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Wernicke's Encephalopathy Complicated by Postoperative Digestive Fistula and Severe Nutritional Deficits

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Abstract | **Case Report**

Wernicke's encephalopathy (WE) is a neurological disorder caused by thiamine (vitamin B1) deficiency, often seen in patients with chronic alcoholism, malnutrition, or conditions impairing thiamine absorption. This case report discusses a 50-year-old woman with no significant medical history who developed WE following surgery for ileo-colic intussusception, complicated by a postoperative digestive fistula and severe nutritional deficits. The patient's clinical course emphasizes the need to consider WE in the differential diagnosis of neurological symptoms in patients with gastrointestinal diseases and postoperative complications, especially when risk factors for thiamine deficiency are present.

Keywords: Wernicke's encephalopathy (WE), Thiamine deficiency, Postoperative complications, Intestinal malabsorption, Ileo-colic intussusception.

INTRODUCTION

Wernicke's encephalopathy (WE) is an acute neurological condition caused by a deficiency in thiamine (vitamin B1). It is classically associated with chronic alcoholism but can also occur in patients with severe malnutrition, intestinal malabsorption, or prolonged vitamin losses, particularly in postoperative settings. Diagnosis is often delayed due to the polymorphic clinical presentation and low suspicion in non-alcoholic patients.

We report the case of a 50-year-old woman, with no notable medical history, who developed WE following surgery for ileo-colic intussusception, complicated by an enterocutaneous fistula leading to severe malnutrition. This case highlights the importance of considering WE in patients with serious digestive complications and neurological disorders, to ensure early thiamine treatment and prevent irreversible neurological damage.

CLINICAL CASE

The patient, aged 50 with no prior medical or surgical history, presented with signs of bowel obstruction. Abdominal CT revealed an acute ileo-cecal intussusception causing mechanical small bowel obstruction, without signs of digestive ischemia. Emergency surgery was performed.

Operative findings revealed terminal ileum intussusception into the right colon, which could not be manually reduced. An extended right colectomy was performed, removing the terminal ileum, cecum, ascending colon, hepatic flexure, and part of the transverse colon. A side-to-side ileo-transverse anastomosis was created.

Histopathological examination showed ischemic and hemorrhagic changes in the ileo-colic segment, consistent with intussusception on a submucosal inflammatory nodule, with no signs of malignancy. Lymph node analysis showed no metastatic adenopathy (16N-/16N).

Postoperatively, the patient developed feculent discharge from former drain sites, suggestive of an enterocutaneous fistula. Conservative management was initiated, including biological monitoring, nutritional support, and correction of metabolic imbalances.

Biological work-up revealed moderate anemia, leukocytosis, and very high CRP levels, indicating severe inflammation. Hypoalbuminemia and electrolyte disturbances were also noted.

A few days later, the patient developed neurological symptoms including altered consciousness, nystagmus, and ataxia. Neurological examination and brain imaging were compatible with Wernicke's encephalopathy, likely secondary to thiamine deficiency due to acute malnutrition and prolonged digestive losses. Intravenous thiamine treatment was immediately started, resulting in gradual improvement of the neurological symptoms.

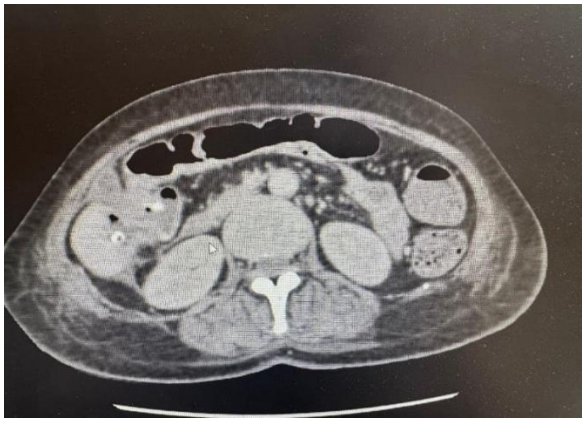

Figure 1: Axial CT scan showing a digestive fistula

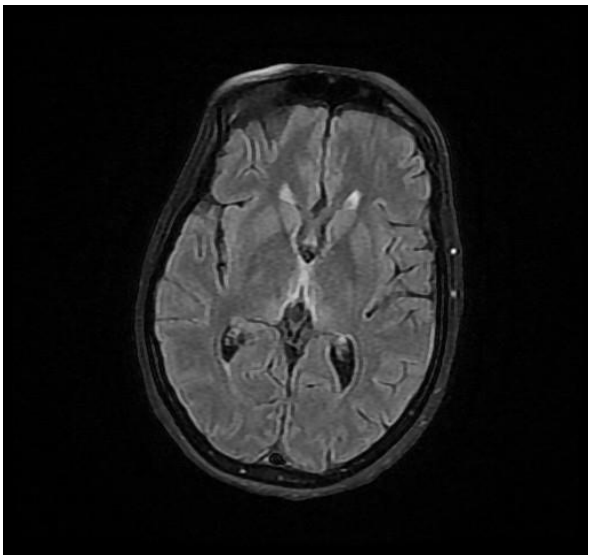

Figure 2: MRI slice showing FLAIR hyperintensity of the thalamus

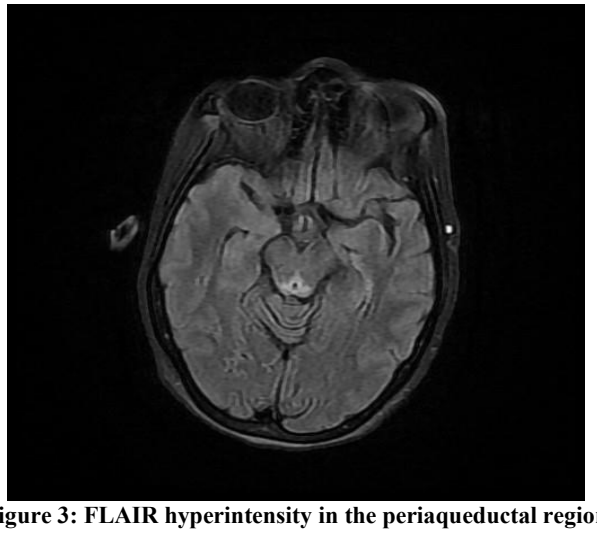

Figure 3: FLAIR hyperintensity in the periaqueductal region

DISCUSSION

Wernicke's encephalopathy (WE) is a neurological emergency caused by thiamine (vitamin B1) deficiency, often underdiagnosed in non-alcoholic settings. It typically occurs in high-risk patients such as those with malnutrition, after major gastrointestinal surgery, or with prolonged digestive losses. The classical clinical triad includes confusion, ataxia, and nystagmus—though this full triad is present in only a third of cases, making diagnosis challenging.

In our case, the patient had no history of alcoholism or digestive disease. The trigger was acute malnutrition secondary to a severe postoperative complication: an enterocutaneous fistula following extended right colectomy for ileo-colic intussusception. This led to significant nutritional imbalance and an uncorrected thiamine deficiency.

Similar non-alcoholic WE cases have been reported in the literature, especially in patients with complex postoperative courses after digestive surgery. Digestive fistulas cause major fluid, electrolyte, and vitamin losses, and early postoperative malabsorption, particularly in the absence of early supplementation, can rapidly lead to vitamin B1 deficiency.

Diagnosis is mainly clinical, supported, when possible, by magnetic resonance imaging (MRI), which may show characteristic lesions in periventricular areas such as the mammillary bodies, thalamus, and floor of the fourth ventricle. However, MRI can be normal and should not delay treatment. A rapid clinical response to parenteral thiamine further supports the diagnosis.

Management relies on immediate high-dose parenteral thiamine supplementation. In our case, early vitamin B1 administration led to progressive neurological improvement. This case highlights the importance of preventing malnutrition in at-risk patients, especially those with postoperative digestive complications, and the need to consider WE in any unexplained acute neurological symptoms.

CONCLUSION

This case illustrates the occurrence of Wernicke's encephalopathy in an unusual context: a non-alcoholic patient following digestive surgery complicated by an enterocutaneous fistula. It stresses the importance of clinical vigilance in patients at risk of thiamine deficiency who present with suggestive neurological symptoms, even if mild. Early consideration of the diagnosis and prompt thiamine administration are essential to prevent irreversible neurological damage. The case also emphasizes the importance of rigorous nutritional management in postoperative care, particularly in patients with major digestive complications.

REFERENCES

1. Ammouri W, Harmouche H, Alaoui M, Tazi ZM, Maamar MM, Adnaoui M. *Gayet-Wernicke encephalopathy in non-alcoholic patients: A serious complication.* Rare Diseases Journal.
2. Hudong Liang, Lan Wu, Ling-ling Liu, Jinming Han, Jie Zhu, Tao Jin. *A case report: Non-alcoholic Wernicke encephalopathy associated with polyneuropathy.* SAGE Open Medical Case Reports.
3. Yin H, Xu Q, Cao Y, Qi Y, Yu T, Lu W. *Nonalcoholic Wernicke's encephalopathy: a retrospective study of 17 cases.* SAGE Open Medical Case Reports.
4. Okafor C, Nimmagadda M, Soin S, Lanka L. *Non-alcoholic Wernicke's encephalopathy: great masquerader.* BMJ Case Reports.