Uncommon Alliance: A Case Report on Concurrent Ulcerative Colitis and Myocarditis

consultant360.com/case-point/uncommon-alliance-case-report-concurrent-ulcerative-colitis-and-myocarditis

Case in Point

Introduction. Myocarditis is an inflammatory injury to the myocardial or pericardial layers of the heart that typically presents within one month of the inciting event. Symptoms are characterized by sudden onset of chest pain, shortness of breath, and palpitations[1]. The clinical presentation ranges from mild disease to severe complications, including cardiogenic shock and sudden cardiac death. There are several known causes of myocarditis including viruses, autoimmune disorders, and drugs or toxins[2]. The definitive diagnostic approach to myocarditis is endomyocardial biopsy, though non-invasive testing with cardiac magnetic resonance imaging (c-MRI)[1] is commonly used.

Prognosis varies by the severity of left ventricular dysfunction (LVEF) at presentation. Most patients with mild dysfunction (LVEF = 40%-50%) recover fully, but 50% of patients with moderate to severe dysfunction (LVEF < 35%) progress to chronic heart failure, and only 25% of these patients recover fully.[2] Thus, myocarditis is a serious cardiac condition, and clinicians must be vigilant in patients presenting with nonspecific cardiac symptoms. Treatment depends on the cause of injury and the severity of cardiac dysfunction but may include immunosuppression through steroid or biologic therapy[2]. A timely diagnosis is important so proper management can occur.

Patients with inflammatory bowel disease (IBD) are at higher risk of myocarditis compared to the general population[3]. In a Swedish study of 83,264 cases of IBD, the long-term risk of myocarditis was found to be higher compared to the general population over a 20-year follow-up period. During a median follow up of 12 years, there were 256 cases of myocarditis in those with IBD. This increased risk was found 20 years after date of diagnosis, corresponding to 1 additional case of myocarditis per 735 patients with IBD compared to the general population. This increased risk was seen in patients with IBD, irrespective of their specific diagnosis (Crohn's versus ulcerative colitis [UC])[4]. In patients with IBD, myocarditis arises from three potential etiologies: drug-induced myocarditis (e.g., 5-ASA therapy), viral infection, or as an extraintestinal manifestation (EIM) of IBD.[3]

Here, we present a case of a young man with UC who transferred from an outside hospital in cardiogenic shock, ultimately diagnosed with acute myocarditis. This case is important because it demonstrates an initial presentation that was consistent with the three most common causes of IBD myocarditis: he had been on 5-ASA therapy, he had a viral

prodrome, and he was in the middle of a flare of his UC. This case also illustrates that presentations of cardiogenic shock due to IBD myocarditis should be closely monitored as their clinical courses have been shown to be more severe[4].

Case Presentation. A 28-year-old man with a history of UC (diagnosed in 2022), and no prior cardiac history, presented to an outside hospitalwith chest pain and shortness of breath. One week prior, he was admitted for a UC flare where he was treated with steroids. He was discharged home with a prednisone taper and his home balsalazide dose.

Several days after discharge, he developed intermittent fevers to 101°F, a productive cough, emesis, and bloody diarrhea. He tried over-the-counter decongestants without relief. On the day of presentation, he awoke suddenly with dyspnea, orthopnea, and left-sided chest pain. In the emergency department, his vitals showed a blood pressure of 89/60, heart rate in the 130s, respiratory rate of 24, O2 saturation of 85% on room air, and a temperature of 99.1 °F. A transthoracic echocardiogram (TTE) showed severely reduced systolic dysfunction (ejection fraction [EF] = 20%). The patient was transferred to our institution for escalation of care.

Upon transfer, the patient was in cardiogenic shock and on dobutamine 10 mcg/kg/min. Infectious work-up was unremarkable, including blood and urinary cultures. A respiratory polymerase chain reaction panel was also negative. His laboratory tests **(Table 1)** were notable for:

- Erythrocyte sedimentation rate (ESR): >119 mm/h
- C-reactive protein (CRP): 262.2 mg/L
- Troponin: 0.54 ng/mL
- B-type natriuretic peptide (BNP): 2302 pg/mL

Table 1: Admission laboratory tests. Initial laboratory tests upon transfer from outside hospital. Data outside the normal range are highlighted in red.

Admission laboratory test	Test results (range)
Sodium	137 (Normal range; 136-144)
Potassium	3.2 (Normal range; 3.3-5.1)
Chloride	100 (Normal range; 98-108)
Carbon dioxide	24 (Normal range; 20-32)

Blood urea nitrogen	7 (Normal range; 7-22)
Creatinine	0.68 (Normal range; 0.6-1.4)
Estimated glomerular filtration rate	130 (Normal range; l > 59)
BNP	2302 (Normal range; 1-100)
Calcium	7.6 (Normal range; 8.9-10.3)
White blood cells	13.6 (Normal range; 3.5-10.5)
Hemoglobin	10.4 (Normal range; 13-17.5)
Platelet count	222 (Normal range; 150-400)
Albumin blood test	2.6 (Normal range; 3.6-5.0)
Total protein	6.1 (Normal range; 6.5-8.3)
Alkaline phosphatase	38 (Normal range; 30-110)
Aspartate aminotransferase	23 (Normal range; 10-40)
Alanine aminotransferase	15 (Normal range; 15-45)
Total bilirubin	0.5 (Normal range; 0.2-1.4)
Prothrombin time	9.6 (Normal range; 9.5-13.6)
Troponin	0.54 (Normal range; < 0.4)

The patient's electrocardiogram was notable for sinus tachycardia. The TTE was notable for LV enlargement, severe reduction in left EF (30%), reduced right systolic function, mild mitral regurgitation, and mild tricuspid regurgitation. Right and left heart catheterization (RHC/LHC)

showed normal filling pressures and coronary arteries. The patient was on dobutamine 10 mcg/kg/min at the time of the studies. An endomyocardial biopsy was also completed which showed active myocarditis with mixed lymphohistiocytic and neutrophilic inflammation with multiple foci of myocyte injury. After 5 days, he underwent cardiac magnetic resonance imaging, which showed an EF of 49% and findings suggestive of interstitial fibrosis and consistent with myocarditis.

Due to his history of UC, the concern for drug-induced myocarditis and extraintestinal manifestation of UC were raised. Gastroenterologists recommended discontinuation of his balsalazide, and he started on 40 mg daily of prednisone. His bloody bowel movements resolved during the next few days. In the cardiac care unit, he was weaned off dobutamine with oral afterload reduction, with resolution of his dyspnea and chest pain. Ultimately, he was discharged home 8 days after presentation on the following guideline-directed medical therapy: metoprolol 25 mg daily, lisinopril 10 mg daily, spironolactone 12.5 mg daily, dapagliflozin 10 mg daily.

Follow-up TTE 3 months after discharge showed ejection fraction of 55% with mild left atrial enlargement. At 4 months post discharge, he was back to his normal exercise capacity. His UC therapy was changed to vedolizumab with resolution of his GI symptoms.

Discussion. Acute myocarditis can be a life-threatening cardiac condition. Common causes include viruses, autoimmune disorders, and drugs or toxins (particularly medications containing 5-ASA)[2]. The pathogenesis of viral myocarditis is thought to be from immune cell over-activation leading to pathogenic remodeling of the myo- or epicardium[2]. Balsalazide, a commonly prescribed first-line therapy for UC, has also been associated with reports of myocarditis[5]. The mechanism of injury is unclear, although there is suspicion of cell-mediated hypersensitivity[5].

In addition to viral and drug-induced myocarditis, EIM is also a consideration when patients with IBD present with symptoms of myocarditis. More than 30% of patients with IBD present with EIM manifestations of their disease, and myocarditis is one that can be overlooked in this population[6].

EIM may be associated with an ongoing intestinal flare (commonly seen with erythema nodosum)[7]. However, some EIM can occur independent of intestinal symptoms (such as ankylosing spondylitis)[7]. It is unclear whether myocarditis is associated with uncontrolled IBD or can present independent of an intestinal flare. Approximately 6 in 15,000 patients with IBD can suffer from myocarditis as an EIM[6]. Studies have shown that patients with IBD have an increased risk of myocarditis compared with those of the general population, even up to 20 years after diagnosis[4]. One proposed mechanism includes upregulation of the interleukin-23 axis in IBD, disrupting the “gut-heart” axis and inhibiting accumulation of regulatory T-cells[4].

The diagnostic and treatment guidelines of myocarditis as an EIM are still very limited. Gruenhagen and colleagues[8] treated a simultaneous exacerbation with prednisone and mesalamine with resolution of both GI and cardiac symptoms. In our patient, his balasalzide was held, and he was treated with prednisone for his IBD flare, with plans to initiate biologic therapy as an outpatient. The biopsy results did not show a definitive cause of his myocarditis, as both EIM and drug-induced myocarditis indicated lymphocytic predominant myocarditis. It is unknown whether our patient would still tolerate balsalazide therapy going forward.

As stated, there have been reports of myocarditis as an EIM of IBD, but this is one of the first case reports to have a patient present with myocarditis as the possible result of a recent IBD flare. Additionally, he had a viral prodrome and had been on 5-ASA therapy for his UC. This case illustrates the differential for myocarditis that clinicians must consider in patients with IBD. Clinicians must be vigilant in patients with IBD who present with chest pain and should consider a more extensive cardiac work-up if warranted, including routine electrocardiograms and echocardiograms. Furthermore, if these patients present with chest pain, further reconciliation of their IBD medications must be discussed, especially if they are on 5-ASA therapy. Further research should investigate the diagnostic and treatment strategies of myocarditis as an EIM of IBD.

AUTHORS:
Daniel Sweeney MD[1] • Mingxi Dennis Yu MD[1]

AFFILITATIONS:
Loyola University Medical Center, Maywood, United States of America

CITATION:
Uncommon alliance: a case report on concurrent ulcerative colitis and myocarditis. *Consultant.* Published online xx. doi:xx
Received August 23, 2024. Accepted December 11, 2024.

DISCLOSURES:
The authors report no relevant financial relationships.

ACKNOWLEDGEMENTS:
None.

CORRESPONDENCE:
Daniel Sweeney, MD, 2160 S 1st Ave, Maywood, IL 60153 (daniel.sweeney@luhs.org)

References

1. Ammirati E, Moslehi JJ. Diagnosis and treatment of acute myocarditis: a review. *JAMA.* 2023;329(13):1098-1113. doi:10.1001/jama.2023.3371.
2. Olejniczak M, Schwartz M, Webber E, Shaffer A, Perry TE. Viral myocarditis—incidence, diagnosis, and management. *J Cardiothorac Vasc Anesth.* 2020;34(6):1591-1601. doi:10.1053/j.jvca.2019.12.052.
3. Wang YY, Shi W, Wang J, Li Y, Tian Z, Jiao Y. Myocarditis as an extraintestinal manifestation of ulcerative colitis: a case report and review of the literature. *World J Clin Cases.* 2022;10(27):9750-9759. doi:10.12998/wjcc.v10.i27.9750.
4. Sun J, Yao J, Olén O, et al. Long-term risk of myocarditis in patients with inflammatory bowel disease: a nationwide cohort study in Sweden. *Am J Gastroenterol.* 2024;119(9):1866-1874. doi:10.14309/ajg.0000000000002701.
5. Radhakrishnan ST, Mohanaruban A, Hoque S. Mesalazine-induced myocarditis: a case report. *J Med Case Rep.* 2018;12(1):44. doi:10.1186/s13256-017-1557-z.
6. Caio G, Lungaro L, Caputo F, et al. Recurrent myocarditis in a patient with active ulcerative colitis: a case report and review of the literature. *BMJ Open Gastroenterol.* 2021;8(1):e000587. doi:10.1136/bmjgast-2020-000587.
7. Malik TF, Aurelio DM. Extraintestinal manifestations of inflammatory bowel disease. *StatPearls.* Published March 6, 2023. Accessed March 2024. https://www.ncbi.nlm.nih.gov/books/NBK568797/.
8. Gruenhagen B, Alraies MC, Vakil KP, March SK. Ulcerative colitis-induced myocarditis. *BMJ Case Rep.* 2014;2014:bcr2014204818. doi:10.1136/bcr-2014-204818.