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Letter to the Editor / Editöre Mektup

Two Cases of Wernicke Encephalopathy Related with Obesity Surgery and Recurrent Emesis

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Dear editor;

Wernicke encephalopathy (WE) is a neurological syndrome associated with thiamine deficiency, often related with alcoholism (1,2). If signs and symptoms are recognized, the condition can be treated in an early stage; if they are overlooked, it may develop into Korsakoff syndrome (KS) with a mortality rate of 20% (1). The entire range of clinical findings with disturbance of consciousness, ataxia, ophthalmoplegia, and nystagmus is only seen in a small fraction of 10-20%. Therefore, especially in cases with no history of alcoholism, diagnosis can be difficult (3,4). While cranial magnetic resonance imaging (MRI) results are helpful in the diagnosis, they are not always requested in clinical practice. Several publications of cases and studies in the last years have shown that this disease can be seen more frequently than previously thought in persons without alcohol addiction, e.g. after bariatric surgery, parenteral nutrition, hyperemesis gravidarum, anorexia nervosa, or hunger strike (1,5,6). Data from the literature show that in 20% of nonalcoholic cases the condition can be diagnosed but mostly escaped attention (4). In the literature, WE cases related with recurrent and long-term vomiting are only reported with pregnancy-related hyperemesis gravidarum. We present 2 cases of WE resulting from recurrent emesis in previously healthy persons with no nutritional problems.

Case 1: A 52-year-old woman, no known history of disease. After sleeve gastrectomy operation for obesity, the patient developed a complaint of unstoppable vomiting on day 2 post-op. After 1 month of parenteral nutrition, when the patient resumed enteral nutrition, vomiting decreased but did not

resolve completely. In month 3 post-op, the patient presented with complaints of disturbed consciousness, confused speech, and imbalance. In the neurologic examination, her consciousness was confused, spatial, temporal, and personal orientation were impaired. Her speech was dysarthric, the abduction of the eyeballs from the median line was deficient. She had bilateral dysmetria and was ataxic in each direction when standing, unable to be mobilized without support. Biochemical tests found no pathologies. In the cranial diffusion MRI, bilateral symmetric restricted diffusion was found in the crura mesencephali. The patient was admitted with a diagnosis of WE. Her clinical picture completely resolved within 1 week with vitamin B complex support in the form of thiamine 100mg/day.

Case 2: A 19-year-old male had begun vomiting 4 months previously without seeking medical attention, developing vertigo, double vision, and deficiencies in ocular motion 3 months ago. The patient was examined in an external neurology clinic. A gastroenterological consultation and examination results did not find any pathology to explain the cause of vomiting. The cranial MRI showed bilateral thalamic and periaqueductal T2 and FLAIR hyperintense lesions. The patient was diagnosed with WE. For 1 week, he received parenteral thiamine followed by oral maintenance treatment. Around 2 weeks after discharge, as vomiting continued and double vision and defective ocular motility, which had decreased after thiamine therapy, increased again, the patient presented to us. In the cranial MRI, it was found that the previously existing lesions partly persisted. Therefore, the patient was admitted to our clinic for parenteral thiamine replacement. In the neurological examination, no abnormality other than bilateral total external ophthalmoplegia was found. With high-dose parenteral thiamine treatment, the ophthalmoplegia substantially improved, and in the cranial MRI control it was seen that the lesions had regressed.

It is now known that WE is not exclusively related to alcoholism. Persons with eating disorders and hyperemesis gravidarum, individuals undergoing surgical operations for obesity, and patients receiving long-term parenteral nutrition or dialysis as well as carriers of the human immunodeficiency virus (HIV) can be at risk due to the development of thiamine deficiency (1,5). One of the situations for non-alcoholic development of WE is long-term recurrent vomiting, as was the case with our patients. In the diagnosis of WE, it is important not only to recognize clinical signs and symptoms, but also to identify the potential risk groups. Rather than the classical triad of ocular involvement (ophthalmoplegia, nystagmus), ataxia, and disturbance of consciousness, the patient may also present with a single sign. In case of delayed diagnosis, it is likely that all three symptoms occur (3,4). In the literature, there are also rare reports of cases with loss of vision, and in some cases this loss of vision is the initial symptom (7). In the first of our cases, the entire clinical triad was present, while in the second case only ophthalmoplegia was found.

The body of a healthy person contains only 30-50mg of thiamine. As the daily requirement of thiamin is 1-2mg, we can see that if for whatever reason enteral nutrition is not possible, any person's thiamine reservoir will be depleted within 3-4 weeks and signs of WE start to appear. After the onset of the first symptoms, 7-10 days later a rapid clinical deterioration may develop, followed by deep coma. The first of our patients began to show clinical signs around 8 days after parenteral nutrition support, while the second one presented symptoms 1 month after the onset of vomiting.

Typical MRI findings for WE show a symmetric bilateral involvement of the medial thalamus, mammillary bodies, mesencephalon, and tegmentum as well as the periaqueductal area. These lesions can bind gadolinium and show restricted or increased diffusion. Atypical findings may also be added. In many cases, MRI assessment can be normal (8,9). Of our patients, the first one only had a bilateral lesion in the crura mesencephali, the other in the thalamic and periaqueductal area.

Reportedly, WE responds well to treatment especially in the early stage (10). Hospitalized patients have to be treated urgently with IV thiamine 100mg; in patients with alcoholism, the dose needs to be

increased (11). At the end of 1 week, treatment is continued with an oral maintenance dose.

In conclusion, WE can develop in relation with a number of causes other than alcoholism, one of those being recurrent vomiting, as was the case in our patients. In particular with the increase of slimming surgery, it is important to make patients aware of the issue and to provide vitamin replacement (12,13). Keeping these causes in mind is important for an early detection of this presentation that is responsible for significant mortality and morbidity, which allows for an early start of therapy.

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