Hypoxic ischaemic encephalopathy: lesions on magnetic resonance

Encefalopatía hipóxico-isquémica: lesiones en resonancia magnética

Dear Editor:

Hypoxic ischaemic encephalopathy (HIE) is a clinical and anatomical condition characterized by motor and neuropsyschological sequelae secondary to the lack of oxygen due to the cessation of blood flow in the brain. The severity of the lesions correlates to the duration of the lack of oxygen and it is estimated that, after more than 4-5 minutes of anoxia, the lesions are irreversible.[1]

We present the case of a 41-year-old male who suffered, under the effects of toxic substances, a fall of 5 metres in height causing cranioencephalic trauma and cardiac arrest. Failure was reversed after cardiopulmonary resuscitation, with total anoxia duration estimated at 20 minutes. On admission to the ICU, the toxicological study was positive for ethanol, cocaine and cannabis. On suspension of the initial sedation, the patient presented myoclonic status that reverted after treatment with levetiracetam, midazolam and valproic acid.

On examination 3 months after admission, the patient is in a vegetative state with spontaneous eye opening and unreactive intermediate pupils, presents slow horizontal eye movements and retained oculocephalic reflexes while maintaining good respiratory mechanics, without motor response to visual or painful stimuli.

The nuclear magnetic resonance (NMR) scan performed 15 days after admission showed the following lesions: diffusion-weighted hyperintensity in the splenium of the corpus callosum, hyperintensity of basal nodes in T1, T2 (Figure 1A) and FLAIR (Figure 1B) sequences compatible with sub-acute haemorrhagic infarctions, gyral images of hypersignal in both occipital lobes suggesting pseudo-laminar necrosis in T1, cortical and sub-cortical foci suggesting sub-acute infarction in the territory bordering the medial and anterior cerebral arteries at the bilateral frontal and insular level, with blurring of grey and white matter (Figure 1) and a normal ventricular pattern.

NMR imaging is a complementary test to be requested in HIE and shows a suite of characteristic lesions: in less than 24 hours, an increase in restriction is observed in the diffusion-weighted sequence as a consequence of cytotoxic oedema. Over the first 15 days, diffusion restriction gradually reduced and the T2-weighted and FLAIR sequences show hyperintensity in the areas affected and an image of sulci absent due to swelling. Haemorrhagic infarctions are seen as petechial hyperintensity and there is widespread blurring of the grey and white matter. Subsequently, it is possible to observe atrophic changes and signs of laminar cortical necrosis in T2.[2]

Involvement of the brain in HIE is characteristically bilateral and symmetric, but there are structures such as the hippocampus (region CA1), the medial layer of the cerebral cortex, the corpus striatum, the thalamus and the Purkinje cells that are more intensely affected.[2,3] Certain characteristics of these areas contribute to their selective vulnerability to deprival of oxygen: a greater relative metabolic demand, a larger amount of excitatory neurotransmitters, a more modern phylogenetic origin justifying the involvement of the corpus striatum, but not the globus pallidus, and the location in two arterial territories.[2]

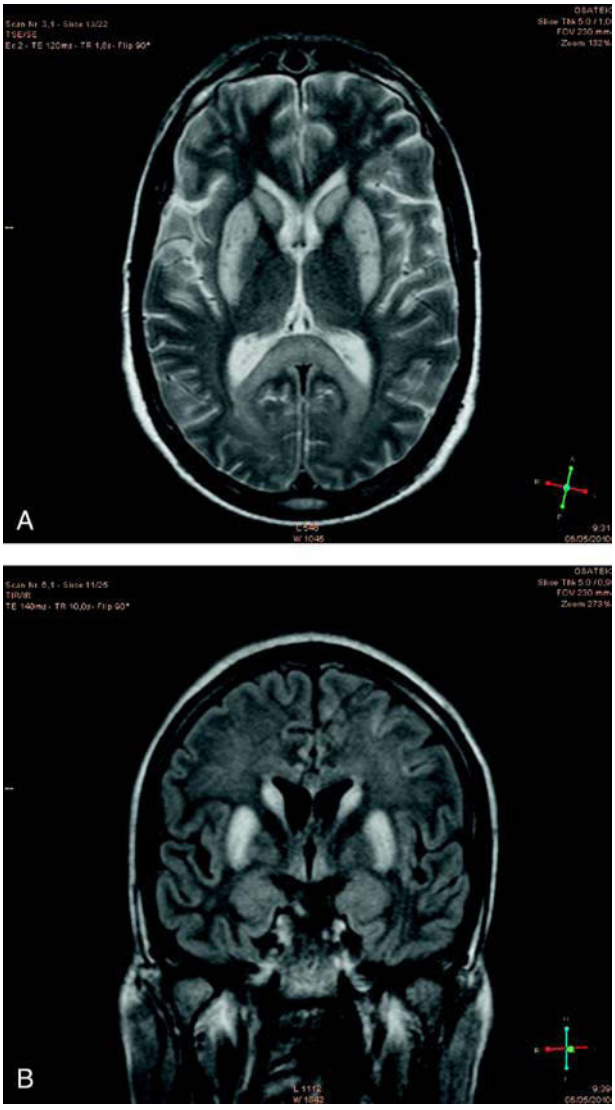

Figure 1 Cranial MRI performed 15 days after admission. A. T2 sequence axial slice; notable hyperintensity on basal nodes and blurring of white and grey matter. B. FLAIR coronal slice; in addition to the hyperintensity on basal nodes and blurring of white and grey matter, widespread gyral swelling is visible giving an image of atrophied sulci.

The lesions observed on the MRI scan for this patient do not point to any toxic aetiology, as the encephalopathies secondary to the substances consumed by the patient mainly affect other structures such as the globus pallidus and the supratentorial white matter in the case of cocaine,[4] and the third ventricle and mamillary bodies in the case of

Wernicke's acute encephalopathy due to a B1 deficit secondary to alcohol intake.[5,6]

In terms of prognosis, the MRI findings of laminar cortical necrosis and hyperintensity at basal nodes, together with clinical data, duration of the anoxia, absence of pupil response,[7] absence of motor response[7] and early myoclonic status[8] allowed a poor evolution to be forecast for this patient.

In conclusion, the progression and dissemination of CPR techniques has generated an increase in the number of patients surviving CPR with HIE sequelae, and this has increased the need to determine clinical, electrophysiological or radiological markers allowing short-term and long-term prognoses to be established. NMR imaging provides prognostic information from the acute moment[3,9,10] and it is particularly helpful in cases of patients under sedation, where clinical parameters are less useful.[5]

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Association of peripheral facial nerve palsy and seropositivity of HTLV 1, a case report

Asociación entre parálisis facial periférica recurrente y seropositividad HTLV-1: a propósito de un caso

Dear Editor:

The aetiological diagnosis of peripheral facial nerve palsy (PFNP) covers a broad spectrum of pathologies; despite this, a highly variable proportion of between 62 and 93% are considered to be idiopathic or Bell's palsy.[1] We must not lose sight of the fact that this is a diagnosis by exclusion, and be alert to the data that lead us to suspect other causes in order to avoid overestimating the figures of idiopathic facial nerve palsy (IPFNP). One of these aetiologies is as yet emerging and still under study: seropositivity for the type 1 human T lymphotropic virus (HTLV-1).

We report the case of a 56-year-old male, originally from Peru and a resident of Spain for the last 18 years. High blood pressure and obesity were the most salient findings in his medical history. He initially presented PFNP on the left side that presented an incomplete response to steroid treatment; the mild erasure of the left nasogenian sulcus and Bell's sign persisted. Approximately 6 months later, the patient presented at the Emergency Department due to contralateral PFNP; the cerebral computerized tomography (CT) performed was normal. The patient was therefore sent home with a decreasing dosing scheme of steroids. One month later, he consulted due to clinical worsening, at which time the examination revealed that he could not close his right eye, Bell's sign, epiphora, and scant mobility in the lower right region of the face, which made it particularly difficult for him to eat and speak. The rest of the neurological examination was normal and there were no signs of lingua plicata or angiooedema. On admission, several complementary studies were carried out but failed to exhibit any significant alteration: blood panel, biochemistry, vitamin B_{12}, folic acid, autoimmunity, cell immunity,