Case Report

DOI: 10.5582/irdr.2017.01000

Silence pancreatitis in systemic lupus erythematosus

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Summary **We present here a systemic lupus erythematosus (SLE) related biochemically silent pancreatitis which was assessed *via* computed tomography in a 35-year-old woman. A patient with a twelve-year history of SLE presented with exacerbation of symptoms of the basic disease, with SLE Disease Activity Index > 15. She was referred to inpatient care. Dosage of corticosteroid and azathioprine for SLE was increased; subclinically and biochemically silent pancreatitis had developed, and was not diagnosed within an appropriate time. On the 15th hospital day, the patient died due to multisystem organ failure, which was defined as a consequence of clinically and biochemically silent pancreatitis in systemic lupus erythematosus.**

Keywords: Pancreatitis, systemic lupus erythematosus, pancreatic enzymes, computed tomography

1. Introduction

Systemic lupus erythematosus (also known as lupus or SLE) is a chronic inflammatory disease that can affect various parts of the body. The cause of lupus is not well understood; it is an autoimmune condition, meaning that the body's immune system attacks its own tissues as if they are foreign. Virtually every system and organ can be affected by SLE. The gastrointestinal tract is one of the most commonly affected systems in SLE, and the incidence of gastrointestinal manifestations may be underestimated clinically because some of these are indistinct and may not have abdominal symptoms (*1,2*).

About 160 cases of SLE related acute pancreatitis have been reported in the literature (*2*). The main causes of pancreatitis are mechanical obstructions of the pancreatic duct and toxic metabolites such as alcohol intake and certain drugs. However, a number of patients develop "idiopathic" pancreatitis, in which no aetiology other than SLE itself can be identified. The involvement of the pancreas in SLE is rare; elevated serum amylase and lipase are the most commonly detected biochemical abnormalities. Other biochemical abnormalities include hypertriglyceridemia, hypocalcaemia, hypoalbuminemia, abnormal liver function tests and elevated serum creatinine. The exact pathogenic mechanisms of SLE related pancreatitis are not clear, but are probably mediated by immune complex-induced microangiitis (*2-4*).

The aim of this work is to report on an SLE related clinically and biochemically silent pancreatitis, which was assessed *via* diagnostic imaging in a 35-year-old woman.

2. Case Report

A 35-year-old woman with a 12-year history of SLE presented because of exacerbation of symptoms of the basic disease such us joint pain, headache, myositis, rash, *etc*. (SLE Disease Activity Index > 15). She was referred to inpatient care.

She had no history of other disease or surgery. In the last twelve years treatment had been based mostly on azathioprine (50 mg/day) and corticosteroids (20 mg/day).

The patient had been well until seven days before admission, when fatigue and malaise developed. On

Released online in J-STAGE as advance publication April 16, 2017.

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Table 1. Complete blood count, laboratory analysis and serology during hospital stay

Items	On admission	4th day	9th day	13th day
Hemoglobin 12-15 g/dL	8.1	7.4	9.1	8.3
Hematocrit 36-47%	28	26	33	24
Mean corpuscular volume 80-100 fL	71	67	76	68
Red Blood Cells 4.2 -5.4 million/mcL	3.1	2.7	3.8	2.8
White blood cells 4-10 × 10^9/L	12.6	13.4	15.9	15.4
Neutrophils (%)	76	79	84	86
Lymphocytes (%)	14	12	11	11
Monocytes (%)	7.5	6.9	4	2
Platelets 150-400 × 10^9/L	112	99	88	75
Erythrocyte sedimentation rate in first hour	60		105	120
C-reactive protein < 0.5 mg/dL	75	98	120	177
International normalizedratio 0.9-1.2		1.1		
Glucose 4.1-6.1 mmol/L	5.5	5.9	6.3	7.6
Urea nitrogen 2.9-7.1 mmol/L	10.30	12.90	14.8	10.5
Creatinine 61.9-115 µmol/L	290	330.7	462	295
Triglycerides < 2.82 mmol/L	1.7		1.6	1.5
Total cholesterol 3-5.5 mmol/L	5.1		5.0	5.2
Sodium 135-145 mmol/L	144		149	140
Potassium 3.5-5 mmol/L	5.4	5.7	6.6	5.5
Total calcium 2-2.6 mmol/L	2.26	2.12	1.79	1.37
Phosphate 0.8-1.5 mmol/L			1.2	
pH 7.35-7.45	7.39	7.41	7.41	7.44
Bicarbonate 18-22 mmol/L	34	40	41	49
Total protein 60-80 g/L	64			55
Albumin 35-50 g/L	31			24
Aspartateaminotransferase 5-30 U/L	38		60	110
Alanineaminotransferase 5-30 U/L	61		75	158
Gammaglutamyltransferase 6-50 U/L	35		49	68
Alkalinephosphatase 50-100 U/L	68		94	115
Amylase 30-125 U/L		70	95	120
Lipase 10-150 U/L		90		148
Total bilirubin: 2-20 µmol/L		17.8		25
Direct bilirubin: 0-6 µmol/L		5		9
Antinuclear antibodies			positive	
Lactatedehydrogenase 60-160 U/L	390	378	420	385
Urine mL/day		900	800	450

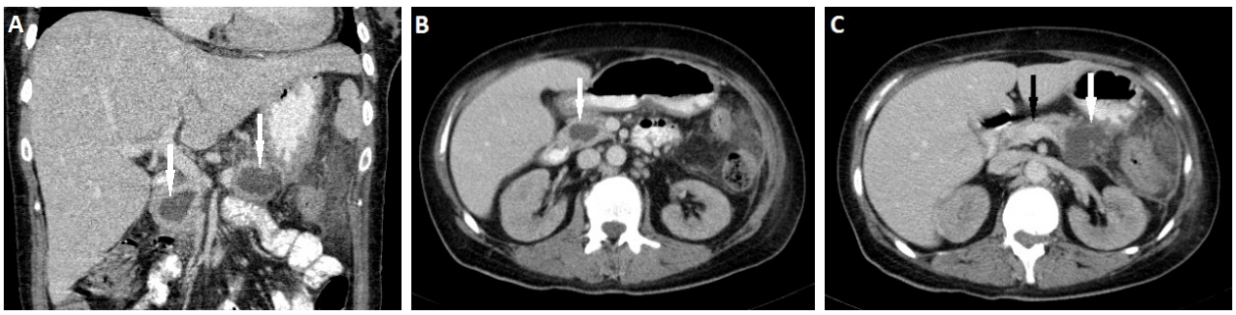

Figure 1. Computed tomography scans (A), (B), (C) show enlarged pancreas with irregular echogenicity and fluid collection (white arrows), (C) scan shows slightly dilated pancreatic duct (black arrow).

examination she felt feverish. Temperature was 37.8°C, blood pressure 110/70 mmHg, pulse 90 beats per minute and respiratory rate 16 breaths per minute. She was alert and oriented.

Physical examination showed swollen joints (elbow, wrist, knee), facies and skin rash. There were no meaningful symptoms from the chest and abdominal organs. Complete blood count (CBC), laboratory analysis and serology markers on admission and during hospital stays are shown in Table 1.

Initially, imaging assessment *via* real-time sonography of abdominal organs (pancreas, liver, gallbladder, spleen, both kidneys) and chest X-ray showed normal appearance.

Within the next few days, besides increased doses of azathioprine (150 mg/day) and corticosteroids (60 mg/day) for SLE, the patient underwent ciprofloxacin (1 g/day), pantoprazole (40 mg/day), NSAID and physiological solution (500 mL/day for the first three days). On the eighth day two units of packed red cells

were transfused. Over this time, she had oral intake of food and drugs.

During inpatient care, the general aspect of the patient worsened, and renal failure significantly increased with mild general swelling. On the 11th hospital day, due to renal failure, the patient was referred to haemodialysis *via* central venous catheter (CVK), and a control chest X-ray showed the appropriate position of the catheter in the superior cava vein. On the same day, real-time sonography showed an enlarged pancreas with irregular morphology and dilated pancreatic duct, which was additionally confirmed by computed tomography (CT) - Figure 1. A day later, pancreatic enzymes remained in the referral range.

On the 15th hospital day, the patient died due to multisystem organ failure, defined as a consequence of clinically and biochemically silent pancreatitis in SLE.

3. Discussion

The diagnosis of acute pancreatitis is based on clinical presentation, laboratory tests where serum amylase and/or lipase are more than three times the upper limit of normal, and CT scan findings. It is difficult to assess the disease due to the lack of accurate and uniformly accepted definitions of disease severity and commonly encountered complications of acute pancreatitis (*5*).

The incidence of SLE-related pancreatitis may be underestimated, because cases of subclinical pancreatitis with elevated pancreatic enzymes but no symptoms are not diagnosed or reported (*2*).

In SLE-related pancreatitis, approximately 90% have abdominal pain, almost 75% of patients have nausea and vomiting, and about 50% have fever (*2,4,6*).

During 12 days of inpatient care, our patient did not have or did not report abdominal or epigastric pain; there was no vomiting, and throughout this time she had an oral intake.

In SLE related pancreatitis, increased values for serum amylase and lipase are the most commonly detected (*2,6*). Normal serum amylase levels have been reported in some cases of acute pancreatitis, but serum lipase levels are usually elevated. Normal serum lipase in the setting of acute pancreatitis is an extremely rare occurrence. A literature review by Shah *et al*. found only two case reports of clinical and radiological evidence of acute pancreatitis with a normal serum lipase level (*7*).

In our case, both pancreatic enzymes, amylase and lipase, remained within normal limits during the hospital stay.

Additional biochemical abnormalities in SLE related pancreatitis include hypoalbuminemia in 78%, abnormal liver function tests in 65% and elevated serum creatinine 44% (*2,4*). Biochemical values for albumin, serum creatinine, liver function tests, *etc*. are presented in Table 1.

Pascual-Ramos *et al*. (*8*) reported that the SLE disease activity index was significantly increased in patients with idiopathic pancreatitis. Their study ruled out the assumption that corticosteroids and azathioprine could cause SLE related pancreatitis. Our patient was admitted in the active phase of the basic disease, and the dosage of corticosteroid and azathioprine for SLE was increased. Abnormalities in CBC are also common: anaemia, leukopenia, and thrombocytopenia are relatively common (81%, 59%, and 48% respectively), while leukocytosis is infrequent (only 15%) (*4*). In our case she had a CBC all the time with severe anemia and thrombocytopenia; leukocytosis existed during inpatient care, and non-specific inflammatory parameters such as C-reactive protein and erythrocyte sedimentation rate were also elevated. Based on imaging assessment of the pancreatic disorder and high SLE disease activity in addition to anemia, thrombocytopenia, hypoalbuminemia, abnormal liver function test and elevated serum creatinine; SLE related pancreatitis had developed, and unfortunately was not diagnosed within an appropriate time.

The high mortality rate in SLE pancreatitis is associated with severe disease activity, thrombocytopenia and acute renal failure (*3,4,6*). Due to clinically significant renal failure, our patient was referred to haemodialysis treatment *via* CVK. On the same day, CT revealed enlarged and irregular morphology of the pancreas, and four days later she died due to multisystem organ damage.

Our case showed an SLE related biochemically silent pancreatitis with normal serum lipase and amylase, which was mainly diagnosed *via* contrast enhanced CT. There are no cases in the recent literature which describe SLE related pancreatitis with normal levels of serum lipase and amylase.

In conclusion, although uncommon, acute pancreatitis should be considered in the differential diagnosis of abdominal pain in SLE patients. Mechanical obstruction (most frequently a result of choledocholithiasis) and toxic-metabolic aetiologies (secondary to alcohol intake, certain drugs, hypocalcaemia or hypertriglyceridemia) should be ruled out. The early diagnosis of acute pancreatitis in SLE patients, especially those with abdominal pain, and appropriate treatment, is beneficial for a better therapeutic outcome in the majority of patients. Acute pancreatitis can have a variable presentation, and physicians caring for patients who are presented to the hospital with epigastric pain should be aware despite normal amylase and lipase levels. In appropriate clinical conditions, further imaging modalities such as CT scans may be helpful.

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(Received January 14, 2017; Revised March 29, 2017; Accepted April 2, 2017)