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Wernicke Encephalopathy after Sleeve Gastrectomy: A Case Report

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Abstract

The number of obese patient's increases, and bariatric procedures have become more common. The total number of these procedures in Poland was 1958 in 2016. Malabsorption, rapid weight loss and vomiting can predispose patients to nutritional deficiencies following surgery. After bariatric surgery, the majority of vitamin and mineral deficiencies seem to be asymptomatic. However, in some cases both reversible and irreversible neurological symptoms may be observed. Punchai et al. observed that the incidence of neurological complications due to vitamin B deficiency was 0.7%. Wernicke Encephalopathy (WE) is an acute disorder resulting from thiamine deficiency and is characterized by neuropsychiatric symptoms such as ataxia, ophthalmoplegia and confusion. We present a case of a 29-year-old woman who developed WE after sleeve gastrectomy.

Keywords: Encephalopathy; Gastrectomy; Deficiency

Introduction

A 29-year-old obese woman was admitted to the Department of Neurology in Zabrze, Poland due to dizziness and visual disturbances. The patient had undergone sleeve gastrectomy two months earlier. As a result, the patient lost 25 kg following the procedure and the body mass index (BMI) was reduced from 46.5 to 36.5. The postoperative course was complicated by prolonged vomiting and anastomotic stricture was suspected. The patient had a history of Lyme disease and was treated with ceftriaxone. Esophagogastroduodenal transit showed good contrast pass, abdominal ultrasound revealed no abnormalities and surgical complications were ruled out. During hospitalization, the patient developed oscillopsia and an episode of high blood pressure. The subject was assessed by an otolaryngologist and labyrinthine vertigo was ruled out. The patient was started on betahistine (10 mg 3 times/day) and vinpocetine (25 mg twice/day). However, no improvement was observed. A brain computed tomography (CT) with contrast revealed decreased white matter density in the occipital lobe. The posterior reversible encephalopathy syndrome (PRES) was suspected.

Case Report

After neurological examination, the patient was transferred to the Department of Neurology in Zabrze. On admission, neurological assessment revealed oscillopsia, nystagmus in all gaze positions (with predominant vertical nystagmus) and abduction deficit in the right eye. Brain magnetic resonance imaging (MRI) was performed and the PRES syndrome and brain tumour were ruled out. Electroencephalogram, blood flow in transcranial Doppler and carotid duplex-Doppler ultrasound were within normal ranges. Several days later, the patient developed mild ataxia of the right limbs and paresthesia of the lower limbs. Muscular strength was decreased in the lower limbs and the patient could not ambulate even with assistance. The patellar reflexes were absent. Extensor plantar reflex was increased on the right (positive Babinski sign). Examination of superficial sensation revealed damage at the D8-D11 levels on the left side. Electroneurography showed slow conduction velocity in the peroneal nerves and femoral nerve axonal loss. The patient was also assessed by an ophthalmologist and an endocrinologist. At that time, the differential diagnosis included paraneoplastic syndrome, autoimmune diseases, neuro infection and WE. Abdominal ultrasound, chest X-ray and *abdominal and pelvic CT* were normal. The patient was tested for the presence of onconeural antibodies (anti-Hu, anti-Ri, anti-Yo). The results were negative. The paraneoplastic syndrome was ruled out.

Lumbar puncture was performed and the analysis of the cerebrospinal fluid (CSF) was within the normal range. Latex agglutination test was negative. Test results for infectious diseases are presented in Table 1. The results of other laboratory tests are presented in Table 2. Visual evoked potentials revealed bilateral abnormal latency of P100 (up to 115 ms in the left eye and 114 ms in the right eye). The patient was started on methylprednisolone. However, no improvement was observed. Oligoclonal bands were not found in the serum or the CSF. Myelin oligodendrocyte glycoprotein (MOG) antibodies, aquaporin-4 antibodies (AQP4) and N-methyl-D-aspartate receptor (NMDA) antibodies were negative. The patent was diagnosed with WE and lumbar radiculomyelopathy. Supplementation of vitamins (B1, B12, B6, C, folic acid), calcium, iron, copper and zinc were given (Table 3). Two weeks after IV administration of thiamine, nystagmus improved, and the patient condition improved. Electroneurography, the analysis of the CSF and brain MRI were normal. No adverse effects were observed except for allergic skin reaction. Due to the occurrence of this reaction, the dose of thiamine was reduced, and the oral route of administration was chosen. At discharge, the patient was able to ambulate with minimal assistance. Residual nystagmus and ataxia were

Test	Material	Results
HIV test	Serum	Negative
Borrelia IgG antibodies (Western Blot)	CSF	Negative
Borrelia IgM antibodies (Western Blot)	CSF	Negative
Borrelia IgG antibodies (Western Blot)	Serum	Positive
Borrelia IgM antibodies (Western Blot)	Serum	Negative
HSV IgG Ab	Serum	Positive
HSV IgM Ab	Serum	Negative
HSV IgG Ab	CSF	Positive
HSV IgM Ab	CSF	Negative
CSF: Cerebrospinal Fluid		

Table 1: Test results for infectious diseases.

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still present. The patient was prescribed 50 mg thiamine P.O. 3 times/day. Rehabilitation was recommended.

Discussion

Wernicke Encephalopathy (WE) is a disease that results from vitamin B1 deficiency. Most commonly, it occurs in patients with chronic alcohol abuse. It was also reported following bariatric surgical procedures, including sleeve gastrectomy. Higher risk is observed in young women with vomiting [1-6]. Ziegler et al. indicated the need for vigorous supplementation of thiamine in patients with persistent vomiting after surgical bariatric procedures [7]. The condition usually develops within 4 to 12 weeks after bariatric procedure. However, it was found that it also occurs many years after surgery [2,8]. The disease is observed mainly in patients with weight loss greater than 7 kg per month [6]. Thiamine (vitamin B1) is absorbed mostly in the upper jejunum [9]. Its biologic half-life ranges from 10 to 20 days [10]. The total amount of thiamine in the body is approximately 30 mg [11]. For the maintenance of thiamine reserve, regular intake is required. In the case of malnutrition, intestinal thiamine absorption can be reduced by 70% [9].

The classic clinical symptoms of WE include ataxia, mental confusion and eye movement abnormalities [12]. There is no clinical test to diagnose WE. Caine et al. proposed diagnostic criteria with high sensitivity and specificity (85% to 100%) [13] which were later confirmed by Galvin et al. They recommended the same criteria for alcoholic and non-alcoholic patients [14]. To be diagnosed with WE, the patient needs to meet two of the following criteria:

1) Dietary deficiencies

2) Ocular abnormalities

3) Cerebellar dysfunction and

4) Either an altered mental state or mild memory impairment [14,15].

The diagnosis of WE can be supported by MRI and the determination of vitamin B1. Reversible cytotoxic oedema is the most characteristic MRI lesion in this syndrome [16]. On MRI imaging, the pathologic alternations are symmetrical. They are located in the thalami, tectal plate, mamillary bodies and periaqueductal area [14,16]. A normal MRI does not rule out the diagnosis of WE [17,18]. Our patient presented with weakness, oscillopsia and vertigo followed by development of ataxia and paresthesia. The brain MRI was normal. There are no established treatment recommendations. The recommendations given by different authors are provided in Table 4.

Test	Results	Range
B12 [pg/ml]	540.9	191-663
Fe [umol/l]	24.8	5.83-34.5
Ferritin (4th July) [umol/l]	796.9	15-150
Ferritin (8th August)	354.6	15-150
CRP (17th June) [mg/l]	77.35	<5
CRP (3rd July) [mg/l]	3.14	<5
Mg [mmol/l]	1.01	0.75-1.25
Ca [mmol/l]	2.3	2.15-2.5
Ca^{2+} [mmol/l]	1.24	1.1-1.35
ALT [U/L]	40.4	<41
AST [U/l]	58.6	<40
RBC [mln/ul]	5.40	4.2-5.4
MCV fl	79	78-100
MCH pg	27	27-31
MCHC g/dl	33.6	32.0-36.0

CRP: C-Reactive Protein; ALT: Alanine Transaminase; AST: Aspartate Transaminase; RBC: Red Blood Cells; MCV: Mean Corpuscular Volume; MCH: Mean Corpuscular Haemoglobin; MCHC: Mean Corpuscular Haemoglobin Concentration

Table 2: Laboratory test results.

Supplements	Dose
B1	500 mg 3 times/day (3 weeks)
B1/B6	100 mg/100 mg/day
B12	1000 mg/day (1 week)
Folic acid	15 mg/day
Vitamin C	500 mg/day
Zinc gluconate/copper gluconate	38 mg/2.13 mg/day
$FeSO_3$	100 mg/day
$CaCO_3$	1000 mg twice/day
Lactobacillus rhamnosus R0011, *Lactobacillus helveticus* R0052	Twice/day

Table 3: Therapeutic supplementation in our patient.

Recommendations	Dose	Application	Duration
Sechi et al.	500 mg 250 mg if effective response is observed	Dissolved in 100 ml of normal saline, 3/day IV 1/day IV/IM	2-3 days 3-5 days, or until no further improvement
Ziegler et al.	100 mg Later 100 mg	1/day parenteral 1/day PO	7-14 days Until neurological symptoms resolve
Galvin et al.	200 mg	3/day IV	Until no further improvement

Table 4: Recommendations given by different authors.

Conclusion

According to some authors, thiamine doses lower than 200 mg/d are insufficient. Our patient was given 500 mg of thiamine 3 times/day and after 5 days the improvement was still unsatisfactory. The treatment was continued with a high dose of thiamine. Patient's condition improved gradually, particularly after two weeks. However, after 3 weeks following the administration of thiamine, we observed skin allergic reaction and thiamine was discontinued. The dose of thiamine was reduced, and the oral administration route was chosen. Other adverse effects were not observed. The upper tolerable intake level for thiamine has not been established. According to the literature data, oral application of 500 mg/day of thiamine for more than one month showed no adverse effects. Insufficient or delayed administration of thiamine may contribute to residual symptoms even after treatment. In patients with untreated WE, the mortality rate reaches 20%. Although WE seem to be a rare complication of bariatric procedures, prevention of nutritional deficiencies is crucial. Delay in the diagnosis and treatment of WE result in a worse prognosis. As the number of patients after bariatric surgeries gradually increases, awareness of the potential adverse effects is of great importance.

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