Leaky gut and silent kidneys – a challenging presentation in systemic lupus erythematosus – What, Why and How?

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Abstract

Systemic lupus erythematosus (SLE) is an autoimmune multisystem disease predominantly affecting young females. Its presentation varies from mild musculoskeletal, muco-cutaneous involvement to life and organ threatening manifestations like renal failure, diffuse alveolar haemorrhage, hemophagocytic lymphohistiocytosis (HLH) and organic brain syndrome. Even though it is a female predominant disease, males tend to be higher severity, more renal involvement, higher mortality and delay in diagnosis. Acute presentation of gastroenteritis with renal failure have differentials like infections causing pre-renal azotemia, hemolytic uremic syndrome, systemic vasculitis and certain toxins, poisonings or drugs. We present case of a young male with diarrhea and acute renal failure, later diagnosed and managed as systemic lupus erythematosus.

Key words: lupus, lupus enteritis, lupus nephritis, belimumab, mimics

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Introduction

Systemic lupus erythematosus (SLE) is a systemic autoimmune rheumatic disease predominantly affecting young females[1]. Its presentation varies from mild musculoskeletal, muco-cutaneous involvement to life and organ threatening manifestations like renal failure, diffuse alveolar haemorrhage, hemophagocytic lymphohistiocytosis (HLH) and organic brain syndrome[2]. Even though it is a female predominant disease, males tend to be higher severity, more renal involvement, higher mortality and delay in diagnosis.[1,2] Acute presentation of gastroenteritis with renal failure have differentials like infections causing pre-renal azotemia, hemolytic uremic syndrome, autoimmune diseases and certain toxins, poisonings or drugs[3]. We present case of a young male with diarrhea and acute renal failure, later diagnosed and managed as systemic lupus erythematosus.

Case summary

30 year Indian male, came to emergency with history of non-bloody, non-foul smelling, scanty amount, diarrhea for 5 days with mild post prandial abdominal pain, without tenesmus or abdominal distension. He also complained of reduced urine output for 3 days. He had fever for 2 days prior to onset of these symptoms, however, he denied any skin rash, yellowish discoloration of sclera or urine, hematuria, froth urine, pedal edema or facial puffiness. He also denied any recent travel, over the counter or unauthorized medications or drugs usage, any illicit substance usage and promiscuity. On examination, he had tachycardia and tachypnea, without any positive abdominal findings. On investigation, there were normocytic normochromic anemia, leucopenia, thrombocytopenia, raised creatinine, hyperkalemia, hyponatremia and elevated liver enzymes (aspartate and alanine transaminases). On ultrasound imaging, there was increased echogenicity of bilateral kidneys with maintained cortico-medullary differentiation, mild bilateral pleural effusion and ascites. In view of acute gastroenteritis and renal failure with dyselectrolytemia, he was suspected to have infection as underlying cause and treated accordingly with antibiotics along with initiation of hemodialysis. On further work up, he had high lactate dehydrogenase, low complements level (both C3 and C4), normal stool analysis with normal fecal calprotectin, normal echocardiogram and negative culture in blood and stool. Based on these, suspicion of hemolytic uremic syndrome (HUS), thrombotic thrombocytopenic purpura (TTP) or autoimmune pathologies were considered. His plasmic score was 4 however, in view of high mortality associated with TTP, he was initiated on daily plasma exchange therapy along with intravenous methylprednisolone (1000mg) pulse therapy.

In the mean time, his anti-nuclear antibody (ANA) by indirect immunofluorescence was positive of 4+ intensity in mixed homogenous and fine speckled pattern, corresponding titer was 1:5120. He also had 3+ positivity for direct Coomb's test along with very high titer of anti-double stranded deoxyribonucleic acid (ds-DNA) levels. On retrospective inquiry, he affirmed to having multiple episodes of painless lingual and palatal ulcer for 1 year however, he denied history of any lupus like rash, photosensitivity, arthralgia, alopecia, Raynaud's phenomenon, muscle weakness or sicca symptoms. His extractable nuclear antigens (ENA) panel by line blot assay showed positivity for anti ds-DNA, anti SSA, anti-histone and anti-nucleosome antibodies. His peripheral smear was not shown of any schistocytes with normal bilirubin and haptoglobin, which essentially excluded TTP and HUS. Hence after 3 session of plasma exchange, it was discontinued. It was beyond doubt that patient was active systemic lupus erythematosus (SLE) with SLE-disease activity index2k (SLEDAI2K = 18). His loose stool were persistent despite antibiotics and steroid, hence computed tomography (CT) of abdomen was done which showed ascites with small bowel and omental thickening along with mesenteric congestion suggestive of mesentric vasculitis – likely lupus enteritis (Figure 1). His colonoscopy showed diffuse edema without any ulcerations or erosions, biopsy of same revealed mild lymphocytic infilteration without any granuloma, cryptitis, crypt abcess, microscopic colitis or malignant changes. He also underwent renal biopsy which confirmed class III/IV lupus nephritis (Figure 2).

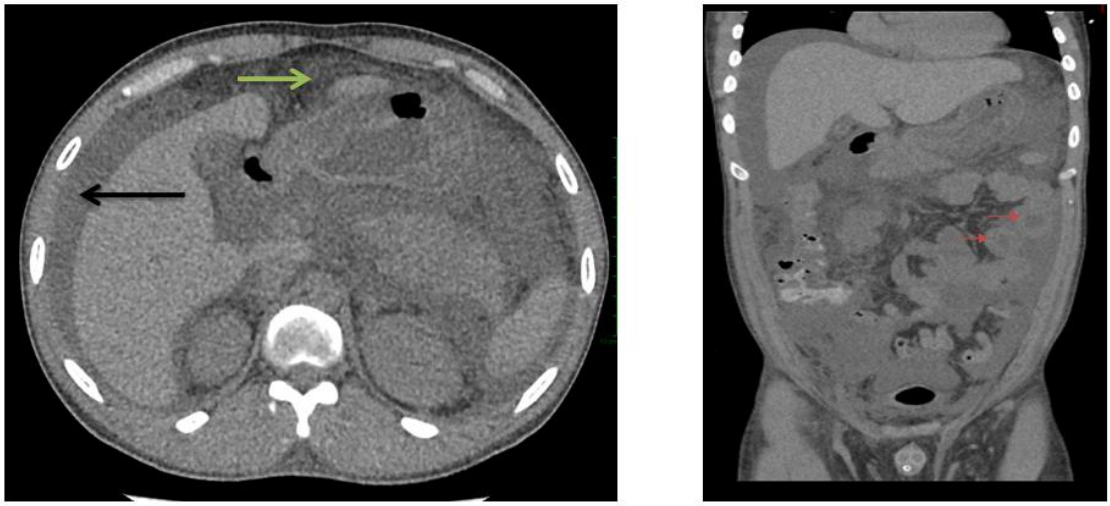


Figure 1 A: Abdominal CT axial view showing diffuse ascites (black arrow) with omental thickening (green arrow) and mesenteric congestion.

Figure 1 B: Abdominal CT coronal CT view showing small bowel thickening with ascites and mesenteric congestion, giving Target Sign appearance (red arrows).

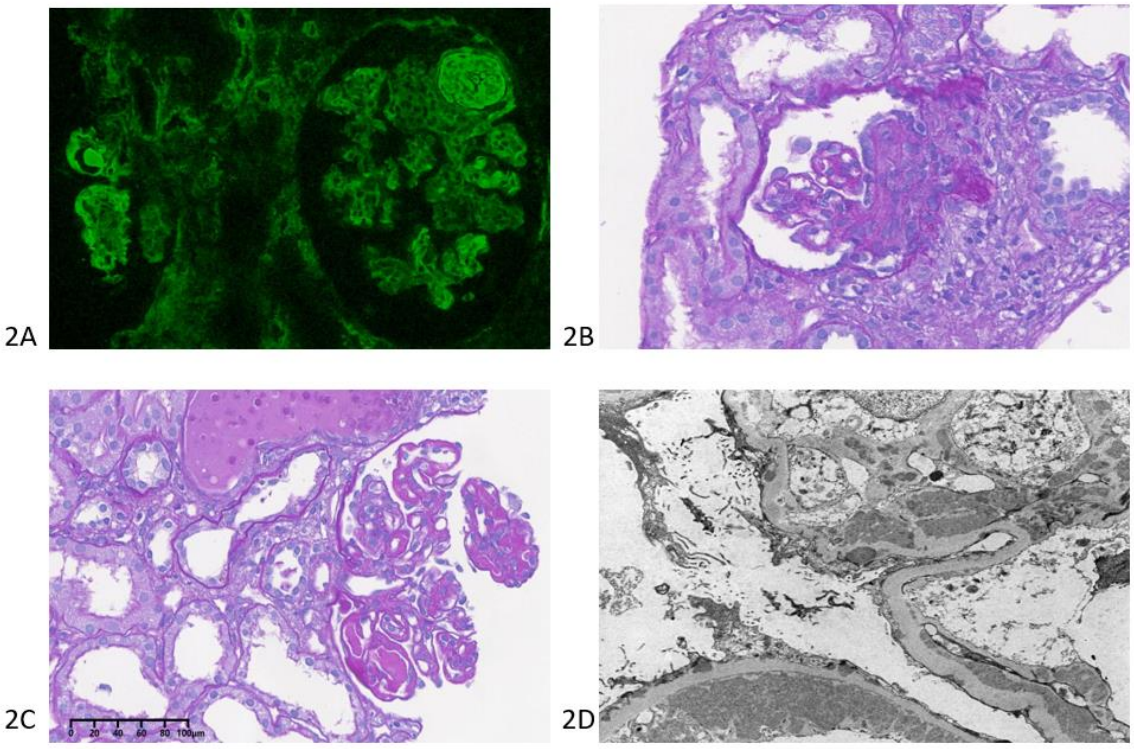


Figure 2 The renal biopsy contained three glomeruli only, in which wire loop lesion and hyaline pseudo thrombi (A) were appreciated, along with equivocal fibro cellular crescent (B). The immunofluorescence study has shown a full-house staining of IgG, IgM, IgA, C3, C1q (C), in addition to Kappa and Lambda light chains. The ultrastructural examination revealed diffuse mesangial and sub endothelial electron dense deposits (D). The histopathologic diagnosis is proliferative glomerulonephritis, highly suggestive for lupus nephritis (ISP/RPS classes between III and IV. No significant tubulointerstitial scarring or vascular disease were identified.

By day 5 of admission, his leucocyte count had normalised, with improvement in hemoglobin and platelets level. Once diagnosis of SLE was confirmed, he was continued on oral prednisolone along with hydroxychloroquine. He received renal dose modified injection of cyclophosphamide (700 mg.) for remission induction on day 5 along with injection of belimumab (10 mg/kg.) on day 10. By day 13, his altered bowel movements had settled along with complete normalisation of his hemoglobin, leucocyte and platelets count. His urine output had improved as well, however he still required few more sessions of hemodialysis prior to his discharge on day 18.

He is under follow up for last 1 year and has completed his 6 monthly pulses of high dose injection of cyclophosphamide. He was later maintained on oral mycophenolate mofetil and monthly doses of injection of belimumab. His renal functions normalised by fourth month and he had no further episodes of altered bowel or fall in blood counts. His immunological parameters also showed gradual improvement over time.

Table 1 Laboratory parameters on admission, during hospital stay and follow up of index case

Parameters	Admission	Day 3	Day 5 (post 3 days steroid pulse and 3 sessions of plasma exchange)	Discharge – Day 18	Follow up – 3rd month	Follow up - 6th month	Follow up - 12th month
Hemoglobin (gm/dl)	7.2	8.3	8.6	9.4	10.2	10.8	12.4
Platelets (number x 10^9 / uL)	18	26	54	96	118	220	236
C- reactive protein (mg/dl)	21.8	18.4	11.2	7.4	5.3	2.07	2.03
Creatinine (µmol/L)	342	365	329	216	184	118	108
Lactate dehydrogenase (IU/ml)	637	621	328	239	184	162	164
Aspartate aminotransferase (U/L)	128	184	96	72	38	47	41
Alanine aminotransferase (U/L)	106	117	64	41	26	32	38
C3 (mg/dl)	45			56	74	106	112
C4 (mg/dl)	2			6	10	12	9
Anti DsDNA (IU/ml)	389			312	246	94	87

Discussion

Unlike lupus nephritis, which occurs due to deposition of immune mediated complexes in the glomerulus leading to inflammation, crescents formation and necrosis, lupus enteritis is a vasculitis affecting mesenteric vasculature. It can present as non infectious gastroenteritis and colitis to severe manifestation like pseudo-obstruction, intestinal hemorrhage, perforation and infarctions[4]. With acute renal failure, cytopenias and high inflammatory burden, identification of lupus enteritis becomes challenging and etiologies like hemolytic uremic syndrome, thrombotic thrombocytopenic purpura, disseminated intravascular coagulation, catastrophic antiphospholipid syndrome, thrombotic microangiopathy (TMA) – complement mediated or post infectious, infections like bacterial endocarditis, cytomegalovirus or toxins and poisoning related multi-organ dysfunction need to be ruled out[5,6,7,8].

Lupus enteritis (LE) has been pathologically categorized as inflammatory or thrombotic. Inflammatory LE is classically vasculitis in the mesenteric vasculature due to immune complex

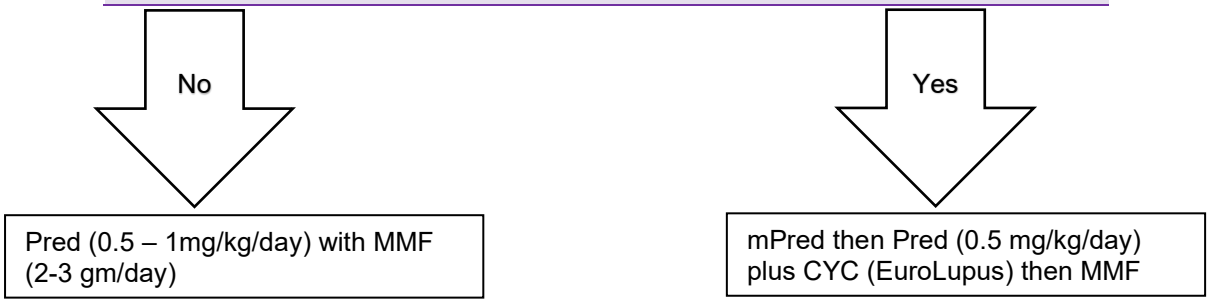
deposition causing inflammation, complement activation and increasing gut permeability. Thrombotic LE occurs in the setting of identified anti-phospholipid (aPL) antibodies or thrombus in the blood vessels. However, current data on correlation between aPL antibodies and occurrence of LE is not robust.[4,9]

As per recent review of literature[4], half of patients with LE have loose stools as a presenting complaint. Most common complaints are abdominal pain, nausea and vomiting. Recurrence can occur in 20-30% patients, mostly within two years of disease onset. Presence of target sign (representing bowel wall thickening), comb sign (mesenteric vessel fullness and enhancement) and attenuation of mesenteric fat are three most commonly described findings on contrast enhanced computed tomographic imaging in cases with suspected LE. These signs are however, not specific for LE and have also been described with other intestinal pathologies like intestinal pseudo-obstruction, inflammatory bowel disease, peritonitis, pancreatitis and intestinal obstructions. Occurrence of target sign with renal failure and pancytopenia in our index case lead to suspicion for some underlying systemic disease.

A recent consensus[10] on management of rare SLE manifestations have laid down few clinical features that suggest severity of LE (table 2). As per these elements, our case had severe LE requiring hospitalization with abdominal pain, diarrhea, inability to eat and dyselectrolytemia. Management of LE is also guided based on severity with intravenous (IV)/oral steroid, cyclophosphamide, mycophenolate and/or rituximab as alternative.[10]

Table 2 Severity criteria based management of Lupus Enteritis as per ERN-ReCONNET- SLICC-SLEuro expert consensus[7]

Presence of Severity Criteria
1) Digestive perforation or ischemia
2) Digestive hemorrhage
3) Severity of symptoms, generalized edema, weight loss and inability to eat
4) Sepsis due to bacterial translocation
5) Extent and severity at imaging
6) Severe hypoalbuminemia (eg < 20G/L) or severe dehydration or ion abnormalities
7) Requiring hospitalization



mPred – methylprednisolone infusion, Pred- Prednisolone oral (or equivalent), CYC- cyclophosphamide, MMF- mycophenolate mofetil

Other intestinal diseases that can have similar presentation in SLE patients are microscopic colitis, protein losing enteropathy, cytomegalovirus related colitis and inflammatory bowel disease. Also to remember that infections, toxins, drugs and extra-intestinal causes like nephrotic syndrome, endocarditis, anti-phospholipid syndrome can also mimic LE. A close observation of all available evidences may lead to early diagnosis and management in such challenging scenarios (table 3)

Table 3 Mimics of Lupus enteritis – clinical features, endoscopic, imaging and histopathology findings to differentiate them

CLINICAL CONDITION	MANIFESTATION	INVESTIGATION	ENDOSCOPY[1]/ RADIOLOGY[2]	HISTOPATHOLOGY
CMV COLITIS[14]	Bloody diarrhoea, abdominal pain, fever, weight loss, diffuse tenderness, malnutrition	Cytopenia, positive CMV PCR	1. Mucosal ulcers, Pseudo membranes, Diffuse inflammation 2. Bowel wall thickening and ulcers	Inclusions (OWL EYE) in endothelial; and stromal cells and necrosis
SMALL INTESTINAL BACTERIAL OVERGROWTH (SIBO)[18]	Chronic diarrhoea, Bloating, Malabsorbtion, Worsning after meal, Nail changes, hair fall, Weight loss, Arthralgia, Muscle loss	Positive Hydrogen-methane breath test, Elevated folate, Low B12	1. Normal or mild mucosal edema 2. Normal or mild small bowel dilation	Non-specific mild inflammation
ISCHEMIC COLITIS[19]	Acute abdominal post prandial pain, Bloody diarrhoea, Urgency in passing stools, Abdominal tenderness, Rectal bleeding, Ischaemic Hepatitis, ascites	Anaemia, Leucocytosis, Elevated Lactate	1. Segmental inflammation, mucosal edema, and “thumb-printing” due to submucosal hemorrhage; possible ulceration 2. “Thumbprinting” (submucosal edema) bowel wall thickening, pneumatosis intestinalis	Mucosal necrosis, hemorrhagic lamina propria, hyalinization of vessels; no granulomas and chronic inflammation
CELIAC DISEASE[15]	Chronic Diarrhoea, Bloating, Weight loss, Dermatitis Herpetiformis, weight loss,malnutrition, Myalgias, Arthralgias	Positive-anti tTG igA, anti endomysial antibody, Low iron, Vit-D	1. Scalloping of duodenal folds, mosaic pattern, or loss of folds; possible erosions in severe cases 2. Small bowel dilation and reversal of normal jejunal-ileal fold pattern	Villous atrophy, increased intraepithelial lymphocytes, crypt hyperplasia
INFLAMMATORY BOWEL DISEASE (IBD)[16]	Chronic diarrhoea (Bloody in UC, Watery in crohns), Abdominal pain, weight loss, arthritis, Uveitis, Erythema nodosum, Pyoderma gangrenosum, Perianal fistulae	Anaemia, Elevated fecal calpotectin, Elevated ESR, CRP	1-Ulcers, skip lesions, and cobblestone appearance (CD); continuous mucosal inflammation with friability and pseudopolyps (UC) 2-wall thickening, fistulae, or strictures (CD); Pancolitis (UC)	Chronic inflammation- crypt distortion (Most important finding), granulomas (CD), or crypt abscesses (UC), transmural inflammation
MICROSCOPIC COLITIS[11]	Chronic watery diarrhoea, Abdominal pain, Weight loss, Nocturnal diarrhoea, Worsened by medications	Non specific or high (CRP, ESR)	1. Normal or mild erythema 2. Bowel thickning or non specific structural changes	increased intraepithelial lymphocytes, thickened sub epithelial collagen

				band or mixed inflammation
PROTEIN LOSING ENTEROPATHY[13]	Chronic diarrhoea, ascites, anasarca, Weightloss, malneutrition, Peripheral oedema	Hypoalbuminaemia, Lymphopenia, anaemia, Electrolyte imbalance,	1. Lymphangiectasia, Normal or mild oedema 2. Mild oedema, Dilated lymphatic vessels	Dilated lymphatic channels, Mucosal oedema, Loss of villous architexture
MEDICATIONS INDUCED DIARRHEA	Diarrhoea due to medications (NSAIDs, Mycophenolate, Hydroxychloroquine), Mild abdominal discomfort, Not much signs	Normal	1. Normal or mild non-specific inflammation (e.g., NSAID-induced erosions) 2. mild non-specific bowel wall changes and erosions on imaging	Non-specific mild inflammation, drug-specific injury (apoptosis with mycophenolate)
IRRITABLE BOWEL SYNDROME (IBS)	Chronic diarrhoea, Mixed bowel habits, Bloating, No weight loss	Normal	1. Normal; no structural abnormalities 2. Normal	Normal mucosa

CMV – cytomegalovirus, PCR – polymerase chain reaction, CD- Crohn's disease, UC- ulcerative colitis, IgA tTG – IgA tissue transglutaminase antibody, ESR – erytheocyte sedimentation rate, CRP – C reactive protein, NSAID – non steroidal anti inflammatory drugs

Microscopic colitis which includes lymphocytic colitis and collagenous colitis is a rare occurrence in SLE.[11] Of the two variants, collagenous colitis occurs more commonly in autoimmune diseases including SLE. It is characterized by chronic watery diarrhea with normal colonic mucosa on endoscopy. Microscopic examination shows either subepithelial collagen deposition (collagenous) or intraepithelial lymphocytic infilteration (lymphocytic). Non steroidal anti-inflammatory drugs (NSAID) are implicated as potential offending agent. Management aims at stopping any offending agent and symptomatic treatment. Some patients may require steroid, cholestyramine or sulfasalazine. Severe intractable cases may need surgery.[11]

Protein losing enteropathy (PLE) is another clinical condition that requires high index of suspicion and is always a diagnosis of exclusion. It is characterized by severe hypoalbuminemia (serum albumin < 3 gm/L) with normal urinary protein excretion (24 hour urinary protein < 0.8 gm/day). Patients can present with diarrhea, abdominal pain or distension, pedal or peri-orbital edema and in severe cases as anasarca. Routine tests like endoscopy, CT abdomen, gastrointestinal mucosal biopsy are either normal or have non specific changes. Diagnosis is established by advanced imaging like Technetium (Tc)-99 albumin scintigraphy and stool alpha-1 anti trypsin clearance. Prevalence in SLE is reported to be only 3.2%, mostly seen in Asians.[12,13] Other similar conditions like celiac disease, tropical sprue, Menetrier's disease, small intestinal bacterial overgrowth (SIBO), congestive cardiac failure and intestinal lymphangiectesia needs to be ruled out.[13] Most patients with PLE associated with SLE have a dramatic response to corticosteroid, rarely requiring other immunosuppressants. [12]

Cytomegalovirus (CMV) infection is a great masquerader of SLE. It can not only mimic SLE but also can trigger flares and relapses in SLE patients. SLE patients are prone for infections even without any immunosuppressive medications, risk of which is definitely multiplied after treatment. CMV can cause multisystem disease including colitis and pancytopenia. Diagnosis requires high titer of CMV IgM antibodies or positive CMV polymerase chain reaction (PCR) with/out inclusion bodies either in the colonic or bone marrow biopsy. Management requires use of anti viral agents like ganciclovir, valganciclovir, foscarnet and cidofovir.[14]

Inflammatory bowel disease (IBD) and celiac disease are other immune mediated diseases that can coexist in SLE patients. As per recent meta-analysis prevalence celiac disease in SLE patients is comparable to general population and routine screening is not recommended.[15] Literature describes ulcerative colitis in < 1% SLE patients, Crohn's disease is even rare. Treatment used in managing SLE like steroid, azathioprine control IBD as well. Nevertheless, drugs used in managing IBD like sulfasalazine or anti tumor necrosis factor therapy (TNF) can trigger drug induced lupus or lupus like phenomenon in few patients. Endoscopy and intestinal biopsy can confirm the diagnosis of IBD and celiac disease.[16]

On the other hand, LE has non specific endoscopic and biopsy findings. Management of LE requires steroid along with other immunosuppression, decision of which is individualized based on severity and systemic involvement.[10] Most patients with only LE improve with steroid and hydroxychloroquine. In refractory cases or recurrent exacerbations of LE, other therapy like mycophenolate mofetil, ciclosporin, tacrolimus, rituximab, plasmapheresis have also been used[4,9]. Intestinal perforation, hemorrhage or ischemia leading to gangrene may warrant urgent surgical management.[4,9] A recent observational study [17] showed that SLE patients with mesenteric vasculitis had more abdominal pain, higher ESR, LDH and bowel thickening with other CT abnormalities.

Conclusion

This case highlights that lupus can have acute presentation affecting multiple organs simultaneously. High index of suspicion for an autoimmune pathology is crucial for early diagnosis and management. For rare phenomenon like LE, mimics need to be ruled out especially infections. Always to remember that time is the driving factor in management of multi-system diseases that portend high mortality and morbidity risk.

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Statement of ethics

The above study involving human participants were planned, conducted, and reported in accordance with the World Medical Association (WMA) Declaration of Helsinki

Patient informed consent: Written informed consent was obtained from the patient to publish their case and any accompanied images

Disclosure statement: Affirm that there is no financial disclosure, non-financial relationship and activities.

Conflict of interest

The authors declare that there is no conflict of interest.

Artificial intelligence (AI) disclosure statement

The authors confirmed that this is an AI unassisted work

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Authors' contribution

DM, ML, AP, AT, PG were involved in active management of this case. SS and AA reported the histopathology images. MA reported the radiology scans. DM, ML and AP were involved in drafting the manuscript. All authors were involved in proof reading, editing and finalizing the manuscript**.**

Data sharing statement

Data sets are not available publicly because of legal/security/privacy/policy reasons. However, it is available by request from the correspondence author.

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