Introduction

Wernicke encephalopathy (WE) is a syndrome that arises from thiamine (vitamin B1) deficiency and is typically characterized by nystagmus, ataxia, and confusion, with at least two of these characteristics being present [1]. Magnetic resonance imaging (MRI) is not required for the diagnosis of WE, as it has been shown to have an estimated sensitivity of 53% in revealing WE [2]. WE and thiamine deficiency are commonly known as complications of chronic alcoholism. However, non-alcoholic causes are becoming more recognized, including hyperemesis gravidarum, chemotherapy-induced hyperemesis, and bariatric surgery [3]. WE after bariatric surgery has been shown to present with atypical neurological features, outside the classic triad, including third and sixth cranial nerve palsies [4]. However, hearing loss is considered rare [3,4].

In this paper we describe the case of a 28-year-old female who developed sensorineural hearing loss (SNHL) in the setting of thiamine deficiency and acute WE after bariatric surgery, requiring hearing aids despite thiamine replacement.

Case Report

A 28-year-old female presented to a clinic with difficulty hearing, which had been ongoing without change for one year since her hospitalization for hyperemesis status three months post-sleeve gastrectomy. She had no history of ototoxic medications, auditory or physical trauma, or ear surgery, no recurrent history of otitis media or congenital anomalies, and no family history of early-onset hearing loss. During her hospitalization one year prior to visiting the clinic, she was given intravenous (IV) 5% dextrose in half-normal saline due to her five-day history of hyperemesis and starvation keto. On day four of hospitalization, she developed new-onset diplopia and gait ataxia. Her exam was significant for horizontal nystagmus and ataxic gait, including difficulty with tandem walking. Her brain MRI was normal. Laboratory workup revealed a thiamine deficiency (59 nmol/L, with the normal reference range being 70–180 nmol/L).

Having met the diagnostic criteria, a clinical diagnosis of WE was established [1]. Treatment with IV thiamine was initiated (500 mg infused over 30 minutes three times daily for three days followed by 250 mg once daily for an additional five days). Her nystagmus and ataxia improved the follow-
The patient was fitted with bilateral hearing aids and subjective improvement in symptomatic hearing loss was re-

![Fig. 1. Audiogram showing mild and slight sensorineural hearing loss in the right and left ear, respectively, status one year post-acute Wernicke encephalopathy and thiamine repletion.](image-url)
ported. Given her unremarkable personal and family history, genetic testing was deferred. She continued with routine vitamin supplementation and close monitoring and her thiamine levels remained within normal limits.

**Discussion**

Sensorineural hearing loss has been attributed to various types of vitamin deficiencies, such as vitamin D [5]. However, it is rarely associated with thiamine deficiency and it is generally not considered a syndrome of WE. Nevertheless, there is a growing number of case reports describing hearing loss associated with WE. Our literature review yielded ten case reports of SNHL occurring with WE prior to thiamine repletion (Table 1). The SNHL resolved within two weeks or less after thiamine replacement treatment in 4/10 cases [3,6-8], improved but persisted within three months or less in 5/10 cases [9-13], and was not reported in one case [14]. Only 4/10 cases performed an audiogram at the time of presentation, all showing moderate SNHL [3,10,12,13], and a follow-up audiogram was performed after thiamine replacement in only one case. Nakamura, et al. [12] presented a moderate SNHL which improved to a mild SNHL status three months post-thiamine repletion. None of the cases reported DPOAEs. However, Prosperini, et al. [13] did report on brainstem auditory-evoked response testing that demonstrated bilateral prolonged I–III interpeak latencies, elicited by auditory stimuli. In these ten reports, the hearing loss accompanied by WE recovered relatively rapidly after thiamine repletion. Our case reports hearing loss occurring after WE and ongoing SNHL at one-year follow-up after acute WE despite standard IV thiamine therapy and confirmation of serum thiamine repletion. Although the hearing impairment of our 28-year-old patient was classified by audiogram as mild sensorineural hearing loss, it was still significant enough for her to find that the bilateral hearing aid devices improved her quality of life.

Bariatric surgery increases the risk of thiamine deficiency, as thiamine absorption primarily occurs in the proximal small intestine. Thiamine is a coenzyme required by most cells for energy production in the form of adenosine triphosphate (ATP). When glucose is administered prior to thiamine repletion, ATP cannot be generated through the Krebs cycle, causing an increase in lactic acid production and eventual cell death [15]. In mice, an auditory neuropathy phenotype was created by deleting the high-affinity thiamine transporter encoded by SLC19A2, which is expressed in the inner hair cells within the cochlea. Cochlear histological analysis showed selective loss of inner hair cells one to two weeks after a low thiamine diet and significantly greater inner than outer hair cell loss after longer low-thiamine challenges (lasting three

| Case            | Age | Sex | Risk factor for B1 deficiency          | B1 level | SNHL after B1 repletion | Audiogram PTA |
|-----------------|-----|-----|----------------------------------------|----------|-------------------------|---------------|
| Kondo, et al.   | 28  | F   | Gastric carcinoma, chemotherapy        | NR       | Resolved prior to hospital discharge | NR            |
| Buscaglia, et al. | 17  | F   | Hyperemesis gravidarm                   | NR       | Improved prior to hospital discharge | NR            |
| Foster, et al.  | 35  | F   | Gastric bypass                          | NR       | NR                      | NR            |
| Jethava, et al. | 44  | F   | Gastric bypass                          | 33 nmol/L| Improved at 8 week follow up | Moderate (40 dB R, 50 dB L) |
| Zhang, et al.   | 23  | M   | Acute pancreatitis, 1 month limited diet| NR       | Improved 2 days after treatment | NR            |
| Walker, et al.  | 61  | M   | Diabetic gastroparesis                  | 35 nmol/L| Resolved after 24 hours of treatment | Moderate (40 dB R, 42 dB L) |
| Moussa, et al.  | 49  | M   | Emesis 3 months, unknown cause           | 36 nmol/L| Resolved after 2 weeks | NR            |
| Nakamura, et al.| 61  | M   | Alcoholism, gastric resection for ulcer  | 33 nmol/L| Improved after 3 months | Moderate (40 dB R, 50 dB L) |
| Nguyen, et al.  | 35  | F   | Gastric sleeve                          | NR       | Resolved prior to hospital discharge | NR            |
| Prosperini, et al. | 27  | M   | Gastric sleeve                          | NR       | Improved prior to hospital discharge | Moderate (30 dB R, 50 dB L) |

B1: Thiamine, SNHL: sensorineural hearing loss, PTA: pure tone average, NR: not reported
weeks). When the mice on three-week low-thiamine challenge returned to a normal diet, slight improvements in average thresholds were noted after five weeks. However, the thresholds did not return to normal levels [15]. This mouse model suggests that hearing loss induced by thiamine deficiency may improve after the repletion of thiamine but it may not completely return to baseline. To help prevent thiamine deficiency, WE, and possibly irreversible hearing loss, it is vital that patients at risk of thiamine deficiency be repleted prior to initiation of infusions containing dextrose. It is important for providers to recognize non-alcoholic patients at high risk for thiamine deficiency, including those who have recently undergone bariatric surgery.

In the primary care setting, it is uncommon for young adults to endorse hearing loss. However, the number of patients undergoing bariatric surgery is growing. These patients are at an increased risk of vitamin deficiencies, along with their associated neurological and auditory complications, such as those occurring in WE. While some signs and symptoms resolve after immediate treatment, others, such as our patient’s sensorineural hearing loss, can persist. Primary care and other providers caring for patients after bariatric surgery should refer symptomatic patients for audiological evaluations so that any hearing impairment can be diagnosed and treated to help improve patient care and quality of life.

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Conflicts of interest
The authors have no financial conflicts of interest.

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REFERENCES
1) Caine D, Halliday GM, Kril JJ, Harper CG. Operational criteria for the classification of chronic alcoholics: identification of Wernicke’s encephalopathy. J Neurol Neurosurg Psychiatry 1997;62:51-60.
2) Antunez E, Estruch R, Cardenal C, Nicolas JM, Fernandez-Sola J, Urbano-Marquez A. Usefulness of CT and MR imaging in the diagnosis of acute Wernicke’s encephalopathy. AJR Am J Roentgenol 1998;171:1131-7.
3) Walker MA, Zepeda R, Afari HA, Cohen AB. Hearing loss in Wernicke encephalopathy. Neuro Clin Pract 2014;4:511-5.
4) Singh S, Kumar A. Wernicke encephalopathy after obesity surgery: a systematic review. Neurology 2007;68:807-11.
5) Büki B, Jünger H, Zhang Y, Lundberg YW. The price of immune responses and the role of vitamin d in the inner ear. Otol Neurotol 2019;40;701-9.
6) Kondo K, Fujimura M, Murase M, Koderia Y, Akiyama S, Ito K, et al. Severe acute metabolic acidosis and Wernicke's encephalopathy following chemotherapy with 5-fluorouracil and cisplatin: case report and review of the literature. Jpn J Clin Oncol 1996;26:234-6.
7) Moussa TD, Hamid A, Fatimata HD, Sofiane T. An unusual mode of revelation of Wernicke's encephalopathy: bilateral blindness with bilateral hypoacusia. Neuro India 2017;65:1406-7.
8) Nguyen JTT, Franconi C, Prentice A, Wycoo V. Wernicke encephalopathy hearing loss and palinacousis. Intern Med J 2019;49:536-9.
9) Buscaglia J, Faris J. Unsteady, unfocused and unable to hear. Am J Med 2005;118:1215-7.
10) Jethava A, Dasanu CA. Acute Wernicke encephalopathy and sensorineural hearing loss complicating bariatric surgery. Conn Med 2012;76:603-5.
11) Zhang SQ, Guan YT. Acute bilateral deafness as the first symptom of Wernicke encephalopathy. AJNR Am J Neuroradiol 2012;33:E44-5.
12) Nakamura T, Imai K, Hamanaka M, Yamazaki H, Yamada T, Mizuno T. A case of Wernicke encephalopathy with hypoacusia and MR high intensity of the inferior colliculi that normalized after thiamine. Rinsho Shinkeigaku 2018;58:100-4.
13) Prosperini L, Stasolla A, Greco G, Gerace C, Tortorella C. Non-alcoholic Wernicke encephalopathy presenting as bilateral hearing loss: a case report. J Neurol 2019;266:1027-30.
14) Foster D, Falah M, Kadom N, Mandler R. Wernicke encephalopathy after bariatric surgery: losing more than just weight. Neurology 2005;65;1847: discussion 1847.
15) Liberman MC, Tartaglini E, Fleming JC, Neufeld EJ. Deletion of SLC19A2, the high affinity thiamine transporter, causes selective inner hair cell loss and an auditory neuropathy phenotype. J Assoc Res Otolaryngol 2006;7:211-7.