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A Fatal Case of Wernicke's Encephalopathy Attributed to Intractable Emesis During Tirzepatide Initiation

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Patient: Male, 69-year-old
Final Diagnosis: Wernicke's encephalopathy
Symptoms: Emesis • loss of consciousness
Clinical Procedure: —
Specialty: General and Internal Medicine
Objective: Unusual clinical course

Background: Tirzepatide is a once-weekly injectable therapy for type 2 diabetes mellitus and obesity that acts as a dual glucagon-like peptide-1 (GLP-1) and glucose-dependent insulinotropic polypeptide receptor agonist. This medication is commonly used for weight loss, but gastrointestinal adverse effects are common among patients receiving tirzepatide. Intractable emesis is among several conditions that can lead to thiamine deficiency and subsequent Wernicke's encephalopathy (involving ophthalmoplegia, ataxia, and confusion). Recent studies have suggested an association between Wernicke's encephalopathy and GLP-1 receptor agonists; causality has not been established.

Case Report: A 69-year-old man experienced months of intractable emesis after initiation of tirzepatide therapy. His clinical course progressed to a reduced level of consciousness, requiring intubation for airway protection. Brain magnetic resonance imaging (MRI) subsequently revealed findings consistent with Wernicke's encephalopathy. The patient's mental status failed to improve despite high-dose intravenous thiamine replacement, and extensive diagnostic evaluation failed to identify an alternative cause of his encephalopathy. The patient ultimately died while receiving hospice care.

Conclusions: This patient experienced intractable emesis after initiation of tirzepatide therapy and subsequently developed Wernicke's encephalopathy attributed to thiamine deficiency. His case underscores the importance of close monitoring during treatment initiation to identify severe adverse effects and potentially harmful dietary changes. The patient's imaging findings demonstrate severe neurological damage that can occur with prolonged Wernicke's encephalopathy. Prompt diagnosis and treatment with high-dose intravenous thiamine are necessary to prevent progression to irreversible neurological deficits or death. Wernicke's encephalopathy was diagnosed based on the clinical course and characteristic MRI findings; biochemical thiamine deficiency was not independently confirmed.

Keywords: Fatal Outcome • Tirzepatide • Wernicke Encephalopathy
Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/954396

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Introduction

Glucagon-like peptide-1 (GLP-1) receptor agonists are rapidly gaining popularity as an adjunctive treatment for type 2 diabetes mellitus and obesity. Tirzepatide is a commonly used agent that acts as a dual GLP-1 and glucose-dependent insulinotropic polypeptide receptor agonist; it is typically administered as a once-weekly injection. Both hormones are considered nutrient-stimulated hormones released by the intestines in response to nutrients. They affect multiple organs involved in metabolic and gastrointestinal regulation, including the gastrointestinal tract, pancreas, central nervous system, kidneys, heart, and vasculature [1].

When GLP-1 and glucose-dependent insulinotropic polypeptide receptors are stimulated by these hormones or agonists such as tirzepatide, insulin levels increase, glucagon levels decrease, and gastric emptying slows [1]. Tirzepatide has been approved by the US Food and Drug Administration (FDA) for both diabetes management and weight loss in patients with obesity. Studies have also demonstrated potential benefits for patients exhibiting heart failure with preserved ejection fraction [1]. Tirzepatide has been shown to significantly reduce body weight and hemoglobin A1c levels; in some cases, it has been associated with diabetes remission that persists after treatment discontinuation [2].

An analysis of online medication reviews indicated that around 62% of patients taking GLP-1 receptor agonists experience gastrointestinal adverse effects, such as nausea, vomiting, constipation, diarrhea, or loss of appetite [3]. Approximately 12% of tirzepatide users experience vomiting, an adverse effect that becomes more common at higher doses [4]. Such effects are generally attributed to delayed gastric emptying caused by these medications [1]. Although usually mild to moderate, the effects can occasionally be severe enough to require treatment discontinuation [5]. One study demonstrated that nausea and vomiting were responsible for up to 43.8% of treatment discontinuations [6]. However, among patients who continue taking GLP-1 receptor agonists, overall satisfaction with treatment is similar between those who experience gastrointestinal adverse effects and those who do not [3].

Prolonged intractable emesis can adversely affect nutrition in multiple ways, including the development of thiamine deficiency. Thiamine stores can be depleted in as little as 2 weeks of inadequate intake [7]. This vitamin is essential for cerebral metabolism, and deficiencies initially affect areas with high metabolic demands, including the mammillary bodies, medial thalami, and periaqueductal gray matter [7]. Wernicke's encephalopathy is a devastating consequence of thiamine deficiency, characterized by the classic triad of ophthalmoplegia, ataxia, and confusion. Rarely, patients may present with hypotension or a reduced level of consciousness [7]. Diagnosis is typically based on clinical presentation, although symptoms can vary; approximately 16% of patients present with all 3 components of the classic triad [7]. This condition is usually reversible with aggressive thiamine replacement, but neuropsychiatric symptoms can persist in many cases [7].

Wernicke's encephalopathy is frequently associated with alcohol use, but many other conditions can cause nutritional deficits and subsequent thiamine deficiency. This association contributes to the underdiagnosis of nonalcoholic cases, and autopsy studies suggest that only 16% of nonalcoholic cases of Wernicke's encephalopathy are identified before death [7].

There is increasing documentation of a rare association between GLP-1 receptor agonist use and Wernicke's encephalopathy. An analysis of the FDA Adverse Event Reporting System database from 2005 to 2024 identified 28 953 neurological adverse events associated with GLP-1 receptor agonist use, of which 14 were classified as Wernicke's encephalopathy [8]. A more comprehensive literature review identified 14 cases of Wernicke's encephalopathy associated with GLP-1 receptor agonist use from 2023 to 2024, predominantly involving semaglutide or tirzepatide [9]. Although such findings suggest a possible association between Wernicke's encephalopathy and GLP-1 receptor agonist use, they do not establish causality or fully characterize the severity and outcomes of these cases.

Case Report

A 69-year-old man had a medical history of heart failure with preserved ejection fraction, hypertension, hyperlipidemia, and type 2 diabetes mellitus; he presented to the emergency department with generalized weakness and dizziness. He had begun tirzepatide injections 2 months before presentation and almost immediately developed severe nausea, loss of appetite, and emesis. He continued the medication despite these intractable adverse effects because he was satisfied with his weight loss, reporting that he had lost 13 lbs in the first 9 days, although he could not recall his starting weight.

He completed the first month of 2.5-mg tirzepatide injections and received at least one 5-mg dose before discontinuing tirzepatide 3 weeks prior to presentation because of lightheadedness and persistent emesis. He had discontinued the medication independently without discussing these symptoms with his primary care physician. He had no history of gastrointestinal disorders and denied alcohol, tobacco, or illicit drug use. His family confirmed that he did not consume alcohol, and a thorough chart review revealed no evidence of prior alcohol use. He had maintained a normal, well-balanced diet before symptom onset. His family medical history was unremarkable.

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His other home medications included metoprolol succinate, losartan, metformin, and a statin; there were no other recent medication changes.

Upon initial presentation to the emergency department, he had mild bradycardia (46 beats/min), with otherwise normal vital signs. He weighed 280 lbs (127 kg) and displayed a body mass index of 35.18 kg/m^2. Physical examination findings were unremarkable except for mild diffuse abdominal tenderness. He had no focal neurological deficits and responded appropriately to orientation questions but exhibited mild confusion, including an inability to recall when he had discontinued tirzepatide or his weight before treatment initiation. Laboratory findings were notable for mild leukocytosis (11 000 cells/µL; reference range, 4500-11 000 cells/µL) with neutrophilic predominance and anion gap metabolic acidosis; the bicarbonate level was 17 mmol/L (reference range, 23-29 mmol/L) and anion gap was 13 mmol/L (reference range, 4-12 mmol/L). His blood glucose level was within normal limits, and his beta-hydroxybutyrate level was 1.0 mmol/L (reference range, < 0.5 mmol/L).

His symptoms were attributed to starvation ketosis and dehydration resulting from persistent gastrointestinal adverse effects after the initiation of GLP-1 receptor agonist therapy, based on the timing of symptom onset; causality could not be established. He was treated with gentle intravenous fluid resuscitation, oral nutritional supplements, and antiemetic therapy. His symptoms improved, and he was able to tolerate a regular diet. Further gastrointestinal testing to rule out alternative causes of persistent vomiting was not performed because his symptoms had resolved. After a 1-week-long hospitalization, he was discharged to a skilled nursing facility.

Two weeks later, he returned to the emergency department from the nursing facility with new-onset left-sided facial droop, leftward gaze deviation, and a reduced level of consciousness; his Glasgow Coma Scale (GCS) score was 6. He underwent emergency intubation for airway protection and was admitted to the intensive care unit. He was hypotensive on admission, with a blood pressure of 74/48 mm Hg, which required vasopressor support with a norepinephrine infusion.

Initial laboratory findings during his second hospitalization revealed leukocytosis (19 400 cells/µL); hypernatremia (153 mmol/L; reference range, 135-145 mmol/L); acute kidney injury with a creatinine level of 5.49 mg/dL (reference range, 0.6-1.1 mg/dL); and mildly elevated liver enzymes, with an aspartate aminotransferase level of 46 U/L (reference range, 8-48 U/L) and an alanine aminotransferase level of 112 U/L (reference range, 7-55 U/L). He had recurrent anion gap metabolic acidosis, with a bicarbonate level of 13 mmol/L and an anion gap of 20 mmol/L. His lactate level was normal, and his beta-hydroxybutyrate level was 1.4 mmol/L. His initial high-sensitivity troponin level was elevated at 138 ng/L (reference range, ≤ 22 ng/L in men), followed by 126 ng/L, which was attributed to demand ischemia.

Computed tomography scans revealed no acute intracranial, thoracic, abdominal, or pelvic abnormalities. Continuous electroencephalographic monitoring for 24 hours revealed no seizure activity. Intravenous cefepime was initiated pending an infectious workup, which ultimately revealed no evidence of respiratory or urinary tract infection, and blood culture findings were negative. A lumbar puncture was performed, and cerebrospinal fluid analysis results were unremarkable, with negative infectious and autoimmune evaluation findings.

Brain magnetic resonance imaging (MRI) revealed abnormal diffusion and T2 signal intensity involving medial portions of the thalami, hypothalamus, periaqueductal gray matter, and mammillary bodies (**Figure 1**; diffusion-weighted images not shown). Combined with the patient's reduced level of consciousness and recent nutritional deficiency resulting from intractable emesis, these MRI findings supported a diagnosis of Wernicke's encephalopathy.

The decision was made to initiate emergency treatment for Wernicke's encephalopathy without first measuring whole-blood thiamine levels or erythrocyte transketolase activity because the results would have taken several days to obtain. The patient immediately received high-dose intravenous thiamine replacement at 400 mg every 8 hours for 3 days, followed by thiamine tablets administered through a nasogastric tube for 7 days. His hypotension resolved, and he no longer required vasopressor support. His electrolyte abnormalities resolved with dialysis, which was subsequently discontinued as his renal function recovered. Unfortunately, the patient's level of consciousness did not improve despite thiamine replacement, and his GCS score remained at 6T (E1, verbal not testable [VNT], M4). Repeat brain MRI with and without contrast, performed 5 days after the initial scan, demonstrated differential anatomical progression characterized by near-complete resolution of hyperintensities in the periaqueductal gray matter and superior colliculi, accompanied by early subacute mammillary body atrophy with mild residual hyperintensity, without other interval abnormalities (**Figure 2**; diffusion-weighted images not shown).

The patient remained unable to protect his airway because of persistent neurological dysfunction and would have required prolonged mechanical ventilation. After 11 days in the intensive care unit and extensive goals-of-care discussions with the patient's family, the family elected to proceed with inpatient hospice care because the patient would not have wanted a tracheostomy for prolonged ventilatory support. He underwent palliative extubation and died peacefully the following day.

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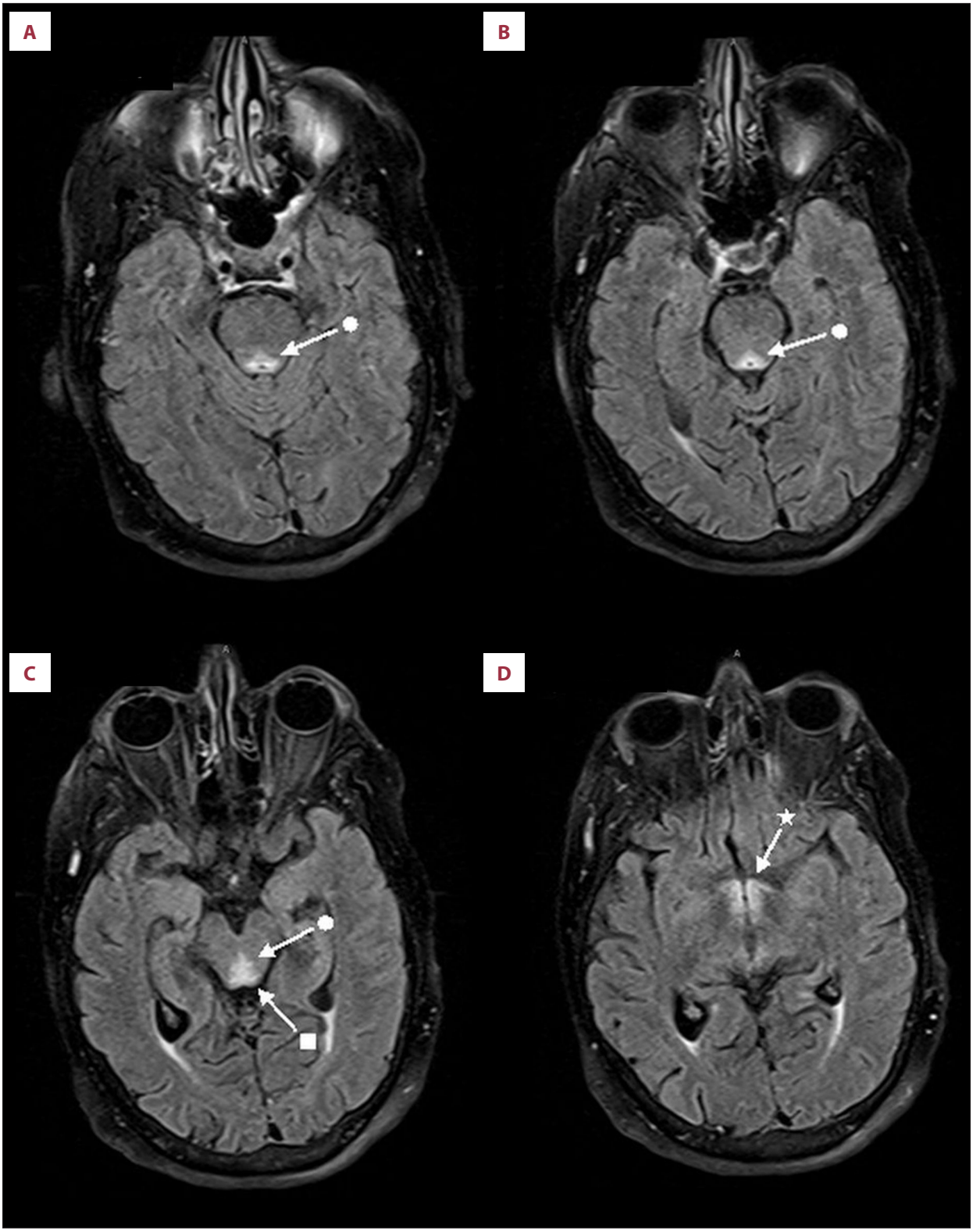
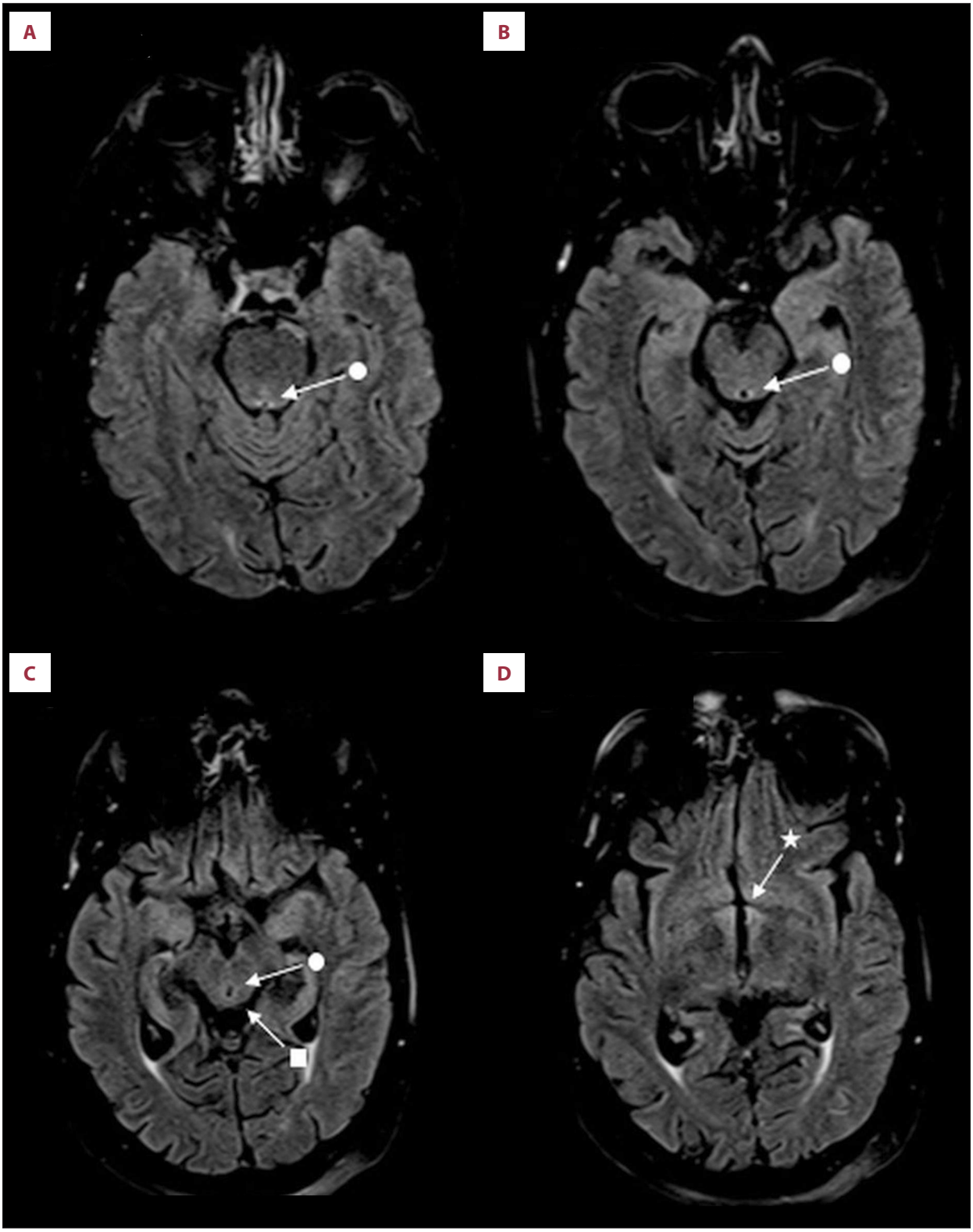


Figure 1. Initial brain magnetic resonance imaging. Ascending axial fluid-attenuated inversion recovery (FLAIR) brain magnetic resonance imaging slices demonstrating bilateral, symmetric hyperintensities in the periaqueductal gray matter (circles in **A-C**), superior colliculi (square in **C**), and mammillary bodies (star in **D**), consistent with Wernicke's encephalopathy.

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Figure 2. Follow-up brain magnetic resonance imaging. Ascending axial fluid-attenuated inversion recovery (FLAIR) brain magnetic resonance imaging slices obtained 5 days after the initial scan (Figure 1), demonstrating near-complete resolution of the hyperintensities in the periaqueductal gray matter (circles in **A-C**) and superior colliculi (square in **C**), along with the development of mammillary body atrophy and mild improvement in hyperintensity (star in **D**).

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Discussion

Our patient experienced severe nutritional deficiencies associated with tirzepatide initiation, culminating in fatal Wernicke's encephalopathy. This case provides an opportunity to examine the subtle early signs of Wernicke's encephalopathy in a patient with nutritional deficits and the progression to possibly permanent neurological injury, as reflected by changes on serial brain MRI scans.

Wernicke's encephalopathy can be challenging to diagnose because symptoms are highly variable, and most patients do not present with the classic triad that many physicians expect [7]. Our patient initially exhibited mild confusion, as reflected by an inconsistent timeline regarding when he had discontinued tirzepatide and an inability to recall his starting weight. His atypical presentation delayed diagnosis for 3 weeks until his second admission, when he presented with more characteristic symptoms and MRI findings consistent with Wernicke's encephalopathy. This condition should be considered in any patient with suspected nutritional deficits because thiamine deficiency can develop in as little as 2 weeks of inadequate thiamine intake, and 19% of patients exhibit none of the symptoms of the classic triad [7]. Notably, our patient's Wernicke's encephalopathy was diagnosed based on the clinical course and characteristic MRI findings. However, whole-blood thiamine levels and erythrocyte transketolase activity were not measured; therefore, biochemical thiamine deficiency was not independently confirmed.

Although causality is difficult to establish from a single case, our patient's nutritional deficiency was likely attributable to the months of frequent, intractable emesis he experienced after tirzepatide initiation. Limited data are available regarding the duration of gastrointestinal adverse effects after tirzepatide discontinuation. However, based on tirzepatide's half-life of 5 days, approximately 6% of the patient's last dose would have remained after 3 weeks, potentially explaining the persistence of his symptoms after treatment discontinuation [10].

The present case exemplifies the willingness of some individuals to tolerate serious adverse effects in pursuit of weight loss, amid a concerning trend of disordered eating and unhealthy weight loss among GLP-1 receptor agonist users [11]. Clinicians prescribing GLP-1 receptor agonists should advocate for healthy weight loss strategies and consider discontinuing treatment in patients who exhibit excessive weight loss or inadequate nutrition. These medications offer numerous benefits for patients with obesity, but close monitoring during treatment initiation is essential to identify intolerable gastrointestinal symptoms and unhealthy dietary habits, while preventing severe nutritional deficiencies and complications such as Wernicke's encephalopathy.

Although other manifestations of thiamine deficiency can occur with more prolonged deficiency, our patient may have developed only Wernicke's encephalopathy because of his relatively short illness duration. Thiamine deficiency initially affects areas with the highest thiamine requirements, primarily including the brain regions involved in Wernicke's encephalopathy [12]. Had his deficiency progressed without treatment or death, he might have subsequently developed cardiovascular manifestations of thiamine deficiency, including pulmonary hypertension and right-sided heart failure [12].

In the present case, the diagnosis of Wernicke's encephalopathy was ultimately supported by brain MRI findings (**Figure 1**). Thiamine is essential for cerebral metabolism; deficiency initially affects areas of high metabolic activity, including the mammillary bodies, anterior and medial thalamic nuclei, periaquaductal gray matter, and inferior and superior colliculi and, rarely, the cerebellum and pons [3,9]. In Wernicke's encephalopathy, these regions become edematous and exhibit bilateral, symmetric hyperintensities on T2-weighted and fluid-attenuated inversion recovery (FLAIR) images [13]. These MRI findings have low sensitivity (53%) but high specificity (93%) for Wernicke's encephalopathy, making them useful support for the diagnosis when present [13]. Furthermore, the extent of abnormalities visible on brain MRI is correlated with disease severity. Patients with more severe neurological symptoms are more likely to exhibit neuroradiological evidence of Wernicke's encephalopathy, whereas those without MRI abnormalities often have a better prognosis [13]. Our patient's clinical course was consistent with this pattern, encompassing severe neurological symptoms, prominent neuroradiological findings, and a poor clinical outcome.

Substantial evidence indicates that timely thiamine replacement is necessary to improve the likelihood of recovery from Wernicke's encephalopathy without permanent neurological deficits [14]. After the acute phase of Wernicke's encephalopathy, characterized by hyperintensities in various cerebral structures, subsequent MRI scans have demonstrated atrophy, particularly in the mammillary bodies and thalamus [13]. This atrophy has most frequently been observed in patients with persistent neurological deficits after Wernicke's encephalopathy and represents irreversible brain damage caused by the condition [13]. The second brain MRI in the present case further supported this association (**Figure 2**). Although hyperintensities associated with edema had improved, the scan revealed early atrophy of the mammillary bodies, suggesting that the patient's neurological deficits may have been permanent. This atrophy might help explain the persistence of his neurological symptoms, which ultimately preceded his death.

Resumption of an adequate diet after his first admission failed to resolve our patient's thiamine deficiency. Intestinal

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absorption of thiamine is substantially lower than the amounts provided by high-dose intravenous thiamine therapy recommended for patients with Wernicke's encephalopathy. One study demonstrated that for any oral dose exceeding 30 mg, only approximately 4.5 mg of thiamine is absorbed by the intestines [15]. There are no clear guidelines regarding the optimal thiamine dose for patients with Wernicke's encephalopathy; however, available sources consistently recommend high-dose intravenous or intramuscular thiamine replacement. Specific recommendations range from 200 mg twice daily to 500 mg three times daily [7,15]. The duration of treatment is similarly uncertain; many sources recommend at least 3 days of intravenous or intramuscular therapy, whereas others advise continuing thiamine replacement until the patient demonstrates clinical improvement [7]. Despite variation in terms of recommendations, intravenous or intramuscular thiamine replacement is consistently recommended for patients with Wernicke's encephalopathy because oral thiamine is insufficient to rapidly replenish thiamine stores and treat this condition. The consequences of delayed thiamine replacement were evident in the present case: persistent severe neurological impairment, palliative extubation, and death the following day.

Existing literature suggests a rare association between Wernicke's encephalopathy and GLP-1 receptor agonist use based on large adverse event reporting databases but provides limited information regarding the severity and outcomes of these events. The present case provides another example of Wernicke's encephalopathy associated with tirzepatide use, although it does not establish causality. Notably, it illustrates how this typically reversible condition can progress to permanent neurological injury and subsequent death, with serial brain MRI findings documenting disease progression.

Conclusions

This case highlights the need for heightened vigilance during tirzepatide initiation in patients experiencing severe emesis, loss of appetite, rapid weight loss, confusion, or other signs of possible nutritional deficiencies. Prolonged malnutrition resulting from conditions such as the intractable emesis experienced by our patient increases the risk of Wernicke's encephalopathy. Early diagnosis and prompt intravenous thiamine replacement are essential for patients with Wernicke's encephalopathy to prevent severe neurological damage, progression to irreversible atrophy, permanent neurological deficits, or death.

Institution Where Work Was Done

Orlando Regional Medical Center, Orlando, FL, USA.

Patient Consent

The patient provided informed consent for the publication of this report.

AI Declaration

This manuscript was created and revised without the use of AI by any authors.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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References:

1. Galindo RJ, Cheng AYY, Longuet C, et al. Insights into the mechanism of action of tirzepatide: a narrative review. Diabetes Ther. 2026;17:19-40
2. Sawamura T, Ohmori A, Kometani M, et al. Two cases of diabetes remission through temporary tirzepatide treatment. Intern Med. 2026;65(5):666-72
3. Armanious A, Hunter RM, Griffiths K, et al. Patient perceptions of Ozempic® (semaglutide) for weight loss: A mixed methods analysis of online medication reviews. J Med Internet Res. 2026;28:e78391
4. Alhazmi A, le Roux CW. Do no harm: Managing nausea and vomiting in GLP-1 based obesity therapies. Front Endocrinol (Lausanne). 2026;17:1788698
5. Collins L, Costello RA. Glucagon-like peptide-1 receptor agonists. [Updated 2024 Feb 29]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026. Available from: https://www.ncbi.nlm.nih.gov/books/NBK551568/
6. Ceriello A, Prattichizzo F, Mastan Sheik Abdullah AR, et al. Causes and consequences of discontinuation of GLP1RAs or tirzepatide. Nat Rev Endocrinol. 2026;22(9):530-42
7. Singh J, Regina AC. Wernicke encephalopathy. [Updated 2026 Jan 10]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026. Available from: https://www.ncbi.nlm.nih.gov/books/NBK470344/
8. Chen H, Liu S, Gao S, et al. Pharmacovigilance analysis of neurological adverse events associated with GLP-1 receptor agonists based on the FDA Adverse Event Reporting System. Sci Rep. 2025;15:18063
9. Lev D, Leibowitz A, Lang A, et al. Glucagon-like peptide-1 receptor agonists and Wernicke encephalopathy: A pharmacovigilance study and literature review. Clin Nutr. 2026;57:106571
10. Chavda VP, Ajabiya J, Teli D, et al. Tirzepatide, a new era of dual-targeted treatment for diabetes and obesity: A mini-review. Molecules. 2022;27(13):4315
11. Dushay J. My biggest GLP-1 ethical problem: patients who don't want to stop. STAT [Internet]. 2026 [cited 2026 Jun 1]. Available from: https://www.statnews.com/2026/02/19/glp-1-weight-loss-drugs-eating-disorders-doctor/
12. Smith TJ, Johnson CR, Koshy R, et al. Thiamine deficiency disorders: A clinical perspective. Ann N Y Acad Sci. 2021;1498(1):9-28
13. Sullivan EV, Pfefferbaum A. Neuroimaging of the Wernicke-Korsakoff syndrome. Alcohol Alcohol. 2009;44:155-65
14. Thomson AD, Guerrini I, Marshall EJ. The evolution and treatment of Korsakoff's syndrome: Out of sight, out of mind? Neuropsychol Rev. 2012;22:81-92
15. Nakamura ZM, Tatreau JR, Rosenstein DL, Park EM. Clinical characteristics and outcomes associated with high-dose intravenous thiamine administration in patients with encephalopathy. Psychosomatics. 2018;59:379-87

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