



Lupus Enteritis as an Initial Manifestation in a Previously Undiagnosed Case of Systemic Lupus Erythematosus: A Case Report

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Indian J Radiol Imaging 2026;36:258–261.

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Abstract

Systemic lupus erythematosus (SLE) is an autoimmune condition with the potential to impact all bodily organ systems. Lupus enteritis represents a rare complication observed in a subset of patients who exhibit nonspecific gastrointestinal symptoms. Typically, the diagnosis relies on imaging findings such as localized or widespread thickening of the bowel wall, abnormal enhancement of the bowel wall, a distinctive “comb sign” indicating engorged mesenteric vessels, the presence of ascites, and lymph node enlargement. Treatment usually involves the administration of corticosteroids and other immunosuppressive agents as second-line options, which have demonstrated efficacy in managing lupus enteritis. This report details a case of previously undiagnosed SLE in a patient presenting with abdominal pain and diarrhea, with diagnostic assessments confirming the presence of lupus enteritis. Timely identification and appropriate treatment are crucial in managing lupus enteritis to mitigate potential complications and enhance long-term survival. Additionally, we explore the clinical characteristics of lupus enteritis, underscoring its underlying mechanisms, diagnostic approaches, and therapeutic strategies.

Keywords

- ▶ comb sign
- ▶ lupus enteritis
- ▶ target sign

Introduction

Systemic lupus erythematosus (SLE) is a chronic autoimmune condition that can affect any organ. Although uncommon, it can also impact the gastrointestinal system, with enteritis being one such manifestation characterized by inflammation of the small blood vessels within the gut.¹ The chief presenting symptom is typically abdominal pain. The rarity of lupus enteritis as the first sign of SLE, coupled with its potential for misdiagnosis, can result in delayed treatment. Here, we describe a case of enteritis as the inaugural sign of new-onset SLE, accompanied by a review of the literature to identify key diagnostic indicators.

Case Presentation

A 27-year-old Asian woman with a history of hypothyroidism was admitted to a private hospital with complaints of abdominal pain, diarrhea, vomiting, and fever. Initially, the patient was managed symptomatically with antibiotics for 2 days, after which she developed difficulty in passing stool for 1 day and was referred to our tertiary care center for further management. On examination, the patient was pale and afebrile, and the abdomen was distended; however, no guarding or rigidity was present. The patient also had bilateral lower limb swelling and painful right knee swelling. Other systemic examinations and vitals were normal. Initial

article published online
March 27, 2025

DOI <https://doi.org/10.1055/s-0045-1806748>.
ISSN 0971-3026.

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Table 1 Laboratory tests with reference values of the patient done on the day of admission

Routine hematological investigations			
Laboratory parameter	Test results	Reference values	Unit
White blood cells (WBC)	5.51×10^3	$4-10 \times 10^3$	Cells/ μ L
Differential leucocyte counts	91	44-76	%
Neutrophils	05	20-40	%
Lymphocytes	02	2-10	%
Monocytes	02	1-6	%
Eosinophils			
Red blood cells (RBC)	2.96×10^6	$3.5-5.5 \times 10^6$	Cells/ μ L
Fasting blood glucose	92	70-100	mg%
Hemoglobin	8.6	12-16	g/dL
Platelet count	282,000	150,000-450,000	/ mm^3
Erythrocyte sedimentation rate (ESR)	95	0-19	mm/h
C-reactive protein (CRP)	38.2	≤ 5	mg/L
Serum creatinine	1.6	0.6-1.4	mg/dL
Urine protein	Trace	Nil	-
Serum albumin	2.8	3.0-4.5	g/dL
Total protein	5.8	6.0-7.5	g/dL

laboratory tests (**►Table 1**) showed anemia (Hb: 8.6 g/dL), normal WBC count with lymphopenia (total leukocyte count [TLC]: 5510 cell/ μ L with 5% lymphocytes), increased C-reactive protein (38.2 mg/L), raised creatinine (1.6 mg/dL), and hypoproteinemia (total protein: 5.8 g/dL and serum albumin: 2.8 g/dL). Trace protein was also detected in the patients' urine.

One month prior to the development of the present symptoms, the patient reported a history of trivial trauma to her right knee, following which she developed swelling in the same knee. Joint fluid aspiration was performed at a different institution and sent for microbiological analysis. It revealed the growth of salmonella species, and appropriate antibiotics were given for the same, which resulted in resolution of the swelling.

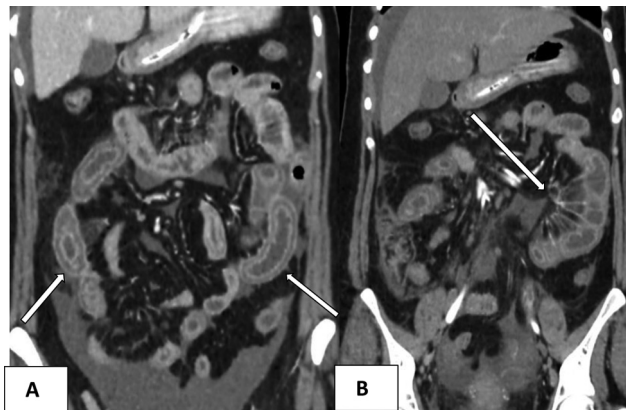


Fig. 1 Coronal sections of contrast-enhanced computed tomography (CECT) of the abdomen performed on the day of admission. (A) Bowel wall thickening with heterogeneous enhancement giving target appearance (arrows). (B) Engorged mesenteric vessels arranged in a comb-like arrangement, comb sign (arrows).

Due to ambiguity of clinical symptoms, imaging was required to provide additional diagnostic clarity. Estimated glomerular filtration rate (eGFR) (36 mL/min/1.73 m²) was above the threshold for safe contrast administration²; however, in view of raised serum creatinine (1.6 mg/dL), consent from treating physician and a nephrologist was obtained before proceeding with contrast-enhanced computed tomography (CECT) of the abdomen, which revealed small bowel distention with diffuse bowel wall thickening right from the duodenum to the ileum with heterogeneous enhancement giving a target appearance, engorged mesenteric vessels in a comb-like arrangement (comb sign), ascites, and lymphadenopathy (**►Fig. 1**). Additionally, the patient also had bilateral hydronephrosis with bilateral ureteric narrowing and urothelial enhancement (**►Fig. 2**).

The patient was treated initially with intravenous (IV) fluids, antibiotics, gastric decompression via a nasogastric tube, and a parenteral diet. The stool culture was negative.

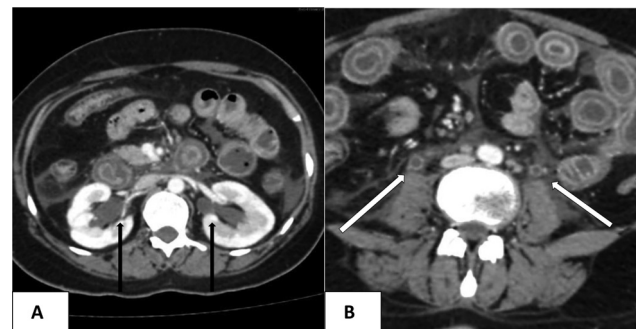


Fig. 2 Axial sections of contrast-enhanced computed tomography (CECT) of the abdomen performed on the day of admission. (A) Bilateral hydronephrosis (arrows). (B) Hydronephrosis with urothelial enhancement (arrows).

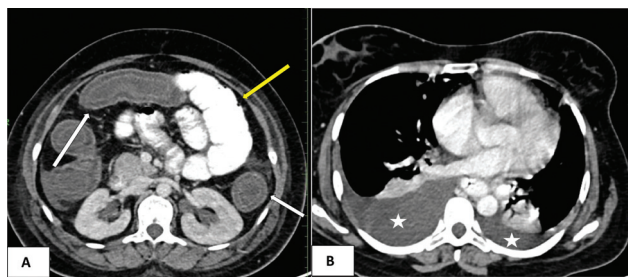


Fig. 3 Axial sections of repeat contrast-enhanced computed tomography (CECT) of the abdomen performed on day 6 of admission after the patient worsened clinically. (A) Involvement of the large bowel (white arrows) with resolution of the proximal jejunal bowel wall thickening (yellow arrow). (B) Bilateral pleural effusion (white asterisks).

Abdominal pain worsened and clinically perforation was suspected, so a CECT of the abdomen with positive oral contrast was repeated on day 6 of hospitalization (►Fig. 3), which revealed diffuse circumferential thickening of the entire colon as well as engorged mesenteric vessels. While there was resolution of proximal jejunal wall thickening (►Fig. 3A), mild wall thickening persisted throughout the remaining small bowel. The patient also developed bilateral pleural effusion (►Fig. 3B).

In view of mesenteric ischemic changes occurring in a young patient, with concomitant involvement of the small and large intestine and associated genitourinary involvement, the possibility of vasculitic enteritis was considered, and an autoimmune workup was sent (►Table 2), which showed positive p-ANCA (1:80) and low C3 and C4. ANA blot was positive for antibodies against nucleosomes, which are specific for SLE. The patient denied other lupus symptoms.

Treatment was started with methylprednisolone and cyclosporine, with a complete resolution of symptoms. Follow-up CECT of the abdomen (►Fig. 4) performed 7 days after starting the treatment (i.e., on day 13 of hospitalization) also showed resolution of bowel wall thickening, ascites, and bilateral pleural effusion. Bilateral hydronephrosis

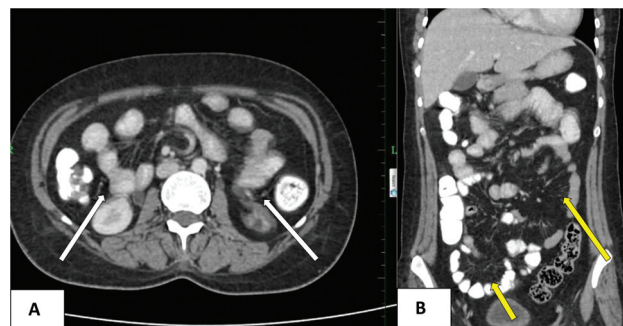


Fig. 4 Follow-up contrast-enhanced computed tomography (CECT) of the abdomen performed 1 week after commencement of therapy. (A) Axial section showing complete resolution of bowel wall thickening (arrows). (B) Coronal section showing remission of mesenteric engorgement (arrows) and ascites.

was persistent, but there was resolution of urothelial enhancement.

Discussion

Lupus enteritis, a rare complication of SLE, is diagnosed in approximately 1% of lupus patients who present with abdominal pain. Symptoms are nonspecific and include abdominal pain (97%), ascites (78%), nausea (49%), vomiting (42%), and diarrhea (32%).³

According to Janssens et al, only 13% of lupus enteritis cases are initially diagnosed alongside SLE.⁴ The exact pathophysiology remains uncertain, though many experts suggest that immunocomplex deposits may lead to microvascular lesions, potentially causing intestinal ischemia.^{5,6} The common laboratory findings include hematologic abnormalities (leukopenia, lymphopenia, and anemia), positive ANA (92%), anti-dsDNA (74%), low complement (88%), anti-RNP (28%), anti-SSA (26%), and anti-Sm (24%).

An abdominal CT scan performed after injecting IV contrast is the primary diagnostic tool that facilitates the identification of abnormal bowel wall enhancement (target sign) and engorged mesenteric vessels (comb sign). Other findings that can be seen in the abdominal CT scan are ascites

Table 2 Autoimmune workup with reference values of the patient

Laboratory parameter	Test results	Reference values	Unit
Complement 3 (C3)	48.3	90–180	mg/dL
Complement 4 (C4)	5.2	10–40	mg/dL
p-ANCA (ANCA-MPO)	1:80	Negative: <1:10 Weak positive: 1:10 Positive: ≥1:20	Qualitative
c-ANCA (ANCA-PR3)	Negative	Negative: <1:10 Weak positive: 1:10 Positive: ≥1:20	Qualitative
ANA	1:320 +4, homogenous	Negative: <1:80 Weak positive: 1:80 Positive: ≥1:160	Qualitative
Nucleosomes	52++	Qualitative	

and lymphadenopathy.¹ Contrast administration is generally considered safe when eGFR is above 30 mL/min/1.73 m²; however, in individual high-risk circumstances (e.g., numerous risk factors, recent AKI, borderline eGFR), prophylaxis with isotonic volume expansion may be considered in patients with eGFR of 30 to 44 mL/min/1.73 m² at the discretion of the ordering clinician.³

The multifocal nature of bowel wall thickening, not confined to a single vascular territory, suggests mesenteric vasculitis affecting multiple vessels simultaneously. Similar findings can also be seen in mechanical small bowel obstruction, inflammatory bowel disease especially Crohn's (where terminal ileum and ileocecal junction is more commonly involved and additionally skip lesions are present), and hypoproteinemia (which also shows edematous changes in subcutaneous tissues, peritoneum, and retroperitoneum).⁷ Thus, rather than bowel wall changes, the ancillary findings occurring in other organs may suggest the diagnosis as was in our case, with the involvement of genitourinary system resulting in ureteritis and hydronephrosis.

Ureteritis and hydronephrosis are rare complications of SLE, and the exact incidence is not known; however, most cases have been reported in the Southeast Asian population. The underlying pathology of ureteritis and hydronephrosis in SLE may involve diffuse small vessel vasculitis, which can lead to neuritis and dysfunction of the smooth muscle in the bladder and ureter.⁸ Other possible causes for hydronephrosis are detrusor muscle spasm with subsequent vesicoureteric reflux or fibrosis of the ureterovesical junction.⁹ Hydronephrosis also occurs in other types of vasculitis such as polyarteritis nodosa and Henoch-Schönlein syndrome.^{10,11}

Ischemia is potentially reversible, and initial management involves systemic steroids. For severe and unresponsive cases, cyclophosphamide, azathioprine, or mycophenolate mofetil can be added, along with hydroxychloroquine for long-term maintenance therapy.¹²⁻¹⁴ In our case, the patient showed clinical and radiological improvement after initiating bowel rest and receiving IV pulses of methylprednisolone and cyclophosphamide.

While surgical intervention was not required in our case, perforation is a potential complication, necessitating caution in management.

Conclusion

In conclusion, lupus enteritis is a rare complication of SLE, and it is even rarer to have it as an initial presentation. Being

clinically nonspecific, the diagnosis is difficult, and thus imaging plays a crucial role in its detection. Its remarkable response to immunosuppressive treatment underscores the necessity of early diagnosis.

Conflict of Interest

None declared.

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