Case Report

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Protein-losing enteropathy as the first presentation of systemic lupus erythematosus the first case reported in Palestine with systemic review

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Introduction: Systemic lupus erythematosus is a chronic inflammatory disease affecting women, causing gastrointestinal issues like acute pancreatitis, esophagitis, and protein-losing enteropathy. Protein loss is uncommon, but a case study shows protein-losing enteropathy as a first sign.

Importance: Protein-losing enteropathy (PLE) is a rare gastrointestinal manifestation of SLE, often seen years before diagnosis. Suspecting hypoalbuminemia without protein loss is crucial, but diagnosis is challenging due to imaging and histological findings.

Case presentation: A 22-year-old woman with epigastric pain experienced sudden abdominal pain, vomiting, and arthralgia. An abdominal CT scan revealed moderate bilateral hydronephrosis, pelvic free fluid, a kidney stone, and a cyst. Laboratory tests showed normal hemoglobin levels, platelets, white blood cells, C-reactive protein, and ESR. Diagnosis of SLE was confirmed, and pulse steroid therapy and hydroxychloroquine were initiated. Severe ascites required pigtail insertion, and ceftazidime was added. The patient's condition improved, and she was discharged with regular follow-ups.

Clinical discussion: A 22-year-old female was diagnosed with protein-losing enteropathy, a rare gastrointestinal manifestation of systemic lupus erythematosus (SLE). The disease is divided into mesenteric vasculitis, pseudo-obstruction, and protein loss enteropathy. The patient also had severe enteritis, abdominal pain, nausea, and diarrhea. The study found that the main complaint was abdominal pain with dysphagia, mainly due to active SLE inflammation. The patient responded well to treatment, with a 62.5% rapid improvement in pulse steroids and a cure for underlying causes through DMARDS or immunosuppressant drugs.

Conclusions: The case presents a rare SLE diagnosis with gastrointestinal involvement, pleural effusion, and progressive swelling. Despite correct diagnosis and aggressive treatment, clinical improvement occurred, requiring high clinical suspicion.

Keywords: ascities, hypoalbuminemia, low-income nation, Meigs syndrome, protein-losing enteropathy, pancytopenia, systemic lupus erythematosus

Introduction

Systemic lupus erythematosus (SLE) is a chronic multisystemic inflammatory disease of unknown etiology that predominantly affects women of reproductive age, marked by antibodies against nuclear and cytoplasmic antigens[1]. Up to 40% of SLE patients experience gastrointestinal issues at some point in their lives due to active disease, side effects of the medication, or other intercurrent processes, involving any organ along the gastrointestinal tract, most commonly the pancreas (e.g. acute pancreatitis). Other manifestations include esophagitis, intestinal pseudo-obstruction, protein-losing enteropathy (PLE), lupus hepatitis, mesenteric vasculitis or ischemia, and peritonitis[2]. PLE is characterized by a severe loss of serum proteins in the gastrointestinal tract, resulting in hypoalbuminemia manifested as ascites, diarrhea, and generalized edema[3]. PLE is suspected when serum albumin is <3.0 g/l with normal urinary protein excretion.

HIGHLIGHTS

- Systemic lupus erythematosus is a multisystem autoimmune disease with a diverse array of clinical and serological manifestations.
- Majority of gastrointestinal symptoms are related to adverse medication reactions or infection.
- Protein-losing enteropathy is, although rare, it has been reported as the initial presentation of systemic lupus erythematosus.
- Protein-losing enteropathy associated systemic lupus erythematosus flare has the good and rapid response to pulse steroids.

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Protein loss through GIT can be confirmed by increased alpha1-antitrypsin clearance in the stool or by Tc-99m albumin scintigraphy, when these tests are not available, PLE is a diagnosis of exclusion[4]. It is uncommon for SLE to present with protein-losing enteropathy as the initial presentation. Therefore, we would like to present a case study of a young female patient who exhibited protein-losing enteropathy as the first sign of SLE[4]. 'This case report has been reported in line with the Surgical CAse Report (SCARE) criteria[5]'.

Case presentation

A 22-year-old woman with a free past medical history, presented with a 3-day history of epigastric pain, of sudden onset, sharp and colicky, radiated to all over the abdomen and more to the flanks, gradual in the course, worsened with time, as it became worse in the day of admission, exacerbated by movement and relived partially with analgesics, accompanying with loss of appetite, decrease oral intake, constipation for 2–3 days with no passing of stool or flatus and vomiting up to 6 times of gastric content. Three weeks before admission, she had bilateral flank pain associated with vomiting. An abdominal CT scan was done and she was found to have moderate bilateral hydronephrosis, mild pelvic free fluid, a left nonobstructing kidney stone, and an adnexal mass suggestive of a simple cyst (Fig. 1). Therefore, she was admitted to the urology department and treated conservatively as a case of UTI vs PID. The patient has reported having joint pain involving the MCP, PIP, wrists, and knees, pain is worse in the morning and is accompanying with swelling and erythema. There is a history of autoimmune disorders in her family. Her aunt has rheumatoid arthritis. The patient has no significant hair loss, photosensitivity rashes, or malar rash. There is no history of miscarriages. There is no history of Raynaud's phenomenon, dry eyes, or redness of the eyes. Examination revealed generalized abdomen tenderness, particularly in the hypogastric area. She has a petechial rash on her chin. She has no pallor or jaundice. No photosensitivity rashes or malar rashes were noted. There are no oral ulcers or palpable lymph nodes. Heart auscultation revealed a dual rhythm with no murmurs, and jugular venous pressure was not raised. Respiratory examination revealed bilateral equal vesicular breath sounds without adventitious sounds. Neurological examinations showed no abnormalities, including assessments of the upper and lower limbs and cranial nerves. Joint examinations did not reveal any signs of joint swelling, tenderness, or deformity.

Laboratory studies (Table 1) revealed hemoglobin of 11.8 g/dl, platelet of 60 000/μl, white blood cell (WBC) of 4400/μl, c-reactive protein (CRP) was 2.7 mg/l, and erythrocyte sedimentation rate (ESR) was 20 mm/h. Her serum creatinine was 0.7 mg/dl. Her alanine aminotransferase (ALT) and aspartate aminotransferase (AST) were normal. Her serum albumin was low (2.2 g/dl) with a total protein of 5.2 g/dl. Urine analysis reveals no proteinuria or hematuria. ANA was positive. She underwent an abdomen and pelvis CT scan with IV contrast which showed diffuse circumferential wall thickening of the small bowel (Fig. 2). Intra-abdominal free fluid. The right adnexal cystic lesion measuring about 2.2 cm mostly represents an ovarian cyst. Moderate bilateral hydronephrosis. Bulky uterus with congested pelvic vessels. Multiple prominent parailiac lymph nodes. There is a left adnexal hyperdense rounded structure measuring 2.2 cm, suggestive of a hemorrhagic cyst. Additionally, bilateral well-defined hypoechoic breast lesions, primarily fibroadenomas, were identified on breast ultrasound. Consultation with the gynecology team indicated normal folliculogenesis, with a 2.2 cm ovarian follicle considered a typical finding for the patient's age, ruling out a ruptured ovarian cyst and ectopic pregnancy. No gynecological procedures were deemed necessary. Since the patient had clinical features

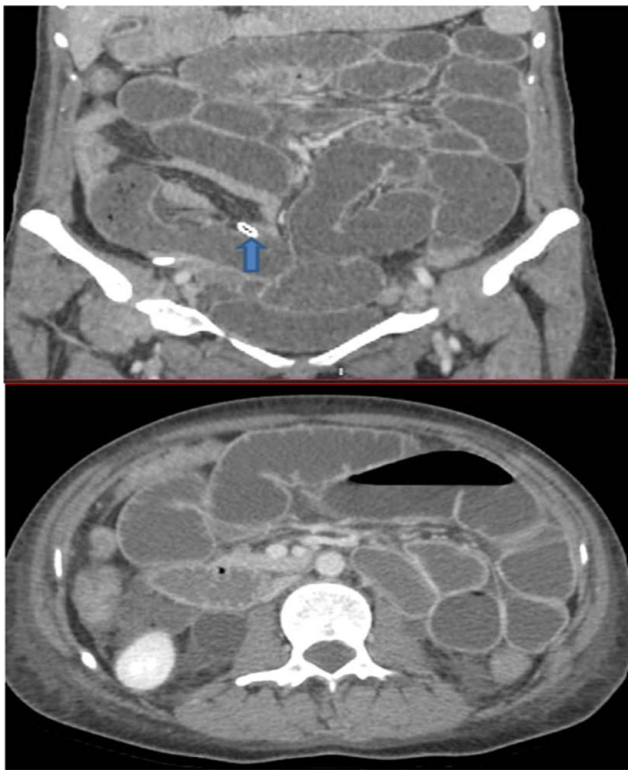

Figure 1. Abdomen and pelvis CT scan with IV contrast (axial view). Moderate bilateral hydronephrosis (black arrow) associated with thickening and enhancement of both ureters along their course, mainly at the right side, accompanied by periureteric fluid tracking along the course of the right ureter; however, no definite obstructive cause was detected. Multiple prominent parailiac lymph nodes up to 0.9 cm in the short axis are likely reactionary.

Table 1

Summary of clinical investigations and values.

Labs	Values
Hb%	11.8 g/dl
PLt	60 000/μl
WBC	4400/μl
CRP	2.7 mg/l
ESR	20 mm/H
Cr	0.7 mg/dl
AST and ALT	Normal
Albumin	2.2 g/dl
Total protein	5.2 g/dl
ANA	Positive (more than 10 index)
Antidouble strand DNA (ds-DNA)	Positive (5.1)
C3 and C4	26 mg/dl and 2.9 mg/dl, respectively
U1- snRNP	Positive
Anti-SSA and Anti-SSB	Positive
Anti-SM	Positive
Anticardiolipin	Positive

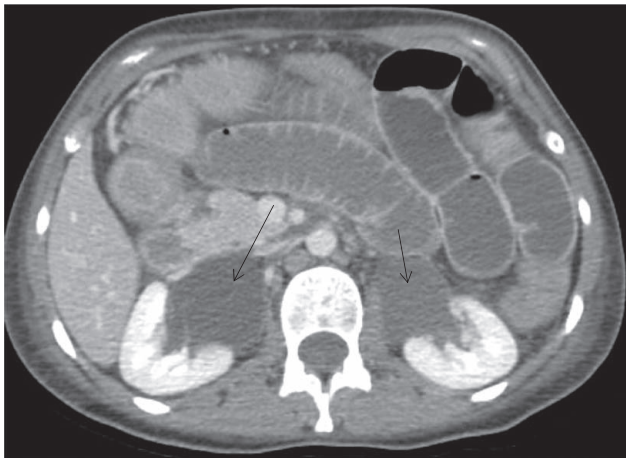

Figure 2. Abdomen and pelvis CT scan with IV contrast (coronal view and axial view, respectively), which showed diffuse circumferential wall thickening with a white pattern of enhancement involving the small bowel. The blue arrow showed ultrasound-guided pigtail catheter drainage for severe to moderate amounts of ascetic fluid.

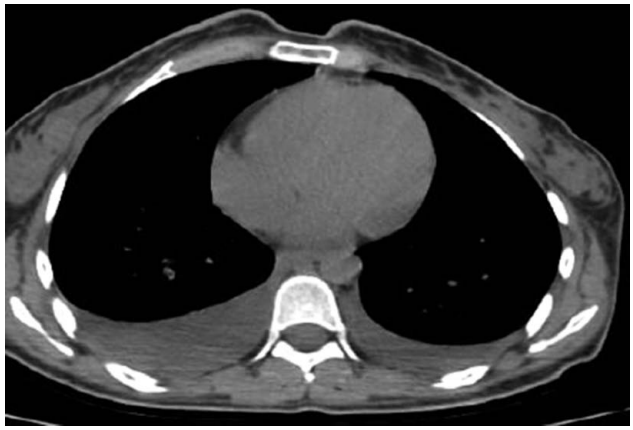

Figure 3. Upper endoscopy showed diffuse gastric mucosal edema and erythema, along with duodenal mucosal edema suggesting sever duodenitis and gastritis.

consistent with suspected SLE, diagnostic investigations were done to diagnose SLE. The patient's antinuclear antibody (ANA) tested positive with a titer of (nucleolar pattern) more than 10 index, also her antidouble-stranded DNA (ds-DNA) yielded positive results with 5.1 titer. Furthermore, both complement-3 (C3) and complement-4 (C4) levels were low, measuring 26 mg/dl and 2.9 mg/dl, respectively. ENA profile: U1-snRNP+ve (4.9), anti-Jo1–ve, anti-SS-A +ve (5.4), anti-SS-B +ve (5.2), anti-Scl 70 –ve, anti-SM +ve (4), SNRNP/SM –ve, anti-CENB –ve, lupus antibody -ve, anticardiolipin positive (20), anti-CCP –ve direct coombs and indirect –ve. Based on the patient's clinical presentation and investigative results (Table 1) aligning with the criteria outlined by the European Alliance of Associations for Rheumatology (EULAR) and the American College of Rheumatology (ACR), achieving a score of 27, a diagnosis of SLE was confirmed.

She was started on pulse steroid therapy of 1 g daily of methylprednisolone, and hydroxychloroquine 200 mg. Regarding pancytopenia, a blood film revealed no blasts. Endoscopy revealed diffuse gastric mucosal and duodenal edema (Fig. 3), with negative H. pylori results. Treatment commenced with esomeprazole 80 mg, metoclopramide, and ondansetron as needed. Colonoscopy findings were unremarkable. Follow-up ultrasound demonstrated severe ascites, prompting paracentesis with drainage of 170 cc of ascitic fluid, revealing nonportal HTN parameters with normal culture and cytology. Persistent abdominal distension led to further ultrasound evaluation, confirming severe ascites and necessitating pigtail insertion under radiologic guidance (Fig. 2). Analysis of ascitic fluid showed neutrophils of 331/, prompting the addition of ceftazidime. Culture and cytology results came back and showed no growth and cell count, respectively. Over 3 days, 3800 cc of fluid was drained before pigtail removal. Subsequent complaints of diarrhea prompted stool analysis, revealing trophozoites of entamoeba histolytica, leading to the initiation of metronidazole therapy. Abdomen and pelvis CT scan with IV contrast follow-up showed a marked interval decrease in intrabdominal free fluid, likely due to drainage, with only residual minimal free fluid remaining in the pelvis. There is a less appreciated thickening of the small bowel loops compared to the previous examination. Parailiac lymph nodes appear less prominent, with measurements of up to 0.4 cm in the short axis, decreased from 0.9 cm previously. Also during the hospital course, the patient underwent high-resolution chest CT scan, which showed bilateral pleural effusion (Fig. 4). The patient clinically and laboratory were improved and stable her vital signs then discharge with regular follow up for rheumatologist.

Discussion

Before discussing protein-losing enteropathy, it is important to note that our patient was admitted to our hospital with severe abdominal pain, vomiting, and diarrhea. Upon admission to the hospital, the patient received routine lab tests, such as a complete blood count (CBC), which revealed microcytic anemia, leukopenia, and thrombocytopenia. Based on previous findings, we suspect an autoinflammatory process, so we requested ANA, complement, double-strand DNA, and extractable nuclear antigen profiles, which revealed positive ANA, hypocomplementemia, and high-titer double-strand DNA with a negative ENA profile. So the patient was diagnosed with SLE flare. The patient also complained of abdominal distention, mild lower limb edema, and hypoalbuminemia.

Systemic lupus erythematosus (SLE) can affect almost any organ; however, gastrointestinal system manifestations are uncommon compared to other autoimmune diseases like systemic sclerosis and inflammatory bowel disease. The development of gastrointestinal involvement in SLE is mainly divided into three categories, for example, mesenteric vasculitis, pseudo-obstruction, and protein loss enteropathy. The current case involves a 22-year-old female who was clinically diagnosed with protein-losing enteropathy as the first manifestation of SLE. Since Palestine is regarded as a low-income nation, PLE is an exclusionary diagnosis that ought to be considered when making a differential diagnosis for any unexplainable ascites. Technetium 99 albumin scintigraphy, MR lymphangiography, and 24 h fecal al antitrypsin clearance was necessary for the diagnosis of protein-losing enteropathy. At our facility, these investigations were not

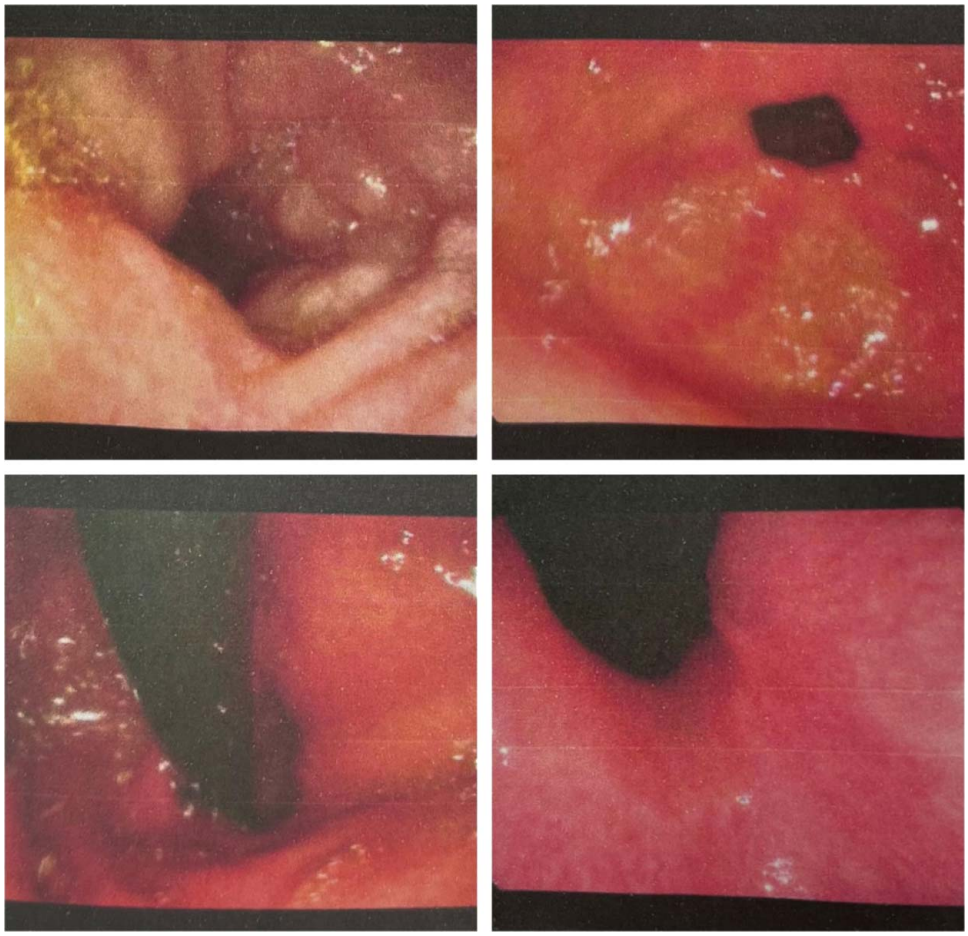

Figure 4. High resolution CT scan (axial view) was showed mild to moderate bilateral pleural effusions noted most evident at right side, associated with adjacent collapse consolidation, mainly at the right lower lobe along with few atelectatic bands. Both lungs fields appear normal without any evidence of masses or lung nodules with clear hila and mediastinum. There is no pericardial effusion and cardiomegaly.

accessible for a definitive diagnosis[6]. Nonetheless, given the positive response of serum albumin to steroid therapy, we could safely conclude that PLE caused hypoalbuminemia. A rare disorder called-pseudo-Meigs syndrome (PPMS), sometimes referred to as Tjalma syndrome, affects people with systemic lupus erythematosus (SLE) who have abnormal kidney function[6]. Typically, there is no suspicion of an ovarian tumor, and the patient comes with pleural effusion, ascites, and a high CA 125. In the differential diagnosis of SLE ascites, this illness ought to be included. However, in cases of Tjalma syndrome, there will not be any digestive issues like diarrhea, and the ascites will only be exudates with pleural effusion. Although half of SLE patients experience GI symptoms, lupus-associated protein-losing enteropathy is a rare manifestation with limited literature (case reports and series)[6].

Lupus-associated protein-losing enteropathy is diagnosed after ruling out other causes of hypoalbuminemia, such as lupus nephritis with proteinuria, liver dysfunction, malabsorption, or malnutrition[7]. Lupus enteritis (LE) was also considered a diagnosis; our patient experienced abdominal pain and nausea/vomiting (common in 97 and 45% of lupus enteritis patients, respectively), which were consistent with this disease process[8]. He also has diarrhea. This goes along with LE. Furthermore, although her CT scan demonstrated moderate to severe intra-abdominal free fluid in the setting of hypoalbuminemia and diffuse circumferential wall thickening with a white pattern of enhancement involving the small bowel suggested enteritis, there was a right adnexal cyst, mostly benign simple ovarian cyst, moderate bilateral hydronephrosis without any obvious source of obstruction and mild to moderate bilateral pleural effusion mostly secondary to SLE flare. Upper and lower endoscopies were done and showed diffuse gastric and duodenal edema with negative H. pylori, making sure there is severe enteritis concomitant with SLE flare and grossly normal through colonoscopy. It is worth mentioning that her CT demonstrates findings consistent with lupus enteritis, showing prominent parailiac lymph nodes, increasing mesenteric fat attenuation, and congested pelvic vessels.

We tried during the writing of our study to collect all cases that were like ours, meaning that protein-losing enteropathy was the first presentation of SLE, where we collected all cases written through the previous 22 years till today. Eight cases were gathered, and our case was one of them (Table 2). We have attached a table containing the case and other data that helped us highlight the importance of publishing cases like ours in terms of primary symptoms, physical examinations,

Table 2

Similar cases from the literature.

Authors	Year	Age	Sex	Country	Associated illness	1st presentation	Ascitic fluid albumin	SAAG	Serum albumin	Lipid profile	CBC	Autoimmune panel	Complement	Proteinuria	A1 antitrypsin	Steroid response	Treatment
Our case	2024	23	Female	Palestine	Hydronephrosis& bilateral pleural effusion	Esophagitis, gastritis, ascites	2.1	0.8	2.9	HDL 24 LDL 57 Triglycerides 169 Cholesterol 105 Cholesterol post pulse steroid 210	Panctopenia	Direct coombs +ve Antiphospholipid antibodies +ve ANA +ve anti-dsDNA +ve U1-snRNP +ve Smith antibody +ve anti-RO +ve anti- LA +ve	Low C3.C4	Absent	*	Rapid improvement	Hydroxychloroquine 200 mg/ daily
Elham Abdalla[1]	2023	33	Male	Sudan	Gastritis IBS Depression	Ascites	2.4 g/dl	0.9	1.5 g/dl	LDH 249 Total cholesterol 306 mg \dl	Leukopenia Thrombocytopenia	ANA +VE anti-RNP/Sm +VE anti-Sm +VE	Normal	Absent	*	Rapid improvement	Hydroxychloroquine 400 mg/day Azathioprine 100 mg/ day
Ramanathan Ramesh[4]	2023	38	Female	South Asia	Hypothyroidism	Progressive generalized body swelling	*	†	30g/dl	Normal	Leukopenia	ANA +VE anti-ds-DNA -VE anti-cariolipin -VE Lupus anticoagulanti-VE anti-beta2 glycoprotein –VE anti-TTG –VE HIV 1 and 2 –VE CMV –VE anti-TPO –VE	Low C3.C4	Absent	*	Rapid improvement in 2-weeks	Hydroxychloroquine 200 mg/ daily
Abdulrahman M. Aljebreen[9]	2013	41	Female	Saudi Arabia	Hypothyroidism Raynaud's phenomenon	Ascites	†	Nine and 10 In two-occasions	†	†	Normal	ANA – VE ASMA –VE anti-dsDNA —VE anti-AMA –VE	†	Normal U\A	*	Improved on 60 mg prednisone orally	100 mg Azathioprine
Murad Bektas[7]	2022	29	Female	Turkey	Chronic diarrhea	Ileus	‡	‡	‡	‡	‡	ANA +VE Anti-ds-DNA Anti-Sm\ribonuceoprotein +VE	Low C3,C4	‡	‡	‡	‡
Chang-keun lee[10]	2002	47	Female	Korea	–	Ascites Weakness Urinary frequency edema	†	0.8	1.9	LDH 512 Cholesterol 180 Triglyceride 102	Leukopenia Anemia	ANA +VE Anti-Ro (SSA) +VE anti-La (SSB),anti-RNP antibody, anti-Sm antibody, and anti-Scl-70 antibody were negative. Antiphospholipid antibodies including the lupus anticoagu- lant (LAC), anticardiolipin antibodies (aCL), and ß2-glycoprotein I (ß2- GPI) antibodies were negative	Low C3,C4	U\A not done	31.4 ml/24	Failed	Responded to 3-cycles of cyclophosphamide
		68	Male	Korea	–	Generalized body swelling	†	0.5	2	LDH 890 Cholesterol 136 Triglyceride 124	Anemia thrombocytosis	ANA +VE anti-Ro (SSA) +VE Anti-La (SSB) –VE anti-RNP –VE Anti-Sm –VE Anti-Scl-70 –VE Antiphsophplipid abs –VE HIV abs –VE	Low C3,C4	24 h urine protein 839	84.9 ml/24	Improved but succumbed to infectious complication And died	†

Marco fernandes Sri[11]	2023	39	Female	America	–	Endometritis, hemophagocytic lymphophagocytosis	†	†	2.3	LDH 879 U/l Triglyceride 446 mg/dl	Bicytopenia	ANA +VE anti-dsDNA +VE anti-SSA +VE anti-SSB +VE anti-PL7 +VE anti-RNP +VE anti-U1-SnRNP +VE anti-Pm-Scl75 +VE	Low C3, C4	U/A +++ Protein 1404 mg/dl	*	Rapid improvement	Fourth cycle cyclophosphamide, Anakinra

*Not done due to not available.
†Not available data.
‡Limited data access
- There is no associated illness.

appropriate laboratory tests, and treatment for such cases. One systematic review found that 82% of the reported cases of Lupus-associated protein-losing enteropathy occur in females, with symptoms of peripheral edema in 94%, ascites in 58%, pleural effusions in 54%, and diarrhea in 40%; our patient demonstrated these typical clinical characteristics. But according to our collected data, Ascites in 50%, general body swelling in 37.5%, mild to moderate pleural effusion in 62.5%, and diarrhea in 40%. Also, the incidence in females is higher than in males 3:1, that is, by a percentage of 75%. It is worth mentioning that our case is considered the youngest patient compared to other collected cases, which means that although gastrointestinal symptoms are rare as the first presentation of SLE. They can come at a young age, as in our case, which distinguishes our case from others the first and main chief complaint was abdominal pain with dysphagia, mostly due to the inflammatory process of active SLE (lupus enteritis related to SLE flare). In other cases where main chief complaints were abdominal distension or progressive body swelling. According to available collected data, four cases have SAAG less than 1.1 according to Ascitic fluid analysis, which is mean exudative. Our case had a SAAG of 0.8 despite hypoalbuminemia being 2.9 and serum protein being more than 2.5. According to these values, we excluded Ascites secondary to portal HTN causes, nephrotic syndrome, and severe malnutrition. The results go with the inflammatory process related to SLE flare, protein-losing enteropathy (lupus nephritis), but the main problem facing us remains limited to specific investigation tests, as we mentioned above. Most of the collected cases diagnosed with the disease by excluded due to the lack of specific investigation tests and high costs, such as albumin scintigraphy or fecal alpha-1-antitrypsin levels, with sensitivities of 97% and 94%, respectively. So, in our case, we were diagnosed by exclusion. As for why we doubted SLE as a diagnosis for collected cases, we found pancytopenia or bicytopenia in their CBC and hypocomplementemia c3 and c4. As for the response to treatment, there is a 62.5% rapid improvement in pulse steroids, certainly with a cure for underlying causes by either DMARDS or immunosuppressant drugs. Our patient started on a pulse steroid with elevated serum cholesterol. Initial high serum total cholesterol could be a predictive factor for a favorable steroid response[12]. And azathioprine with a good response to treatment, which means clinical and laboratory improvement. For lupus enteritis, the patient is on a proton pump inhibitor infusion and high-diet protein. Our patient remains under observation with stable vital signs and then discharged on hydroxychloroquine as maintenance therapy for SLE with frequent follow-ups at the rheumatology clinic.

Conclusion

This case describes a rare report of SLE with gastrointestinal involvement, specifically PLE, accompanied by other complications. SLE-associated PLE is difficult to diagnose, and limited information exists due to its rarity. This case presented a diagnostic challenge and required supportive treatment until the full effect of immunosuppressive drugs was achieved. Early diagnosis and high clinical suspicion were crucial for successful treatment.

Methods

The work has been reported in line with the Surgical CAse Report (SCARE) 2023 criteria[12].

Ethical approval

The study is exempt from ethical approval in our institution.

Consent

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

Source of funding

The study did not receive any funding.

Author contribution

A.A.J., T.A., and R.A.: writing the manuscript and designing the figures; A.A., R.A.-A., A.A., R.A., A.A.J., and T.A.: reviewing and editing the manuscript.

Conflicts of interest disclosure

The authors declare no conflicts of interest.

Research registration unique identifying number (UIN)

Not applicable.

Guarantor

Abdelwadod Abuturki.

Data availability statement

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Provenance and peer review

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