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Letter to the Editor

Treatment of refractory enterovirus effusive-constrictive pericarditis with corticosteroids

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A 33-year-old man presented to the emergency department with one week of fever, malaise, sinus congestion, and cough. He had no significant medical, surgical, family, or social history, and was previously on no medications. He did report contacts at work with upper respiratory tract infection symptoms. Three days prior to admission, he was prescribed amoxicillin by his primary care provider for possible bacterial sinusitis, and also started guaifenesin and ibuprofen. However, this failed to address his symptoms, and he returned to the clinic the morning of admission. He was noted to have a new systolic murmur, an EKG was found to be normal, and the patient was then sent home on azithromycin, with instructions to return for fever > 100.5. At home, he spiked a temperature to 102 F, and had an episode of pre-syncope with dizziness and nausea, and he then presented to the emergency department.

In the ED, vitals included temperature of 100.4 F, blood pressure of 98/62 mm Hg, heart rate of 107 beats/minute, and respiratory rate of 20 breaths/minute. Exam revealed a pericardial friction rub, positive pulsus paradoxus to 18–20 mm Hg, positive Kussmaul's sign, and abrogation of radial pulse with Valsalva. Initial labs were notable for a normocytic anemia with hemoglobin of 10.1 g/dL, white blood cell count of 6.4/nL, mild elevation of AST and ALT (66 and 84 IU/L respectively), CRP of 9.7 mg/dL and ESR of 62 mm. Chest X-ray revealed small pleural effusions and a mildly enlarged cardiomediastinal silhouette. Echocardiogram revealed a large pericardial effusion with no chamber collapse, but with respirophasic mitral valve inflow, LVOT/AV Doppler variation, and plethoric noncollapsible IVC concerning for early tamponade (Fig. 1).

A pericardial drain was then placed, and initial analysis of the pericardial fluid revealed an RBC count of 284,000 cells/mm^2, WBC count of 2717 cells/mm^2 with 71% neutrophils, LDH of 746 IU/L, and protein of 5.2 g/dL. An extensive evaluation was performed, ultimately resulting in the isolation of enterovirus via a viral culture of the pericardial fluid. The remained of the workup was negative. Notably, coxsackie A and B serologies were negative. The drain was removed after six days, and he was then started on ibuprofen and colchicine. Repeat echocardiogram revealed findings consistent with effusive — constrictive pericarditis (Fig. 2).

The patient was discharged, but returned to the ED for incessant cough and continued fevers. Due to evidence of persistent constriction on echocardiogram, he was transitioned from ibuprofen to aspirin, but he continued to experience fevers, chills, and headaches. Cardiac MRI revealed diffuse thickening of the pericardium (Fig. 3) with evidence of constriction on cine images. Given the lack of response to approximately two weeks of initial medical therapy, the decision was made to add 1 mg/kg prednisone to his medication regimen, with a taper of 10 mg/month. Follow-up at 1 week from the initiation of prednisone revealed no effusion or evidence of constriction and marked improvement in symptoms. He has since completed several months of his taper without incident, and repeat cardiac MRI has confirmed resolution of pericardial thickening with no evidence of constrictive physiology.

This case of enterovirus effusive-constrictive pericarditis highlights several important points in the diagnosis of pericardial disease, and inspires questions with regard to the management of this entity. This is especially true given the recent emergency of enterovirus D68 causing respiratory symptoms very similar to those afflicting our patient [1,2] Although pericarditis is often attributed to a viral infection, the determination of a specific viral cause is uncommon [3,4]. Furthermore, the viral etiology of a case of pericarditis is most often inferred from serologic studies; isolation from pericardial fluid or tissue is rare [5]. Specific etiologies are more likely to be identified in patients such as ours who develop large effusions or cardiac tamponade [3], as there is a increased diagnostic yield of therapeutic pericardiocentesis for tamponade or hemodynamic derangement [3,6].

Acute pericarditis has a diverse natural history, ranging from resolution with first-line therapy to the development of constriction with or without effusion. Transient cardiac constriction has been reported in

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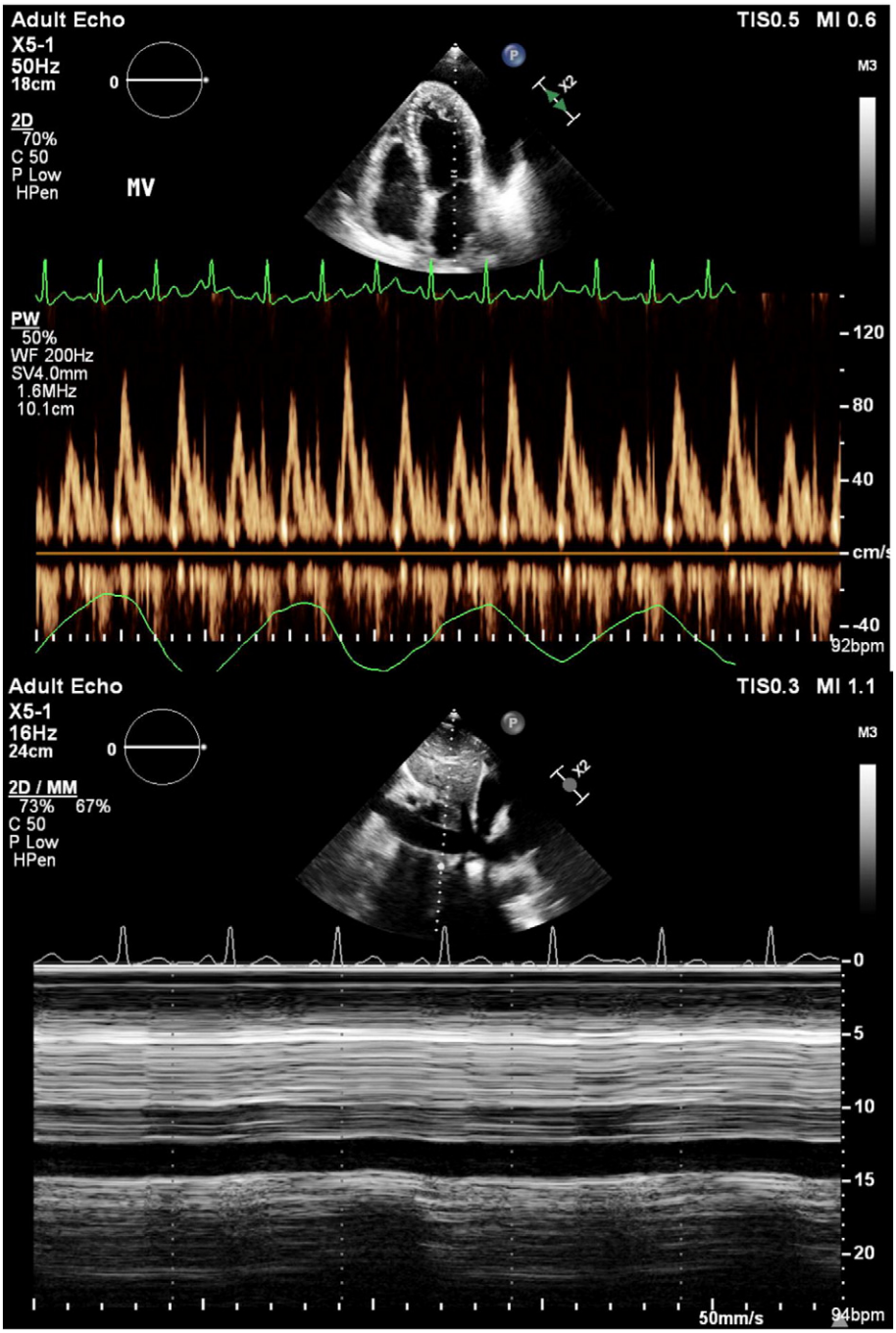

Fig. 1. Transthoracic echocardiogram. Respirophasic variation in mitral valve inflow and plethoric inferior vena cava, without inspiratory collapse.

several studies of patients with idiopathic pericarditis, with resolution averaging from 2 to 9 months [7,8]. It is postulated that constriction is the result of edema, fibrin deposition, or inflammation from pericarditis [9]. Overall, approximately 1% of patients with idiopathic pericarditis require pericardiectomy [10]. The risk does not appear elevated in patients who develop constriction after the resolution of effusion following an episode of effusive pericarditis, as one series followed 16 such patients, none of whom required pericardiectomy [9].

However, pericardiectomy is a frequent outcome in patients with effusive-constrictive pericarditis [10]. This uncommon sequela of acute pericarditis is most often idiopathic [11], and is postulated to represent an intermediate on the spectrum from acute pericarditis to constrictive pericarditis [12]. In a study of 1184 patients with pericarditis by Sagristà-Sauleda et al., 18% had clinical tamponade, and 1% had effusive-constrictive pericarditis. In the 7 patients with idiopathic effusive-constrictive pericarditis, 4 required pericardiectomy [10].

NSAIDs, aspirin, and colchicine remain the standard of care for the treatment of idiopathic or viral acute pericarditis [13,14]. Corticosteroid treatment can yield rapid response in acute pericarditis, but several studies have raised concern about risk of recurrence following such therapy, although evidence is still limited [9]. It is postulated that corticosteroids suppress clearance of the underlying virus and thus promote recurrence. Although there is considerable hesitancy to treat acute pericarditis with steroids, there are reports of definitive resolution of