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WERNICKE'S ENCEPHALOPATHY AS A COMPLICATION OF THIAMINE DEFICIENCY IN A PATIENT FOLLOWING BARIATRIC SURGERY

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ABSTRACT

Purpose: Wernicke's encephalopathy is a severe and life-threatening condition resulting from thiamine deficiency. Among its characteristic neurological symptoms are ataxia, ophthalmoplegia, and altered mental status. This article aims to present Wernicke's encephalopathy as a serious neurological disease, describe its clinical manifestations, diagnostic approach, discuss effective treatment, and emphasize the importance of monitoring vitamin and micronutrient levels in bariatric surgery patients.
Case description: A case of a 29-year-old female patient was presented, showing symptoms of Wernicke's encephalopathy after bariatric surgery. An MRI scan revealed brain changes consistent with Wernicke's encephalopathy. Serum thiamine and retinol levels were markedly reduced. The patient underwent treatment with thiamine and was additionally administered other supplements and medications.
Comment: This case underscores the significance of monitoring vitamin and micronutrient levels in bariatric surgery patients to prevent serious neurological complications. Thiamine replacement therapy was effective in resolving the patient's neurological symptoms.

KEYWORDS

Wernicke's Encephalopathy, Thiamine, Bariatric Surgery

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Introduction

Wernicke's encephalopathy, caused by a deficiency of vitamin B1, was first described in 1881 by Carl Wernicke as a life-threatening neurological disease. This condition is characterized by neurological and psychiatric symptoms, which can vary and lead to misdiagnosis. The characteristic clinical triad of Wernicke's encephalopathy consists of ataxia, nystagmus, and altered consciousness. Ataxia is caused by damage to the cerebellar vermis. Patients often present with a wide-based gait and may even experience complete loss of independent mobility. Limb clumsiness is less common but can occur, as well as flaccid paralysis due to peripheral neuropathy. Oculomotor disturbances result from damage to the oculomotor and abducens nuclei in the midbrain and pons. Horizontal nystagmus is the most common ocular manifestation. Other ocular dysfunctions include abnormalities in pupillary reactions. In the acute phase of the disease, an altered state of consciousness is often observed and can take various forms, such as delirium, dysphoria, or apathy. There may be emotional instability manifested as mood swings, dysphoria, or apathy. The pathology also affects attention, speech, and memory functions. Severe neurological impairments or coma are less frequent manifestations. Untreated Wernicke's encephalopathy can progress to Korsakoff's syndrome or alcoholic amnestic disorder associated with damage to the thalamus. Fresh memory impairment occurs while remote memory and intelligence remain intact. Confabulations or false memories may also occur. Studies indicate that Korsakoff's syndrome occurs in about one-quarter of individuals with non-alcoholic Wernicke's encephalopathy [1, 2]. Unlike symptoms observed in Wernicke's encephalopathy, amnesia in Korsakoff's syndrome is irreversible [3].

The diagnosis of Wernicke's encephalopathy relies on clinical observation of symptoms, since not all patients will exhibit the full triad.

Caine's criteria are helpful for diagnosis: patients should show at least 2 out of 4 listed criteria (table 1) [4].

Table 1. Caine's criteria

CAINE'S CRITERIA
1. Oculomotor abnormalities
2. Dietary deficiencies
3. Cerebellar dysfunction
4. Memory impairments/alterations

Measuring thiamine levels in blood can be useful, but abnormal levels do not always confirm the diagnosis alone. Thiamine deficiency may be indicated by increased lactate/pyruvate ratio of rest or after physical exertion. Reduced transketolase activity in erythrocytes may also be observed. Magnetic resonance imaging (MRI) imaging of the brain is also useful in diagnosis and typically correlates with the patient's clinical condition. Neuroradiological images may show a decrease in white and gray matter volume, as well as changes within the ocular motor nuclei, cerebellar vermis, and vestibular nuclei. The symptoms comprising Wernicke's triad are associated with changes within the ocular motor nuclei, brainstem, cerebellar vermis, vestibular nuclei, reticular activating system of the thalamus, and mammillary bodies. Head MRI reveals hyperintensity on T2-weighted fluid-attenuated inversion recovery (FLAIR) or diffusion-weighted imaging (DWI) around the periventricular region of the thalamus, hypothalamus, floor of fourth ventricle, and periaqueductal area. To differentiate from other CNS disorders, a complete blood count and comprehensive metabolic panel are also recommended [5, 6, 7].

The prevalence of Wernicke's encephalopathy based on postmortem studies is estimated to be 1-3%, but these numbers may be underestimated due to failure to diagnose during life based on clinical symptoms. Wernicke's encephalopathy occurs more frequently in males [3].

The most common cause of Wernicke's encephalopathy is alcohol abuse. This disease along with Korsakoff syndrome occurs in 12.5% of alcoholics [8]. The condition results from impaired thiamine absorption by the gastrointestinal tract, leading to deficiency [9]. An increasing number of cases involve non-alcoholic causes unrelated to alcohol abuse. This disease can occur in individuals with malnutrition, eating disorders, malabsorption syndromes, hypothyroidism, and after bariatric surgery, pregnant women experiencing uncontrollable vomiting, long-term dialysis patients, and those with gastrointestinal tumors or AIDS [3,10].

Patients without an alcoholic etiology exhibit more severe clinical symptoms and greater changes on magnetic resonance imaging (MRI) [11]. Thiamine deficiencies can occur due to increased physiological demand during growth spurts, recovery periods, intense physical exertion, alcoholism, eating disorders, bulimia, nutritional deficit following resection of part of the small intestine, endoscopic tube placement, Crohn's disease, ulcerative colitis, and celiac disease.

The treatment focuses on addressing the underlying deficiencies through intravenous administration of thiamine at a dose of up to 100-300 mg three times a day [12]. The therapy can then be continued orally after stabilization of the patient's condition.

Untreated Wernicke's encephalopathy can lead to irreversible brain changes that account for 20% of patient deaths [13]. Prognosis depends on the severity and advancement of the disease at diagnosis, as well as the duration and effectiveness of treatment. Delayed regression of symptoms has been observed in patients with gait ataxia and balance disturbances. Prompt initiation of treatment leads to resolution of oculomotor symptoms within days or weeks; however, horizontal nystagmus may persist for several months in many patients [14,15].

Case Report

A 29-year-old female patient was transferred from the Surgical Department to the Neurology Department due to headaches, dizziness, and visual disturbances. She had undergone bariatric surgery (roux-en-y gastric bypass) three months prior on the Surgical Department. Two weeks after the surgery, she developed daily vomiting and was treated on the Internal Medicine Department. A CT scan of the head at that time did not show any abnormalities, but an upper gastrointestinal endoscopy revealed gastric ulcers.

Upon readmission to the Surgical Department 11 weeks after the initial surgery, the patient was for ophthalmological consultation due to a sudden deterioration in visual acuity. The examination revealed bilateral optic disc edema. She was transferred to the Neurology Department where a clinical examination showed preserved light perception, horizontal nystagmus when looking sideways, upward gaze-evoked nystagmus, and limited abduction of her left eye. Muscle strength and tone in her upper lower extremities were decreased, with absent knee and ankle reflexes The Romberg's test was positive. The patient was unable to sit independently.

On the fifth day of hospitalization Internal Medicine Department, she started exhibiting altered consciousness and function with drowsiness and illogical thinking. Magnetic Resonance Imaging (MRI) with contrast of her brain revealed changes in the cerebellar vermis and periaqueductal gray matter as well as medial portions of both thalami which correspond to Wernicke's encephalopathy-related lesions. EM ruled out polyneuropathy.

The patient's vitamin levels – thiamine (B1), cobalamin (B12), tocol (E), retinol (A) were measured. Thiamine and retinol levels were significantly low (table 2).

Table 2. Results of thiamine and retinol test of the patients.

Examination:	Range:	Reference range:
Thiamine	35 mmol/l	50-75 mmol/l
Retinol	18 µg/100 ml	30-70 µg/100 ml

Treatment

Wernicke's encephalopathy requires immediate pharmacological intervention due to the possibility of permanent neurological deficit and progression to Korsakoff's syndrome. Causal treatment involves supplementing thiamine deficiency. Before starting treatment, thiamine levels in the blood should be measured, and they should be between 50-75 mmol/L. Thiamine supplementation at a dose of 600 mg per day is recommended, either intravenously or intramuscularly. Benfotiamine (300 mg per day) can be administered instead of thiamine. Glucose should not be given during treatment, as it increases the body's demand for thiamine. After bariatric surgery, thiamine levels should be monitored and supplemented for at least 6 months. [5] [16]

In the described case during hospitalization, anticoagulant therapy was also administered, electrolyte and folic acid deficiencies were corrected. After an ophthalmologic consultation that revealed changes in the eye fundus image corresponding to optic neuritis, steroids (methylprednisolone) were initiated for treatment. The patient was observed to have anxiety disorders during psychiatric assessment; therefore, anxiolytic treatment (fluoxetine 20 mg per day) was used. In neuropsychological evaluation, episodes of heightened anxiety accompanied by visual disturbances, weakened and scattered attention and concentration, difficulty in recalling necessary words, disharmonious learning process when acquiring new information were noted, however other functions remained intact. After assessing vitamin B1 levels in the blood, intramuscular administration of thiamine was initiated with a preparation consisting of a mixture of B-group vitamins – thiamine (100 mg), pyridoxine (100 mg), and cyanocobalamin (1 mg). The medication was given twice daily, resulting in a total daily intake of 200 mg of thiamine for the patient. Quetiapine was also introduced to alleviate psychiatric symptoms. Motor rehabilitation began simultaneously. The patient left the Neurology Department in good general condition with normal visual acuity without oculomotor or atactic disturbances, she could walk unassisted.

Discussion

The presented case represents a severe neurological complication (WE) as a result of thiamine and retinol deficiencies in a patient following bariatric surgery. In our opinion, dysfunctions of the central and peripheral nervous systems will increasingly become a clinical problem in patients undergoing such procedures, as the number of these surgeries continues to rise. Due to significant and life-threatening vitamin and micronutrient deficiencies in these patients, monitoring their serum levels is crucial. In our daily clinical practice, we commonly observe deficiencies in cobalamin (subacute combined degeneration, polyneuropathy, cognitive impairments), thiamine (WE), retinol (blindness), and tocopherol (cerebellar ataxia). It is important to monitor total and ionized calcium and iron levels, as well as zinc, copper, and selenium levels. In the described case, treatment included thiamine supplementation at a daily dose of 200 mg, resulting in rapid regression of ophthalmoplegia symptoms and improvement in visual acuity.

Conclusions

Wernicke's encephalopathy resulting from vitamin B1 deficiency is most commonly observed in patients who abuse alcohol. However, there has been an increasing number of cases caused by other non-alcoholic factors in recent years. A significant proportion of these cases are associated with vitamin and micronutrient deficiencies, due to bariatric surgeries that are being performed more frequently for the treatment of obesity. Therefore, it is important to monitor the serum concentrations of these substances and promptly correct any deficiencies to prevent serious neurological complications. Monitoring and treatment should be conducted for at least 6 months.

Abbreviation:

WE - Wernicke's encephalopathy,
MRI - Magnetic Resonance Imaging,
CT - Computed Tomography
FLAIR - fluid-attenuated inversion recovery,
DWI- diffusion-weighted imaging,
CNS - central nervous system,
AIDS - Acquired Immune Deficiency Syndrome
EMG - Electromyography

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