

# Optic tract and internal capsule lesion in a patient with Wernicke-Korsakoff syndrome

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A 72-year-old man, presented with a one-week history of confusion and an anterograde amnesic disorder accompanied by confabulation, with lack of insight to his symptoms.

Medical history included alcohol abuse and admitted twenty-years of alcohol ingestion (approximately 186 gr/day).

Neurologic examination was notable for slightly decreased consciousness, disorientation to time, severe anterograde amnesia and unsteadiness of stance and gait with four limb ataxia.

A metabolic blood panel including liver profile showed alanine aminotransferase mildly elevated (66 UI/L) with elevated gamma-glutamyl-transpeptidase (gGT: 426 UI/L). Tests for HIV, syphilis and vitamin B12 levels were negative.

Review of initial brain MRI showed a symmetrical, increased fluid-attenuated inversion recovery (FLAIR) signal lesion extending through the hypothalamus, periaqueductal area, mamillary bodies, bilateral anterior thalami, chiasm, both optic tracts and posterior limbs of both internal capsules with restricted diffusion and patchy contrast enhancement (Figure 1, 1a–1b).

A possible Wernicke-Korsakoff syndrome diagnosis was achieved. Following the initial examination, the patient was initiated on prophylactic parenteral thiamine reposition.

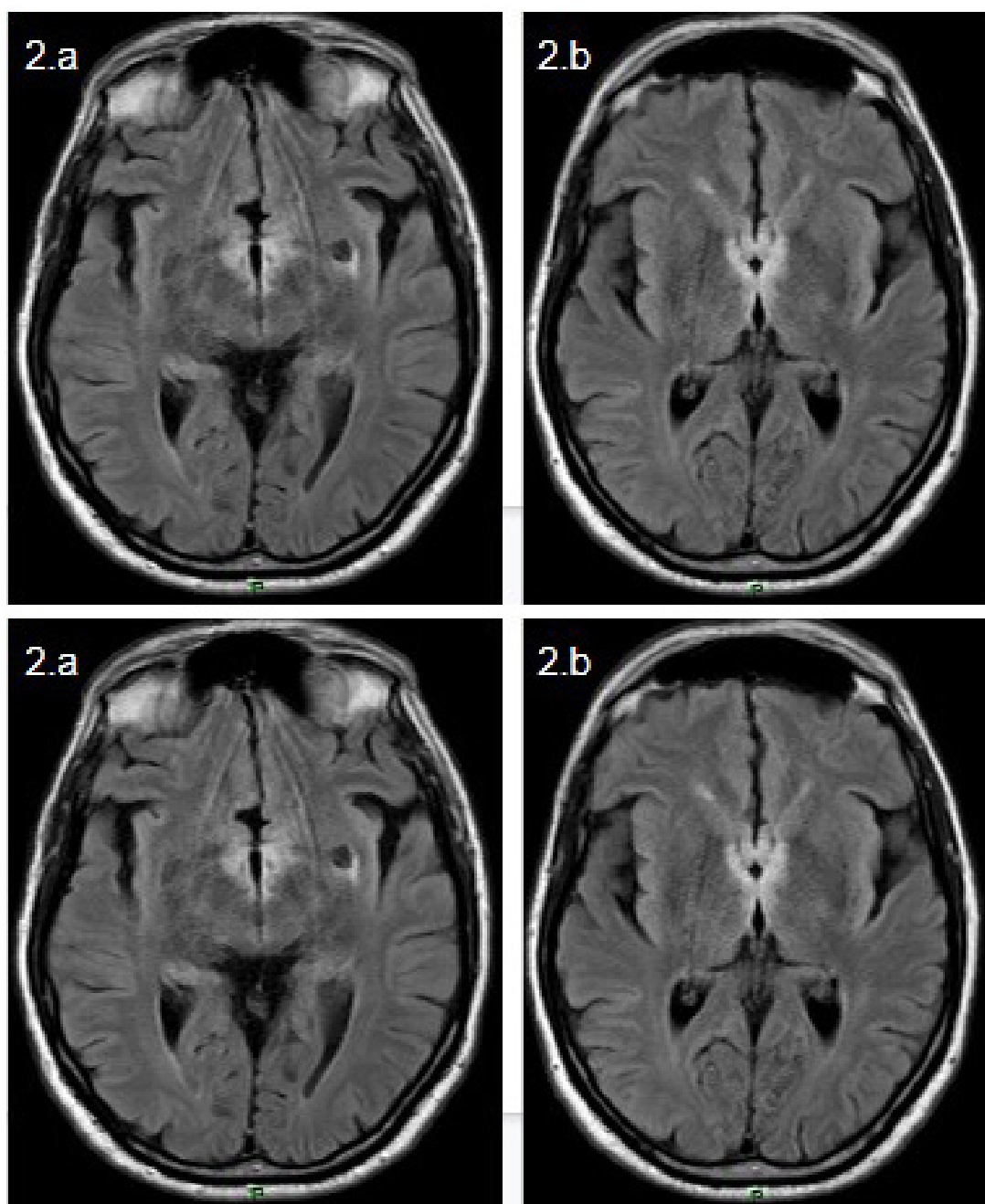
CSF analysis showed elevated proteins (174 mg/dl) and lactate concentration (2.9 mmol/L). Cytologic and immunocytochemical study showed no neoplastic processes. Screening of autoimmune antibodies in CSF and paraneoplastic antibodies in serum were negative. EEG and full-body CT scans were unremarkable.

Thiamine serum levels were normal (16,5 ug/L) (blood sample collected previous to reposition).

Finally, a neurocognitive test indicated malperformance in tasks related to immediate and delayed recall and disturbances in recent and remote memory with confabulation.

A new brain MRI after supplementation showed regression of the previous lesion (Figure 1, 2a-2b).

He was discharged one month later with residual anterograde amnesia and gait instability that are still present eleven months later, at the last follow up.



**Figure 1.** (1) Axial FLAIR MRI sequence showing extensive, symmetrical, hyperintense lesion in chiasm and both optic tracts (1.a) and periaqueductal area, hypothalamus and posterior limb of internal capsules (1.b). (2) Axial FLAIR MRI sequence showing regression of the hyperintense lesion one month after thiamine repositioning in chiasm and both optic tracts (2.a) and periaqueductal area, hypothalamus and posterior limb of internal capsules (2.b)

Source: Authors.

Wernicke's encephalopathy (WE) prototypical clinical triad consists of motor problems such as ataxia or gait incoordination, ocular signs (commonly ophthalmoparesis and nystagmus) and mental status changes (Wicklund & Knopman, 2013; Isenberg-Grzeda, Rahane, DeRosa, Ellis & Nicolson,

2016). Thiamine (vitamin B1) deficiency secondary to alcoholism is the most common etiologic factor (Isenberg et al., 2016). If left untreated or undertreated, there is an increased risk of developing a chronic sequelae: the Korsakoff's Syndrome (KS), characterized with loss of working memory

and confabulation with sparing of remote memories (Sullivan & Pfefferbaum, 2009; Sinha, Kataria, Kolla, Thusius & Loukianova, 2019). Both syndromes together are termed Wernicke-Korsakoff syndrome (WKS).

Even though diagnosis of WKS remains primarily clinical<sup>3</sup>, brain MRI findings in previously reported typical locations (Wicklund & Knopman, 2013; Isenberg, et al., 2016) are highly specific of this syndrome, suggesting MRI is a valuable confirmation tool.

Normal thiamine serum levels shouldn't dismiss the initial suspicion, as its blood concentration does not necessarily reflect brain tissue's concentration (Sinha et al., 2019).

Our case suggests that, even when MRI lesions are not characteristic, intravenous thiamine reposition should be immediately initiated if WKS is suspected, considering the patient's outcome depends on prompt diagnosis and adequate treatment.

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