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Wernicke's Encephalopathy in Crohn's Disease: A Case Report Emphasizing Thiamine Deficiency Risk During Total Parenteral Nutrition

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ABSTRACT

Vitamin B1 (thiamine) is vital for carbohydrate metabolism and neurological functions. Thiamine deficiency, often associated with alcoholism, can arise due to absorption issues, increased metabolic demand, or inadequate intake. While rare in industrialized nations, it poses a risk in conditions like Crohn's disease (CD), particularly during prolonged total parenteral nutrition (TPN) without vitamin supplementation. Wernicke's Encephalopathy (WE), a complication characterized by altered mental status, ocular signs, and ataxia, can lead to Wernicke-Korsakoff syndrome (WKS).

A severely malnourished 50-year-old with CD developed WE after extended TPN without thiamine supplementation. Initial symptoms included weight loss, diarrhea, and anorexia. Despite CD treatment, TPN lacked thiamine supplementation. Neurological symptoms manifested after 15 days, with MRI confirming WE. Thiamine deficiency (40 nmol/L) was treated with 500 mg thiamine injections three times a day. Despite treatment, residual symptoms persisted, evolving into Korsakoff's syndrome.

WE diagnosis is challenging due to a variable symptom triad. WKS, primarily linked to alcohol use, is underdiagnosed in nonalcoholic patients. Patients with malabsorption or malnutrition, including CD, are at risk, especially during TPN without multivitamin injections. Thiamine deficiency leads to neurotoxicity, prompting immediate thiamine administration. MRI aids diagnosis, but treatment should not be delayed. Recommended thiamine doses vary, emphasizing individualized care.

Awareness of WE in CD patients is crucial, emphasizing timely recognition, risk factor understanding, and optimized treatment strategies. Nonalcoholic WKS can be prevented through prophylactic parenteral thiamine treatment in at-risk patients. This case underscores the importance of vigilance, early intervention, and thiamine supplementation during TPN in CD, highlighting the need for tailored management approaches.

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KEYWORDS:

Wernicke's Encephalopathy, Crohn's disease, Total Parenteral Nutrition, Korsakoff syndrome

INTRODUCTION

B1 vitamin, a water-soluble vitamin functions as a coenzyme in the metabolism of carbohydrates and branched-chain amino acids and is involved in neurotransmitter production and glucose metabolism (1).

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The daily recommended dose of thiamine for an average, healthy adult is 1.4 mg (2).

This deficiency is caused by a combination of factors, including defective absorption, excessive elimination, or, rarely, insufficient intake (1). In industrialized countries, where diets are generally varied, thiamine deficiency is uncommon and most frequently associated with alcoholism (3). Pregnancy and critical illness increase the metabolic demand for thiamine and require supplementation (4). Deficiency may also occur in malnourished patients receiving prolonged intravenous (IV) nutrition without vitamin supplementation (5,6).

Such conditions are common in patients with inflammatory bowel disease (IBD), particularly Crohn's disease (CD) (7).
Clinical manifestations of thiamine deficiency in adults vary and can affect the cardiovascular, muscular, nervous, and gastrointestinal systems. Wernicke-Korsakoff syndrome (WKS), a serious neurological disorder, is one of its complications. The diagnosis of WKS includes the acute triad of symptoms (altered mental status, ocular abnormalities, and ataxia) associated with Wernicke's encephalopathy (WE) and the chronic amnestic symptoms of Korsakoff syndrome (8).
Here, we report a case of severely malnourished patient with CD who developed WE following prolonged TPN without thiamin supplementation.

Case report:
A 50-year-old male was admitted to our department because of a 16-kg weight loss over a period of 3 months, he was extremely malnourished with a body mass index (BMI) of 13,3 kg/m2.
He presented a liquid diarrhea without mucus or blood at a rate of 10 stools per day, associated with a Koenig syndrome evolving for one month with a decline of the general state.
Initial laboratory findings were as follows: hemoglobin at 9.8 g/dL; platelet count 253000/µL; CRP level 173 mg/L; fecal calprotectin level 2038 µg/g ; albumin level 18 g/L; cholesterol level 0,67 g/L, LDL level 0,35g/L and HDL level 0,14g/L.
An abdominal CT revealed diffuse bowel wall thickening, more marked in the ascending colon, without stenosis or fistulas *(Figure 1).*

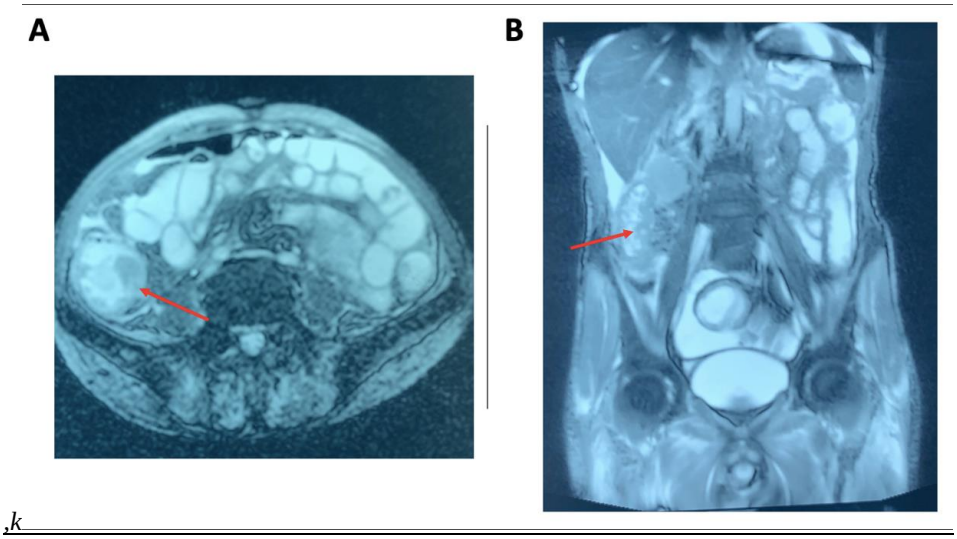

Figure 1: **Abdominal CT scan showing diffuse colonic parietal thickening more marked in the ascending colon measuring 20mm maximum thickness (red arrows). A: axial section; B: coronal section**

Colonoscopy showed ileocecal ulcerations and edema, segmental erythema, and hyperemia of the rectocolonic mucosa, with aphthous erosions, pseudopolyps and loss of vascular pattern, two large and deep ulcers at 40 cm from the anal verge were identified *(Figure 2).*

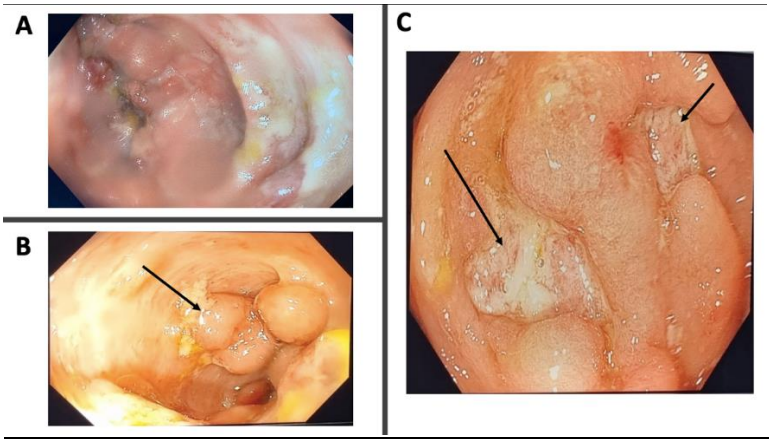

Figure 2: **Colonoscopy showing a congestive, oedematous, polypoid ileo-caecal valve (A) with ulcerated, polyploid rectocolitis (B) with 2 deep ulcerations 40cm from the MA exposing the muscularis (C).**

Investigations led to the diagnosis of colic Crohn's disease in severe relapse which was treated by high dose steroids and antibiotics, with parenteral nutrition and infusion of 5% glucose serum for anorexia and repeated infusions of albumin. The prescription was based on the nutrients contained in a standard three-compartment bag comprising 610 kcal/d, with 3,6 g nitrogen, 80 g glucose, 22.2 g lipid, 21 mmol sodium, 16 mmol potassium, 2 mmol calcium, 2.2 mmol magnesium, and 8.5 mmol phosphate and was given together with 1500 mL of 5% glucose solution. No thiamine or any vitamin substitution was administered during this period.

Due to corticoresistance, the patient required surgical management, but was refused by the patient's family.

After 15 days, his family complained of sudden-onset dizziness and gait ataxia with hypotension and tachycardia. Neurologic examination and brain scan were not able to identify any abnormal neurologic signs except for latent nystagmus in the lateral gaze.

The next day, his symptoms worsened and new symptoms including progressive blurred vision, cognitive dysfunction, intermittent confused speech developed, amnesia, and multidirectional nystagmus.

MRI revealed a high bilateral signal-intensity lesion of the mammillary body, posteromedial thalami and tectum, and the periaqueductal space on a T2-weighted image which was compatible with Wernicke's Encephalopathy (WE) (*Figure 3).*

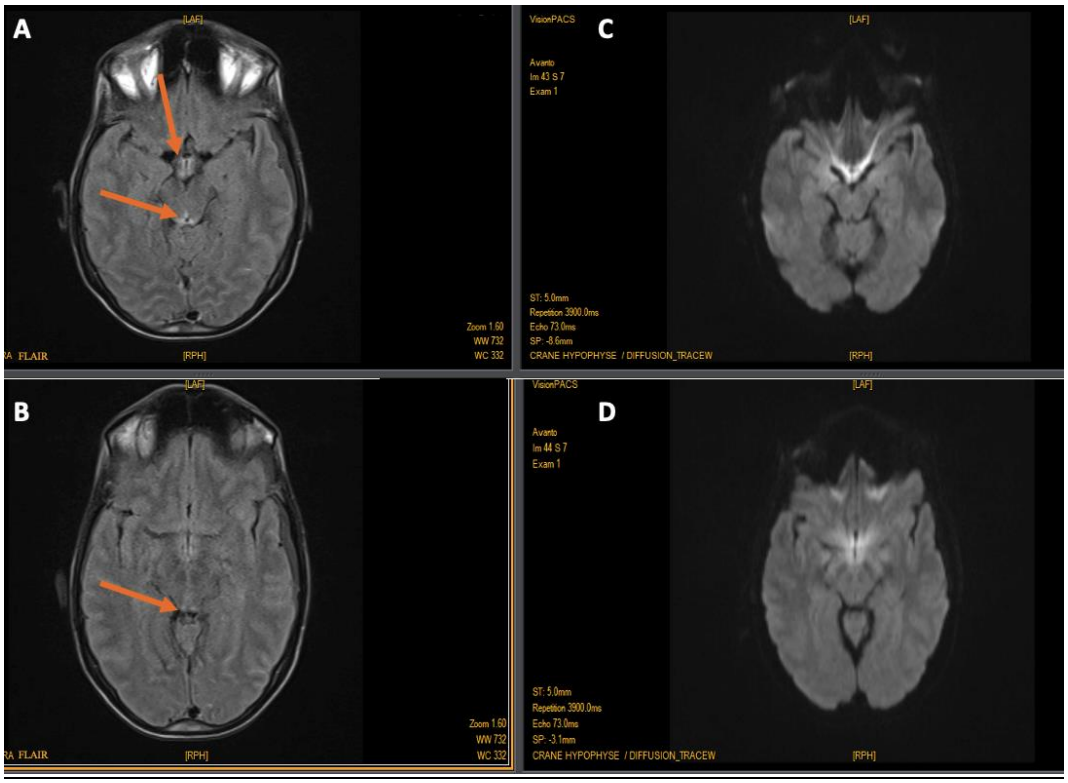

Figure 3: **Brain MRI showing FLAIR hypersignal (A-B) of the tectal lamina, mammillary bodies, periaqueductal, posteromedial thalami and lateral walls of V3, with discrete Diffusion hypersignal (C-D).**

The vitamin B1 deficiency was confirmed with low level 40nmol/L (norms: 83-245 nmol/L).

The patient received immediately thiamine injection at 500 mg 3 times a day.

Even after intravenous thiamine supplementation, his symptoms worsened and left him with sequelae. He kept memory disorders with a muscular weakness of the four limbs having thus evolved towards the chronicity: Wernicke-Korsakoff syndrome.

DISCUSSION

In 1881, Carl Wernicke first described Wernicke encephalopathy (WE) as a set of three main symptoms: confusion, oculomotor disorders (ophthalmoplegia and nystagmus), and cerebellar ataxia. However, the triad was only complete in 8%-30% of cases (9), with the subclinical cases identified in 19% (10), making the diagnosis of WE challenging.

Wernicke-Korsakoff syndrome (WKS), a neurological disorder commonly associated with alcohol use disorder with a prevalence of 12,5% (11) , is less widely recognized for its occurrence in nonalcoholic patients, a fact that is often overlooked (12). According to autopsy-based studies, the disorder is still greatly underdiagnosed, WKS was suspected clinically in less than one-third of alcoholic and only in 6% of nonalcoholic patients. Vitamin B1 deficiency unrelated to

alcohol abuse have an incidence of 0.8%–2.8% in autopsy reports and only 0.04%–0.13% in clinically reported cases (13).

It is essential to consider Wernicke encephalopathy (WE) and thiamine deficiency in patients experiencing malabsorption, malnutrition, or malignancies, even if they present only one symptom of the classic triad. WE may develop when total parenteral nutrition (TPN) is administered without daily multivitamin injections (14).

Micronutrient deficiencies in patients with Crohn's disease (CD) are not uncommon and typically result from a combination of reduced dietary intake, disease-related malabsorption, and a catabolic state due to inflammation. It is important to note that the risk of deficiencies persists even when the disease is in remission (7).

Vitamin B1 is a cofactor for essential enzymes involved in carbohydrate and lipid metabolism, amino-acid production, and neuronal myelination (15). Consequently, thiamine deficiency leads to well-established neurotoxicity, with brain damage occurring within the first week of thiamin deficiency. If untreated, this can result in neuronal necrosis and irreversible neurological impairment, leading to Korsakoff's syndrome after just two weeks (13,16).

Therefore, even a slight suspicion of Wernicke's encephalopathy should prompt the immediate administration of high doses of thiamine parenterally (17).

Magnetic resonance imaging (MRI) is currently considered the most reliable method for confirming the diagnosis, with a high specificity of 93% but a sensitivity of only 53% for diagnosing WE (13). However, MRI should not delay the start of treatment. Typical MRI findings include T2, FLAIR, and DWI hyperintensity in bilaterally symmetrical lesions of the paraventricular regions of the thalamus, hypothalamus, mammillary bodies, periaqueductal region, and the floor of the fourth ventricle (18).

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Nonalcoholic WKS is atypical, and its occurrence can be virtually eliminated through prophylactic parenteral thiamine treatment in patients at risk of malnutrition. Oral thiamine is insufficient to prevent WKS in individuals experiencing vomiting or weight loss (21).

CONCLUSION

In summary, these studies highlight the critical need for heightened clinical awareness of the potential for Wernicke's encephalopathy in patients with Crohn's disease. Early recognition, a thorough understanding of associated risk factors, and the implementation of optimized treatment strategies are essential to effectively prevent and manage this rare but serious neurological complication in the context of Crohn's disease.

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