Clinical History:

A 35-year-old male alcoholic presented with severe upper abdominal pain and vomiting of 2 weeks duration. A CT abdomen was performed. 3 days later he developed nystagmus, ophthalmoplegia and global confusion and was referred for MRI brain.

Imaging Findings:

CT abdomen revealed pancreatic parenchymal necrosis, extensive peripancreatic inflammation, peripancreatic fluid and left pleural effusion in keeping with acute necrotising pancreatitis.

MRI brain after the onset of neurological symptoms revealed areas of T2W and FLAIR hyperintense signal involving the medial aspect of the thalami and the mamillary bodies in a bilaterally symmetrical fashion. Abnormal hyperintense signal was also noted in the peri-aqueductal region. Diffusion weighted imaging revealed bilateral symmetrical areas of restricted diffusion in the medial aspect of bilateral thalami. No other areas of restricted were discernible. The characteristic imaging findings and history helped in confirming a diagnosis of Wernicke’s encephalopathy.

Discussion:

Wernicke’s encephalopathy (WE) is caused by deficiency of vitamin B1 or thiamine. This acute neurological disorder was previously diagnosed only based on the clinical triad of ocular signs, ataxia and altered consciousness [1, 2, 3]. Many cases were however clinically underdiagnosed as the classic triad of symptoms was seen in only about 16 to 38 % of cases [1].

Thiamine is involved in glucose metabolism and is required for maintaining osmotic gradients across cell membrane. Thiamine related metabolism occurs predominantly in the periventricular region of the brain and these areas are preferentially affected in WE [1].

The daily requirement of thiamine is about 1-2 mg and the body reserves are usually depleted within 4 to 6 weeks without regular thiamine intake [1]. Dietary deficiency, alcohol abuse, prolonged vomiting, HIV infection, malignancy and chemotherapy have been implicated as causes for thiamine deficiency [1, 4]. Acute pancreatitis (AP) is associated with a number of factors favouring thiamine deficiency. Alcohol abuse is a common aetiological factor for
WE and AP. Alcoholism is associated with malnourishment, hepatic dysfunction, impaired absorption of thiamine at the mucosal level and increased thiamine metabolism [1]. Prolonged vomiting and parenteral nutrition without thiamine supplementation appear to be responsible for WE in non-alcoholic pancreatitis [4]. Imaging in WE classically reveals symmetrical signal abnormalities in the medial thalami, periaqueductal region, tectal plate and mamillary bodies. Atypical findings include signal abnormalities in cerebellum, cranial nerve nuclei, caudate nucleus, splenium and cerebral cortex. The atypical features are however usually seen in association with other typical findings aiding diagnosis [1]. The signal changes in the medial thalami may be subtle and it has been postulated that diffusion weighted imaging may better depict these lesions in comparison to conventional MRI [2]. Restricted diffusion in the medial thalami was well seen in our case. The presence of caudate nucleus and cortical signal abnormalities has been associated with an increased risk of coma [1, 3].

Treatment of WE involves thiamine administration and the prognosis depends on the stage at which supplementation was initiated. Even treated cases of WE may be associated with residual gait abnormalities, tremors and cognitive difficulties [2]. It is thus essential to regard WE as a possibility in all cases of acute pancreatitis. Some authors suggest initiating thiamine supplementation in cases of acute pancreatitis showing neurological symptoms, even when imaging studies are negative, to avoid the severe complications of this potentially treatable disorder [4].

**Differential Diagnosis List:** Wernicke's encephalopathy in a case of acute pancreatitis., Venous infarction, Viral encephalitis, Creutzfeldt-Jakob disease

**Final Diagnosis:** Wernicke's encephalopathy in a case of acute pancreatitis.

**References:**


Description: Features of pancreatic parenchymal necrosis are seen with extensive peri-pancreatic inflammatory changes and fluid. The stomach appears displaced anteriorly. **Origin:** Department of Radiology and Imaging Sciences, Billroth Hospitals, Chennai, India.
**Description:** Sagittal view reveals pancreatic parenchymal necrosis, mesenteric fat stranding and significant peripancreatic fluid extending into the left anterior pararenal space. Left pleural effusion is also seen. **Origin:** Department of Radiology and Imaging Sciences, Billroth Hospitals, Chennai, India.
**Description:** Diffusion weighted image shows symmetrical areas of restricted diffusion involving the medial aspect of bilateral thalami. **Origin:** Department of Radiology and Imaging Sciences, Billroth Hospitals, Chennai, India.
**Description:** T2 weighted image reveals subtle hyperintense signal involving the medial aspect of bilateral thalami symmetrically. **Origin:** Department of Radiology and Imaging Sciences, Billroth Hospitals, Chennai, India.
Description: FLAIR axial image reveals hyperintense signal in the peri-aqueductal region and in the mamillary bodies. Origin: Department of Radiology and Imaging Sciences, Billroth Hospitals, Chennai, India.
Description: FLAIR axial image at a higher level reveals bilateral symmetrical areas of hyperintense signal involving the medial aspect of thalami. Origin: Department of Radiology and Imaging Sciences, Billroth Hospitals, Chennai, India.
Description: The areas of hyperintense signal involving the medial aspect of bilateral thalami are well seen. Origin: Department of Radiology and Imaging Sciences, Billroth Hospitals, Chennai, India.
**Description:** Hyperintense signal is seen in the peri-aqueductal region of the midbrain. **Origin:** Department of Radiology and Imaging Sciences, Billroth Hospitals, Chennai, India.