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LETTER TO THE EDITOR

# A Late Onset of Wernicke-Korsakoff Encephalopathy After Biliopancreatic Diversion: a Case Report

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## Introduction

Wernicke-Korsakoff encephalopathy (WKE) is a neurologic disease due to a severe thiamine deficiency. This vitamin, an essential cofactor for cellular metabolism, is not produced by the human organism, so its total supply comes from diet, being absorbed from the duodenum.

Wernicke described its acute onset in 1881; its classic form is characterized by the triad of ataxia, abnormal mental state, and ocular abnormalities (especially nystagmus).

Although it is most commonly associated with chronic alcohol misuse (90% of all cases in USA), other medical conditions resulting in inadequate thiamine intake, such as gastrointestinal disease (vomiting, diarrhea), hyperemesis gravidarum, hemodialysis, sepsis, GI cancer, Crohn's disease, psychiatric disorders, HIV infection, and malnutrition can determinate the onset of the disease [1, 2].

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Another well-established cause of WKE is bariatric surgery. However, in most bariatric surgery-related cases, there were no specific neurological symptoms and no definitive neuroimaging markers were established. Moreover, a recent review [3] upon the most common bariatric procedures showed that the large majority of cases develop in a range of onset between 4 and 12 weeks post-surgery (12 days up to 18 months). No significant difference in the time of onset in the different surgical procedures was observed. Roux-en-Y gastric bypass was the most related procedure (52%), followed by sleeve gastrectomy (21%); biliopancreatic diversion was associated with a small percentage of cases of WKE (3%). Biliopancreatic diversion is a single surgical procedure combining a sleeve gastrectomy and gut bypass, connecting the reduced stomach (about the 20% of the former organ) and the second tract of duodenum with the last part of the small intestine, with the aim to reduce calorie and nutrient absorption [4].

Precipitating factors were persistent vomiting, diarrhea, rapid weight loss, anorexia, minimal food intake or glucose-containing intravenous feeding, alcohol misuse, and noncompliance with vitamin supplements.

## Case Study

### History

Patient was a 62-year-old man with type 2 diabetes since he was 30 years old, on basal-bolus insulinic regimen. For morbid obesity (BMI 40, body weight 123 Kg), in 2012, he had bariatric surgery (BPD—Biliopancreatic diversion).

The intervention was without major complications, and the patient was discharged after a few observation days. After surgery (weight 78 Kg, 63.4% TWL, BMI 26), patient

stopped therapy for diabetes (last HbA1c 5.2%). He did not follow any particular diet and was in good health until the end of 2017.

In early 2018, his symptoms started with a walking disorder, mainly secondary to a balance disorder with a subjective instability feeling, especially in the changes of position, associated with some difficulties to deambulate on disjointed terrain (two accidental falling episodes in the last 12 months). Such condition developed in a slowly progressive trend over time and was accentuated in the last year, when a slowdown in the speech appeared.

In the last year, he started therapy with probiotics for recurrent diarrhea.

For all these symptoms, he underwent a series of specialist visits and instrumental examinations, including an EMG/ENG (initial axonal neuropathy in the lower limbs) and a brain MRI (showing an "empty sella").

Then, he came to our attention for deepening of the diagnostic framework.

Neurological examination: patient alert and cooperative, slow-down, dysprosodic speech, normal ocular movements, some nystagmus shocks in the look of laterality and upward and to the right; osteotendinous reflexes diffusely well evocable, symmetrical, bilateral hallux, a tendency to extend the big toe to the Chaddok; no muscular hypotrophy, no deficiency of global or segmentary force in the limbs, ataxic ambulation, and heel; in Romberg, with closed eyes, tendency to right lateropulsion, performs some steps in tandem, albeit with difficulty; He could not stand on the tips and with difficulty on their heels. No tremor at rest or postural, no hypertone.

A series of tests and blood exams were performed. The neurophysiological investigations (EMG/ENG and SSEP/MEP) documented an involvement of central motor and somatosensory pathways, in the absence of axonal sensory neuropathy, with evidence of initial myelinopathic suffering of the motor fibers pertaining to the lower limbs.

A new brain MRI study showed mild T2-signal hyperintensity of the PAG (Peri-Aqueductal Gray) (Fig. 1).

In the light of these findings and in view of the clinical symptomatology (ataxia, nystagmus, and paresthesia), given the history of BPD and the test results, in the suspicion of a carential genesis of the disorder linked mainly to a deficiency of thiamine absorption, treatment with a parenteral regimen of B1 was undertaken.

Objectively, the patient reports, since he started treatment, an improvement of the levels of attention and a less uncertainty in walking.

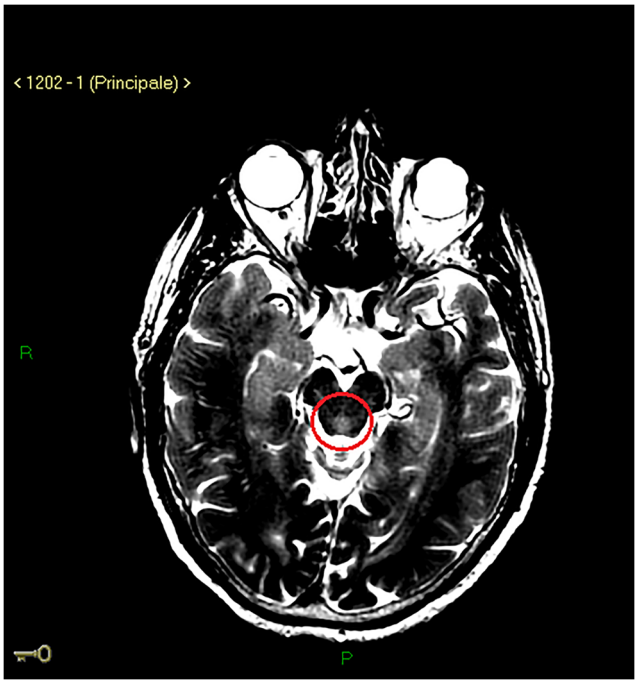


**Fig. 1** Cerebral MRI scan. T2-FLAIR sequence of cerebral MRI presenting mild hyperintensity in peri-acqueductal gray (PAG), without DWI and breaks of hematoencephalic barrier. Not altered signals in thalamus and mammillary body, bilaterally

## Conclusions

This case report shows a late onset (6 years) of WKE, a rare condition, which typically occurs after 4–12 weeks from surgery (up to 18 months). In particular, symptoms onset started after persistent diarrhea, already known as a precipitant factor for thiamine deficiency. In literature, only one case of a late onset WKE was reported after vertical banded gastroplasty [5], due to vomiting related to a stricture at the level of the gastric band. No late onset WKE reports after BPD were present.

Therefore, independent from timing after surgery, in presence of a known trigger for WKE and suggestive clinical/radiological features, suspicion of WKE needs to be considered and thiamine supplementation should be started.

### Compliance with Ethical Standards

**Conflict of Interest** The authors declare that they have no conflict of interest.

**Ethical Approval** For this type of study, formal consent is not required.

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