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• *CASE REPORT* •

Acute pancreatitis as an initial symptom of systemic lupus erythematosus: A case report and review of the literature

Feng Wang, Nian-Song Wang, Bing-Hui Zhao, Ling-Quan Tang

Feng Wang, Nian-Song Wang, Ling-Quan Tang, Department of Nephrology, the Sixth Hospital attached to Shanghai Jiaotong University, 600 Yishan Road, Shanghai 200233, China
Bing-Hui Zhao, Department of Radiology, the Sixth Hospital attached to Shanghai Jiaotong University, 600 Yishan Road, Shanghai 200233, China
Correspondence to: Feng Wang, Department of Nephrology, the Sixth Hospital attached to Shanghai Jiaotong University, 600 Yishan Road, Shanghai 200233, China. zyzwq1030@sohu.com
Telephone: +86-21-64369181-8379
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Abstract

Acute pancreatitis as an initial symptom of systemic lupus erythematosus (SLE) is rare. We present a report of a 46-year-old female patient who had fever, abdominal pain and vomiting, elevated pancreatic enzyme levels, hypocalcemia, hypoxemia, and various other laboratory abnormalities. She was first diagnosed with acute severe pancreatitis and then with SLE after further investigations. After a 2-mo treatment with somatostatin, the patient recovered.

Key words: Systemic lupus erythematosus; Pancreatitis

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INTRODUCTION

Systemic lupus erythematosus (SLE) is a multi-system, autoimmune disorder characterized by a broad range of manifestations, including presence of antibodies against cell nuclei. The initial manifestations of SLE, however, can involve many organ systems either singly or in combination, which frequently makes diagnosis difficult. The American Rheumatism Association recommends that the diagnosis of SLE can be confirmed if it meets 4 of the following 11 revised criteria: malar rash, discoid rash, photo-sensitivity, oral ulcers, arthritis, serositis, renal disorder, neurologic disorder, hematologic disorder, immunologic disorder, and antinuclear antibodies.

We report a 46-year-old female patient with fever, abdominal pain and vomiting, elevated levels of pancreatic enzyme, hypocalcemia, hypoxemia, and other laboratory abnormalities. She was first diagnosed with acute severe pancreatitis and then with SLE, after further investigations.

CASE REPORT

A 46-year-old woman came to the Sixth Hospital attached to Shanghai Jiaotong University, complaining of a 6-d history of persistent abdominal pain, loss of appetite, occasional nausea and vomiting without obvious reasons. She also had a 3-d history of fever and generalized weakness.

She reported bilateral thigh pain, finger pain and swelling 5 mo ago, which were abated after 7-d treatment with hydroxy-chloroquine. Four months ago, she had elevated alanine aminotransferase, and received no treatment. She also reported some thinning of her hair and a weight loss of 5 kg in the past 2 mo. Her family history was unremarkable for auto-immune disorders.

Her body temperature was 38 ℃, pulse rate 100 beats per minute, blood pressure 120/80 mmHg. She had no skin lesions, but had swollen and painless lymph nodes. She had fine rales on bilateral lungs, and 3-degree systolic murmur in the mitral valve area and a pericardial friction sound. Her abdomen was soft with mild epigastric tenderness, minimal guarding, and no rebound tenderness.

Her blood chemistry was as follows: 126 mmol/L sodium, 4.4 mmol/L potassium, 97 mmol/L chloride, 22.5 mmol/L bicarbonate, 1.6 mmol/L blood urea nitrogen, 31 μmol/L creatinine, 5.1 mmol/L glucose, 83 U/L aspartate aminotransferase, 16 U/L alanine aminotransferase, 66 g/L protein, 37 g/L albumin, 1.70 mmol/L calcium, and 18 μmol/L total bilirubin. A complete blood count showed a white cell count of 3.0×10^9/L with 78.9% neutrophils, 19.8% lymphocytes, 2.3% monocytes. Hemoglobin was 89 g/L, hematocrit 33.8%, and platelets 112×10^9/L. Pancreatic enzyme determinations disclosed a blood amylase level of 1 000 U/L (normal 20-115 U/L) and urine amylase level of 830 U/L, and a lipase level of 690 U/L. Urinalysis showed cloudy urine with a protein level of 260 mg/dL, a white cell count of 3-4/μL, granular casts 0. Nitrite and leukocyte esterase were negative. Blood culture was negative.

Computed tomographic (CT) scan and ultrasonic scan of the abdomen with contrast, showed swollen pancreas but no gallbladder stones (Figure 1A). Another CT scan of the bosom showed effusion both in right and in left pleurae (Figure 1B).

Connective tissue showed an antinuclear antibody (ANA) titer of 1:640, positive anti-double-stranded DNA (dsDNA) antibody, negative anti-Smith antibody, anti-ribonucleoprotein, anti-SSA, and anti-SSB. The erythrocyte sedimentation rate was 92, complement 3 (C_3) was 0.75 g/L (normal 1.0-1.8 g/L),

anti-cardiolipin antibodies and lupus anti-coagulant were negative.

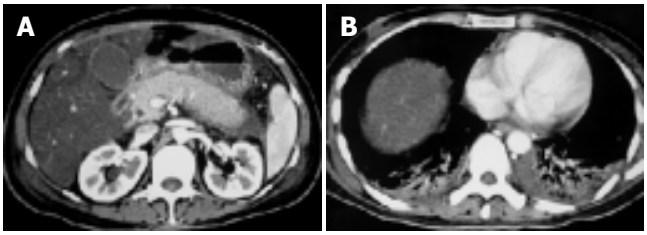

Figure 1 Swollen pancreas (**A**) and pleural effusion (**B**) before treatment with somatostatin.

After a 2-mo treatment with somatostatin, the pancreatic enzyme was 29 U/L. Repeated CT scan revealed a less swollen pancreas (Figure 2A) and still bilateral pleural effusion (Figure 2B). But the patient still had fever and palpitation. A diagnosis of SLE pancreatitis was made on the 66th d of admission, and 40 mg methylprednisolone intravenous injection qd was started. Once steroid therapy began, the heart rate declined to 90 beats per minute. Two weeks after continuous steroid therapy, the heart rate was 78 beats per minute and the temperature was 36.6 ℃. Pleural effusion disappeared.

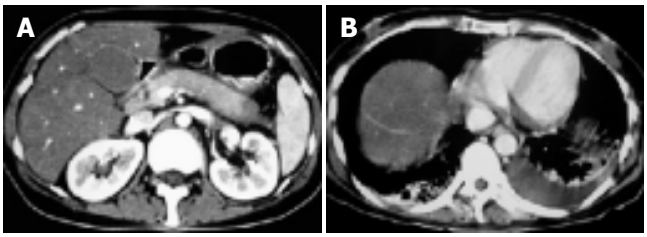

Figure 2 Less swollen pancreas (**A**) and pleural effusion (**B**) after treatment with somatostatin.

DISCUSSION

Although the patient on admission was positive for ANA and anti-dsDNA, arthritic, serositic and blood cells reduced, which fulfills the American Rheumatism Association criteria for the diagnosis of SLE. Our case illustrates the protean manifestations of SLE and a rare initial manifestation as acute pancreatitis. Over 50% SLE patients have manifestations of digestive system, but pancreatitis is few, and such acute pancreatitis as an initial manifestation is even rare. The association between SLE and pancreatitis was first documented in 1939[1]. Since then, only 70 cases of SLE pancreatitis have been documented in the literature, and only 10 of these cases, including this one, have pancreatitis as their initial manifestation[2-5].

The mechanisms involved in the origin of SLE pancreatitis are not clear. SLE pancreatitis might result from vasculitis, microthrombi, anti-pancreas autoantibody, side-effects of medicine, intimal thickening and virus infection. Most lupus pancreatitis is found in patients with long-standing SLE who have multi-organ involvement and are already on steroid, diuretic, or immunosuppressive therapy, all of which have been implicated in the etiology of pancreatitis. Since the initial description of SLE pancreatitis, whether steroid or SLE is the primary cause has been controversial. Recent studies support that SLE is the primary etiologic factor of SLE pancreatitis, and drug toxicity may also play a role[5-9].

Pathological changes of SLE pancreatitis bear similarities to those of pancreatitis due to other causes. SLE pancreatitis can occur during a generalized flare-ups and disease quiescence, though the latter seems more likely[3]. Its manifestation can be acute, severe, or chronic, self-limiting or fulminant. It is believed that subclinical pancreatitis which has an elevation of pancreatic enzymes without clinical symptoms is much more frequent than symptomatic pancreatitis[10-14]. About 30.5% of asymptomatic SLE patients have hyperamylasemia; therefore subclinical pancreatic damage might occur very frequently in SLE[15].

Within the context of SLE, the diagnosis of SLE pancreatitis is usually based on clinical findings as abdominal pain, nausea, and vomiting as well as laboratory findings like abnormal pancreatic enzymes and suggestive tomographic findings. Etiology diagnosis is fairly important, which decides whether steroid can be used. If drug toxicity is not considered as the origin, SLE pancreatitis is treated with steroid. Of course, steroid or other drugs should be stopped once they are regarded as the cause[16,17].

In conclusions, it is important to determine whether drug toxicity is the cause of SLE pancreatitis. If the condition of patients with SLE pancreatitis can be improved with somatostatin, steroid might not be necessary during the acute episode.

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