

Case Report

Rare Cause of Central Vertigo of Metabolic Origin Due to Vitamin B1 Deficiency: Case Report

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Abbreviations:

EFI: eye fixation index; VA: visual acuity; BPPV: benign positional paroxysmic vertigo; VVOR: visual vestibular optokinetic reflex; VOR: vestibulo ocular reflex; COR: cervico ocular reflex; MRI: magnetic resonance imaging; WE: Wernicke encephalopathy.

Introduction

Vitamin B1 is a cofactor for three major enzymes (the pyruvate dehydrogenase complex, α -ketoglutarate dehydrogenase, and transketolase) that are involved in cellular energy metabolism[1].

The deficiency of thiamin causes a Wernicke-type encephalopathy, but in rare cases it can manifest itself by rotary vertigo attacks in an acute context associated with an instability that may be confusing to the clinician.

Patient and observation

This patient was 27 years old, originally from Morocco, followed for severe celiac disease with asthenia, jaundice and general deterioration. and without any notion of particular otological history. The patient presented to the ENT consultation for acute vertigo crisis evolving for 4 days, with instability, tinnitus and uncontrollable vomiting.

Clinical examination returned to normal bilateral otoscopy. Her visual acuity (VA) measured 10/10 in both eyes. Pupil reflexes were brisk and consensual with no relative afferent pupil defect. Slit lamp exam and ophthalmoscopy were normal, including both optic

Abstract

This presentation shares an extremely rare case, patient with central acute dizziness of metabolic origin for lack of vitamin B1. A 27-year-old patient hospitalized for severe celiac disease with asthenia, jaundice and deterioration of general condition, presenting a rotary vertigo, tinnitus for 4 days and uncontrollable vomiting. The clinical examination revealed an up-beating nystagmus on primary gaze that increased slightly on lateral gaze, increased markedly on upgaze and decreased significantly in amplitude and frequency on downgaze. At the videonystagmography, oculomotricity test was pathological, the kinetic test was normal except the Eye Fixation Index (EFI) at 0,44. The caloric test was normal. The brain MRI was normal, the blood test of vitamin B1 was decreased. The patient benefited from an intravenous B vitamin therapy with a clear improvement of her symptomatology after 3 weeks of treatment. Vertigo of central origin is a rare cause and often unknown, the management is often difficult.

Keywords: Videonystagmography; central vertigo; Thiamine deficiency

nerves appearance.

External examination revealed small amplitude and moderate frequency up-beating nystagmus on primary gaze that increased slightly on lateral gaze, increased markedly on upgaze and decreased significantly in amplitude and frequency on downgaze.

Absence of nystagmus in search of benign positional paroxysmic vertigo (BPPV).

At the VNG (videonystagmography)

Oculomotricity test: Pathological, with a staircase curve in the eye tracking test, and a nystagmus change of direction to the Gaze nystagmus. (Figure 1)

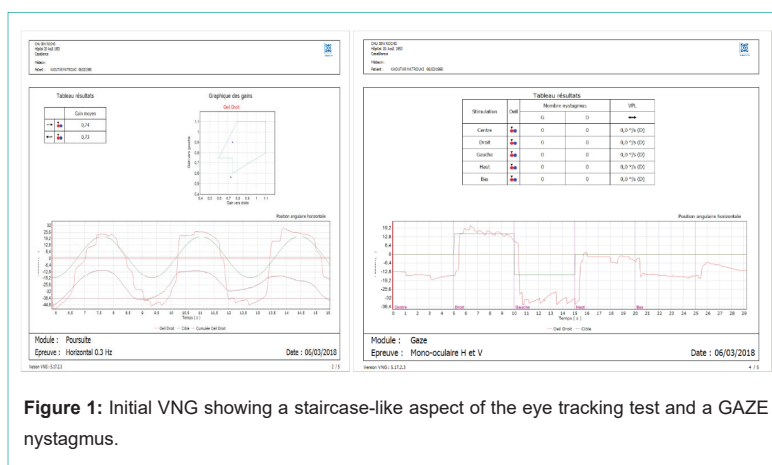


Figure 1: Initial VNG showing a staircase-like aspect of the eye tracking test and a GAZE nystagmus.

Kinetic test: A normal visual vestibular optokinetic reflex (VVOR), a normal vestibulo ocular reflex (VOR), a normal cervico-ocular reflex (COR) with an Eye Fixation Index (EFI) at 0,44.

Normal caloric test:

The clinical examination and the results of the VNG were in favor of a central vertigo indicating the realization of a brain magnetic resonance imaging (MRI) which was normal.

The patient's file was staffed in medical neurology who asked for a blood test for vitamin B1, and asked to start a treatment before waiting for the result of the B1 vitamin test which has only a reliability and limited interest.

The treatment was B1 intravenous vitamin therapy at 500 mg thiamine hydrochloride diluted in 100 mL of saline, passage over 30 min, 2 times / day, during 5 days. Then 250 mg / d until clinical improvement, then relay orally, associated with magnesium because it is a cofactor of thiamine and deficiency is a cause of inefficiency.

The blood test of vitamin B1 was decreased.

The patient described an improvement after one week of treatment, and a complete normalization of her symptomatology after three weeks, with a normal VNG (Figure 2) of control including oculomotor tests and the eye fixation index (EFI).

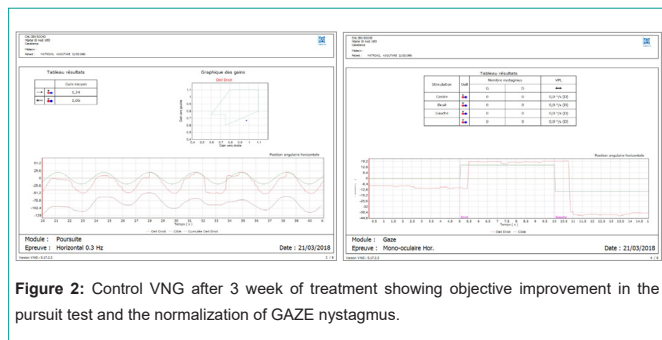


Figure 2: Control VNG after 3 week of treatment showing objective improvement in the pursuit test and the normalization of GAZE nystagmus.

Discussion

Vitamin B1 (thiamine) deficiency has many reasons, such as chronic alcoholism, recurrent vomiting, parenteral nutrition, gastrointestinal surgery, cancer, liver diseases and so on[2].

Sometimes this deficiency can lead to Wernicke encephalopathy (WE) which is an acute neurological emergency resulting from a deficiency of thiamine (vitamin B1) [3], and consists of a triad of ataxia, mental confusion and ocular motor dysfunction [4]. It is worth noting that the three symptoms are present, concurrently, in only 16% of patients which could make the diagnosis undisclosed.

In our case, uncontrollable vomiting caused vitamin depletion of the patient. Since body storage of thiamine lies between 30 and 50mg, reservations may become extinct as early as four to six weeks[5]. Thiamine, known as vitamin B1, is an essential molecule involved in several metabolic steps of chain energy cell production[6]. Thiamine deficiency demonstrated to affect brain structures with high thiamine turnover rate as cerebellum, medulla, pons, hypothalamus, midbrain and thalamic area[7].

These regions are involved in the mechanism of controlling eye movements and steady fixation which are committed during Thiamine deficiency[8]. The main ophthalmological signs include abduction paresis, horizontal and vertical gaze palsy, internuclear ophthalmoplegia, horizontal and vertical nystagmus and optic neuropathy[4]. While a presumptive diagnosis can be made by measuring the erythrocyte transketolase activity, it is limited by its

lack of specificity and cost. Prompt administration of thiamine leads to an improvement in the ocular signs within hours to days, vestibular function and gait ataxia during the second week, and confusion subsides over days to weeks[9]. In case of neurological signs suggestive, the treatment must be started without delay, parenterally. There is no validated supplementation scheme, however all reported cases and meta-analyses show that prescribed doses are usually important : 500 mg thiamine hydrochloride in 100 mL of physiological serum for 30 minutes, three times a day for three days, then 250 mg / day, until symptoms improve, then oral relay[10].

Conclusion

Vertigo of central origin is a rare cause and often unknown, the management is difficult. Vitamin B1 deficiency can manifest itself as dizziness, ataxia or confusion, hence the importance of thiamine supplementation in patients with a particular area of severe undernutrition. The absence of toxicity, even at high doses, and the imperfection of available biomarkers encourage prevention and treatment empirically.

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