WERNICKE'S ENCEPHALOPATHY IN PEDIATRIC PATIENTS AFTER LIVER TRANSPLANT SURGERY: CLINICAL CASE REPORT

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ABSTRACT

Pediatric liver transplantation has become increasingly important in the management of chronic liver diseases in children. During the postoperative intensive care period, several risk factors may impair absorption and alter the metabolism of essential vitamins, including vitamin B1. Deficiency of vitamin B1 can lead to Wernicke encephalopathy, which often presents clinically in a manner similar to acute encephalopathy. In this report, we describe a case of a 6-year-old girl who underwent living-donor liver transplantation from an unrelated donor. Nineteen days after surgery, the patient developed seizures accompanied by a decreased level of consciousness. Brain MRI revealed hyperintense lesions around the aqueduct and in the occipital region. After excluding other potential causes of impaired consciousness, blood samples were obtained for vitamin B1 quantification and the patient was administered intramuscular vitamin B1. Within two days of treatment, the patient showed marked neurological improvement and complete cessation of seizures. Subsequent testing confirmed a severe deficiency of vitamin B1. This is a rare and easily overlooked complication after liver transplantation.

I. INTRODUCTION

Wernicke encephalopathy (WE) is an acute or subacute encephalopathy causing nervous system damage due to Thiamine (Vitamin B1) deficiency. This is a medical emergency; early treatment can reverse the condition with a very good response. If left untreated, the patient may suffer irreversible neurological damage and it can even be life-threatening. The first case of Wernicke encephalopathy was reported by Carl Wernicke and colleagues in 1881 as an acute neurological disorder due to vitamin B1 deficiency, with classic symptoms including ataxia, nystagmus, ophthalmoplegia and impaired consciousness.[1]

The prevalence of WE in studies ranges from 0.4% to 2.8% and is higher in alcoholics. WE is often considered a condition associated with alcohol abuse. However, currently, an increasing number of WE cases are being detected that are unrelated to alcohol addiction [2]. A study in Guangzhou, China, by Li Min Ding et al. on a group of liver transplant patients showed that between 2011 and 2021, 23 patients were diagnosed with WE according to Caine's criteria [3].

II. CASE REPORT

The patient is a 6-year-old female, first child, born via normal full-term delivery, diagnosed with biliary atresia and underwent the Kasai procedure at two months of age. Postoperatively, bile drainage was poor and the disease progressed to biliary cirrhosis with recurrent episodes of cholangitis; the Model for End-Stage Liver Disease (MELD) score was 31. At the time of admission, the child was in 1st grade with normal psychomotor development. The patient underwent living-donor left lobe liver transplantation from an unrelated donor on September 8, 2025. Postoperatively, the patient received total parenteral nutrition and was treated with Tazocin antibiotics and immunosuppressants including Tacrolimus and Corticosteroids to prevent rejection. Respiratory

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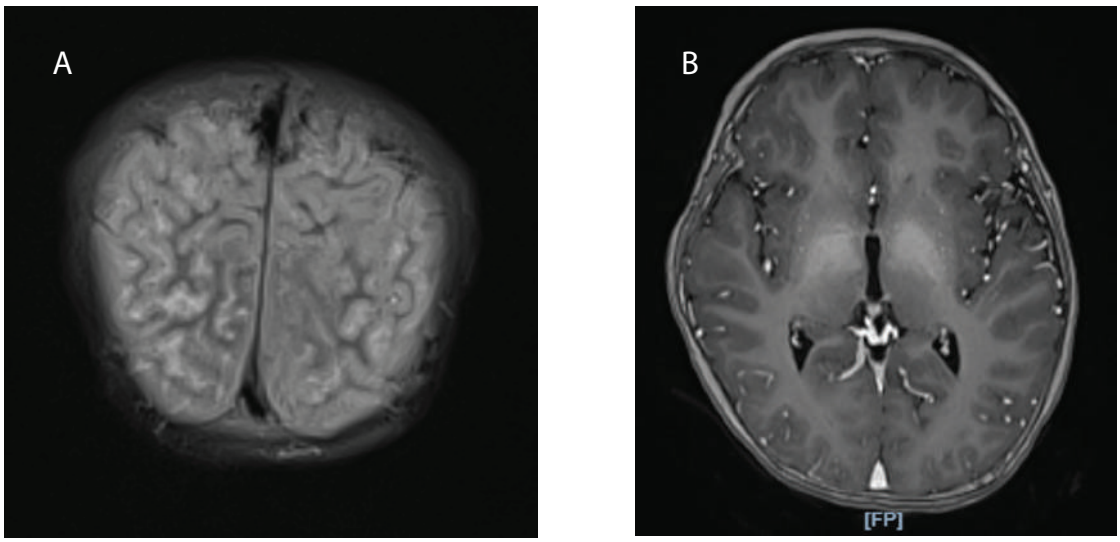
and hemodynamic parameters were stable, except for a trend of hypertension requiring control with oral Amlodipine; liver function tests improved over time. Bowel movements returned and Peptamen feeding was initiated on postoperative day 7. However, abdominal fluid subsequently increased with chylous characteristics, so oral intake was reduced and parenteral nutrition was increased. Infusion components included Glucose, electrolytes, phosphorus and magnesium, combined with Peptamen feeding.

The patient experienced two episodes of acute neurological symptoms:

- Episode 1: On postoperative day 8, the patient developed sudden seizures involving the face and both arms, accompanied by impaired consciousness and ophthalmoplegia; blood pressure at the time of seizure was high for age. Tests at the time of seizure showed no abnormalities in blood glucose, electrolytes, or acid-base balance. Brain MRI on the same day revealed abnormal hyperintense lesions in the subcortical occipital region and supratentorial area. Seizures were terminated with intravenous Midazolam; Nicardipine was maintained for blood pressure control and oral Levetiracetam (20 mg/kg/dose) was administered. Subsequently, the patient's consciousness fully recovered after 2 hours, with no further seizures in the following days.



A: Abnormal hyperintense lesions in the parieto-occipital cortex on FLAIR and T2W

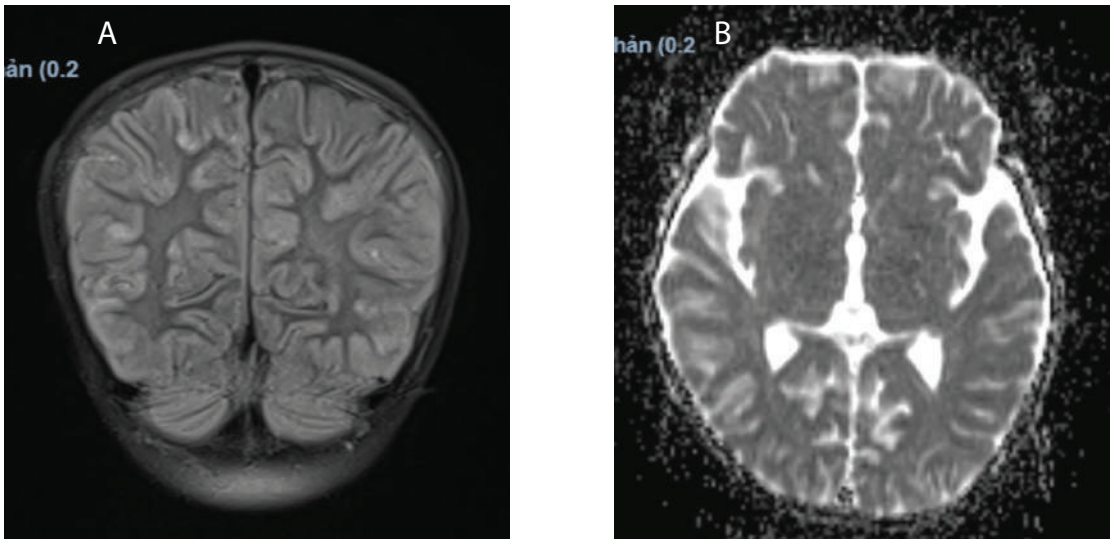
B: Hyperintensity in bilateral globus pallidus on T1W.

Figure 1. Brain MRI lesions on postoperative day 8

- Episode 2: On postoperative day 16, the patient developed high fever (39°C) accompanied by abdominal pain, loose stools and sudden focal seizures in both arms, with fixed gaze deviation to the left, no nystagmus, bilateral pupils 2.5 mm with reactive light reflex, accompanied by impaired consciousness: Glasgow Coma Scale 10 (Eyes: 4, Verbal: 3, Motor: 3). Clinical examination showed good air entry in lungs, SpO2 100%, heart rate 115 bpm, blood pressure 113/70 mmHg. Blood tests obtained at the time of seizure are presented in Table 1, including vitamin B1 quantification which would yield results after a few days. Brain MRI results showed scattered subcortical hyperintense lesions on T2W in the parieto-occipital region, with no structural or signal abnormalities in the thalamus or basal ganglia.

Table 1. Patient's laboratory results at the time of seizure

Test	Result	Unit
White blood cells	30.8	G/l
Hemoglobin	126	g/l
INR	1.43	
APTT	36.3	Second
Fibrinogen	3.35	g/l
NH_3	24	µmol/l
Total/Direct Bilirubin	28.3/11.1	mmol/l
GOT/GPT	34.4/27.2	U/l
Calcium	2.42	mmol/l
Sodium	133	mmol/l
Phosphorus	0.84	mmol/l
Blood glucose	6.2	mmol/l
Tacrolimus trough level	5.26	ng/ml
CSF cells	5	Cells/µL
CSF glucose	4.5	mmol/l
CSF protein	0.39	g/l
CSF chloride	120	mmol/l



A: Scattered hyperintensity in bilateral subcortical parieto-occipital regions on T2W sequence

B: Normal signal in thalamus and bilateral basal ganglia

Figure 2. Brain MRI lesions on postoperative day 16

After controlling blood pressure and trough Tacrolimus levels and excluding other causes such as electrolyte disturbances and blood glucose issues, we identified that the patient had nutritional risk factors leading to micronutrient deficiencies, including Wernicke encephalopathy, which could explain

the clinical symptoms despite the unavailability of Thiamine level results and atypical MRI findings. The patient was administered 100 mg of intramuscular vitamin B1 daily, along with intravenous Phosphorus supplementation to normalize blood Phosphorus levels. Within less than 24 hours, the patient showed marked improvement in consciousness and complete cessation of seizures. The patient's level of consciousness returned to completely normal after 48 hours. Quantitative results of blood vitamin B1 showed severe deficiency: 1.2 ng/ml (normal range: 20-51 ng/ml).

The patient continued with oral vitamin B1 supplementation and full oral feeding. No further seizures were recorded and the child was fully alert until discharge 3 weeks later.

III. DISCUSSION

3.1. Pathophysiology

Thiamine (vitamin B1) is one of the 8 B-group vitamins. It is water-soluble and the body cannot synthesize it; it must be supplemented through diet. Thiamine-rich foods include brown rice, pork, poultry, bread, cereals and infant formulas. The primary concern for Thiamine deficiency is alcohol abuse; however, there are many non-alcoholic causes leading to impaired supply and absorption of Thiamine:

- Cancer.
- Gastrointestinal surgery.
- Hyperemesis.
- Fasting.
- Gastrointestinal diseases.
- AIDS.
- Malnutrition.
- Dialysis and renal diseases.
- Prolonged parenteral nutrition.
- Psychiatric eating disorders.

Other causes: infection, poisoning, thyroid disease, unbalanced diet, unknown etiology...[4]

Physiologically, Thiamine deficiency causes dysfunction of the Krebs cycle and the Pentose phosphate pathways:

- It prevents pyruvate from converting to Acetyl CoA to generate energy for cells. Instead, pyruvate is converted to lactate.

- α Ketoglutarate Dehydrogenase cannot enter the Krebs cycle, causing reduced ATP production.

The consequence of ATP deficiency causes the Sodium-Potassium-ATP pump on the neuronal membrane to fail, reducing the transport of Sodium out of the cytoplasm and Potassium into the cell, altering the concentration gradient and potential. This leads to intracellular Sodium accumulation and intracellular lactate stasis, finally causing neuronal cell death via cytotoxic mechanisms. [5]

- Reduced Thiamine affects the pentose phosphate pathway: reduced production of NADPH, which is essential for nucleic acid and glutathione synthesis, leads to the accumulation of reactive oxygen species and reduced synthesis of nucleotides and myelin. This decreases the oxidative stress tolerance of neurons. Simultaneously, it disrupts the blood-brain barrier due to astrocyte damage. The result is cerebral edema due to vasogenic mechanisms. [6]

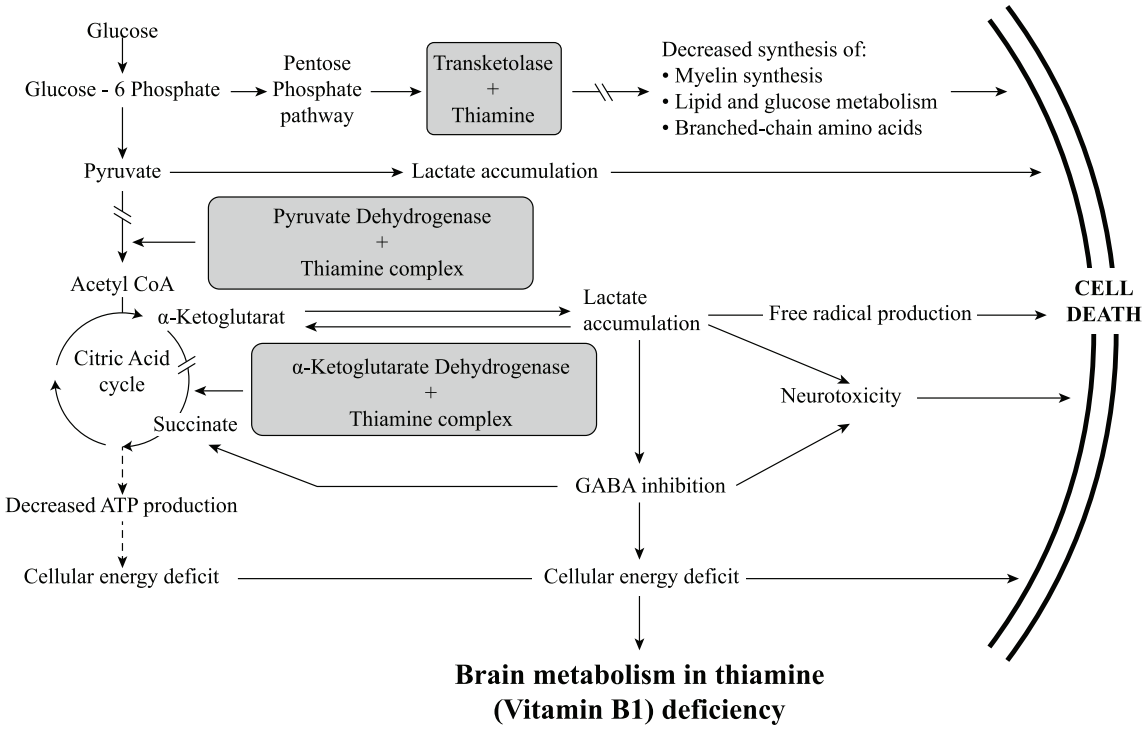


Figure 3. Pathophysiology of Thiamine deficiency [5,6]

3.2. Diagnosis

Despite differences in typical symptoms between the alcoholic patient group and patients with other causes and notably limited studies on children, most literature uses the diagnostic criteria by Caine et al., presented in the 2010 European Federation of Neurological Societies (EFNS) guidelines. Diagnosis of Wernicke encephalopathy has a sensitivity of 85% when 2 of the following 4 criteria are met (Level B Recommendation): [2]

1. Nutritional deficiency and a history of alcohol abuse or any other condition of vitamin B1 supply deficiency.

2. Ocular signs: nystagmus, ophthalmoplegia.

3. Cerebellar dysfunction: gait ataxia, coordination disorders.

4. Altered mental status or mild memory impairment.

Direct quantitative testing of blood Thiamine concentration has been performed in some countries, including Vietnam. The normal range of blood Thiamine concentration in adults is 20 – 51 ng/ml. However, blood contains only 0.8% of total body thiamine. Therefore, blood thiamine concentration cannot accurately assess the degree of thiamine deficiency in the body.

A normal Computed Tomography (CT) scan is not a criterion to exclude WE. Brain MRI helps support the diagnosis of WE. Brain MRI has a low sensitivity of about 53% but a high specificity of 93%. However, it is crucial not to delay Thiamine treatment while awaiting results. For similar reasons, the quantification of thiamine or thiamine pyrophosphate levels, while useful, is not routinely performed. Signs of WE on brain MRI include:

- Symmetrical hyperintensity on T2, FLAIR and DWI in periventricular areas: thalamus and periventricular region of the third ventricle (80%), periaqueductal area (59%), mammillary bodies (45%), cranial nerve nuclei (18%) and periventricular gray matter of the fourth ventricle (7%).

- In addition, MRI may show lesions in rare locations: putamen, caudate nucleus, corpus callosum, medulla, pons, red nucleus, substantia nigra of the midbrain, pre- and post-central gyri.

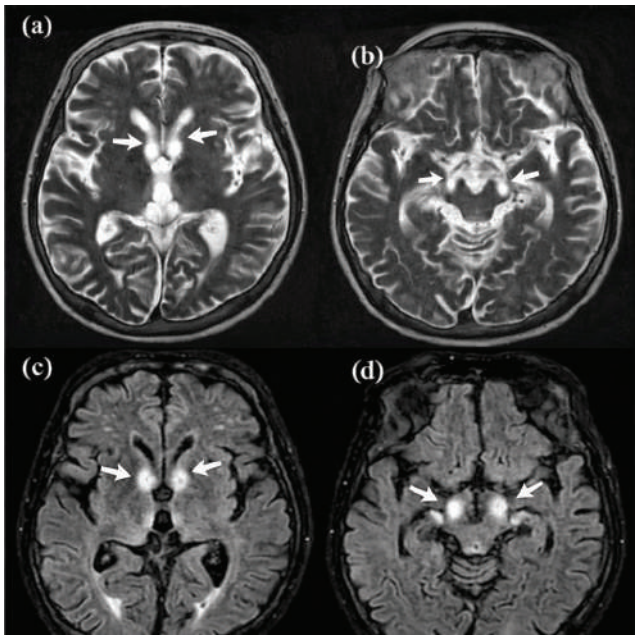


Figure 4. Typical lesions on MRI of Wernicke encephalopathy[6]

a,b: Bilateral symmetrical hyperintensity on D2W sequence in the thalamus, mammillary bodies and periaqueductal area

c,d: Lesions shown more clearly on FLAIR

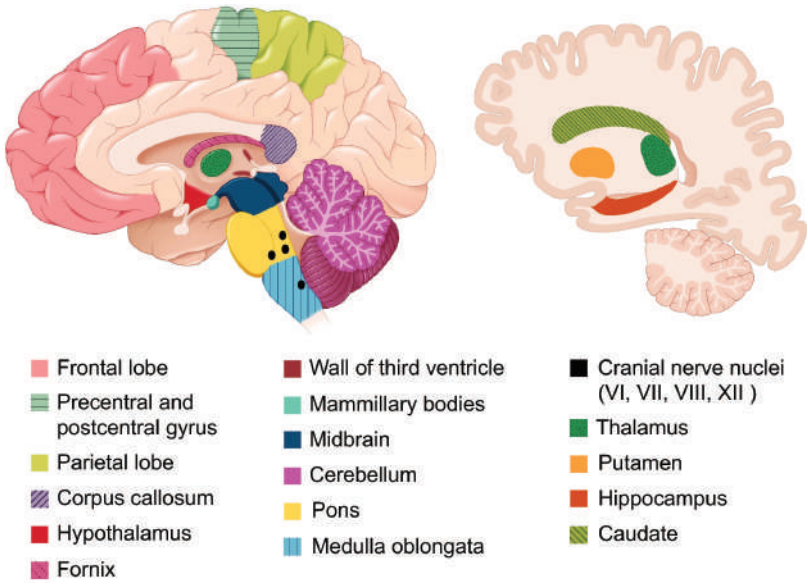


Figure 5. Distribution of brain lesions in Wernicke encephalopathy6

Our patient presented with neurological symptoms manifesting as seizures, ophthalmoplegia, impaired consciousness accompanied by fever and loose stools on a background of normal mental history. MRI did not show specific WE lesions like T2W hyperintensity in the thalamus or subcortical gray nuclei but showed small subcortical hyperintense areas in the bilateral parietal regions. Although the clinical symptoms and MRI were atypical, the patient had risks regarding parenteral nutrition, so we

included Thiamine deficiency encephalopathy as a differential diagnosis alongside infection, PRES syndrome and electrolyte disturbances.

Subsequently, although the patient's blood pressure and blood Tacrolimus levels were well controlled, the clinical condition did not improve. Blood samples were obtained for vitamin B1 quantification and the patient was simultaneously administered 100 mg of intramuscular Vitamin B1 daily. Three days later, the patient's consciousness improved markedly, with resolution of ophthalmoplegia and cessation of seizures. The patient's blood vitamin B1 quantification result showed a distinct deficiency: 1.2 ng/ml compared to the normal range of 20 – 51 ng/ml.

3.3. Treatment of Wernicke encephalopathy

Currently, there is no literature on specific treatment dosages or routes of administration for Thiamine supplementation in pediatric WE. For adults, the European Federation of Neurological Societies (EFNS) guidelines recommend using Thiamine 200 mg three times daily and intravenous administration is preferred over intramuscular injection (Level C Recommendation). There is currently limited documentation regarding the risk of anaphylaxis to Thiamine. However, many opinions suggest administering Thiamine where resuscitation facilities are available. Furthermore, delaying Thiamine administration can lead to irreversible encephalopathy and threaten life. Therefore, it should be implemented early [2].

Another guideline from the Royal Australasian College of Physicians (RACP) recommends:

- Emergency: Intramuscular or intravenous injection of 500 - 1500 mg daily.

- Therapeutic dose: For patients diagnosed with WE: Intravenous or intramuscular injection of 200 - 500 mg three times daily for 5 - 7 days, followed by 100 mg orally per dose, 3 times daily for 1-2 weeks, then reduced to 100 mg daily.

- Prophylactic dose: Intravenous or intramuscular injection of 100 mg - 200 mg three times daily, followed by oral administration of 100 mg three times daily for 1 - 2 weeks, then reduced to 100 mg daily.

IV. CONCLUSION

Wernicke encephalopathy is a rare neurological complication following liver transplantation. The main cause is reduced Vitamin B1 supply due to fasting or prolonged parenteral nutrition. Common symptoms include mental confusion, ophthalmoplegia, or cerebellar dysfunction. Treatment for Wernicke encephalopathy should be initiated immediately upon suspicion, as delayed supplementation can lead to irreversible neurological damage or be life-threatening.

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