

CASE REPORT



Wernicke's encephalopathy mimicking multiple sclerosis in a young female patient post-bariatric gastric sleeve surgery

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ABSTRACT

We describe a case of Wernicke's encephalopathy secondary to thiamine (B1) deficiency in a patient status post-bariatric sleeve gastrectomy. The presenting symptoms of new-onset weakness, diplopia, and confusion in a young female patient raised suspicion for multiple sclerosis (MS), but given a history of bariatric surgery, thiamine levels were checked, revealing significant Vitamin B1 (thiamine) deficiency. This case highlights the importance of thorough history taking, as a misdiagnosis of MS in this case could have resulted in irreversible neurological deterioration and hematological and infectious consequences associated with the inappropriate administration of disease-modifying therapies. It is also important to note that severe vitamin deficiency occurred despite medication compliance.

ARTICLE HISTORY

Received 30 December 2019
Accepted 25 June 2021

KEYWORDS

Wernicke's encephalopathy (WE); thiamine deficiency; B1 deficiency; multiple sclerosis (MS); bariatric sleeve gastrectomy

1. Introduction

In 2018, the American Academy of Neurology updated their multiple sclerosis (MS) guidelines, recommending that physicians consider prescribing disease-modifying therapies for patients with MS very early in the disease course [1]. The emphasis on early treatment in turn necessitated the early detection and diagnosis of MS by primary care physicians. The initial symptoms and signs of MS often include focal neurological deficits including vision loss, diplopia, vertigo, imbalance, and weakness in the legs. However, a number of non-specific symptoms like fatigue and numbness are also present in MS, making the diagnosis challenging. Typically, MS occurs in relatively young individuals in the third and fourth decades of life and is seen more often in women with a ratio of 2:1 (higher prevalence in females versus males) [2]. Therefore, any young female presenting with neurological symptoms raises concern for possible MS diagnosis. This unfortunately has resulted in the over-diagnosis and misdiagnosis of MS for many patients [3]. A recent study showed that nearly 1 in 5 patients with a diagnosis of MS did not actually have MS, and this included patients who were on treatment for many years [4].

Data from the World Health Organization indicate that worldwide obesity in adults and children has tripled since the 1970s, with 650 million adults classified as obese [5]. 35% of adults and 17% of children are obese in the USA [6]. Obesity is defined as body mass index (BMI) ≥ 30 kg/m² in adults and BMI ≥ 95 percentile for children and teens within the same age and sex groups [7,8]. The designation of obesity

compared to overweight is associated with elevated risks of serious health problems in both adults and children, including type II diabetes, hypertension, dyslipidemia, depression, obstructive sleep apnea, cardiovascular disease, stroke, osteoarthritis, various cancers, liver disease, and mortality [7–10]. Bariatric surgery is becoming increasingly more common to manage obesity and its associated conditions in adolescents and adults in the USA. It has been shown to reduce all-cause mortality up to 10 years post-surgery [11]. The total number of bariatric surgeries has increased from 158,000 procedures in 2011 to 228,000 procedures in 2017 with sleeve gastrectomy and Roux-en-Y gastric bypass as the most common bariatric surgeries in the USA [12,13].

Even though bariatric surgery confers significant benefits to obese patients, post-operatively many patients experience adverse effects related to deficiencies of iron, calcium, vitamin D, and vitamin B12, sometimes even despite taking prescribed supplements. Other less common nutritional deficiencies of essential micronutrients post-bariatric surgery have also been described including deficiencies of vitamins B1, B6, folic acid, Vitamin A, Vitamin K, copper, zinc, and selenium. Several of these deficiencies have potential debilitating neurological consequences. In a single center study spanning 3 years, the most common neurological complications in post-bariatric surgery patients were sensorimotor polyneuropathy, compression mononeuropathy, Wernicke's encephalopathy, ocular myasthenia, worsening of pre-existing autonomic neuropathy, monophasic CNS demyelination and relapsing remitting MS [14].

We present a case of a young female who presented with atypical neurological symptoms after bariatric surgery, whose clinical presentation raised suspicion for MS, but fortunately her surgical history was taken into account, and she was appropriately diagnosed with thiamine deficiency and treated with supplements to good effect.

2. Case

A 22-year-old female with morbid obesity and bipolar disorder presented to the emergency room with new onset bilateral nystagmus, and bilateral lower extremity weakness progressing over 4 weeks making her bedbound. Her family also reported mild confusion. There was no known family history of neurological disease, and she had not reported any recent diarrheal illness preceding her symptoms. She was admitted to the hospital with initial diagnosis of possible MS. The patient also had dysuria and urinalysis was consistent with urinary tract infection on admission. She was started on intravenous antibiotics and IV fluid resuscitation for the urinary tract infection.

While on the floors, a detailed history and physical examination was done. The patient had a history of a gastric sleeve surgery 2 months prior and was taking her supplements (including vitamins) as prescribed. Comprehensive neurological examination revealed the evidence of psychomotor slowing with disorientation to time, poor recall and recollection, and short attention span for age. She was also noted to have 4/5 muscle strength proximally, decreased hip flexion and extension with appropriate effort. The range of movements of knee were intact but poorly sustained. Plantar reflexes showed normal flexor response bilaterally. Eye examination showed intact visual fields and no afferent or efferent pupillary defects but was positive for prominent vertical upbeat nystagmus, horizontal gaze evoked nystagmus with torsion bilaterally.

The initial differential diagnosis for her neurological symptoms included possible MS. High-dose intravenous steroids were considered but were deferred initially because of the active urinary tract infection. MRI brain showed non-specific T2 FLAIR signals in periventricular, juxta-cortical, and bilateral medial thalamic regions. Cervical and thoracic spine MRI were normal. Lumbar puncture showed no evidence of infection or malignancy. No oligoclonal bands were observed in cerebrospinal fluid making MS unlikely.

Meanwhile, given her history of recent gastric sleeve surgery, there was concern for vitamin B1 or B12 deficiencies and/or other mineral (Copper) deficiencies - and therefore levels were checked. She was given multiple vitamin supplementations including Vitamin B1 and B12 at admission. Thiamine level returned low

at 28 nmol/L (reference range 78–185 nmol/L). She was diagnosed with Wernicke's encephalopathy and started on high-dose intravenous thiamine (500 mg twice a day). Over her hospital course, both her mentation and gait slowly improved. On discharge to acute rehabilitation, she was ambulating with a rolling walker. At 5-month follow up, she was ambulating without assistance, nystagmus had resolved, but short-term memory impairment persisted.

3. Discussion

The patients with obesity have a higher prevalence of nutritional deficiencies, with some patients having baseline deficiencies prior to surgery, and others developing it following surgical procedures. This risk of developing new deficiencies is multifactorial and is due in part to dietary changes, in addition to post-surgical changes in absorptive surfaces in the GI tract [15,16].

Vitamin deficiency is a known complication of bariatric surgery with Vitamin C, B1, and B6 deficiencies among the most common [17]. Thiamine deficiency status post-vertical sleeve gastrectomy has been reported in 25% of the cases in adult populations [18]. Affected patients are predominantly African American females compliant with their prescribed vitamin regimens [18]. Wernicke encephalopathy, a serious disorder of thiamine deficiency, has been shown to occur in fewer than 1% of all patients after bariatric surgery, although this is likely to be a vast underestimation [19]. Interestingly, while men are more frequently diagnosed with WE, women seem to have heightened susceptibility [20,21].

MS is the most common CNS disorder resulting in irreversible disability in young adults [22,23]. An estimate of MS prevalence in the USA in 2010 was 309.2 per 100,000 people [24]. It disproportionately affects women at increasing incidence rates over time for reasons that are unclear [25,26]. The mean age of onset is 28–31 with women affected at earlier ages than men [22,27].

Classic signs of WE include the triad of encephalopathy, oculomotor dysfunction, and gait ataxia, especially in patients with alcohol use disorder. However, all three components of this triad have only been present in 33% of patients with many patients demonstrating only one or two of the three features [21,28]. For this reason, WE is likely vastly underdiagnosed [29,30]. One study showed that only 20% of patients found to have chronic WE lesions at autopsy had been diagnosed prior to death. [30] In this study, only 16% of patients had all three characteristic features of the WE triad, while 19% of patients had no features [30].

Some of the symptoms including incoordination, gait instabilities, double vision, abnormal eye

movements, can be seen in both MS and Wernicke's encephalopathy.

Although typical MRI lesions involving symmetric alterations in the thalami and periaqueductal area are commonly seen in patients with WE secondary to chronic alcohol use, it is more common to see atypical lesions in WE secondary to nonalcoholic causes. Some of the atypical lesions described include symmetric alterations of the cerebellum, cranial nerve nuclei, periventricular region, and cerebral cortex [31]. These suspicious overlaps in both clinical and neuroimaging between MS and WE make it paramount to differentiate the two before initiating treatment.

Two aspects of our case stand out severe Vitamin B1 deficiency occurred despite patient adherence to prescribed supplement regimen. The MRI brain also showed non-specific periventricular and thalamic lesions that could have been misdiagnosed as demyelinating lesions of MS. Additionally, such changes are often present in Wernicke's encephalopathy [31].

4. Conclusion

This case illustrates the importance of proper recognition of thiamine deficiency post-bariatric surgeries. Unfortunately, a subset of similar patients has been diagnosed and treated as MS [4]. Although, WE can appear a few weeks to several months after bariatric surgery, these patients are at lifelong risk of developing deficiencies [32]. The recommended initial treatment is parenteral thiamine 200 mg three times a day to 500 mg once or twice daily for 3 to 5 days followed by 250 mg daily for 3 to 5 days, and dose of 100 mg daily [33]. Prompt initiation of treatment is critical for prevention of irreversible Korsakoff syndrome.

Primary care physicians should maintain a high index of suspicion for micronutrient deficiencies in patients presenting with neurological symptoms. Neurological conditions caused by vitamin and micronutrient deficiencies are reversible when diagnosed early and treated with appropriate supplementation. Obtaining an outpatient neurology referral, although necessary, may be time consuming, further delaying treatment while permanent neurological sequelae may result [34]. This case underscores the importance for primary care physicians to consider nutritional deficiencies as possible etiologies for neurological problems as they occur not uncommonly after bariatric surgery.

Acknowledgments

The authors have received no funding for this work.

Disclosure statement

No potential conflict of interest was reported by the author(s).

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References

- [1] Rae-Grant A, Day GS, Marrie RA, et al. Practice guideline recommendations summary: disease-modifying therapies for adults with multiple sclerosis: report of the guideline development, dissemination, and implementation subcommittee of the American academy of neurology. *Neurology*. 2018;90(17):777–788.
- [2] Harbo HF, Gold R, Tintoré M. Sex and gender issues in multiple sclerosis. *Ther Adv Neurol Disord*. 2013;6(4):237–248.
- [3] Nielsen JM, Korteweg T, Barkhof F, et al. Overdiagnosis of multiple sclerosis and magnetic resonance imaging criteria. *Ann Neurol*. 2005;58(5):781–783.
- [4] Kaisey M, Solomon AJ, Luu M, et al. Incidence of multiple sclerosis misdiagnosis in referrals to two academic centers. *Mult Scler Relat Disord*. 2019;30:51–56.
- [5] Obesity and Overweight. Published 2018. Accessed 2019. <https://www.who.int/en/news-room/fact-sheets/detail/obesity-and-overweight>
- [6] Ogden CL, Carroll MD, Kit BK, et al. Prevalence of childhood and adult obesity in the USA, 2011–2012. *JAMA*. 2014;311(8):806–814.
- [7] Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults--The evidence report. National institutes of health. *Obes Res*. 1998;6(Suppl 2):51S–209S.
- [8] Barlow SE, Committee E. Expert committee recommendations regarding the prevention, assessment, and treatment of child and adolescent overweight and obesity: summary report. *Pediatrics*. 2007;120(Suppl 4):S164–192.
- [9] Aune D, Sen A, Norat T, et al. Body mass index, abdominal fatness, and heart failure incidence and mortality: a systematic review and dose-response meta-analysis of prospective studies. *Circulation*. 2016;133(7):639–649.
- [10] Steele CB, Thomas CC, Henley SJ, et al. Vital signs: trends in incidence of cancers associated with overweight and obesity - USA, 2005–2014. *MMWR Morb Mortal Wkly Rep*. 2017;66(39):1052–1058.
- [11] Arterburn DE, Olsen MK, Smith VA, et al. Association between bariatric surgery and long-term survival. *JAMA*. 2015;313(1):62–70.
- [12] Koh CY, Inaba CS, Sujatha-Bhaskar S, et al. Laparoscopic adjustable gastric band explantation and implantation at academic centers. *J Am Coll Surg*. 2017;225(4):532–537.
- [13] English WJ, DeMaria EJ, Brethauer SA, et al. American society for metabolic and bariatric surgery estimation of metabolic and bariatric procedures performed in the USA in 2016. *Surg Obes Relat Dis*. 2018;14(3):259–263.
- [14] Berger JR. The neurological complications of bariatric surgery. *Arch Neurol*. 2004;61(8):1185–1189.
- [15] Tucker ON, Szomstein S, Rosenthal RJ. Nutritional consequences of weight-loss surgery. *Med Clin North Am*. 2007;91(3):499–514, xii.
- [16] Flancbaum L, Belsley S, Drake V, et al. Preoperative nutritional status of patients undergoing Roux-en-Y

- gastric bypass for morbid obesity. *J Gastrointest Surg.* 2006;10(7):1033–1037.
- [17] Clements RH, Katasani VG, Palepu R, et al. Incidence of vitamin deficiency after laparoscopic Roux-en-Y gastric bypass in a university hospital setting. *Am Surg.* 2006;72(12):1196–1202. discussion 1203–1194.
- [18] Tang L, Alsulaim HA, Canner JK, et al. Prevalence and predictors of postoperative thiamine deficiency after vertical sleeve gastrectomy. *Surg Obes Relat Dis.* 2018;14(7):943–950.
- [19] Aasheim ET. Wernicke encephalopathy after bariatric surgery: a systematic review. *Ann Surg.* 2008;248(5):714–720.
- [20] Harper C. The incidence of Wernicke's encephalopathy in Australia—a neuropathological study of 131 cases. *J Neurol Neurosurg Psychiatry.* 1983;46(7):593–598.
- [21] M V, R A, G C. The Wernicke-Korsakoff syndrome and related disorders due to alcoholism and malnutrition. Philadelphia: FA Davis; 1989.
- [22] SV R, Ad S. *Epidemiol Multiple Scler.* 2011;29(2):207–217.
- [23] Noseworthy JH, Lucchinetti C, Rodriguez M, et al. Multiple sclerosis. *N Engl J Med.* 2000;343(13):938–952.
- [24] Wallin MT, Culpepper WJ, Campbell JD, et al. The prevalence of MS in the USA: a population-based estimate using health claims data. *Neurology.* 2019;92(10):e1029–e1040. .
- [25] Alonso A, Hernán MA. Temporal trends in the incidence of multiple sclerosis: a systematic review. *Neurology.* 2008;71(2):129–135.
- [26] Dunn SE, Steinman L. The gender gap in multiple sclerosis: intersection of science and society. *JAMA Neurol.* 2013;70(5):634–635.
- [27] Goodin DS. The epidemiology of multiple sclerosis: insights to disease pathogenesis. *Handb Clin Neurol.* 2014;122:231–266.
- [28] A A, J H, I K, S G. Wernicke's encephalopathy as a mimicker of a Brainstem Relapse in a Multiple Sclerosis Patient. 2015.
- [29] Torvik A, Lindboe CF, Rogde S. Brain lesions in alcoholics. A neuropathological study with clinical correlations. *J Neurol Sci.* 1982;56(2–3):233–248.
- [30] Harper CG, Giles M, Finlay-Jones R. Clinical signs in the Wernicke-Korsakoff complex: a retrospective analysis of 131 cases diagnosed at necropsy. *J Neurol Neurosurg Psychiatry.* 1986;49(4):341–345.
- [31] Zuccoli G, Pipitone N, G Z, N P. Neuroimaging findings in acute Wernicke's encephalopathy: review of the literature. *AJR Am J Roentgenol.* 2009;192(2):501–508. .
- [32] Eric Oudman JW, Mirjam van Dam W, Ulas Biter L, Albert Postma Preventing Wernicke Encephalopathy after Bariatric surgery.
- [33] Galvin R, Bråthen G, Ivashynka A, et al. EFNS guidelines for diagnosis, therapy and prevention of Wernicke encephalopathy. *Eur J Neurol.* 2010;17(12):1408–1418. .
- [34] Nourazari S, Hoch DB, Capawanna S, et al. Can improved specialty access moderate emergency department overuse?: effect of neurology appointment delays on ED visits. *Neurol Clin Pract.* 2016;6(6):498–505.