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Case Report

Acute Severely Decompensated Heart Failure in a 23-year-old Guatemalan Immigrant: What about the "Forgotten Disease"?

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Abstract

We are presenting a case of a young immigrant patient from Guatemala with severe acute decompensated heart failure. The differential diagnosis is extensive, including but not limited to viral myocarditis, alcohol-induced or cocaine-induced cardiomyopathy, dilated cardiomyopathy, hypertrophic cardiomyopathy, ischemic cardiomyopathy, toxic cardiomyopathy, thyroid disease, nutritional deficiencies, idiopathic cardiomyopathy, and infiltrative conditions such as amyloidosis or sarcoidosis. One other condition to consider based on this patient's region of origin is Chagas cardiomyopathy, often referred to as the "forgotten disease" because it disproportionately affects marginalized and underprivileged populations in Latin America, where access to healthcare, education, and public health infrastructure is limited, and the disease often goes undiagnosed and untreated. This article will focus primarily on Chagas disease epidemiology, clinical manifestations, challenges in detection and management, and the importance of prevention and control strategies for this disease.

Keywords: Congestive Heart Failure; Chagas Disease; Cardiomyopathy; Immigrant; Serology; *Trypanosoma Cruzi*

Introduction

Although the etiology of acute decompensated cardiomyopathy is well recognized and studied, Chagas disease is a significant cause of cardiomyopathy in Latin America. However, due to limited awareness and diagnostic resources, it is often not considered in the differential diagnosis of cardiomyopathy outside of endemic regions. This lack of recognition can lead to delayed diagnosis and treatment, resulting in significant morbidity and mortality. Herein, we present a case of a young immigrant from Guatemala with severe acute decompensated cardiomyopathy. His laboratory workup and medical management were incomplete; however, could this presentation have been due to Chagas cardiomyopathy? Despite the lack of resolution in this case, we believe it is crucial to underscore the need for heightened public awareness surrounding the underlying causes of typical clinical presentations among populations with atypical epidemiological backgrounds. In this case, we would like to highlight "the forgotten disease," Chagas cardiomyopathy.

Case Presentation

Our patient was a 23-year-old male with no known past medical history who presented to the emergency department with a one-week history of worsening back, abdominal, and testicular pain. He also reported swelling in the upper and lower extremities, as well as abdominal and testicular swelling. The patient also experienced shortness of breath and lethargy. He denied fevers, chills, chest pain, nausea, or vomiting. He also denied trauma, recent animal or insect bites, or close contacts with similar symptoms.

The social and demographic history review was consistent with a Spanish-speaking immigrant from Guatemala who had been

in the United States for less than a year. He had spent more than 3 months crossing borders and deserts from his native country, into Mexico, and then to the United States. The patient was undocumented and worked as a farmhand in North Florida. He stated that he had not seen a medical professional since he was a child.

Although he had a history of binge drinking, he denied any current use of alcohol or illicit substances.

During admission, his vital signs were as follows: pulse 131 beats/min, respiratory rate: 18 breaths/min, blood pressure: 109/57 mmHg, and oxygen saturation: 95%. Electrocardiography was consistent with atrial fibrillation with rapid ventricular response, treated with diltiazem and amiodarone, with conversion to normal sinus rhythm. Initial laboratory findings are shown in Table 1. The toxicology screen was negative, and the urinalysis was normal.

Tests	Results	References	Units
White blood count	9.3	4.0 - 10.5	10^3 uL
Hemoglobin	14.7	13.7 - 17.5	g/dL
Hematocrit	41.9	40.1 - 51	%
Platelets	145	150-400	10^3 uL
MCV	82.3	79.0 - 92.2	fl
ESR	62	0 - 10	mm/hr
CRP	5.56	0 - 0.29	mg/dL
Sodium	118	136-145	mmol/L
Potassium	5.2	3.5-5.1	mmol/L
Chloride	85	98-107	mmol/L
Carbon dioxide	23	21-32	meq/L
Glucose	94	74-106	mg/dL
BUN	82	18-Jul	mg/dL
Creatinine	2.31	0.6-1.30	mg/dL
Protein total	6.3	6.4-8.2	g/dL
Albumin	2.8	3.4-5.0	g/dL
Calcium	7.6	8.5-10.1	mg/dL
Phosphorus	4.7	2.5-4.9	mg/dL
Total bilirubin	5	0.2-1.0	mg/dL
AST	258	15-37	units/L
ALT	543	13-56	units/L
AlkPhos	114	45-117	units/L
Magnesium	2.6	1.8-2.4	mg/dL
Lactic acid	7.3	0.4-2.0	mmol/L
Troponin	0.131	0 - 0.045	ng/mL
NT-Pro-BNP	43561	0 - 900	pg/mL

Table 1: Pertinent laboratory data at the time of presentation.

Physical examination demonstrated a distressed, critically ill patient with findings consistent with anasarca and prominent scrotal swelling. Cardiopulmonary examination demonstrated distant heart sounds, tachycardia, diminished peripheral pulses, pronounced jugular venous distention, and crackles in all lung fields. His abdomen was distended, non-tender to palpation, with a positive fluid wave. His eyes and sclera were normal, with moist mucosal membranes and no palpable lymphadenopathy.

Plain radiography and computed tomography (CT) of the chest, shown in Figure 1, revealed cardiomegaly with vascular congestion, pulmonary consolidations, and left pleural effusion, with CT of the abdomen and pelvis without contrast showing anasarca without bowel obstruction. An ultrasound of the abdomen was positive for diffuse ascites, right pleural fluid, and gallbladder sludge. Transthoracic echocardiogram (TTE) showed a severely dilated left ventricle with reduced systolic function of 10-15%. Right ventricular systolic pressure was estimated to be 86 mmHg, with interatrial septum blowing from right to left, consistent with increased right atrial pressure.

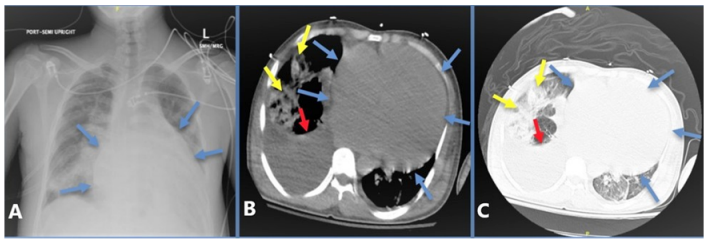

Figure 1: Plain radiography and axial slide of a chest CT-scan without contrast (mediastinal and lung windows, respectively), demonstrating severe cardiomegaly (blue arrows), pulmonary consolidations (yellow arrows), and a large pleural effusion (red arrows). (Panel A) Chest radiography demonstrating severe cardiomegaly (blue arrows). (Panel B) Mediastinal window of CT-chest without contrast revealing severe cardiomegaly (blue arrows), right-sided pleural effusions (red arrows), and pulmonary consolidations (yellow arrows). (Panel C) Lung window of a CT of the chest without contrast revealing severe cardiomegaly (blue arrows), right-sided pleural effusion (red arrow), and pulmonary consolidations (yellow arrows).

The patient was then admitted to the intensive care unit (ICU) with a diagnosis of cardio-septic shock in the setting of decompensated congestive heart failure and community-acquired pneumonia. Appropriate broad-spectrum antibiotic therapy was commenced. Additional assessments included congestive hepatopathy and acute kidney injury, likely cardiorenal syndrome. A chest tube was placed via right-sided thoracentesis, yielding approximately 3.7 liters of transudative fluid over the next several days. The patient was later started on vasopressors and inotropic cardiac support consistent with norepinephrine, vasopressin, and

dobutamine. Additionally, the patient received several doses of intravenous 5% human albumin.

The patient's clinical course was complicated during ICU day 2 with bleeding from the chest tube insertion site and vascular peripheral accesses. Coagulation studies are shown in Table 2. These were consistent with disseminated intravascular coagulation, likely precipitated by the overwhelming sepsis. This complication was treated conservatively with intravenous fluids, monitoring hemodynamics, and a continued effort to treat the infection. The patient ultimately required three units of platelet transfusions as his platelets reached a critical value of 7 x 10^3/uL.

On ICU day 3, blood cultures were positive for *Klebsiella pneumoniae* in 2 out of 2 bottles. At this point, antibiotic treatment was adjusted down to the narrowest spectrum possible. Additional serological studies for hepatitis B and C, Adenovirus, *Bordetella pertussis*, HIV, influenza, parainfluenza, RSV, rhinovirus, *Legionella*, and *Streptococcus pneumoniae* were negative. A comprehensive antinuclear antibody panel (ANA) was also negative.

Tests	Results	References	Units
D-Dimer	25255	<250	ng/mL
Fibrinogen	159	200-400	mg/dL
Pt	52	11.0-12.5	seconds
aPTT	59.7	30.0-40.0	seconds
INR	4.5	<1.1	ratio

Table 2: Coagulation studies.

Trypanosoma cruzi antibodies were sent to the lab to evaluate for possible Chagas disease, given the patient's demographics and clinical characteristics, having migrated from an endemic area of the disease and the unusual presentation of cardiomegaly and congestive heart failure in such a young individual. The antibodies resulted in being non-reactive just two days after the patient had left the hospital before medically advised, still in critical condition. His family members supported his decision due to his socio-economic and immigration concerns, despite reassurances from the medical staff that he was safe. Just a few days later, we were informed that the patient had expired at home. Before his departure, our team was arranging for a medical transfer to a different facility for cardiac magnetic resonance imaging (MRI) and endomyocardial biopsy to further assess the cause of the cardiac pathology.

Discussion

Congestive heart failure and cardiomyopathy

Congestive heart failure (CHF) is a medical condition characterized by structural or functional abnormalities in the heart that result in insufficient blood flow to meet the body's demands, leading to significant morbidity and mortality. It is a prevalent disorder globally and has been associated with high healthcare costs. According to reports, African Americans, Hispanic Americans, and recent immigrants from developing countries have the highest incidence and prevalence of heart failure (HF) among ethnic groups [1]. According to the Global Health Data Exchange registry, the current estimated worldwide prevalence of CHF is 64 million cases, which translates to a substantial financial burden on healthcare systems [1].

Cardiomyopathy (CM) is a disease of the heart muscle that can lead to significant structural and functional abnormalities, resulting in heart failure and arrhythmias. CM is classified into five main types: hypertrophic CM, dilated CM, restrictive CM, arrhythmogenic right ventricular CM, and unclassified CM [2]. CM is a major cause of heart failure and sudden cardiac death worldwide, and its prevalence is increasing due to aging populations, lifestyle changes, and improved diagnostic tools [3]. While the incidence and prevalence of CM may vary depending on the specific type and population studied, it is estimated that millions of people worldwide are affected by this disease [3]. Out of the main etiologies and comorbidities of CM, such as hypertension, coronary artery disease, diabetes mellitus, obesity, chronic kidney disease, and thyroid disorder, among others, Chagas CM is also an important but often overlooked cause, particularly in endemic regions of Latin America. Given the significant burden of Chagas disease and its potential to cause chronic heart disease, it is important for healthcare providers to consider Chagas disease in the differential diagnosis of patients presenting with CM, especially those with a history of travel to or residence in endemic regions. Early diagnosis and treatment of Chagas disease can help to prevent or slow the progression of heart disease and improve patient outcomes.

Despite not performing cardiac MRI, biopsy, or genetic testing, an extensive cardiac evaluation was conducted on the patient in question, yet failed to identify the underlying cause of the severely decompensated cardiomyopathy. Apart from the patient's country of origin and recent immigration history, there was no convincing evidence of the common comorbidities mentioned above, nor did he have any apparent risk factors for coronary artery disease or diabetes mellitus. Furthermore, there was no current use of illicit drugs or alcohol by the patient. Autoimmune processes were ruled out via ANA testing, while viral infections, specifically hepatitis B and C, adenovirus, HIV, influenza, parainfluenza, and rhinovirus, were also ruled out, making other infectious etiologies such as Chagas disease very high on our differential diagnosis.

Chagas Disease

Trypanosoma cruzi is a unicellular parasite known to be the cause of American trypanosomiasis or Chagas disease (CD)

and is found in the urine and feces of the infected triatomine insect (commonly known as the kissing bug) [4]. Other important modes of transmission include food-borne, blood transfusions, vertical maternal-fetal transmission, organ transplantation, and unintentional laboratory exposure [4-6]. Once infected, CD becomes a potentially lifelong illness, leading to the development of chronic sequelae such as enlarged colon, enlarged esophagus, and dilated CM. The latter is the most debilitating disease manifestation, occurring in about a third of these patients [5]. Other acute and subacute CD symptoms are fever, fatigue, body aches, hepatomegaly, splenomegaly, emesis, and diarrhea [4-8].

CD has an acute and a chronic phase. The acute phase of CD is often asymptomatic or characterized by a mild illness consistent with fever, malaise, hepatosplenomegaly, generalized edema, or edema of one eyelid (commonly known as the Romaña sign) that occurs over the first few weeks to months after the infection [7-9]. During this phase, the parasite is easily detected, causing a cascade of immunologic reactions and eventually leading to a rise in detectable immunoglobulin (Ig) M followed by IgG over the following weeks to months [10]. Though the aforementioned immunologic mechanisms reduce the parasite load, *Trypanosoma cruzi* usually persists in deep tissues for the duration of the host's lifespan.

Following the acute phase, the majority of infected patients enter a prolonged, asymptomatic form of the disease called chronic indeterminate. During this time, few or no parasites are found in the blood [5,6,10]. On the other hand, the determinate chronic form comprises an estimated 20-30% of infected people who will develop severe and often fatal problems throughout their lives. These are clinically divided into cardiac, digestive, and cardio-digestive based on their clinical presentations [5,9,11].

It is estimated that 6 to 8 million people are infected with *Trypanosoma cruzi* worldwide [5,12-14], resulting in over 10,000 deaths annually [5,13,14], with the highest prevalence in Mexico, Central America, and South America [4]. Around 300,000 carriers of Chagas disease live in the United States [5-8], and most of them are asymptomatic. Chagas-induced nonischemic CM (NICM) is the leading cause of death in Latin America [5].

Chagas disease diagnosis

The gold standard for diagnosis is based on positive serological evidence of specific IgG class antibodies against T. cruzi through various methods including ELISA, indirect immunofluorescence, or hemagglutination with at least two positive tests using two different antigens, with ELISA being sufficient for diagnosis [7,10,11]. False positives can occur with infections caused by leishmaniasis [11], malaria, and syphilis which is why the World Health Organization (WHO) recommends two different serological tests to confirm the diagnosis [7]. Using blood smears to directly visualize the trypomastigotes is usually only helpful in acute disease and congenital forms of the disease as they are rarely observed in the chronic forms of the disease, with the exception of patients who are immunosuppressed or have a history of HIV [10-11]. In cases of inconclusive results, or serological tests that are discordant, a third assay such as western blot or polymerase chain reaction (PCR) can then be used to confirm infection [7,10,11].

Chagas Disease Waning Antibody Response

We have concluded from the literature review that one of the possible reasons why we did not have a diagnosis of CD in our patient is because of declining antibodies as a result of spontaneous parasite clearance earlier in our patient's life. An example of this event is a published study by Buss, et. al [12] where the association between a downward antibody trajectory, PCR positivity, and symptoms of CD in untreated individuals was analyzed. They found that when asymptomatic, chronically infected individuals were screened for Chagas disease, only a third had seropositive results, while others had laboratory evidence of declining and low serological antibody levels [12]. It is hypothesized that the antibody response generated by the body is directly proportional to the quantitative parasite burden present in the body. Therefore, as the parasite is cleared, the antibody levels diminish [12]. This was also evident with patients who were undergoing pharmaceutical therapy for chronic Chagas disease during a study performed by Murphy, et. al. [15], who found that falling levels of IgG1 against T. Cruzi were suggestive of successful treatment and clearance of the parasite.

We also found that during clinical research performed by Munoz et al. [16] consisting of serological surveillance between treated and untreated CD patients, an interesting finding was reported: about 3% of untreated patients achieved spontaneous seronegative status, which is likely attributed to a highly efficacious immune response against the parasite. Additionally, the overall result of this study revealed that treated patients had to wean serological markers over time as well.

With the above information taken into account, we predict the likelihood that our patient suffered from Chagas disease at a very early age and developed irreversible CM from the disease. He was in turn able to hemodynamically compensate against this cardiac burden throughout the years due to his rather young and intact physiology. Given that he was young and healthy with no underlying immunocompromising conditions, it is likely that his immune system was able to mount a strong response to the parasite over time, resulting in waning antibodies and a complete resolution of parasitemia. This would explain why our single serological test yielded a negative result.

When he presented to the hospital, our patient had severe pneumonia, which likely made his underlying dilated CM worse and eventually resulted in a mixture of cardio-septic shock with congestive hepatomegaly and multi-organ failure. Although uncommon, clinicians should be aware that immunocompetent patients with long-standing untreated Chagas disease can be seronegative, and Chagas disease cannot be ruled out in these patients, especially when there is strong clinical and epidemiological evidence to consider CD as a diagnosis of exclusion.

Although insufficient laboratory testing for CD was performed during his hospital stay, this patient's clinical presentation suited the diagnosis of Chagas CM with subsequent multi-organ failure. If we were able to successfully diagnose CD, it likely would have been in the late chronic phase, where anti-Trypanosoma therapy would be close to ineffective, and it is unclear how often a cure is achieved [8].

Conclusion

Chagas disease continues to be a significant health risk in Latin America and a concern in developed countries. Despite being discovered long ago, it remains one of the most important endemic infections. With a large number of individuals affected by chronic Chagas disease globally, including the immigrant population in the United States, it is crucial to establish improved screening methods and conduct further studies to refine diagnostic and therapeutic guidelines. This can help improve clinical outcomes on a global scale.

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