ACUTE PANCREATITIS – AN UNUSUAL PRESENTATION OF SYSTEMIC LUPUS PANCREATITIS AND A REVIEW OF THE LITERATURE

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INTRODUCTION

We report a patient with systemic lupus erythematosus who presented with acute pancreatitis confirmed by increased serum and urinary amylase with enlargement of the pancreas on ultrasound. The patient was not on any medication, and it appeared that the pancreatitis was due to the disease itself rather than being drug induced. Systemic lupus erythematosus (SLE) is a multisystem vasculitis frequently involving skin, joints and kidneys. Although the gastrointestinal tract is not infrequently involved, pancreatitis is a rare manifestation of SLE.

The vast majority of patients with pancreatitis complicating SLE have had longstanding multiorgan involvement and/or are on drug therapies with steroids, diuretics or immunosupressants. Only a small number of cases have been reported where pancreatitis was not related to drug treatment. Only six patients have been reported in the literature with an initial presentation of pancreatitis[1].

CASE REPORT

A 36-year-old woman was admitted with a three-day history of central abdominal pain radiating to the back. The patient was a non-smoker and drank four units of alcohol a week. Eleven years before, she had suffered a DVT complicated by multiple pulmonary emboli (confirmed by ventilation perfusion scan), for which she was treated with warfarin until 10 months prior to this admission. On examination, the heart and lungs were normal, there was no jaundice or lymphadenopathy. The abdomen was soft with a tender epigastrium but no organomegaly or peritonism.

Blood investigations were normal apart from a raised serum amylase (529). Urinary amylase was 1659 (normal <190). Liver function tests revealed an ALT 36, an alkaline phosphatase 148 and a GGT 86. Ultrasound of the abdomen revealed an enlarged pancreas but no stones in the biliary tree. Calcium and fasting lipids were within normal limits. The patient was treated conservatively and sent home with a plan for review in the outpatient clinic.

Two weeks later, the patient was readmitted with a history of slurred speech, weakness in her left arm, fever, vomiting and epigastric pain. On admission the temperature was 39°C, and the pulse rate 110/min. There were no abnormalities in the cardiorespiratory system and the abdomen was soft without any organomegaly or masses. Neurologically, the patient was dyspraxic in her left upper limb and had an expressive dysphasia. There was a mild right-sided upper motor neuron seventh cranial nerve paresis. Tendon reflexes were normal and planters downgoing. There were no signs of meningism.

Haemoglobin was 7.5 gm%, platelet count 73,000, blood film normal, urea, electrolytes, creatitine and amylase were normal. Liver function tests revealed ALT 50, alkaline phosphatase 1,091 and GGT 340. INR 1.6, KCCT >100 (ref range <35) and not corrected by normal plasma. Fibrinogen and fibrin degradation products were normal. The patient was started on cefotaxime and metronidazole to cover the possibility of cerebral abscess. CT brain scan was normal. CT scan of abdomen did not reveal any gallstones and the biliary tree was normal but there was an old haematoma in the right adrenal gland. The neurological symptoms and signs remitted but she had episodes of dysphasia which lasted for three days.

Further investigations revealed strongly positive lupus anticoagulant (confirmed by correction method and DRVVT method). Anticardiolipin antibodies (IgG) were 26 GPLU/ml (range up to 30). Autoimmune screen revealed antinuclear factor to be negative, but anti ds DNA was positive at 1:2,000. Blood, urine, throat swab cultures and viral titres were negative. Repeat liver function tests revealed persistently high ALT, GGT and alkaline phosphatase.

The patient was transfused with four units of packed red cells and her haemoglobin rose to 12 gm%. Aspirin was started (and not warfarin because of the adrenal haematoma) but the liver function tests worsened (alk phos 1,600 and GGT 381). Aspirin was stopped and prednisolone and warfarin were started. The temperature and the liver function tests returned to normal within one week of starting the patient on steroids.

Difficulty was experienced in reducing the dose of steroids and azathioprine was commenced. After the addition of azathioprine, her CRP became normal.

Currently, she is maintained on azathioprine 150 mg once a day and prednisolone 7.5 mg once a day and she is well.

DISCUSSION

Acute pancreatitis in SLE is rare. The commonest cause of pancreatitis in SLE is drug-related, notably diuretics and

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steroids. Necrotizing vasculitis has been documented within the pancreas at autopsy and most patients have clinical evidence of widespread vasculitis[2,3].

Retrospective review of hospitalised patients with SLE showed an incidence of pancreatitis in between 4.5% and 8.5% of patients[4,5]. Only 75 cases of SLE have been reported to have developed acute pancreatitis which was due to the disease itself[2,6-8]. In the last few years, many authors have recognised the association between antiphospholipid syndrome and vascular occlusion (both arterial and venous)[9-11]. It is most likely that pancreatitis may be a part of the catastrophic syndrome characterised by multiorgan dysfunction secondary to vascular occlusions[12]. Interestingly our patient presented with pancreatitis and was later proved to have SLE.

The role of steroids in patients with SLE and pancreatitis is interesting. It is well known that steroids cause[13,14] and are also a treatment for acute pancreatitis[15,16]. Not all patients on steroids develop pancreatitis, raising the suspicion that some amount of vascular compromise is important in its etiology. It is even more confusing to see that most patients with steroid-related pancreatitis settle down with increasing dose of steroids and reducing the dose may have adverse effects[17].

No exact data on mortality from pancreatitis in SLE are available. Reynolds *et al*[4] reported only one death out of 20 patients with pancreatitis and SLE who also had clinical evidence of concomitant cerebral, myocardial and renal involvement. Pollack *et al*[17] described three patients with SLE who died of pancreatitis, of whom one had arteritis, one had thrombus occluding the small arteries and arterioles of the pancreas and the other had pancreatitis associated with end stage renal failure.

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