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Case report

Therapy of acute pancreatitis in systemic lupus erythematosus with plasmapheresis and corticosteroids

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ABSTRACT. *A 24-year-old woman with systemic lupus erythematosus had, after reduction of corticosteroid therapy, a severe relapse of the disease with hepatitis, nephritis and pleurisy. After admission to the hospital, she was given 60-80 mg/day of prednisone and acute pancreatitis developed on the third day.*

Plasmapheresis, followed by injection of 1 g of methylprednisolone, was started. This combined therapy induced a prompt and complete recovery in a few days.

Key words: acute pancreatitis, systemic lupus erythematosus, plasmapheresis, corticosteroids.

Introduction

The occurrence of acute pancreatitis in the course of systemic lupus erythematosus (SLE) has been previously described (1-4). However, because of the concomitant use of drugs that are known to cause pancreatitis, the role of SLE in the development of this complication is still controversial. Reports of SLE patients with pancreatitis prior to the use of corticosteroids or without steroid therapy have suggested that pancreatitis may be caused by active lupus vasculitis (5). In view of this, corticosteroid therapy should not be discontinued, but increased.

On the other hand, plasmapheresis has recently been considered as a potentially valuable adjunct in the treatment of patients with active (SLE) (6), though concurrent drug therapy appears to have an important role in the duration of the effect of plasmapheresis (7). A case of acute pancreatitis in SLE is reported. A prompt recovery was obtained with plasmapheresis combined with pulse corticosteroid administration.

Case report

A 24-year-old woman. known to have had SLE for two years, showed an acute severe relapse of the disease with hepatitis, nephritis, pleurisy and pericarditis one month after reduction of corticosteroid therapy (from 16 to 4 mg/day of methylprednisolone). On admission to our hospital, physical examination revealed fever of 39°C, a pulse of 110, dyspnea, dullness and diminished breath sounds over the right pulmonary base, pericardial friction rubs, a gallop sound, mild edema of the ankles, moderate liver enlargement, face and malar rash.

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Laboratory evaluation revealed: WBC 2,500/mm³, Hb 10 g/dl, BUN 81 g/l, serum creatinine 1.6 mg/dl, serum protein 4.5 g/dl, albumin 2.2 g/dl, hematuria 2+, proteinuria 5 g/l, SGOT 318 U/l (normal values < 40 U/l), SGPT 204 U/l (normal values < 55 U/l), LDH 1033 U/l (normal values < 360 U/l), CPK 181 U/l (normal values < 190 U/l) and alkaline phosphatase 703 U/l (normal values < 78 U/l). Other results were: antinuclear antibodies present at a titer of 1:640 with a shaggy pattern, anti ds-DNA (*Crithidia Luciliae*) at 1:320, complement activity (measured as CH_{50}) zero, circulating immune complexes, by the Raji cell method (8), present at high titer, mostly of the IgG class. Serum amylase was not tested. A chest radiograph showed a pleural effusion on the right side; an abdominal sonogram was normal with the exception of a thin fluid layer between the right lobe of the liver and the diaphragm.

Therapy was started with 60 mg of oral methylprednisolone and cimetidine 1 g/day. Antibiotics, started two days prior to admission, were continued. The steroids were increased to 80 mg on the second day. Suddenly on the third day, the patient complained of nausea, vomiting and severe epigastric pain radiating to the back. Serum amylase was high (470 U/l, normal values < 36 U/l and the transaminases SGOT and SGPT increased further. A repeat abdominal sonogram revealed an enlarged pancreas head of 5 cm not present on the previous study.

Nasogastric suction was applied and the medication was continued unaltered. The next day the serum amylase increased (685 U/l) and the patient was severely ill.

Because of the pancreatitis, it was considered risky to increase the steroid dosage. Plasmapheresis was therefore commenced with the removal of 70% of total patient plasma. The plasma was replaced by saline and albumine 5%. After the first plasmapheresis on the same day, pulse steroid therapy was begun with the i.v. injection of 1 g of methylprednisolone. The plasma exchange was repeated three times in the first week and another time in the second week after an interval of 7 days. Corticosteroids were increased during ten days until a dosage of 40 mg/day was reached. The patient had a prompt remission of her abdominal discomfort. Furthermore, the sonogram showed a remarkable reduction in the pancreas size.

In eight days both the pancreatitis and the hepatitis had resolved. The other SLE manifestations (pleurisy, nephritis and pericarditis) improved in ten days.

Slight proteinuria (± 1 g/24 hours) persisted for another 4 months. The levels of transaminases, amylase, circulating immune complexes and antinuclear antibodies rapidly declined. Complement activity was detectable five days after the first plasmapheresis. Immune complexes, mostly IgM, (IgM titer 1:64, and IgG 1:16) increased again fourteen days after the first plasmapheresis.

Treatment with cyclophosphamide (75 mg/day) was started, and corticosteroids were further reduced to 12 mg/day. The patient is still continuing this therapy, but the corticosteroid dose is 4 mg/day after one year. She feels very well, and is about to graduate from the University.

Discussion

Acute pancreatitis became manifest in this patient during increased corticosteroid therapy for a severe exacerbation of SLE. Both SLE vasculitis and drug administration could have been the cause of such a complication. Indeed, several case reports have implicated corticosteroid therapy as a cause of pancreatitis (9-12). It was considered unwise to continue with corticosteroids alone, and therefore a combined therapy was attempted. The response to this combined treatment was impressive, all signs and symptoms improving within a few days. It is hard to say which therapeutic procedure was the more effective in our patient. It is certain that the administration of a steroid bolus did not aggravate the pancreatitis suggesting that this complication was related to the SLE rather than to corticosteroid therapy. Although biopsy confirmation was not obtained, it is likely that vasculitis was the pathogenetic event in the development of pancreatitis in our patient.

There is some evidence that immune complex deposition may be enhanced by corticosteroids (13, 14), and it seems reasonable to assume that such deposition could also occur in the pancreas. Since it has been shown that immune complexes can be removed by plasmapheresis, this therapy would seem to be helpful in preventing this potentially dangerous effect of corticosteroids.

The dramatic clinical result, with full recovery within a few days, suggests that this combined (and possibly synergic) therapy may be a promising approach in the treatment of overwhelming SLE.

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