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Myopericarditis and Intracavitary Thrombus as a Manifestation of Ulcerative Colitis: a case report

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Case Report

Keywords: Colitis, Ulcerative, Myocarditis, Thrombosis, Inflammatory Bowel Diseases, Case Reports

Posted Date: April 28th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-9351588/v1

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Additional Declarations: No competing interests reported.

Abstract

Background: Ulcerative colitis (UC) is a chronic inflammatory bowel disease with a systemic nature, primarily affecting the rectum and colon. However, it can also manifest with extraintestinal symptoms. In rare cases, there may be cardiac involvement, such as myopericarditis, which is a rarely described complication. The diagnosis of this condition poses a clinical challenge and requires specialised imaging studies for confirmation and timely management.

Case presentation: A 26-year-old female with a 5-year history of ulcerative colitis presented with a severe relapse, characterised by bloody diarrhea, weight loss, abdominal and chest pain, as well as joint symptoms. During the patient's hospitalisation, she developed hypotension, fever, and elevated cardiac troponin levels (1.078 ng/L). Cardiac magnetic resonance confirmed the presence of myopericarditis with right ventricular dysfunction and a right atrial thrombus. The patient was successfully treated with intravenous methylprednisolone, enoxaparin, colchicine, and infliximab. This case illustrates a rare extraintestinal manifestation and highlights the importance of considering cardiac complications in patients with inflammatory bowel disease (IBD). Furthermore, the potential roles of systemic inflammation and medication-related adverse effects in the aetiology of this complication are discussed.

Conclusions: Myopericarditis and intracavitary thrombi are uncommon and life-threatening manifestations of active UC. Clinical suspicion and advanced imaging such as cardiac magnetic resonance are essential for timely diagnosis and management.

Background

Ulcerative colitis (UC) is an inflammatory disease that affects the rectum and colon, with variable extent. It can result in significant long-term complications and is often associated with an impaired quality of life. Research indicates that the prevalence of UC is comparable in men and women. However, some studies suggest that this may be slightly higher in men. The diagnosis is most frequently made between the second and fourth decades of life, although it can occur at any age. The clinical manifestations may be gastrointestinal or extraintestinal (1).

With regard to the gastrointestinal level, symptoms are dependent on the location and degree of intestinal inflammation. Patients commonly present with symptoms such as hematochezia or rectal bleeding (up to 90%), decreased stool consistency, increased stool output (in more than 90%), rectal tenesmus, and abdominal pain (2).

Extraintestinal manifestations occur in approximately one-quarter of patients and may be dependent or independent of intestinal activity. Common presentations include peripheral arthritis (11%), primary sclerosing cholangitis (2.5%), and ocular manifestations such as episcleritis or uveitis (2%) (2). Cardiac involvement is less common. Myocarditis has been described as an extraintestinal manifestation (3–5), but it can also result from adverse effects of medications used in UC treatment (e.g., 5-aminosalicylates) (6–8).

However, a critical, less common feature is the systemic hypercoagulability inherent to IBD. This condition is driven by an inflammatory burden, where cytokines (such as tumour necrosis factor-α [TNF-α], interleukin-1β [IL-1β], and interleukin-6 [IL-6]) trigger a prothrombotic shift with platelet activation and aggregation (9). There is also an imbalance of coagulation factors (elevated fibrinogen, Factor V, Factor VIII, and von Willebrand factor) and hypofibrinolysis (enhanced by plasminogen activator inhibitor-1 and thrombin-activatable fibrinolysis inhibitor) (9).

The prevalence of thrombotic events is not clearly defined, but the risk of thrombosis in individuals with IBD is estimated to be 3 to 4 times higher than in the general population. This risk is particularly striking in young patients during active flares, potentially leading to spontaneous thrombosis in unusual locations. This report presents a case of myocarditis and intracavitary thrombus as rare, life-threatening extraintestinal manifestations of UC (10–12).

Case presentation

A 26-year-old female university student with a 5-year history of UC presented to the emergency department with a one-week history of severe relapse symptoms. The patient reported approximately six bloody stools per day, diffuse abdominal pain, and an unintentional weight loss of 10 kg over the previous six weeks. Additionally, she exhibited inflammatory arthralgia in the wrists and ankles. Notably, she complained of oppressive retrosternal chest pain for five days, which worsened with deep inspiration.

Since being diagnosed, she has been taking oral mesalazine (information regarding dosage was not available). Two months prior to this admission, she had a moderate exacerbation treated with prednisone (50 mg/day for 4 weeks) and an intensified mesalazine regimen (2 g orally every 12 hours and 500 mg rectally every 12 hours). The patient reported strict adherence to treatment and denied allergies, toxin exposure, or family history of cardiac abnormalities.

At admission, the patient's vital signs were within acceptable limits, with a blood pressure (BP) of 90/60 mmHg, heart rate of 64 beats/min, respiratory rate of 18 breaths/min, and temperature of 36.8°C. She appeared pale, with a moderately distended and tender abdomen, without signs of peritoneal irritation. At 18 hours after admission, the patient developed hypotension (BP 73/34 mmHg) and fever (38°C).

Laboratory investigations (see Table 1) revealed elevated high-sensitivity cardiac troponin (initial 0.5 ng/L, increasing to 1.078 ng/L; reference range 0–0.26 ng/L). Acute-phase reactants were found to be significantly elevated, with an erythrocyte sedimentation rate of 89 mm/h and high-sensitivity C-reactive protein of 1.32 mg/L. The complete blood count revealed anemia (hemoglobin level of 9.4 g/dL), but all other parameters were within normal limits.

Table 1
Relevant admission laboratory tests

Laboratory test (units)	Result	Reference range
White blood cells (×10³/μL)	10.28	5.0–10.0
Neutrophils (×10³/μL)	7.59	2.0–7.0
Lymphocytes (×10³/μL)	1.3	1.5–4.0
Hemoglobin (g/dL)	9.4	12.0–16.0
Platelets (×10³/μL)	630	150–450
C-reactive protein (mg/dL)	1.32	< 0.03
Erythrocyte sedimentation rate (mm/h)	80	0–15

The diagnosis was facilitated by the use of imaging studies. Although transthoracic echocardiography was limited by a poor acoustic window, it showed preserved ventricular function. Further cardiac magnetic resonance imaging (MRI) revealed late gadolinium enhancement in the basal and mid segments of the inferior and inferolateral walls, a small pericardial effusion, and an image in the right atrium suggestive of a thrombus (see Fig. 1). A colonoscopy confirmed severe active ulcerative colitis (Mayo score 3) from the sigmoid colon to the hepatic flexure (see Fig. 2).

The patient underwent a multidisciplinary evaluation by the gastroenterology, cardiology, and infectious diseases teams. The final diagnosis was severe acute ulcerative colitis with extraintestinal manifestations, including myopericarditis and an intracavitary thrombus.

Treatment was initiated with intravenous methylprednisolone, leading to progressive gastrointestinal improvement. In regard to the cardiac complications, the following therapeutic measures were initiated: anticoagulation with subcutaneous enoxaparin (1 mg/kg every 12 hours) and anti-inflammatory therapy with colchicine (0.5 mg daily).

Concomitant infections were excluded. Stool culture, a set of blood cultures, and a gastrointestinal pathogen panel were all negative. In addition, screening for human immunodeficiency virus, hepatitis B and C, syphilis by non-treponemal testing, and tuberculosis by purified protein derivative testing was negative. Biological therapy with infliximab was then initiated to manage severe ulcerative colitis activity.

The patient demonstrated a favourable response and was subsequently discharged. During a subsequent 12-week follow-up, the primary thrombophilia testing was found to be negative. Following an episode of community-acquired pneumonia, the patient made a full recovery within six months. She is currently in clinical remission and undergoing maintenance therapy with infliximab every two months. Follow-up cardiac magnetic resonance imaging revealed no gadolinium enhancement, with preserved left ventricular ejection fraction.

Discussion and Conclusions

Myopericarditis is a rare extraintestinal manifestation of UC (6–8, 13–18), characterised by a heterogeneous clinical presentation that often delays diagnosis, thereby increasing morbidity and mortality risks. In addition, intracardiac thrombosis represents an even less frequent but clinically significant complication reported in patients with active disease (3–5, 19). Several cases of cardiac involvement in UC —including both inflammatory and thrombotic manifestations—have been described in the literature (see Table 2). Notably, the present case uniquely combines both myopericardial inflammation and intracavitary thrombus formation.

Table 2
Summarizing reported cases of cardiac complications in ulcerative colitis.

Author	Year	Age (years)	Sex	IBD type / activity	Cardiac involvement	Clinical Presentation	Treatment	Outcome
Chin et al	1988	30	Male	UC (active)	LV apical thrombus (1.5 cm); normal LV size/function	Bloody diarrhea; tachycardia (120 bpm); dysarthria; right-sided weakness; cold, painful left foot	Anticoagulation: IV heparin → warfarin; popliteal embolectomy; prednisone; sulfasalazine	Thrombus resolved (3 weeks); transmetatarsal amputation; stable at discharge
Urgesi et al	2010	38	Female	UC (active)	Right ventricular thrombus; pulmonary embolism	Dyspnea; tachycardia (110 bpm); anemia (Hb 6.9 g/dL)	Anticoagulation: LMWH → warfarin; anti-TNF therapy	Thrombus resolution; clinical improvement
Freeman et al	2010	26	Male	UC (active)	Recurrent myopericarditis	Chest pain; fever; tachycardia (120 bpm); anemia (Hb 11.9 g/dL); elevated troponin; nonspecific ST-T wave changes on ECG	Corticosteroids; discontinuation of mesalamine	Resolution; recurrent episodes over time
Saleh et al	2010	39	Male	UC (active)	Large LV apical thrombus (3.5 cm); normal LV size/function	Abdominal pain; bloody diarrhea; tachycardia (108 bpm); anemia (Hb 6.9 g/dL); transient dysarthria; right-sided weakness	Anticoagulation: IV heparin → warfarin; aspirin; prednisone; mesalazine	Stable; discharged after 1 week; abdominal pain resolved; hemoglobin stabilized
Varnavas et al	2014	30	Male	UC (active)	Lymphocytic myocarditis; severe LV dysfunction (LVEF 13%); pericardial effusion	Abdominal pain; bloody diarrhea; heart failure; chest pain; elevated troponin	Inotropes; IABP; piperacillin tazobactam; prednisone; mesalazine; heart failure therapy (bisoprolol, ramipril, spironolactone)	Clinical improvement; recovery of LV function (LVEF 50%) at 6 months
Gruenhagen et al		24	Male	UC (active)	Acute myocarditis; subepicardial LGE (apical inferior and inferolateral segments)	Abdominal pain; bloody diarrhea; chest pain; dyspnea; elevated troponin	Prednisone; mesalamine	Complete symptom resolution
Willner et al	2015	21	Female	UC (active)	Multiple LV apical thrombi (1.18, 0.38, 0.35 cm); normal LV size/function	Abdominal pain; bloody diarrhea; tachycardia (120 bpm); anemia (Hb 7.24 g/dL); T-wave inversions on ECG	Anticoagulation: warfarin; prednisone; 6-mercaptopurine; hydrocortisone; metronidazole	Symptoms resolved; residual small thrombus at 1 month; complete resolution at 6 months
Kim et al	2016	28	Female	UC (active)	Fulminant myocarditis; severe LV dysfunction (LVEF 10%)	Dyspnea; tachycardia (138 bpm); elevated troponin	ECMO; methylprednisolone; infliximab	Full recovery with normalization of LV function

Author	Year	Age (years)	Sex	IBD type / activity	Cardiac involvement	Clinical Presentation	Treatment	Outcome
Murphy et al	2017	42	Male	Suspected IBD (new diagnosis)	Eosinophilic myocarditis; severe LV dysfunction (LVEF 29%); anterior pericardial effusion; LGE (mesocardial); no thrombus;	Diarrhea; weight loss; hypovolemic shock; heart failure; eosinophilia	Corticosteroids; ICD placement; heart failure therapy	Partial recovery (LVEF 42% at 1 year)
Caio et al	2021	26	Male	UC (active)	Recurrent myocarditis; LV hypokinesis (LVEF ~ 50%); myocardial oedema (T2) in apical anterior and septal segments	Fever; abdominal pain; bloody diarrhea; chest pain; dyspnea; elevated troponin; elevated CRP	Corticosteroids; azathioprine; later vedolizumab	Resolution after first episode; recurrence during flare; improved with immunosuppression
Jena et al	2022	22	Male	UC (active)	Mesalazine-induced myopericarditis; mild LV hypokinesis (LVEF 50–55%)	Palpitations; bradycardia (38 bpm); hypotension (80/60 mmHg); anemia (Hb 8.2 g/dL); elevated troponin T	Discontinuation of mesalazine; IV steroids; inotropes	Hemodynamic improvement within 48 h; normal follow-up echocardiogram; discharged on steroids and azathioprine
Andrei et al	2023	28	Female	UC (recently diagnosed)	Mesalazine-induced myopericarditis; increased T1/T2 signal; LGE (inferolateral); minimal pericardial effusion	Fever; chest pain; leukocytosis; elevated CRP; elevated troponin T and NT-proBNP; incomplete RBBB	Discontinuation of 5-ASA (rechallenge positive); infliximab; permanent 5-ASA cessation	Good clinical response; CMR at 6 months showed complete recovery
Andrei et al	2023	79	Male	Suspected IBD (new diagnosis)	Myocarditis; biventricular dysfunction (LVEF 20%); epi-mesocardial LGE; pleuro-pericardial effusion; global LV hypokinesis	Exertional dyspnea; peripheral edema	Discontinuation of 5-ASA; heart failure therapy (bisoprolol, ramipril, eplerenone, furosemide)	LVEF improved to 40%; recovery of RV function at 1 month; splenic lymphoma diagnosed
Littlewood et al	2024	28	Female	UC (post-flare)	Mesalazine-induced myocarditis; extensive patchy LGE (mid-to-epicardial, basal/lateral wall); LVEF 50%-55%	Sharp pleuritic chest pain; diaphoresis; elevated troponin T; elevated CRP	Discontinuation of mesalazine; ibuprofen (3 days); colchicine (3 months)	Symptoms resolved; discharged after 5 days; follow-up CMR at 4 months showed near-complete resolution

Note: References for individual cases are provided in the main reference list.

Abbreviations: CRP, C-reactive protein; CMR, cardiac magnetic resonance; DOACs, direct oral anticoagulants; ECG, electrocardiogram; ECMO, extracorporeal membrane oxygenation; GI, gastrointestinal; Hb, hemoglobin; IABP, intra-aortic balloon pump; IBD, inflammatory bowel disease; ICD, implantable cardioverter-defibrillator; LGE, late gadolinium enhancement; LMWH, low-molecular-weight heparin; LVEF, left ventricular ejection fraction; NT-proBNP, N-terminal pro–B-type natriuretic peptide; RBBB, right bundle branch block; RV, right ventricle; UC, ulcerative colitis.

The initial diagnostic workup typically involves electrocardiography (showing sinus tachycardia or ST-T wave abnormalities), elevated cardiac biomarkers, and transthoracic echocardiography. However, as demonstrated in this case, echocardiography may be inconclusive due to

technical limitations, such as poor acoustic window. In modern clinical practice, cardiac MRI has become the non-invasive gold standard, surpassing endomyocardial biopsy in routine use. Characteristic findings, including focal myocardial oedema (T2-weighted sequences), early gadolinium enhancement, and regional ventricular dysfunction (20), were instrumental in confirming myopericarditis in this patient.

A critical diagnostic challenge is distinguishing extraintestinal manifestations from alternative etiologies of myopericarditis. UC-related systemic inflammation was considered the most likely cause, supported by the temporal association with an active flare and the absence of alternative triggers. Mesalazine-induced myopericarditis, although well described —typically occurs within the first 28 days of therapy (6)—was deemed unlikely given the patient's long-term exposure without prior adverse events. Paraneoplastic and autoimmune causes were not supported by clinical, laboratory, or imaging findings. Idiopathic myocarditis could not be entirely excluded but was considered less likely in the presence of a clear inflammatory trigger.

During moderate-to-severe disease activity, this state resembles a "cytokine storm," driven by elevated levels of TNF-α, IL-1β, and IL-6, which promote both myocardial and pericardial inflammation and activation of the coagulation cascade through endothelial dysfunction, tissue factor upregulation, thrombin generation, and platelet activation (21, 22). This prothrombotic milieu is further supported by sustained elevations in key coagulation factors and platelet counts beyond the acute phase, contributing to a persistent shift in the global hemostatic balance toward thrombosis (23). TNF-α acts as a central link between inflammation and coagulation, creating a self-amplifying loop that culminates in a "perfect storm", in which cardiac inflammation and thrombus formation represent parallel manifestations of the same systemic inflammatory–prothrombotic axis (24, 25).

Regarding the intracavitary thrombus, patients suffering from inflammatory bowel disease (IBD) have a 3- to 4-fold higher risk of thromboembolic events compared with the general population, with approximately 50% occurring during active flares (26, 27). While common sites include the lungs and deep veins, right atrial thrombi are exceedingly rare and predominantly affect young individuals (19). The negative result of the thrombophilia workup performed 12 weeks post-event further supports an inflammation-driven mechanism, arguing against an underlying inherited or persistent prothrombotic condition.

Achieving the correct balance in therapeutic management was essential. Given the potential risks associated with erratic absorption and gastrointestinal (GI) bleeding during an active flare, parenteral anticoagulation with enoxaparin was preferred over direct oral anticoagulants (DOACs) (28, 29). Enoxaparin is favored for venous thromboembolism prophylaxis in active IBD due to predictable pharmacokinetics via parenteral administration and established safety even in the presence of active GI bleeding, whereas DOACs raise concerns regarding absorption variability and increased bleeding risk in this setting (30–32).

Furthermore, the decision to initiate infliximab was based on its proven efficacy in moderate-to-severe UC (33, 34), particularly in biologic-naïve patients compared with standard-dose adalimumab or golimumab (35, 36). Infliximab induces and maintains remission, promotes mucosal healing, and exerts steroid-sparing effects (1, 37), with emerging evidence of antithrombotic properties (38). However, data specifically addressing its role in UC-associated myopericarditis remain limited.

This combined approach targets both the immediate thrombotic risk (via enoxaparin) and the underlying inflammatory-thrombotic axis (via infliximab), while avoiding medications that may exacerbate hypercoagulability (corticosteroids) or pose concerns regarding absorption and bleeding risk (DOACs) in the setting of active mucosal disease (30, 39, 40).

Myopericarditis and intracavitary thrombosis are rare but life-threatening extraintestinal manifestations of UC. Their non-specific clinical presentation requires a high level of clinical suspicion, particularly in patients with active disease and atypical chest pain. This case underscores the importance of a multidisciplinary approach and the crucial role of advanced imaging, such as cardiac MRI, in preventing adverse outcomes. Early recognition of these cardiovascular complications is crucial, as they may reflect the severity of the systemic inflammatory response rather than isolated cardiac disease.

Abbreviations

BP
blood pressure
DOACs
direct oral anticoagulants
GI
gastrointestinal
IBD
inflammatory bowel disease
IL
interleukin

MRI
magnetic resonance imaging
TNF-α
tumour necrosis factor alpha
UC
ulcerative colitis

Declarations

Ethics approval and consent to participate

This study was reviewed and approved by the Ethics and Research Committee of the Fundación Cardioinfantil-Instituto de Cardiología (Bogota, Colombia). The patient provided written informed consent to participate in this study.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Availability of data and materials

All data generated or analyzed during this study are included in this published article.

Competing interests

The authors declare that they have no competing interests.

Funding

The authors received no financial support for the research, authorship, and/or publication of this article.

Authors' contributions

LDD, MBG, JB, and JM were involved in the clinical care of the patient and data collection. NA performed the cardiovascular assessment and interpreted the cardiac imaging. DRK supervised the gastroenterological management and provided critical revision of the manuscript for important intellectual content. LDD, MBG and DRK were major contributors in writing the manuscript. All authors read and approved the final manuscript.

Acknowledgements

The authors thank Dr. José Cruz, internist at Fundación Cardioinfantil–Instituto de Cardiología, for his support in editing and translating this manuscript.

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Figures

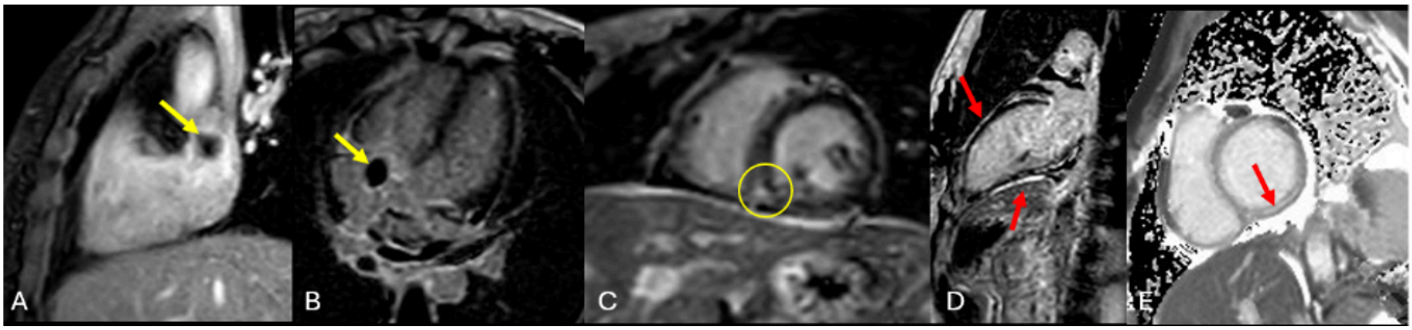


Figure 1

Cardiac magnetic resonance imaging.

(A) Early gadolinium enhancement (right ventricular inflow–outflow view) showing a round, homogeneous hypointense mass in the right atrium (yellow arrow). **(B)** Phase-sensitive inversion recovery (PSIR) imaging demonstrating the same mass without late gadolinium enhancement, consistent with intracardiac thrombus (yellow arrow). **(C)** Short-axis PSIR image showing a mid-wall triangular area of left ventricular (LV) late gadolinium enhancement in the mid-septal region (yellow circle). **(D)**Mild to moderate pericardial enhancement (red arrows). **(E)** T2-weighted short tau inversion recovery (T2w-STIR) sequence showing myocardial edema in the basal inferior LV wall (red arrow). Findings consistent with myopericarditis and intracardiac thrombus.

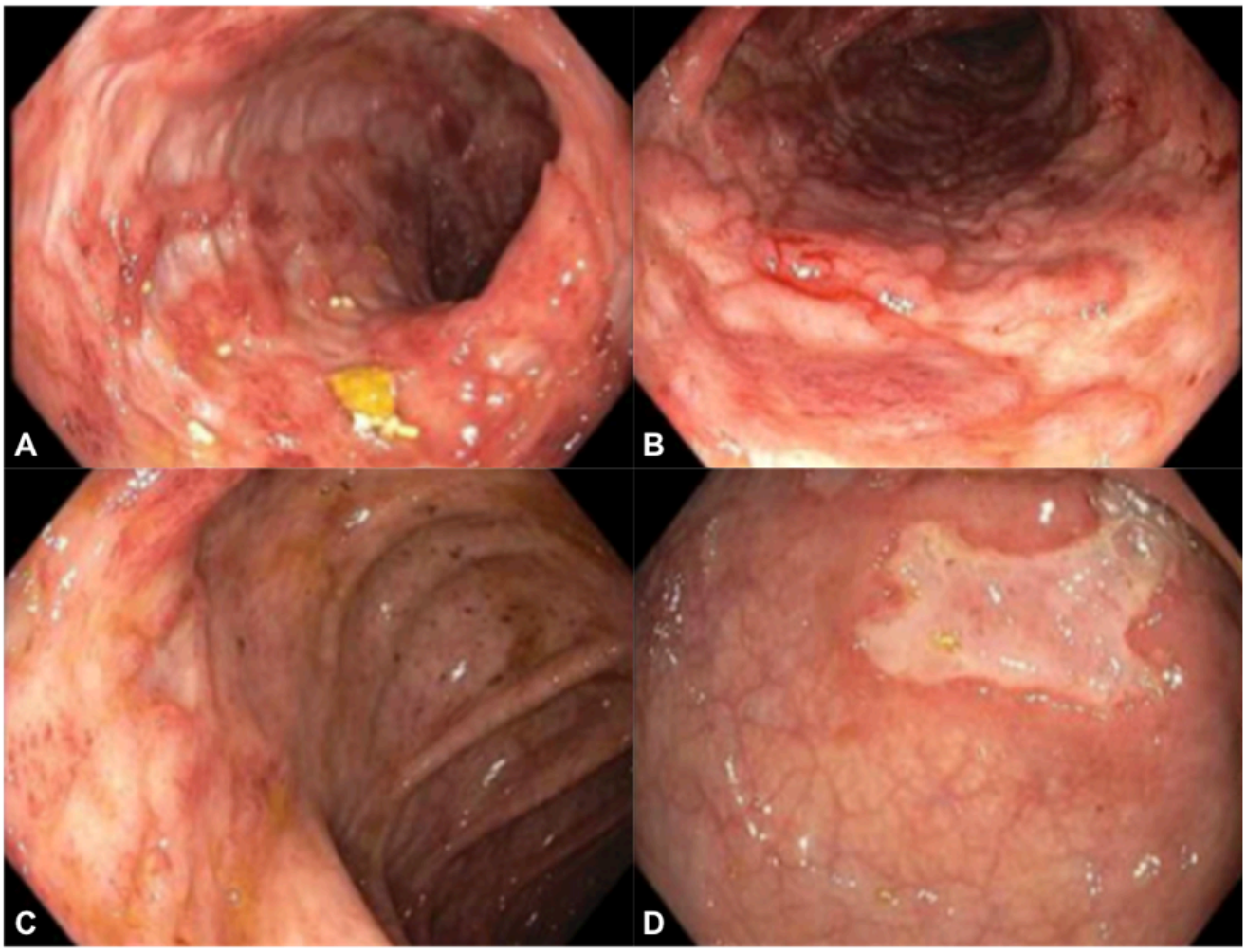


Figure 2

Ileocolonoscopy findings consistent with severe active ulcerative colitis.

(A) Descending colon: marked mucosal edema, erythema, and loss of vascular pattern. **(B)** Transverse colon: loss of haustration with diffuse erythema, erosions, and stigmata of recent bleeding. **(C)** Hepatic flexure: transition from severely inflamed transverse colon (edematous, eroded mucosa with fibrin-covered ulcers) to normal-appearing ascending colon. **(D)**Descending colon: patchy distribution of deep ulcers with fibrin base.