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**CLINICAL NOTE**

# Wernicke's encephalopathy after cephalic pancreaticoduodenectomy

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## ABSTRACT

Wernicke's encephalopathy is an acute neurological disorder resulting from thiamine deficiency. We report a case in a young patient who underwent a cephalic duodenopancreatectomy with a bleeding duodenal ulcer refractory to endoscopic and surgical treatment, requiring total parenteral nutrition, without thiamine supplementation.

***Key words***: Wernicke's encephalopathy. Cephalic duodenopancreatectomy. Gastrointestinal Hemorrhage. Peptic ulcer hemorrhage.

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## INTRODUCTION

Wernicke's encephalopathy is an acute neurological disorder resulting from thiamine deficiency. The main cause is alcohol abuse. Among other causes described in literature, we would be able to find chronic dietetic deficiency, as in total parenteral nutrition without thiamine addition, or increased nutrients requirements as in septic shock.

We report a case in a 27 year old male, occasional drinker, who developed the disease after undergoing multiple surgical operations due to a bleeding duodenal ulcer, and finally required a cephalic duodenopancreatectomy.

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## CLINICAL CASE

A 27 years old male with history of upper gastrointestinal bleeding three years ago, due to a gastric ulcer caused by non-steroidal antiinflamatory drugs use. The patient was transferred to our hospital's intensive care unit from a regional hospital, because of hemodynamic instability caused by an upper gastrointestinal bleeding. During the upper endoscopy we appreciated the presence of bleeding from duodenum, treated by clips placement and epinephrine injection. Twenty-four hours later the patient suffered syncope and presented blood within the nasogastric tube; therefore, we decided to carry out an emergency surgical operation. During the operation an upper gastrointestinal bleeding caused by duodenal ulcer penetrating the pancreas was evidenced, so we decided to perform a longitudinal pyloromyotomy, ulcer over sewing after clips withdrawal, achieving hemostasis and adding a bilateral trunk vagotomy, Heineke-Mikulicz pyloroplasty and omentoplasty. Twenty-four hours later, the patient's hemoglobin level dropped 4 g/dL and presented tachycardia; so, we decided to reoperate performing an endoscopic exploration through the pylorus, finding active bleeding. The pyloric artery was ligated and a new pyloroplasty with omentoplasty was performed. Forty eight hours later, a drop in hemoglobin level, hypotension and tachycardia, forced a new emergency operation. Findings were a big gastric clot and a bleeding point in the posterior pyloric aspect next to the pyloric artery ligature, and hemostasis was achieved after haemostatic stitches.

During the postoperative course, the patient developed a peripancreatic collection, requiring percutaneous drainage, and a biliary fistulae treated by conservative management. The patient also presented an acute respiratory distress syndrome with satisfactory outcome. Twelve days after last operation was performed, a new bleeding episode with hemodinamic instability, pushed the patient through an emergency operation. New findings were a gallbladder perforation due to ulcer's decubitus, and severe bleeding orig-

inated in the upper duodenal knee. We performed an antrectomy and resection of the first portion of duodenum. Hemostasis of the pancreas penetrating ulcus was accomplished. We also peformed colecistectomy and intraoperative cholangiography obtaining normal results. Roux-en-Y reconstruction was done.

One week later a cephalic duodenopancreatectomy was performed leaving a tutor within the pacreatoduodenal anastomosis. After this surgical operation, the patient's hemodinamic status improved and bleeding was finally controlled.

During the postoperative course the patient suffered septic shock caused by a nosocomial pneumonia treated with broad spectrum antibiotics achieving a satisfactory response. Later the patient developed spatial and temporal disorientation, ataxia with horizontal nystagmus and upper left limb weakness, requiring endotracheal intubation and mechanical ventilation. There were no findings in the two cranial CT scans so we performed a brain MRI showing deviation in signal intensity from encephalic trunk, mainly on the fourth ventricle floor, the central protuberance area, periaqueductal gray matter, paramedian regions of the cerebral peduncles, third ventricle walls (medial region of both thalami), mammillary tubercles, bilateral and symmetrical involvement of the confluence of internal and external white capsule, some frontal cortical and subcortical areas compatible with Wernicke's encephalopathy (Fig.1A).

Thiamine treatment was initiated, achieving a significant improvement in neurological symptoms and lesions found on control brain MRI (Fig.1B). Shortly after starting treatment, blood thiamine value reached 2 mg/dL (2.0 to 7.5 mg/dL).

The patient was discharged, after 60 days in the intensive care unit, being hemodynamically stable, with adequate digestive tolerance and resolving pneumonia. Definitive discharge from our hospital took place 27 days later.

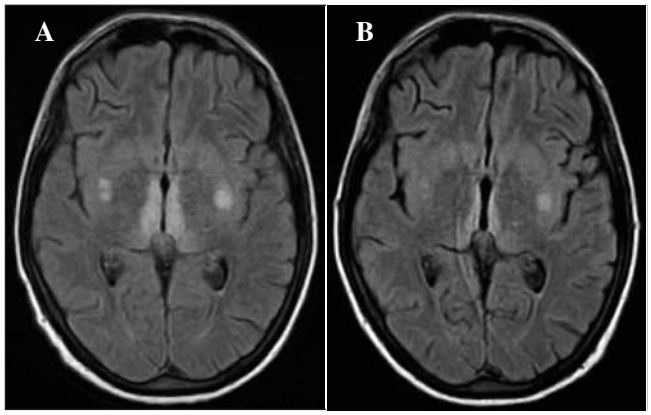


Fig. 1.

## DISCUSSION

Wernicke's encephalopathy is an acute neurological disorder resulting from thiamine deficiency. Thiamine is a water-soluble vitamine that is absorved in the duodenum by an active mechanism and breaking through the blood-brain barrier also by active and passive mechanisms (1). The main sources of thiamine are yeast, pork, vegetables, beef, grains and nuts. It is deposited in muscle, heart, liver, kidneys and brain, although its most important storage site is muscle, where it reaches levels of 30 mg (1). Its biological half-life is 9 to 18 days.

The main cause of its lack is alcohol abuse although is not the only one. Among other causes, can be highlighted the chronic dietary deficiency (unbalanced diet or parenteral nutrition without thiamine), bad absorption or low intake (celiac disease, pyloric obstruction, recurrent vomiting or gravidic hyperemesis), excessive intake of carbohydrates in relation to the supply of thiamine or a greater need for nutrients (growth, exercise, pregnancy or infection) (1,2).

The first author who described this syndrome was Carl Werkicke in 1881. Classic triad consists of abnormal eye movements, ataxia and mental status changes (only 20% of patients present with the triad) (3,4). The most frequent situation is to find a patient deeply disoriented, unresponsive, and, if untreated, can progress to coma and even death. The ophthalmoplegy is a horizontal nystagmus on lateral gaze, lateral rectus palsy, conjugate gaze palsy and rarely ptosis. Ataxia is postural and affects gait (5). In our case there was a clinical setting of disorientation in time and space, accompanied by ataxia, horizontal nystagmus and left upper limb weakness. Diagnosis is mainly clinical and can be done if the patient has at least two of the following four signs: a) dietary deficiencies; b) ocular disorders c) cerebellar dysfunction; and d) mental manifestations or memory impairment (6).

Within the complementary explorations available to achieve the diagnosis we can find the levels of thiamine in blood and urine or the MRI. Within the lab test the determination of thiamine concentration in blood and the measurement of transketolase activity of red blood cells can confirm the diagnosis.

MRI is considered the most important test for early diagnosis of the syndrome, prognosis and detection of atypical presentations (sensibility 53 % and specificity 93%) (3,7). Findings in our case consisted of deviation in signal intensity from encephalic trunk, mainly on the fourth ventricle floor, the central protuberance area, periaqueductal gray matter (8), paramedian regions of the cerebral peduncles, third ventricle walls (medial region of both thalami), mammillary tubercles, bilateral y symmetrical involvement of the confluence of internal and external white capsule, and some frontal cortical and subcortical areas.

The treatment is based on immediate parenteral administration of thiamine (100 mg a day for 2 weeks), followed by oral support of thiamine (100-300 mg a day for 3-12 months).

Development of Wernicke encephalopathy in this patient could be explained by several causes: by a deficiency of thiamine absorption after performing a cephalic duo-

denopancreatectomy, exacerbated by lack of supply of it in parenteral nutrition and increased requirement of this vitamin secondary to septic shock. After supplying therapeutic thiamine levels within parenteral nutrition, we achieved a significant improvement in the clinical status and radiologic imaging, allowing the patient discharge from the Intensive Care Unit to the Pneumology Service in order to treat the pneumonia and later on, final discharge from the hospital.

Wernicke's encephalopathy is a rare disease that should be considered for its prevention in patients with prolonged parenteral nutrition therapy by adding appropriate supplements.

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