

# Acute pancreatitis- As a Presenting Manifestation of Systemic Lupus Erythematosus

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**Abstract** 15 years aged young girl was admitted with acute pain abdomen with distension, vomiting and fever. Patient was very sick and investigations, both blood and imaging of abdomen were suggestive of pancreatitis. This acute condition was soon followed by acute nephritis within a day or two and was established due to systemic lupus erythematosus through various investigations. Then we started treatment with high dose steroid and patient showed dramatic improvement. As a rare complication of SLE, acute pancreatitis presents as generalized flare-ups in most patients previously diagnosed with SLE. But acute pancreatitis as an initial feature of a multisystem disorder of SLE is rare and physician should remain aware of this clinical entity.

**Keywords:** systemic lupus erythematosus (SLE), acute pancreatitis, lupus nephritis

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## 1. Introduction

Here we report an interesting case of acute pain abdomen due to pancreatitis, the cause of which was detected later to be due to systemic lupus erythematosus.

## 2. Case Report

A fifteen year old female patient was admitted with acute pain abdomen and distension for last five days associated with continuous, low grade fever, nausea, vomiting for last seven days. On general examination, patient was febrile (the temperature varied from 99 to 102°F), tachycardic, hypotensive (BP- 100/60mm Hg). There were mild pedal pitting edema and moderate pallor without any skin or mucosal involvement or lymphadenopathy.

Gastrointestinal examination revealed a distended abdomen with diffuse tenderness, sluggish peristalsis without any shifting dullness and empty rectum. She was kept on nothing per mouth, intravenous fluids and empirical antibiotics (metronidazole and fluroquinolones). Provisional diagnosis was acute abdomen and straight x-ray in erect posture did not show any free gas under diaphragm, but gut loops were distended with a ground glass appearance. On next day patient developed puffiness of face with acute respiratory distress and blood pressure went upto 160/90 mmHg. She also developed bilateral pleural effusion and tender ascites.

Investigations showed neutrophilic leucocytosis (total leucocyte count-24000/cmm; neutrophils<sub>76</sub>, lymphocytes<sub>20</sub>, monocytes<sub>2</sub>, eosinophils<sub>2</sub>), elevated urea(160mg/dl), creatinine(2.2mg/dl), potassium(5.8mmol/L), aspartate amino transferase(300U/L), alanine amino transferase(150 U/L), lactate dehydrogenase(LDH-650U/L), amylase (2142U/L) and lipase (1203U/L). At this turn of events the provisional diagnosis was acute pancreatitis complicated by prerenal azotemia and septicemia. Within two days the patient developed huge, tense ascites; pleural effusion with severe dyspnoea and marked swelling of face and both legs. Routine lab investigations revealed progressive rising values of serum urea, creatinine, and decline in hemoglobin(from 12.6gm/dl to 7.5gm/dl), hematocrit(20% reduction), total protein(2.2gm/dl) and serum albumin(0.8gm/dl). Ultrasonography showed a bulky, hypoechoic pancreas, bilateral kidney enlargement with raised cortical echogenicity, moderate ascites and bilateral pleural effusion which biochemically showed exudative (LDH-577U/L, total protein-2gm/dl). Cultures of blood, pleural and peritoneal fluid showed no growth. A serial routine urine analysis revealed plenty of RBC and elevated 24 hours urinary protein (4gm/dl). With a view to a young female continued to have occasional fever, persistent hematuria and proteinuria; and serositis we went for serological tests. The connective tissue workup showed a strongly positive antinuclear antibody (ANA) titer (1:320), anti-double-stranded DNA antibody positivity, anti-Smith antibody positivity and anti-histone antibody positivity while anti-SSA and anti-SSB were

negative. As patient's condition was deteriorating even after third day with elevation of biochemical markers, leucocytosis and fluid accumulation we opted for CT scan abdomen. Computerized tomographic scan of the abdomen showed diffuse pancreatic necrosis especially of the body and tail, bilateral enlarged kidney (Figure 1 & Figure 2). Serum IgG4 antibody level was negative. Renal biopsy was done with immunofluorescence study which

showed features of class 4 lupus nephritis. The patient was put on intravenous methylprednisolone for three days then oral prednisolone at a dose of 1 mg/kg body weight. Patient showed dramatic improvement in clinical as well as biochemical parameters within one week starting steroid. Patient was discharged in stable condition after three weeks of admission.



Figure 1. CT scan (horizontal section) showing necrosis of pancreas



Figure 2. CT scan (Vertical section) showing dilated gut loops and necrosis of pancreas

### 3. Discussion

Systemic lupus erythematosus patient may present with protean manifestation. When it presented with classical symptoms and signs it is not difficult to stamp the diagnosis of systemic lupus erythematosus. Presenting manifestation may sometimes make a clinician highly confused and if not diagnosed promptly we may lose the patient. In our case patient presented with features of acute pancreatitis with clinical suggestion of acute intestinal obstruction.

SLE is a multisystem disease, including the gastrointestinal system in about 50% of all SLE patients. As a rare complication of SLE, the frequency of pancreatitis in SLE patients is only about 0.2%–8.2% and presents as generalized flare-ups in most cases of patients previously diagnosed with SLE. [1] Only 100 cases of SLE pancreatitis had been documented in the literature and only 12 cases including ours had reported pancreatitis as an initial presentation of SLE [1].

Patients with acute pancreatitis had higher systemic lupus erythematosus disease activity index (SLEDAI) scores and higher mortality, compared to those SLE patients without pancreatitis. [2,3] Mortality associated with SLE related to acute pancreatitis is ranging from 27.5 to 45% according to different studies. [4,5] Delayed diagnosis and improper treatment is the main reason for unfavorable prognosis, even mortality [6]. Likewise, the mortality rate of the Hopkins Lupus Cohort (3%) was considerably lower than average of other reported studies due to close monitoring, early diagnosis, and treatment [7].

In SLE patients with acute pancreatitis, one should search for the common causes of pancreatitis (obstruction of the pancreatic duct, toxi-metabolic aetiologies and medications). After the exclusion of these aetiologies, the diagnosis of lupus pancreatitis can be established. In retrospect, although our patient fulfilled the American College of Rheumatology (ACR) classification criteria for lupus, the initial impression was an infectious process. Our patient had multiorgan involvement of SLE, namely, hematologic, renal, and gastrointestinal; however, the most distinguishing feature of our patient's condition was pancreatitis.

The pathogenic mechanism of SLE-related acute pancreatitis is very complex and multifactor. Vascular damage (including vasculitis, intimal thickening, immune complex deposition, occlusion of arteries, and arterioles), autoantibody production, abnormal cellular immune response, and drug toxicity may be responsible for the development of pancreatitis [8].

Since the initial description of SLE pancreatitis, there has been debate as to its origin, namely, steroid vs SLE as the primary cause. Most cases of pancreatitis in lupus have been in patients with long-standing SLE with multiorgan involvement. The arguments against steroid-induced pancreatitis in SLE include the rare occurrence of pancreatitis in non-SLE patients on steroids, the finding of vasculitic lesions in the pancreas on autopsy, and the resolution of acute pancreatitis on continued steroid

therapy. However in SLE patients, subclinical pancreatitis with elevation of pancreatic enzymes without clinical symptoms seems to be more frequent than clinical pancreatitis. Ranson found that hyperamylasaemia occurs in 30.5% of asymptomatic SLE patients, suggesting that subclinical pancreatic damage might occur frequently in SLE [9].

Our patient had never previously used corticosteroid. Conversely, increasingly accumulated evidence showed that steroids do not trigger acute pancreatitis or cause increased mortality on acute pancreatitis, but instead, they have a possible therapeutic effect on SLE-related pancreatitis. [3] In Hopkins cohort, appropriate treatment with corticosteroids added a survival benefit in SLE-related acute pancreatitis. [7] Our patient showed a dramatic improvement of pancreatitis after she was put on steroids with a rapid decline in pancreatic enzymes and disappearance of CT findings.

Currently our patient is receiving monthly cycles of intravenous cyclophosphamide therapy.

### 4. Conclusion

SLE can involve any organ system. It is important that the family physician, who treats patients as a whole, suspect SLE when a straightforward diagnosis is associated with inexplicable multiple concomitant abnormalities. Clinician must consider pancreatitis in a patient of SLE with pain abdomen and elevated pancreatic enzymes. This is first case we had experienced in our hospital and use of high dose steroid showed survival benefit.

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