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REVIEW ARTICLE

Wernicke Encephalopathy: a Future Problem Even After Sleeve Gastrectomy? A Systematic Literature Review

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Abstract Wernicke encephalopathy (WE) is a serious complication of bariatric surgery with significant morbidity and mortality. A few cases have been reported in the literature, mainly in patients after a Roux-en-Y gastric bypass. Since sleeve gastrectomy (SG) has become a more established and popular bariatric procedure, WE is expected to appear more frequently after SG. We performed a literature review on WE after SG, and 13 cases have been found to be sufficiently documented. The risk of WE needs to be considered in patients with a prolonged vomiting episode and any type of neurological symptoms, independent of the presence of any surgical complications.

Keywords Sleeve gastrectomy · Wernicke encephalopathy · Thiamine deficiency · Morbid obesity · Bariatric surgery

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Introduction

In recent years, sleeve gastrectomy (SG) has become popular as a standard single bariatric procedure. Its frequency is increasing worldwide, representing the majority of bariatric surgeries in many states [1–3]. Nevertheless, such methods may potentially lead to severe nutritional deficiencies with chronic disability. Long-term data on nutritional deficiencies are limited. Currently, little is known about the impact of the post-bariatric course of macro-and micronutrient deficiencies after SG, especially the B-group vitamins, and associated increased incidence of neurological complications [4, 5]. Thiamine (vitamin B1) is a core enzyme in several biochemical pathways, particularly in the central and peripheral nervous system because of its sensitivity to energy metabolism [6]. Thiamine deficiency is considered to be the most significant cause of micronutrient-associated cerebral dysfunctions and leads to early onset of peripheral polyneuropathy or Wernicke encephalopathy (WE), which requires immediate treatment [7]. Thiamine deficiency affects various other organ systems, including the gastrointestinal tract and heart. WE is characterized by severe cognitive and psychotic disorders, confusion, ataxia, and dysfunction of cranial nerves (especially in the oculomotor part) [8].

WE can occur after all types of bariatric surgical procedures, including Roux-en-Y gastric bypass, or vertical banded gastroplasty and biliopancreatic diversions (BPD), similar techniques to SG, but performed only in fewer hospitals. These procedures can produce true vomiting rather than regurgitation. Other essential predisposing risk factors for bariatric surgery-associated WE are excessive alcohol consumption, rapid post-bariatric weight loss, or long-term parenteral nutrition [9].

In this case study, we report a case of severe thiamine deficiency following SG in a high-risk patient and systemically discuss published data.

Materials and Methods

From January 2007 to April 2015, only one of 255 patients who were treated with SG developed micronutrient-associated encephalopathy in our center. Pre-hospitalization data, inpatient chart records, and radiological and endoscopic findings were analyzed for the reported patients. In addition, we performed a literature review on WE after SG using PUBMED, OVID, EMBASE, and GOOGLE SCHOLAR, searching for MeSH terms, such as WE, Korsakoff syndrome, encephalopathy, beriberi, thiamine deficiency, vitamin B1, restrictive weight loss surgery, sleeve gastrectomy, and vertical gastrectomy. Studies reporting on patients with an objective diagnosis of WE after SG were included. There were no language restrictions. Cases of other restrictive weight loss interventions (vertical banded gastroplasty, intragastric balloon, gastric plication or gastric band surgery) were excluded. We reviewed the full text and the abstracts of these articles and reviewed the data on age, sex, comorbidities, onset duration, weight loss, predisposing factors, assumed reasons, radiographic and neurologic findings and outcomes. All of the included studies were case reports; no meta-analyses were used. Continuous variables were reported as medians (range). Dichotomous variables were reported as percentages. All of the statistics were conducted using the SPSS 16.0 software package (IBM Cooperation, Armonk, New York, USA).

Case Report

A 55-year-old man with a body mass index (BMI) of 67 kg/m^2 underwent SG at our clinic. The patient's medical record included metabolic syndrome with arterial hypertension, type 2 diabetes mellitus, dyslipidemia, non-alcoholic steatohepatitis (NASH), gastro-esophageal reflux disease (GERD; Los Angeles classification A), and obstructive sleep apnea syndrome requiring continuous positive airway pressure.

The first routine visit in our outpatient clinic 4 weeks after surgery was uneventful. Two months later, the patient arrived at the emergency care unit with nausea and recurrent vomiting, dysphagia, reflux, and the classic triad of WE and Korsakoff syndrome. Intravenous thiamine (vitamin B1; 300 mg three times daily i.v.) was administered immediately without waiting for results of a magnetic resonance image (MRI) and laboratory assessment. Gastroscopy combined with a contrast swallow excluded a stenosis, obstruction, or kinking of the gastric remnant, but demonstrated severe GERD (Los Angeles classification D). The patient continued losing weight, up to 50 kg (42 % EWL) 3 months after the surgical intervention.

Approximately 6 months after the SG, the patient was able to walk with minimal assistance, and his neurological status further improved. Impaired short- and long-term memory was still present, and the patient needed social support in a special care facility. One year after the SG, we decided to convert the SG into a Roux-en-Y gastric bypass (RYGB) because of the persistent GERD. Two years after the SG, the patient is still closely monitored in our outpatient clinic and receives daily supplementation of multivitamins and minerals. At this time, the patient's neurological status is partially recovered and his actual BMI is approximately 42 kg/m^2.

Results

Similar to our patient, we identified 13 cases of WE after SG in the literature. One case report was excluded because it was not published in a recognized journal. Among the 13 included patients, six were women with a median age of 36 years (range 21–55); the median BMI reported in nine studies was 49.7 kg/m^2 (range 41.5–67). Six cases noted a type 2 diabetes mellitus or glucose intolerance as a comorbidity. The onset of WE occurred between 12 days and up to 7 months after surgery. When symptoms of WE developed, patients had lost between 19.5 and 90 kg of body weight (reported in eight cases). The most common neurological sign was altered mental status (n=11). Eight cases confirmed vomiting as a postoperative risk factor. The onset of vomiting and the duration after the SG differ in most instances or were insufficiently reported. Furthermore, three patients were non-compliant with multivitamin supplementation. Psychogenic anorexia (n=1) and parenteral nutrition (n=1) were other assumed predisposing factors. The classical triad of WE was present in six cases (46 %).

Peripheral polyneuropathy (PNP) was reported in the lower limbs in five patients. Other symptoms and signs were weakness (n=7), dysarthria (n=2), and impaired memory (n=8). Typical findings of WE on brain MRIs were found in six of ten cases (60 %). The CT scan did not reveal significant cerebral alterations in four cases. In one instance, the electroencephalogram showed diffuse non-specific changes. Electromyography was performed in five patients; the results showed peripheral polyneuropathy. The serum thiamine levels were low in six patients; the thiamine levels in urinary samples were low in one case. Among patients who underwent esophagogastroduodenoscopy (EGD) (n=6) and upper gastrointestinal (UGI) endoscopy (n=5), the findings included one functional stenosis, three cases of esophagitis, and one case of delayed gastric emptying. Most patients had no mechanical cause of vomiting (n=6). One gastrojejunostomy resulting from a functional stenosis was performed and completed, one mini-bypass was performed in the early

postoperative period, one stent was inserted because of leakage, and one switch to a RYGB was performed because of reflux esophagitis LA grade D. Most patients were treated with parenteral thiamine; thiamine supplementation management was different in most reports. In all cases, treatment with thiamine was started soon after WE diagnosis. The duration and dose after starting i.v. thiamine treatment were different in the reports. Clinical outcome was reported in all patients; median follow-up ranged from 3 weeks to 12 months. Most patients showed partial recovery; only three patients were reported to achieve a complete recovery. Several patients had residual neurologic deficits, including Korsakoff syndrome (n=2), persistent amnestic state with anterograde amnesia and memory loss (n=2), memory problems, gait disturbance (n=5), nystagmus (n=2), and persisting neuropathy (n=1). Two patients died during hospitalization from multiorgan or respiratory failure.

Discussion

To our knowledge, this is the 13th case report on WE following SG [10–21] (Table 1). Our systemic review is the first to overview the data of patients who have developed WE after SG. Although all of the information is based on case reports, the number of cases seems to have increased in recent years, most likely because SG is an established bariatric procedure [22, 23]. Therefore, one can assume that the current number of identified patients suffering from WE after SG may be underreported. Contrawise, the risk of developing macro- and micronutrient deficiencies in combination with neurological symptoms is considered to be quite small in SG [5, 24]. There are fewer studies on the incidence and development of micronutrient deficiency and metabolic encephalopathy after SG [13]. Although the diagnosis of WE after bariatric surgery can be difficult, the leading symptoms and signs may appear at any time and may progress even over years [8].

Risk Factors

The most important predisposing risk factor for post-bariatric WE is an inadequate vitamin B1 supply resulting from either non-compliance of vitamin supplementation, prolonged postoperative vomiting, or both. Repeated vomiting is thought to deplete thiamine reserves within weeks after bariatric surgery in the early period [8]. Eight cases in the literature confirmed recurrent vomiting as a postoperative risk factor. Nausea and vomiting are seen relatively often after bariatric surgery in the early postoperative phase. The association between the vomiting onset, duration, and frequency of vomiting after surgery and the development of neurological symptoms are insufficiently described in the analyzed cases and are not entirely understood so far. Furthermore, recurrent postoperative emesis that occurs a relatively long time after surgery remains unclear, except for one case with functional stenosis. This result highlights the fact that WE has to be considered even in the absence of any surgical complication, e.g., stenosis/obstruction at EGD or UGI and at later onset. The development of symptoms of WE widely varied among the reviewed cases. Weight loss ranged from 20 to 90 kg (% EWL 30–117 %) and was secondary to anorexia and bulimia (two cases). It was not possible to identify a cutoff value between the amount of post-bariatric weight loss and the increased risk of WE.

Low preoperative serum levels of thiamine are known to be associated with an increased risk of WE after bariatric surgery. Caucasians usually show a smaller incidence of low serum thiamine levels. In women, serum thiamine levels are lower than in men [25]. It has been reported that 5–30 % of patients undergoing bariatric surgery suffered from subnormal serum thiamine levels prior to the treatment [26, 27]. After the bariatric procedure, low serum levels of thiamine are detected in up to 25 % of the patients within 2 years and in up to 30 % 5 years after bariatric surgery [28, 29]. Preoperative thiamine screening is not mandatory under the current nutrition guidelines; therefore, in our case, the thiamine levels were not determined.

Various coexisting morbidities, including diabetes mellitus and hepatic steatosis, were considered to be involved in the onset of WE. Thiamine is assumed to be depleted faster in patients with type 2 diabetes mellitus and hepatic steatosis [30]. Among the analyzed cases, six patients suffered from diabetes mellitus or glucose intolerance.

Other risk factors for the development of WE are excessive alcohol consumption and long-term parenteral nutrition (two cases), which is often seen in bariatric patients with surgical complications in the intensive care unit [8]. The role of post-bariatric alcohol consumption in bariatric surgery, especially in men, remains under debate because a reliable method to verify alcohol consumption, such as measurements of the serum CDT levels, is not widely used [31]. Korsakoff syndrome is more likely to manifest when WE results from a history of alcohol abuse. This condition may contribute to the occurrence of WE years after bariatric surgery [32]. Delayed gastric emptying because of gastric paresis especially seen in patients with type 2 diabetes mellitus, as well as a high-calorie, high-carbohydrate diet, can be significant risk factors as well [33].

Frequency

Thiamine deficiency is reported to occur more often after RYGB [7]. This could be related to the fact that maximal absorption of thiamine takes place in the jejunum, which can be affected by bacterial overgrowth or anatomical changes after a RYGB. Clements et al. noted a significant incidence rate up to 18 % 2 years after a RYGB [34]. However, the

Table 1 WE cases after SG: demographics, clinical features, and outcomes

Number	Author/published date	Age /sex	BMI preoperative	Onset of symptoms	Weight loss between surgery and WE	Follow-up	Classical neurology	Other neurological symptoms	Assumed predisposing factors	Outcome
		Year	kg/m^2	Months after surgery	kg	Months after WE	N/C/A			
1	Coyette 2007	21/F	nr	2.5	nr	12	N/C/A	Weakness	nr	Neurological deficits
2	Makarewicz et al. 2007	38/F	62	12 days	nr	6	N/C/-	Weakness, hypokinesis, tibial nerve paresis	Vomiting, non-compliance	Full recovery
3	Jeong et al. 2011	24/M	47.1	7	60	nr	N/-/A	Dysarthria, diplopia, dysmetria	Parenteral nutrition	Partial recovery
4	Abouaf 2011	44/M	nr	5	40	12	N/-/A	Blurred vision	Vomiting	Partial recovery
5	Sharabi et al. 2012	43/F	nr	3	30	2.5	N/C/A	Weakness	Vomiting	Partial recovery, death
6	Moizé et al. 2012	35/F	49.8	2.5	15	12	N/-/A	Weakness, dizziness	Non-compliance	Full recovery
7	Scarano et al. 2012	27/F	43.7	3	nr	6	N/C/A	Hearing impairment, lower limbs paresthesia, stuporous, tetraparesis	Unclear	Partial recovery
8	Goselink et al. 2012	49/M	42	4	25	7	N/C/A	PNP lower limbs, dysphagia	Vomiting, non-compliance	Partial recovery
9	Cerutti et al. 2013	33/M	47	4.5	91	nr	-/C-/-	Dysarthria, tetraparesis	Psychogenic anorexia	partial recovery
10	Saab et al. 2013	27/F	nr	0.75	nr	0.75	N/C/-	Lethargy	Unclear	Full recovery
11	Manatakis 2014	51/M	41.5	2	nr	2	-/C/-	Weakness, drowsiness	Vomiting	Death
12	Landais et al. 2014	31/M	47	2	38	5	N/C/A	Papillary hemorragy, photophobia, anxious episodes	Vomiting, non-compliance	Partial recovery
13	Kroell et al. 2015	55/M	67	3	50	24	N/C/A	PNP lower limbs, dysphagia	Vomiting	Partial recovery

N nystagmus, *C* confabulation, *A* ataxia, *PNP* peripheral neuropathy, *nr* not reported

impact of thiamine deficiency after SG is unknown. In the last 8 years, only 13 cases of WE have been reported after SG. WE after bariatric surgery seems to occur more often in women, but whether there is a gender-specific predisposition is currently not clearly understood [9]. In the published data, the patients were between 24 and 55 years old.

Time of Neurological Symptoms

Early Onset WE occurred within the first 2 weeks after SG because of functional gastric stenosis, dietary non-compliance, and prolonged postoperative vomiting. A laparoscopic gastrojejunostomy was performed, and the SG was converted to a mini-gastric bypass. All cognitive impairments disappeared after 6 months [13]. Saab et al. also reported a case of WE after SG within the first 3 weeks, but without the presence of vomiting or any other possible explanation [15]. These early cases are often related to gastric outlet obstruction similar to the reported cases after gastric bypass.

Late Onset In all other circumstances, WE occurred later after SG, within the first 7 months, and after bariatric surgery without any signs of stenosis, obstruction, or kinking of the gastric remnant. The median onset of clinical symptoms was 3 months after surgery. This wide variability with respect to timing of onset is also not understood and requires further studies.

Clinical Manifestations of WE

The classic triad of WE includes encephalopathy, oculomotor abnormalities (nystagmus, ophthalmoparesis, conjugate gaze palsies), and gait ataxia, as presented in our case. This triad was only reported in 46 % of identified SG cases. The most common sign was altered mental status. However, WE might be underestimated because the classic triad has been seen in only 16 % of patients; frequently, not all of the elements are present and a subclinical deficiency with nonspecific symptoms is common [6]. Singh et al. highlighted the frequency of atypical neurologic symptoms [9]. Peripheral neuropathy (dry beriberi) and a sensorimotor distal axonopathy are often associated with calf cramps, muscle tenderness, burning feet, and autonomic features and have also been described to predominantly involve the lower extremities. The diagnosis of WE can be missed when alertness is placed in the evidence of typical symptoms [9, 35]. A wide variety of symptoms are described. The symptoms of subclinical thiamine deficiency are often nonspecific and include fatigue, lethargy, restlessness, and headaches, as mentioned in most case reports. Other associated atypical clinical signs and symptoms are peripheral neuropathy, vestibular dysfunction without hearing loss, dysphagia, and depression; Korsakoff psychosis reflected the combination of the symptoms and identified the challenge of making the diagnosis. Cardiac involvement may manifest as congestive cardiac failure (wet beriberi) but was not documented in the reviewed case reports.

Differential Diagnosis

Neurological side effects of bariatric surgery are mainly associated with deficiencies in vitamins of the B-group, in particular vitamin B 12, folic acids, vitamin D, and minerals copper and zinc [36]. Depending on the type of nutritional deficiency, any part of the central or peripheral nervous system may be involved and can lead to ongoing unresolved, debilitating neurological complications. Acute post-gastric reduction surgery (APGARS) neuropathy was used to describe such phenomenon [37]. Disturbance of the peripheral nervous system is mainly peripheral (poly)neuropathy, myelopathy, or myeloneuropathy. It is important that bariatric surgical teams need to impart this information to patients and their primary care teams, because the presence of signs and symptom of WE can be misdiagnosed and treated inappropriately while progressive neurological damage occurs.

Diagnostic Tools

Post-bariatric WE mainly remains a clinical diagnosis. Clinical symptoms and signs of WE can be very vague early after bariatric surgery but mostly relate to prolonged postoperative vomiting and a rapidly altered mental status. Mainly, it is not possible to detect direct connection between nutritional deficiencies and the occurrence of neurological disorders [38]. There are no laboratory examinations that are specific for WE. In most of the reported cases, serum thiamine levels or urinary thiamine excretion were reduced [8]. In contrast, an average serum thiamine level does not exclude the presence of WE. The sensitivity and specificity of blood and urinary tests in symptomatic patients are limited, technically difficult, time-consuming, and unreliable [9, 33]. In our patient, serum thiamine analysis was not performed before treatment.

Radiological examination by MRI is the diagnostic tool of choice, with a sensitivity of 53 % and a specificity of 93 % for WE [39, 40]. Typical MRI findings include increased T2, proton density, or fluid-attenuated inversion recovery (FLAIR) signals in the periventricular regions. In our patient, typical findings included areas of increased T2 signal surrounding the aqueduct and third ventricle and within the medial thalamus and mammillary bodies, as mentioned in another six cases. Additionally, MRI may differ among patients with and without alcohol abuse [41]. MRI findings were not pathbreaking in all cases. In four cases, no radiographic abnormalities were present. In general, CT is an insensitive body imaging for the diagnosis of WE [42]. Cerebrospinal fluid tests and electroencephalograms can be within normal ranges.

Treatment

Immediate parenteral thiamine administration is the treatment of choice [43]. The optimal regimen is a matter of debate, but should be performed early and aggressively. In early stages, the syndrome is very responsive to thiamine treatment. The treatment management differed and was not uniform in published cases. A standard regimen in severe deficiencies of thiamine therapy is 500 mg i.v. every 8 h for two consecutive days and 500 mg i.v. intramuscularly once daily for an additional 5 days in combination with magnesium and other vitamins of the B-group. In general, infusions containing dextrose without thiamine should be avoided because they can trigger or worsen WE. Long-term oral administration of 50 to 100 mg of thiamine per day is recommended after the parenteral therapy with thiamine for the first weeks [44].

The prevention of WE syndrome could be improved by oral administration of thiamine to patients before and after SG and notably to patients at risk; prophylactic thiamine should be initiated in patients with symptoms of gastrointestinal distress after bariatric surgery [7].

Outcomes

The course of post-bariatric WE varies widely and is unpredictable [8]. Only one third of the patients with WE after SG achieved complete recovery. In addition, most patients had residual neurologic deficits, such as short- and long-term memory loss, ataxia, and peripheral neuropathy that could persist over months. Despite early initiation of treatment, complete recovery is rare. In the overviewed case reports, two patients died because of multiorgan and respiratory failure. Our patient had an incomplete recovery even after 16 months. His sequela was impaired memory and learning capabilities. In general, approximately 80 % of patients with WE who survive develop Korsakoff syndrome.

Several limitations exist in this study. It is a retrospective study that reviews a small sample size over a short period of time. A systemic review of case reports is also limited by the lack of complete information or publication or selection bias. However, it addresses an important issue about prompt diagnosis, including a workup of the clinical essentials of WE in patients undergoing SG to ensure a better outcome in understanding this topic (Table 2). The predictors of this important

Table 2 Overview red flags of WE after SG

Risk factors	Recurrent emesis
	Non-compliance and inadequate vitamin supplementation
	Preoperative vitamin B deficiencies
	Surgical complications (stenosis)
	Parenteral feeding, caloric carbohydrate diet
	Comorbidities: alcohol consumption, type 2 diabetes, hepatic steatosis (NAFLD), delayed gastric emptying
Time of neurological symptoms	Early: within 2–6 weeks (stores can be depleted)
	Late: within 7 months with variability (usually 3–5 months)
Clinical manifestations	Wernicke encephalopathy (ocular dysfunction, gait ataxia, encephalopathy) classic triad not often seen
	Altered mental status changes
	Korsakoff syndrome (amnestic-confabulatory syndrome)
	Peripheral neuropathy and polyradiculopathy
	Nonspecific symptoms: fatigue, lethargy, restlessness
	Atypical symptoms: vestibular dysfunction without hearing loss, dysphagia, depression
Diagnostic tools	Clinical diagnosis
	Laboratory examinations may be helpful, but not specific, serum thiamine levels may be reduced
	MRI may show increased T2 signals in periventricular regions
Differential diagnosis	Other nutrient deficiencies: vitamin B12, copper (folate, niacin, vitamin E)
Treatment	Wernicke encephalopathy: 500 mg of thiamine IV 3 times a day for 2 days, followed by 500 mg/day IV or IM for 5 days in combination with magnesium and other vitamins of the B group, followed by long-term oral administration of 50 or 100 mg/day [33]
	Periodic follow-up visits with targeted implementation of nutrient deficiencies
Outcome	Complete recovery is rare

NAFLD non-alcoholic fatty liver disease

neurological complication need to be evaluated in long-term studies.

Moreover, this review article highlighted the importance of thorough examination and supervision of bariatric patients after surgery. Knowledge of the clinical picture, early identification, and appropriate treatment of thiamin deficiencies may help to reduce the development of neurological disturbances.

Conclusion

Health professionals need to be vigilant for the heterogeneity symptoms of WE and the likelihood of progression over time. The highest suspicion for WE remains for patients with prolonged postoperative vomiting and a rapidly altered mental status, even at any time, and without signs of obstruction or functional stenosis after SG. The supplementation of thiamine using multivitamin compounds might be beneficial to lower potential risks for severe deficiencies of thiamine leading to a WE.

Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no competing interests.

Statement of Informed Consent Informed consent was obtained from all of the individual participants included in the study.

Statement of Human Rights All of the procedures performed in the study involving human participants were in accordance with the ethical standards of the institutional research committee and the 1964 Helsinki.

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