Wernicke's encephalopathy: a complication of acute pancreatitis?

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INTRODUCTION

Wernicke's encephalopathy is a common and preventable disorder. It is a result of diencephalic and mesencephalic dysfunction of central gray matter surrounding the third and fourth ventricles secondary to thiamine deficiency. Patients who are fasting, receiving parenteral nutrition, recovering from gastrointestinal surgery, being fed after a period of starvation, undergoing haemodialysis or suffering from advanced cancer are particularly susceptible to this disorder. There are many reports of Wernicke's encephalopathy occurring in alcoholics but it is rare in non-alcoholics and within the literature there is no definite conclusion as to whether or not Wernicke's encephalopathy is a complication of acute pancreatitis. This article reports a case of Wernicke's encephalopathy in association with acute pancreatitis in a non-alcoholic patient.

DISCUSSION

This article describes a case of Wernicke's encephalopathy in association with acute pancreatitis in a non-alcoholic patient. To the best of the authors' knowledge, Wernicke's encephalopathy complicating acute pancreatitis in a non-alcoholic patient is rare, the only other report being from Winslet et al (1990).

CASE REPORT

A 60-year-old woman was admitted to the authors' hospital having suffered from upper abdominal pain for 4 days. The pain was steady in the mid-epigastrium with a band-like radiation to the back. There was no previous history of alcohol abuse. Blood pressure was 120/80 mmHg, her pulse was 82 beats per minute, and her axillary temperature was 37.8°C. On examination, she was found to be acutely ill, jaundiced and to have epigastric tenderness. Decreased bowel sounds were heard on auscultation of the abdomen.

Complete blood count on admission showed a haemoglobin of 14.1 g/dl, white blood cell count 13 500/mm^3, platelet count 136 000/mm^3, and erythrocyte sedimentation rate 51 mm/hr. Serum chemistries were within normal ranges with the exceptions of: aspartate aminotransferase 45 IU/litre (normal 5–40 IU/litre), protein 5.5 g/dl (normal 6.7–8.3 g/dl), albumin 3.4 g/dl (normal 3.5–5.3 g/dl), total bilirubin 1.5 mg/dl (normal 0.2–1.0 mg/dl), direct bilirubin 0.78 mg/dl (normal <0.4 mg/dl), total calcium 7.7 mg/dl (normal 8.4–10.2 mg/dl), glucose 137 mg/dl (normal 76–110 mg/dl), amylase 465 U/litre (normal <100 U/litre), lipase 1087 U/litre (normal <190 U/litre).

Abdominal plain film showed localized ileus of a segment of small intestine ('sentinel loop'). Abdominal ultrasonography showed a diffusely enlarged, hypoechoic pancreas and peripancreatic fluid collection but no gallstones. Computed tomography (CT) scan of the abdomen showed diffuse enlargement of the pancreas and necrosis of the body. She was treated with supportive care including pain control, intravenous fluids and nothing by mouth. Ceftriaxone, amikacin and metronidazole were given intravenously for 10 days. A proteinase inhibitor (gabexate mesilate) and a somatostatin analogue (octreotide) were given. On day 22, abdominal pain and tenderness subsided and serum amylase and lipase reduced to 305 U/litre and 408 U/litre respectively. Oral feeding was initiated with a liquid diet. On day 35, apathy, impaired awareness, disorientation and ataxia occured. On neurological examination, there was horizontal nystagmus. The upper and lower limbs were hypotonic with absent ankle jerks. Complete blood count, urea and electrolyte, and blood sugar estimation were normal. CT scan of the brain showed no mass lesion. A differential diagnosis of a brain lesion or Wernicke's encephalopathy was made by the neurologist, and the patient was treated with intravenously with thiamine 25 mg twice daily for 20 days. The horizontal nystagmus resolved at 48 hours and after 14 days there were no neurological signs. The patient was discharged fully recovered on day 60.

Wernicke's encephalopathy is caused by an underlying deficiency of thiamine, an essential coenzyme in intermediate carbohydrate metabolism, and is classically associated with chronic alcoholism. Although the mechanism by which the neurological disorder is produced is unknown, proposed mechanisms of Wernicke's encephalopathy are altered cerebral energy metabolism resulting from decreases in transketolase, pyruvate and acetylcholine; diminished nerve-impulse transmission at synapses; and impaired DNA synthesis (Schenker et al, 1980).

Wernicke's encephalopathy is characterized by the triad of ocular abnormalities, ataxia and a global confusional state (*Table 1*). Symptoms that may occur in addition to or in place of the classic triad are hypothermia (Donnan and Seeman, 1980) resulting from involvement of the temperature-regulating centre, and hypotension (Ackerman, 1974) caused by a defect in efferent sympathetic outflow and decreased peripheral resistance. Coma is a particularly important feature for early recognition of Wernicke's encephalopathy (Torvik et al, 1982), because it may be the sole manifestation.

Wernicke's encephalopathy is poorly recognized even when features of the classic triad are present (Harper, 1983), and remains a clinical diagnosis because there are no characteristic abnormalities in diagnostic studies of CSF, nuclide and computed tomographic brain scans, or electroencephalograms and evoked poten-

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tials (Handler and Perkins, 1982). Although thiamine deficiency may be apparent following an enzyme assay, such as erythrocyte transketolase activity or the effect of transketolase activity on exogenous thiamine pyrophosphate, intravenous thiamine should be given without delay, to avoid potential morbidity and mortality, if a diagnosis of Wernicke's encephalopathy is suspected.

Although the adult daily requirement seldom exceeds 2 mg, limited body storage (approximately 30 mg) means that inadequate intake can produce symptomatic deficiency in only a few weeks or months. Chronic consumption of large amounts of alcohol in conjunction with marginal thiamine intake in the diet may result in the development of Wernicke's encephalopathy (*Table 2*). Wernicke's encephalopathy may also be seen in association with carbohydrate loading in the presence of a marginal thiamine store. In this patient this occured after 13 days of feeding on a liquid diet, following the previous 22 days where she had been given nothing by mouth. The patient was maintained on a type of liquid diet made of polished rice which is deficient in thiamine. The development of Wernicke's encephalopathy may be iatrogenic in some cases.

TABLE 1.
Clinical features of Wernicke's encephalopathy

Triad	Ophthalmoplegia, ataxia, global confusional state
Ocular abnormalities	Horizontal nystagmus, bilateral rectus palsy, conjugate gaze palsies
Ataxia	Wide-based ataxic gait, lower limb intention tremor, dysmetria
Mentation	Inattentiveness, abulia, impaired memory, lethargy, coma
Autonomous nervous system	Tachycardia, postural hypotension

TABLE 2.
Susceptibility to Wernicke's encephalopathy

Most common	Alcoholics
Others	Malnutrition as a result of hyperemesis, fasting, starvation, haemodialysis, gastric plication, advanced cancer, AIDS
	Prolonged parenteral nutrition

CONCLUSION

The diagnosis of Wernicke's encephalopathy can be missed if a detailed history is not elicited. Consideration of Wernicke's encephalopathy in high-risk groups will prevent this disorder and lead to the earlier detection and treatment of this reversible but nonetheless potentially fatal condition. HM

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