

Lupus Enteritis as the Initial Presentation of Systemic Lupus Erythematosus: A Diagnostic Challenge Case Report

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Abstract

Background: Gastrointestinal (GI) involvement in systemic lupus erythematosus (SLE) is uncommon and often presents with nonspecific symptoms that mimic primary gastrointestinal disorders or postoperative complications, resulting in delayed diagnosis and inappropriate management. Lupus enteritis is a rare but potentially life-threatening manifestation of SLE and may occasionally represent its initial presentation, further complicating the diagnostic process. **Case Presentation:** A 27-year-old woman presented with a one-year history of persistent nausea, vomiting, and progressive weight loss leading to severe malnutrition. She had previously undergone abdominal surgery for presumed gastrointestinal disease without clinical improvement. During

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hospitalization, she developed systemic manifestations including alopecia, pancytopenia, hypocomplementemia, and positive antinuclear antibodies, raising suspicion for active SLE. Contrast-enhanced abdominal computed tomography demonstrated small-bowel wall thickening with a target sign and mesenteric vessel engorgement, findings consistent with lupus enteritis. Endoscopic evaluation excluded structural obstruction and malignancy. Based on the integration of clinical, serological, and radiological findings, a diagnosis of lupus enteritis as the initial presentation of SLE was established. Treatment with high-dose corticosteroids followed by cyclophosphamide resulted in rapid clinical, nutritional, and hematologic improvement. Conclusion: Lupus enteritis should be considered in patients with persistent unexplained gastrointestinal symptoms, particularly when accompanied by systemic autoimmune features. Recognition of characteristic radiologic findings together with serologic evidence of disease activity is crucial for timely diagnosis. Early immunosuppressive therapy may prevent unnecessary surgical interventions and reduce the risk of serious complications.

Keywords: systemic lupus erythematosus, lupus enteritis, mesenteric vasculitis, immunosuppressive therapy, diagnostic challenge

1. Introduction

Systemic lupus erythematosus (SLE) is a chronic multisystem autoimmune disease characterized by autoantibody production and immune complex deposition, leading to diverse clinical manifestations [1]. Approximately 40–60% of patients with SLE experience gastrointestinal symptoms; however, only a minority of these manifestations are directly attributable to active lupus-related gastrointestinal disease [2]. These manifestations often pose significant diagnostic challenges, as they may mimic primary GI disorders or adverse drug reactions [3].

Lupus enteritis represents one of the rare but clinically important GI manifestations of SLE [4]. It is characterized by immune-mediated inflammation and vasculopathy affecting the mesentery and intestinal wall [5]. Recent studies highlight that although its incidence is low, lupus enteritis can lead to severe complications such as bowel ischemia, perforation, pseudo-obstruction, and malnutrition if not recognized promptly. The nonspecific nature of its clinical presentation frequently results in delayed diagnosis and, in some cases, unnecessary surgical interventions [6].

Contrast-enhanced abdominal computed tomography (CT) has become the imaging modality of choice for diagnosing lupus enteritis. Typical findings include small-bowel wall thickening with a “target sign” and engorgement of mesenteric vessels, which help distinguish lupus enteritis from infectious or mechanical causes of abdominal pain. Nevertheless, radiologic findings must be interpreted in conjunction with systemic features of lupus, such as cytopenia, hypocomplementemia, and positive autoantibody serology, to establish a comprehensive diagnosis [7].

This case report aims to highlight the diagnostic challenges of lupus enteritis as a manifestation of SLE, particularly in patients with chronic GI symptoms unresponsive to surgical intervention. By presenting this case, we seek to raise clinical awareness of lupus enteritis, emphasize the importance of recognizing characteristic imaging findings, and underscore the role of timely immunosuppressive therapy in preventing morbidity and unnecessary surgical procedures.

2. Case Presentation

A 27-year-old woman presented with a one-year history of persistent daily vomiting and progressive weight loss. Her symptoms began in 2024 with abdominal pain initially treated as gastritis without improvement. She was later hospitalized for abdominal pain, low-grade fever, and black vomitus, and diagnosed with a GI infection. In October 2024, she underwent an appendectomy with partial bowel resection; however, nausea and vomiting persisted postoperatively despite preserved appetite and swallowing. She also reported alopecia and intermittent arthralgia. The patient had no previous history of blood transfusion apart from perioperative transfusion during abdominal surgery. She reported normal bowel habits and denied fever, seizures, or other neurological symptoms.

On admission, the patient appeared weak and severely undernourished (weight 35 kg, height 157 cm, BMI 14.2 kg/m²). Vital signs were stable with blood pressure 97/58 mmHg, heart rate 89 beats/min, respiratory rate 20 breaths/min, temperature 36.3°C, and oxygen saturation 99% on room air. Conjunctival pallor was noted without scleral icterus. Cardiorespiratory examination was unremarkable. Abdominal examination revealed increased bowel sounds, diffuse tenderness, and ascites without shifting dullness. Bilateral lower limb edema was present.

Initial laboratory evaluation (16/09/2025) revealed normocytic normochromic anemia with bicytopenia and marked inflammatory markers. Hypoalbuminemia and mild transaminase elevation were

observed, while renal function remained preserved despite mild albuminuria. Immunologic testing showed low complement C3 with normal C4 and positive ANA, supporting immune-mediated disease activity. Tumor markers and viral hepatitis serology were negative. Detailed laboratory results are presented in Table 1.

Table 1. Laboratory Findings.

Type	Parameter	Result	Reference range
Hematology	Hemoglobin	9.2 g/dL	12.0–16.0 g/dL
	Hematocrit	25.8 %	36–46 %
	Erythrocytes	$3.02 \times 10^6/\mu\text{L}$	$3.8\text{--}5.2 \times 10^6/\mu\text{L}$
	Leukocytes	$3.52 \times 10^3/\mu\text{L}$	$4.0\text{--}10.0 \times 10^3/\mu\text{L}$
	Platelets	$115 \times 10^3/\mu\text{L}$	$150\text{--}400 \times 10^3/\mu\text{L}$
	MCV	85.4 fL	80–100 fL
	MCH	30.5 pg	27–33 pg
	MCHC	35.7 g/dL	32–36 g/dL
	RDW-CV	16.0 %	11.5–14.5 %
	ESR	>140 mm/h	<20 mm/h
Differential count	Neutrophils	62.5 %	40–70 %
	Lymphocytes	31.5 %	20–40 %
	Monocytes	5.7 %	2–8 %
	Eosinophils	0.3 %	1–4 %
	Basophils	0 %	0–1 %
Clinical chemistry	Albumin	2.78 g/dL	3.5–5.0 g/dL
	AST	48 U/L	<35 U/L
	ALT	37 U/L	<35 U/L
	BUN	12.0 mg/dL	7–20 mg/dL
	Creatinine	0.17 mg/dL	0.6–1.1 mg/dL
	CRP	0.78 mg/dL	<0.3 mg/dL
	Electrolytes	Within normal limits	—
Immunology	Complement C3	49.0 mg/dL	90–180 mg/dL
	Complement C4	20.8 mg/dL	10–40 mg/dL
	ANA	43.5 (positive)	Negative
Tumor markers / serology	CEA	Within normal range	<5.0 ng/mL
	CA 15-3	Within normal range	<30 U/mL
	Anti-HCV	Non-reactive	Non-reactive
Urinalysis	Urine albumin	30 mg/L	<30 mg/L

Abbreviations: MCV, mean corpuscular volume; MCH, mean corpuscular haemoglobin; MCHC, mean corpuscular haemoglobin concentration; RDW-CV, red cell distribution width – coefficient of variation; ESR, erythrocyte sedimentation rate; AST, aspartate aminotransferase; ALT, alanine aminotransferase; BUN, blood urea nitrogen; CRP, C-reactive protein; C3, complement component 3; C4,

complement component 4; ANA, antinuclear antibodies; CEA, carcinoembryonic antigen; CA 15-3, cancer antigen 15-3; anti-HCV, antibodies against hepatitis C virus.

Contrast-enhanced abdominal CT demonstrated a characteristic target sign in the mid-jejunum, suggestive of lupus enteritis, along with multiple strictures in the duodenum (pars II–III) and jejunum causing proximal dilatation. Multiple enlarged lymph nodes were observed in para-aortic and mesenteric regions. The CT findings are shown in Figure 1.

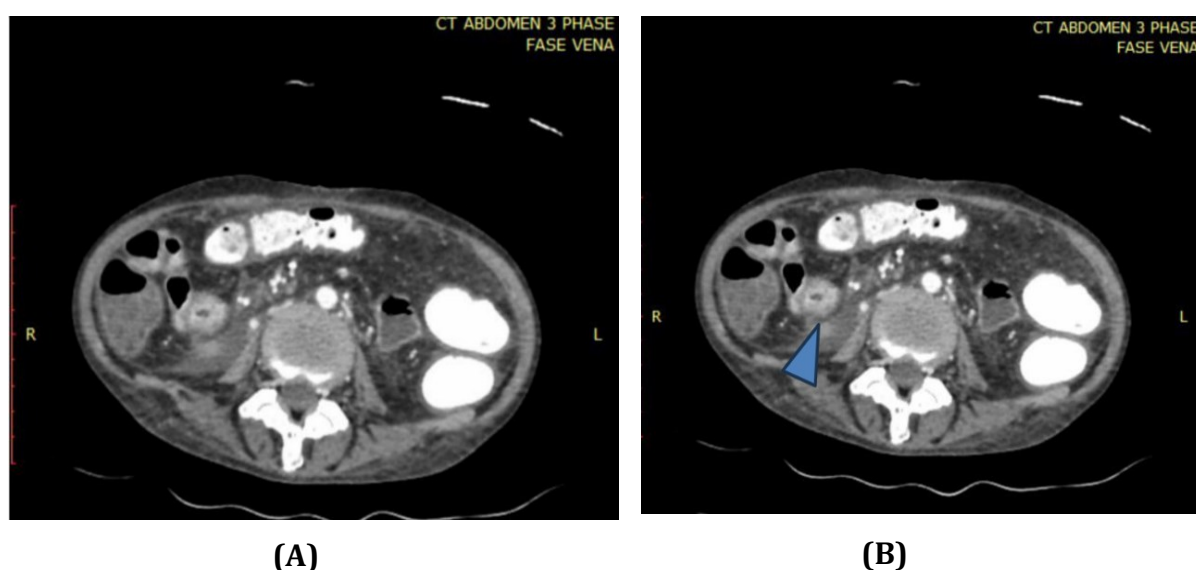


Figure 1. (A) Patient's Abdominal CT scan; (B) Blue arrow shows bowel wall thickening seen in Patient's Abdominal CT scan.

Based on the integration of clinical, laboratory, and radiologic findings, the patient was diagnosed with SLE with gastrointestinal involvement consistent with lupus enteritis. According to the 2019 European League Against Rheumatism/American College of Rheumatology (EULAR/ACR) classification criteria, the patient fulfilled the entry criterion of positive antinuclear antibodies (ANA) and accumulated a total score of 12 points (2 points for non-scarring alopecia, 3 points for leukopenia, 4 points for thrombocytopenia, and 3 points for low complement C3), thereby meeting the classification criteria for SLE. Additional clinical problems included severe malnutrition, suspected gastric outlet obstruction, chronic wound dehiscence, anemia, and hypoalbuminemia-related edema [8].

Given the patient's prolonged history of persistent nausea, vomiting, severe weight loss, previous abdominal surgery, and bowel wall thickening on CT imaging, several differential diagnoses were considered before establishing the diagnosis of lupus enteritis. Histopathological records from the

previous appendectomy were unavailable for review, precluding retrospective assessment of the initial pathological diagnosis. These diagnoses and the rationale for their exclusion are summarized in Table 2.

Table 2. Differential Diagnosis of Lupus Enteritis and the Clinical Findings Supporting or Excluding Each Diagnosis

Differential diagnosis	Findings supporting the diagnosis	Findings leading to exclusion
Mechanical bowel obstruction	Persistent vomiting, history of abdominal surgery	No obstructive lesion on endoscopy; CT favored inflammatory bowel wall thickening rather than transition point
Intestinal tuberculosis	Chronic symptoms, weight loss, mesenteric lymphadenopathy	No microbiological, histopathological, or radiological evidence suggestive of tuberculosis
Crohn's disease	Small bowel wall thickening	Endoscopy showed no characteristic inflammatory lesions; clinical and laboratory findings favored SLE
Gastrointestinal malignancy	Severe weight loss and anemia	Endoscopy showed no evidence of malignancy; imaging did not demonstrate a mass lesion
Chronic intestinal pseudo-obstruction	Persistent nausea and vomiting without clear mechanical obstruction	Characteristic CT findings, positive ANA, hypocomplementemia, and favorable response to immunosuppressive therapy supported lupus enteritis
Lupus enteritis	Positive ANA, hypocomplementemia, bowel wall thickening with target sign, systemic manifestations of SLE	Final diagnosis

Abbreviations: CT, computed tomography; SLE, systemic lupus erythematosus; ANA, antinuclear antibodies.

Initial management consisted of intravenous fluids (1,000 mL/24 h), intravenous lipid emulsion every 48 hours, and intravenous methylprednisolone 62.5 mg daily. Symptomatic therapy included metoclopramide, sucralfate, and omeprazole. One unit of packed red blood

cells (PRC) was transfused. Esophagogastroduodenoscopy (EGD) was planned for further evaluation.

On Day 4 (19/09/2025), the patient experienced one episode of postprandial vomiting and persistent weakness. Laboratory evaluation revealed progression to severe pancytopenia (hemoglobin 7.9 g/dL, leukocytes $2.57 \times 10^3/\mu\text{L}$, platelets $62 \times 10^3/\mu\text{L}$). A central venous catheter was inserted due to difficult access. Two additional PRC units were transfused, and corticosteroid therapy was escalated to high-dose intravenous methylprednisolone 500 mg/day.

By Day 8 (23/09/2025), clinical improvement was noted with reduced edema and fewer vomiting episodes. Laboratory results showed significant hemoglobin and leukocyte recovery (14.0 g/dL and $5.23 \times 10^3/\mu\text{L}$, respectively), although thrombocytopenia persisted ($60 \times 10^3/\mu\text{L}$). Serum albumin improved to 3.28 g/dL. Steroids were tapered to intravenous methylprednisolone 31.25 mg/day.

On Day 10 (25/09/2025), EGD revealed gastroesophageal reflux disease with Los Angeles grade C esophagitis, erosive pangastritis, and duodenitis without mechanical obstruction. Histopathology showed non-specific inflammatory changes and was negative for *Helicobacter pylori*. Endoscopic findings are presented in Figure 2. Follow-up laboratory tests showed recurrent leukopenia and thrombocytopenia, suggesting ongoing immune-mediated marrow suppression. Intravenous cyclophosphamide 250 mg was administered.

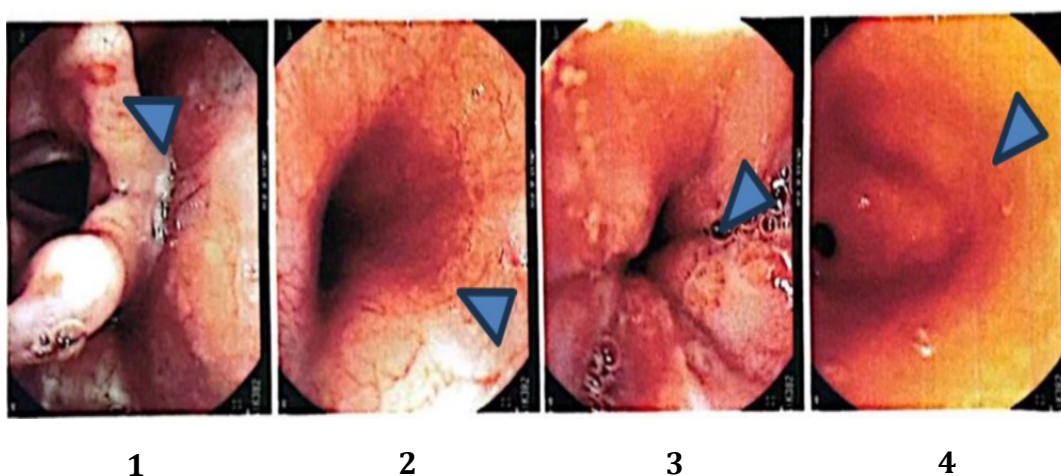


Figure 2. Patient's EGD Result (1) Larynx: Hyperemic, edematous mucosa with prominent vascularity; (2) and (3) Esophagus: Diffuse hyperemia from proximal

to distal segments with erosions in the middle to distal third; EGJ showed a mucosal break with granular changes; (4) Stomach (Antrum) The mucosa appeared hyperemic, edematous, and erosive.

The patient was discharged on oral methylprednisolone 16 mg twice daily, sucralfate, omeprazole, ondansetron, and domperidone. At outpatient follow-up (7/10/2025), GI symptoms had markedly improved with better oral intake. Hematologic parameters showed recovery (hemoglobin 13.5 g/dL, leukocytes $5.71 \times 10^3/\mu\text{L}$, platelets $87 \times 10^3/\mu\text{L}$), and albumin normalized to 4.34 g/dL. The patient was scheduled to receive six monthly intravenous cyclophosphamide 250mg pulses followed by maintenance therapy with hydroxychloroquine 200mg daily and tapering corticosteroids.

3. Discussion

Chronic recurrent nausea and vomiting with progressive weight loss require a structured diagnostic approach prioritizing common GI etiologies before systemic autoimmune causes are considered [9,10]. Mechanical obstruction including peptic ulcer-related stenosis or postoperative adhesions must be excluded, particularly in patients with prior abdominal surgery [11]. Functional disorders such as gastroparesis or intestinal pseudo-obstruction may clinically mimic obstruction without structural blockage [12]. In this case, persistence of symptoms despite prior surgery, combined with severe malnutrition and systemic manifestations, prompted reassessment beyond mechanical pathology toward inflammatory systemic disease.

Although gastrointestinal manifestations are relatively uncommon in SLE, they may occasionally represent the earliest manifestation of the disease. Research reported that gastrointestinal complaints were frequently recalled as prodromal symptoms preceding the diagnosis of SLE [13]. This observation is consistent with our case, in which persistent nausea and vomiting preceded the diagnosis of SLE by approximately one year and initially led to an alternative surgical diagnosis. Therefore, persistent unexplained gastrointestinal symptoms, particularly when accompanied by systemic abnormalities such as cytopenia or hypocomplementemia, should prompt consideration of underlying autoimmune disease even in the absence of classic lupus manifestations.

Gastrointestinal involvement occurs in 40–60% of patients with SLE, although only a minority of cases are directly attributable to active lupus [3]. Lupus enteritis represents a rare but potentially life-threatening

manifestation characterized by immune-mediated small-bowel inflammation. Because GI manifestations are not incorporated into SLEDAI scoring, disease activity may be underestimated [2,3]. In this patient, chronic vomiting, severe malnutrition (BMI 14.2 kg/m²), alopecia, anemia, and progressive pancytopenia indicated multisystem disease. Positive ANA and low complement C3 supported active immune complex-mediated activity, while hematologic abnormalities reinforced systemic involvement rather than isolated GI pathology.

The pathogenesis of lupus enteritis is driven by immune complex deposition and complement activation within mesenteric vessels, resulting in submucosal edema, vascular congestion, and segmental ischemia. Complement consumption correlates with disease activity, consistent with the low C3 level observed in this patient [7]. In addition to vasculitis, enteric neuropathy and smooth muscle dysfunction may contribute to reversible dysmotility, explaining chronic vomiting in the absence of complete obstruction [14]. The absence of perforation or hemorrhage suggests inflammatory involvement without transmural necrosis.

Contrast-enhanced abdominal CT remains the diagnostic cornerstone for lupus enteritis. Typical findings include bowel wall thickening with a “target sign” and mesenteric vascular engorgement (“comb sign”) [7]. In patients with prior abdominal surgery, distinguishing inflammatory vasculitic changes from mechanical obstruction represents a significant diagnostic challenge [14]. In this case, integration of imaging findings with serologic activity and systemic features favored active lupus enteritis over postoperative obstruction.

Endoscopy plays a complementary role primarily to exclude structural obstruction or malignancy, as superficial biopsies often fail to detect submucosal vascular lesions characteristic of lupus enteritis [15]. In this patient, erosive gastritis and nonspecific duodenitis were observed without obstructive pathology. The discordance between persistent vomiting and minimal mucosal abnormalities further supported a motility-dominant autoimmune process.

Hematologic involvement complicated the clinical course. Pancytopenia in SLE may result from immune-mediated destruction or inflammation-related marrow suppression [16]. The temporal association between high-dose corticosteroid and cyclophosphamide therapy and hematologic recovery supports immune-mediated suppression rather than drug toxicity. The coexistence of lupus enteritis and pancytopenia likely reflects

heightened systemic immune activation and may indicate more severe disease.

The management of lupus enteritis depends on disease severity, the presence of systemic involvement, and the patient's clinical response. High-dose corticosteroids remain the first-line treatment, while additional immunosuppressive agents are recommended for severe, recurrent, or refractory disease. The currently available therapeutic options and their clinical indications are summarized in Table 3.

Table 3. Therapeutic options for lupus enteritis and their clinical indications

Treatment	Clinical indication	Role in management
High-dose corticosteroids	First-line treatment for active lupus enteritis	Rapid control of intestinal inflammation
Intravenous cyclophosphamide	Severe disease, recurrent lupus enteritis, or major organ involvement	Induction therapy in severe SLE
Mycophenolate mofetil	Steroid-sparing agent or refractory disease	Maintenance or alternative immunosuppression
Azathioprine	Maintenance therapy after remission	Prevention of relapse
Rituximab	Refractory or relapsing disease	Rescue therapy in selected patients
Hydroxychloroquine	All patients with SLE unless contraindicated	Long-term disease control and relapse prevention
Nutritional support	Malnutrition or prolonged gastrointestinal symptoms	Supportive management

In the present case, high-dose intravenous methylprednisolone was initiated as first-line therapy because of active lupus enteritis. Intravenous cyclophosphamide was subsequently administered due to the severity of gastrointestinal involvement, prolonged disease course, marked malnutrition, pancytopenia, and active systemic disease. The patient demonstrated rapid clinical improvement following immunosuppressive therapy, supporting the diagnosis of lupus enteritis. The patient remained clinically stable during outpatient follow-up and was scheduled to complete six monthly intravenous cyclophosphamide pulses, followed by maintenance therapy with hydroxychloroquine and tapering corticosteroids.

High-dose corticosteroids constitute first-line therapy for lupus enteritis, with response rates approaching 70–90% [7]. Additional

immunosuppressive agents, including cyclophosphamide, are recommended in severe or refractory cases. Although standardized treatment algorithms are lacking, early immunosuppression is associated with improved outcomes [2,3]. In this patient, pulse methylprednisolone followed by cyclophosphamide resulted in rapid symptom resolution and hematologic recovery, supporting inflammatory rather than structural etiology.

Nutritional optimization is critical, as prolonged vomiting and intestinal inflammation frequently result in severe protein-calorie malnutrition [14]. Temporary parenteral support facilitates intestinal recovery and stabilization. Following immunosuppressive therapy, this patient demonstrated restored oral intake and normalization of albumin levels.

Although prognosis is generally favourable with prompt treatment, relapse occurs in 20–30% of cases, particularly in the presence of persistent hypocomplementemia [3]. Ongoing serologic monitoring and maintenance therapy are therefore essential. Given the initial complement consumption observed in this patient, continued hydroxychloroquine therapy and gradual steroid tapering are warranted to reduce recurrence risk.

Strengths and Limitations

This case illustrates the diagnostic challenge of lupus enteritis presenting as the initial manifestation of SLE. However, several limitations should be acknowledged. Extended autoantibody profiling, including anti-dsDNA, anti-Sm, anti-Ro/SSA, anti-La/SSB, and antiphospholipid antibodies, was not available in this case. In addition, histopathological findings from the previous appendectomy were unavailable, and long-term follow-up after cyclophosphamide therapy was limited. Nevertheless, the diagnosis was supported by fulfilment of the 2019 EULAR/ACR classification criteria, characteristic clinical manifestations, hypocomplementemia, positive ANA, typical radiologic findings, and a favourable response to immunosuppressive therapy. The ANA immunofluorescence pattern using HEp-2 cells was not available because the test was reported only qualitatively at our institution. Direct antiglobulin testing (Coombs test) was not performed because autoimmune hemolytic anemia was not clinically suspected.

4. Conclusions

Gastrointestinal involvement in SLE remains a diagnostic challenge due to its nonspecific presentation and frequent misclassification as functional or

mechanical disease. Persistent GI symptoms accompanied by serologic evidence of active lupus and characteristic radiologic findings should prompt early suspicion of lupus enteritis. A rapid response to immunosuppressive therapy further supports an inflammatory rather than structural etiology. Timely recognition of mesenteric vasculitis-related pathophysiology and implementation of a multidisciplinary diagnostic strategy are essential to prevent catastrophic complications, including intestinal ischemia and perforation.

Declarations

Author Contributions

Conceptualization: F.V., A.A.; Methodology: F.V., A.A.; Formal analysis: F.V.; Investigation: F.V., A.A.; Writing—original draft: F.V.; Writing—review & editing: F.V., A.A.

Artificial Intelligence Statement

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Institutional Review Board Statement

Not applicable.

Informed Consent Statement

Informed consent was obtained from the patient prior to the publication of this case report and accompanying clinical data/images. The case report was conducted in accordance with the Declaration of Helsinki.

Data Availability Statement

The data presented in this study are available on reasonable request from the corresponding author.

Conflicts of Interest

The authors declare no conflict of interest.

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