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Case Report

Clinical dilemma of acute abdomen in patients with systemic lupus erythematosus: A case report

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Abstract

Introduction: Gastrointestinal (GI) symptoms and signs may be seen in approximately one third of patients with rheumatologic disorders as primary presentation. Some of these findings may be nondiagnostic and may be clinical diagnostic challenge. GI tract involvement by systemic lupus erythematosus (SLE) must be differentiated from adverse drug reactions of treatment agents. Abdominal pain, associated with nausea and vomiting, is seen in up to 30 percent of patients with SLE. The cause of abdominal pain does not differ significantly from that in patients without lupus. Special attention should be given to conditions that may accompany lupus such as lupus peritonitis and infection**.** Lupus peritonitis is very unusual clinical finding and it is worth reporting.

Case Report: Our patient was a young female with definite rheumatologic disorders with acute abdomen as the dominant clinical finding. Imaging findings confirmed peritoneum and small intestine involvement. Paraclinical work-up including blood analysis confirmed SLE. She was managed with prednisone and non-steroidal anti-inflammatory agents, and discharged with partial improvement.

Conclusion: This case report shows that patients with symptoms consistent with acute abdomen in SLE remind us a clinical dilemma. There have been few reports of acute abdomen in patients with SLE in the literature. And more case reports are needed.

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Introduction

Gastrointestinal (GI) presentations may be seen in approximately one third of patients with rheumatologic disorders. Some of clinicopathologic findings may be mostly nondiagnostic and usually show intestinal tract involvement by rheumatologic inflammation, and adverse drug reactions of treatment agents. Bowel discomfort, associated by emesis and anorexia, is seen in up to one third of cases with SLE. The cause of abdominal pain does not differ significantly from that in patients without SLE. Special attention should be given to disorders that may accompany lupus such as lupus peritonitis, infection, inflammatory bowel disease, pancreatitis, and mesenteric vasculitis with intestinal infarction. In immunocompromised cases, infestation with opportunistic microorganisms like cytomegalovirus may cause abdominal pain and GI catastrophes like upper GI bleeding.[1,2]

A usually forgotten etiology of abdominal pain in SLE is lupus peritonitis. It is rarely reported, but autopsy studies have shown that 60 to 70 percent of patients with SLE have had an episode of peritoneal attack at some time of disease history. If peritoneal involvement is asscociated with frank rebound tenderness on physical examination and computed tomography (CT) scan documented intraperitoneal fluid, fluid tap is warranted to rule out infection. In literature review, sometimes abdominal pain of undiagnosed pathology responds to glucocorticoids, proposing an inflammatory

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origin. Thus, if a high gradient is found on peritoneal examination, a course of steroids (60 mg of prednisone per day) may be suggested if clinical finding is moderate to severe. Presence of peritoneal fluid is unusual in SLE. When present, visceral perforation with and without peritonitis should be excluded by paracentesis. Other causes of ascites like congestive heart failure and hypoalbuminemia related to lupus must be excluded. These causes may be due to the nephrotic syndrome or protein-losing enteropathy in some patients.[3,4]

Dysphagia is the most prevalent GI symptom in SLE and is usually due to organ hypomotility. The other presentations include esophageal stricture, gastroesophageal reflux, esophageal candidiasis, esophageal ulcers, and medication side-effects. Radiographic and manometric evaluation of the esophagus may disclose abnormal motility, while endoscopy and radiography of the esophagus can show other curable pathologies of dysphagia. The treatment of esophageal symptoms depends on predisposing cause.[5]

Mesenteric vasculitis is potentially life-threatening. The presentation may be insidious with lower abdominal pain. Radiographies and abdominal CT scans may show nonspecific but suggestive findings, but arteriography is usually required for diagnosis. Treatment includes antibiotics, high dose glucocorticoids, and intravenous cyclophosphamide.[6,7]

Pancreatitis occurs in less than 10 percent of patients, usually in those with active SLE elsewhere. The presentation does not differ from patients without SLE and includes upper abdominal pain, nausea and vomiting, and an increase in serum amylase. The differential diagnosis of abdominal pain and an elevated amylase should include mesenteric infarction, perforated peptic ulcer, ruptured ectopic pregnancy, some tumors, and renal failure. Imaging studies (e.g., CT scan or ultrasound) often help to confirm the diagnosis. Some patients require glucocorticoids in addition to usual medical treatment.[8,9]

The potential causes of either liver enzyme abnormalities or overt liver disease include SLE itself ("lupus hepatitis"), nonsteroidal anti-inflammatory drugs (NSAIDs), and coincidental disease. Liver chemistry abnormalities may resolve with cessation of NSAID or treatment of active SLE. Liver abnormalities are common; jaundice is rare and may reflect hemolysis rather than liver disease. Some liver diseases in SLE may be severe and progressive. The term “lupoid hepatitis” refers to autoimmune hepatitis rather than liver involvement in SLE.[10,11]

Protein-losing enteropathy in patients with SLE has been noted in a number of small series and case reports. It is estimated that half of patients have diarrhea. It may represent the first manifestation of SLE. Patients typically respond well to glucocorticoids or immunosuppressants.[12]

Methods

A young woman was visited at our center because of acute belly discomfort; she had been well until 4 weeks ago from her presentation. She developed generalized abdominal pain accompanied with nausea and vomiting, and complaining of abdominal discomfort. She was visited at the emergency department by a general surgeon. The musculoskeletal examination disclosed peripheral polyarthralgia, and based on the findings acute abdomen was taken into account. She was managed with nasogastric aspiration, rehydration, antibiotics, and nutritional support treatment. The differential diagnosis was discussed and she was finally diagnosed with acute appendicitis. She was transferred to operating room. After explorative laparotomy, emergent appendectomy was performed with diagnosis of perforated appendicitis. But her abdominal pain with nausea and vomiting was continued and she was transferred to our center.

The previous laboratory findings of patient are shown in the table 1-4.

Special consideration was directed to disorders that might have be associated with

Table 1. Laboratory findings of the patient

Teste	Results	Reference value
White blood cells (× 1000/mm³)	2.5 (Low)	4.0-10
Red blood cells (× 10^6/mm³)	3.40 (Low)	M: 4.5-6.3, F: 4.2-5.4
Hemoglobin (g/dl)	8.3 (Low)	M: 14-18, F: 12-16
Hematocrit (%)	26.7 (Low)	M: 39-52, F: 36-46
Mean corpuscular volume (fl)	79	77-97
Mean corpuscular hemoglobin (pg)	24 (Low)	26-32
Mean corpuscular hemoglobin concentration (%)	31 (Low)	32-36
Platelet (× 1000/mm³)	145	140-440
RDW (%)	15.3	11-16
PDW (fl)	10.6	10-17
MPV (fl)	8.9	8.5-12.5
P-LCR (%)	18.6	17-45
Segment (%)	75.5	
Lymphocyte (%)	17.9	
Mixed (%)	6.6	

RDW: Red blood cell distribution width; PDW: Platelet distribution width; MPV: Mean platelet volume; P-LCR: Platelet large cell ratio; M: Male; F: Female

lupus such as infection and lupus peritonitis. She was diagnosed to have SLE with skin and joint involvement and pancytopenia and was treated with steroids and chloroquine and was stable until 4 weeks ago when she presented to our center. With using the American Rheumatism Association (ARA) criteria for the diagnosis of SLE, it was suggested that patient had classical SLE. After 2 years regular follow up, unfortunately she did not continue medical visits. Until 4 weeks ago her present presentation appeared as discussed above.

It is clear that the patient's major presentations were due to SLE peritonitis. Laboratory findings including of ascites fluid analysis are shown in the table 4.

Table 2. Laboratory findings of the patient

Tests	Results	Reference value
Antiphospholipid (IgG) (U/ml)	1.3	< 12 Negative, 12-18 Borderline, >18 Positive
Antiphospholipid (IgM) (U/ml)	0.7	< 12 Negative 12-18 Borderline > 18 Positive
Antinuclear antibody (Index)	0.1	< 1 Negative > 1 Positive
Anti-DNA (Index)	2.5 (High)	< 0.8 Negative, 0.8-1.2 Equivocal > 1.2 Positive
C3 (mg/dl)	22 (Low)	90-180
C4 (mg/dl)	5.4 (Low)	10-40
CH50 (Units)	32 (Low)	101-300
Anti-cardiolipin antibodies (IgG) (U/ml)	0.1	< 12 Negative 12-18 Borderline > 18 Positive
Anti-cardiolipin antibodies (IgM) (U/ml)	0.1	< 12 Negative > 18 Positive 12-18 Borderline
Antiphospholipid (IgG) (U/ml)	6	< 12 Negative 12-18 Borderline > 18 Positive
Antiphospholipid (IgM) (U/ml)	0.3	< 12 Negative 12-18 Borderline > 18 Positive
White blood cells (× 1000/mm³)	10-12	Complete urinalysis
Red blood cells (× 1000/mm³)	16-18	
Epithelial	3-5	

Table 3. Laboratory findings of the patient

Tests	Results	Reference value
Urea (mg/dl)	16	M: 19-44, F: 15-40
Creatinine (mg/dl)	0.72	0.7-1.4
Aspartate aminotransferase (IU/l)	13	M: 0-31, F: 0-37
Alanine aminotransferase (IU/l)	11	M: 0-41, F: 0-31
Alkaline phosphatase (IU/l)	55 L	Adult: 64-306, Children: 180-1200
Amylase (IU/l)	122 (High)	Up to 100
Serum Sodium (mEq/l)	128 (Low)	136-145
Potassium (mEq/l)	3.8	3.6-5
Lipase (IU/l)	68 (High)	Up to 60
Urine volume/24hrs (ml/24 hours)	1900	800-2000
Urine creatinine/24hrs (g/24 hours)	0.5	0.5-1.5
Urine protein /24hrs (mg/24 hours)	62	40-150
Direct Coombs	Negative	Negative
Indirect Coombs	Negative	Negative

M: Male; F: Female

Plain chest X-ray showed bilateral basal pleural effusion. Imaging findings are described below (Figure 1).

Discussion

SLE can affect the entire GI tract.[1] GI presentations may be seen in approximately one third of cases. Some of clinicopathologic findings may be mostly nondiagnostic and usually show intestinal tract involvement by lupus and adverse drug reactions of treatment agents.

The most important problem is excluding surgical abdomen. The cause of abdominal pain does not differ significantly from that in patients without lupus.[4]

Table 4. Laboratory findings of the patient

	Many
Color	Yellow
Appearance	Semi turbid
pH	6
Protein	+3
Blood/hemoglobin	+2
Glucose	Negative
Ascorbic acid	Negative
Urobilinogen	Negative
Bilirubin	Negative
Nitrite	+2
Ketone	Negative
Glucose	132 pleural fluid analysis and cell-count
Protein	1.5
Albumin	1
Lactate dehydrogenase (Peritoneal)	264
White blood cell (mm^3)	70
Red blood cell (mm^3)	300
Peritoneal cell count	
White blood cells (mm^3)	6000
	Segment 90%
	Mononuclear 10%
Red blood cells (mm^3)	1200000
Hematocrit (%)	10.0
Glucose (mg/dl)	67
Protein (g/dl)	1.4
Lactate dehydrogenase (IU/l)	254

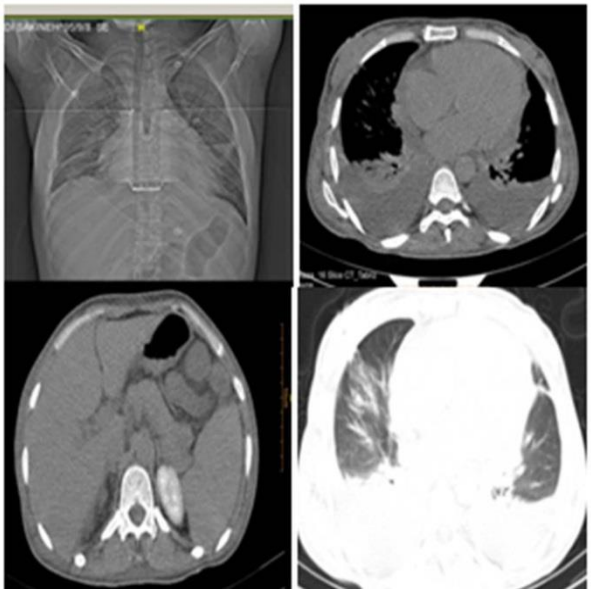

Figure 1. Plain chest X-ray showed bilateral basal pleural effusion with passive lung collapse, abdominal CT scan showed intestinal wall edema and circumferential thickening of small and large bowels wall, splenomegaly and ascites

Special attention should be given to disorders that may accompany lupus such as lupus peritonitis, infection, inflammatory bowel disease, dyspepsia, pancreatitis, and mesenteric vasculitis with intestinal infarction.In immunocompromised cases, infestation with opportunistic microorganisms like cytomegalovirus may cause abdominal pain and GI catastrophes like upper GI bleeding.[5]

An often overlooked cause of abdominal pain in SLE is lupus peritonitis. It is rarely suspected, but autopsy studies show that 60 to 70 percent of patients with SLE have had an episode of peritoneal attack at some time of disease history. If peritoneal involvement attack is with frank rebound tenderness on physical examination and CT scan documented intraperitoneal fluid, fluid tap is warranted to rule out infection. In literature review, sometimes abdominal pain of undiagnosed pathology responds to glucocorticoids, showing an inflammatory cause.[6] Thus, if a high gradient is found on peritoneal examination, a course of steroids (60 mg of prednisone per day) may be suggested if clinical finding is moderate to severe. Presence of peritoneal fluid is unusual in SLE. When present, visceral perforation with and without peritonitis should be excluded by paracentesis. Other causes of ascites like congestive heart failure, hypoalbuminemia related to lupus must be excluded. These causes may be due to the nephrotic syndrome or protein-losing enteropathy in these patients. Dyspepsia has been noted in 11 to 50 percent of patients with SLE, while peptic ulcers (usually gastric) are present in 4 to 21 percent. These complications are more common in patients treated with NSAIDs, but SLE itself may also predispose to ulcer formation. Glucocorticoids also increase the incidence of dyspepsia.[8,9] Mesenteric vasculitis is potentially life-threatening.[13] Radiographs and abdominal CT scans may show nonspecific but suggestive findings, but arteriography is usually required for diagnosis. Treatment includes antibiotics, high dose glucocorticoids, and intravenous cyclophosphamide.[10,11]

Pancreatitis occurs in less than 10 percent of patients, usually in those with active SLE elsewhere. The presentation does not differ from patients without SLE and includes upper abdominal pain, nausea and vomiting, and an increase in serum amylase.[12,14] The differential diagnosis of abdominal pain and an elevated amylase should include mesenteric infarction, perforated peptic ulcer, ruptured ectopic pregnancy, some tumors, and renal failure. Imaging studies (e.g. CT scan or ultrasound) often help to confirm the diagnosis. Some patients require glucocorticoids in addition to usual medical treatment.[13,15,16]

Conclusion

GI symptoms and signs may be seen in approximately one third of patients with rheumatologic disorders as primary presentation. Some of these findings may be nondiagnostic and may be clinical diagnostic challenge. GI tract involvement by SLE must be differentiated from adverse drug reactions of treatment agents. Abdominal pain, associated with nausea and vomiting, is seen in up to 30 percent of

patients with SLE. Special attention should be given to disorders that may accompany lupus such as lupus peritonitis and infection. If the physician has a high clinical suspicion of this diagnosis, prompt treatment with corticosteroids is very important. This suspicion may prevent the catastrophic result in patients with GI involvement of SLE and improve prognosis in this high-risk patient population.

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Authors' Contribution
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Conflict of Interest
None.

Ethical Approval
The author has made his best effort to not reveal any information showing the identification of the patient and keep it confidential.

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