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Cerebral Fat Embolism as a Rare Possible Complication of Traumatic Pancreatitis

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ABSTRACT

Context Neurological complications following an acute attack of pancreatitis have been described. They are mainly in the form of encephalopathy, retinopathy and rarely polyneuropathy. Cerebral infarction following an acute attack of pancreatitis is very rare.

Case report We report the case of a 25 year old male who developed multiple cerebral infarcts (both hemorrhagic and non-hemorrhagic) as a complication of acute pancreatitis.

Conclusion This is probably the first case report where a cerebral fat embolism during the course of acute pancreatitis has been hypothesized as a cause of cerebral infarction leading to hemiplegia.

INTRODUCTION

Acute severe pancreatitis is a serious condition which can result in both local and systemic complications. Systemic complications are usually referable to the gastrointestinal, cardiovascular, renal, hepatic and neurological systems and their occurrence adds to the morbidity and mortality. Neurological complications associated with acute pancreatitis are uncommon and are mainly in the form of encephalopathy, retinopathy and polyneuropathy. Hemiplegia following acute pancreatitis has not been reported earlier.

CASE REPORT

A 25 year old male, an occasional smoker and a social drinker had fallen off a motorcycle 7 days prior to hospitalization. He was examined at a local hospital and was discharged after first aid as there was no evidence of head injury or trauma to major organs. He presented at our hospital with complaints of acute pain in the upper abdomen and sitophobia 7 days after the accident. The pain was dull, aching and radiating to the back, and was aggravated upon eating. He had one episode of vomiting on the day of admission. On examination, he was febrile and had tachycardia. The abdomen was distended and there was mild tenderness in the epigastric area. A chest examination revealed signs of left pleural effusion. Fundus oculi examination and the other systemic examinations were unremarkable. Investigations revealed normal hemoglobin with elevated total counts and normal blood sugar, electrolytes, lipid profile, renal and liver functions. Serum amylase was 422 IU/L. Ultrasonography of the abdomen and chest revealed a bulky, hyperechoic pancreas with a few hypoechoic areas, ascites and left sided pleural effusion. Pleural and ascitic fluid amylase were 27,790 IU/L and 21,070 IU/L, respectively. A CT scan of the abdomen revealed grade E pancreatitis (Figures 1 and 2). An ERCP examination revealed pancreatic ductal disruption communicating with a cavity into which a nasopancreatic drain was placed. One week after hospitalization, he developed sudden

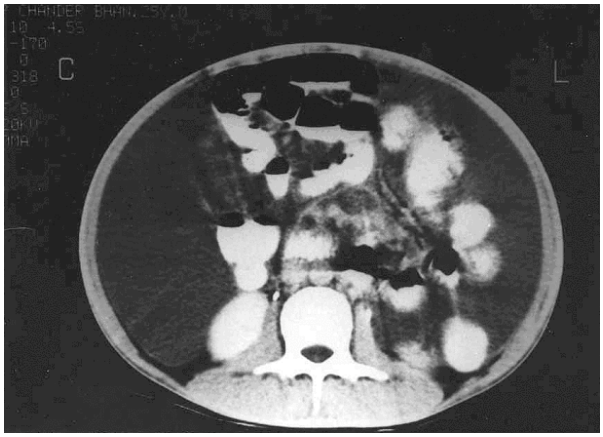

Figure 1. Abdominal CT scan showing acute pancreatitis (Grade E).

onset hemiplegia and right supranuclear facial nerve palsy of the right side. There was no loss of consciousness. A repeat fundus oculi examination was normal. A CT scan of the brain revealed multiple infarcts (hemorrhagic and non-hemorrhagic) in the left middle cerebral artery territory without evidence of skull fracture or subdural haemorrhage (Figure 3). Echocardiography, carotid doppler study, coagulation profile (prothrombin time, partial thrombin index, activated partial thromboplastin time), platelet count, venereal disease laboratory test, and workup for disseminated intravascular coagulation were all normal. He was managed conservatively. Surgery for pancreatic necrosectomy was planned but was refused by the patient. A feeding jejunostomy was done and he was treated with antibiotics, analgesics, jejunostomy feeds and aggressive physiotherapy.

After 3 weeks of therapy, his pain decreased and he started improving neurologically. On follow-up, there were no further episodes of pain; he continued to improve neurologically and could move without support. The tests for anti-phospholipid antibodies, Protein C and S levels and antithrombin III levels were normal.

DISCUSSION

Trauma is a rare cause of acute pancreatitis. Cholelithiasis, ethanol ingestion and idiopathic pancreatitis constitute 90% of all causes of acute pancreatitis. Our patient had traumatic pancreatitis as was evident from the history and was proven on ERCP which disclosed a ductal disruption.

Neurological complications following acute pancreatitis are uncommon. They are usually in the form of encephalopathy, polyneuropathy and/or visual loss due to retinopathy or cerebral infarction. All these complications have been more commonly reported in acute alcoholic pancreatitis.

Encephalopathy, the commonest neurological abnormality, is multifactorial and is generally related to abnormal metabolic disturbances [1, 2, 3]. Polyneuropathy, rarely described with acute pancreatitis, is either due to metabolic

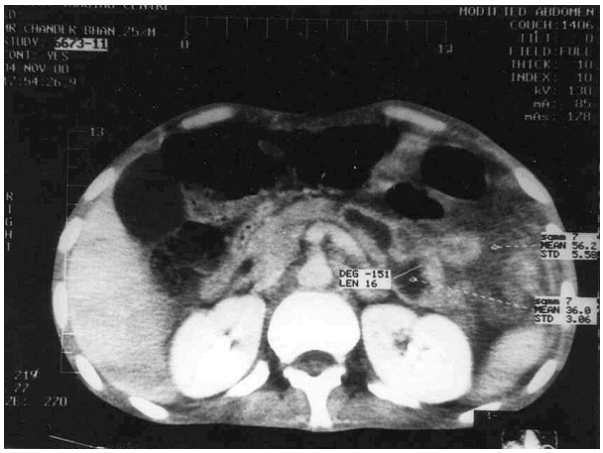

Figure 2. Abdominal CT scan showing acute pancreatitis with focal hypo densities.

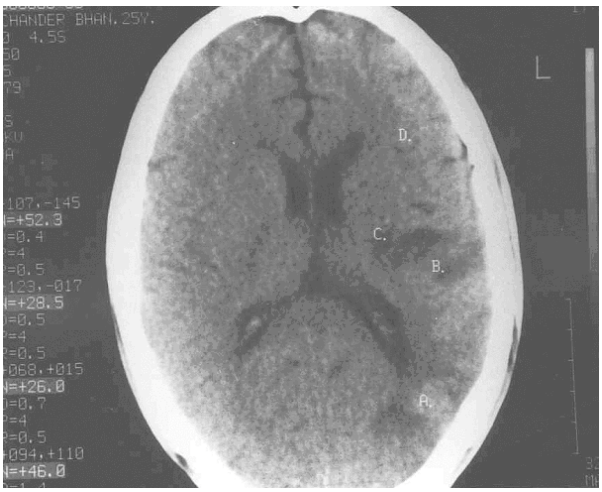

Figure 3. CT scan of the brain showing focal hypo densities suggestive of cerebral infarction.

causes, i.e. deficiency of vitamins (vitamin E) in diet during acute attack, or critical illness polyneuropathy seen in patients with severe pancreatitis [4].Loss of vision, described with alcoholic pancreatitis, is mostly due to retinopathy [5], rarely due to cortical infarction [6] and is attributed to non-traumatic cerebral embolism (cholesterol/fat embolism). Hemiplegia due to cerebral infarction, resulting from a fat embolism, after an attack of acute pancreatitis has not been reported earlier.

Cerebral fat embolism has been reported as a cause of cerebral infarction and multiple brain hemorrhages in autopsy studies involving acute pancreatitis [1, 2]. In one patient having a cerebral infarction and presenting with visual loss following acute pancreatitis, Sudan III positive material suggestive of a fat embolism was demonstrated on cerebro-spinal fluid examination [6].

Pathophysiologically, chylomicrons and VLDL have been shown to develop calcium-dependent agglutination by C-reactive proteins in acute pancreatitis, which could result in vascular occlusion and resultant infarction [4]. In our patient, a similar mechanism could have been responsible for vascular occlusion and resultant infarction.

This is probably the first case report, where a cerebral fat embolism has been suggested as a cause of cerebral infarction leading to hemiplegia. Our patient had acute traumatic pancreatitis. The absence of thrombophilia, a hypercoagulable state and any lesions in the heart or carotids, makes cholesterol embolism secondary to pancreatitis as the most plausible mechanism for this unusual complication in a young patient.

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