Wernicke encephalopathy in a patient with severe acute pancreatitis

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To the Editor,

36-years-old male patient with history of biliary severe acute pancreatitis (SAP) before 4 months. Endoscopic retrograde cholangiopancreatography (ERCP) was done for the patient at that time and common bile duct stones removed and the patient was discharged. He came before 1 month complaining of fever, abdominal pain and swelling. Physical examination revealed abdominal tenderness and ascites. Chest auscultation showed bilateral decreased air entry. Glucose : 177 mg/dL, AST : 64 U/L, ALT : 114 U/L, LDH : 986 U/L, total/conjugated bilirubin : 2.8 mg/dL/ 1.6 mg/dL, amylase : 1552 U/L, lipase : 1238 U/L, CRP : 36.2 mg/dl, WBC : 21,550 mm^3, hematocrit : 36% and platelets : 366,000 mm^3. Computed Tomography showed acute pancreatitis with areas of necrosis at the level of the head and tail of pancreas. There was a 18 × 5 cm cystic degeneration in the body of the pancreas with air bubbles observed inside it (Fig. 1). The patient was hydrated and meropenem started. The patient then after started to complain of drowsiness. He was transferred to the intensive care unit and intubated. Cranial magnetic resonance imaging (MRI) showed obvious limitation of diffusion in the anteromedial part of both thalamui, mamillary bodies, the left caudate nucleus, putamen and in the left precentral gyrus (Fig. 2). These findings were diagnosed with Wernicke's encephalopathy (WE). The patient's blood and abdominal fluid cultures showed growth of klebsiella pneumonia and enterococci. Vancomycin were added in addition to meropenem. Electrolyte abnormalities were corrected and thiamine treatment started. A stent was inserted in through the necrotic tissue inside the

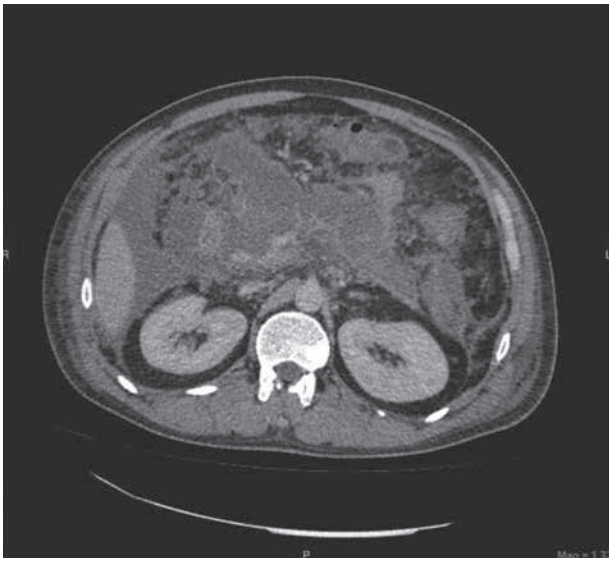

Fig. 1. — Acute pancreatitis with areas of necrosis at the level of the head and tail of pancreas. There is a 18 × 5 cm cystic degeneration in the body of the pancreas with liquid and air bubbles observed inside it. There ismoderate amount of free fluid within the abdomen.

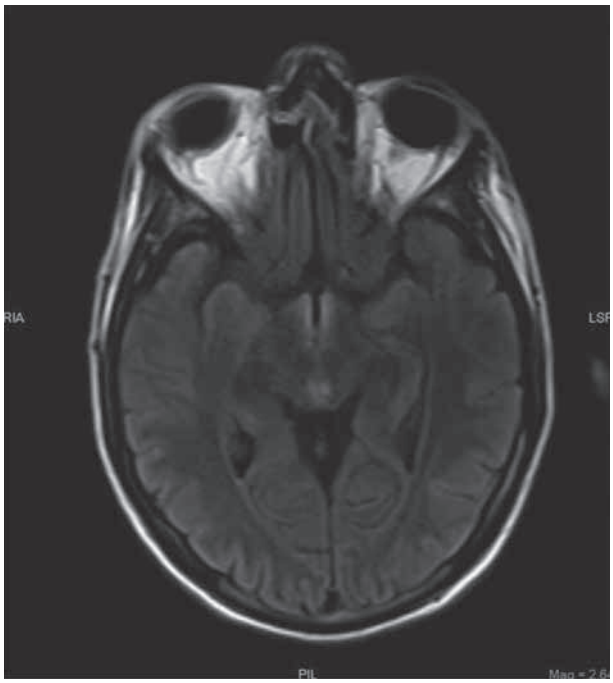

Fig. 2. — Limitation of diffusion in the anteromedial part of both thalamui, mamillary bodies, the left caudate nucleus, putamen and in the left precentralgyrus.

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duodenum. In a second session, necrotectomy was done and two abdominal drains placed. However patient who stay two months in the intensive care unit was died.

Pancreatic encephalopathy (PE) is the occurrence of neuropsychiatric abnormalities in setting of SAP, is difficult to diagnose and treat and it has a high mortality (1). Sun *et al*. reported an incidence of 0.7% of WE in patients with SAP (2). Many factors were involved in the pathogenesis of PE in SAP. Pancreatin activation, excessive release of cytokines and oxygen free radicals, hemodynamic disturbance, hypoxemia, bacterial infection, electrolyte imbalance and vitamin B1 deficiency participated in the development of PE in SAP (2,3).

WE is a neuropsychiatric condition associated with thiamine deficiency, presenting as a combination of acute onset ataxia, ophthalmoplegia and confusion (1). MRI is the preferred imaging method employed for the diagnosis of WE and findings commonly include affectation of mammillary bodies, thalamus, periventricular areas (3). Treatment of WE consists of prompt and intravenous thiamine administration (4).

In conclusion there are several factors which may cause encephalopathy in patients with SAP. Although the correction of underlying factors, we should consider that the disease can be mortal.

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