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Lupus enteritis in a tuberculosis-endemic region of India: Diagnostic pitfalls and radiologic insights

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Abstract:
Systemic lupus erythematosus (SLE) may rarely present as lupus enteritis, a gastrointestinal manifestation that can closely mimic abdominal tuberculosis in endemic regions. A 22-year-old woman with prolonged fever, weight loss, abdominal pain, vomiting, diarrhoea and pancytopenia was initially treated for disseminated tuberculosis but showed no clinical improvement. Contrast-enhanced computed tomography showed characteristic bowel wall thickening with a target sign, while autoimmune evaluation revealed a strongly positive ANA, elevated anti-dsDNA antibodies, hypocomplementemia and multiple extractable nuclear antigen antibodies, confirming lupus enteritis secondary to SLE. Prompt treatment with corticosteroids, hydroxychloroquine and cyclophosphamide resulted in marked clinical recovery. Thus, data shows the importance of integrating characteristic radiological findings with autoimmune investigations for early diagnosis and appropriate immunosuppressive therapy in tuberculosis-endemic settings.

Keywords: Systemic lupus erythematosus (SLE), lupus enteritis, tuberculosis mimicry, autoimmune vasculitis, computed tomography, target sign, mesenteric vasculitis, hypocomplementemia

Background:
Systemic lupus erythematosus (SLE) is a chronic autoimmune and systemic disorder of the connective tissues with a variety of gastrointestinal manifestations observed in many patients over the course of their disease **[1]**. Lupus enteritis is a rare gastrointestinal manifestation of SLE, characterised by immune-mediated inflammation of the bowel wall and mesenteric vessels **[2, 3]**. Abdominal pain, diarrhoea, ascites and constitutional signs are common presentations in patients with Lupus enteritis, many of which can mimic the clinical presentation of infectious gastrointestinal disorders **[4]**. The use of computerised tomography with contrast administration allows identification of unusual features or "signs" associated with lupus enteritis, including bowel wall thickening, a "target" sign, mesenteric fat stranding and ascites **[5]**. The combination of descriptions of general signs associated with abdominal tuberculosis in regions where the disease is endemic and radiological signs of lupus enteritis creates a strong potential for misdiagnosis and delay in initiating appropriate immunosuppressive therapy **[6]**. Because of these factors, misdiagnosis or delay in the initiation of treatment for lupus enteritis can lead to severe complications, including bowel ischemia, bowel perforation, haemorrhage, sepsis and increased morbidity **[7]**. Therefore, it is of interest to report a case of lupus enteritis initially misdiagnosed as abdominal tuberculosis in a tuberculosis-endemic region.

Materials and Methods:
The purpose of this research is to provide a thorough description of a confirmed case of TB infection that is clinically diagnosed with lupus enteritis in a geographic area that has an unusually high incidence of the bacterial disease Tuberculosis (TB) and was initially diagnosed and treated as an invasive type of TB within the abdomen of this patient based on clinical findings. A 22-year-old woman presented with prolonged fever, weight loss, abdominal pain, vomiting, diarrhoea and pancytopenia and a pleural effusion. The patient underwent extensive clinical, laboratory, radiological and autoimmune investigations to confirm the diagnosis. Laboratory testing consisted of baseline haematology and biochemistry investigations (including haemoglobin, leukocyte count, platelet count, erythrocyte sedimentation rate, C-reactive protein, serum albumin, renal function tests and urine protein/creatinine ratio). The patient underwent radiological evaluation, including a non-contrast CT scan of the thoracic region and a contrast-enhanced CT scan of the abdominal region, with oral contrast, to evaluate for bowel and mesenteric involvement, ascites and lymphadenopathy. An upper GI endoscopy was performed and biopsies and ascetic fluid analyses were obtained during the procedure. The Autoimmune evaluation included ANA and anti-double-stranded DNA antibody testing, complement levels, a direct Combs test and an extractable nuclear antigen profile. For computed tomography scans, the severity of bowel wall thickening, the extent of bowel involvement, the target sign, mesenteric fat stranding and ascitic involvement were documented. Finally, the clinical response to treatment with corticosteroids, hydroxychloroquine and cyclophosphamide was documented and compared with the radiological and immunological findings.

Results:
The patient presented with prolonged fever, weight loss, abdominal pain, vomiting, diarrhoea and constitutional symptoms, along with significant haematological abnormalities. Baseline laboratory investigations demonstrated severe anaemia with a haemoglobin level of 5.8 g/dL, leukopenia with a total leukocyte count of 2740/µL and thrombocytopenia with a platelet count of 104,000/µL. The erythrocyte sedimentation rate was markedly elevated at 110 mm/hour, while C-reactive protein was elevated at 14.9 mg/L. Hypoalbuminemia and proteinuria were also present, with a urine protein-creatinine ratio of 1.54. Serum creatinine was within the normal range and the Mantoux test was negative. The detailed baseline laboratory findings are presented in **Table 1**. The CT findings were further assessed using the CT-based severity parameters presented in **Table 2**. Diffuse bowel-wall thickening greater than 3 mm was present, with extensive involvement of the stomach, small bowel and colon. The characteristic target sign was identified, along with mesenteric vessel engorgement and mesenteric fat

stranding. Ascites was also present. Each of these findings contributed one point, resulting in a total CT severity score of 5. The findings collectively indicated extensive gastrointestinal involvement with prominent bowel-wall and mesenteric abnormalities, consistent with severe lupus enteritis. The autoimmune and immunological findings are summarised in **Table 3**. ANA was positive with a homogeneous pattern, while the anti-dsDNA antibody titre was markedly elevated at >800 IU/mL. Complement levels were reduced, with C3 of 40 mg/dL and C4 of 5 mg/dL, indicating hypocomplementemia. The extractable nuclear antigen profile was positive for anti-Sm, anti-RNP, anti-histone, anti-SSA (Ro), anti-SSB (La) and anti-nucleosome antibodies. Lupus anticoagulant was negative, while the direct Coombs test was positive. The urine protein-creatinine ratio was 1.54, indicating proteinuria. Taken together, these findings provided strong immunological evidence of active systemic lupus erythematosus with multisystem involvement and supported the diagnosis of lupus enteritis. Contrast-enhanced computed tomography demonstrated extensive gastrointestinal involvement with diffuse circumferential bowel-wall thickening and a characteristic layered mural enhancement pattern. **Figure 1** shows diffuse bowel-wall thickening with a characteristic target appearance on contrast-enhanced CT, suggestive of lupus enteritis. The bowel wall shows circumferential thickening with enhancement of the mucosal and outer mural layers and relative hypoattenuation of the intervening submucosal layer. This characteristic appearance, in association with the clinical presentation and other radiological findings, supported the diagnosis of lupus enteritis. A more detailed demonstration of the characteristic target appearance is shown in **Figure 2**. The image shows circumferential mural thickening involving the transverse colon with a characteristic target or double-halo appearance. The inner hyper enhancing mucosal layer, middle hypodense submucosal layer representing oedema and outer enhancing muscular is propria/serosal layer are clearly delineated. The presence of these distinct mural layers reflects bowel-wall oedema and inflammatory involvement and represents an important radiological feature of lupus enteritis. **Figure 3** further shows the layered mural enhancement pattern involving the small bowel. The image shows dilated jejunal and ileal loops with circumferential bowel-wall thickening and a characteristic target appearance. The mural layers are clearly visualised, with the circumferential wall thickening producing the characteristic layered enhancement pattern. The associated small-bowel dilatation indicates extensive intestinal involvement in this patient. In addition to small-bowel involvement, colonic involvement was also showed. **Figure 4** shows circumferential mural thickening involving the sigmoid colon with associated luminal gaseous distension. This finding shows that the inflammatory bowel involvement extended to the colon and was not restricted to the small intestine. The combination of bowel wall thickening, mural enhancement and bowel dilatation across multiple gastrointestinal segments showed the extent of intestinal involvement in this patient. The combination of systemic symptoms, haematological abnormalities, characteristic CT findings and positive autoimmune investigations supported the diagnosis of lupus enteritis associated with systemic lupus erythematosus. The patient had initially received empirical treatment for abdominal tuberculosis because of the prolonged fever, weight loss, gastrointestinal symptoms, pleural effusion, ascites and abdominal imaging abnormalities in a tuberculosis-endemic setting. However, the absence of microbiological evidence supporting tuberculosis and the persistence of clinical and radiological abnormalities prompted further autoimmune evaluation. Following establishment of the diagnosis, treatment with corticosteroids, hydroxychloroquine and cyclophosphamide resulted in significant improvement in the patient's symptoms and overall clinical condition.

Table 1: Baseline Laboratory Investigations

Investigation	Result	Reference Range	Interpretation
Hemoglobin	5.8 g/dL	12–15 g/dL	Severe anemia
Total leukocyte count	2740/µL	4000–11,000/µL	Leukopenia
Platelet count	104,000/µL	150,000–450,000/µL	Thrombocytopenia
Erythrocyte sedimentation rate (ESR)	110 mm/hour	<20 mm/hour	Elevated
C-reactive protein (CRP)	14.9 mg/L	<5 mg/L	Mildly elevated
Serum albumin	Low	3.5–5.0 g/dL	Hypoalbuminemia
Serum creatinine	Normal	0.6–1.2 mg/dL	Normal renal function
Mantoux test	Negative	Negative	No evidence of tuberculosis exposure
Urine protein-creatinine ratio	1.54	<0.2	Proteinuria

Table 2: CT-Based Severity Assessment in Lupus Enteritis

Parameter	Finding in this patient	Score
Bowel wall thickening (>3 mm)	Present (diffuse)	1
Extent of bowel involvement	Extensive (stomach, small bowel, colon)	1
Target sign (layered enhancement)	Present	1
Mesenteric vessel engorgement / fat stranding	Present	1
Ascites	Present	1
Total CT score		5

Table 3: Autoimmune Workup and Immunological Profile

Test	Result	Interpretation
ANA	Positive – homogeneous pattern	Supports autoimmune connective tissue disease and is mandatory entry criterion for SLE classification
Anti-dsDNA	>800 IU/mL	SLE-specific antibody indicating high disease activity

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C3 complement	40 mg/dL	Hypocomplementemia consistent with active immune-complex mediated disease
C4 complement	5 mg/dL	Further supports complement consumption in active SLE
LIA anti-Sm	Positive	Highly specific for SLE
LIA anti-RNP	Positive	Associated with connective tissue disorders and SLE
LIA anti-histone	Positive	Seen in SLE and drug-induced lupus
LIA anti-SSA (Ro)	Positive	Associated with SLE and Sjögren syndrome
LIA anti-SSB (La)	Positive	Supports systemic autoimmunity
LIA anti-nucleosome	Positive	Associated with active SLE and lupus nephritis
Lupus anticoagulant	Negative	No evidence of antiphospholipid syndrome
Direct Coombs test	Positive	Indicates autoimmune hemolysis
Urine protein-creatinine ratio	1.54	Low-grade proteinuria suggestive of early lupus nephritis

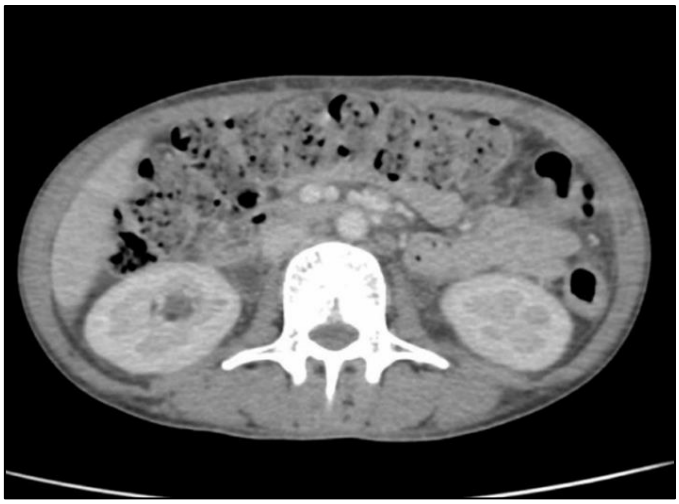

Figure 1: Contrast-enhanced image showing diffuse bowel wall thickening and target appearance, suggesting lupus enteritis

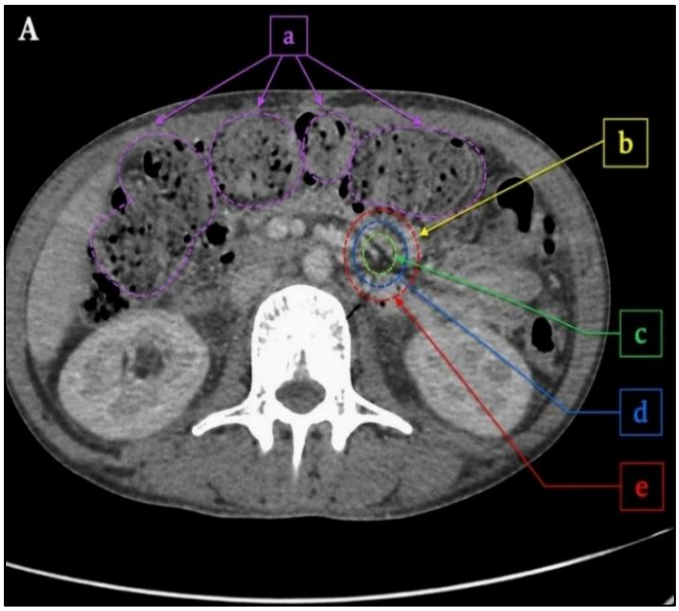

Figure 2: Target sign in lupus enteritis. The image shows circumferential mural thickening involving the transverse colon (**a**), producing a characteristic target or double-halo appearance (**b**). The target appearance is composed of an inner hyperenhancing mucosal layer (**c**), a middle hypodense submucosal layer representing oedema (**d**) and an outer enhancing muscularis propria/serosal layer (**e**).

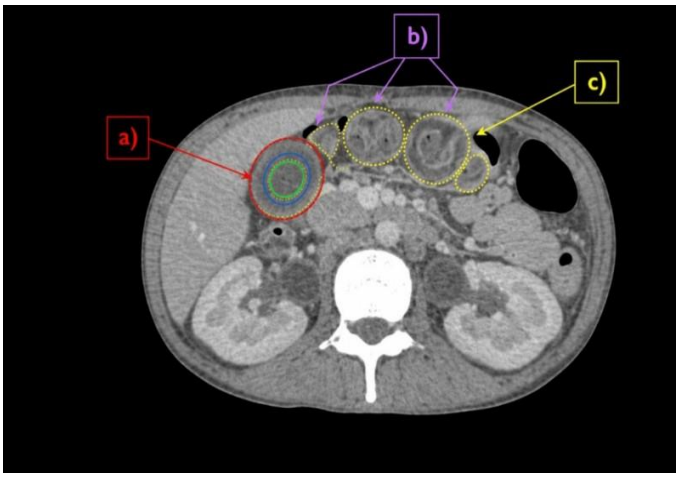

Figure 3: Layered mural enhancement in lupus enteritis. The image shows dilated small-intestinal loops involving the jejunum/ileum (a), with a characteristic target or double-halo appearance (b) and circumferential bowel wall thickening (**c**).

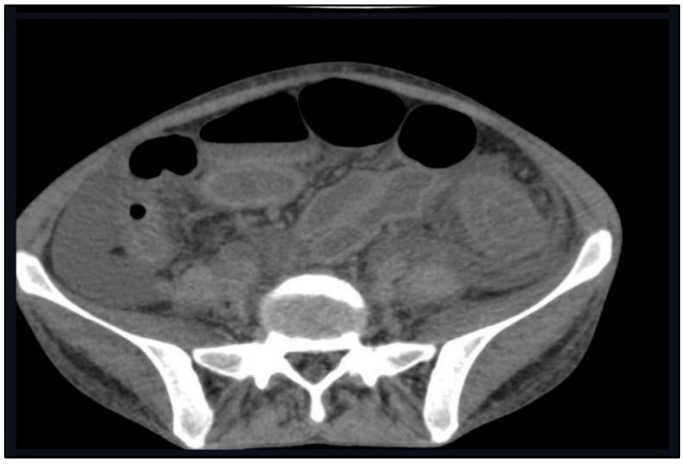

Figure 4: Sigmoid colonic involvement in lupus enteritis

Discussion:

Lupus enteritis is a rare, but clinically relevant, complication of systemic lupus erythematosus. This form of lupus is characterised by an immune-mediated inflammation of the bowel wall and vascular supply **[8, 9]**. The initial presentation of lupus enteritis is usually non-specific gastrointestinal complaints such as abdominal pain, vomiting, diarrhoea, abdominal distension, nausea and ascites, which can make diagnosis difficult in routine clinical practice **[10]**. In regions where

tuberculosis (TB) is endemic, the presence of systemic signs or symptoms like fever, weight loss, loss of appetite, serositis, lymphadenopathy, bowel-wall thickening can frequently mimic abdominal TB, delaying recognition of lupus enteritis and leading to inappropriate empirical anti-TB treatment **[11]**. Similarly, Huynh *et al.* reported an atypical presentation of systemic lupus erythematosus with gastrointestinal involvement in a tuberculosis-endemic region, where the disease closely mimicked abdominal tuberculosis and posed a significant diagnostic challenge before autoimmune evaluation established the correct diagnosis **[12]**.The patient presented in this case received empirical anti-TB treatment due to systemic signs/symptoms, pleural effusion, ascites and abdominal imaging findings indicating widespread TB. The absence of microbiological evidence and the gradual worsening of radiological findings raised suspicion for an autoimmune process. The pathogenesis of lupus enteritis is thought to involve immune-complex-mediated vasculitis, activation of complement, direct injury to the endothelial lining of the mesenteric vessels, thrombosis of mesenteric vessels and inflammatory cell infiltration of the mesenteric vascular system, resulting in increased bowel wall oedema and subsequent compromise to the blood supply to the bowel **[12]**. Persistent or extensive inflammation may ultimately lead to complications such as ischaemia, infarction of the bowel, perforations, bleeding diarrhoea and ultimately sepsis if diagnosis and treatment are delayed **[13]**. Anaemia, leucopenia, thrombocytopenia and hypocomplementemia are common haematological abnormalities associated with active systemic lupus erythematosus and all were present in the patient described in this case. This patient had severe bone marrow suppression, as evidenced by a haemoglobin level of 5.8 g/dL, leukopenia, thrombocytopenia, elevated inflammatory markers, decreased complement levels and substantial immunological stimulation. Radiological studies are very important for diagnosing lupus enteritis, especially when clinical symptoms are not definitive **[14]**. The best imaging technique for determining this diagnosis is by using contrast-enhanced computed tomography scans, which can provide characteristic features of lupus enteritis, such as bowel wall thickening, a layered mural enhancement "target sign", mesenteric vascular engorgement, mesenteric fat stranding and ascitic fluid, all of which support the diagnosis very strongly **[15]**. In this patient, a repeat contrast-enhanced CT performed to evaluate for lupus enteritis showed diffuse circumferential bowel wall thickening involving the stomach, small intestine and colon, with a characteristic target appearance and significant submucosal oedema. The presence of necrotising mesenteric adenitis and persistent ascites was a sign that pointed towards a diagnosis of lupus enteritis as opposed to abdominal TB. In addition, the CT severity score of 5 calculated from this image evaluation also indicated that this patient's gastrointestinal involvement was extensive enough to warrant aggressive immunosuppressive therapy. A serological test to screen for autoimmune disease in this patient was crucial for confirming the diagnosis **[16]**. The patient exhibited strong ANA positivity with a homogeneous pattern, a very high anti-dsDNA antibody titer (>800 IU/mL) and positive anti-Sm, RNP, SSA, SSB, histone and nucleosome antibodies, as well as depressed C3 and C4 complement levels. The presence of a positive direct Coombs test and low-grade proteinuria provides additional evidence for the active, multisystemic component of Systemic Lupus Erythematosus in this patient. The extensive autoimmune positivity and gastrointestinal findings strongly support the diagnosis of lupus enteritis. The primary treatment of lupus enteritis is prompt initiation of systemic corticosteroid and immunosuppressive therapy to prevent irreversible bowel injury and systemic complications **[17]**. Severe cases of lupus enteritis often require IV pulse methylprednisolone, followed by corticosteroids and additional immunosuppressive agents such as cyclophosphamide **[18]**. Severe lupus enteritis was effectively treated with IV pulse methylprednisolone, dexamethasone, hydroxychloroquine and cyclophosphamide, resulting in rapid improvement for this patient, with complete resolution of abdominal pain, vomiting, obstruction, oedema and constitutional symptoms, thus avoiding surgical intervention and validating the effectiveness of early, aggressive immunosuppressive therapy **[19, 20]**. Recent literature further supports the importance of recognising gastrointestinal involvement as a clinically significant manifestation of systemic lupus erythematosus. A comprehensive review published in 2026 highlighted lupus enteritis and mesenteric vasculitis as major gastrointestinal manifestations of SLE and emphasised that nonspecific symptoms such as abdominal pain, diarrhoea, vomiting and weight loss may delay recognition of the underlying autoimmune disease **[21]**. These observations are particularly relevant to the present case, in which constitutional and gastrointestinal manifestations initially suggested abdominal tuberculosis. A 2026 case report and literature review of lupus enteritis further demonstrated that acute abdominal presentations may represent active lupus-related intestinal disease and reinforced the importance of integrating clinical findings with characteristic imaging and immunological investigations **[22]**. The present case is consistent with these recent observations and further highlights the diagnostic challenge posed by the coexistence of a tuberculosis-endemic epidemiology and radiological features of intestinal inflammation. Early recognition of the target sign, assessment of complement levels and lupus-specific autoantibodies and consideration of multisystem manifestations may therefore facilitate differentiation of lupus enteritis from infectious abdominal conditions and allow timely immunosuppressive treatment **[23]**. This case emphasises the need for young women with unexplained abdominal symptoms, cytopenia, serositis and constitutional symptoms in a region where TB is endemic to maintain a very high index of suspicion for autoimmune GI disease. Through clinic radiological correlation, extensive autoimmune evaluation and expedited initiation of immunosuppressive therapy can significantly lower morbidity and prevent life-threatening complications resulting from lupus enteritis.

Conclusion:

Lupus enteritis should be considered in patients with unexplained gastrointestinal symptoms and systemic features suggestive of SLE, particularly in tuberculosis-endemic regions. Early recognition using characteristic CT findings together with autoimmune serology facilitates timely immunosuppressive treatment, prevents unnecessary antitubercular therapy and reduces the risk of serious complications.

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