Un caso de síndrome de Webino

A case of Webino Syndrome

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ABSTRACT

Wall-eyed bilateral internuclear ophthalmoplegia is a rare neuroophthalmological condition characterized by dissociated abducting nystagmus, a large angle exotropia in primary gaze and supranuclear vertical gaze palsy.
The authors present the case of an 83-year-old man admitted to the internal medicine ward with the diagnosis of cholangitis that suddenly starts with complaints of diplopia. Based on the clinical findings and results of the diagnostic workup, was established the diagnosis of Wall-eyed bilateral internuclear ophthalmoplegia syndrome.

Keywords: Exotropia; Medial longitudinal fasciculus; Wall-eyed bilateral internuclear ophthalmoplegia syndrome; Stroke.

RESUMEN

La oftalmoplejía internuclear bilateral es una rara condición neurooftalmológica caracterizada por nistagmo en abducción disociado, una exotropía de gran ángulo en la mirada primaria y parálisis supranuclear de la mirada vertical.
Los autores presentan el caso de un hombre de 83 años que ingresa en el servicio de medicina interna con el diagnóstico de colangitis que se inicia repentinamente con quejas de diplopía. Con base en los hallazgos clínicos y los resultados del estudio diagnóstico, se estableció el diagnóstico de síndrome de oftalmoplejía internuclear bilateral (síndrome WEBINO).

Palabras clave: Exotropía; Fascículo longitudinal medial; infarto cerebral; síndrome de oftalmoplejía internuclear bilateral de Wall-Eyed.

INTRODUCTION

Wall-eyed bilateral internuclear ophthalmoplegia (WEBINO) is a relatively rare neuroophthalmological condition characterized by dissociated abducting nystagmus, a large angle exotropia in primary gaze and supranuclear vertical gaze palsy.[1,2,3]

WEBINO is a rare manifestation due to pontine or midbrain lesions leading to a particular form of bilateral internuclear ophthalmoplegia.[4] Anatomically, results from the disruption of bilateral medial longitudinal fascicle, which is supplied by the anteromedial perforators of the posterior cerebral artery.[1,4,5] It carries fibers from the abducens nucleus in the pons to the contralateral medial rectus subnucleus of the oculomotor nerve, generating adduction when the other eye abducts.[6]

CASE DESCRIPTION

The authors present the case of an 83-year-old man with past medical history of arterial hypertension, dementia and had been cholecystecomized. The patient was admitted to the hospital due to abdominal pain, fever and jaundice with 3 days of evolution. On the initial physical exam, the patient presented jaundice, his vitals were normal, and he had a soft, depressing abdomen, without pain on palpation. Other systemic examinations were unremarkable.

Laboratory investigations revealed raised inflammatory markers (C-reactive protein 109mg/L), conjugated hyperbilirubinemia (total bilirubin of 64 µmol/L), and increased liver enzymes (Alanine transaminase 93 U/L; Aspartate transaminase 214 U/L; Gamma-glutamyl transpeptidase 1212 U/L; Alkaline phosphatase 589 U/L;). Ultrasound and abdominal CT showed no changes. Subsequently MRI was performed and revealed the presence of choledocholithiasis, leading to mild biliary ectasia, which showed diffuse parietal thickening. The diagnosis of cholangitis was established, and empirical antibiotic therapy was started.

On the third day of hospitalization, the patient suddenly starts with complaints of diplopia.

Initial neurological examination revealed that in the primary gaze position, the patient showed alternating exotropia (Figure 1b). On right lateral gaze, the right eye abducted fully, but the left eye failed to adduct (Figure 1a). When gazing to the left side, the left eye completely abducted and there was an adduction deficit of the right eye (Figure 1c). Convergence was impaired. There were no changes in normal acuity and visual fields. No eyelid ptosis or pupillary changes. In the remaining neurological examination, the patient did not had ataxia, decreased muscle strength or sensory changes.

Urgent cerebral CT and CT angiography were performed, which not identified ischemic lesions or vessel occlusion.

Magnetic resonance showed multi-infarction anterior lenticular-capsular lacunar, more extensive on the left with extension to the radiate corona and the frontal and parietal regions of radiate coronas and semioval centers, and bilateral pontine infarction (more extended to the left) (Figure 2, white arrow).

A transthoracic echocardiogram was performed and showed no structural or functional cardiac alterations.

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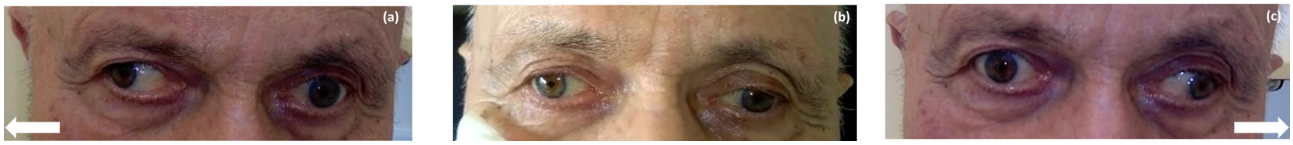
Figure 1



Examinations did not reveal any embolic sources, so we hypothesized that he was likely to have a brainstem stroke due to occlusion of a small penetrating artery. Based on this clinical findings and results of the diagnostic workup, was established the final diagnosis of WEBINO syndrome due to pontine infarction. Patient was discharged from the hospital and on a clinical follow-up showed a slow but progressive recovery of normal oculomotor function.

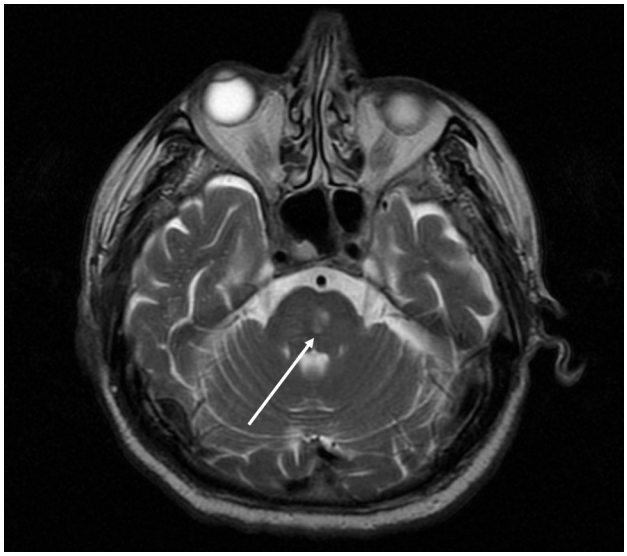
DISCUSSION

A lesion from any source that damages the medial longitudinal fasciculus bilaterally can result in WEBINO.[6] It could be caused by multiple sclerosis, infectious, inflammatory, toxic, nutritional, traumatic, and metabolic.[2,3,4] WEBINO seems to be most frequently associated with multiple sclerosis, while unilateral INO is most frequently associated with brainstem ischemia.[6] Because the median dorsal pons is supplied by multiple small arteries such as the paramedian, short circumferential, and long circumferential arteries, most of the infarcts in this area due to occlusion of one small artery cause unilateral, not bilateral, medial longitudinal fasciculus syndrome.[2]

WEBINO has several characteristic clinical signs, namely, large exotropia in primary gaze, impairment of adduction, dissociated nystagmus in the abducting eye, vertical gaze-evoked nystagmus, and impaired vertical vestibulo-ocular reflex.[3] Most reported stroke patients with WEBINO exhibited other manifestations such as vertical gauze palsy, consciousness disturbance, and ataxia.[2]

The prognosis of WEBINO and other types of internuclear ophthalmoplegia has not been well-documented, perhaps due to the lack of a consistent pool of patients available for long-term follow-up.[3] Besides treatment of the underlying systemic condition, management focuses on improving diplopia, and either surgery or botulinum toxin injections.[3] Alternatively, the use of prisms to correct exotropia and occlusion therapy may be useful.[3] Clinicians should be aware of such a condition in patients with brainstem infarction.

Figure 2



CONFLICT OF INTEREST
The authors declare that they have no conflict of interests.

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This research had no funding sources.

ETHICAL ASPECTS
All participants submitted a consent form to be included in this study.

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