# Wernicke's encephalopathy following tirzepatide therapy in a non-alcoholic patient with autoimmune hepatitis/primary biliary cholangitis and diabetes: a case report

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## Background

Wernicke's encephalopathy (WE) is a neurological emergency most commonly associated with chronic alcohol misuse. However, it can also develop in individuals who are malnourished for reasons unrelated to alcohol, where timely diagnosis is often overlooked. WE has also been reported following bariatric surgeries such as Roux-en-Y gastric bypass and sleeve gastrectomy, particularly in patients with persistent vomiting and reduced oral intake. These procedures may increase risk by bypassing portions of the proximal small intestine, including the duodenum and jejunum, which are important sites of thiamine absorption.[1,2] GLP-1/GIP receptor agonists are increasingly used in the management of diabetes and obesity. Although highly effective, these agents commonly cause appetite suppression and vomiting, which may predispose susceptible individuals to nutritional deficiencies and their associated complications.

## Case presentation

A 49-year-old woman with a complex medical history, including type 2 diabetes (T2DM), overlap syndrome of autoimmune hepatitis (AIH) and primary biliary cholangitis (PBC), and iron-deficiency anaemia was treated with tirzepatide. Following an increase in her tirzepatide dosage, she began experiencing persistent vomiting and marked appetite suppression. She was subsequently admitted to hospital with vomiting and ketosis. During her hospital stay, she developed progressive neurological symptoms including drowsiness, confusion,

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**Timeline of clinical events**

| Date / Period | Event |
|---|---|
| 2004–2021 | Diagnosis of T2DM (2004), initiation of insulin therapy (2008), diagnosis of PBC (2015), diagnosis of AIH (2021), background retinopathy identified (2008) |
| 2016 and 2019 | Two documented self-reported episodes of poor oral intake and vomiting. Gastroparesis was ruled out. Further details, including blood test results for these episodes, are not available |
| 10 Sep 2018 | Liraglutide initiated |
| 1 Aug 2019 | Semaglutide initiated |
| March 2022 | Admission for diabetic ketoacidosis (DKA) and vomiting; gastroparesis excluded. Further details, including blood test results for this episode, are not available |
| Dec 2022 – May 2023 | GLP-1 therapy restarted. Semaglutide was switched to exenatide due to a national supply shortage |
| June 2024 – March 2025 | Initiation and escalation of tirzepatide up to 10 mg as per recommended protocol every four weeks |
| April 2025 | Tirzepatide discontinued due to ongoing nausea |
| May 2025 | Severe vomiting and development of starvation ketosis, resulting in hospital admission. Endoscopy normal |
| 19 June 2025 | Re-admitted with vomiting and borderline DKA |
| 20 July 2025 | Inpatient mental health team involved to assess for eating disorders (including avoidant/restrictive food intake disorder, ARFID) |
| | Ferritin (190 µg/L), folate (3.5 µg/L) and vitamin B12 (242 pmol/L) |
| | CT abdomen and pelvis excluded intestinal obstruction. CT head normal. MRI showed T2 signal changes in the periaqueductal grey, thalamus and tectum, which are classical features of Wernicke's encephalopathy; thiamine treatment was started |
| 13 August 2025 | Follow-up MRI demonstrated resolution of previous abnormalities |
| October–November 2025 | Inpatient rehabilitation led to improvements in cognition and balance, and ability to perform activities of daily living. Patient was discharged home |
| 2 December 2025 | Bilateral optic neuropathy secondary to Wernicke's encephalopathy diagnosed by ophthalmology |

difficulty walking, nystagmus and visual disturbances.

An MRI scan revealed characteristic signal changes consistent with WE, and she showed neurological improvement after commencing thiamine therapy. Despite early treatment ophthalmological assessment later revealed bilateral optic neuropathy. Psychiatric evaluation was conducted due to concerns regarding avoidant/restrictive intake patterns, but the patient did not report any psychological or emotional factors related to eating and opted not to pursue further mental health follow-up. Through a multidisciplinary inpatient rehabilitation programme, she experienced improvements in cognition, mobility and nutritional status.

## Discussion

This case highlights the growing safety considerations associated with GLP-1/GIP receptor agonists and the potential risk of non-alcoholic Wernicke's encephalopathy in patients with multiple predisposing factors. While the temporal relationship with tirzepatide suggests a possible association, the presentation is likely multifactorial. The patient had a history of recurrent vomiting and probable underlying nutritional vulnerability, which may have contributed to the development of thiamine deficiency. These pre-existing factors, together with reduced oral intake, are likely to have increased the risk of Wernicke's encephalopathy. Recent pharmacovigilance analyses have identified a safety signal for Wernicke's encephalopathy with GLP-1 receptor agonists, including tirzepatide. Data from the FDA Adverse Event Reporting System (FAERS) indicate that Wernicke's encephalopathy was disproportionately reported in association with semaglutide, tirzepatide and the GLP-1 receptor agonist class as a whole.[3,4] Among reported cases, 68% occurred alongside symptoms such as nausea, vomiting or reduced food intake, with weight loss ranging from 3.5 to 13.3 kg per month over three to six months.[3]

While bariatric surgery protocols mandate structured nutritional monitoring, there are currently no equivalent frameworks in place for those receiving GLP-1RA therapy. Individuals using GLP-1 agonists for obesity management experience caloric reductions of 16%–39%.[5] Such large, rapid reductions can result in inadequate intake of essential vitamins and minerals, particularly when energy intake falls below 1,200 kcal per day for females and 1,800 kcal per day for males.[6] Observational studies have shown that those on GLP-1 therapy may develop nutritional deficiencies within 12 months, most frequently vitamin D, followed by thiamine and other B vitamins.[7] A recent narrative review including 480,825 adults reported that micronutrient deficiencies may occur in patients receiving GLP-1 receptor agonist therapy. Vitamin D deficiency was the most common abnormality, affecting 7.5% of patients at six months and 13.6% at 12 months. Iron depletion was also frequently observed, with GLP-1 receptor agonist users demonstrating 26–30% lower ferritin levels compared with SGLT2 inhibitor users. Protein and calcium insufficiency were associated with lean mass loss, and deficiencies in thiamine and cobalamin increased over time.[8]

### Key messages

- Wernicke's encephalopathy can occur in non-alcoholic patients who experience prolonged vomiting and malnutrition
- GLP-1/GIP receptor agonists, such as tirzepatide, may contribute to nutritional deficiencies by suppressing appetite in susceptible individuals
- Early recognition and prompt thiamine treatment are crucial, as delays may lead to irreversible neurological complications, including optic neuropathy
- Comprehensive medication reviews and holistic assessments are essential in patients with complex multimorbidity to identify evolving risks and prevent adverse outcomes

Wernicke's encephalopathy is a medical emergency requiring prompt empirical thiamine therapy. Early thiamine supplementation should be considered in high-risk patients presenting with prolonged vomiting, significant weight loss or neurological symptoms even prior to imaging or laboratory confirmation. Empirical treatment dose is 500 mg intravenous thiamine (dissolved in 100 mL normal saline) infused over 30 minutes, three times daily for 2-3 days.[9] In such cases, intravenous glucose should not be administered without concurrent thiamine replacement. IV glucose may aggravate acute thiamine deficiency and Wernicke's encephalopathy.[10]

Optic neuropathy is a rare but well-documented sequela of Wernicke's encephalopathy. Classic ophthalmic manifestations include nystagmus, ophthalmoplegia and conjugate gaze palsies (occurring in approximately 29% of patients), but bilateral visual disturbances with optic disc oedema and retinal haemorrhages can be presenting features.[9]

## Patient perspective

The patient described her experience as frightening, especially given the sudden onset of cognitive and visual symptoms. She felt that her initial concerns were not fully acknowledged, resulting in distress due to delays in diagnosis. Nevertheless, she expressed gratitude for the support and care provided by the rehabilitation team. She continues to adapt to her ongoing visual impairment, reliance on walking aids, and difficulties with delayed memory recall. Motivated by her experiences, she is keen to raise awareness about the nutritional risks linked to medications such as tirzepatide.

## Conclusion

This case highlights the potential for serious nutritional and neurological complications associated with GLP-1/GIP therapies, even in those without a history of alcohol misuse. Prompt recognition of Wernicke's encephalopathy and admission to

hospital for swift administration of thiamine are essential. The management of such complex cases involving chronic diseases necessitates a multidisciplinary approach.



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**Patient consent** Written informed consent was obtained from the patient for the publication of this case report and any accompanying images or clinical information.

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