Subcortical reversible T2-weighed hypointensities in seizures secondary to diabetic ketoacidosis

Hipointensidad subcortical reversible en secuencia T2 en crisis convulsivas secundarias a cetoacidosis diabética

Dear Editor:

Seizures are frequent in patients with non-ketotic hyperosmolar hyperglycaemia and more unusual in ketoacidotic decompensations.[1]

After an epileptic crisis, it is possible observe transient alterations in the cerebral magnetic resonance images (MRI), namely hyperintensities in T2 and FLAIR.[2,3] The presence of T2-weighted hypointense subcortical focal images have recently been pointed to as a characteristic finding of epileptic seizures associated with non-ketotic hyperosmolar hyperglycaemic states.[4]

We report the case of a patient suffering epileptic seizures in the context of uncontrolled diabetic ketoacidosis (DKA), in whom a reversible focal hypointensity was noted in the right parietal white matter on the cranial MRI (T2-weighted sequence).

A 76-year-old woman who did not know she was diabetic was admitted due to DKA. During her admission, she suffered 4 partial motor crises with secondary generalization, starting with left hemicorporal motor issues and generalizing into a tonic-clonic seizure lasting between 1 and 3 minutes, with recovery of the level of consciousness between crises and in a period of 5 hours, followed by an intense post-critical right hemisphere deficit, with left hemiparesis, hemihypoaesthesia, hemianopsia and hemisensory neglect, recovering in the next 24 hours. Bloodwork revealed 596 mg/dL glycaemia, pH 7.30, glycosuria and ketonuria, with serum osmolarity calculated at 318 mOsm/L. Treatment was begun with levetiracetam (500 mg *per os* every 12 h), and the seizures have not recurred. A cranial MRI scan was performed 18 hours after the first seizure, showing T2 hypointensity in right parietal subcortical white matter with slight cortical hyperintensity in FLAIR, tenuous gyriform uptake of contrast and slight restriction of diffusion in this location (fig. 1). The electroencephalogram (EEG) showed a right frontotemporal focus of delta waves. The patient was discharged after the ketoacidotic imbalance was resolved without neurological focality. An MRI scan performed 3 months later showed the resolution of the anomalies detected in the initial MRI (fig. 2). Anti-epileptic treatment was suspended without the seizures recurring.

Seizures were associated with non-ketotic hyperglycaemic imbalances (NKHI) in 15-40% of patients. Motor focal crises and partial continuous epilepsy are particularly common in these patients.[5] Epileptic seizures have also been described in ketotic hyperglycaemic crises, albeit less frequently.[6] The pathogenesis of the seizures associated with metabolic disorders has not been completely clarified. Hyperglycaemia may precipitate the crises by reducing the levels of gamma-aminobutyric acid (GABA), thus lowering the seizure threshold. Intracellular ketosis and acidosis might increase this threshold.[7] the most widely accepted hypothesis to explain this increase in the seizure threshold is that the ketone bodies increase the synthesis of GABA by raising the

Figure 1 Hyposignal in the subcortical white matter on the right parietal lobe in T2-weighted and FLAIR sequences (A and B) with slight restriction of diffusion in the cortex (C) and gyriform uptake of contrast in T1-weighted sequence (D).

Figure 2 Resolution of the lesions observed. A) T2-weighted sequence; B) FLAIR sequence; C) diffusion sequence; D) T1 after administration of gadolinium.

activity of the glutamic acid decarboxylase enzyme in the Krebs cycle.[8] The patient reported here presented, paradoxically, an accumulation of crises in the context of a DKA, with mild acidosis. The MRI findings correlated well with both the changes in the EEG and her clinical symptoms.

Neuro-radiological alterations have been described after isolated or recurrent epileptic crises. The MRI anomalies are typically hyperintense lesions in the white matter in the T2 or FLAIR sequences.[2,3] Some authors have recently described transient subcortical T2 hypointensities in patients with crises and NKHI in both retrospective and prospective studies.[4,9,10] There is one reported case of ketotic hyperglycaemia associated with partial continuous epilepsy with reversible hypointensity of the subcortical white matter in T2 sequences.[6] Attention has also been called to the existence of diffusion restriction in patients with visual crises in the course of a NKHI.[11] Diffusion restriction suggests the presence of cytotoxic oedema. Local cytotoxic oedema may be related to both the seizure itself and the existence of focal ischaemia or hyperviscosity.[12] The slight gyriform uptake of contrast has also been described in crises associated with NKHI. During the crises, metabolic changes such as hypoxaemia, oedema, acidosis and cell membrane alterations, associated with endothelial dysfunction in diabetic patients, may lead to a disruption of the blood-brain barrier.[7]

In conclusion, patients with seizures symptomatic of DKA may present focal hypointensity in T2-weighted white matter with diffusion restriction, as well as cortical hyperintensities in FLAIR with gadolinium uptake. The semiology of the seizure and post-crisis stages and the changes in EEG correlate with these findings. Acknowledging these changes will facilitate the differential diagnosis when studying these patients.

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Selective IgA deficiency and multiple sclerosis

Déficit selectivo de IgA y esclerosis múltiple

Dear Editor:

Multiple sclerosis (MS) is the most common demyelinating autoimmune disease of the central nervous system in young adults and is one of the leading causes of non-traumatic, neurological disability.[1-3]

Selective IgA deficiency is the most frequent primary immunodeficiency.[4] Generally speaking, this deficiency is not associated with disease and is only revealed when routine laboratory studies are performed. However, the deficit of IgA is usually associated with infections of the respiratory and gastrointestinal tracts, and less often, with allergic and autoimmune diseases; these latter associations are a bit hazy from a physiopathological point of view.[5-8] Specifically, the association between selective IgA deficiency and autoimmune phenomena has been reported in both systemic, as well as organ-specific processes, the most widely reported of which are haematological disorders (idiopathic thrombocytopenic purpura), diseases of the gastrointestinal tract (ulcerative colitis), endocrine diseases (autoimmune thyroiditis), and rheumatological diseases.[9] To date, there have been no case reports of the association between IgA deficiency and MS.