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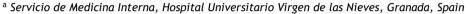
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## MEDICINE IN IMAGES

## Wernicke's encephalopathy<sup>☆</sup> Encefalopatía de Wernicke

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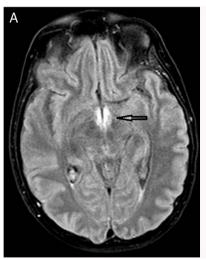




Figure 1

A 52-year-old woman with a known history of active tobacco use and alcoholism was admitted to the emergency department after discovering her at home in a reduced state of consciousness and urinary incontinence. Laboratory tests revealed a slight increase in gamma-glutamyl

transpeptidase (GGT), with no ion or red blood cell abnormalities. The physical examination revealed left eye esotropia, significant temporal and spatial disorientation and agitation. The patient underwent cranial computed tomography and lumbar puncture, both without findings, as well as an electroencephalogram that showed no epileptiform findings. Based on suspected Wernicke encephalopathy, we started treatment with high-dose thiamine. The patient was hospitalized to monitor her progress and maintain surveillance. Magnetic resonance imaging revealed T1 hypointensity and FLAIR and T2 hyperintensity at the periaqueductal, pulvinar, bilateral medial thalamic

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and diencephalic floor level (Fig. 1A and B, FLAIR sequence), consistent with the suspected diagnosis. Treatment with thiamine was maintained at descending doses, with the patient's condition significantly improving, with only certain anterograde amnesia remaining at discharge.

Wernicke encephalopathy is a syndrome with neurological impairment produced by vitamin B1 (thiamine) deficiency, which requires early identification and treatment to prevent irreversible damage and progression to Korsakoff syndrome. Wernicke encephalopathy should be suspected in conditions of alcoholism and malnutrition when faced with the onset of oculomotor impairment or nystagmus, ataxia and impaired state of consciousness. Treatment with thiamine should be started as soon as possible.