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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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

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

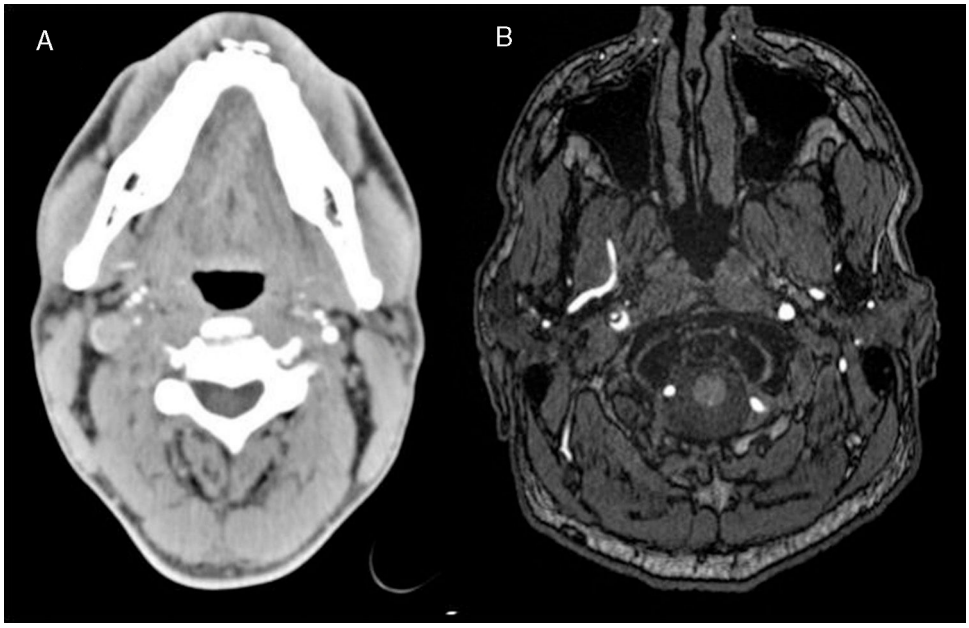

Figure 1 (A) CT angiography showing reduced blood flow in the right internal carotid artery at the level of the cervical spine. (B) MRI angiography (time-of-flight sequence) showing reduced blood flow in the same artery (this sequence was taken at a higher level than the CT angiography sequence) and a false lumen in the medial region.

to hospital 24 hours later. The examination revealed right miosis with preserved pupillary light reflex and right ptosis; the patient reported diplopia with extreme right gaze positions. The neurological and general examination revealed no other relevant findings. A blood analysis detected no remarkable alterations. A brain CT scan and a CT angiography study showed no parenchymal alterations and revealed reduced contrast flow in the extracranial section of the right internal carotid artery (after the bifurcation), with occlusion of the arterial lumen (Fig. 1A). Brain MRI and MRI angiography findings suggested right internal carotid artery dissection at the cervical level, associated with a lack of blood flow from the carotid canal to the venous sinus on time-of-flight sequences (Fig. 1B), and punctiform cortical—subcortical ischaemic lesions in the watershed territory of the right anterior and middle cerebral arteries. The patient was diagnosed with Horner syndrome and headache secondary to traumatic right internal carotid artery dissection, and was discharged with oral anticoagulants and conventional analgesics. After several weeks of rest, and resuming work and daily activities, including exercise, he became concerned by his symptoms and sent us a photograph of himself (Fig. 2). He presented changes in facial skin colouration after exercise: the left side of his face was flushed and sweaty, whereas the right side was pale, showing anhidrosis. A 3-month follow-up examination revealed clinical improvements, with mild right ptosis and miosis persisting. Additional MRI and MRI angiography studies revealed no alterations; acenocoumarol was switched for acetylsalicylic acid.

Horner syndrome is caused by disruption of sympathetic nervous system fibres. The first-order neuron emerges from the hypothalamus and descends through the brainstem to the spinal cord, where it synapses in the Clarke column, at the C8-T1 level. The second-order neuron exits

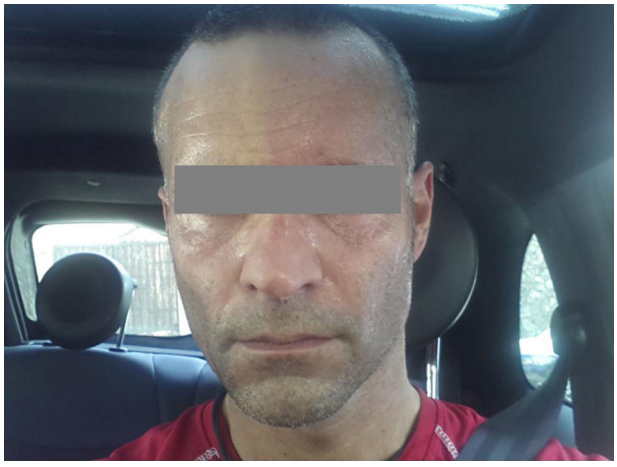

Figure 2 Photograph sent by the patient after exercise, showing anhidrosis and pallor on the right side of his face, ipsilateral to carotid artery dissection, and flushing and sweating on the left side.

the spinal cord via the T1 nerve root, ascending through the cervical chain ganglia and synapsing in the superior cervical ganglion (C1-C2). The third-order (postganglionic) neuron travels along the carotid artery to the iris dilator muscle.[5] First-order neuron lesions affect sweating on the same side of the body, whereas second-order neuron lesions cause ipsilateral facial alterations and third-order neuron lesions either do not affect sweating or affect sweating only in the upper part of the face.[6] In our patient, compression of the cervical sympathetic plexus was located at the level of the internal carotid artery, immediately below the superior cervical ganglion. Insufficient evidence is available to confirm that anticoagulants

are more effective than antiplatelets.[7,8] However, early treatment is essential to prevent ischaemic brain lesions; associated mortality may reach 20%. Around 30% of patients are left with permanent neurological sequelae.[9] In our patient, the acute episode was managed with anticoagulants due to MRI evidence of atheroembolic lesions; once symptoms had resolved, the patient received conventional antiplatelets.

We present the case of a patient with rare though characteristic imaging signs of cervical sympathetic chain involvement. The association between anhidrosis or hypohidrosis and skin colouration changes is a typical sign of the disease that helps locate the lesions to the cervical sympathetic chain.

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We wish to express our sincere thanks to our patient for consenting to the publication of his case and images; this study would have not been possible without his cooperation.

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Chorea/ballism secondary to non-ketotic hyperglycaemia: report of 4 cases☆

Corea/balismo secundaria a hiperglucemia no cetósica: serie de 4 casos

Dear Editor:

Chorea or ballism is a hyperkinetic disorder characterised by involuntary, abrupt, irregular, large-amplitude movements due to basal ganglia lesions. Causes vary greatly, and include vascular, metabolic, degenerative, and infectious aetiologies; deficiency diseases, etc. Among the metabolic causes, chorea associated with hyperglycaemia is noteworthy: despite being an infrequent disease, it is potentially reversible when treated correctly.[1]

We present the cases of 4 patients (3 women and one man) who attended the emergency department due to uncontrollable choreic movements (predominantly left-sided in 3 and bilateral in one) progressing for more than 24 hours (Table 1). All 4 cases were assessed by a neurologist in the emergency department. The rest of the neurological examination was normal. An emergency blood analysis including ions, urea, creatinine, glucose, complete blood count, coagulation, and venous gases revealed high glucose levels (ranging from 196 to 939 mg/dL) and pH > 7.3 in all cases. A urine analysis detected high glucose levels and absence of ketone bodies in 3 patients (trace values were detected in patient 2). Patients 1 and 3 had previously been diagnosed with diabetes mellitus, whereas patients 2 and 4 had not. All patients presented arterial hypertension and dyslipidaemia under treatment. All 4 cases were admitted for study. During admission, a blood test including liver and kidney function tests, lipid profile, ions, thyroid hormones, vitamin B_{12}, folic acid, complete blood count, iron profile, and coagulation returned normal results (except in one case, showing known kidney failure, which was under follow-up); serology tests for HIV, HCV, HBV, and *Treponema pallidum* were negative in all cases. All patients showed glycated haemoglobin levels far above normal limits (13.8%-16.5%). All patients underwent emergency CT and MRI scans

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