Cardiac Disease in Seropositive Chagasic Patients in Panama

To the Editor:

Numerous investigations have shown that Chagas disease is endemic in most of Panama, mainly in rural areas near the Panama Canal where the specific ecological and epidemiological conditions for transmitting this zoonosis are present.1,2 Despite a relatively low prevalence (1%-3%) compared with other South American countries, it is recognised as a public health problem in several regions of the country. However, there are no current clinical studies describing the clinical characteristics manifested by Chagas disease in this small area (3 million inhabitants) of the Americas.

We present a descriptive study of a series of cases in order to point out the principal cardiac disorders observed in infected Panamanians from different endemic zones. 61 patients with chronic Chagas disease were examined: 24 (39.3%) were referred as a result of blood bank screenings; 22 (36.1%) were discovered through field research in endemic communities and 15 (24.6%) due to a clinical diagnosis compatible with Chagas disease. Most patients (39; 63.9%) came from the main endemic region (La Chorrera, Capira, and Arraiján districts in the central part of the country) and the rest (22; 36.1%) came from other scattered zones with demonstrated active transmission (Santa Fe, Chepo, and Chilibre districts). All patients tested positive for infection with Trypanosoma cruzi using 3 serological tests (ELISA, immunofluorescence assay, and Western blot). In these patients, parasitaemia was analysed using haemocultures. Each patient received a complete clinical examination, chest x-ray, electrocardiogram, and echocardiogram.

The results indicate that there were asymptomatic forms in the patients examined, but clinical forms with aberrant ventricular conduction, sinus bradycardia, left ventricular hypertrophy, enlarged heart, and other types of heart disease were predominant. We did not find any gastrointestinal symptoms related to Chagas disease.

Prevalent conduction alterations were incomplete (28; 45.9%) and complete (12; 19.7%) right bundle branch block, and left anterior fascicular block (10; 16.4%). This corresponds with findings from other studies including higher numbers of chronic Chagas disease patients in other endemic regions which register a higher prevalence and incidence rate than that found in Panama. We must stress that complete right bundle block is an important disorder related with increased mortality in patients with chronic Chagas cardiomyopathy (CCC). This means that chance finding of this electrocardiograph alteration in Panamanian patients should be linked to Chagas disease, especially if the patient comes from an area where it is considered endemic. On the other hand, slow heart rate (<50 beats/min) was found in 25 (41%) of the electrocardiograms from the patients who were examined. This sinus bradycardia could be a topic for future research on cardiac dysautonomia in Panamanian Chagas disease patients. Echocardiographic studies documented a left ventricular ejection fraction of 40%, in conjunction with segmentary and general alterations reflecting a systolic dysfunction that is characteristic of advanced stages of Chagas disease. In addition, we found 2 apical aneurisms, which is the most noticeable segmentary disorder in CCC, considered to be a predicting factor of sudden death and related to higher mortality in prospective studies.3 Another symptom indicating the seriousness of CCC is enlarged heart. Radiology examinations found an enlarged heart in 16 (26.3%) of the patients who were evaluated.

As in previously published studies,1 this study showed no or very low parasitaemia in haemocultures. This indicates that there is a significant difference from findings in South American countries, where it is frequently possible (0%-50%) to demonstrate the parasite’s presence in the blood during the chronic phase.4

The series of Chagas infections that we studied contains patients from the main parts of the country where the illness is currently described as being endemic. However, we will need to study a larger, more heterogeneous patient group in order to establish accurate statistics on the frequency and severity of cardiac alterations observed in patients infected with T cruzi in the different endemic regions of Panama.

These results, in conjunction with the efforts of health authorities, will contribute to improve clinical diagnosis, treatment and control of Chagas disease in Panama.

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REFERENCES

Letters to the Editor


Purulent Pericarditis Complicated by Cardiac Tamponade Secondary to a Hydatid Cyst-Associated Hepatic Abscess

To the Editor:

Purulent pericarditis is a rarely-seen clinical entity characterized by a purulent pericardial effusion, and which is generally produced by the extension of a nearby bacterial infection locus or by blood dissemination. Primary infection is rare.1 The clinical profile is very severe and tends to progress to cardiac tamponade2 or else it is the first clinical manifestation.

We present the case of an 81 year old female patient with no relevant medical history who was admitted with the diagnosis of heart failure triggered by a respiratory infection and with a non-specific condition of discomfort and weakness that had been developing over 1 month, and dyspnea with effort, which progressed to dyspnea at rest, had developed in the days before she was admitted. She experienced occasional unproductive coughing and had no measurable fever.

Laboratory tests showed: hemoglobin 11.9 g/dL, haematocrit 36.8%, and leukocytosis, with 16.8 × 10⁹/L (3% band neutrophils), 705 × 10⁹ platelets/L, and fibrinogen, 464 mg/dL. Thoracic radiography showed an enlarged heart with a slight redistribution in both bases. The ECG showed low QRS voltage and diffuse T-wave flattening.

Twenty-four hours later, she had deteriorated clinically and showed clinical and hemodynamic signs of cardiac tamponade, hypertension, pulsus paradoxus, and high venous pressure data. A transthoracic echocardiogram was performed, which confirmed the severe pericardial effusion and the data reflecting cardiac tamponade with collapsed right cavities.

After several failed attempts to perform echocardiogram-guided pericardiocentesis using the subxiphoid approach, which served to extract a small amount of purulent-looking matter but did not confirm its location in the pericardial cavity, we observed a left-side submammary mass extending caudally toward the abdomen. We then ran a thoraco-abdominal CT scan, and it revealed a large cystic lesion on the left hepatic lobe which extended to the pericardium (Figure).

The surgeon then performed another evacuation pericardiocentesis guided through the left parasternal approach and extracted 1000 mL of purulent matter. An emergency open laparotomy was then performed, revealing the abscessed hydatid liver cyst destroying the diaphragm and communicating with the pericardium.

After draining, a pericystectomy was performed and the pericardium was cleansed.