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Case Report

Acute pancreatitis complicated by encephalopathy

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ABSTRACT

Pancreatic encephalopathy is a frequently under-diagnosed and rare complication of acute pancreatitis. It denotes the occurrence of neuropsychiatric abnormalities in the setting of acute pancreatic inflammation, and presents with neurological symptoms that may persist even after the resolution of all metabolic parameters. Here we present the case of a 42 year old male patient presenting with altered sensorium and focal neurological deficit during the course of acute pancreatitis. The patient was treated conservatively, and later improved with supportive care.

Keywords: Acute Pancreatitis, Encephalopathy, Focal Neurological deficit, Pancreatic inflammation

INTRODUCTION

Pancreatic encephalopathy is a frequently under-diagnosed and rare complication of acute pancreatitis. It denotes the occurrence of neuropsychiatric abnormalities in the setting of acute pancreatic inflammation, and presents with neurological symptoms that may persist even after the resolution of all metabolic parameters.[1,2] It was first described by Rothermich and Von Haam in 1941, and the pathogenesis first hypothesized by Vogel in 1950. The diagnosis is difficult in early stages, and so is the differentiation from Wernicke's encephalopathy. Some patients develop encephalopathy in spite of successful treatment, and the mortality rate can reach as high as 67%.[3] Although pancreatic encephalopathy can develop in any case of acute pancreatitis, the incidence is much higher in cases of severe complicated/ acute necrotizing pancreatitis.

CASE REPORT

A 42 year old male diabetic presented to the medicine emergency with three days history of abdominal pain and vomiting, associated with altered sensorium for one day prior to admission. He had been on metformin for the past 7 years, and there was no complaint of blurred vision, tingling sensation in any limb or decreased urine output. There was no history of fever, abdominal distension, bleeding manifestations, hypertension or tuberculosis. No significant history of alcohol abuse, smoking or illicit drug use could be elicited.

On examination, the patient was altered. His blood pressure was 130/80 mm Hg and pulse was 78 beats per minute. There was no pallor, edema, cyanosis, clubbing or lymphadenopathy. CNS examination revealed a Glasgow Coma score of 9/15. Pupils were mid-dilated and sluggishly reacting. Reflexes were normal and the Bilateral Plantar Response was Extensor/ Babinski's Positive. Rigidity was noted in both the upper and lower limbs. Power and Sensory System could not be assessed in view of altered sensorium. Abdominal examination revealed a tender abdomen with mild hepatomegaly. Respiratory and cardiac examinations were unremarkable.

Routine blood counts revealed elevated liver enzymes with SGOT (AST) and SGPT (ALT) being 774 U/L and 3990 U/L respectively, on admission. The total bilirubin was 6.1 mg/dl, with the direct fraction being 1.8 mg/dl. Alkaline phosphatase was 185 IU/L. A Provisional diagnosis of Fulminant Hepatitis was made and

immediate treatment was started with IV fluids and necessary antibiotic cover with I.V Ceftriaxone and Metronidazole. Tests for HIV, HBV and HCV done by ELISA were negative and IgM antibodies to HAV and HEV were not found. A urine toxicology screen obtained was negative and an emergency Ultrasound of the whole abdomen was suggestive of an enlarged liver. A Lumbar puncture and CSF analysis was done, which revealed an acellular picture with sugar and protein levels of 85 mg/dl and 34mg/dl respectively. Serum Ceruloplasmin and 24 hour Urinary copper levels were normal.

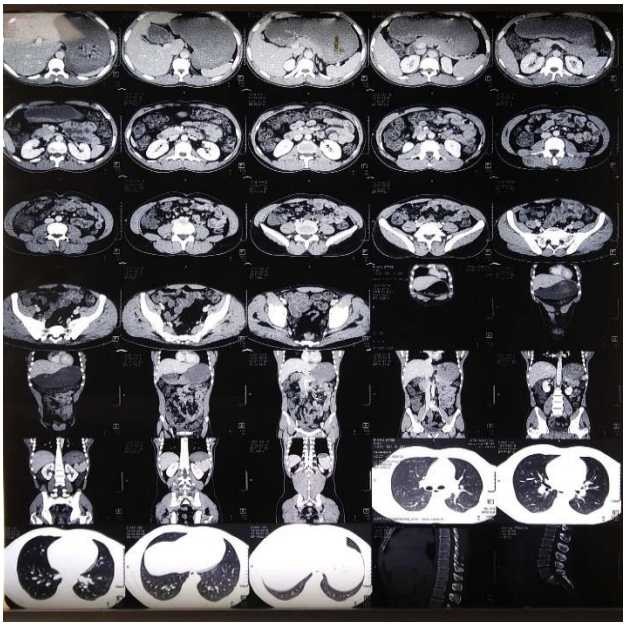

Figure 1: CECT abdomen acute interstitial edematous pancreatitis involving the pancreatic tail.

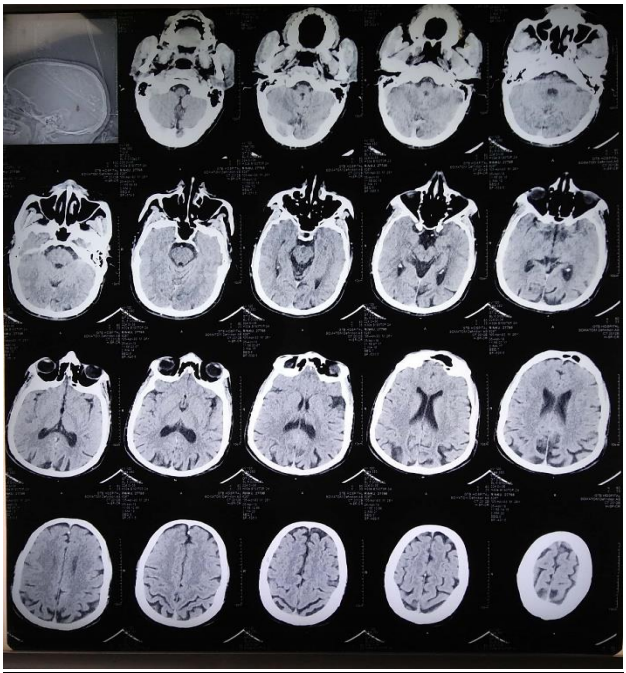

Figure 2: NCCT brain a normal scan.

On the third day of admission, the patient's serum amylase and lipase levels were 280 U/L and 400 U/L respectively which increased to 396 U/L and 670 U/L the next day. An emergency CECT Abdomen revealed an Acute Early Interstitial Edematous Pancreatitis, involving the pancreatic tail (Figure 1). NCCT Head was not suggestive of any significant abnormality (Figure 2). Treatment was supplemented with Thiamine 300mg stat as well as on maintenance with dextrose, along with Intravenous fluids. The patient's sensorium gradually improved and the patient gradually became fully conscious and oriented on the fifth day of admission.

DISCUSSION

Pancreatic Encephalopathy is a rare presentation in patients of acute pancreatitis. It usually occurs in the early stages of the disease and has a mortality rate of 48.5% to 67%.[4,5] The neurological complications in the restorative or later stages of severe and simple acute pancreatitis are usually secondary to long fasting, multiple vomiting episodes and Total Parenteral Nutrition (TPN) without any thiamine supplementation, collectively termed as Wernicke's encephalopathy.[4,6] Studies have shown that pancreatic encephalopathy is poorly recognized by most physicians, and delay in recognition and necessary supportive care can prove to be fatal in these cases.[6]

The pathogenesis of pancreatic encephalopathy has been poorly understood. During severe acute pancreatitis, high levels of pancreatic enzymes including trypsin, elastin, lipase and phospholipase A2 (PLA2) enter the circulation and alter the permeability of the Blood Brain

Barrier. PLA2, with potent neurotropism, can directly act on the phospholipid layers of brain cells, causing neuronal edema, focal hemorrhagic necrosis, meningeal inflammation with severe demyelinating changes in axons and secondary cell dysmetabolism of neurons, leading to various neuropsychiatric symptoms.[2,7,8] The increased blood-brain barrier permeability can also cause sensitized blood T-cells to enter the brain tissue contributing to demyelination.[7] These patients have also been found to have intracerebral capillary engorgement with cell infiltration and broadened intercellular spaces in the brain parenchyma.[2]

During severe acute pancreatitis, hypoxemia and intravascular fluid depletion can activate the renin angiotensin system, which also plays an important role in the occurrence and progression of pancreatic encephalopathy.[9-11] Additionally, Pro-inflammatory cytokines such as TNF-a, IL-1b, and IL-6 increase the permeability of the blood-brain barrier by damaging it.[12] In addition to these factors, reduced myocardial pumping due to the release of myocardial depressant factor and deterioration of circulatory capacity due to vomiting and third spacing of fluid leads to cerebral ischemia and hypoxia.[13,14] Encephalopathy complicating pancreatitis

may also occur due to hypoxia secondary to pulmonary fat embolism, cerebral fat embolism, disseminated intravascular coagulation or hyperosmolality.[15] Brain imaging obtained during the acute phase of pancreatic encephalopathy may essentially be normal.[1]

The early stage of pancreatic encephalopathy is seen within 15 days (usually 2-5 days) after onset of symptoms of acute pancreatitis.[7] The symptoms include confusion, dysarthria, anxiety, muscular aches, short lucid periods and a cyclic progression with remission and relapses. Examination findings include rigidity of all four limbs, hyper-reflexia and asterixis with an occasionally positive Babinski's sign.[2,3] Though the encephalopathy often resolves without leaving any focal deficits, an important long term manifestation of this condition might be cognitive impairment which is demonstrable on the MRI as diffuse cortical and subcortical atrophy. A close differential diagnosis of pancreatic encephalopathy is Wernicke's encephalopathy, which may occur late in the course of acute pancreatitis as a result of prolonged fasting and thiamine depletion.[6]

Treatment of pancreatic encephalopathy mainly comprises of proper management of severe acute pancreatitis and its complications. In the early stages of disease, it is necessary to control pancreatic secretion and drainage, correct fluid and electrolyte imbalance as well as hypo-proteinemia, and provide sufficient caloric support.[2] There is no specific treatment for this condition except supportive care and thiamine.[1] With proper treatment, recovery is uneventful among young patients without co-morbidities but may have sequelae such as cerebral infarction in the elderly, especially above 60 years of age.

Pancreatic encephalopathy carries a high mortality among high risk and untreated patients; the most common causes of death secondary to encephalopathy being shock, keto-acidosis and multi-organ dysfunction syndrome.[16]

CONCLUSION

Pancreatic encephalopathy is a rarely reported but devastating complication in patients having Acute Pancreatitis. The patients may present with altered behaviour and neurological deficits either early or late in the progression of the disease, and even during resolution. Therefore it becomes necessary to anticipate this phenomenon in all complicated cases of pancreatitis, and provide early and adequate conservative management with fluid support and judicious antibiotic cover, if and as required. The treatment of acute pancreatitis is primarily non-operative except in cases of severe acute biliary pancreatitis with resultant complications such as fluid sequestration and phlegmon formation, in which effective and timely drainage leads to a low mortality rate in the patients who receive surgery.

There is also sufficient evidence which suggests that pancreatic encephalopathy can be prevented by administration of Vitamin B1/Thiamine as well as good nutritional replenishment in the early stages of the disease.

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