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CASE REPORT

About a case of Wernicke's encephalopathy after sleeve gastrectomy

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Introduction

We report a case of Wernicke's encephalopathy (WE) after sleeve gastrectomy (SG).

A 27-year-old woman underwent SG two months earlier. She had no treatment (no vitaminic supplementation), no medical history except ear surgery and no history of alcoholism. Before surgery, her body mass index was 42.1. Since her SG, she had lost 23 kg in two months, from 108 to 85 kg.

One month after surgery, she arrived at the emergency care unit with nausea and recurrent vomiting. Gastro-abdominal explorations (abdominal ultrasonography, abdominal CT-scan, gastroscopy) were normal. She was sent home with specific diet (fractioned meals) and vitaminic supplementation. Due to lack of compliance, vitaminic supplementation was not taken.

The situation worsened with inconspicuous appearance of confusion, swallowing difficulty, ataxia, dizziness and visual troubles (diplopia and blurred vision) but she did not contact her bariatric team.

Confusion was first attributed to psychiatric illness because of hard psychological context (parent's divorce, ex-boyfriend's illness), and she was hospitalized in a psychiatric unit.

During this hospitalisation, as she rapidly (three days) developed tetraparesis, facial diparesis, horizontal nystagmus and vigilance troubles, she was transferred and admitted to our neurological department. These troubles appear nearly two months after the SG.

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The lumbar puncture showed a normal glycorrhachia and no cellular abnormalities, but the proteinorachie was increased at 509 mg/l (150–450 mg/l). The electroneuromyography was normal in four limbs.

The cerebral MRI showed high-intensity Flair in the periaqueductal region and bilateral paraventricular regions of thalami (Figs. 1 and 2). All blood analysis was normal except thiamine level reduced to 15.9 ng/ml (20–100 ng/ml).

We began parenteral thiamine supplementation (750 mg IV per day during two weeks followed by 500 mg oral per day). MRI normalized after two weeks of treatment. One month after supplementation, the patient recovered a normal vigilance. Diplopia and visual abnormalities, facial diparesis and dysphagia disappeared. She underwent five months of rehabilitation. After a follow-up of fourteen months, she still had ataxic and cognitive deficit (episodic memory is severely affected).

Discussion

WE is an acute neurological syndrome resulting from thiamine deficiency characterized by (1) nystagmus and ophthalmoparesis, (2) cognitive impairment and (3) unsteadiness of stance and gait. However, this classic triad is seen in only 16 % of patients, which can explain that WE is under-recognised, especially in alcoholic patients [1].

Alcoholism is the common cause for this disease. However, WE has also been reported in patients with any condition of unbalanced nutrition that lasts for 2–3 weeks like gastrointestinal surgical procedures (including SG), staple diet of polished rice, recurrent vomiting as in hyperemesis gravidarum or chronic diarrhoea, cancer and chemotherapeutic treatment, systemic disease and

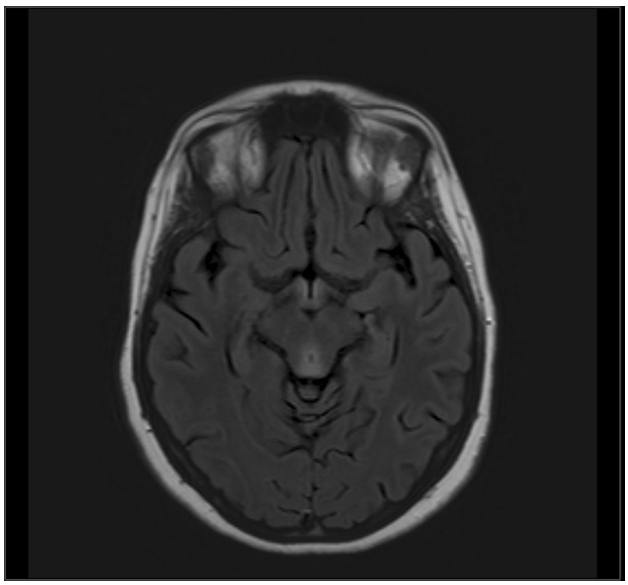

Fig. 1 Flair weighted imaging demonstrating high-intensity in periaqueductal region

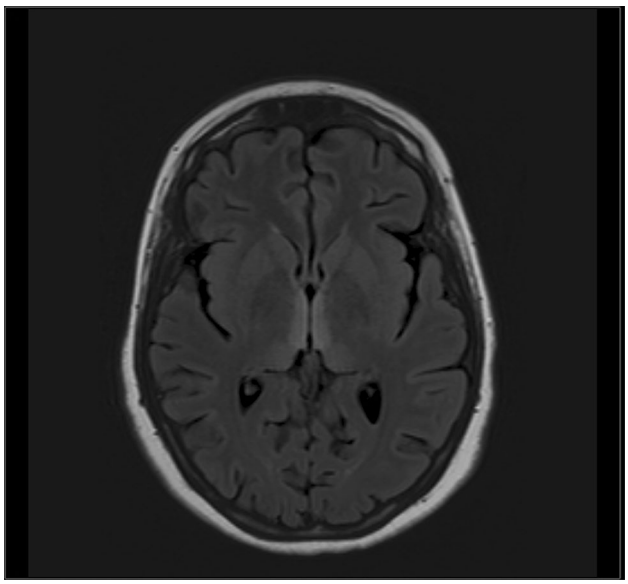

Fig. 2 Flair weighted imaging demonstrating high-intensity in bilateral paraventricular regions of thalamus

magnesium depletion [2]. Thiamine deficiency has been discovered in up to 49 % patients after gastric bypass and WE was well reported following vertical banded gastroplasty (VBG). VBG and SG are two procedures that can produce true vomiting rather than regurgitation and now that SG has become the most commonly used bariatric procedure, rate of WE after SG increased. Nonetheless Kröll performed on October 2015 a recent literature review on WE after SG, only 13 cases have been published [3].

Among paraclinical studies, cerebral MRI is the most valuable method to confirm the diagnosis of WE with a high specificity of 93 % but a poor sensitivity of 53 %. Classically, acute thiamine deficiency causes neuropathological lesions in tectal plate, thalamus, hypothalamic nuclei and periventricular nuclei which may result in WE's cardinal signs of ataxia, nystagmus, ophthalmoparesis and confusion. In cerebral MRIs of patients with WE and in our patient's MRI, acute bilateral edematous lesions are typically seen in mammillary bodies, periaqueductal and periventricular gray matter, collicular bodies and thalamus [4]. However, in case of WE after SG, only 60 % of patients have these typical findings [3].

In the later phase of thiamine deficiency, targeted structures become atrophic and assume a different neuroradiological signature from the acute stage (disappearance of edematous lesions and loss of volume in the mammillary bodies, thalamus, pons, cerebellar hemispheres and anterior superior vermis) [3].

Treatment is parenteral thiamine supplementation, but there is no available evidence from randomised controlled trials neither about dose, frequency or duration of supplementation nor about prevention of WE in alcoholic people or after bariatric surgery.

According to the US guidelines, patients with bariatric surgery should be provided with oral multivitamin supplement that contains thiamine.

In case of persistent vomiting, aggressive supplementation with thiamine is imperative and intravenous glucose should be administered carefully because it can worsen thiamine deficiency.

In patients with symptoms of WE, aggressive parenteral supplementation with thiamine (100 mg/d) should be administered for 7–14 days and subsequent oral administration (100 mg/d) should be continued until neurological symptoms resolve [5].

The estimated mortality is 17 % without treatment. Korsakoff syndrome is the chronic form of WE, characterized by irreversible cognitive impairment, typically striking loss of working memory with relatively little loss of reference memory. It usually follows or accompanies WE, like in our case.

We want to highlight the possibility of WE after SG. The diagnostic is still difficult because of a poor sensitivity of cerebral MRI and a rare classic clinical triad. It is important that patients and their primary care teams have to be informed of WE by the bariatric surgical team, because the presence of signs and symptoms of WE can be misdiagnosed and treated inappropriately. Any neurological symptoms following bariatric surgery must suggest WE and must be treated by correct thiamin supplementation. We also remind that delay of treatment can lead to irreversible neurological impairment or death.

Compliance with ethical standards

Conflict of interest On behalf of all authors, the corresponding author states that there is no conflict of interest.

Ethical approval This article does not contain any studies with human participants or animals performed by any of the authors.

Informed consent For this type of study formal consent is not required.

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