Wernicke Encephalopathy in Adolescents After Bariatric Surgery: Case Report and Review

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abstract

Roughly 1% of all weight loss surgery is performed in adolescents. There is strong evidence demonstrating significant postsurgical weight loss, improvement in quality of life, and reduction in comorbidities such as hypertension and diabetes. Reports of postoperative complications in adolescents are few because of the small sample size in most series. Despite vitamin supplementation, nutritional deficiencies requiring hospitalization occur occasionally after Roux-en-Y gastric bypass. Wernicke encephalopathy, a triad of ophthalmoplegia, ataxia, and altered mental status, is a serious consequence of thiamine (vitamin B_1) deficiency. Few cases of Wernicke encephalopathy after weight loss surgery have been reported in the literature and even fewer in the pediatric population. Here we describe a teenage girl who develops vomiting after Roux-en-Y gastric bypass and presented with nystagmus, irritability, and ataxia. The clinical presentation, diagnosis, and treatment of Wernicke encephalopathy in adolescents after bariatric surgery are discussed.

Nearly 6 million children in the United States are obese, with a BMI ≥95th percentile. The highest prevalence of childhood obesity is among youth aged 12 to 19 years. Nonsurgical weight loss treatments for the 2% to 6% of children with extreme obesity, defined as a BMI >99th percentile, are often ineffective.[1,2] As more bariatric procedures are being performed on adolescents, evidence demonstrating the long-term benefits continues to grow.

Complications from bariatric surgery are reported as moderate to severe in 5% to 13% of adolescents in the first 3 postoperative years.[3–5] Nutritional deficiencies, although often minor, occur in the majority of these patients.[6–8] Wernicke encephalopathy, a triad of ataxia, altered mental status, and ophthalmoplegia due to thiamine (vitamin B_1) deficiency, is a potentially fatal condition that may be underdiagnosed in this population. In cases that are identified and treated, neurologic deficits still persist in nearly half.[9] Here we review the case of 17-year-old girl with a recent history of gastric bypass diagnosed with Wernicke encephalopathy after presenting with weeks of vomiting followed by headache, ophthalmoplegia, and ataxia.

CASE PRESENTATION

A 15-year-old Caucasian girl presented to the Massachusetts General Hospital pediatric weight center for evaluation of her obesity complicated by juvenile rheumatoid arthritis, depression, hepatic steatosis, amenorrhea, and headaches. She was found to have a BMI of 36.4 (>99th percentile) and was placed into a medical management program. She participated in a comprehensive

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adolescent group weight loss surgery program and was evaluated by nutrition, psychology, and pediatrics monthly. After 18 months her weight had increased to 120 kg with a BMI of 45.5 despite maximum medical management. At age 17, she underwent laparoscopic Roux-en-Y gastric bypass (RYGB) at the same institution. The operation involved creation of a 30cc gastric pouch attached (hand sewn) to a 100 cm roux limb. The biliopancreatic limb measured 60 cm in length. She reported taking her multivitamins, vitamin B_{12}, vitamin D, and calcium supplements postoperatively.

Five weeks after her RYGB, she was admitted to the hospital for abdominal pain, nausea, vomiting, and dehydration and was diagnosed with pancreatitis and anastomotic stenosis. Her weight at this time was 108 kg with a BMI of 40.8. Abdominal and pelvic computed tomography scans and a barium swallow study showed no abnormalities or evidence of leak. She subsequently underwent upper endoscopy with dilation of an anastomotic stricture. She improved clinically and was discharged from the hospital 5 days after admission.

Ten days later, she re-presented to the emergency department for continued abdominal pain, worsening vomiting, and new headaches. She had mild epigastric tenderness and a normal neurologic examination. She received intravenous (IV) fluids, ondansetron, and omeprazole, after which she was able to tolerate fluids and was discharged from the hospital.

She returned to the emergency department 2 days later for worsening headache, frequent vomiting, and new diplopia. She endorsed bilateral blurry vision and pain with eye movements. Her headache was bifrontal, constant, and severe and was not reminiscent of her typical headaches. She also reported photophobia, phonophobia, and abdominal pain but denied numbness, weakness, fevers, or meningismus. Her weight had decreased further to 94 kg with a BMI of 35.6.

Her general examination was notable for a severely obese teenager lying in bed in a dark room with her eyes covered. She was easily upset when approached and refused to turn her head or walk. She was fully alert, oriented, and followed all other commands. She had pain on lateral gaze with incomplete abduction bilaterally and prominent nystagmus. The remainder of her neurologic examination, including strength, sensation, and reflexes, was normal. Her admission laboratories were remarkable only for a lipase of 220 U/L (reference range 13–60 U/L) and potassium of 3.1 mmol/L (reference range 3.4–5.0 mmol/L).

She was admitted to the surgical service and started on lactated Ringer's in 5% dextrose. At this time, the differential diagnosis for her neurologic symptoms included migraine headache, pseudotumor cerebri, and venous sinus thrombosis. The neurology service recommended metoclopramide and magnesium in addition to IV fluids for her headaches. She had a brain MRI with venous imaging and an abdominal computed tomography, both of which were normal. On hospital day 2, she underwent an upper endoscopy with dilatation of a mild recurrent anastomotic stricture. On examination by the neuroophthalmology service, she had mildly reduced visual acuity bilaterally, prominent end gaze nystagmus, and bilateral sixth cranial nerve palsies.

Although her headaches were improving, on hospital day 6 she continued to experience diplopia and nystagmus. She had also developed lower extremity paresthesias and was only able to ambulate with assistance. Given her progressive neurologic symptoms and recent RYGB, Wernicke encephalopathy was suspected. A thiamine level was ordered, and IV supplementation was promptly initiated. Dextrose was removed from her IV fluids. On day 7, she developed truncal ataxia and a wide-based gait. Her thiamine level returned to 39 nmol/L, with a normal reference range of 70 to 180 nmol/L. Just before bypass surgery, her thiamine level was 129 nmol/L. She was continued on IV thiamine 500 mg 3 times a day for 3 days followed by every-other-day dosing. A repeat brain MRI was again normal. Her gait improved by day 9 of her hospitalization, and she was discharged from the hospital on 50-mg oral thiamine for an indefinite amount of time.

In neurology clinic follow-up 1 month later, her headaches, diplopia, and nystagmus had improved but she continued to use a cane for ambulation. At her 6-month follow-up, she had mild residual nystagmus and intermittent diplopia but was ambulating without significant difficulty.

DISCUSSION

Since the early 2000s, bariatric surgery for adolescents has been used more widely. In 2009, the BMI cutoffs for weight loss surgery were reduced to match those of adult patients, with eligible adolescents having a BMI ≥35 with major comorbidities or >40 with minor comorbidities.[7,10] Current guidelines emphasize that eligible children should achieve physical maturity, typically age 13 for girls and 15 for boys, and undergo extensive preoperative preparation and an informed consent process.[7,11] Although controversy regarding elective surgery in minors continues, a recent prospective observational study demonstrated significant reductions in weight as well as in premorbid diabetes, hypertension, and dyslipidemia up to 3 years after gastric bypass surgery in

adolescents.[3] Several studies have also revealed an improvement in overall quality of life and depression postoperatively.[12–14]

The RYGB involves bypassing the entire duodenum and the first portion of the jejunum, where thiamine is best absorbed. Reduced absorption and bacterial overgrowth contribute to the development of thiamine deficiency in these patients. Severely obese adults can have poor overall nutrition and micronutrient depletion even before surgery, but preoperative nutritional status in adolescents has not been thoroughly investigated.[15,16] The half-life of thiamine is only 18 to 20 days, and thus stores are quickly depleted in cases of recurrent vomiting, inadequate nutrition, or malabsorption.

Wernicke encephalopathy occurs in <1% of all patients after bariatric surgery, although the condition is likely underdiagnosed.[9,17] Only 10% to 20% demonstrate the complete triad of gait ataxia, ophthalmoplegia, and altered mental status and even fewer in children and adolescents.[18–20] Altered consciousness is the most common presenting symptom in children. The most common ocular abnormalities include nystagmus, bilateral sixth cranial nerve palsies, and conjugate gaze palsy.[19,21] Serum testing for thiamine and transketolase levels is often, but not always, abnormal. Thus, a high level of suspicion must occur for the proper diagnosis to be made.

Literature review revealed only 9 definitive cases of Wernicke encephalopathy in children undergoing weight loss surgery, several of which were lacking valuable clinical information (Table 1)[3,5,22–25] As in adults, frequent vomiting with or without gastrojejunal stenosis as well as medication noncompliance are present in the majority of these patients.[25,26] The most frequent presenting signs and symptoms included nystagmus, paresthesias, and hyporeflexia. Although symptom onset is most often between 4 to 12 weeks after surgery in adults, 94% develop Wernicke encephalopathy within 6 months postoperatively, which we found in the limited cases of adolescents described in the literature.[9,26] Importantly, our patient case highlights the danger in providing dextrose-containing fluids without thiamine supplementation to at-risk populations, such as children with cancer, anorexia, or recent weight loss surgery.[21] Glucose metabolism heavily utilizes thiamine,

TABLE 1 Adolescent Cases of Wernicke Encephalopathy After Bariatric Surgery

Ref	Age (years)/ Sex	Weight Loss %	Risk Factors	Onset, months[a]	Symptoms	Signs
3[b]	13–19, NR	NR	NR	<36	NR	NR
5	13–21, NR	NR	NR	<12	Mild	NR
5	13–21, NR	NR	NR	<12	Severe	NR
22	15 F	30	Medication noncompliance	4	Dizziness, diplopia, hearing loss, tinnitus, numbness and burning of hands, lower extremity pain, falls	Nystagmus, hearing loss, hyperesthesia in stocking distribution, decreased vibratory sensation of fingers and lower extremities, lower extremity hyporeflexia
22	14 F	34	Vomiting, anastomotic stenosis	2	Dizziness, burning foot pain, lower extremity weakness and numbness, finger numbness, impaired ambulation	Nystagmus, hypoesthesia and reduced vibratory sensation in stocking distribution, lower extremity areflexia and weakness, waddling gait
22	17 F	42	Dehydration, anastomotic stenosis	6	Headaches, burning foot pain	Hyperesthesia of feet
23	14-19, NR	NR	Poor nutrient intake	<12	Lower extremity pain and numbness	NR
24	17 F	21	Vomiting, anastomotic stenosis, medication noncompliance	2.5	Confusion, lower extremity pain in stocking distribution, weakness, falls	Nystagmus, lower extremity areflexia, weakness and numbness, unstable gait
25	17 F	11	Nausea, medication noncompliance	1.5	Confusion, drowsiness, diplopia, lower extremity numbness, impaired ambulation	Nystagmus, bilateral sixth cranial nerve palsies, lower extremity hypoesthesia, areflexia, broad-based gait
Current case	17 F	22	Vomiting, pancreatitis, anastomotic stenosis, medication noncompliance	2	Irritability, headache, diplopia, pain with eye movements, lower extremity numbness, impaired ambulation	Nystagmus, bilateral sixth cranial nerve palsies, lower extremity hypoesthesia, truncal ataxia, wide-based gait

F, female; NR, not reported.

[a] Months until symptom onset after bariatric surgery.

[b] Unclear whether reported case had baseline thiamine deficiency.

so further increasing glucose levels can deplete already-low thiamine stores.

MRI can support the diagnosis of Wernicke encephalopathy. As in adults, children with Wernicke encephalopathy may demonstrate symmetric hyperintensity of the mammillary bodies, periaqueductal gray, cerebellar vermis, and thalamus on MRI. Hyperintensity of the putamen has also been detected only in children.[27] As with our patient, brain MRI is normal in up to 53% with proven Wernicke encephalopathy.[9,28]

Wernicke encephalopathy is a medical emergency, with a mortality rate up to 20%. Recovery is incomplete in 49% of adults, who experience residual cognitive impairments, gait imbalance, and nystagmus.[9,29] Once the diagnosis is suspected, treatment should be rapidly initiated with parenteral thiamine. Although dosage guidelines vary, 500 mg IV thiamine 3 times a day for 2 to 3 days followed by 250 to 500 mg IV once daily for 5 days is frequently recommended.[25,30,31] Afterward, 50 to 100 mg oral thiamine daily should be initiated and continued for at least 6 months postoperatively.[24,31–34] Given that 10% to 20% of obese individuals are moderately to severely thiamine deficient at baseline, some have also suggested starting a multivitamin before surgery.[22,34]

The adolescent population presents additional treatment challenges compared with adults. Only 13% to 33% of adolescents are fully compliant with vitamin supplementation after weight reduction surgery.[35,36] Forgetting to take vitamins and difficulty swallowing pills were commonly cited reasons for noncompliance.[35] Comorbid depression is particularly common in obese teens and, if not properly managed, can lead to further poor compliance.[7,24]

Prevention of Wernicke encephalopathy can be improved through close postsurgical follow-up with a multidisciplinary team, especially in those with significant or persistent vomiting. Despite such follow-up, however, compliance is overreported. Recognition of the signs and symptoms is critical to early diagnosis and treatment of this potentially fatal and easily treatable condition.

ABBREVIATIONS

IV: intravenous
RYGB: Roux-en-Y gastric bypass

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