Journal of Community Hospital Internal Medicine Perspectives

Volume 13 | Issue 4 Article 12

2023

Myocarditis With Cardiogenic Shock In A Young Female With Severe Ulcerative Colitis Flare

Ramez Alyacoub
Winchester Medical Center, Winchester Virginia, alyacoubramiz@yahoo.com

Harshwardhan Khandait
Department Of Internal Medicine, RWJBH/ Trinitas Regional Medical Center, Elizabeth NJ

Hardik Fichadiya
Department Of Internal Medicine, RWJBH/ Trinitas Regional Medical Center, Elizabeth NJ

Meherwan Joshi
Department Of Cardiology, RWJBH/ Trinitas Regional Medical Center, Elizabeth NJ

Recommended Citation

Alyacoub, Ramez; Khandait, Harshwardhan; Fichadiya, Hardik; and Joshi, Meherwan (2023) "Myocarditis With Cardiogenic Shock In A Young Female With Severe Ulcerative Colitis Flare," *Journal of Community Hospital Internal Medicine Perspectives*: Vol. 13: Iss. 4, Article 12.
DOI: 10.55729/2000-9666.1209
Available at: https://scholarlycommons.gbmc.org/jchimp/vol13/iss4/12

CASE REPORT

Myocarditis With Cardiogenic Shock in a Young Female With Severe Ulcerative Colitis Flare

Ramez Alyacoub [a,b,*], Harshwardhan Khandait [b], Hardik Fichadiya [b], Meherwan Joshi [c]

[a] Winchester Medical Center, Winchester, VA, USA
[b] Department of Internal Medicine, RWJBH/ Trinitas Regional Medical Center, Elizabeth, NJ, USA
[c] Department of Cardiology, RWJBH/ Trinitas Regional Medical Center, Elizabeth, NJ, USA

Abstract

Myocarditis is a rare extraintestinal manifestation of inflammatory bowel disease. Myopericarditis-associated inflammatory bowel disease can be a side effect of the medications used to treat inflammatory bowel disease or the disease process. We present a 25-year-old female with history of ulcerative colitis presented with abdominal pain associated with sharp and central chest pain. She was in a flare of ulcerative colitis with bloody diarrhea. She developed shock and was in intensive care unit. Echocardiogram showed reduced ejection fraction and pericardial effusion. Coronary artery disease, sepsis, thyroid disease were ruled out. She was treated with systemic antibiotics, intravenous steroids, and guideline-directed medical therapy for presumed ulerative colitis associated with myopericarditis and had symptomatic improvement. Treatment of IBD-associated myopericarditis includes the standard induction treatment for IBD with steroids and guideline-directed medical therapy for heart failure.

Keywords: Heart failure, Inflammatory bowel diseases, Myocarditis

1. Background

Myocarditis is an inflammatory disease that affects cardiac myocytes. It has a wide range of clinical presentations with different etiologies. It can be caused by infectious agents, medications, toxins, hypersensitivity, or systemic inflammatory syndromes. Inflammatory bowel disease has a wide range of extraintestinal manifestations. Reported cardiovascular manifestations include pericarditis, myocarditis, endocarditis, valvulopathies, arterial and venous thromboembolism, and coronary artery disease.[1] Myocarditis is a rare extraintestinal manifestation of inflammatory bowel disease; Incidence of myocarditis is rising and is reported to be 25 per 100,000 hospitalizations in 2019.[2] Myopericarditis-associated inflammatory bowel disease can be a side effect of the medications used to treat inflammatory bowel disease or the inflammatory disease process itself. Here we present a 25-year-old female patient with a severe flare of Ulcerative Pancolitis with backwash ileitis complicated with myopericarditis.

2. Case presentation

A 25-year-old female with a medical history of ulcerative colitis (UC), idiopathic thrombocytopenic purpura (ITP), and hypothyroidism presented to the hospital with complaints of chest pain, described as sharp central, non-exertional. She also complained of abdominal, bloody diarrhea (up to 6 episodes per day) and a low-grade fever. On examination, vitals were HR 120 bpm, max temp 101 F, RR 20, and BP 100/50. Lung fields were clear. The cardiac exam revealed a 2/6 systolic murmur but no JVD or pericardial rub. The abdomen was soft and non-tender, normoactive bowel sounds were heard, and there was no organomegaly. There was a trace of bilateral symmetrical pitting edema. She had a cesarean delivery two months ago. Home medications included mesalamine as needed and levothyroxine. She had no recent vaccinations.

Received 16 February 2023; revised 14 April 2023; accepted 21 April 2023.
Available online 29 June 2023

* Corresponding author at. Department Of Internal Medicine, RWJ-BH/ Trinitas Regional Medical Center, 228 Woodberry Ln, apt 134, Winchester, VA, 22601, USA.
E-mail address: Alyacoubramiz@yahoo.com (R. Alyacoub).

https://doi.org/10.55729/2000-9666.1209

On admission, initial laboratory findings are presented in Table 1. Stool culture and stool Clostridioides difficile were negative. TSH and fT4 were normal. She tested negative for COVID-19 and influenza. EKG showed sinus tachycardia and nonspecific T wave changes. Chest X-ray was normal (Fig. 1A). CT pulmonary angiogram was negative for pulmonary embolism. The patient was given a 2 L fluid bolus in the emergency department due to concerns of hypovolemia and possible sepsis in the setting of diarrhea and ulcerative colitis. Her potassium was replaced. She was admitted to the telemetry unit. Empiric antibiotics with Vancomycin and Piperacillin-tazobactam were also started.

The patient developed worsening hypotension and did not respond to fluids. She was transferred to the ICU for vasopressor support. Workup for sources of sepsis, including blood cultures and stool studies, was negative. BNP was 1095 pg/ml, ESR 60 mm/h, and CRP 26.6 ng/dl. The patient also developed jugular venous distension. An echocardiogram showed reduced LV systolic function (LVEF 35%) with moderately dilated and hypokinetic RV and elevated pulmonary artery pressures, normal ventricular wall thickness, small pericardial effusion, diastolic grading could not be performed due to severe fusion of E/A waves. Repeat CXR showed bilateral perihilar and lower lobe alveolar infiltrates (Fig. 1B). CT abdomen and pelvis showed moderate right-sided and small left-sided pleural effusion, bilateral consolidations, and colon wall thickening (Fig. 2).

Table 1. Initial Laboratory studies with reference range.

Laboratory investigation	Lab value	Reference range
Hb	11.7 g/dl	12–16
WBC	8.7 K/UL	4-10 k/L
Platelets	324 × 10^9/L,	250–450 × 10^9/L
AST	71 U/L	15–41 U/L
ALT	29 U/L	14–54 U/L
ALK	127 U/L	38–126 U/L
BUN	7 mg/dl	8–20 mg/dl
Cr	0.99 mg/dl	0.4–1 mg/dl
K	2.8 mmol/L	3.5–5 mmol/L
Na	138 mmol/L	135–145 mmol/L
Albumin	2.6 g/dl	3.5–5.5 g/dl
Lactic acid	2.3 mmol/l	0.5–2 mmol/l
troponin	0.47 ng/ml (0.00 ng/ml 4 days prior)	<0.5 ng/ml
D-dimer	361 ug/L	<230 ug/L DDU

In the ICU, she required Norepinephrine for vasopressor support briefly. The patient was started on colchicine and ibuprofen for concerns of pericarditis. She was started on Lasix 20 mg twice a day for heart failure and volume overload. Guideline-directed medical therapy was slowly introduced with metoprolol, ivabradine, and ramipril. Methylprednisone 60 mg intravenous daily was started for UC flare after ruling out infectious colitis. Cardiac catheterization showed normal coronary arteries (Fig. 3), ruling out coronary artery disease. A cardiac MRI was not done due to unavailability in our institution. Colonoscopy showed diffusely friable mucosa in the sigmoid colon, transverse colon, ascending colon, rectum, and cecum with evidence of backwash ileitis. Biopsy confirmed pancolitis with backwash ileitis and was negative for CMV. Testing for hepatitis panel, as well as Quantiferon for TB, were negative. She was not a candidate for TNF alpha inhibitors due to heart failure. Therefore, she received one dose of Vedolizumab. A repeat echocardiogram showed improvement in LV function and normalization of PA pressures. Ibuprofen was stopped as it is associated with worsening colitis.

The patient met the criteria for clinically suspected myocarditis and was diagnosed with myopericarditis due to a flare of ulcerative colitis. Her heart failure symptoms, as well as diarrhea, improved. She was discharged on colchicine for pericarditis, on guideline-directed medical therapy with metoprolol succinate, ramipril, spironolactone, and ivabradine, and on prolonged steroid taper for

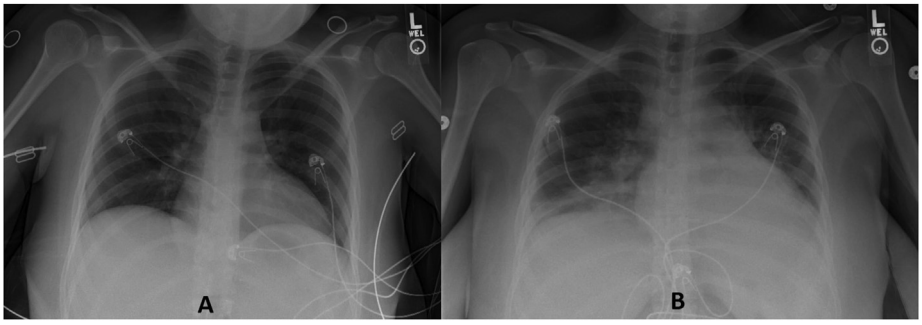

Fig. 1. A: Initial Chest X-Ray normal finndings B: Repeat Chest X-Ray showed Bilateral Alveolar infiltrates.

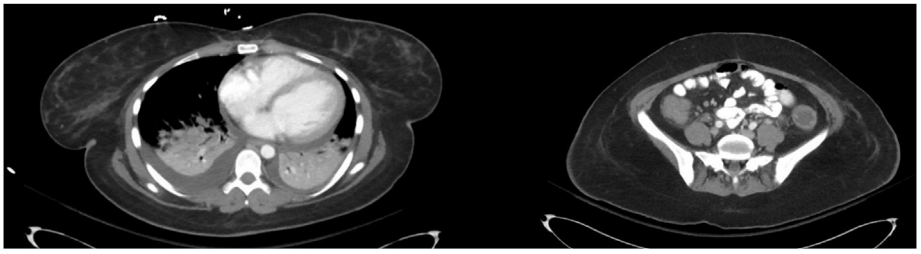

Fig. 2. CT abdomen and pelvis showed moderate right sided and small left-sided pleural effusion, bilateral consolidations, and colon wall thickening.

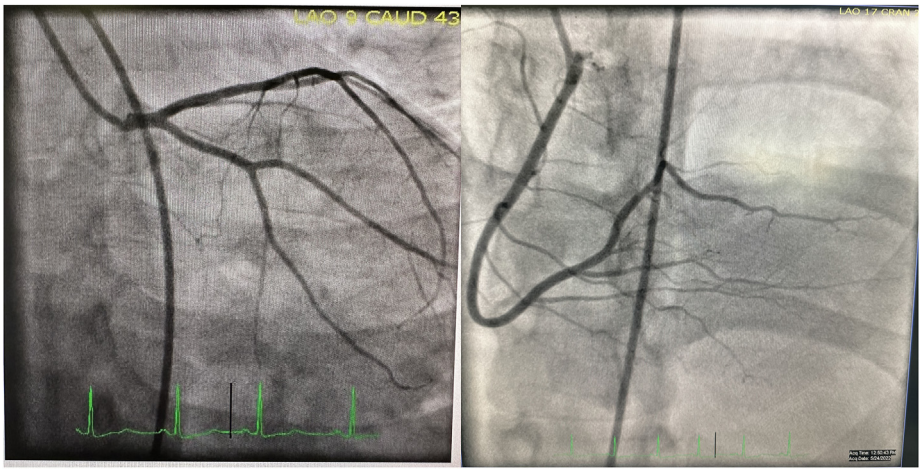

Fig. 3. Cardiac catheterization showing normal coronary arteries.

ulcerative colitis with the arrangement to continue further doses of Vedolizumab as an outpatient.

3. Discussion

Inflammatory bowel diseases are a group of disorders affecting the gastrointestinal tract. The two types include ulcerative colitis and Crohn's disease, even though they primarily affect the GI tract. IBD is multisystem inflammation with a variety of extra-intestinal manifestations. Cardiovascular manifestations of inflammatory diseases are rare and include; Pericarditis, myocarditis, heart failure, venous thromboembolism, arterial thromboembolism, arrhythmias, conduction abnormalities, and endocarditis valvulopathies.[1]

The incidence of myocarditis and pericarditis in inflammatory bowel disease is rare but has been trending. In addition, myocarditis is more common in ulcerative colitis than Crohn's disease.[2,3] The annual incidence of myocarditis in UC over five years per 100,000 hospitalizations was 25.1 in 2019 compared to 13.6 in 2016 in a study by Dasu.[2]

Myocarditis in inflammatory bowel disease has two main pathophysiologic mechanisms; the first is due to ASA medications. Proposed mechanisms include IgE–mediated allergic reactions, direct cardiac toxicity, cell-mediated hypersensitivity due to the overproduction of eosinophil-stimulating cytokines, or humoral cross-reactivity with the peri- and myocardium leading to widespread inflammation.[4] This type of myocarditis usually manifests weeks after starting therapy and resolves after stopping it. The second mechanism is related to autoimmune inflammation. IBD arises from a dysregulated immune response to gut flora and its products as a result of multiple environmental and genetic factors that impairs the mucosal barrier; this, in turn, results in the activation of T-Helper and NK cells leading to the production of TNF alpha and proinflammatory cytokines, autoantibodies.[5] This disturbance and imbalance of the immune system subsequently lead to exposure to autoantigens during the acute flare and may cause direct cytotoxicity on myocytes by activating cytotoxic T cells, causing the release of inflammatory mediators and activation of the immune system. This sequence of events can lead to acute myocarditis.[5,6] The myocarditis type secondary to UC disease activity tends to occur in patients with a long history of ulcerative colitis.

Myocarditis in inflammatory bowel disease can have various presentations similar to other etiologies of myocarditis, from shortness of breath to

fulminant heart failure and cardiogenic shock.[7] It can present with chest pain in concomitant pericarditis[8,9] and can even lead to pericardial effusion and tamponade.[10] Elevated cardiac troponin level is expected in patients with myocarditis, but the absence of such elevation does not exclude myocarditis. The ECG in patients with myocarditis may be normal or show nonspecific abnormalities. Findings may include nonspecific ST segment changes, single atrial or ventricular ectopic beats, and ventricular arrhythmias. It may also be associated with regional ST elevations and Q waves. Viral studies are not routinely obtained in the workup of myocarditis or pericarditis as they are of low diagnostic yield and do not affect management. Acute phase reactants are typically elevated, particularly in autoimmune myopericarditis. A coronary angiogram is usually necessary to exclude coronary artery disease.[11]

Diagnosing clinically suspected myocarditis requires at least one of the clinical presentations of myocarditis and at least one diagnostic criteria. However, at least two diagnostic criteria are required if the patient is asymptomatic.[11,12] Clinical presentations encompass acute chest pain (pericarditis or pseudo-ischemic); New-onset or worsening of shortness at rest or exertional, and/or fatigue, with or without signs of heart failure; Palpitations and/or unexplained arrhythmia symptoms and/or syncope, and/or aborted sudden cardiac death; Unexplained cardiogenic shock. Diagnostic criteria include electrocardiographic criteria such as findings mentioned in the prior paragraph; Elevated troponin; Functional and structural abnormalities present on cardiac imaging (can be seen on an echocardiogram, an angiogram, or Cardiovascular Magnetic Resonance); or Tissue characterization by CMR. A definitive diagnosis of myocarditis requires an endomyocardial biopsy.[12]

All patients with acute myocarditis should receive guideline-directed medical treatment for heart failure and arrhythmias, if applicable, as per AHA guidelines.[13,14] In the case of autoimmune-mediated myocarditis, steroids with or without immunosuppressive medications are the mainstay treatment. Steroids often lead to the resolution of cardiac manifestations regardless if the myocarditis was due to mesalamine or UC disease activity.[10] Colchicine and nonsteroidal anti-inflammatory drugs are also used when pericarditis is coexistent, termed myopericarditis. However, in IBD, NSAIDs are typically avoided as they are associated with worsening diarrhea and disease activity exacerbation.[15] Steroids are also part of the induction treatment for ulcerative colitis. In case of moderate-severe exacerbations of ulcerative colitis with inadequate response to steroids in 5–7 days, TNF alpha inhibitors are typically used. However, they are contraindicated in patients with reduced ejection fraction,[16] which makes managing such patients challenging. Besides TNF inhibitors, the monoclonal antibody Vedolizumab which binds integrin resulting in gut-selective anti-inflammatory activity is an option for steroid-refractory cases in which TNF inhibitors are ineffective or contraindicated.[17] Vedolizumab was used in our patient as well as in a similar case of a young male with myocarditis and ulcerative colitis by Caio.[18]

The timing of the patient's presentation coincided with an acute episode of colitis. Her myocarditis and bowel symptoms improved after initiating steroids and heart failure therapy. A repeat echocardiogram showed that left ventricular systolic function and pulmonary artery pressures had normalized. This suggests that the acute increase in pulmonary pressure was Group 2 pulmonary hypertension secondary to underlying myocarditis. Sepsis work up was unremarkable and thyroid tests were normal. Another differential to consider in this case was perinatal myocarditis, as she had delivered two months before her presentation. Delivery was at 35 weeks gestation. However, her presentation with chest pain and low-level troponin leakage during an acute flare of ulcerative colitis and response to steroids makes myopericarditis secondary to her Ulcerative Colitis the likely diagnosis.

4. Conclusions

New onset chest pain or shortness of breath may indicate myopericarditis in patients with ulcerative colitis. Diagnosis is by applying diagnostic criteria of myocarditis and exclusion of other causes of myocarditis. Early recognition and management of this rare complication are essential to avoid associated life-threatening complications.

Financial support

None declared.

Patient permission/consent declarations

Patient gave consent to publication of information present in case report.

Conflict of interest

None declared.

Acknowledgement

Work was done in RWJBH/Trinitas Regional Medical Center. Author Ramez Alyacoub was affiliated with RWJBH/Trinitas Regional Medical Center at the time of work but now affiliated with Winchester Medical Center.

References

1. Bunu DM, Timofte CE, Ciocoiu M, et al. Cardiovascular manifestations of inflammatory bowel disease: pathogenesis, diagnosis, and preventive strategies. *Gastroenterol Res Pract*. 2019;2019, 3012509.
2. Dasu N, Khalid Y, Blair B, Chhoun C, Joo L. Complications of myocarditis in inflammatory bowel disease (IBD): a population-based comparative study between ulcerative colitis and crohn's disease. *Inflamm Bowel Dis*. 2022;28(Supplement_1): S38−S39.
3. Patil S, Gonuguntla K, Kumar M, et al. Myocarditis complicating inflammatory bowel disease: a comparison between ulcerative colitis and crohn's disease from the national inpatient sample. *J Am Coll Cardiol*. 2020;75(11):1077.
4. Mitchell NE, Harrison N, Junga Z, Singla M. Heart under attack: cardiac manifestations of inflammatory bowel disease. *Inflamm Bowel Dis*. 2018;24(11):2322−2326.
5. Bracamonte-Baran W, Čiháková D. Cardiac autoimmunity: myocarditis. *Adv Exp Med Biol*. 2017;1003:187−221.
6. Bruestle K, Hackner K, Kreye G, Heidecker B. Autoimmunity in acute myocarditis: how immunopathogenesis steers New directions for diagnosis and treatment. *Curr Cardiol Rep*. 2020; 22(5):28.
7. Varnavas VC, Reinsch N, Perrey M, et al. Recurrent lymphocytic myocarditis in a young male with ulcerative colitis. *Eur J Med Res*. 2014;19(1):11.
8. Buckholz A, Shen NT, Gupta V, Abramowitz M. Myocarditis in a patient with ulcerative colitis: an uncommon but important manifestation of disease or drug effect. *Am J Gastroenterol*. 2017;112:S1104−S1105.
9. Freeman HJ, Salh B. Recurrent myopericarditis with extensive ulcerative colitis. *Can J Cardiol*. 2010;26(10):549−550.
10. Bansal D, Chahoud G, Ison K, et al. Pleuropericarditis and pericardial tamponade associated with inflammatory bowel disease. *J Ark Med Soc*. 2005;102(1):16−19.
11. Lampejo T, Durkin SM, Bhatt N, Guttmann O. Acute myocarditis: aetiology, diagnosis and management. *Clin Med*. 2021 Sep;21(5):e505−e510.
12. Caforio AL, Pankuweit S, Arbustini E, et al. Current state of knowledge on aetiology, diagnosis, management, and therapy of myocarditis: a position statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. *Eur Heart J*. 2013;34(33):2636−2648d.
13. Bozkurt B, Colvin M, Cook J, et al. Current diagnostic and treatment strategies for specific dilated cardiomyopathies: a scientific statement from the American heart association [published correction appears in circulation. *Circulation*. 2016; 134(23):e579−e646.
14. Tschöpe C, Cooper LT, Torre-Amione G, Van Linthout S. Management of myocarditis-related cardiomyopathy in adults. *Circ Res*. 2019;124(11):1568−1583.
15. Kvasnovsky CL, Aujla U, Bjarnason I. Nonsteroidal anti-inflammatory drugs and exacerbations of inflammatory bowel disease. *Scand J Gastroenterol*. 2015;50(3):255−263.
16. Chung ES, Packer M, Lo KH, Fasanmade AA, Willerson JT. Anti-TNF Therapy against Congestive Heart Failure Investigators. Randomized, double-blind, placebo-controlled, pilot trial of infliximab, a chimeric monoclonal antibody to tumor necrosis factor-alpha, in patients with moderate-to-severe heart failure: results of the anti-TNF Therapy against Congestive Heart Failure (ATTACH) trial. *Circulation*. 2003; 107(25):3133−3140.
17. Wyant T, Fedyk E, Abhyankar B. An overview of the mechanism of action of the monoclonal antibody Vedolizumab. *J Crohns Colitis*. 2016 Dec;10(12):1437−1444.
18. 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.