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Gastrointestinal Manifestations Leading to Diagnosis of Systemic Lupus Erythematosus During Flare: A Case of Lupus Enteritis

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Abstract

Lupus enteritis (LE) is an uncommon cause of abdominal pain in individuals with systemic lupus erythematosus (SLE). This condition is secondary to the inflammation of the intestinal wall caused by the activation of the complement system and accumulation of immune complexes and resultant ischemic injury and vasculitis. Herein, we present a 13 years old female presenting with pain abdomen for 15 days which was diffuse ,colicky and sharp, predominantly peri-umbilical and involving lower abdomen with severity 10 out of 10 with passage of loose stools with mucus , non-bloody in nature since 7 days .There was low grade fever on and off for last 1 month and anorexia. There was no known comorbid condition or history suggestive of chronic liver disease or prior bowel pathology. On asking further , there was history of symmetrical joint pain involving both hands and oral ulcer. Her abdomen was soft on palpation, with diffuse tenderness. The patient took treatment in a local hospital for these symptoms prior to visiting our hospital for which there was no significant response to empirical antibiotics initially. On general examination , patient was alert, conscious and oriented with toxic appearance during febrile episodes. Mild pallor and dehydration was present. Systemic examination reveals abdomen diffusely tender ,bowel sound was present with mild guarding without hepatosplenomegaly. Her initial laboratory investigation revealed anemia with hemoglobin of 9.9 gram /dL with mild elevation in liver enzymes with lipase of 543 U/L(<3times upper normal limit). Stool routine examination was unremarkable. A computed tomography (CT) study of whole abdomen showed diffuse edematous wall thickening of small and large bowel loops giving water target sign along with abdominal lymphadenopathy , mild ascites and mild pleural effusion. On further evaluation, anti nuclear antibody (ANA) was found to be positive (1:320) , nuclear speckled, with anti -Smith (Sm) antibody and U1RNP , SS-A positive. Her complement levels , C3 (less than 40 mg/dL) and C4 (less than 8 mg/dL) levels were low . The patient was managed with intravenous(IV) fluids ,IV corticosteroids and cyclophosphamide and followed up at one month . She didnot report any new symptoms or relapse of lupus enteritis. In the absence of previous suspicion of lupus, this diagnosis of LE was challenging. Also, the major clinical manifestations which include abdominal discomfort, vomiting, and diarrhea are often can be mistaken for several acute and chronic gastrointestinal disorders, posing a diagnostic problem.

Keywords: NIL

Introduction

Lupus enteritis (LE) is a rare manifestation of systemic lupus erythematosus (SLE). The pathophysiology of LE has not been fully elucidated, although inflammatory and thrombotic processes are likely

important factors [1] . It is characterised by intestinal wall inflammation secondary to the deposition of immune complexes and complement system activation, resulting in vasculitis and ischaemic injury [2] .The worldwide prevalence of LE varies from 0.2% to 9.7% among all patients with SLE, and from 29% to 65% among SLE patients with acute abdominal discomfort [3] . It is very rarely seen as an presenting sole manifestation of the disease flare and thus investigating LE is crucial to avoid disease complications and rule out other differentials .

Case Presentation

A 13 year old female presented to Emergency department complaining of pain abdomen for 15 days which was diffuse ,colicky and sharp, predominantly peri-umbilical and involving lower abdomen with severity 10 out of 10 with passage of loose stools with mucus , non-bloody in nature since 7 days .There was low grade fever on and off for last 1 month and anorexia. There was no known comorbid condition or history suggestive of chronic liver disease or prior bowel pathology. On asking further , there was history of symmetrical arthralgia involving both hands and oral ulcer. There was no history suggestive of photosensitive rash, alopecia, sicca symptoms, genital ulcer, Raynaud's phenomenon, red coloured urine , chest pain or breathing difficulty. She was not on any medications previously. There was no family history suggestive of any rheumatological disease or malignancies. The patient received treatment in a local hospital for these symptoms prior to visiting our hospital for which there was no significant response to empirical antibiotics , intravenous fluids , proton pump inhibitor or probiotics initially. On general examination , patient was alert, conscious and oriented with toxic appearance during febrile episodes. Mild pallor and dehydration was present. Systemic examination revealed diffusely tender abdomen with presence of bowel sound with mild guarding and absence of hepatosplenomegaly.

Table 1 shows her laboratory investigation results. It revealed mild anemia with hemoglobin of 9.9 gram/dL,mildly elevated liver enzymes and lipase less than 3 times upper normal limit. Anti nuclear antibody (ANA) was found to be positive (1:320) , nuclear speckled, with anti -Smith (Sm) antibody and U1RNP, SS-A and Ro positive. Her complement levels , C3 (less than 40) and C4 levels (less than 8) were low . Immunoglobulins were found to be in normal range. A computed tomography (CT) study of whole abdomen showed diffuse edematous wall thickening of small and large bowel loops giving water target sign along with abdominal lymphadenopathy , mild ascites.(figure 1)

TABLE 1: laboratory investigation of the patient showing anemia , mild elevation in liver enzymes and positive ANA and low complements

Test Name	Result	Unit	Reference Range
COMPLETE BLOOD COUNT			
WBC	4.57	10^3/µL	4.00–11.00
Haemoglobin (Hb)	9.9	g/dL	12–16
Neutrophils	77.2	%	40–70
Lymphocytes	21.7	%	20–40
Monocytes	1.1	%	2–10
Eosinophils	0	%	1–6
Basophils	0	%	0–2
Platelet Count	397	10^3/µL	150–400

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BIOCHEMICAL INVESTIGATIONS			
Blood Urea	21	mg/dL	14.98–36.38
Serum Creatinine	0.62	mg/dL	0.52–1.05
Sodium	149	mmol/L	137–145
Potassium	3.51	mmol/L	3.5–5.1
Amylase	160.5	U/L	30–110
Lipase	543	U/L	23–300
CRP	<5.0	mg/L	0–10
Procalcitonin	0.08	ng/mL	<0.1

URINE ANALYSIS			
Urine Colour	Pale Yellow	—	Pale Yellow/Amber
Urine Transparency	Clear	—	Clear
Specific Gravity	1.01	—	1.010–1.030
Urine pH	Acidic	—	4.6–8.0
Urine Protein	trace	—	Negative
Nitrite	Negative	—	Negative
Blood	Negative	—	Negative
Urobilinogen	Normal	—	Negative
Leucocyte Esterase	Negative	—	Negative
Urine Pus Cells	0–1/HPF	—	1–5/HPF
Urine RBC	Absent	—	Absent
Urine Epithelial Cells	0–1/HPF	—	Upto 20/HPF
Urine Crystals	Absent	—	Absent
Urine Casts	Absent	—	Absent

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STOOL ANALYSIS			
Stool Colour	Yellow	—	—
Stool Consistency	Semi solid	—	—
Stool Mucus	Present	—	—
Stool Blood	Absent	—	—
Stool Reaction	Alkaline	—	—
Stool Pus Cells	1–2/HPF	—	—
Vegetable Cells	Few	—	—
Bacteria	Few	—	—
Stool RBC	Not seen	—	—
Fungal Elements	Plenty/HPF	—	—
Epithelial Cells (Stool)	Occasional	—	—

AUTOIMMUNE PROFILE			
ANA HEP-2	Positive	—	Negative
ANA Pattern	Nuclear speckled (AC 4,5)	—	—
ANA Intensity	2+	—	—
ANA Primary Titre	1:100	—	—
ANA End Point Titre	1:320	—	—
Serum C3	<40	mg/dL	88–165
Serum C4	<8.0	mg/dL	14–44
ANA PROFILE			
U1-nRNP/Sm	Strong Positive	—	Negative
Sm	Positive	—	Negative
SS-A	Strong Positive	—	Negative
Ro-52	Strong Positive	—	Negative

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SS-B/La	Negative	—	Negative
Scl-70	Negative	—	Negative
PM-Scl	Negative	—	Negative
Jo-1	Negative	—	Negative
CENP-B	Negative	—	Negative
PCNA	Negative	—	Negative
ds-DNA	Negative	—	Negative
Nucleosomes	Negative	—	Negative
Histones	Negative	—	Negative
Rib-P Protein	Negative	—	Negative
AMA-M2	Negative	—	Negative
CECT Whole Abdomen	DIFFUSE EDEMATOUS WALL THICKENING OF SMALL AND LARGE BOWEL LOOPS GIVING WATER TARGET SIGN ALONG WITH ABDOMINAL LYMPHADENOPATHY, MILD ASCITES AND RIGHT-SIDED MILD PLEURAL EFFUSION		
HbsAg	non reactive		
Anti HCV	non reactive		
HIV I & II	non reactive		
stool C. difficile toxin	absent		

A computed tomography (CT) study of whole abdomen showed diffuse edematous wall thickening of small and large bowel loops giving water target sign along with abdominal lymphadenopathy , mild ascites.(figure 1)

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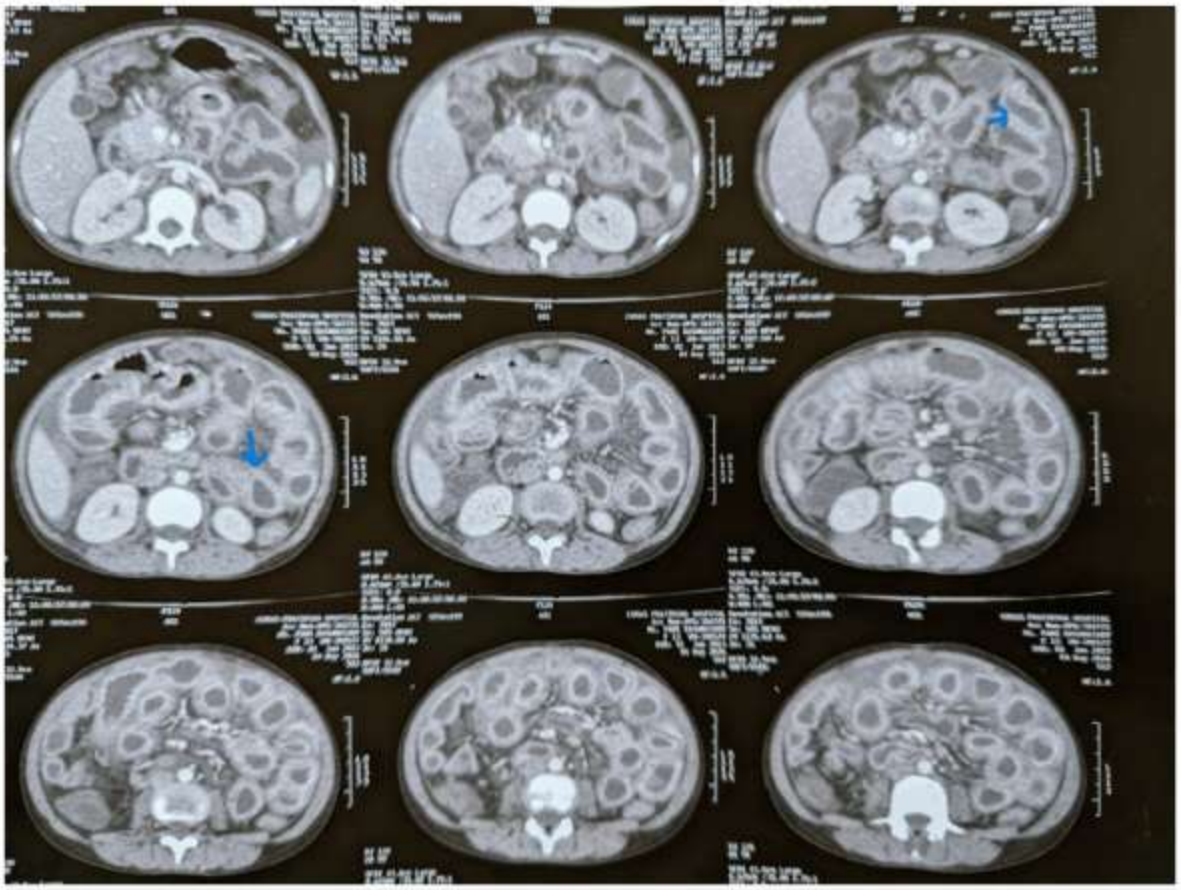

FIGURE 1: target sign as seen on CT abdomen

Therefore, after detailed evaluation of history , clinical examination, laboratory and radiological investigations , the patient was diagnosed to be a case of Lupus enteritis (LE). She was started on IV methylprednisolone 1mg/kg for 5 days followed by injection cyclophosphamide as per Euro Lupus protocol with significant improvement in her gastrointestinal symptoms. On discharge she was advised tab hydroxychloroquine after baseline eye examination and tab prednisolone 1mg per kg body weight and shewas followed up at one month , she didnot report any new symptoms or relapse of lupus enteritis.

Discussion

Gastrointestinal symptoms in SLE patients are not uncommon. They are usually attributable to medication side effects, infections, or other associated conditions (autoimmune hepatitis, primary biliary cirrhosis, inflammatory bowel disease, or celiac disease)[4]. Rarely, it may be due to SLE itself. Our patient was not a previously diagnosed case of SLE, which made the initial diagnosis of lupus enteritis challenging. Clinical presentation is typically nonspecific and most frequently includes abdominal pain (97%), ascites (78%), nausea (49%), vomiting (42%), and diarrhea (32%) [5]. LE is commonly associated with hematologic abnormalities, including leukopenia, lymphopenia, and anemia, as well as frequent immunological findings such as ANA positivity (92%), anti-dsDNA antibodies (74%), and hypocomplementemia (88%). Anti-RNP (28%), anti-SSA (26%), and anti-Sm (24%) antibodies are less commonly detected. Notably, C-reactive protein elevation is not a typical feature of this entity. [5]

Contrast-enhanced CT remains the gold-standard imaging diagnostic tool, often demonstrating the classic “target sign” (concentric bowel wall thickening where the hyperenhancing mucosa contrasts with the hypoenhancing and thickened submucosal layer)[6] .

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Other etiologies to be considered are Gastrointestinal infections, including opportunistic infections in immunosuppressed patients, other immune-mediated diseases, such as inflammatory bowel disease (IBD) and ANCA-associated vasculitis, and drug-induced toxicity, namely those associated with immunosuppressive agents. These causes need to be excluded by the combination of clinical, laboratorial, radiological, and histological features. Management of LE relies on controlling the underlying SLE flare.

High-dose corticosteroids are the mainstay of acute treatment, often leading to rapid improvement of bowel symptoms [7] and refractory cases or when concomitant organ involvement exists, additional immunosuppressive agents may be employed. The choice most often falls on cyclophosphamide, MMF, or rituximab, the latter being the most effective one in treating unresponsive patients. [8]

Long-term monitoring is important, as recurrence of GI manifestations may portend poor overall disease. [9]

Conclusions

The diagnosis of LE in our case was challenging because of the absence of a prior diagnosis of SLE. The major clinical manifestations of the patient included abdominal discomfort and diarrhea, which are quite non-specific and can be mistaken for several chronic gastrointestinal disorders, posing a diagnostic problem. A detailed clinical history unmasked several symptoms and signs related to SLE and laboratory and radiological evaluation associated with exclusion of possibilty of other differentials helped in diagnosis and further management of the patient . Studies on treatment with Belimumab is emerging. Our patient has been advised regular follow up care.

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