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Case report

Constrictive Pericarditis Presenting as Isolated Ascites ☆,☆☆

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ABSTRACT

Constrictive pericarditis is a rare cause of right-sided heart failure secondary to a stiff, non-compliant pericardium. Clinical presentation can vary considerably and requires a high suspicion for diagnosis. A 31-year-old male presented to the emergency department with complaints of abdominal distension. An abdominal ultrasound revealed large volume ascites; thus, it was initially suspected he had underlying cirrhosis. However, an echocardiogram revealed a diagnosis of constrictive pericarditis. It's important for clinicians to consider constrictive pericarditis in a patient presenting with unexplained right-sided heart failure.

Introduction

Constrictive pericarditis is characterized by a rigid pericardium which impedes cardiac filling leading to diastolic heart failure. Clinical presentation can vary dramatically but typically patients present with complaints of volume overload. In our case a 31-year-old male presented to the emergency department with complaints of worsening abdominal distension. While he was initially suspected to have cirrhosis, multiple imaging modalities demonstrated a thickened pericardium causing hemodynamic compromise consistent with a diagnosis of constrictive pericarditis. Constrictive pericarditis is extremely rare; however, it should always be on the differential as symptomatic burden can be relieved through pericardiectomy.

Case presentation

A 31-year-old man presented to the emergency department with progressively worsening abdominal swelling and unintentional weight loss for five months. He denied any fever,

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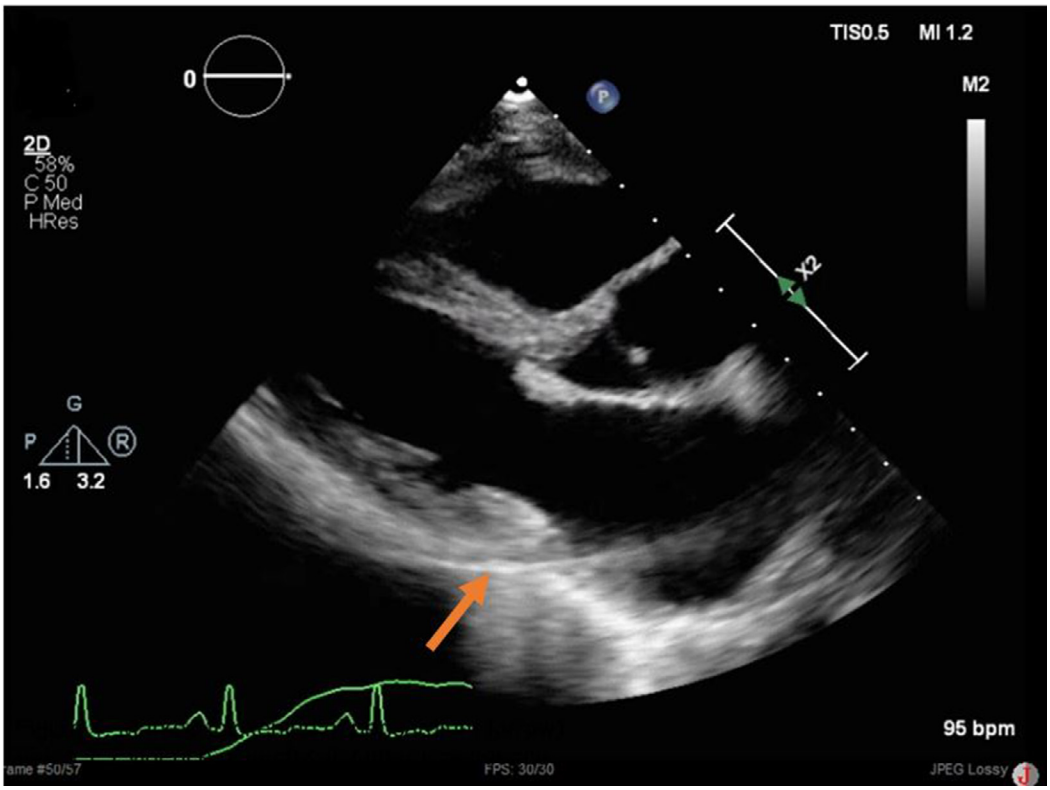

Fig. 1 – Marked thickening of pericardium (arrow)

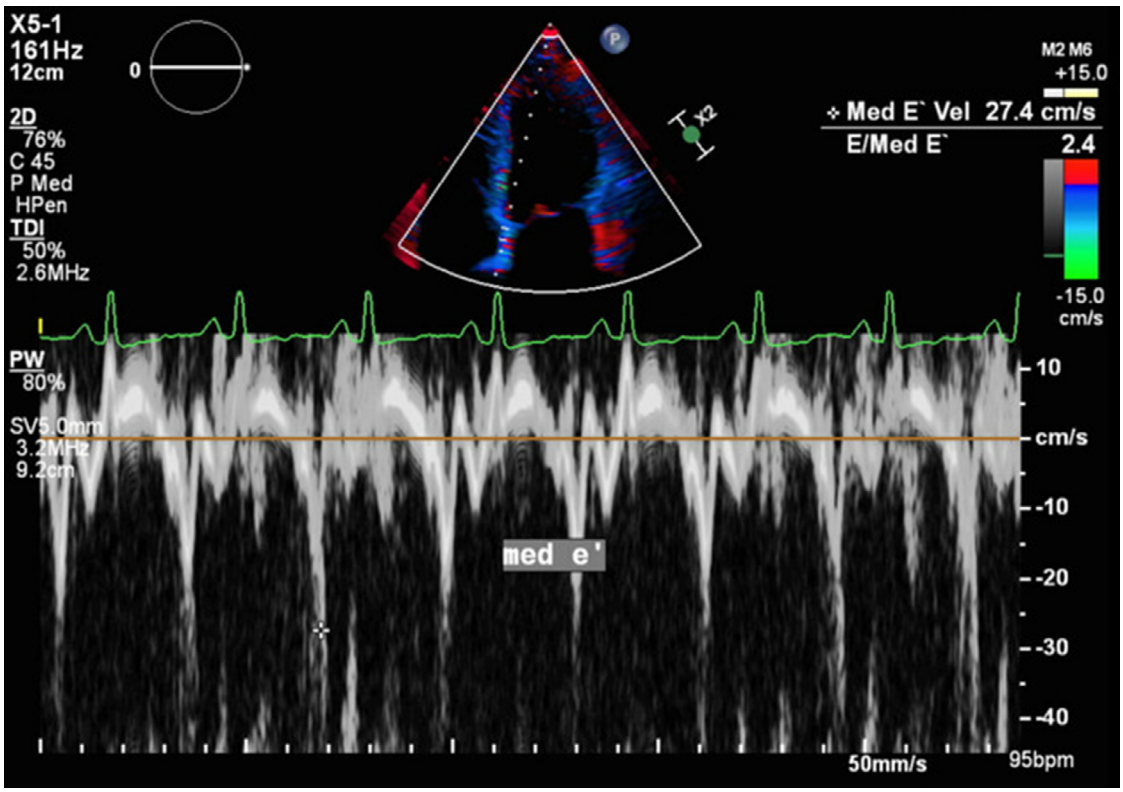

Fig. 2 – Demonstrating medial E' velocity of 27.4cm/s, exceeding Mayo Clinic criteria of 9.0 cm/s for constrictive pericarditis

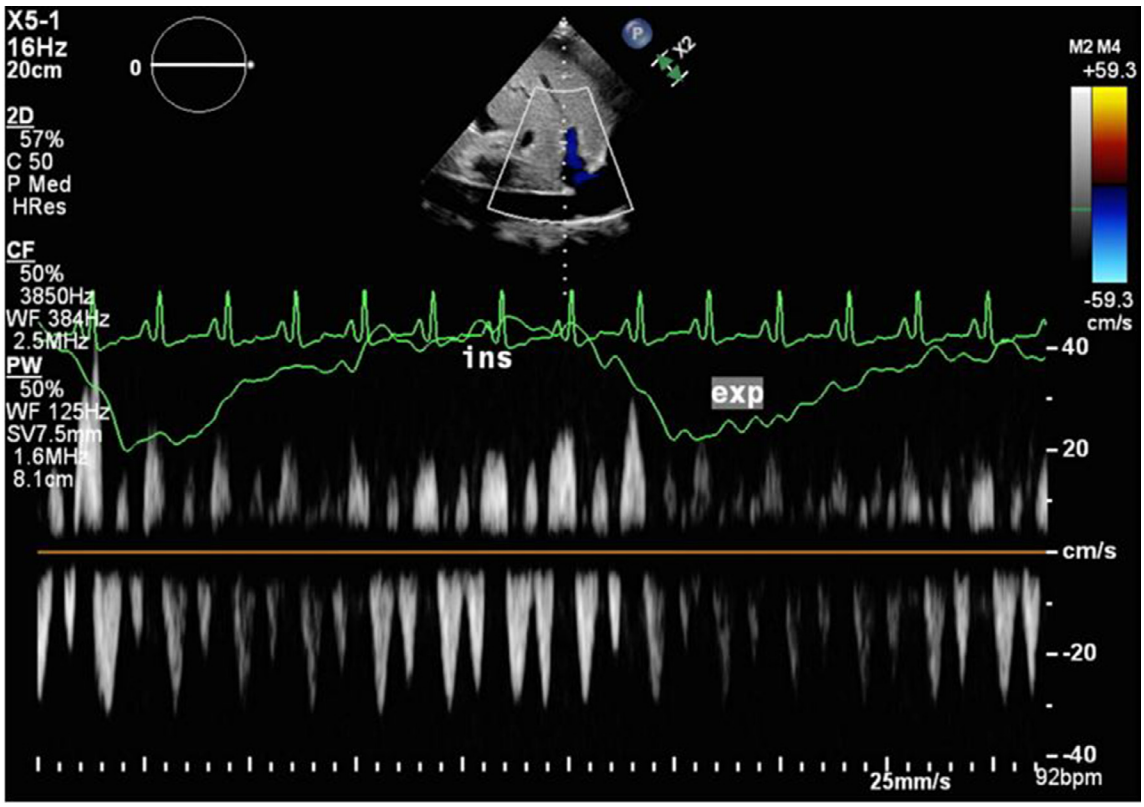

Fig. 3 – The hepatic venous index of diastolic reversal velocity divided by systolic forward velocity equals 0.82 (NR = 0.78)

chills, night sweats, chest pain, shortness of breath, palpitations, orthopnea, paroxysmal nocturnal dyspnea, or syncope. The patient had a history of generalized anxiety and major depression. He was not on any medications and denied drug or alcohol use. He was adopted and unable to provide any family history.

On presentation his blood pressure was 105/68 mmHg and heart rate was 106 beats per minute. On physical examination his abdomen was non-tender but distended with shifting dullness. His cardiac exam revealed normal S1/S2 heart sounds with no murmur, rub, or gallop. His lungs were clear to auscultation. There was trace pitting edema of his lower extremities and jugular venous distension (JVD).

Abdominal ultrasound was suggestive of cirrhosis with large volume ascites. A diagnostic and therapeutic paracentesis was performed with 11 liters removed. His JVD improved after his paracentesis suggesting his ascites was the predominant cause of his distended jugular veins. Peritoneal fluid studies yielded an albumin of 2.4 mg/dL, serum ascites albumin gradient was 1.5. Purified protein derivative (PPD) skin test was non-reactive. Standard cirrhosis work-up, including viral hepatitis and autoimmune panels were negative. The focus instead shifted to a cardiac etiology for his ascites.

A transthoracic echocardiogram (TTE) showed left ventricular ejection fraction (LVEF) of 50-55% and thickened pericardium (Fig. 1). There was evidence of ventricular interdependence with resulting respiratory variation in aortic flow velocity (Video 1). Medial mitral annular e' velocity was 27.4 cm/s and hepatic vein expiratory diastolic reversal ratio was 0.82(Fig. 2 and Fig. 3). Given his clinical presentation and

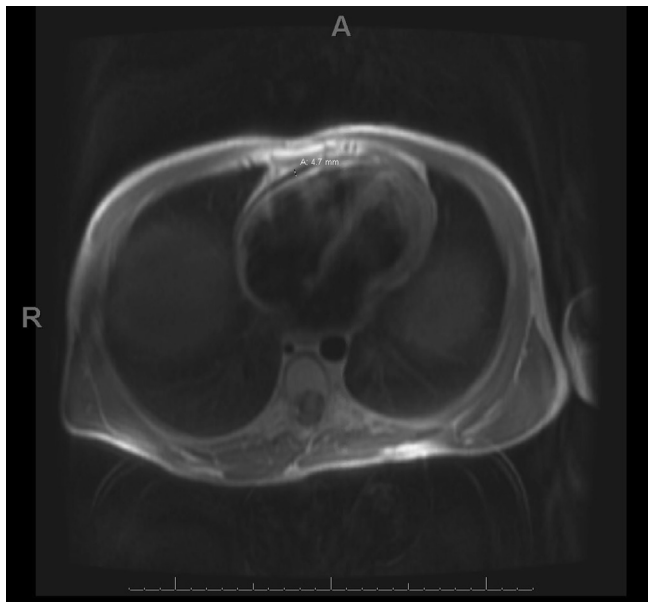

Fig. 4 – Diffuse, circumfrential pericardial thickening is seen measuring up to 6 mm without pericardial effusion or calcification

echocardiogram he was suspected to have constrictive pericarditis as the cause of his ascites. He subsequently completed a right and left heart catheterization as a confirmatory test prior to intervention.

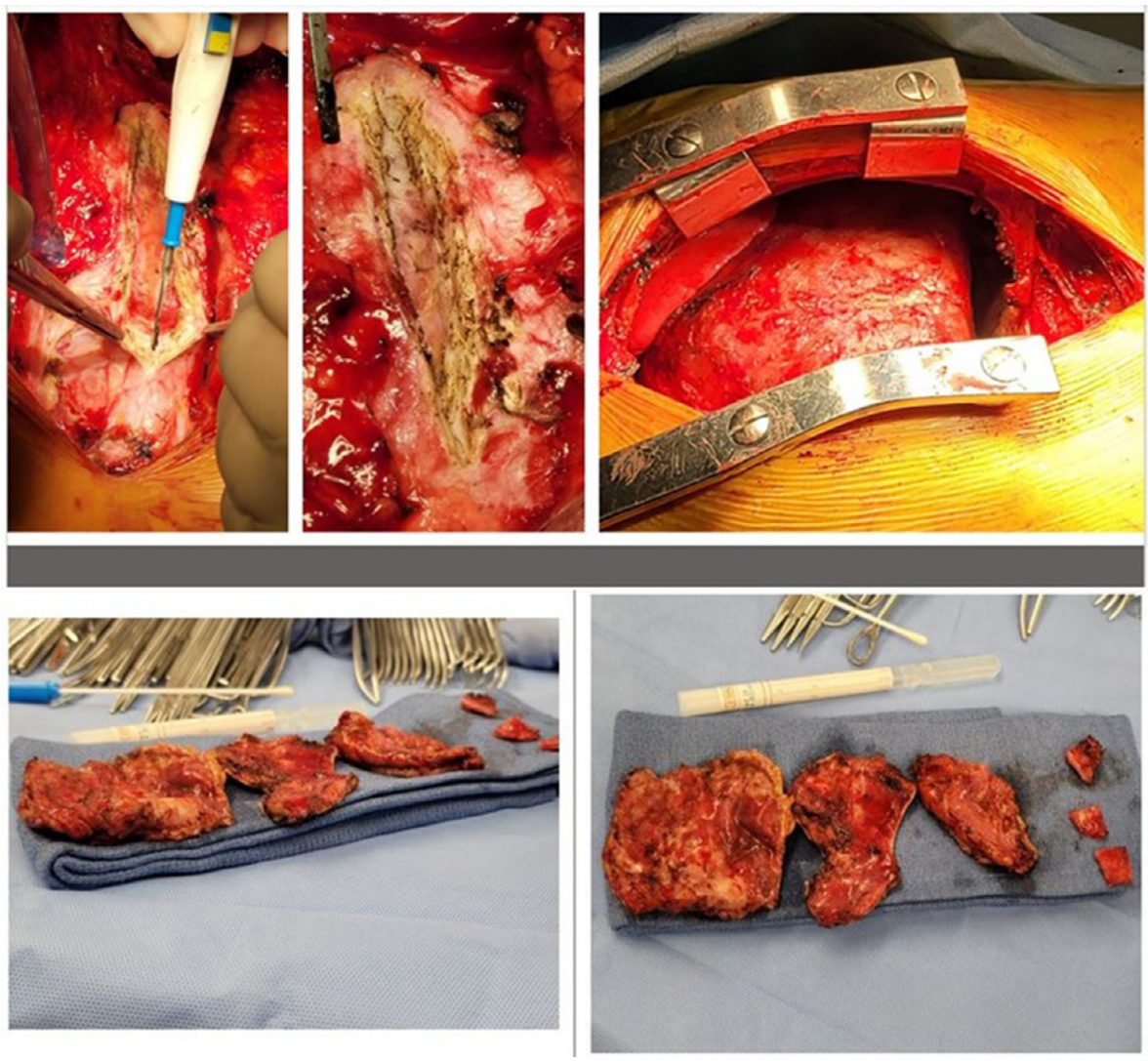

Fig. 5 – The pericardium was dense and fibrotic completely encapsulating the heart. At the beginning of the procedure, the central venous pressure (CVP) was 25 mmHg. After the pericardium was incised to the level of the pericardial fat and removed the CVP was 15 mmHg

Simultaneous left and right ventricular pressures were obtained which demonstrated elevated filling pressures. However, no prominent ventricular interdependence was demonstrated. While he had evidence of hemodynamic compromise during his initial echocardiogram, we were unable to reproduce it during invasive hemodynamic catheterization. Given the high clinical suspicion for constrictive pericarditis we decided to complete cardiac magnetic resonance (CMR) imaging as a second-line imaging modality to confirm his thickened pericardium and abnormal interventricular septal motion we initially saw on echocardiogram.

CMR imaging redemonstrated a thickened pericardium without evidence of any pericardial effusion or calcification (Fig. 4). A prominent diastolic interventricular septal bounce and LVEF of 53% was noted on CMR (Video 2).

The patient was referred for pericardiectomy. In the operating room thickened pericardium was directly visualized, and pericardial stripping was performed (Fig. 5).

The patient recovered rapidly after pericardial stripping and was discharged home four days later. At three-month follow-up he had complete resolution of his ascites. Histologic findings are shown in Figure 6. The cause of his constrictive pericarditis was determined to be idiopathic.

Discussion

Constrictive pericarditis arises secondary to chronic inflammatory changes resulting in fibrous thickening of the pericardium [1]. Scarring can progressively restrict ventricular filling past early diastole. The list of etiologies for constrictive pericarditis is extensive, but the differential varies based on geography. In developing countries tuberculosis is the most common cause worldwide, meanwhile pericardial diseases following a viral illness, cardiac surgery, or radiation therapy are more common in developed countries [2–4].

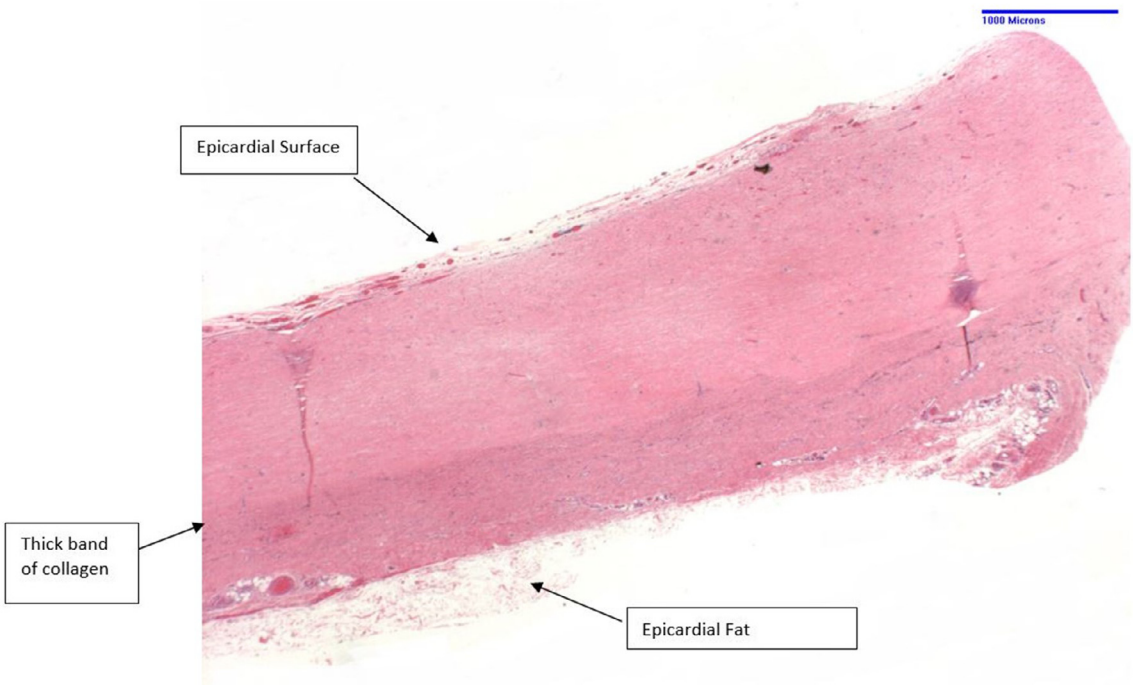

Fig. 6 – Histopathologic examination revealed dense pericardial fibrosis with minimal microscopic calcification and epicardial neovascularization consistent with idiopathic constrictive pericarditis

Clinical presentation can vary but patients tend to complain of chronic symptoms secondary to volume overload including peripheral edema, dyspnea on exertion, or worsening abdominal distension as seen in this case. Patients can present with elevated JVD and peripheral edema secondary to elevated right-sided pressures and a high-pitched early diastolic sound can be appreciated from the sudden cessation of ventricular filling, known as a pericardial knock.

According to 2015 ESC Guidelines the first line imaging modality for patients with suspected constrictive pericarditis is TTE [4]. Key imaging findings include ventricular interdependence – a result of non-compliant pericardium preventing diastolic ventricular filling. Normally inspiration decreases intrathoracic pressure allowing inflow of blood into both ventricles. In constrictive pericarditis, inspiration results in decreased intrathoracic pressure without significant change in the intracardiac pressures. Intrathoracic-intracardiac dissociation leads to right ventricular (RV) expansion and shifts the interventricular septum to the left as the stiff pericardium limits left ventricular (LV) filling. In expiration the septum shifts towards the right lowering the RV's ability to fill. Additional echocardiographic findings suggestive of constrictive pericarditis include expiratory hepatic vein diastolic flow reversal ratio (≥ 0.79 cm/s) and elevated medial e' velocities (≥ 9cm/s). Each of these findings are independently associated with the diagnosis of constrictive pericarditis and were seen in our patient. The Mayo Clinic Criteria for echocardiographic evidence of constrictive pericarditis found the combination of these three variables had a diagnostic specificity of 97% for constrictive pericarditis [5].

Accordingly, ventricular interdependence seen on echocardiogram can be demonstrated on cardiac catheterization. Simultaneous RV and LV pressure tracings with discordant respiratory changes are pathognomonic for and are considered the most sensitive and specific finding for constrictive pericarditis [6]. While in a restrictive cardiomyopathy, or even a normal heart, concordant respiratory variation is seen instead.

Although direct visualization of pressure tracings is classically the gold standard when confirming constrictive pericarditis, in our patient we were unable to observe ventricular interdependence during cardiac catheterization. Hypovolemia can mask typical pressure tracings, but our patient had been optimized prior to catheterization and filling pressures were elevated [5].

CMR offers a comprehensive imaging modality. Not only does it allow visualization of the pericardium, but also impact on the structure and function of the myocardium. Real-time imaging during free breathing can evaluate hemodynamics during cardiac filling which emulates what we elicit during cardiac catheterization. Another novel approach, Biderman et al., described the utility of a tagged myocardium study measuring visceral-parietal pericardial adherence to determine constriction offering another alternative method when trying to confirm an elusive diagnosis such as constrictive pericarditis [7]. Over time cardiac MRI will likely replace catheterization as the most sensitive and specific modality for diagnosis of constrictive pericarditis.

Conclusions

Constrictive pericarditis remains a difficult, but important diagnosis given surgical interventions available for treatment.

Reliance on a single test to confirm or exclude the diagnosis may lower the sensitivity for detection as illustrated by the absence of convincing ventricular interdependence during respiration observed during his cardiac catheterization. The use of multiple imaging modalities allowed us to confirm the diagnosis of idiopathic chronic constrictive pericarditis followed by successful surgical intervention.

Informed consent

Informed consent for publication of this case was obtained from the patient.

Authorship

All authors had full access to the data when designing and drafting the manuscript.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.radcr.2021.10.047.

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