CLINICAL VIGNETTE

Insect Induced Heart Failure

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Presentation

A 49-year-old El Salvadoran male with no significant past medical history presented to the emergency room complaining of 2 weeks of progressive abdominal pain with jaundice and a 10-pound weight loss. Pain was described as achy and bloated, with episodic sharp sensations in the epigastric and umbilical areas, with dyspnea and pleuritic pain especially when supine. Although anorexic secondary to nausea, the patient did not report abdominal pain related to food or movement. He denied fevers, sweats, chest pain, headaches, vision changes, dizziness, joint pain, rashes, sick contacts, and recent travel. He lived in El Salvador until moving to Southern California 5 years ago. He worked installing drywall, had smoked 10 pack years and drank 12 cans of beer on the weekends. His last alcoholic drink was 10 days prior to presentation.

On presentation the patient was hemodynamically stable and afebrile. He was jaundiced and tremulous with jugular venous distension. Lung examination revealed fine crackles in bilateral bases. Cardiac examination was remarkable for tachycardia with frequent premature ventricular contractions. His left ventricular impulse was enlarged but not displaced. He had a normal S1 and S2 without murmurs or gallops. Abdominal examination revealed a distended, soft abdomen with mild hepatomegaly and positive hepatojuglar reflex. No stigmata of chronic liver disease or fluid wave was present. There was 1+ edema in both ankles and many scabs on the lower legs from localized pruritis.

Although suspicious of liver disease, new onset right heart failure secondary to left heart failure was the working diagnosis. Sinus tachycardia arrhythmia with multiple PVC's, low voltage frontal leads, and Q waves in V2 and V3 were present on the electrocardiogram (EKG) (Figure 1). Chest radiograph showed congestive heart failure (CHF) with minimal pleural effusions, and an enlarged heart. Abdominal ultrasound revealed ascites, bilateral pleural effusions, and normal flow through the portal vein, suggestive of right heart failure. Liver failure was not initially ruled out given mild transaminitis, hepatomegaly, and history of alcoholism. He was diuresed with furosemide, and was ruled out for acute coronary syndrome. Transthoracic echo revealed an ejection fraction of 20% to 25%, mild to moderate global

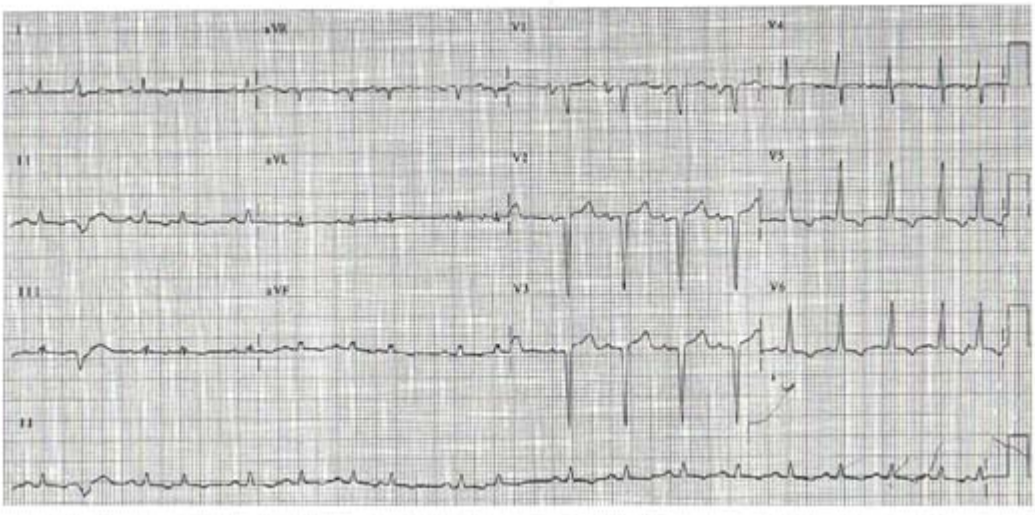

Figure 1 Admission EKG

hypokinesis, mild to moderate mitral and tricuspid regurgitation, and moderate left atrial enlargement. Aggressive diuresis continued with clinical improvement of abdominal pain, and patient was additionally started on spironolactone, statin, ace-inhibitor, and fish oil. Left heart catheterization revealed no flow-limiting coronary artery disease. Paroxysmal ectopic atrial rhythm was present on telemetry monitoring. Because Chagas disease is increasing in Los Angeles County, *Trypanosoma cruzi* serologies were sent and confirmed the diagnosis. After discharge, the patient continued to experience worsening heart failure marked by non-sustained ventricular tachycardia which required ICU admission days after initial discharge, and the placement of an automated internal cardiac defibrillator (AICD).

Discussion

T cruzi is the protozoan parasite that causes Chagas disease, usually transmitted by an infected Reduviid bug ("kissing bug"), found mostly in Central and South America. The infected bug defecates on a person's skin while taking a blood meal and subsequent rubbing of the bite site introduces the contaminated feces into the open bite wound, eyes, or mouth[1]. Although vector-borne transmission is most common, transmission can also occur with blood transfusion, organ transplantation, and vertically (mother-baby)[2-4]. An estimated 8 to 10 million people currently have Chagas disease in the Americas, where poor housing conditions facilitate vector-borne transmission[2]. With worldwide migration beyond the endemic areas, Chagas is becoming more common in countries where it was previously unknown, and approximately 100,000 people are currently infected in the United States, though most were infected abroad. *T cruzi* infection in endemic areas usually occurs in childhood and resolves in several months. However, 5% of cases are fatal with cardiac involvement present in more[1] than 90%. Most individuals stay in the latent phase (seropositive) of the disease for the rest of their life, but still have subclinical cardiac involvement on electrophysiologic examination of the heart. However, in 20% to 30% of cases the chronic symptoms of the disease develop, after a latent period of 10 to 30 years, usually affecting the heart[2]. Chagas heart disease can present with heart failure (typically biventricular), arrhythmia, and vascular emboli. There is often a prominent apical impulse and regurgitant mitral and tricuspid valves, as seen in our patient[1]. Autonomic dysfunction, especially bradycardia, is common, and EKG commonly shows premature contractions (atrial or ventricular), non-sustained ventricular tachycardia, atrial fibrillation, abnormal Q waves, and/or differing degrees of heart block[1]. Unfortunately, cardiac dysfunction associated with Chagas disease is associated with a higher mortality than for other causes, with 5-year mortality rates[5] over 50%. In patients with left ventricular ejection fraction of less than 30%, mortality is over 70% at 2 to 4 years[2]. Death often occurs from ventricular arrhythmias, complete heart block, embolism, and refractory congestive heart failure[2].

Anti-Trypanosomal medications (benznidazole and nifurtimox) are effective in curing or slowing the progression of Chagas disease only if advanced cardiomyopathy has not yet developed, therefore treatment was not an option in our patient. Treatment of Chagas-induced heart failure is symptomatic and similar to other causes of heart failure[1]. Amiodarone appears more effective as an anti-arrhythmic than other medications in Chagas patients[6]. Heart transplantation has been shown to better increase survival in Chagas patients with end-stage disease compared to non-Chagas recipients[7]. In patients with ventricular arrhythmias, automated internal cardiac defibrillators (AICD) should be considered, as patients with hemodynamically unstable ventricular tachycardia (VT) have higher mortality rates compared to those with tolerated VT or no VT (69% vs 22% and 26%, respectively)[8]. Over 90% of patients in one study reported the device to be discharged within a year of implantation[9], while in another study 85% reported defibrillation within 6 months, significantly higher than the 51% in a control group of coronary artery disease patients[10]. Chagas patients with dilated cardiomyopathy and chronic atrial fibrillation also benefited from AICD implantation [11].

Conclusion

Our case illustrates both an unusual presenting complaint leading to the diagnosis of Chagas disease, and a reminder that what once was a rarely seen disease is becoming more common in non-endemic areas such as Los Angeles county due to an influx of Central and South American immigrants. Specialized Chagas cardiomyopathy clinics have been established to promote the diagnosis and treatment of Chagas disease in the United States, given the relative lack of awareness of the condition in non-endemic areas[12]. Chagas is often insidious in onset, taking decades for symptoms to present. However, once clinical findings are present, deterioration can be extremely rapid and include unstable cardiac arrhythmias that are often difficult to treat.

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