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*Case Report*

# Childhood Lupus-Associated Protein-Losing Enteropathy (LUPLE): A Case Report and Review of the Literature

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**Abstract**

Protein-losing enteropathy (PLE) is a rare condition characterized by clinical findings such as edema, ascites, pleural effusion, and diarrhea due to excessive protein loss from the gastrointestinal system. Although systemic lupus erythematosus (SLE) is rare in childhood, PLE can be the first presenting feature; this condition is referred to as lupus-associated protein-losing enteropathy (LUPLE). Protein-losing enteropathy (PLE) is an uncommon condition resulting from excessive protein loss in the gastrointestinal system. Our case shows that PLE can be the initial presentation of SLE, which is a rare manifestation in childhood. PLE, a rare complication of lupus, tends to be more severe in children, and the diagnostic process can be challenging. This case report presents a 7-year-old girl who presented with abdominal distension, generalized edema, chronic diarrhea, and weakness. Despite treatment, the recurrence of symptoms and the addition of new joint findings led to further investigations, which revealed positive anti-dsDNA and low complement levels, resulting in a diagnosis of systemic lupus erythematosus. The patient's clinical condition improved with steroid, azathioprine, and hydroxychloroquine treatments. This case highlights the importance of considering SLE in the differential diagnosis of PLE and underscores the significance of recognizing the rare presentations of childhood lupus.

**Keywords**

LUPLE, SLE, pediatric, diarrhea, hypoalbuminemia, protein-losing enteropathy

## Introduction

Protein-losing enteropathy (PLE) is a rare disorder resulting in excessive protein loss from the gastrointestinal tract, presenting with clinical features such as edema, ascites, diarrhea, and pleural effusion.[1] Pediatric-onset systemic lupus erythematosus (SLE) is a multisystemic autoimmune disease characterized by various clinical findings and immunological features. Although SLE is a rare condition in childhood, PLE can be its first presenting feature. PLE can occur in conditions with increased intestinal lymphatic pressure, erosions, or ulcerations of the intestinal mucosa or when the mucosal surface remains intact, but damage occurs in tight junctions.[2] The diagnosis is made based on the presence of hypoalbuminemia and an increase in fecal alpha-1 antitrypsin.[1] Herein, we report a 7-year-old female patient who presented with PLE and was finally diagnosed with SLE-associated protein-losing enteropathy after detailed investigations and successfully treated with systemic corticosteroids, azathioprine and hydroxychloroquine. This case highlights the importance of considering SLE in the differential diagnosis of PLE.

## Case Report

A 7-year-old girl presented with abdominal distension, diarrhea, weakness, and difficulty in walking. She had visited another medical center 7 months prior due to complaints of profuse watery diarrhea leading to weight loss, oral ulcers, and abdominal distension. Her medical history revealed that she was born full-term following an uncomplicated pregnancy. At birth, her weight was 3500 grams (+0.5 SD). At the age of 4, she was diagnosed with Hashimoto's thyroiditis during routine examinations, which revealed hypothyroidism and positive thyroid autoantibodies. There was neither a

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family history of rheumatic diseases nor consanguinity between the parents. In laboratory work-up, celiac serology was negative (tissue transglutaminase IgA and IgG), and duodenal/bulb biopsies showed no increase in intraepithelial lymphocytes. Her immunoglobulin (Ig) A level was 208 mg/dl (normal range: 36–268). Stool analysis revealed a pH of 6 with negative findings for fecal fat and reducing substances. Due to elevated fecal calprotectin (**FC** > 1000 µg/l), a colonoscopy was performed, which showed normal mucosa throughout the colon and terminal ileum. However, histopathological examination revealed cryptitis, with crypt abscesses observed in the ascending, descending, and sigmoid colon, leading to a diagnosis of inflammatory bowel disease. When the patient was started on 60 mg of corticosteroid therapy, she responded dramatically. A plan to gradually taper off the corticosteroids was implemented, but 2 days after stopping the treatment, the patient's symptoms recurred. Therefore, corticosteroid treatment had been restarted. The patient came to our center, still receiving corticosteroid treatment. On physical examination at the time of admission to our hospital, the patient weighed 28 kg (89th percentile), was 120 cm tall (44th percentile), and had a body mass index of 19 (+1.5 SD). Hypertrichosis, a cushingoid appearance, abdominal distension, and edema around the back of the hands and periorbital area were observed. Other system examinations were unremarkable. Laboratory tests revealed a hemoglobin level of 12 g/dL (reference range: 10.8–13.1 g/dL); total white blood cell count of $8.7 \times 10^3$/µL (reference range: 4.2–11.4 $\times 10^3$/µL); platelet count of 279,000/µL (reference range: 150,000–350,000/µL); aspartate aminotransferase of 22 IU/L (reference range: 27–49 IU/L); alanine aminotransferase of 12 IU/L (reference range: 15–35 IU/L); total bilirubin of 0.2 mg/dL (reference range: 0.3–1.2 mg/dL); total serum protein of 4.1 g/dL (reference range: 6.5–7.7 g/dL); serum albumin of 1.6 g/dL (reference range: 3.4–4.8 g/dL); serum creatinine of 0.54 mg/dL (reference range: 0.3–0.6 mg/dL); sodium of 140 mmol/L (reference range: 139–146 mmol/L); potassium of 4 mmol/L (reference range: 3.8–5.4 mmol/L); and calcium of 6.7 mg/dL (reference range: 9.1–10.2 mg/dL). After treatment for hypoalbuminemia, the adjusted calcium level was 8.6 mg/dL. C-reactive protein level was elevated at 22 mg/dL (normal <5 mg/dL), and the erythrocyte sedimentation rate was 21 mm/h (reference range: 0–15 mm/hour). Urine analysis results were normal. Viruses, bacteria, or parasites were not detected in the stool samples. Protein electrophoresis revealed normal levels of IgG (1110 mg/dL; reference range: 837–1243 mg/dL), IgA (208 mg/dL; reference range: 36–268 mg/dL), and IgM (78.5 mg/dL (reference range: 30–220 mg/dL). The lipid profile was within normal limits. Thyroid-stimulating hormone was measured at 4.79 µIU/mL (reference range: 0.50–5.0 µIU/mL), and thyroxine at 1.73 ng/dL (reference range: 0.93–1.71 ng/dL). Serum anti-thyroglobulin levels were elevated to 185 IU/mL (reference range, <20 IU/mL). Serological tests for cytomegalovirus, hepatitis B, hepatitis C, and other viruses were negative. Endoscopy showed pangastritis with no evidence of *Helicobacter pylori* on special stains, while colonoscopy revealed normal mucosa in both the terminal ileum and colon. Histopathological examination revealed blunted and atrophic villi in the terminal ileum, while the colonic mucosa was consistent with chronic active colitis. Duodenal biopsy showed villous atrophy with intraepithelial lymphocytes and neutrophilic infiltration, with no evidence of giardiasis or Whipple's disease (Figure 1). Anti-enterocyte antibodies were negative. Fecal alpha-1 antitrypsin levels, measured while the patient was on steroid therapy, were within normal limits. Ultrasonography revealed hepatosteatosis with normal vascular structure. Echocardiography and chest radiography were normal. In contrast, abdominal computed tomography revealed slight dilation of the colon and small bowel loops.

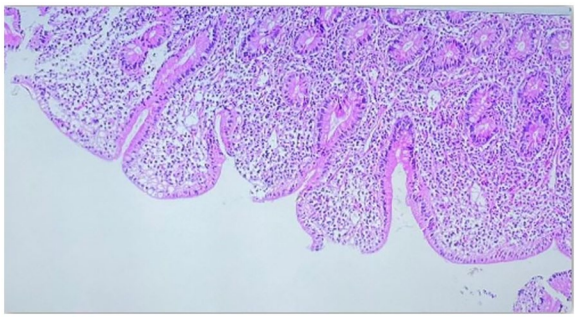


**Figure 1.** Duodenum, HE (×400): Villous atrophy with intraepithelial lymphocytosis and neutrophilic infiltration. No evidence of giardiasis or Whipple's disease.

During the patient's follow-up, albumin replacement and a protein-rich diet resolved ascites and edema, but ankle swelling and difficulty in walking persisted. Serological testing revealed a normal rheumatoid factor level of 9.1 IU/mL (normal <15 IU/mL) and a positive antinuclear antibody (ANA) at a titer of 1:160 with a speckled pattern. The ANA panel revealed +1 positivity for anti-nucleosome antibodies and +2 positivity for anti-proliferating cell nuclear antigens. Anti-dsDNA was elevated at 298 IU/mL (reference range: 0–200 IU/mL), whereas anti-Smith and anti-neutrophil cytoplasmic antibodies were negative. Complement levels (C3 and C4) were decreased at 27.2 mg/dL (reference range: 74–152 mg/dL) and <0.074 mg/dL (reference range: 0.1–0.4 mg/dL), respectively. The patient was diagnosed with SLE and was started on a high-protein and medium-chain triglyceride diet, systemic corticosteroid (0.5 mg/kg/day), azathioprine (2 mg/kg/day), and hydroxychloroquine (100 mg/day). Corticosteroid therapy was tapered to a low-maintenance dose, with the patient remaining asymptomatic for 1 year.

## Discussion

Protein-losing enteropathy (PLE) is an uncommon manifestation of rare diseases such as pediatric-onset SLE, and

lupus-associated protein-losing enteropathy (LUPLE) describes PLE secondary to SLE. SLE is an autoimmune systemic disease affecting various organs and systems. It is more common in girls, similar to its prevalence in adults.[3] Adult patients presenting with PLE as the first clinical manifestation of lupus have also been reported.[4] Upon reviewing pediatric lupus cases, we identified 8 cases in which PLE was the first symptom of SLE.[5-12] Literature reports that only 3 patients were diagnosed with LUPLE before age 10 years old.[8,13,14] Table 1 summarizes pediatric patients with LUPLE. This suggests that, in early childhood, PLE may present as the sole symptom for many years before the diagnosis of SLE is made.

In patients with a clinical presentation of LUPLE, PLE is often associated with chronic diarrhea. PLE is a rare condition in which excessive protein loss from the gastrointestinal tract results in symptoms such as edema, ascites, diarrhea, and pleural effusion. Its etiology covers a wide spectrum, including primary gastrointestinal diseases (e.g., Crohn's disease, celiac disease), causes of lymphatic obstruction (e.g., intestinal lymphangiectasia, heart failure), infections (e.g., Whipple's disease, tuberculosis), and immunological disorders (e.g., systemic lupus erythematosus, immunoglobulin deficiencies). Several mechanisms have been proposed to explain how lupus causes this intestinal protein loss. Including increased capillary permeability due to complement activation,[4,19] vasculitis-induced endothelial damage from immune complex deposition, cytokine-mediated intestinal permeability, and intestinal lymphangiectasia leading to lymphatic leakage.[4,6,19,20] Yazici et al[19] proposed complement activation as a mechanism for PLE in SLE, with decreased levels of C3 and C4 observed in a subset of patients. Similarly, our patient had low C3 and C4 levels, supporting the role of complement-mediated increased capillary permeability in the pathogenesis of lupus-associated PLE. However, the exact pathophysiological mechanism remains unclear. In patients presenting with PLE, an extensive and broad differential diagnosis often requires clinicians to perform advanced diagnostic investigations.[1] Therefore, making a diagnosis can be challenging and time-consuming. A patient's medical history and clinical findings are important in guiding the diagnosis. In this case, the presence of Hashimoto's thyroiditis in the patient, along with a family history of autoimmune diseases, specifically the patient's mother having Hashimoto's thyroiditis, initially suggested autoimmune enteropathy. However, this diagnosis was ruled out after the anti-enterocyte antibodies tested negative. During follow-up, the emergence of joint symptoms led to an evaluation for SLE based on the 2019 classification criteria established by the European League Against Rheumatism and the American College of Rheumatology. The patient fulfilled 2 clinical criteria (oral ulcers and joint involvement) and met 2 immunological criteria (low C3, low C4, and positive anti-dsDNA antibodies), achieving a total score of 18 points, which confirmed the diagnosis of SLE.[21]

Hypoalbuminemia is the most important characteristic laboratory finding of LUPLE.[22] Edema, ascites, and pleural effusion are the common clinical manifestations of hypoalbuminemia. Hypoalbuminemia can result from liver, kidney and intestinal diseases, poor protein intake due to malnutrition, chronic infections as a negative acute phase reactant or conditions such as analbuminemia.[23] The causes of hypoalbuminemia are shown in Table 2. For hypoalbuminemia to be attributed to PLE, it must be demonstrated that no other causes are present, or it must be shown that protein loss is directly of gastrointestinal origin.[1] The approach for hypoalbuminemia is shown in Figure 2. To confirm that hypoalbuminemia is due to PLE, tests such as fecal alpha-1-antitrypsin levels or Tc-99m human serum albumin scintigraphy are used.[1] We could not use this test because the Tc-99m HSAS was unavailable at our center. The patient presented to our center while on steroid therapy, and during this time, fecal alpha-1-antitrypsin levels were normal. Therefore, at the beginning of the differential diagnostic process, other potential causes were ruled out, and the patient was diagnosed with PLE.

SLE cases that begin in childhood tend to have a more severe course than adults.[22,24,25] The difficulty in diagnosing pediatric-onset SLE is that clinical findings are often unclear and may overlap with other pediatric diseases. However, early recognition of lupus and initiation of treatment are critical because they can significantly improve the disease course.[25] In the literature, the demographic and clinical characteristics of pediatric lupus cases where PLE was either the presentation of initial or subsequent findings are presented in Table 1.[5-18]

Most pediatric cases diagnosed with LUPLE present with classic clinical symptoms of significant hypoproteinemia, such as edema and ascites.[6-12] Childhood SLE can present with typical findings as well as nonspecific and atypical symptoms.[25] Very few cases of gastrointestinal involvement in lupus have been reported in the literature. Any organ in the gastrointestinal system, such as the liver, pancreas, or intestine, can be directly affected. The most common symptom is abdominal pain with various underlying causes. In children, after mesenteric vasculitis and intestinal pseudo-obstruction, PLE is the third most common gastrointestinal manifestation of lupus.[22] In childhood, there is no clear data on LUPLE as an initial presenting symptom; only case reports are available for this age group.

Endoscopic findings in patients with LUPLE are variable and nonspecific.[2] Findings such as mucosal edema, erythema, duodenitis, microscopic colitis, and lymphangiectasia highlight various features of gastrointestinal involvement in SLE.[2,6,7,12,26] In our patient, the duodenum exhibited blunted and atrophic villi along with an increase in intraepithelial lymphocytes (Figure 1). Although the colonoscopic appearance was normal, histopathological examination revealed focal neutrophilic infiltration and cryptitis.

**Table 1.** Summary of the Reported Pediatric Cases of Protein-losing Enteropathy Associated With SLE From 1980 to 2024.

| Year ref. | Authors | Age / Sex | Diagnosis | First presentation | Clinical findings of PLE | Alb | Aabs at PLE onset | Endoscopy/colonoscopy findings | Histological findings | Accompanying conditions | Treatment of PLE |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 1980[12] | Tsukahara et al | 12y M | SLE | PLE[a] | Generalized edema and ascites, pleural effusion, weight gain | 1.3 | ANA, Anti-DNA | N.d | N.d | Facial butterfly rash | Steroids |
| 1982[6] | Chase et al | 12y F | SLE | PLE[a] | Periorbital edema | 1.2 | ANA, Anti-DNA | Open ileal biopsy | Intestinal lymphangiectasia | Lupus nephritis, Libman-Sacks endocarditis, hypothyroidism | Steroids |
| 1990[7] | Edworthy et al | 16y F | SLE | PLE[a] | Periorbital and leg edema | 2.0 | ANA, Anti-ds-DNA, Anti-microsomal, Anti-thyroglobulin | N.d | Intestinal lymphangiectasia | Lupus nephritis, hematological manifestations | Steroids |
| 1991[15] | Tsutsumi et al | 16y F | SLE and SjS | Collagen disease | Pretibial edema, weight loss, mild ascites, pleural effusion | 0.9 | ANA, Anti-DNA | N.d | Edema and mononuclear infiltration at small intestine, C3 deposits in capillary walls | Facial rash, pleuritis | Steroids |
| 1992[16] | Garcia-Consuegra et al | 15y F | SLE | Collagen disease | Malaise, dyspnea, and leg edema | 2.4 | ANA, Anti-histones, Anti-cardiolipin | N.d | Normal | Neurological and hematological symptoms, pleuropericarditis, arthritis, thrombophlebitis, shrinking lung | Steroids CTX |
| 1996[9] | Molina et al | 13y F | SLE | PLE[a] | Abdominal pain, diarrhea, edema | 0.7 | ANA, Anti-SSA/SSB | N.d | Minimal villus blunting at small intestine | Pericarditis, myocarditis | Steroids AZA |
| 2002[13] | Gattorno et al | 6y F | CTD | Collagen disease | Periorbital and pretibial edema, abdominal pain, asthenia | 1.27 | ANA | Mucosa fragile and mildly edematous<br>Diffuse purpuric lesions | Alteration of duodenal villous architecture; colonic mucosal edema and extravasation | Evans' syndrome, mesangial glomerulonephritis | CTX |
| 2004[17] | Park et al | 11y F | SLE | Concomitant | Periorbital edema, weight loss | 2.9 | ANA, Anti-dsDNA, Anti-cardiolipin Anti-SSA/SSB | N.d | Chronic duodenitis | Lupus nephritis, pericarditis, oral ulcers | Steroids, CTX |
| 2011[14] | Hedrich et al | 11moF | SLE | Concomitant | Generalized edema<br>Arthritis, fever | 1.3 | ANA, Anti-ss-DNA, anti-SMA, anti-cardiolipin anti-SSA/SSB | N.d | Acute duodenitis, villous atrophy | Lupus nephritis, arthritis, hematological symptoms, serositis | Steroid AZA |
| 2012[11] | Ranawaka et al | 13y F | SLE | PLE[a] | Generalized edema, ascites, and diarrhea | 1.8 | ANA | N.d | Normal | Hematological symptoms, malar rash, neuropsychiatric lupus | Steroid AZA |
| 2012[5] | Carneiro et al | 18y F | SLE | PLE[a] | Limb edema, abdominal pain, and diarrhea | 1.6 | ANA, Anti- Sm | Normal<br>Normal | Chronic duodenitis | Photosensitivity<br>Malar rash | Steroid AZA |
| 2015[8] | Fontana et al | 8y M | SLE | PLE[a] | Generalized edema | N.d | ANA, Anti-dsDNA | Gastric hypertrophic folds with mucosal nodularity, lymphoid nodular hyperplasia in the distal ileum | A gastric mucosal chronic inflammation with foveolar hyperplasia and microscopic lymphangiectasias | Neuro-psychiatric lupus | Diet Steroid AZA |
| 2016[18] | Sansinanea et al | 17y F | SLE | Concomitant | Generalized edema, ascites, chronic abdominal pain and diarrhea | 1.7 | ANA, anti-cardiolipin<br>Lupus anticoagulant | Edema at duodenum | Chronic duodenitis | Malar rash, polyarthritis, alopecia, oral ulcers | Steroid CTX AZA RTX |
| 2020[10] | Naddei et al | 10y M | SLE | PLE[a] | Generalized edema | N.d | ANA, Anti-ds-DNA, anti-SSA/B | Gastric hypertrophic folds with mucosal nodularity Lymphoid nodular hyperplasia at the distal ileum | H.P. gastritis, lymphangiectasia, chronic colitis | Hematological symptoms | Diet + Steroid ZA |
| 2024 | Our case | 7y F | SLE | PLE[a] | Edema, diarrhea | 2.2 | ANA, Anti ds-DNA | Pangastritis<br>Normal | Terminal ileum villi are blunted and atrophic in appearance (chronic ileitis)<br>Colon, focal neutrophilic infiltration and cryptitis | Arthritis<br>Hashimoto thyroiditis | Diet + Steroid AZA Hydroxy chloroquine |

Abbreviations: AAbs, autoantibodies; Alb, albumin; AZA, azathioprine; CTD, connective tissue disease; CTX, cyclophosphamide; Diet+, high-protein diet and medium-chain triglycerides; F, female; H.P, Helicobacter Pylori; N.d, not detected; M, male; mo, month; PLE, protein-losing enteropathy; Ref, reference; RTX, rituximab; SLE, systemic lupus erythematosus; SjS, Sjogren's syndrome; y, year.
[a]First presentation PLE.

**Table 2.** Differential Diagnoses of Hypoalbuminemia: An Overview.

| Pathomechanism | Differential diagnosis | Diagnostic approach |
|---|---|---|
| Reduced synthesis | Genetic abnormalities (analbuminemia)<br>Hepatic dysfunction (cirrhosis, acute lıver failure, acute, or chronic hepatitis<br>Malabsorption<br>Congestive heart failure<br>Nutritional deficiencies | Genetic analysis<br>Liver function tests,<br>Transglutaminase Ig A, fecal steatosis, etc.<br>Brain natriuretic peptide<br>Nutritional parameters<br>Low protein diet |
| Increased renal albumin loss | Nephrotic syndrome<br>Chronic kidney disease | Serum creatinine<br>proteinuria |
| Increased catabolism | Infection, sepsis, burn | Acute phase reactants |
| Alteration in distribution | Cancer<br>İnfections, sepsis, shock, ischemia/ reperfusion, hypothyroidism, | Tumor Markers, etc |
| Increased gut albumin loss | Protein-losing enteropathy | α-1 antitrypsin level |

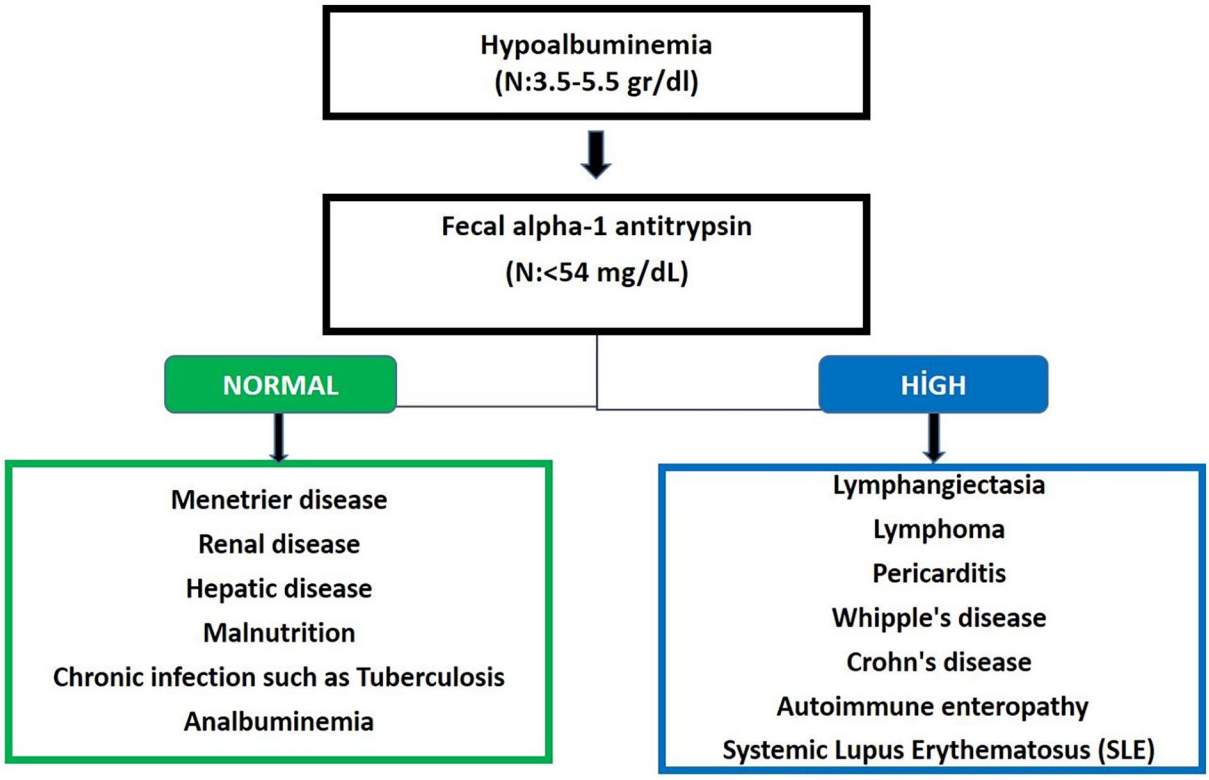



**Figure 2.** Differentiation of hypoalbuminemia according to fecal alpha-1 antitrypsin level.

In conclusion, our patient, initially monitored with a preliminary diagnosis of PLE, was later diagnosed with LUPLE after detailed investigations following the appearance of joint symptoms during follow-up. Chronic diarrhea can be a rare and early manifestation of SLE. Clinicians should not only focus on the classical findings of SLE but should also consider rare SLE-associated conditions such as LUPLE and keep LUPLE in mind when determining a differential diagnosis of PLE.

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## Informed Consent

Written informed consent was obtained from the patient's legal guardian for the publication of this case report, including any accompanying data and images.

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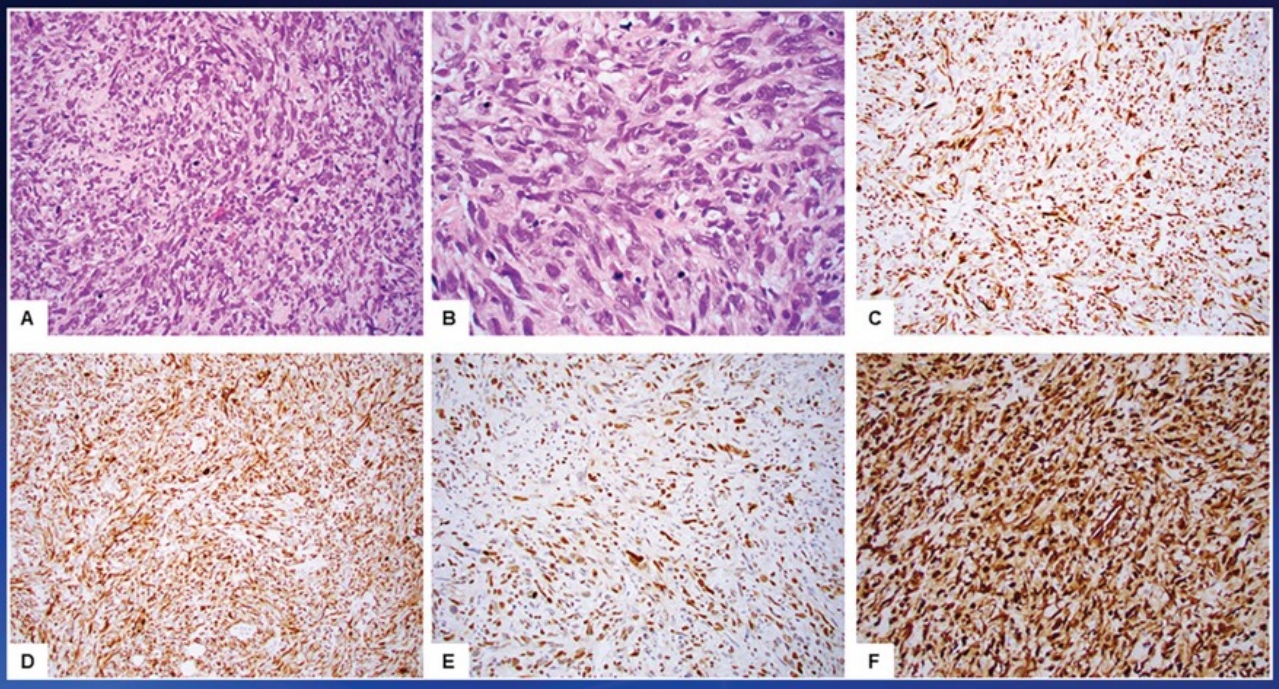
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