



# Mild symptomatic Wernicke's Encephalopathy: A case report

Maria Sofia Cotelli\*, Patrizia Civelli, Marinella Turla

Neurological Unit, ASST Valcamonica, Esine, Brescia, Italy

## ABSTRACT

Wernicke's encephalopathy (WE) is an acute, neuropsychiatric syndrome which results from a deficiency in Vitamin B1 (thiamine), which in its biologically active form, thiamine pyrophosphate, is an essential coenzyme in several biochemical pathways in the brain, often due to alcohol abuse (alcoholic WE) (1). Non-alcoholic WE variant manifests in many different clinical settings, such as gastrointestinal tumors, hyperemesis gravidarum, chemotherapy, acquired immunodeficiency syndrome, prolonged therapeutic fasting, prolonged parenteral nutrition and bariatric surgery, and anorexia nervosa and can even be secondary to socioeconomic factors. The classic triad of encephalopathy, oculomotor dysfunction, and gait ataxia is only seen in approximately one-third of patients and is more common in alcoholics; only some of these symptoms are usually present. Here, we describe a case of an occasional neuroradiological finding of WE not related to symptoms or signs.

**Key words:** Wernicke; encephalopathy; alcohol; thiamine

## BACKGROUND

Wernicke's encephalopathy (WE) is an acute, neuropsychiatric syndrome which results from a deficiency in Vitamin B1 (thiamine), which in its biologically active form, thiamine pyrophosphate, is an essential coenzyme in several biochemical pathways in the brain, often due to alcohol abuse (alcoholic WE) (1). Non-alcoholic WE variant manifests in many different clinical settings, such as gastrointestinal tumors, hyperemesis gravidarum, chemotherapy, acquired immunodeficiency syndrome, prolonged therapeutic fasting, prolonged parenteral

nutrition and bariatric surgery, and anorexia nervosa and can even be secondary to socioeconomic factors (1). The incidence of WE is believed to be higher in developing countries due to vitamin deficiencies and malnutrition. The female-to-male ratio for WE is 1:1.7, and there are no studies that show a particular race predisposed to WE (2).

## CASE REPORT

We report the case of a 57-year-old woman with typical radiological features for WE, despite her negative neurological examination and her unremarkable medical history. In particular, the patient denied alcohol abuse (she used to drink half a glass of wine only with meals). She reported a various diet, with a good appetite and normal digestion. She also denied weight loss and typical symptoms of malabsorption or gastrointestinal symptoms such

\*Corresponding author: Maria Sofia Cotelli,  
Neurological Unit, ASST Valcamonica, Esine, Brescia, Italy.  
E-mail: [cotellim@gmail.com](mailto:cotellim@gmail.com)

Submitted: 1 December 2018/Accepted: 15 December 2018

DOI: <https://doi.org/10.17532/jhsci.2018.269>

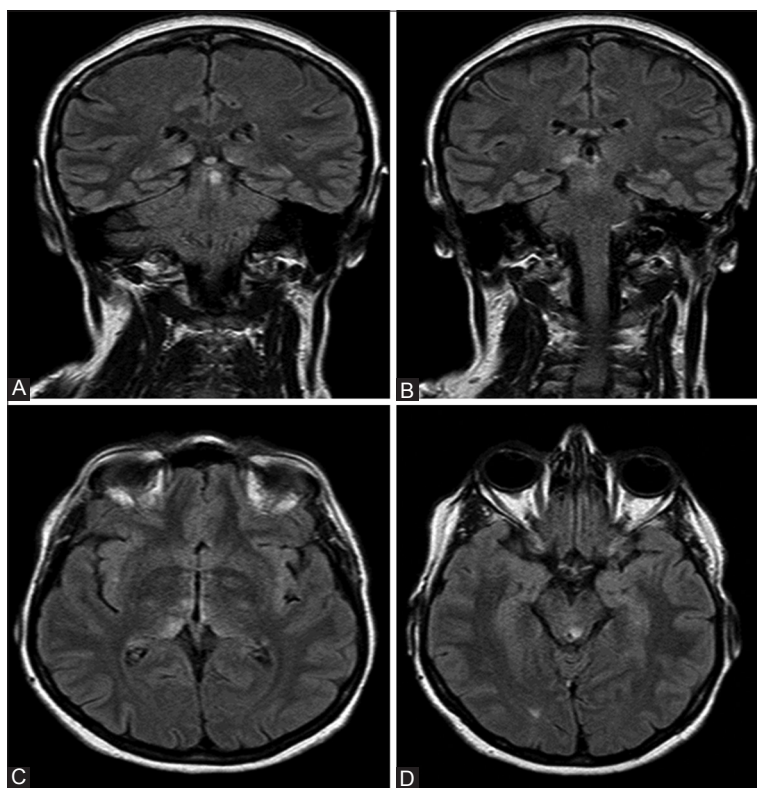


as chronic diarrhea, nausea, vomit, and abdominal pain. Her body mass index (2) was normal. She was never diagnosed a cancer or had a gastrointestinal surgical procedure. Her medical history was also unremarkable except for high blood pressure (but in good control with amlodipine).

She was admitted to the Emergency Medicine Department due to persistent dizziness and objective vertigo, with nausea, which started from about 7 days, and 4–5 episodes of vomit from 1 day, and was subjected to a brain computerized tomography, which was normal. Symptoms quickly improved after administration of intravenous levosulpiride in the Emergency Department. Neurological examination was normal except for bilateral horizontal nystagmus with torsional components, for which she was hospitalized. Brain magnetic resonance imaging (MRI) showed multiple hyperintense lesions

localized in the periaqueductal gray matter (mainly on the left side) and the ventral portion of both the posterior and medial thalami. No restriction of diffusion or pathological enhancement after contrast gadolinium was reported. These neuroradiological features were reported as suspicious for a radiological pattern of WE (Figure 1).

Blood examinations (blood count, glucose, electrolytes, serum creatinine, thyroid function, transaminases, tumor markers, amylase, insulin, bilirubin and Vitamin B12, folate and Vitamin B1, immunological test such as complement activity, antinuclear antibodies, extractable nuclear antigen, anticytoplasmic antibody, lupus anticoagulant, human immunodeficiency virus, hepatitis B and C virus, Treponema Pallidum and Borrelia Burgdorferi serology, serum Immunoglobulin assay were all normal. Blood alcohol and drug test were



**FIGURE 1.** Brain magnetic resonance imaging (MRI) showed multiple hyperintense lesions localized in the periaqueductal gray matter (mainly on the left side) and the ventral portion of both the posterior and medial thalami. No restriction of diffusion or pathological enhancement after contrast gadolinium was reported. These neuroradiological features were reported as suspicious for a radiological pattern of WE. (A) Coronal T2/fluid attenuated inversion recovery image. (B) Coronal T2/fluid attenuated inversion recovery image. (C) Axial T2/fluid attenuated inversion recovery image. (D) Axial T2/fluid attenuated inversion recovery image.

also normal. Doppler carotid US, chest X-ray, and ultrasound of the abdomen resulted in all normal. Blood pressure resulted in normal (125/80 mmHg). Neuropsychological assessment, performed in the 2<sup>nd</sup> day of in-hospitalization, resulted in all normal.

Intravenous thiamine supplementation 600 mg/day was administered for a month, while otorhinolaryngologists suggested levosulpiride 15 drops 3 times in a day for 7 days. Neurological examination resulted in normal after 3 days of in-hospitalization with complete absence of nystagmus.

## DISCUSSION

WE is a neurologic condition as a consequence of thiamine (Vitamin B1) deficiency. It was first described by Carl Wernicke in 1881; he noted the classic triad of altered mentation, ataxia, and ocular signs in association with brain lesions on autopsy (3). The classic triad of encephalopathy, oculomotor dysfunction, and gait ataxia is only seen in approximately one-third of patients and is more common in alcoholics (AL) (4). The onset of the disease can be characterized by several other findings such as cardiac failure with hypotension and tachycardia, gastrointestinal symptoms such as abdominal pain and nausea, hypothermia due to the involvement of the posterior hypothalamic regions, deafness due to thalamic involvement, and epileptic seizures in case of glutamatergic hyperactivity (5,6).

The prevalence of WE in the general population has been estimated from autopsy studies and varies from 0.4 to 2.8 (7), and it seems to be much higher in AL than in non-alcoholics. WE is often misdiagnosed, leading to persistent dysfunctions and, in some cases, to death. Nowadays, MRI of the brain, showing T2 and fluid attenuated inversion recovery hyperintensities in typical (thalami, mammillary bodies, tectal plate, and periaqueductal area) and atypical areas (cerebellum, cranial nerve nuclei, and cerebral cortex), is surely the most important and effective tool in the diagnostic assessment of WE (8). To date, high specificity (93%) but low sensitivity (53%) is attributed to MRI in WE diagnosis (9).

WE is a medical emergency and any therapeutic delay may result in permanent neurological damage or death. The treatment of suspected or manifest WE is based on the administration of thiamine. To

date, there is still no consensus on its optimal dose, modality of administration, and treatment time. The traditional recommendation is a parenteral dosage of at least 100 mg of thiamine per day (10). Recently, some authors have recommended that patients should be given 200 mg of thiamine 3 times a day (4). It should be given before or concomitantly with any carbohydrates because glucose can precipitate the disorder. Duration of treatment also remains an enigma; it should be continued until there is no further improvement in signs and symptoms (1).

We report the case of a “pure radiological diagnosis” of non-alcoholic and actually idiopathic WE as we have not been able to identify predisposing factor for the manifestation of the syndrome. We decided to follow our patient with strict neurological and neuroradiological examinations due to the improvement of vertigo and nystagmus with levosulpiride. MRI follow-up at 3, 6, and 9 months has shown a stable neuroradiological pattern. She also repeated the previous panel of blood examinations every 6 months, which resulted in normal. After 5 years of follow-up, she is still asymptomatic.

We think that this neuroradiological finding was not related with the referred symptoms at the admission, probably it was only an occasional finding (the typical triad symptoms characterized by ophthalmoplegia, ataxia, and confusion were all absent and vertigo and nystagmus disappeared after symptomatic treatment with levosulpiride, so she was discharged with diagnosis of acute labyrinthitis).

Moreover, it should be difficult, in our opinion, to find this neuroradiological pattern after few episodes of vomit, even if both vertigo and vomit can be considered possible precursors of WE (11).

Nystagmus improved during the 3<sup>rd</sup> day of in-hospitalization. In WE, gaze-evoked nystagmus, spontaneous upbeat nystagmus, and horizontal or vertical ophthalmoplegia have been described (12). These signs occur probably due to selective vulnerability to thiamine deficiency in high-energy-dependent glia and neurons in the gray matter that surrounds the third ventricle, the Sylvian aqueduct, and the floor of the fourth (13). Our Colleagues Otorhinolaryngologists considered nystagmus seen in our patient (horizontal with torsional component) as probably linked to acute labyrinthitis and suggested levosulpiride for

nausea and dizziness. However, nowadays, there are not still diagnostic criteria for non- alcoholic WE: European Federation of Neurological Societies guidelines (4) report only a list of eight signs and symptoms divided into eight clinical domains with sensitivity of each domain (recalculated from the paper) ranging from 20% (seizures) to 75% and (cerebellar signs). Sensitivity of the classic triad was about 23% but rose to 85% if the patients had at least two of the four following features: Dietary deficiencies, eye signs, cerebellar signs, and either mild memory impairment or an altered mental state (4). Our patient's symptoms and sign are not specific nor sensitive, and without brain MRI, a definite diagnosis would never have been performed.

## CONCLUSIONS

Wernicke's encephalopathy (WE) is an acute, neuropsychiatric syndrome typically associated with a peculiar neuroradiological pattern. In presence of brain imaging suggestive for WE, thiamine deficiency should be first excluded, but there are lots of other potentially pathogenetic factors that should be considered as possible differential diagnosis. It is also possible to find a typical neuroradiological pattern, such as in our case, apparently not related to typical presentation. In our opinion therapy with thiamine should be anyway administered following guidelines and brain magnetic resonance follow up should be performed.

## REFERENCES

1. Sechi G, Serra A. Wernicke's encephalopathy: New clinical settings and recent advances in diagnosis and management. *Lancet Neurol* 2007;6(5):442-55.  
[https://doi.org/10.1016/S1474-4422\(07\)70104-7](https://doi.org/10.1016/S1474-4422(07)70104-7).
2. Arts NJ, Walvoort SJ, Kessels RP. Korsakoff's syndrome: A critical review. *Neuropsychiatr Dis Treat* 2017;13:2875-90.  
<https://doi.org/10.2147/NDT.S130078>.
3. Thomson AD, Cook CC, Guerrini I, Sheedy D, Harper C, Marshall EJ, et al. Wernicke's encephalopathy revisited. Translation of the case history section of the original manuscript by Carl Wernicke 'Lehrbuch der Gehirnkrankheiten für Aerzte und Studierende' (1881) with a commentary. *Alcohol Alcohol* 2008;43(2):174-9.  
<https://doi.org/10.1093/alcalc/agn144>.
4. Galvin R, Bräthen G, Ivashynka A, Hillbom M, Tanasescu R, Leone MA, et al. EFNS guidelines for diagnosis, therapy and prevention of wernicke encephalopathy. *Eur J Neurol* 2010;17(12):1408-18.  
<https://doi.org/10.1111/j.1468-1331.2010.03153.x>.
5. Reuler JB, Girard DE, Cooney TG. Current concepts. Wernicke's encephalopathy. *N Engl J Med* 1985;312(16):1035-9.  
<https://doi.org/10.1056/NEJM198504183121606>.
6. Doss A, Mahad D, Romanowski CA. Wernicke encephalopathy: Unusual findings in nonalcoholic patients. *J Comput Assist Tomogr* 2003;27(2):235-40.  
<https://doi.org/10.1097/00004728-200303000-00022>.
7. Harper C, Fomes P, Duyckaerts C, Lecomte D, Hauw JJ. An international perspective on the prevalence of the wernicke-korsakoff syndrome. *Metab Brain Dis* 1995;10(1):17-24.  
<https://doi.org/10.1007/BF01991779>.
8. Manzo G, De Gennaro A, Cozzolino A, Serino A, Fenza G, Manto A, et al. MR imaging findings in alcoholic and nonalcoholic acute Wernicke's encephalopathy: A review. *Biomed Res Int* 2014;2014:503596.  
<https://doi.org/10.1155/2014/503596>.
9. Antunez E, Estruch R, Cardenal C, Nicolas JM, Fernandez-Sola J, Urbano-Marquez A, et al. Usefulness of CT and MR imaging in the diagnosis of acute Wernicke's encephalopathy. *AJR Am J Roentgenol* 1998;171:1131-7.  
<https://doi.org/10.2214/ajr.171.4.9763009>.
10. Donnino MW, Vega J, Miller J, Walsh M. Myths and misconceptions of Wernicke's encephalopathy: What every emergency physician should know. *Ann Emerg Med* 2007;50(6):715-21.  
<https://doi.org/10.1016/j.annemergmed.2007.02.007>.
11. Oudman E, Wijnia JW, van Dam M, Biter LU, Postma A. Preventing wernicke encephalopathy after bariatric surgery. *Obes Surg* 2018;28(7):2060-8.  
<https://doi.org/10.1007/s11695-018-3262-4>.
12. Anand P, Gold DR. Nystagmus from Wernicke's encephalopathy. *N Engl J Med* 2017;377(4):e5.  
<https://doi.org/10.1056/NEJMicm1615499>.
13. Kattah JC. The spectrum of vestibular and ocular motor abnormalities in thiamine deficiency. *Curr Neurol Neurosci Rep* 2017;17:40.  
<https://doi.org/10.1007/s11910-017-0747-9>.