

In April 2019, in the countryside of the Brazilian Northeast, a new outbreak of ACD occurred, resulting in the infection of 40 people in a group of 77, all of whom had been hosted at a local school during a spiritual retreat. Although the source of infection is still under investigation, the case drew attention due to the number of infected individuals at the same time and the multiplicity of symptoms, suggesting oral transmission. Therefore, the present article will discuss a singular cardiac manifestation of ACD in one of the victims of the event.

Case Description

A 34-year-old, white male went to a countryside in the Brazilian’s Northeast on April 18th, 2019. Two weeks later, he reported fatigue, dyspnea, dizziness, with a darkened view during a soccer match. Three days later, he still had a headache, which lasted two weeks, accompanied by fever and chills, and chose to self-medicate himself with dipyrone. Due to the persisting symptoms, he sought out medical help at an emergency room, where hypotheses of dengue and upper airway infection were contemplated. One week after the first symptoms, he reported a dry cough, which lasted one month, as well as vomiting and lower back pain in a single episode that suddenly woke him up, but he reported no irradiation. The patient expressed a high degree of pain, with an intensity of nine on a scale of ten, characterized as perforating, which lasted 5 minutes, but without associated symptoms. In the fourth week of symptoms, he presented a painless edema in his lower limbs. Approximately 40 days later, he received the epidemiological and parasitological diagnosis of Chagas Disease, and was prescribed with antiparasitic treatment (benznidazole – 300 mg/day for 60 days), although the patient presented no important alteration by the clinical, electro, and/or echocardiographic view. The patient underwent a physical examination with no abnormal findings. One week later, his symptoms remitted. A 12-lead Electrocardiography (ECG) was performed one month after the beginning of the treatment (Figure 1). A transthoracic echocardiogram was also performed with no sign of abnormalities (Figure 2). In the following month, the patient continued to feel well and was subjected to a new ECG (Figure 3).
Discussion

The patient has shown a nonspecific clinical picture of fatigue and dyspnea, evolving with dizziness, fever, chills, dry cough, vomiting, one episode of lower back pain, and a painless edema in his lower limbs prior to the clinical examination, which showed no changes. The diffuse symptoms made it difficult to elucidate differential diagnoses, although, considering the epidemiological context in which he was inserted, the PCR for Trypanossoma Cruzi was performed, confirming the ACD. There were no clinical cardiac manifestations on the day of the examination; nevertheless, an ECG was conducted, considering that, in other outbreaks, perimyocarditis and/or acute heart failure, though less frequently, has been described at this stage of the disease.\(^4\)

Even though the patient has followed the same clinical pattern, he presented an ECG (Figure 1) with ST segment elevation from V2 to V5 associated with depression of the PR interval. In the second ECG (Figure 3), performed one month after the first, the findings were maintained. Such findings are also poorly described in other studies, which elucidated ventricular repolarization alterations, electrical axis deviation, low voltage, left anterior fascicular block, and right bundle branch block, complete or not, as the most common changes in those patient’s exams.\(^2,6,7\) In some DCA outbreaks, however, Pinto-Dias et al.,\(^8\) Alarcon et al.,\(^9\) and Alvarado-Tapias et al.,\(^10\) have reported recurrent ST-elevations, especially in the population under 18, but PR interval depression was not mentioned, as is the case herein.\(^8-10\) Even with the echocardiogram performed by professionals experienced in the pathology, the exam was unable to add any relevant information.

Causes of ST-elevation are left bundle branch block, left ventricular overload, early repolarization, Prinzmetal angina, ventricular aneurysm, Brugada syndrome, myocarditis, pulmonary thromboembolism, cerebral hemorrhage, hyperkalemia, and heart injury.\(^11\) The main differential diagnoses, however, are acute myocardial infarction and acute pericarditis stemming from subepicardial ischemia.\(^11-13\) In the first, pathological Q waves are present, which are not present in the second.\(^12\) Furthermore, the presence of PR depression, when associated with diffuse ST-elevation with superior concavity, is pathognomonic for acute pericarditis.\(^14\)
Figure 2 – Transthoracic echocardiogram - a) longitudinal parasternal view b) four-chamber apical view: left ventricle (LV) diastolic diameter: 45 mm; left atrium (volume): 34 ml/m$^2$; normal doppler fluxometry; $E'$: 9; ejection fraction LV (Simpson): 55%; right ventricle with preserved systolic dimensions and function; global longitudinal strain: 17.9%.

Figure 3 – Calibration is correct. The ECG was analyzed by the Portuguese acronym “REFASA”: with a sinus rhythm, normal axis, heart rate of 85 bpm; normal amplitude, duration, and polarity of P, QRS, and T; ST segment elevation was shown from V2 to V5; PR interval was down (DII, aVF, V1 – V4) (PR duration = 188 ms); normal QT interval (QT duration = 344 ms), and no inactive electric area was shown.
Conclusion

The diffuse ST-elevation associated with PR depression presented itself as a finding on the ECG of a patient with ACD. Since such an electrocardiographic diagnosis is pathognomonic for acute pericarditis, and the patient was subclinical for this type of cardiac involvement, ECG seems to be an important tool for the management of ACD patients.

The medical community’s attention must be drawn to the fact that, within a specific epidemiological context, the ST-elevation, even in asymptomatic patients, should lead to Acute Chagas Disease as a differential diagnosis.

Author contributions

Conception and design of the research: Barros MNDS. Acquisition of data: Pinto GDC, Pimentel JHM, Medeiros CA. Analysis and interpretation of the data: Pinto GDC, Pimentel JHM. Writing of the manuscript: Pinto GDC, Pimentel JHM. Critical revision of the manuscript for intellectual content: Martins SM, Barros MNDS, Junior WAO, Carrazzone CFV.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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Study Association

This study is not associated with any thesis or dissertation work.

Ethics approval and consent to participate

This study was approved by the Ethics Committee of the Complexo Hospitalar HUOC/PROCAPE under the protocol number 23852719.2.0000.5192. All the procedures in this study were in accordance with the 1975 Helsinki Declaration, updated in 2013. Informed consent was obtained from all participants included in the study.

References

1. Pérez-Molina JA, Molina I: Chagas disease. Lancet. 2018; 391(10115):82-94.
2. Bastos CJ, Aras R, Mota G, Reis F, Dias JP, Jesus RS, et al. Clinical outcomes of thirteen patients with acute chagas disease acquired through oral transmission from two urban outbreaks in northeastern Brazil. PLoS Negl Trop Dis, 2010, 4(6):e711. 10.1371/journal.pntd.0000711.
3. Toso, M A, Vial U F, Galanti N. Oral transmission of Chagas' disease. Rev Méd Chile. 2011;139:258-66.
4. Dias JCP, Ramos Jr AN, Contijoch, et al: 2 Nd Brazilian Consensus on Chagas Disease. Rev Soc Bras Med Trop. 2015, 49:3-60.
5. Barros, MNDS, Silva MCA, Neto NRO, Escarião AG, Albuquerque ALT. New ECG Training Methodology: Demystifying Theory in Practice – Practical Teaching of ECG. Rev Bras Educ Med. 2016, 40:751-6.
6. Ortiz J, Pereira BVM, Couceiro KN, Hossamah R, Doria SS, Silva PRL. Cardiac evaluation in the acute phase of Chagas’ disease with post-treatment evolution in patients attended in the state of Amazonas, Brazil. Arq Bras Cardiol. 2019;112(3):240-6.
7. Colantonio LD, Prado N, Segura EL, Sosa-Estani S: Electrocardiographic abnormalities and treatment with benznidazole among children with chronic infection by trypanosoma cruzi: A retrospective cohort study. PLoS Negl Trop Dis. 2016;10(5):e0004651. 10.1371/journal.pntd.0004651.
8. Pinto-Dias JC. General revision and evolution of acute cases of Chagas Disease studied at the Emmanuel Dias Research Centre between 1940 and 1969. Rev Med Minas Gerais. 2009; 19:25-35.
9. Alarcón de Noya B, Díaz Bello Z, Colmen R, ares C,Ruiz-Guevara R, Mauriello L, Zavale-Jaspe et al. Large urban outbreak of orally acquired acute Chagas disease at a school in Caracas, Venezuela. J Infect Dis. 2010; 201(9):1306-15.
10. Alvarado-Tapias E, Miranda-Pacheco R, Rodriguez-Bonfante C, Velasquez G, Loyo J, Gil-Oviedo M, et al. Electrocardiography repolarization abnormalities are characteristic signs of acute chagasic cardiomyopathy. Invest Clin. 2012;53(4):378-394.
11. ST-segment elevation in conditions other than acute myocardial infarction. N Engl J Med. 2003; 349(22):2128-35. 10.1056/NEJMra022580.
12. Surawicz B, Lasseter KC. Electrocardiogram in pericarditis. Am J Cardiol. 1970; 26(5):471-4. 10.1016/0002-9149(70)90704-6.
13. De Micheli A, Medrano GA: [On the electrophysiopathological concept and ECG manifestations of ischemia, injury and necrosis]. Arch Cardiol Mex. 2009;79:2-4.
14. Friedmann AA. ECG no Hospital Geral.São Paulo:Editora Manole;2016.