# Wernicke Encephalopathy and Lactic Acidosis in Thiamine Deficiency

Thiamine deficiency can cause encephalopathy (Wernicke) and lactic acidosis. Herein we report a 6-year-old girl on total parenteral nutrition (TPN) who developed lactic acidosis and neurological symptoms due to improper vitamin replacement, which responded to thiamine injection. The MRI brain findings were not typical for Wernicke encephalopathy.

Thiamine acts as a co-enzyme for decarboxylation and transketolase reactions. Blood pyruvate and lactate can increase in thiamine deprivation [1]. Wernicke encephalopathy is a neurological complication of thiamine deficiency characterized by nystagmus, ophthalmoplegia, ataxia, and confusion. It is mostly reported in chronic alcoholic adults and in few pediatric patients with other etiologies [2].

A 6-year-old girl was referred for encephalopathy, tachycardia and lactic acidosis. She was diagnosed with megacystis microcolon intestinal hypoperistalsis syndrome and had previously undergone several surgeries including total colectomy and subtotal ileum resection. She was admitted for six months with dehydration, electrolyte imbalance, poor oral intake and was receiving total parenteral nutrition (TPN) since then. She had developed encephalopathy and blurred vision three days back and, tachycardia and lactic acidosis several hours before admission to the intensive care unit.

On admission, the heart rate was 145/min, blood pressure was 100/65 mmHg with normal capillary refill time and no edema. She was lethargic, with Glasgow coma scale of ten. Neurological examination showed nystagmus and ankle clonus. Rest of the tendon reflexes and sensory system were normal.

Preliminary laboratory examination showed hemoglobin 9.7 g/dL, white cell count $10\times10^3$ cells/µL, aspartate transaminase 108 U/L, alkaline phosphatase 352 U/L, serum procalcitonin 1.42 ng/mL, CRP 9.5 mg/L and lactic acidosis (pH 7.32, $PCO_2$ 26.6 mmHg, $HCO_3$ 15.9 mmol/L, lactate 7.3 mmol/L), Chest *X*-ray showed normal heart size and echocardiography was normal for systolic function (ejection fraction 65%). Her thyroid function test was normal.

Septic shock was diagnosed in the presence of fever, indwelling catheter for TPN and positive blood culture for gram-positive bacteria. Intravenous meropenem and teicoplanin were started empirically. Norepinephrine was added after fluid bolus but later discontinued as she progressively developed tachycardia and high blood pressure, and lactate increased to 13 mmol/L. It was noticed that she had not received water-soluble multivitamin infusion (MVI) due to a hospital-wide shortage for the last one month. Oral multivitamins were not substituted as physicians were unaware of the situation due to an electronic order system error. Thiamine 100 mg was administered intramuscularly. Lactate level decreased to 2 mmol/L within the first hour and tachycardia resolved within 12 hours. Magnetic resonance imaging (MRI) of brain showed diffusion restriction and signal increase on T2 weighted images in bilateral temporal, frontal and parietal cortex, corpus striatum, pons, and medulla oblongata. On the second day of thiamine treatment, her mental status improved, she became responsive to verbal stimuli and nystagmus disappeared. However, clonus persisted. Thiamine replacement (100 mg/day intramuscular) was continued for 14 days in addition to MVI in TPN.

Wernicke's encephalopathy is an acute neurological disorder due to thiamine deficiency. The mental deterioration and cardiovascular instability in the index patient were due to thiamine deficiency as neurological symptoms started one month after MVI shortage and improved after initiation of thiamine replacement. She recovered with neurological sequela due to delayed replacement of thiamine.

The typical radiological finding of Wernicke encephalopathy is symmetric hyperintensities on T2 weighted MRI in the thalamus, mamillary bodies, periaqueductal gray matter, and tectal plate [3]. Atypical cases with cortical involvement are reported similar to index patient [4].

Thiamine deficiency may cause lactic acidosis which is rapidly reversible by vitamin replacement. Cases with profound lactic acidosis traced to thiamine deficiency were reported during a nationwide shortage of intravenous multivitamins in the United States in 1997 [5]. Erythrocyte transketolase activity and thiamine pyrophosphate effect tests were not available to us. However, the dramatic regression of neurological symptoms and lactic acidosis after thiamine replacement was suggestive of thiamine deficiency.

Clinical suspicion of thiamine deficiency might be life-saving, treatment should be started as early as possible before laboratory confirmation. In case of unexplained, refractory lactic acidosis, thiamine deficiency should always be considered in the differential diagnosis.

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# Post-traumatic Pseudoaneurysm of Hepatic Artery: An Unusual Cause of Upper Gastrointestinal Bleeding

Pseudoaneurysm of hepatic artery with upper gastrointestinal bleeding is a rare but life-threatening complication of blunt trauma to the abdomen. An 8-year-old child with this condition was treated successfully with percutaneous coil embolization of the pseudoanysm.

**Keywords:** *Arterial injury, Management, Trauma.*

Pseudoaneurysm of any artery develops due to collection of blood between its two outer layers, the tunica media and the tunica adventitia. It is in contrast with the true aneurysm which involves all three layers of the wall of an artery. Among children sustaining traumatic injuries, 21% have abdominal injuries [1,2]. Rarely, the blunt trauma of the abdomen may be complicated by development of pseudoaneurysm of hepatic artery, which may rupture inside biliary tract, leading to life-threatening complication of hemobilia. Classical signs of hemobilia consist of upper abdominal pain, upper gastrointestinal hemorrhage and jaundice, called Quincke triad. All these three signs are present in only 22% of cases, whereas only upper gastrointestinal bleeding is present in 42% of cases [3].

An 8-year-old child presented in our emergency department with complaint of pain abdomen for 15 days and hematemesis and melena for 10 days. The pain abdomen started when he was punched in his abdomen by one of his schoolmates. He took analgesics for his pain abdomen. There was no history of fever, rash or any bleeding diathesis. He was pale and had tachycardia at admission. There was no history of fever, rashes or any bleeding diathesis. Blood pressure was 113/70 mmHg and there was no petechial/purpuric rash. He was given normal saline bolus and intravenous pantoprazole followed by whole blood transfusion. Blood investigations revealed low hemoglobin (4.8 g/100 mL) with normal leucocyte counts, liver enzymes and renal function tests; International normalized ratio was 0.95. Ultrasonography abdomen done outside had revealed a 9 mm calculus in gall bladder neck. Upper gastrointestinal endoscopy, which had been done prior to coming to our hospital, had documented erosion of mucosa of antrum and pylorus with blood and blood clot inside stomach. Blood was also seen coming out from ampulla of Vater and an impression of erosive gastritis and hemobilia had been reported. The child continued to have hematemesis after admission. A computed tomography (CT) angiography of the abdomen was done which revealed a pseudoaneurysm of the right hepatic artery ***(Fig.* 1*a*)**. Percutaneous coil occlusion of the right hepatic artery was done through the ipsilateral femoral artery ***(Fig.* 1*b*)**, and the hematemesis stopped thereafter. He continued to have intermittent colicky pain abdomen post procedure also, which persisted along with melena, till sixth day of admission. The child became completely asymptomatic on seventh day of admission, when he was discharged. He was asymptomatic, without any pallor, and with normal liver function test on follow up after one month.

Approximately 1.7% of children sustaining blunt