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Visual Loss, Retinal Hemorrhages, and Optic Disc Edema Resulting From Thiamine Deficiency Following Bariatric Surgery Complicated by Prolonged Vomiting

Andrew W. Lawton, MD,[1] Nicholas E. Frisard, MD[2]

[1]Department of Ophthalmology, Ochsner Clinic Foundation, New Orleans, LA [2]Department of Ophthalmology, Louisiana State University Health Sciences Center, New Orleans, LA

Background: Vision loss resulting from thiamine deficiency is a recognized complication of bariatric surgery. Most patients with such vision loss have Wernicke encephalopathy with characteristic changes seen on neuroimaging. Other patients may have retinal hemorrhages, optic disc edema, and peripheral neuropathy without Wernicke encephalopathy. The risk for thiamine deficiency is potentiated by the presence of prolonged vomiting.

Case Report: A 37-year-old female presented with abrupt onset of vision loss and peripheral neuropathy following bariatric surgery. She had a history of prolonged vomiting postoperatively. Examination of the posterior segment of the eye revealed optic disc edema and large retinal hemorrhages bilaterally. Metabolic workup demonstrated thiamine deficiency. She responded quickly to parenteral thiamine therapy with recovery of normal vision and resolution of ophthalmologic findings.

Conclusion: Patients who undergo bariatric surgery and have a thiamine deficiency can present with visual symptoms and ophthalmologic findings only visible by fundoscopy prior to developing more severe and potentially irreversible complications from the vitamin deficiency. Early detection of intraocular changes resulting from thiamine deficiency and initiation of therapy could prevent more devastating neurologic manifestations. Our case supports the consideration of a prospective study aimed at determining the true incidence of ocular and visual changes such as retinal hemorrhage, optic disc edema, and peripapillary telangiectasia in patients following bariatric surgery.

Keywords: *Bariatric surgery, optic nerve diseases, retinal hemorrhage, thiamine deficiency, vomiting*

Address correspondence to Andrew W. Lawton, MD, Department of Ophthalmology, Ochsner Clinic Foundation, 1514 Jefferson Hwy., New Orleans, LA 70121. Tel: (504) 842-3995. Email: andrew.lawton@ochsner.org

INTRODUCTION

In 2007, the incidence of bariatric surgery, both gastric banding and gastric bypass procedures, to treat morbid obesity was reported to be 113,000 cases per year.[1] This number is increasing as a result of new indications for bariatric surgery, including type 2 diabetes mellitus[2] and idiopathic intracranial hypertension.[3] As the number of procedures increases, so does the number of patients with surgical complications.

One potentially significant side effect of bariatric surgery is malabsorption, particularly of lipid- and water-soluble vitamins, and ocular complications can occur with vitamin A, vitamin B12, and thiamine (vitamin B1) deficiencies. Patients with a thiamine deficiency may develop retinal hemorrhages, optic disc edema, peripheral neuropathy, and Wernicke encephalopathy. We present a case of classic ocular changes from a metabolic deficiency and peripheral neuropathy without Wernicke encephalopathy, emphasizing the importance of vigilance and rapid intervention to restore the patient's vision and peripheral nerve function before severe and possibly irreversible neurologic damage occurs.

CASE REPORT

A 37-year-old female underwent a Roux-en-Y laparoscopic procedure on December 20, 2014. On December 30, 2014, her thiamine level was 29 μg/L (the reference range is 38-122 μg/L). On August 13, 2014, her thiamine level had been 43 μg/L. After surgery, she experienced persistent vomiting that required readmission for treatment on January 21, 2015. She was discharged on January 28, 2015, with her nausea controlled by antiemetic suppositories.

The patient presented to the neuroophthalmology service on February 16, 2015, with concerns of sudden, symmetrical, painless vision loss in both eyes beginning 9 days earlier. She again was experiencing nausea and vomiting and could not tolerate oral medications. Her vision had gradually worsened to the point that she could only

distinguish shapes. In addition, she was developing weakness in her legs and found standing difficult. She was alert and oriented to person, place, and time and did not exhibit any signs of cognitive dysfunction. During examination, her visual acuity at distance with correction was 20/400 in her right eye and 20/200E at 3 feet in her left eye. Both pupils constricted sluggishly to light with bilateral 3+ afferent pupillary defects. Confrontation testing revealed dense central visual field defects in both eyes. Extraocular motility was intact in both eyes. Dilated fundus examination revealed 1+ edema of bilateral optic discs and intraretinal hemorrhages in both eyes. The Figure is a fundus photograph showing an intraretinal hemorrhage and optic disc edema in the patient's left eye. Computed tomography (CT) scan of the head was unremarkable for acute intracranial abnormalities. Because of severe nausea and vomiting, the patient was unable to tolerate additional photography or testing. Her thiamine level was 13 μg/L.

The patient was admitted to the hospital on an emergent basis with the diagnosis of thiamine deficiency neuropathy and thiamine optic neuropathy. She initially received intravenous thiamine supplementation, followed by intramuscular thiamine supplementation. She responded well to treatment, had rapid improvement in her vision, and was discharged after 9 days of parenteral therapy and oral vitamin supplementation. Her thiamine dosage was 30 mg intravenously 3 times daily for 7 days and was reduced to oral thiamine 100 mg twice daily at discharge.

The patient returned for an eye examination on March 2, 2015, and reported that she no longer had dark areas in her vision. She was being actively followed in the bariatric surgery clinic, and her daily thiamine dosage at that time was a 100-mg oral maintenance dose. Visual acuity with correction was 20/25 in her right eye and 20/20 in her left eye. Pupillary function was normal. She had minimal residual optic disc edema and intraretinal hemorrhages in each eye. At a repeat test on March 24, 2015, her thiamine level was 93 μg/L.

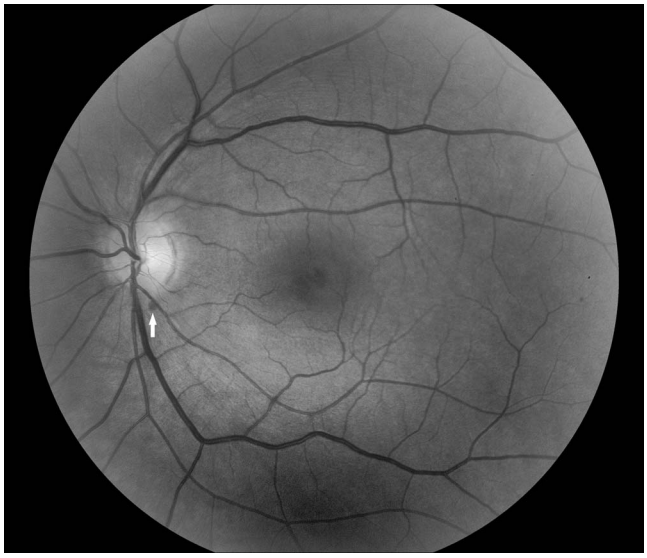

Figure. Fundus photograph of the left eye shows grade 1 optic nerve edema and a retinal hemorrhage (arrow) on the inferior border of the optic nerve.

DISCUSSION

Proper preoperative nutritional screening in patients planning bariatric surgery is crucial because of the potential postoperative complication of malabsorption. The diet of morbidly obese patients may be so heavily skewed toward processed foods that they may be thiamine deficient prior to surgery. Carrodeguas et al found that 47 of 303 patients (15.5%) who presented for bariatric surgery had low thiamine levels on initial screening.[4] Females were more likely to be thiamine deficient at presentation than males. In addition, bariatric surgery can result in other nutritional deficiencies, such as vitamin A, vitamin B12, vitamin E, and copper deficiencies, that can cause ophthalmologic complications.[5]

Vomiting may be a risk factor for thiamine deficiency in otherwise healthy individuals[6,7] and in patients who undergo bariatric surgery.[8] Patients who have had bariatric surgery are particularly prone to vomiting, a symptom that occurs in more than 50% of patients.[9] Patients may vomit because they eat too much at one time, eat too quickly, or have mechanical complications from the surgery.

The true rate of thiamine deficiency after bariatric surgery has not been established, although one author estimated it to be 18.3%.[10] A deficient state can occur rapidly (18-20 days) because thiamine is water soluble, and tissue storage is limited.[11] This process is accelerated in patients with poor nutrition and in patients who do not take prescribed supplementary medications. Routine thiamine screening is not recommended after bariatric surgery but should be considered for patients who use alcohol; receive parenteral nutrition; or experience rapid weight loss, intractable vomiting, neuropathy, encephalopathy, or heart failure. Patients with severe thiamine deficiency, such as Wernicke encephalopathy or Korsakoff syndrome, should be treated with intravenous thiamine, 500 mg per day for 3-5 days, followed by 250 mg per day for 3-5 days or until resolution of symptoms. Mild thiamine deficiency can be treated with intravenous thiamine, 100 mg per day for 7-14 days. When oral intake is resumed, patients can be administered oral thiamine, 100 mg per day, usually indefinitely or until risk factors have resolved. If small bowel bacterial overgrowth is suspected to be the cause of malabsorption, oral antibiotic therapy and oral thiamine, 100 mg twice daily for 2 months, are recommended.[12]

The characteristic presentation of thiamine deficiency in patients following bariatric surgery is Wernicke encephalopathy 4-12 weeks after surgery.[13] Key features of this syndrome include ophthalmoplegia, altered mental status, and ataxia. Patients may also develop depression, visual hallucinations, visual loss, hearing loss, retinal hemorrhages, peripapillary telangiectasia, optic disc edema,[14] and peripheral neuropathy. Generally, CT or magnetic resonance imaging (MRI) of the brain confirms Wernicke encephalopathy.[15] MRI is a more sensitive study than CT for detecting intracranial abnormalities resulting from Wernicke encephalopathy.[15] Our patient was atypical because of the presence of retinal hemorrhages, optic disc edema, and peripheral neuropathy but no clinical evidence of extraocular motility dysfunction, mental status changes, or radiologic abnormalities.

Patients with symptoms of thiamine deficiency require high-dose parenteral thiamine therapy. Treatment should begin as soon as the deficiency is suspected and does not require waiting for documentation of low thiamine levels. Thiamine may be administered intravenously or intramuscularly. Recommended dosages vary greatly, ranging from a minimum of 100 mg daily to as much as 500 mg per day intravenously. Resolution of symptoms may be extremely rapid, and some patients show significant improvement after the initial dose.

Thiamine deficiency after bariatric surgery is a significant postoperative complication, particularly in patients who experience prolonged postoperative vomiting. Diagnosis may be delayed because of absence of characteristic extraocular motility findings or mental status alteration characteristic of Wernicke encephalopathy. Patients are not routinely screened with eye examinations after bariatric surgery, raising the possibility that intraocular changes could precede clinical vision loss or other systemic manifestations of thiamine deficiency.

CONCLUSION

Patients who undergo bariatric surgery and have a thiamine deficiency can present with visual symptoms and ophthalmologic findings only visible by fundoscopy prior to developing more severe and potentially irreversible complications. Early detection of intraocular changes resulting from thiamine deficiency and initiation of therapy could prevent devastating neurologic manifestations. Our case supports the consideration of a prospective study aimed at determining the true incidence of ocular and visual changes such as retinal hemorrhage, optic disc edema, and peripapillary telangiectasia in patients following bariatric surgery.

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REFERENCES

1. Livingston EH. The incidence of bariatric surgery has plateaued in the U.S. *Am J Surg*. 2010 Sep;200(3):378-385. doi: 10.1016/j.amjsurg.2009.11.007.
2. Brethauer SA, Aminian A, Romero-Talamás H, et al. Can diabetes be surgically cured? Long-term metabolic effects of bariatric surgery in obese patients with type 2 diabetes mellitus. *Ann Surg*. 2013 Oct;258(4):628-636; discussion 636-637. doi: 10.1097/SLA.0b013e3182a5034b.
3. Fridley J, Foroozan R, Sherman V, Brandt ML, Yoshor D. Bariatric surgery for the treatment of idiopathic intracranial hypertension. *J Neurosurg*. 2011 Jan;114(1):34-39. doi: 10.3171/2009.12.JNS09953.
4. Carrodeguas L, Kaidar-Person O, Szomstein S, Antozzi P, Rosenthal R. Preoperative thiamine deficiency in obese population undergoing laparoscopic bariatric surgery. *Surg Obes Relat Dis*. 2005 Nov-Dec;1(6):517-522; discussion 522.
5. Becker DA, Balcer LJ, Galetta SL. The neurological complications of nutritional deficiency following bariatric surgery. *J Obes*. 2012;2012:608534. doi: 10.1155/2012/608534.
6. Chitra S, Lath KV. Wernicke's encephalopathy with visual loss in a patient with hyperemesis gravidarum. *J Assoc Physicians India*. 2012 May;60:53-56.
7. Gratton SM, Lam BL. Visual loss and optic nerve head swelling in thiamine deficiency without prolonged dietary deficiency. *Clin Ophthalmol*. 2014 May 22;8:1021-1024. doi: 10.2147/OPTH.S64228. eCollection 2014.
8. Frank P, Crookes PF. Short- and long-term surgical follow-up of the postbariatric surgery patient. *Gastroenterol Clin North Am*. 2010 Mar;39(1):135-146. doi: 10.1016/j.gtc.2009.12.011.
9. DeMaria EJ. Bariatric surgery for morbid obesity. *N Engl J Med*. 2007 May 24;356(21):2176-2183.
10. 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 Dec;72(12):1196-1202; discussion 1203-1204.
11. O'Donnell K. Severe micronutrient deficiencies in RYGB patients: rare but potentially devastating. *Pract Gastroenterol*. 2011 Nov;100:13-27.
12. Mechanick JI, Youdim A, Jones DB, et al; American Association of Clinical Endocrinologists; Obesity Society; American Society for Metabolic & Bariatric Surgery. Clinical practice guidelines for the perioperative nutritional, metabolic, and nonsurgical support of the bariatric surgery patient—2013 update: cosponsored by American Association of Clinical Endocrinologists, The Obesity Society, and American Society for Metabolic & Bariatric Surgery. *Obesity (Silver Spring)*. 2013 Mar;21 Suppl 1:S1-S27. doi: 10.1002/oby.20461.
13. Singh S, Kumar A. Wernicke encephalopathy after obesity surgery: a systematic review. *Neurology*. 2007 Mar 13;68(11):807-811.
14. Bohnsack BL, Patel SS. Peripapillary nerve fiber layer thickening, telangiectasia, and retinal hemorrhages in Wernicke encephalopathy. *J Neuroophthalmol*. 2010 Mar;30(1):54-58. doi: 10.1097/WNO.0b013e3181ceb4d0.
15. Cerase A, Rubenni E, Rufa A, et al. CT and MRI of Wernicke's encephalopathy. *Radiol Med*. 2011 Mar;116(2):319-333. doi: 10.1007/s11547-011-0618-x.

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