

Systemic lupus erythematosus-related acute pancreatitis: a case report

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Systemic lupus erythematosus presenting as acute pancreatitis is rare. We report a case of a 12-year-old girl with a 1-year history of systemic lupus erythematosus who developed active pancreatitis. The pancreatitis was first manifested by nausea, vomiting, fever, and abdominal pain. Elevated serum amylase (578 U/L) and lipase levels (5588 U/L), and pancreatic enlargement on ultrasound and computerized tomography confirmed the diagnosis. She responded well to high-dose corticosteroid. The high titer of antinuclear antibodies (1:1280) and low level of complement components (C3, 42.9 mg/dL; C4, 2.3 mg/dL) during the pancreatitis attack suggested that the pancreatitis may have been due to systemic lupus erythematosus exacerbation and not related to drug therapy.

Key words: Acute pancreatitis, systemic lupus erythematosus

Acute pancreatitis is a rare but often lethal complication in the course of systemic lupus erythematosus (SLE). The role of SLE in the development of acute pancreatitis is unclear because the cause is often obscured by the concomitant use of drugs that can also produce pancreatitis or by coexistent medical problems [1]. Although corticosteroids are an important part of the management of patients with SLE, they have been noted to cause [2,3] or exacerbate [4,5] the course of pancreatitis, which may create a dilemma regarding the choice of therapeutic intervention in this life-threatening condition. However, reports of lupus patients with pancreatitis prior to the use of steroids [1,6-8] have suggested that pancreatitis may be a manifestation of active lupus vasculitis. We report a case of a 12-year-old girl with SLE who developed acute pancreatitis with no apparent cause other than her underlying disease. Prednisolone in a tapering dose was the only drug being taken at the onset of acute pancreatitis. Whether the corticosteroids could have caused the pancreatitis in this lupus patient or the pancreatitis occurred as a consequence of a generalized SLE flare-up could not be definitively established, but there appeared to be a direct relationship between episodes of pancreatitis and SLE activity. The girl's signs and symptoms subsided after high-dose corticosteroids treatment was given.

Early application of sonography or computed tomography to any suspected lesion can help to establish the diagnosis of pancreatitis in patients with SLE.

Case Report

A 12-year-old girl was admitted to our hospital because of nausea, vomiting, fever, and increasingly severe abdominal pain for 2 days. The pain radiated to the back and relieved in the knee-to-chest position. She did not experience these symptoms previously. One year before admission, SLE was diagnosed after she presented with alopecia, arthritis, proteinuria, malar rash, and positive antinuclear antibodies (ANA), and anti-double stranded (ds) DNA were found. Her disease activity was controlled by prednisolone 15 to 60 mg/d. There was no hepatobiliary disease, hypercholesterolemia, or hyperparathyroidism, although a review of her charts found no record of serum amylase values. She had no family history of SLE.

Physical examination revealed a slender ill-looking girl. Her vital signs were a body temperature of 37.8°C, a pulse rate of 120/min, a respiration rate of 18/min, and a blood pressure of 110/65 mm Hg. Pale conjunctivae and malar rash were noted. Abdominal examination showed tenderness during deep palpation in the epigastrium and left upper quadrant. Bowel sounds were reduced. Peripheral blood revealed white blood cell (WBC) count 4410 /mm³, hemoglobin 10.2 g/dL (normal, 12-16 g/dL), platelets 260 000 /mm³, and erythrocyte sedimentation rate 67 mm/h (normal, 0-20 mm/h). Total protein and albumin were 6.7 and 3.1 g/

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dL (normal, 4-5.3 g/dL), respectively. Serum glucose, sodium, potassium, chloride, total and free calcium, cholesterol, triglycerol, blood urea nitrogen (BUN), creatinine, aspartate and alanine aminotransaminase, alkaline phosphatase, total bilirubin, pH, and bicarbonate were within normal. Prothrombin and partial thromboplastin times were within normal limits. Proteinuria was 3+ on urine analysis, and daily protein loss from urine was 1.2 g/d. Blood and urine cultures were negative. Serum amylase and lipase were 578 U/L (normal, 30-150 U/L) and 5588 U/L (normal, 20-250 U/L). The rheumatological profile revealed hypocomplementemia (C3, 42.9 mg/dL [normal, 83-177 mg/dL]; C4, 2.3 mg/dL [normal, 15-45 mg/dL]). Rheumatoid factor and VDRL (Venereal Disease Research Laboratory) were negative. An antibody screen was positive, including ANA 1:1280 (normal, <1:40), with a peripheral pattern. Roentgenogram of the abdomen was unremarkable. Sonography of the abdomen revealed swelling of the pancreas. Contrast-enhanced abdominal computerized axial tomography demonstrated fluid accumulation surrounding the swollen pancreatic tail, which was consistent with a diagnosis of acute pancreatitis (Fig. 1). After administration of adequate hydration, high-dose intravenous methyl-prednisolone (45 mg) daily, and being maintained with nothing by mouth for consecutive 5 days, the abdominal pain improved gradually. The dose of prednisolone was then slowly tapered, and 2 months later her serum amylase, lipase level, and C3 and C4 level were returned to normal range.

Discussion

While more than 90% of lupus patients have abdominal

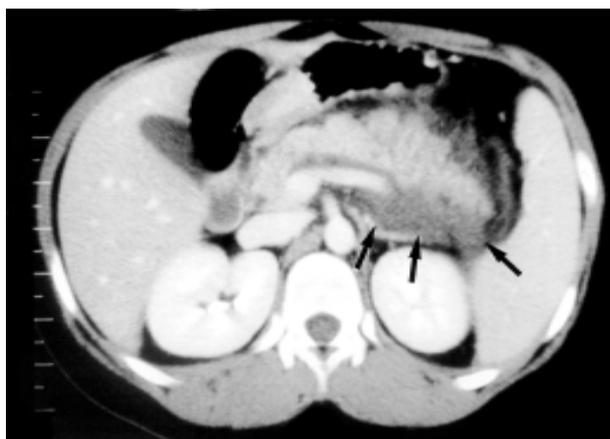


Fig. 1. Contrast-enhanced CT scan at the level of the splenic vein reveals fluid accumulation (arrows) surrounding the swollen pancreatic tail.

pain [8], acute pancreatitis in association with SLE is exceedingly rare. Abdominal pain and elevated serum amylase are the hallmarks of pancreatitis, but occur in only 8% of lupus children with pancreatitis [8]. As the mortality rate for acute pancreatitis in lupus patients is more than 60% [9], the diagnosis of acute pancreatitis should be considered if the lupus patients present with abdominal complaints.

In this case, the common causes of acute pancreatitis were excluded, including trauma history, serological evidence of infectious diseases, history of gallbladder diseases, and alcohol abuse history. The only drug being taken at the onset of acute pancreatitis was steroid.

The existence of steroid-induced pancreatitis remains controversial. Results derived from animal models and human autopsy studies suggest that steroids may cause pancreatic lesions [10,11]. Steroids may enhance immune complex deposition [12], and cause a significant rise in serum amylase [13]. As steroids are such a critical part of the management of lupus patients, side effects of treatment with steroids should be considered for their potential role in the pathogenesis of pancreatitis. Besides, the ulcerogenic effect of steroids may cause abdominal pain. Thus, the use of steroids for treatment of the lupus patients with abdominal pain, and even of lupus patients with suspected acute pancreatitis, poses a dilemma for clinicians.

Steroids treatment was successful in the management of this case. The clinical course of this patient suggested a direct relationship between episodes of pancreatitis and SLE activity. Evidence suggesting an association of SLE disease activity with pancreatitis included low serum C3 and C4 level, and increased ANA titers and anti-ds DNA antibodies. Clinical and biochemical improvement in our patient was clearly related to corticosteroid administration.

In conclusion, this case report may serve as a reminder of important information about the clinical features and causes of acute pancreatitis in patients with SLE. The pathogenesis of pancreatitis in SLE patients remains poorly understood and most likely results from multiple mechanisms. Steroid therapy was unlikely to be the cause of the pancreatitis in this patient, as additional higher dose steroid treatment resulted in relief of her signs and symptoms rather than in exacerbation. The observation suggests that steroid therapy should not be withheld from lupus patients with abdominal pain, especially in those with evidence of a generalized SLE flare-up. Early use of abdominal sonography or computed tomography plays an important role in the

diagnosis of pancreatitis in the lupus patient with the initial manifestation of abdominal pain. Aggressive treatment may save the patient's life.

References

1. Marino C, Lipstein-Kresch E. Pancreatitis in systemic lupus erythematosus. *Arthritis Rheum* 1984;27:118-9.
2. Baron M, Brisson ML. Pancreatitis in systemic lupus erythematosus. *Arthritis Rheum* 1982;25:1006-9.
3. Kapoor D, Mendez E, Espinoza LR. Corticosteroids and SLE pancreatitis. *J Rheumatol* 1999;26:1011-2.
4. Schrier RW, Bulger RJ. Steroid-induced pancreatitis. *JAMA* 1965;194:564-5.
5. Borum M, Steinberg W, Steer M, Freeman S, White P. Chronic pancreatitis: a complication of systemic lupus erythematosus. *Gastroenterology* 1993;104:613-5.
6. Takasaki M, Yorimitsu Y, Takahashi I, Miyake S, Horimi T. Systemic lupus erythematosus presenting with drug-unrelated acute pancreatitis as an initial manifestation. *Am J Gastroenterol* 1995;90:1172-3.
7. Huang JL, Huang CC, Chen CY, Hung IJ. Acute pancreatitis: an early manifestation of systemic lupus erythematosus. *Pediatr Emerg Care* 1994;10:291-3.
8. Reynolds JC, Inman RD, Kimberly RP, Chuong JH, Kovacs JE, Walsh MB. Acute pancreatitis in systemic lupus erythematosus: report of twenty cases and a review of the literature. *Medicine* 1982;61:25-32.
9. Watts RA, Isenberg DA. Pancreatic disease in the autoimmune rheumatic disorders. *Semin Arthritis Rheum* 1989;19:158-65.
10. Pescador R, Manso MA, Rebollo AJ, de Dios I. Effect of chronic administration of hydrocortisone on the induction and evolution of acute pancreatitis induced by cerulein. *Pancreas* 1995;11:165-72.
11. Carone FA, Liebow AA. Acute pancreatitis lesions in patients treated with ACTH and adrenal corticoids. *N Engl J Med* 1957;257:690-7.
12. Haakenstad AO, Case JB, Mannik M. Effect of cortisone on the disappearance kinetics and tissue localization of soluble immune complexes. *J Immunol* 1975;114:1153-60.
13. Dandona P, Junglee D, Katrak A, Fonseca V, Havard CW. Increased serum pancreatic enzymes after treatment with methylprednisolone: possible evidence of subclinical pancreatitis. *Br Med J* 1985;291:24.