

CASE REPORT

INTERMEDIATE

CLINICAL CASE: ACC.23

An Unusual Case of Parasitic Constrictive Pericarditis



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ABSTRACT

Parasitic constrictive pericarditis is a rare entity. We present a case of a 75-year-old man who presented with dyspnea, ascites, and pedal edema and was found to have constrictive pericarditis on multimodality imaging with positive serology for *Strongyloides Stercoralis*. Treatment required ivermectin and radical pericardiectomy with significant clinical improvement. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2023;22:101983) © 2023 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

HISTORY OF PRESENTATION

A 75-year-old man presented to the clinic with the chief symptom shortness of breath, which was associated with abdominal swelling and bilateral pedal edema of 18 months duration. There was no orthopnea or paroxysmal nocturnal dyspnea, fever, cough, or chest pain. The patient reported undergoing biweekly paracentesis for 6 months. A comprehensive physical examination revealed normal vital signs, the presence of a pericardial knock, ascites, and pedal edema bilaterally. His medications included torsemide 40 mg twice daily, aspirin 81 mg once daily,

metoprolol succinate extended release 25 mg once daily, simvastatin 20 mg once daily, and spironolactone 25 mg once daily.

PAST MEDICAL HISTORY

The patient had a past medical history of essential hypertension, dyslipidemia, and coronary artery disease with drug-eluting stent implantation in the right coronary artery. He had a 15-pack year smoking history. The patient further confirmed his travel to Mexico for 2 weeks before symptom onset. No prior history of alcoholism or HIV was noted.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses included heart failure, chronic liver disease, constrictive pericarditis (CP), and restrictive cardiomyopathy.

INVESTIGATIONS

Investigations before the presentation included a right heart catheterization, which demonstrated

LEARNING OBJECTIVES

- To highlight the importance of rare etiologies like parasitic infections for constrictive pericarditis.
- To understand the implication of multimodality imaging in diagnosis and treatment of constrictive pericarditis.

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**ABBREVIATIONS
AND ACRONYMS****CMR** = cardiac magnetic resonance**CP** = constrictive pericarditis

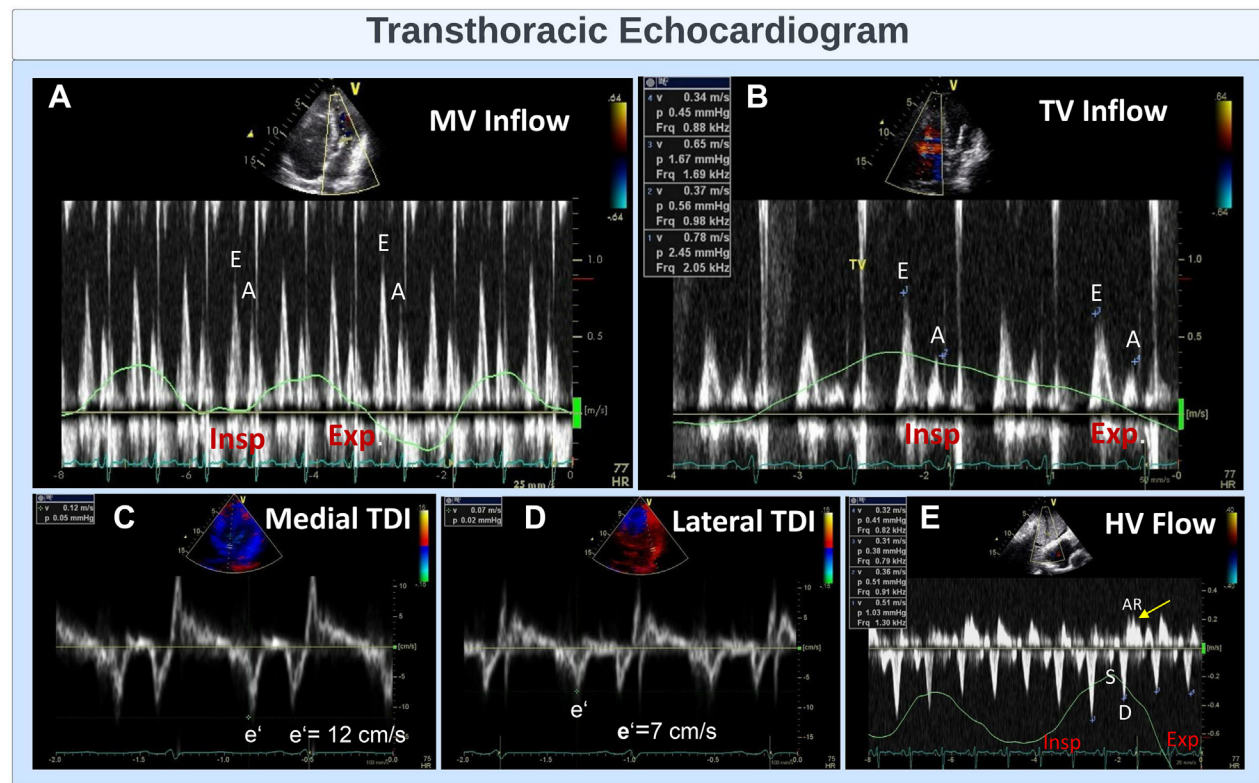
elevated and equalized diastolic cardiac pressures. Left heart catheterization showed patent stents in the right coronary artery. He also underwent a liver biopsy, which showed no evidence of cirrhosis. On presentation, the patient had a normal Westergren sedimentation rate of 10 mm/h (reference 0-10 mm/h), and C-reactive protein 0.5 mg/dL (reference 0-1 mg/dL). Interestingly, the absolute eosinophil count was elevated up to 0.77 k/uL (reference 0.00-0.45 k/uL). The rest of the complete blood counts and metabolic panel were within normal limits. Additional work-up for eosinophilia revealed positive IgG antibodies for *Strongyloides Stercoralis* (3.39 IV; reference ≤ 1.49 IV).

Electrocardiogram demonstrated normal sinus rhythm. Echocardiography showed septal bounce, respirophasic shift of the ventricular septum, tethering of the right atrium, and right and left ventricles as well as pericardial thickening with pericardial calcification, annulus reversus, and hepatic vein

expiratory diastolic flow reversal (**Figure 1**). Cardiac magnetic resonance imaging (CMR) showed diffuse circumferential pericardial thickening measuring up to 6 mm, diastolic septal bounce, respirophasic septal shift, conical ventricular deformity, and dilated inferior vena cava (IVC) of 3.3 cm, all features suggestive of CP. No definitive late gadolinium hyper-enhancement was noted (**Figure 2**).

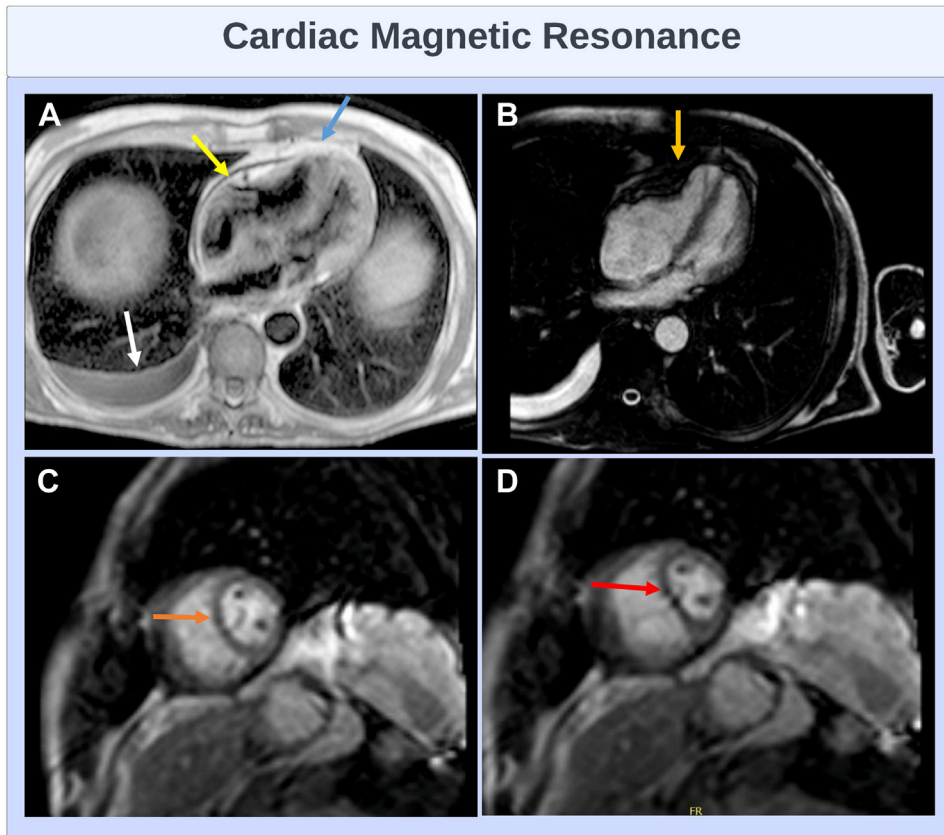
MANAGEMENT

The patient was diagnosed with CP secondary to *Strongyloides Stercoralis* infection. Following a multidisciplinary approach, he was medically treated with oral ivermectin 12 mg once followed by a second dose of 12 mg 2 weeks later as per the infectious disease specialist recommendations and then underwent radical pericardiectomy by the cardiothoracic surgical team. The pericardial pathology revealed marked fibrosis with mild chronic pericarditis.

FIGURE 1 Transthoracic Echocardiogram Findings of Constrictive Pericarditis

Transthoracic echocardiogram showing significant respirophasic flow variation across the MV (**A**) and TV (**B**). Tissue Doppler Imaging (TDI) of the medial (**C**) and lateral (**D**) annulus exhibiting a higher e' (early relaxation velocity) of the medial compared to the lateral annulus ("annulus reversus") and hepatic vein (HV) expiratory (exp) diastolic flow reversal (**E**, yellow arrow). A = peak velocity of blood flow in late diastole from atrial contraction; D = diastolic wave; E = peak velocity of blood flow from left ventricular relaxation in early diastole; Insp = inspiratory; MV = mitral valve; S = systolic wave; TV = tricuspid valve.

FIGURE 2 Cardiac Magnetic Resonance Imaging Findings of Constrictive Pericarditis



Cardiac magnetic resonance imaging demonstrating conical deformity (A, blue arrow), thickened pericardium (A, yellow arrow), pleural effusion (A, white arrow), right ventricular compression (B, yellow arrow), and respirophasic interventricular septal shift on free breathing (C and D, orange and red arrow).

DISCUSSION

Strongyloidiasis is endemic in tropical and subtropical climates. Humans get infected when the filariform larvae penetrate the skin from soil contaminated with feces. They travel in the blood stream to the alveoli in the lungs, from where they migrate up the tracheobronchial tree and are eventually swallowed to habitate the intestine. The larvae then mature into an adult female who lays eggs, which in turn hatches into rhabditiform larvae. It is then excreted in feces and turned into filariform larvae completing the life cycle. Sometimes, the larvae in the intestine can become infective filariform larvae penetrating the intestinal mucosa leading to autoinfection and hyperinfection.¹ Multiorgan systems including pericardial

involvement like pericarditis and pericardial effusion with Strongyloidiasis have been reported.^{2,3}

The causes of CP are multifold, including viral/idiopathic pericarditis, postcardiac surgery, radiation, tuberculosis, and autoimmune diseases like systemic lupus erythematosus. Tuberculosis is a common cause in developing countries, while viral/idiopathic infections are among the common causes in Europe and the United States.⁴ Parasitic etiologies are very rare.

Multimodality imaging plays a key role in the management of CP. Echocardiography is often the first-line imaging modality, which reveals pericardial thickening and calcification. Other features suggestive of CP are diastolic septal bounce, respirophasic shift of the ventricular septum, moderate biatrial enlargement, restricted left ventricular filling, and

dilated IVC and hepatic veins.⁵ A computed tomography (CT) scan is very accurate for detection of pericardial thickness and calcification. Additionally, it can also aid in preoperative planning of pericardiectomy by delineating the severity and exact location of pericardial thickening and calcification.⁵ CMR is often considered as the diagnostic procedure of choice for CP and reveals pericardial thickening, IVC dilation, increased right-sided filling pressures, impaired right ventricular diastolic filling, septal bounce, and conical ventricular deformity.⁵ Our case demonstrated all of the clinical and imaging features of CP. Given the travel history to Mexico, eosinophilia, and positive serology for *Strongyloides Stercoralis*, CP was diagnosed to be secondary from longstanding strongyloidiasis.

The mainstay treatment of CP is surgical pericardiectomy. Medical therapy in CP can be indicated in cases of specific etiologies such as tuberculosis. Antitubercular therapy minimizes the constriction risk from >80% to <10%.^{6,7} Further, medical management can be instituted to resolve the cases of transient constriction.⁸⁻¹⁰ Specifically, although transient constriction can resolve spontaneously, the use of oral anti-inflammatory agents (nonsteroidal anti-inflammatory drugs, steroids, colchicine and now interleukin-1 blockers) may hasten symptom resolution and improve overall outcomes. However, medical therapy should never delay the surgery because advanced cases are associated with poor prognosis and a heightened mortality rate if pericardiectomy is delayed.⁸ Ivermectin is the drug of choice for treatment of strongyloidiasis. It has a strong affinity for glutamate-gated chloride channels in the nerve and muscle cells of the parasite, thereby increasing the permeability of the channels leading to paralysis and death of the parasite.

FOLLOW-UP

The patient showed significant clinical improvement on outpatient follow-up, with remarkable improvement in shortness of breath, ascites, and pedal edema. He completed his scheduled doses of ivermectin.

CONCLUSIONS

In summary, parasitic infection, although very rare, should always be considered as an etiology for constrictive pericarditis. Comprehensive history, including travel history and physical examination, constitute an indispensable part of the patient assessment. Early diagnosis and treatment are crucial to prevent complications like constriction. Multimodality imaging with echocardiography, CT scan, and CMR aid in the diagnosis and management of CP. Radical pericardiectomy remains the treatment of choice.

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