Complex ophthalmoplaegia denoting Wernicke encephalopathy in a non-alcoholic individual

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DESCRIPTION

A 62-year-old man presented with subacute horizontal binocular diplopia and slight gait imbalance, preceded by frequent episodes of vomiting. The patient had undergone two gastric surgeries for gastric ulcer in the past (in 1988 and 2002). Bilateral internuclear ophthalmoplaegia, conjugate vertical gaze palsy and mild tandem gait abnormalities were observed (video 1, part 1; figure 1). Upper gastrointestinal endoscopy and gastric emptying scintigraphy

Video 1 Part 1 demonstrates bilateral internuclear ophthalmoplaegia and conjugate vertical gaze palsy. Part 2 was obtained after administration of intravenous thiamine and shows a full recovery of conjugate eye movements.

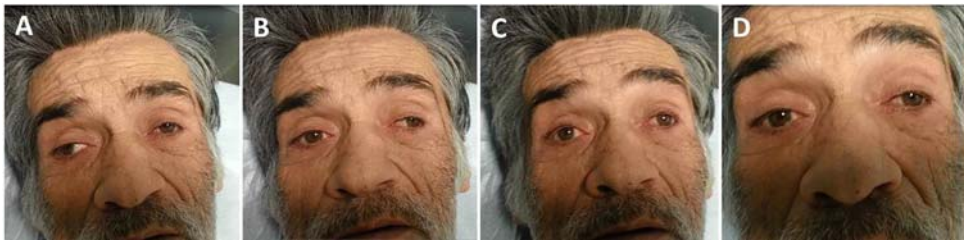

Figure 1 On admission, position of the patient's eyes when asked to look (A) right, (B) left, (C) up and (D) down.

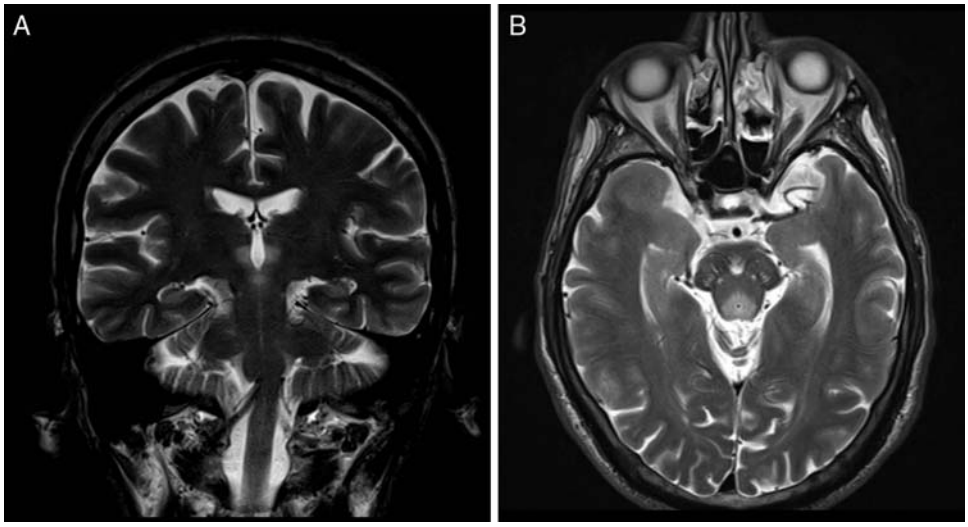

Figure 2 Coronal T2-weighted turbo spin-echo (T2TSE) (A) and axial T2TSE (B) MRI of the brain reveal T2 hyperintense lesions extending symmetrically from the floor of the fourth ventricle cranially along mesencephalic tegmentum and quadrigeminal plate up to the medial thalami, without significant expansion, presenting slight gadolinium enhancement.

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Learning points

- This case demonstrates the clinical heterogeneity of Wernicke encephalopathy, with the classical triad (encephalopathy, ataxia and oculomotor abnormalities—horizontal nystagmus being the most frequent) present in only up to a third of cases.[3]
- Albeit more frequent in alcoholic individuals, it can also occur in patients having a deficit in thiamine absorption for whatever reason, including emesis.
- Brain MRI is the single most important diagnostic tool and it usually depicts hyperintense signal on T2-weighted sequences, with variable enhancement, typically in periventricular and periaqueductal midline locations. Other typical findings include symmetric involvement of the medial thalami, mammillary bodies and tectal plate. Atypical findings are thought to be more frequent in non-alcoholic cases. Also, signal intensity alterations, contrast enhancement and atrophy of the mammillary bodies, as well as atrophy of the cerebellar vermis, are more common in alcoholic than non-alcoholic patients.[3] Keeping these differences in mind is fundamental for an accurate diagnosis of the more uncommon non-alcoholic Wernicke encephalopathy.

suggested a delay in gastric emptying. Brain MRI demonstrated hyperintense symmetric areas in the thalami, midbrain tegmentum, quadrigeminal plate and floor of the fourth ventricle, suggesting Wernicke encephalopathy (figure 2). Intravenous thiamine (200 mg three times a day, as recommended) was administrated for 15 days, followed by oral thiamine.[1] Serum thiamine concentration was impossible to determine (since this measurement is not performed in our centre), but sustained resolution of the patient's neurological signs and symptoms within 4 days, in the absence of additional therapeutic measures besides thiamine supplementation, confirmed the diagnosis of Wernicke encephalopathy (video 1, part 2).[2]

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Competing interests None.

Patient consent Obtained.

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