



An Acute Pancreatitis as a Rare Presentation of Systemic Lupus Erythematosus

Taraneh Dormohammadi-Toosi¹, Abdorahman Rostamian², Ali Khalvat², Fatemeh Shahbazi³

1- Department of Rheumatology, Vali Asr Hospital, Imam Khomeini Hospital Complex AND Center for Research on Occupational Disease, Tehran University of Medical Sciences, Tehran, Iran

2- Department of Rheumatology, Vali Asr Hospital, Imam Khomeini Hospital Complex, Tehran University of Medical Sciences, Tehran, Iran

3- Department of Rheumatology, Vali Asr Hospital, Imam Khomeini Hospital Complex, Tehran University of Medical Sciences AND Department of Biology, Payame Noor University, Tehran, Iran

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Corresponding author:

Taraneh Dormohammadi-Toosi

Email:

dormohammadi@tums.ac.ir

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ABSTRACT

Systemic lupus erythematosus (SLE) is a chronic inflammatory disorder with unknown etiology which can involve various organs in the body. The most common symptoms of this disease are constitutional symptoms along with skin rashes, arthritis, nephrologic, and hematologic manifestations. Acute pancreatitis is an unusual presentation of SLE. Here, we report an interesting case SLE who suffered from fever, weakness, abdominal pain, and weight loss. Finally, lupus pancreatitis was diagnosed based on her symptoms, laboratory, and imaging findings.

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Introduction

Systemic lupus erythematosus (SLE) is a chronic inflammatory disorder with an unknown etiology which can affect various organs such as skin, joints, kidneys, lungs, nervous system, and almost any other organ in the body. SLE is more common in women, especially between ages

20 and 30 years. The most common symptoms of SLE are constitutional symptoms along with skin rashes, arthritis, nephrologic, and hematologic involvement (1, 2). Acute pancreatitis is an uncommon presentation of SLE which manifests as an acute abdominal pain and can occur without other underlying etiology such as gallstone,

drug or alcohol abuse (3). Here, we report an interesting case of SLE, which presented by acute pancreatitis.

Case Report

A 24-year-old woman was admitted to the emergency care ward in 2014 (Imam Khomeini Complex Hospital), with complains of fever, generalized body pain, weakness and weight loss (about 4 kg during 1 month). Her symptoms had begun from 1 month before her admission and it became more severe at presentation.

Her physical examinations were as follows: she was ill and pale. The vital signs: Blood pressure (BP: 100/60 mmHg), pulse rate (PR: 110/min), body temperature (38.5° C oral), and respiratory rate (RR: 20/min). In head and neck examinations multiple, mobile and soft cervical lymphadenopathies (< 2 cm) were detected. There were erythematous plaques over the metacarpophalangeal and proximal interphalangeal joints. The lung examination showed a decrease of breath sounds in the base of the lungs and dullness in percussion. The abdominal examination showed only mild epigastric tenderness in deep palpation. The heart, liver and spleen examinations were normal.

In the extremities, only mild pitting edema in lower limbs was detected. Proximal muscle forces of upper and lower extremities were 4/5. Distal muscle forces were normal without any neurologic problems. Joint examinations were normal.

In the patient's history, there were no histories of malar rash, photosensitivity, abortion, family history of the rheumatologic disorder or other diseases except for a childhood seizure with irregular lamotrigine treatment. The laboratory tests at presentation showed in table 1.

Because of the presence of fever, leukopenia, and a poor general condition, she was treated with broad spectrum antibiotics (vancomycin and imipenem) by impression of sepsis and also investigation for the underlying cause of the leukopenia such as malignancy, collagen vascular disorder, and infection was began.

Two days after her admission, she suffered from sudden onset epigastric pain which spread to the right upper quadrate and periumbilical area of her abdomen along with nausea and vomiting, causing her general condition become worse. We found severe epigastric tenderness and mild grading in her abdominal examination.

Table 1. The laboratory examination data for patient on arrival and on discharge time

Variables	Laboratory examination data on arrival	Laboratory examination data on discharge
Leukocyte count ($\times 10^3/\mu\text{l}$)	2200	10,400
Hemoglobin (g/dl)	8	10.8
Platelet count ($\times 10^3/\mu\text{l}$)	199	253
ESR (mm/h)	98	30
CRP (< 10 mg/ml)	26	10
BUN (mg/dl)	15	10
Cr (mg/dl)	0.7	0.5
ALT (< 35) U/l	110	17
AST (< 35) U/l	450	20
LDH (255-500) U/l	2750	320
CPK (24-195) U/l	550	50
Amylase (480) IU/ml	1090	60
Variables in urine		
WBC (count)	6-8	1-2
RBC (count)	1-2	0-1
Protein	1+	Negative

ESR: Erythrocyte sedimentation rate, CRP: C-reactive protein, BUN: Blood urea nitrogen, Cr: Creatinine, ALT: Alanine aminotransferase, AST: Aspartate aminotransferase, LDH: Lactate dehydrogenase, CPK: Creatine phosphokinase, WBC: White blood cell, RBC: red blood cell, ANA: Anti-nuclear antibody

Our work up showed high serum amylase 1090 IU/unit (480) also the finding of abdominal computed tomography (CT) scan was in favor of acute pancreatitis based on the report of generalized largening of pancreas and haziness and sever fat stranding around the tail of pancreas. There was not any pancreatic duct abnormality, defect or stone. The gallbladder was normal (Figure 1).

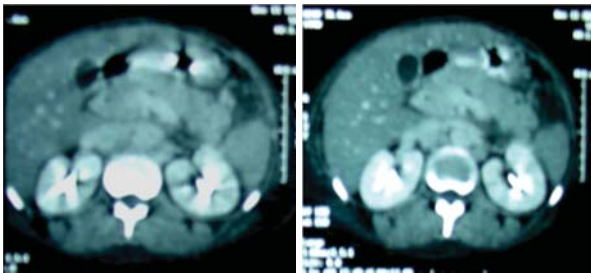


Figure 1. The abdominal computed tomography scans findings compatible with acute pancreatitis

Conservative treatment by impression of acute pancreatic was begun.

After that, other complaints such as dyspnea, orthopnea, tachypnea, and also an episode of seizure were added to her symptoms and her general condition became deteriorated. Her physical examination was as below: RR (130/min), PR (110/min), oral temperature (38° C), and BP (100/70 mmHg).

The heart examination showed tachycardia and also muffling of the heart sounds. Echocardiography showed sever systolic dysfunction and cardiac output of 20%. Furthermore, there was a moderate pericardial effusion. The laboratory data was creatine phosphokinase-MB = 301 (0.3-4 ng/ml), troponin-I = 0.5 (0.2), all of these findings were compatible with myocarditis.

Because of polyserositis, fever, leukopenia and positive serologic markers [anti-double strand DNA > 200 (< 30 IU/ml), anti-nuclear antibody > 100 (< 10 U/ml), the diagnosis of SLE was confirmed]. The patient was treated with methylprednisolone (1 g × 3 days), cyclophosphamide (1 g), and hydroxychloroquine (400 mg/day). She gradually became better and was discharged

after 40 days of her admission. Her laboratory data on discharge have shown in table 1.

Discussion

We reported an interesting case of SLE with a rare manifestation of pancreatitis. Although our patient had some symptoms of SLE, these symptoms were confusing with other diseases such as infection and malignancy which were considered first. As we know, early diagnosis and treatment of SLE patient with major organ involvement are critical because it could be lifesaving and reduce the complication of the disease (4, 5). Our patient showed some symptoms of SLE such as fever, skin rashes, leukopenia, and polyserositis which guided us to evaluate serologic tests of SLE and consider SLE as a cause of lupus pancreatic (LP).

The prevalence of LP varies from 4% to 8.3% based on its underlying etiology such as alcohol usage, gallstone, and surgery (3) and LP just due to SLE is much more uncommon as Reifenstein et al. reported 6 patients out of 69 SLE patients were suffered from acute pancreatitis which was their first presentation (4).

Pathogenic mechanisms of LP are complex and multifactorial. Vessel damages due to vasculitis, immune complex deposition in vessels, thickness of intimal layer of vessels, obstruction of artery or arteriole, autoantibody production, drug toxicity (azathioprine, cyclosporine, steroids, and diuretics), and all are considered (3, 4). Presentation of pancreatitis in SLE patients usually is subclinical but it may be acute, subacute or chronic. It usually occurs in the setting of active disease with major organ involvement. The diagnosis of acute pancreatitis is considered if there is abdominal pain, nausea, vomiting and confirmed with a high level of amylase (≥ 2 -fold) and CT scan findings. The diagnosis of LP will be considered only after rolling out the other cause of pancreatitis such as alcohol use, gall stone, viral infection especially human immunodeficiency virus

(4, 6). The diagnosis of pancreatitis in our patient was confirmed based on its clinical manifestations and high level of serum amylase (≥ 3 -fold) and also CT scan finding (Figure 1). On the other hand, we could not find any other etiologies such as alcohol use, infection, drug use, gallstone, and surgery. We concluded that the acute pancreatitis was occurred due to SLE alone. On the other hand, good response to the treatment was in favor of LP.

Some studies advised steroid and cytotoxic therapy especially cyclophosphamide for the treatment of LP and gastrointestinal vasculitis (7). Although, there were some reports of successful treatment with rituximab in resistance cases (6, 7). Fortunately, our patient had a considerable response to the treatment of steroid and cyclophosphamide and discharged with the acceptable general condition.

This interesting case reminds us that we should consider SLE in the differential diagnosis of acute pancreatitis especially in a young woman without any underlying etiology even if we could not find any symptoms or history of SLE in the presentation.

Conflict of Interests

Authors have no conflict of interests.

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