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Case Report

A Case of Wernicke Encephalopathy in Post Sleeve Gastrectomy: A Case Report

Sultan Mahja Alanazi[1], Hessa Abdullah Alshammari[1], Fahad Khalid Alquwaiay[1], Abdulhafeez Osman Eltoum[1], Shaimaa Moustafa Khairat[2], Atheer Mohammed Alanizy[3], Hussain Gadelkarim Ahmed[4*]

[1]Department of Medicine, King Khalid Hospital, Hail, Saudi Arabia
[2]Department of Neurology, King Khalid Hospital, Hail, Saudi Arabia
[3]College of Medicine, University of Ha'il, Ha'il, Saudi Arabia
[4]Professor, Department of Histopathology and Cytology, FMLS, University of Khartoum, Sudan

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***Corresponding Author:** Hussain Gadelkarim Ahmed
Professor, Department of Histopathology and Cytology, FMLS, University of Khartoum, Sudan

Abstract

Case summary: A 19-year-old Saudi male morbidly obese patient (BMI 48 kg/m^2). The patients underwent sleeve gastrectomy 5 months ago and cholecystitis 1 month ago. The patient presented to our emergency department (ER) with all limb numbness in the last 3 weeks, difficulty walking, blurred vision, headache, and nausea for the last week. The patient was diagnosed with Wernicke encephalopathy based on his symptoms, physical signs, and classical MRI brain findings. ***Conclusion:*** Bariatric surgery can complicate Wernicke encephalopathy, associated with thiamine deficiency. Prophylactic thiamine supplements can prevent the subsequent bariatric WE condition.

Keywords: Wernicke Encephalopathy, Sleeve Gastrectomy, Obesity, Bariatric surgery.

INTRODUCTION

Obesity is a significant health concern worldwide, with increasing prevalence reaching an epidemic state [1]. The rapid increase of morbid obesity among children and adolescents emerged as a public health concern in various geographical areas worldwide [2].

Bariatric surgery is the most successful treatment for morbid obesity. Roux-en-y gastric bypass and laparoscopic sleeve gastrectomy are the most commonly presented surgeries [3]. In recent years there has been an increasing desire for bariatric surgeries in the Middle East and North Africa, though the procedure is not fully understood in the region [4]. Patients subjected to bariatric surgery are at high risk of nutritional deficiencies requiring a proper follow-up and appropriate supplementation [5].

Wernicke encephalopathy (WE) is an acute neurological disorder characterized by a clinical triad of ophthalmoparesis with nystagmus, ataxia, and confusion. WE is regarded as a life-threatening condition triggered by thiamine deficiency that mainly distresses peripheral and central nervous system [6]. It was believed that thiamine deficiency can result from bariatric surgery [7].

CASE REPORT

A 19 years-old Saudi male morbidly obese patient (BMI 48 kg/m^2). The patients underwent sleeve gastrectomy 5 months ago and cholecystitis 1 month ago. The patient presented to our emergency department (ER) with all limb numbness in the last 3 weeks, difficulty walking, blurred vision, headache, and nausea for the last week.

The patient stated that his surgery went uneventful apart from Cholecystitis one month ago, which was treated conservatively. Three weeks ago, he started to have paresthesia, weakness, and even difficulty maintaining balance and difficulty maintaining concentration with others.

There is no history of fever, seizures, syncope, decreased level of consciousness, personality changes, or hallucinations, and no history of trauma, apart from gastric symptoms, vomiting, diarrhea, dysphagia, or odynophagia.

Past Medical History: he was morbidly obese with a BMI of 48 kg/m^2 and lost 25 kg in the last 5 months.

Regarding family and social history: he is a college student, has no record of recent travel, no contact with sick patients, and has no history of alcoholism or substance abuse.

Medication history: he used to take multivitamin tablets and Vitamin b12 injections which he stopped 2 months ago.

On examination:

The patient was vitally stable, with normal BP and Pulse, and he was afebrile.

The patient was conscious and oriented to time, place, and person, but he had difficulty being alert and maintaining concentration with the physician.

He had Restricted extraocular movements in all directions bilaterally. His fundus examination shows bilateral papilledema. His pupils were reactive equally to light.

His Power (using the MCR grading system) is 4/5 in upper limbs and 3/5 in lower limbs.

He had sensory impairment in the lower limb. The sensation was lost up to the umbilicus.

While he has intact sensation in both upper limbs. Proprioception and vibration were unchanged in the upper and lower limbs.

He had hyperreflexia in the upper limbs and hyporeflexia in the lower limbs with a negative Babinski sign.

Cerebellar and gait examinations were challenging to assess as the patient had bilateral lower limb weakness. Systemic examination was unremarkable.

According to his symptoms and clinical findings, a presumed diagnosis of Wernicke encephalopathy was made, and he was admitted by the neurology team as a case of WE for further workup and management.

Basic labs were done (CBC, Renal Profile, LFT, Electrolytes, blood glucose, coagulation profile) within normal range. TFT, VIT b12, VIT D, ESR, CRP, Lipid Profile, and HBA1C were within normal. VIT B1 level was markedly low at 11.4 ng/mL (35–91 ng/mL), CT brain in ER was done, which was unremarkable, MRI brain W/ contrast was made which revealed symmetrical increased T2/flair signal involving the mammillary bodies, dorsomedial thalami, tectal plate, periaqueductal area and to a lesser extent around the third ventricle (see Images 1 & 2).

The patient was diagnosed with Wernicke encephalopathy based on his symptoms, physical signs, and classical MRI brain findings. The patient started on a parenteral Thiamine loading dose of 500 mg three times daily for two consecutive days and 250 mg IV once daily for five days, combined with other vitamin B complex tabs.

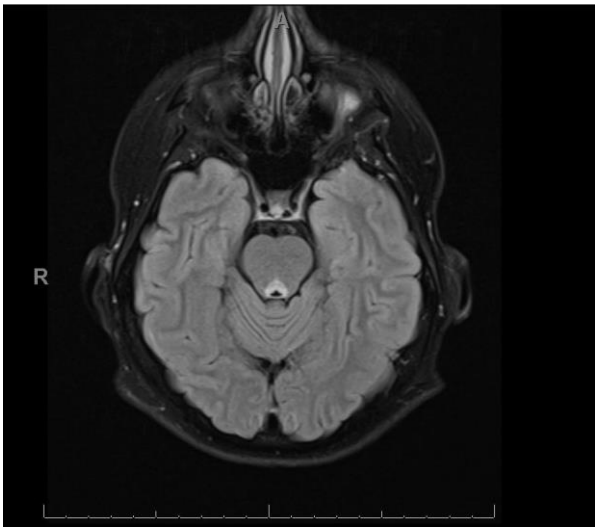

Image 1

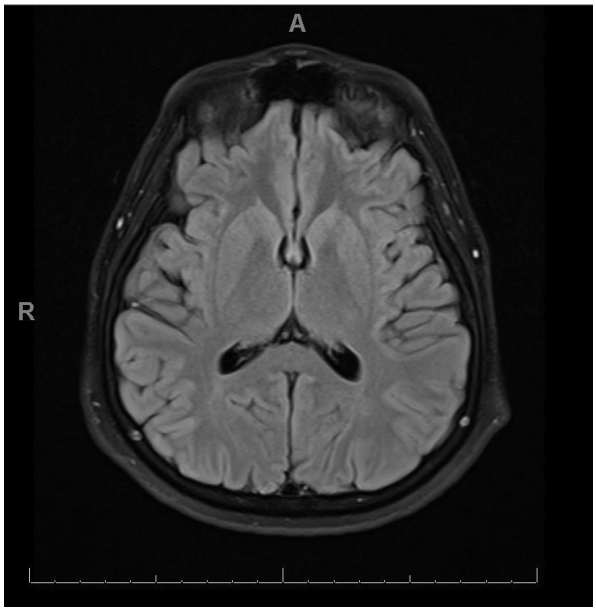

Image 2

DISCUSSION

Thiamine deficiency responsible for Wernicke encephalopathy is believed to be related to bariatric surgery. As the vertical sleeve gastrectomy (VSG) becomes progressively prevalent, its impact on postoperative micronutrient levels, such as thiamine, becomes more crucial. The distressingly high incidence of thiamine deficiency in postoperative SG patients can

lead to dangerous compilations, including WE. Therefore, it is essential to identify predictive demographic, postoperative, and behavioral factors. These necessitate the undertaking of appropriate measures to prevent thiamine deficiency associated with VSG [8]. It was reported that thiamine deficiency can cause permanent neurologic deficits [9].

As thiamine deficiency can be chronic after bariatric surgery, prophylaxis treatment is required. It was found that about 30-35% of bariatric WE patients don't follow bariatric intervention guides that suggest the prophylactic thiamine treatment plans [10]. Moreover, extreme dietary restrictions, hyperemesis, infections at the treatment site, and different somatic situations can result in potential risk for subsequent bariatric WE [11].

Patients with bariatric WE are usually younger and often present with vomiting, ataxia, altered metal status, and eye movement. Younger age appeared to safeguard against mental changes. The WE treatment was habitually inadequate, particularly disregarding small-scale parenteral thiamine levels. Thiamine levels must be checked and treated sufficiently with parenteral thiamine supplementation [10].

CONCLUSION

Bariatric surgery can complicate Wernicke encephalopathy, which is associated with thiamine deficiency. Prophylactic thiamine supplements can prevent the following bariatric WE condition.

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