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Case Report

Crohn's colitis-induced myocarditis

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ABSTRACT

Myocarditis can be idiopathic or arise in response to numerous systemic insults. Myocarditis occurring in the setting of an exacerbation of inflammatory bowel disease is a rare extra-intestinal manifestation of both ulcerative and Crohn's-related colitis. Here, we present a unique case of a 56-year-old female patient presenting with an acute Crohn's colitis flare that was eventually complicated by myocarditis. Our case is unique in that we clearly delineate the clinical course and development of myocarditis in a patient with focal myocardial inflammation in a pattern that is atypical for myocarditis.

<**Learning objective:** To illustrate the clinical course and resolution, along with cardiac magnetic resonance imaging and echocardiographic findings, of Crohn's-induced myocarditis.>

Introduction

Extra-intestinal manifestations of inflammatory bowel disease (IBD) are common and occur in nearly 25–30% of patients [1]. Extra-intestinal complications of IBD include: musculoskeletal, ocular, dermatologic, hepatobiliary, immunologic, hematologic, renal/urologic, pulmonary, and cardiac disorders. Myocarditis is a rare extra-intestinal cardiac complication of IBD. Registry data reported 6 cases of myocarditis in over 15,000 patients [2]. Interestingly, the incidence of myocarditis was slightly higher among patients with IBD relative to the general population [2]. Ulcerative colitis-induced myocarditis has been documented previously [3]. Also, fulminant myocarditis in association with active Crohn's disease has been described [4,5]. We present a case of focal myocarditis occurring secondary to an acute flare of Crohn's colitis. This case is unique in that we illustrate the clinical manifestation and course of myocarditis in a patient with a Crohn's flare. Also, we display atypical cardiac magnetic resonance imaging (MRI) features of myocarditis and illustrate regional wall motion abnormalities and mild global left ventricular systolic dysfunction on echocardiography. To our knowledge, this is the first such case of its type.

Case report

A 56-year-old female with a history of Crohn's disease (not on active maintenance treatment) presented complaining of diffuse abdominal pain (relieved with bowel movements, worsened with eating), diarrhea (5–6 loose bowel movements daily), and bright red blood per rectum for the previous 2 weeks. On day 0 (day of admission), she underwent computed tomography (CT) of the abdomen/pelvis which revealed mural thickening and pericolonic stranding of the ascending colon and cecum as well as mild mural thickening of the descending colon (Fig. 1). On day 2, she underwent MR abdomen enterography which revealed diffuse wall thickening, mucosal hyperenhancement, and surrounding inflammatory changes suggestive of active bowel disease as well as associated acute on chronic inflammation of the cecum, ascending colon, and proximal and mid transverse colon (Fig. 2). The findings on CT and MR were collectively consistent with active Crohn's colitis. Relevant laboratory results revealed white blood cell (WBC) count 13,700 cells/μl, hemoglobin 12.7 g/dL, and platelets 436,000 cells/μl, C-reactive protein (CRP) 22.7 mg/dL (normal 0–0.5 mg/dL), and erythrocyte sedimentation rate (ESR) 47 mm/hr (normal <20 mm/hr). Accordingly, she was diagnosed with an acute Crohn's flare and was treated with solumedrol IV 20 mg BID.

On hospital day 3, she complained of sudden onset, pleuritic chest pain, which she described as a "net-like pain" around her heart with radiation to her back and shoulders bilaterally. It lasted for roughly 10 minutes before self-resolving. Her d-dimer was

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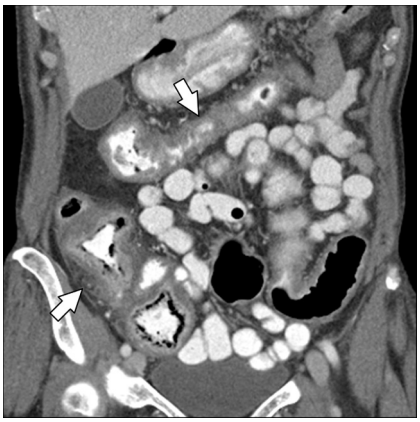

Fig. 1. Coronal reformatted contrast-enhanced computed tomography shows abnormal thickening of the ascending and transverse colon.

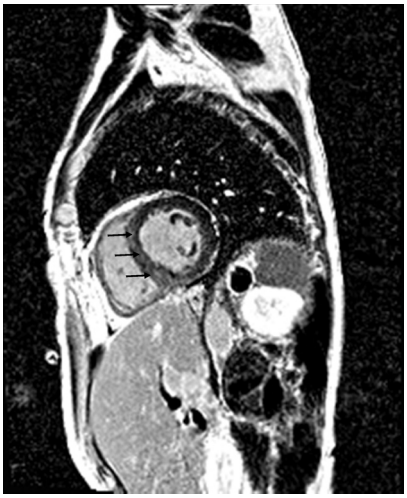

Fig. 3. Cardiac magnetic resonance imaging delayed gadolinium enhancement short-axis view illustrating patchy inflammation within the inferoseptum and anteroseptum (black arrows) in patient with Crohn's colitis flare-induced focal myocarditis.

mildly elevated at 233 ng/mL (range: 0–230 ng/mL). On examination, she was afebrile, normocardic, and normotensive. Physical examination was unremarkable. Electrocardiography revealed normal sinus rhythm, normal intervals, voltage criteria for left ventricular hypertrophy, without ST-T wave changes. Laboratory results revealed troponins of 1.14 ng/mL and 1.63 ng/mL (peak), creatine kinase-MB 2.1 ng/mL, and B-type natriuretic peptide 166 pg/mL (normal 0.2–100 pg/mL). She was transferred to the coronary care unit for presumed non-ST elevation myocardial infarction and treated with ticagrelor, aspirin, and heparin. During continuous telemetry monitoring, she had no atrial or ventricular arrhythmias. Chest X-ray showed clear lung fields and a small left-sided pleural effusion. The initial transthoracic echocardiogram (TTE) revealed a left ventricular ejection fraction (LVEF) of 45–50% and mild systolic LV dysfunction with basal to mid anteroseptal, inferoseptal, and mid inferolateral hypokinesis (Video Clip 1). On day 4, a coronary angiogram displayed angiographically normal coronaries, left heart catheterization revealed normal left ventricular end-diastolic pressure, and left ventriculogram showed normal LV systolic function without regional wall motion abnormalities. On day 5, cardiac MRI showed severe hypokinesis of the basal to mid anteroseptal LV. Delayed gadolinium enhancement (DGE) was notable for elevated extracellular volume in the septum (35–36%, normal 25%), patchy midwall (with sparing of the endocardium), delayed enhancement in the mid anteroseptal and inferoseptal LV in a non-vascular pattern suggestive of myocarditis, and normal pericardial thickness and enhancement (Fig. 3). Cardiac MRI T1 mapping revealed elevated T1 values in the mid septum due to myocardial edema (Fig. 4). Cardiac MRI T2 mapping illustrated high T2 values in the inferoseptum (62) and normal T2 values in the LV apical septum (40) and basal anterolateral LV (42) (Fig. 5A). Dark blood short T1 inversion recovery (STIR) short axis revealed increased signal intensity in mid septum illustrating myocardial edema (Fig. 5B). Collectively, this pattern of DGE, elevated extracellular volume, as well as elevated T1 and T2 values characterize the key cardiac MRI features of myocarditis [6]. Liver function (aspartate transaminase, alanine transaminase, alkaline phosphatase, total bilirubin, unconjugated/conjugated bilirubin) and thyroid function (thyroid stimulating hormone) tests were normal. Her *Clostridium difficile*, stool, and blood cultures were all negative. Stool ova and parasite examination was negative. Cytomegalovirus (CMV) viral load polymerase chain reaction was < 137 IU/mL (negative) as were her hepatitis serologies. Quantiferon gold analysis was indeterminant, but chest X-ray showed no infiltrate.

Given the amalgam of clinical, biochemical, and imaging data and the clinical time course presented above, we believe this is a

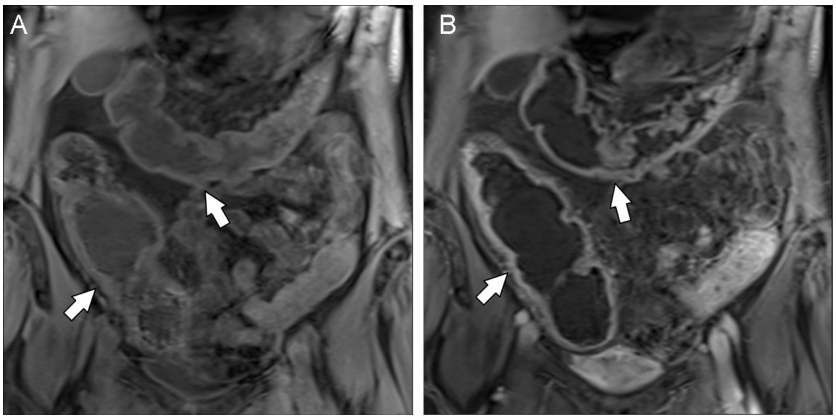

Fig. 2. (A) Coronal fat suppressed magnetic resonance imaging T1-weighted image shows abnormal thickening of the ascending and transverse colon. (B) Following IV contrast, there is enhancement of the wall and vasa recta from active inflammation consistent with colitis.