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Clinical Image

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When the Gut Fails, the Brain Suffers: Wernicke Encephalopathy in Pediatric Inflammatory Bowel Disease

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Case Presentation

A 14-year-old girl presented with growth deceleration, haemorrhagic diarrhea, and vomiting following incidental raw fish intake. Initial laboratory evaluation showed normocytic normochromic anaemia (Hb 9 g/dL, MCV 85 fL) and elevated inflammatory markers (ESR 59 mm/h, CRP 36 mg/L, faecal calprotectin 704 mg/kg). The autoimmune panel was positive (p-ANCA, ANA 1:160 homogeneous, anti-PR3), while infectious causes and celiac disease were excluded. She was diagnosed with moderate-to-severe colonic non-stricturing Crohn's disease (PCDAI 45; SES-CD 15; Mayo endoscopic subscore 3). A diagnostic colonoscopy was complicated by iatrogenic colonic perforation requiring urgent surgery with protective ileostomy. The postoperative course was further complicated by acute pancreatitis. Due to persistent vomiting and intolerance to oral intake, Total Parenteral Nutrition (TPN) was initiated.

After two weeks of exclusive TPN, the patient developed progressive neurological symptoms, including horizontal nystagmus, diplopia, confusion, ataxia, and a positive Romberg sign. Laboratory testing revealed low thiamine levels (19.2 µg/L; normal range 32–95 µg/L). Brain Magnetic Resonance Imaging (MRI) demonstrated symmetrical T2/FLAIR hyperintensities involving the mammillary bodies, hippocampal–forniceal region, and brainstem **(Figure 1)**, supporting the diagnosis of Wernicke encephalopathy. High-dose intravenous thiamine (500 mg twice daily for 7 days) and multivitamin supplementation were promptly initiated, followed by oral therapy (300 mg twice daily for 35 days). The patient showed rapid clinical improvement with normalization of thiamine levels and complete resolution of neurological symptoms, without residual deficits.

Keywords: Inflammatory bowel disease; Wernicke encephalopathy; Malnutrition; Pediatric

Abbreviations & Acronyms: TPN- Total Parenteral Nutrition; ESR- Erythrocyte Sedimentation Rate; CRP- C Reactive Protein; PCDAI- Pediatric Crohn Disease Activity Index; SES-CD - Simple Endoscopic Score for Crohn's Disease; WE- Wernicke Encephalopathy

Discussion

Wernicke encephalopathy is an acute neurological disorder caused by thiamine deficiency, classically characterized by ophthalmoplegia, ataxia, and confusion. Although well described in adults, it is rarely reported in children. Pediatric patients with inflammatory bowel disease are particularly vulnerable due to malnutrition, inflammation, prolonged fasting, and inadequate micronutrient supplementation during total parenteral nutrition **[1–3]**. This case highlights the importance of early recognition of neurological symptoms in high-risk patients. Brain MRI typically shows symmetrical involvement of the mammillary bodies and periventricular regions **[4]**, as observed in this patient **(Figure 1).** Prompt thiamine administration is crucial, as delayed treatment may lead to irreversible neurological damage. Early intervention is associated with rapid and complete recovery **[5].**

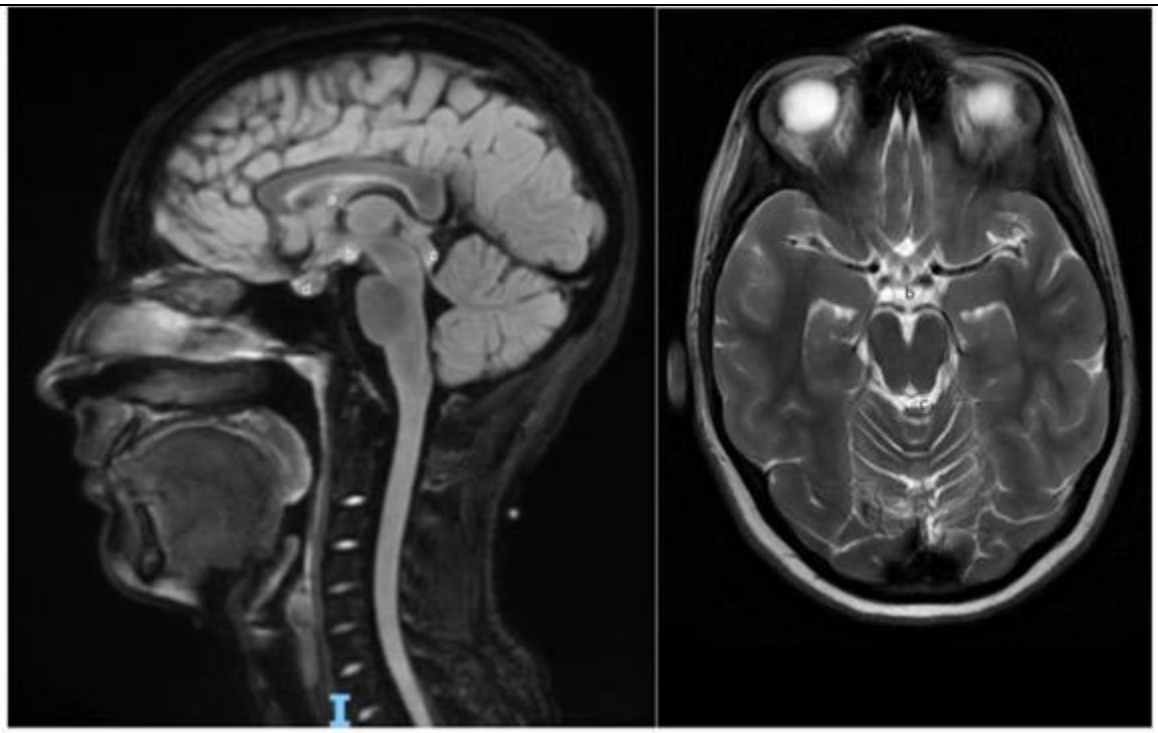

Figure 1: Brain MRI findings in Wernicke encephalopathy.

Brain magnetic resonance imaging showed symmetrical T2/FLAIR hyperintensities in the hippocampus (fornices) (a), quadrigeminal plate -mammillary bodies (b), especially the inferior mammillary bodies (c), the infundibular hypothalamic region (d)-, and pons, consistent with Wernicke's encephalopathy

Key Teaching Point

Wernicke encephalopathy should be suspected in pediatric patients receiving prolonged parenteral nutrition who develop acute neurological symptoms; early thiamine replacement is essential to prevent permanent neurological damage.

References

1. Sechi G, Serra A. Wernicke's encephalopathy: new clinical settings and recent advances in

diagnosis and management. Lancet Neurol. 2007;6(5):442–55.
2. Oudman E, Wijnia JW, Oey MJ, van Dam M, Painter RC, Postma A. Wernicke's encephalopathy in children and adolescents: a systematic review. Eur J Pediatr. 2019;178(4):515–25.
3. Mehanna HM, Moledina J, Travis J. Refeeding syndrome: what it is, and how to prevent and treat it. BMJ. 2008;336(7659):1495–8.
4. Zuccoli G, Pipitone N. Neuroimaging findings in acute Wernicke's encephalopathy: review of the literature. AJR Am J Roentgenol. 2009;192(2):501–8.
5. Thomson AD, Cook CC, Touquet R, Henry JA. The Royal College of Physicians report on alcohol: guidelines for managing Wernicke's encephalopathy in the accident and emergency department. Alcohol Alcohol. 2002;37(6):513–21.

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