Neuroimaging in hypoglycaemic encephalopathy diagnosis and prognosis: A case report☆,☆☆

Neuroimagen en el diagnóstico y pronóstico de la encefalopatía hipoglucémica: a propósito de un caso

Dear Editor:

Hypoglycaemic encephalopathy can be defined as the presence of coma or stupor in patients with glucose levels below 50 mg/dL, persisting for more than 24 hours despite normalisation of blood glucose levels, in the absence of other possible aetiologies.[1] This condition is rare but potentially severe, with a mortality rate of up to 50%, depending on the series.[1–3] We present the case of a patient with typical clinical symptoms and neuroimaging findings.

The patient was a 63-year-old man who had been institutionalised in the previous year due to progressive cognitive impairment (frontotemporal dementia type), with a history of type 2 diabetes mellitus treated with sulphonylureas. During the course of a urinary tract infection, he was found in the early hours of the morning in a coma and with hypoglycaemia (19 mg/dL); the duration of that situation could not be determined, and the patient showed no clinical response to the immediate metabolic correction. Upon arrival at our hospital, the patient presented low-grade fever, a Glasgow Coma Scale score of 3, roving eye movement, and decerebrate rigidity of the left arm, with no other pathological findings; he was admitted to the intermediate care unit. During hospitalisation, complementary tests reliably ruled out an infectious, epileptic, or vascular origin of the symptoms: no alterations were detected in the blood test; CT and CT angiography showed no signs of acute ischaemia or haemorrhage or large-vessel occlusion; CSF analysis with blood formula, culture, and C-reactive protein with negative results for viruses; EEG revealed focal peri-

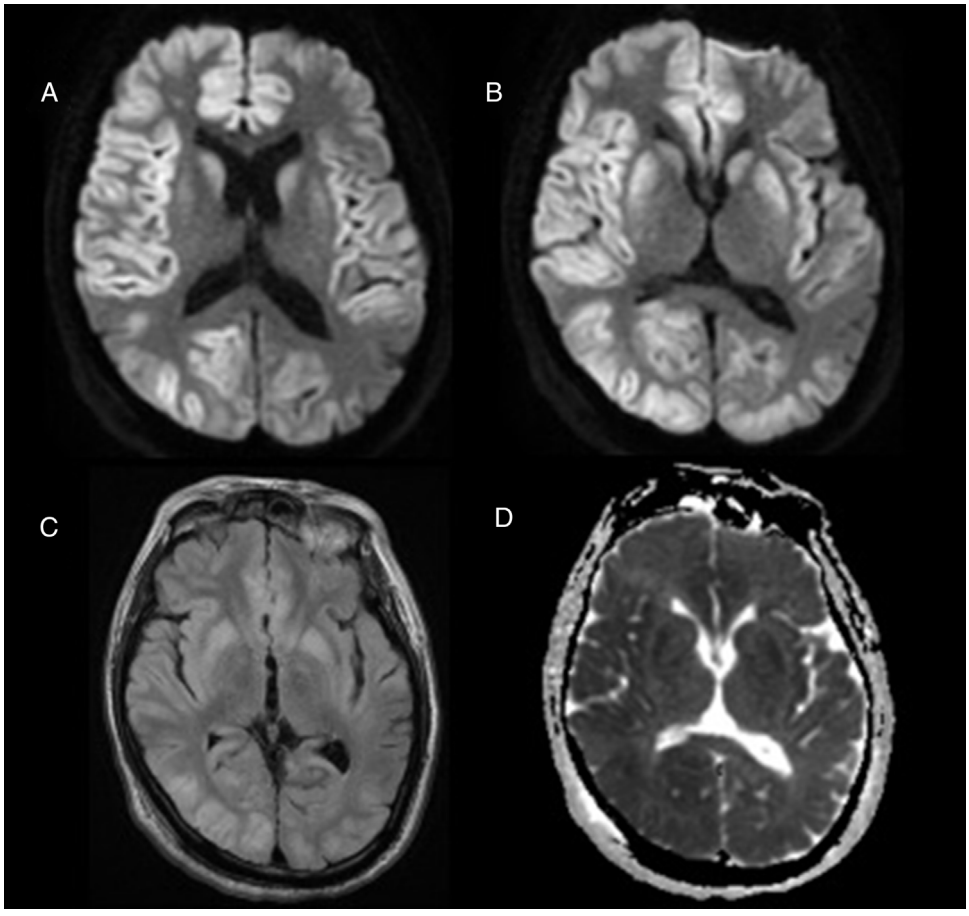

Figure 1 Brain MRI scan performed 48 hours after admission showing hyperintensities on the diffusion-weighted (A and B) and FLAIR sequences (C), with diffusion restriction on ADC maps (D).

☆ Please cite this article as: Rodríguez-Vázquez A, Vicente-Pascual M, Mayà-Casalprim G, Valldeoriola F. Neuroimagen en el diagnóstico y pronóstico de la encefalopatía hipoglucémica: a propósito de un caso. Neurología. 2020;35:131—132.

**This work was submitted for presentation as a poster at the 21st Annual Meeting of the Catalan Society of Neurology.

odic epileptiform activity in the left frontotemporal area and diffuse theta slowing. A brain MRI study performed at 48 hours displayed hyperintensities on T2-weighted, FLAIR, and diffusion-weighted sequences, with ADC maps showing restricted diffusion in cortical and deep grey matter with thalamic preservation; all these findings are compatible with persisting hypoglycaemia (Fig. 1). Despite metabolic correction and support measures, the patient's condition deteriorated and he eventually died.

Hypoglycaemic encephalopathy presents a wide clinical spectrum, and may manifest as epileptic seizures, focal neurological deficits, or decreased level of consciousness. It is important to rule out other causes of encephalopathy, especially toxic and metabolic causes. Diffusion-weighted brain MRI sequences show hyperintensities in the grey matter of the cortex, hippocampus, internal capsule, and basal ganglia in up to 70% of cases.[1–7] Thalamic preservation is characteristic,[2] unlike in the case of hypoxic encephalopathy. The extension of the lesions on MR images may predict prognosis and neurological sequelae,[2,4,5] although the literature includes contradictory data.[1] Several studies have associated basal ganglia involvement with poor prognosis,[2] although some retrospective studies and clinical cases do not report this association.[1] The brain's vulnerability to hypoglycaemia is believed to vary, even between areas of the cerebral cortex, with the parietal occipital cortex being the most vulnerable.[4–6] No reports analyse whether patients with neurodegenerative diseases present a lower tolerability to situations of hypoglycaemia, which would explain the fatal outcome in our patient in spite of glycaemic correction.

In conclusion, hypoglycaemic encephalopathy is a relatively rare entity and should therefore be considered in patients with decreased level of consciousness and serum glucose levels below 50 mg/dL in whom other causes have been ruled out; early glycaemic correction is vital in these cases. Despite the differences in vulnerability between brain areas, it seems clear that greater lesion extension on MR images is associated with higher morbidity and mortality rates; neuroimaging is therefore a useful tool not only for diagnosis but also for neurological prognosis.

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https://doi.org/10.1016/j.nrleng.2017.09.009
2173-5808/

The relevance of anhidrosis in Horner syndrome: analysis of an image☆,☆☆

La relevancia de la anhidrosis en el síndrome de Horner. A propósito de una imagen

Dear Editor:

Horner syndrome is characterised by ptosis secondary to paralysis of the superior tarsal muscle, miosis, pseudoenophthalmos, and, on occasion, anhidrosis or hypohidrosis.[1] The condition may result from a number of causes, including head or neck trauma, brain haemorrhage, cervical disc disease, neck or apical lung tumours, stroke, lateral medullary syndrome, cluster headache, carotid artery dissection, multiple sclerosis, syringomyelia, acute transverse myelopathy, and thoracic aortic aneurysms.[2] Carotid artery dissection is the most frequent cause of painful Horner syndrome.[3,4] Exploring anhidrosis and any other skin changes that may appear may help locate the involvement of the cervical sympathetic chain.[5] We present the case of a patient with Horner syndrome and an infrequent though characteristic change in face colouration that alarmed the patient.

This 44-year-old man was a triathlete and reported no allergies to medications and no alcohol, smoking, or drug habits. During the swimming leg of a triathlon, the patient received a blow to the right side of the neck and began to feel pain in the right side of the face and neck. Pain persisted after finishing the swimming leg, and was associated with right-sided ptosis and blurred vision; the patient went

☆ Please cite this article as: Vicente-Pascual M, Montejo C, Sánchez A, Llull L. La relevancia de la anhidrosis en el síndrome de Horner. A propósito de una imagen. Neurología. 2020;35:132–134.

☆☆This study was submitted to the 6th Competition of Stroke Units, run by the Spanish Society of Neurology.