

An acute neurological complication of Crohn's disease

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A 53-year-old man with a history of Crohn's disease presented acute onset of diplopia, dizziness and confusion accompanied by nausea and vomiting. On admission to the neurology ward, clinical examination revealed the presence of ophthalmoparesis, horizontal nystagmus in the primary position and in both directions of gaze, generalized muscular atrophy, right hemiparesis and cerebellar ataxia.

Laboratory tests showed pancytopenia, with anemia and proteinuria, and normal sodium, potassium, chloride and creatinine levels. A 3 Tesla magnetic resonance imaging (MRI) study of the brain showed, on fluid-attenuated inversion recovery (FLAIR) sequences, marked hyperintensity of the periaqueductal gray matter around the third ventricle, the mammillary bodies, the hypothalamus and the medial part of the thalamus (Fig. 1). Other bilateral hyperintense areas on MRI were located in the posterior medulla and in the posterior wall of the fourth ventricle.

After administration of contrast medium, enhancement was observed at the level of the posterior medulla, periaqueductal gray matter, inferior colliculus, hypothalamus and medial part of the thalamus.

A diagnosis of nonalcoholic Wernicke's encephalopathy as a complication of Crohn's disease was made (Maeda et al., 1995; Liong et al., 2016). Following initiation of replacement therapy with parenteral B1 vitamin (thiamine), there was a significant improvement of the neurological status.

In 1881, Carl Wernicke, in Germany, first described a syndrome of acute confusion, ophthalmoplegia and ataxia, while, only six

years later, Sergej Sergeevič Korsakoff, in Russia, described a series of patients with predominantly anterograde amnesia. However, it was not until several years later (and after several additional cases had been published) that the two conditions were recognized as part of the same clinical spectrum: Wernicke-Korsakoff syndrome, associated with chronic alcoholism and other conditions of malnutrition all leading to thiamine deficiency (Sechi and Serra 2007).

The distinctive MRI features of this often underestimated syndrome make it one of the few cases in clinical neurology in which neuroimaging is pathognomonic.

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Compliance with Ethical Standards.

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Informed consent: Informed consent to publish the material included in the case report was obtained from the patient.

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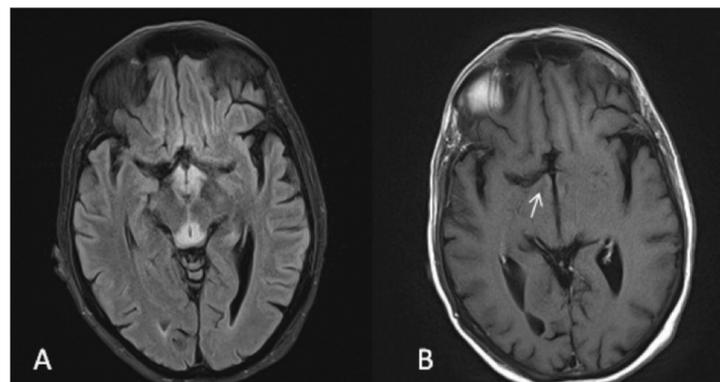


Figure 1 - (A) Brain MRI (axial FLAIR sequences, TR 9000, TE 100) showing marked hyperintensity in several brainstem and diencephalic regions. (B) Brain MRI (axial SE T1 sequences, TR 2280, TE 62) after administration of contrast medium showing slight linear contrast enhancement (arrow) at the level of the hypothalamus.