Case Report

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# Wernicke Encephalopathy in a Patient of Acute Gastroenteritis: A Case Report

Internal Medicine Section

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## ABSTRACT

An acute neurological disease known as Wernicke Encephalopathy (WE) is characterised by the clinical triad of symptoms: ophthalmoparesis with nystagmus, ataxia, and disorientation. Thiamine deficiency is the underlying cause of this potentially fatal condition, which mainly affects the central and peripheral nervous systems. Hereby, the authors present a case of a 47-year-old male who presented with multiple episodes of loose stools and altered consciousness to the emergency ward. He had hyperchloremic metabolic acidosis suspected to be due to acute gastroenteritis, which may have caused his altered consciousness. Despite correction of his metabolic acidosis, his sensorium did not improve. Later, he was diagnosed with WE using Magnetic Resonance Imaging (MRI) brain findings, which showed symmetrical subtle hyperintensities in the medial part of the thalami and periaqueductal grey matter, suggestive of WE; his consciousness improved after thiamine supplementation. There should be a high suspicion of WE for timely diagnosis and treatment. Prompt treatment would be rewarding as WE is reversible, if treated in time.

**Keywords:** Metabolic acidosis, Ophthalmoparesis, Periaqueduct grey matter, Thiamine deficiency

## CASE REPORT

A previously healthy 47-year-old male presented to the Emergency Intensive Care Unit (ICU) with a complaint of loose stools persisting for four days, characterised by watery consistency and absence of foul odor. The patient reported experiencing eight to ten episodes of loose stools per day, accompanied by diffuse, crampy abdominal pain. Additionally, he complained of a high-grade fever persisting for three days, which was accompanied by generalised weakness. The patient had a history of chronic alcohol consumption for around ten years. He used to consume whisky, about 90 mL per day; however, he had abstained from alcohol for the past six months. Relatives revealed that the patient had poor oral intake for the last four days. Furthermore, he had a five-year history of hypertension and type 2 diabetes mellitus, for which he was taking tab. telmisartan 40 mg once daily and tab. metformin 500 mg twice daily.

On the day of admission, he presented with altered sensorium, which developed acutely over the morning. He also complained of losing balance while walking. He had no complaints of loss of consciousness, motor deficit of any limb, involuntary movements of limbs, or incontinence of bowel or bladder. He had no history of head injury.

On examination, the patient was conscious but disoriented to time, place, and person. There were no contusions or abrasions on the body. During the general examination, his pulse was 100 beats per minute, regular in rhythm, and normal in character, while his blood pressure measured 100/80 mmHg. His respiratory rate was 20 cycles per minute, and oxygen saturation was 98% on room air. Nervous system examination revealed that the patient was disoriented to time, place, and person and was not obeying verbal commands. The patient had a normal tone in all four limbs, with deep tendon reflexes 2+ and power grade 5/5 in all joints. He had no nystagmus or restricted eye movements, but there was truncal ataxia. He was not able to stand or walk due to severe loss of balance and ataxia, so Rhomberg's test was not possible. On abdominal examination, he had tenderness diffusely elicited in all areas of the abdomen. There was no hepatomegaly or splenomegaly, and no evidence of ascites.

On day one, his laboratory findings were as follows: haemoglobin was 12 g/dL; white blood cells, platelets, liver function tests, and serum ammonia were in the normal range; however, serum creatinine was 1.98 mg/dL and serum urea was 71 mg/dL. Serum sodium and potassium levels were within normal limits, but serum chloride was elevated around 124 mmol/L (normal value is 98 to 107 mmol/L). Venous blood gas showed hyperchloremic metabolic acidosis with a pH of 7.19 and bicarbonate of 8.0 mmol/L. He was immediately shifted to the Medical ICU and was started on intravenous antibiotics (ceftriaxone 1 gm BD and metronidazole 100 mL TDS), with intravenous fluids (Normal saline 0.9% NaCl) initially one-liter bolus followed by 150 ml/hr. He was also started on intravenous sodium bicarbonate 150 mEq bolus followed by 50 mEq TDS with subcutaneous regular insulin 10 IU before meals.

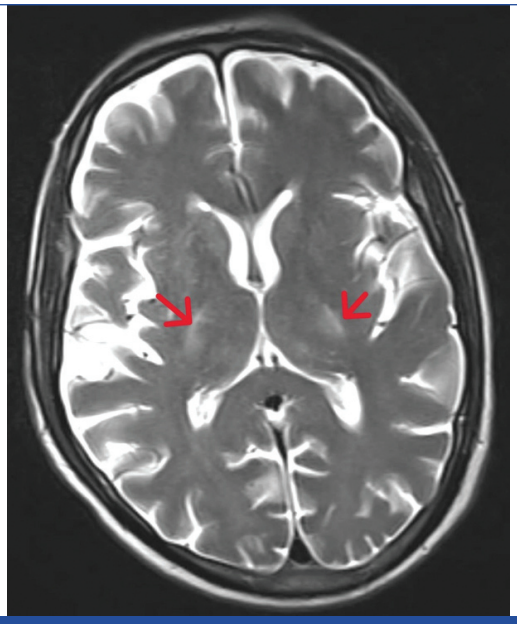
On day 2, his laboratory findings were as follows: serum creatinine decreased to 1.2 mg/dL and serum urea to 56 mg/dL; pH on venous blood gas was 7.30 and bicarbonate 14 mmol/L, with serum chloride at 109 mmol/L. His renal function and metabolic acidosis were improving, with no improvement in his level of consciousness. Ultrasound sonography of the abdomen and pelvis was done, which showed bilateral raised renal echogenicity.

On day 3, his laboratory findings were as follows: serum creatinine became normal at 1.0 mg/dL and serum urea was 50 mg/dL; venous blood gas showed a pH of 7.40 with bicarbonate at 20 mmol/L.

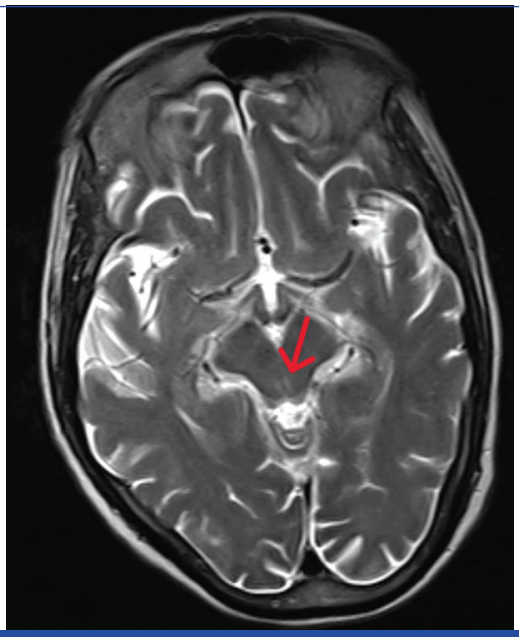
On day 4, MRI brain findings showed symmetrical subtle T2-weighted (T2WI) hyperintensities in the medial part of the bilateral thalami and periaqueduct grey matter, suggestive of WE, along with white matter Ischaemic changes [Table/Fig-1,2]. Based on the radiological findings, the differential diagnosis could be metabolic disorders such as WE, osmotic myelinolysis, Fabry disease, and Wilson disease. Osmotic myelinolysis commonly involves the pons, but extra-pontine osmotic myelinolysis could involve the thalami and basal ganglia. Fabry disease and Wilson disease could involve deep white and grey matter. The patient was promptly treated with intravenous thiamine. He was started on Inj. Thiamine 100 mg TDS for five days.

On day 6, there was an improvement in his consciousness as he became oriented to time, place, and person. He was shifted to the general medical ward on day 8 and started on oral thiamine tablets 100 mg TDS on day 9.

After four days of observation, he was discharged on oral thiamine tablets 100 mg TDS to be continued over a month along with



**[Table/Fig-1]:** MRI Brain (T2-weighted) Axial section showing subtle symmetrical hyperintensities in bilateral thalami (red arrows).



**[Table/Fig-2]:** Magnetic Resonance Imaging (MRI) Brain (T2-weighted axial section) showing subtle hyperintensities in periaqueductal grey matter.

antihypertensive medication, oral hypoglycemics, and insulin. At the follow-up after two weeks, he was able to walk properly and perform his daily activities. He could understand everything, and there was no altered behaviour. He had no loss of balance while walking. Oral thiamine tablets 100 mg TDS were further continued for two more weeks. After four weeks, his condition was similar to before, so he was advised to stop oral thiamine supplements.

## DISCUSSION

The WE is an acute condition characterised by mental confusion, gait ataxia, and ophthalmoplegia. It is a common and preventable disorder due to thiamine deficiency. Ocular palsies improve within hours of thiamine administration, while ataxia improves slowly [1].

In the Western world, WE is found in 0.4 to 2.8% of the general population at autopsy; the majority of these patients were chronic alcoholics and chronic binge drinkers [2]. It is possible that Wernicke lesions are not as well understood clinically as they should be since autopsy investigations have repeatedly shown a greater occurrence of them in the general population than clinical studies predict [3].

The WE can also arise from inadequate nutrition resulting from malabsorption, inadequate food consumption, elevated metabolic demands (as in the case of systemic disorders), or elevated excretion of the water-soluble vitamin thiamine (as in the case of renal dialysis) [4-6]. Causes other than chronic alcohol use include anorexia nervosa, hyperemesis of pregnancy due to nutritional loss, prolonged fasting or starvation due to decreased thiamine intake, systemic malignancy leading to higher metabolism, and in Acquired Immunodeficiency Syndrome (AIDS), it could be drug-induced or due to a catabolic state (acquired immunodeficiency syndrome) [4,5,7,8].

Transketolase, alpha-ketoglutarate dehydrogenase, and pyruvate dehydrogenase are three essential enzymes involved in energy metabolism that require thiamine as a co-factor [9]. Thiamine deficiency-induced neurotoxicity has been linked to mechanisms such as blood-brain barrier disruption, increased reactive oxygen species, and excitotoxicity mediated by N-Methyl-D-Aspartic Acid (NMDA) receptors [10].

Patients with WE are diagnosed when they meet two of the following four Caine criteria: dietary deficiency, oculomotor abnormalities, cerebellar dysfunction, or either altered mental status or mild memory impairment [11].

In a study by Mosch B et al., a non alcoholic 48-year-old male, morbidly obese, hypertensive individual with a poor nutritional diet presented with incontinence of urine and an unsteady gait following an episode of acute gastroenteritis. He was lethargic and confused on presentation. Laboratory results were largely unremarkable, and Computed Tomography (CT) of brain scans showed no significant abnormalities. MRI brain showed bilateral thalami and intraventricular haemorrhage. However, blood thiamine levels were markedly low, leading to the diagnosis of WE [12].

A case report by Zilberman I et al., resembling the scenario above, involved a 47-year-old woman who presented to the emergency room with complaints of acute gastroenteritis accompanied by severe vomiting and inadequate oral intake. She denied any history of alcohol consumption. Upon examination, she exhibited significant lethargy and diffuse weakness and was disoriented to place and date. Although hyponatremia was corrected as per guidelines, the encephalopathy persisted. MRI brain scans revealed Fluid-Attenuated Inversion Recovery (FLAIR) intensities within the dorsal midbrain, bilateral medial thalami, and mamillary bodies, indicative of WE. Serum thiamine levels were assessed before repletion and were found to be 47 nmol/L. Consequently, intravenous thiamine therapy was initiated, and she was transferred to inpatient rehabilitation for further management [13]. Similar findings were seen in present case, such as disorientation and symptoms of acute gastroenteritis.

In a retrospective study by Yin H et al., 17 patients were studied for non alcoholic WE. Dietary deficiency was the most common cause and was found in all 17 patients. Gastrointestinal surgeries were the second most common cause found in five patients, followed by vomiting. In terms of signs and symptoms, altered mental state was found in all patients, and ophthalmic signs were found in five patients. T2WI hyperintensities in the periaqueductal grey matter, medial thalami, and hippocampus were among the MRI brain findings for non alcoholic WE in 13 patients [14].

In present case, the patient initially presented with symptoms of acute gastroenteritis, which subsequently progressed to altered sensorium. Upon admission, hyperchloremic metabolic acidosis was noted, believed to be the underlying cause of the altered sensorium. Despite corrective measures for metabolic acidosis, the altered sensorium persisted. Although renal parameters were showing signs of normalisation with adequate urine output, the level of consciousness did not improve. MRI brain scans revealed characteristic changes indicative of WE. This highlights the importance of maintaining a high degree of suspicion for WE in cases of altered sensorium, particularly in the context of acute gastroenteritis.

## CONCLUSION(S)

The WE is a rare cause of altered sensorium but is reversible, if treated in time. Patients with persistent signs and symptoms of encephalopathy, despite correcting all metabolic parameters, should be considered for WE. The present case report shows that a bout of acute gastroenteritis, if not treated on time, can manifest as WE. Not all patients with WE would have classical signs and symptoms, which might delay the diagnosis. Thus, a high level of suspicion is imperative. Physicians should keep a watchful eye on this, and early treatment plays a pivotal role in managing this complication.

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