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Non-Alcoholic Wernicke's Encephalopathy in Gastroenterology: Clinical Features and Outcomes from a Retrospective Study

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Abstract **Original Research Article**

Background: Wernicke's encephalopathy (WE) is an acute neuropsychiatric disorder caused by thiamine deficiency. Although classically associated with chronic alcohol use, it is increasingly recognized in non-alcoholic patients, particularly in the setting of gastrointestinal diseases. ***Objective:*** To describe the clinical characteristics, diagnostic findings, management, and short-term outcomes of WE in non-alcoholic patients hospitalized in a gastroenterology department. ***Methods:*** A retrospective study was conducted between 2023 and 2025, including patients with suspected WE. Diagnosis was based on the presence of at least two components of the classical triad in a compatible clinical context. Clinical, radiological, and therapeutic data were collected. ***Results:*** Seven patients were included, with a mean age of 27.3 years and a female predominance. Nystagmus, ataxia, and confusion were the most frequent manifestations. Brain MRI confirmed the diagnosis in only one case, while EEG was often normal. Most patients improved after intravenous thiamine, although persistent deficits were observed in some cases. ***Conclusion:*** WE should be suspected in non-alcoholic patients with gastrointestinal diseases and malnutrition. Early administration of intravenous thiamine remains essential to prevent irreversible neurological damage.

Keywords: Wernicke's encephalopathy; thiamine deficiency; non-alcoholic; malnutrition; gastrointestinal diseases; neurological complications.

INTRODUCTION

Wernicke's encephalopathy (WE) is a severe neurological disorder resulting from thiamine deficiency, leading to impaired cerebral metabolism and neuronal injury. Although traditionally associated with chronic alcohol use, WE is increasingly recognized in non-alcoholic patients, particularly in those with gastrointestinal diseases.

In this population, several mechanisms contribute to thiamine depletion, including reduced nutritional intake, prolonged vomiting, intestinal malabsorption, and increased metabolic demands during acute illness. As a result, patients with digestive disorders represent a high-risk group.

The diagnosis remains challenging because the classical triad of confusion, ataxia, and oculomotor abnormalities is present in only a minority of cases. This often leads to delayed recognition and increases the risk of irreversible neurological damage, including Korsakoff syndrome. The present study aimed to describe the clinical presentation, diagnostic approach, management, and outcomes of WE in a non-alcoholic population hospitalized in a gastroenterology department.

MATERIALS AND METHODS

We conducted a retrospective study including patients admitted to the gastroenterology department between 2023 and 2025 for suspected Wernicke's encephalopathy. Patients were included when they presented at least two components of the classical triad, namely ataxia, altered mental status, and oculomotor abnormalities, in a clinical context suggestive of thiamine deficiency.

Data were collected from medical records and included demographic characteristics, underlying gastrointestinal diseases, neurological manifestations, electroencephalogram findings, brain magnetic resonance imaging results, and therapeutic management. Plasma thiamine levels were not available in our setting.

All patients received high-dose intravenous thiamine ranging from 500 to 1000 mg per day, followed by oral supplementation. Clinical outcomes were assessed based on the evolution of neurological symptoms during hospitalization and early follow-up.

RESULT

Seven patients were included in the study, with a mean age of 27.3 years and a predominance of female patients. The underlying conditions were diverse and included acute pancreatitis, Crohn's disease, eosinophilic esophagitis with stenosis, systemic vasculitis, and pancreatic neuroendocrine tumor.

Neurological manifestations were dominated by oculomotor abnormalities, particularly nystagmus, which was observed in the majority of patients. Ataxia and confusion were also frequent but less consistently present. The distribution of these clinical signs is illustrated in Figure 1, which highlights the predominance of oculomotor involvement compared with other neurological features.

Electroencephalogram findings were nonspecific, with most patients showing normal results and only a minority presenting generalized slowing. These findings are summarized in Figure 2, emphasizing the limited diagnostic contribution of EEG in this setting.

This figure illustrates the predominance of normal EEG findings, with a smaller proportion of patients exhibiting diffuse slowing.

Brain magnetic resonance imaging confirmed the diagnosis in only one patient, while remaining cases showed no specific abnormalities.

All patients received intravenous thiamine promptly after clinical suspicion. Clinical outcomes were generally favorable, with rapid neurological improvement observed in most patients, particularly in oculomotor and cognitive symptoms. However, persistent neurological deficits were noted in a subset of patients, as illustrated in Figure 3.

DISCUSSION

Wernicke's encephalopathy in non-alcoholic patients is increasingly recognized but remains underdiagnosed in clinical practice, particularly in gastroenterology. Our findings confirm that gastrointestinal diseases represent a significant risk factor for thiamine deficiency due to a combination of reduced intake, malabsorption, and increased metabolic demand.

The clinical diagnosis of WE remain challenging, mainly because the classical triad is rarely complete. In our study, none of the patients presented all three components simultaneously, which is consistent with previous reports indicating that the full triad is present in less than 20% of cases. This highlights the importance of maintaining a high index of suspicion in patients with risk factors, even when only partial neurological signs are present.

Paraclinical investigations provide limited support for diagnosis. Brain MRI, although highly specific when characteristic lesions are observed, lacks sensitivity, particularly in early or non-alcoholic cases. In our cohort, MRI confirmed the diagnosis in only one patient, illustrating its limited diagnostic yield. Similarly, EEG findings were largely nonspecific and did not contribute significantly to diagnostic decision-making.

The cornerstone of management is the early administration of high-dose intravenous thiamine. Our results demonstrate that prompt treatment leads to rapid clinical improvement in most cases, particularly regarding oculomotor abnormalities and cognitive impairment. However, delayed diagnosis may result in persistent neurological deficits, as observed in some patients in our study. These findings are consistent with the literature, which emphasizes the risk of progression to chronic cognitive disorders, including Korsakoff syndrome, in the absence of timely treatment.

From a clinical perspective, the threshold for initiating thiamine therapy should be low in at-risk populations. Given its safety, low cost, and potential to prevent severe complications, empirical treatment is strongly recommended when WE is suspected.

This study has several limitations, including its retrospective design, small sample size, and the absence of biological confirmation of thiamine deficiency. Nevertheless, it reflects real-world clinical practice and underscores the diagnostic challenges associated with this condition.

CONCLUSION

Wernicke's encephalopathy should be systematically considered in non-alcoholic patients with gastrointestinal diseases and malnutrition. Because clinical presentation is often incomplete and diagnostic tests may be incnclusive, the diagnosis relies primarily on clinical suspicion.

Early administration of intravenous thiamine is essential to prevent irreversible neurological damage and improve patient outcomes.

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