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INTOXICATION WITH ORAL HYPOGLYCEMICS AS A CAUSE OF DIFFUSE BRAIN INJURY

Case report

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ABSTRACT - *Objective:* To present the imaging findings of a patient with diffuse brain injury secondary to intoxication with oral hypoglycemics. *Case report:* A nine years-old boy presented with generalized tonic-clonic convulsions and decreased consciousness. Laboratory investigation demonstrated blood glucose level of 21 mg/dL. The magnetic resonance imaging showed cortical and subcortical temporo-parieto-occipital areas of high signal on T2 and low signal on T1, with high signal on the diffusion-weighted images and low signal on apparent diffusion coefficient maps. *Conclusion:* Cortical and subcortical temporal, parietal and occipital ischemic lesions may be seen in patients with intoxication by oral hypoglycemics.

KEY WORDS: magnetic resonance imaging, diffusion weighted imaging, hypoglycemia.

Intoxicação com hipoglicemiante oral como causa de lesão cerebral difusa: relato de caso

RESUMO - *Objetivo:* Apresentar os achados de imagem em um caso de lesão cerebral difusa secundária a intoxicação por hipoglicemiante oral. *Relato do caso:* Menino de nove anos de idade apresentando crises convulsivas tônico-clônicas e rebaixamento do nível de consciência. Exames laboratoriais revelaram glicose sérica de 21 mg/dL. O exame de ressonância magnética revelou lesões corticais e subcorticais têmporo-parieto-occipitais, com alto sinal em T2 e baixo em T1, apresentando hipersinal nas imagens ponderadas em difusão e hipossinal nos mapas de coeficiente aparente de difusão. *Conclusão:* Lesões isquêmicas corticais e subcorticais nos lobos temporais, parietais e occipitais podem ser identificadas em pacientes com intoxicação por hipoglicemiantes orais.

PALAVRAS-CHAVE: ressonância magnética, imagens ponderadas em difusão, hipoglicemia.

Diffuse brain injury are caused by several disorders, such as hypoglycemia. In addition, different conditions may cause hypoglycemia. In neonates, the most common causes are maladaptive metabolic adjustments in the transition intrauterine to extra uterine life, intrauterine under nutrition, and primary enzymatic or metabolic endocrine abnormalities. Considering another ages, exogenous intoxication and hypoxic-ischemic disorders are the causes of hypoglycemia most frequently demonstrated[1,2]. The histopathological findings seen in patients with transient hypoglycemia and diffuse brain injury have been documented. However, there are only a few studies stressing the imaging findings in these patients[1-4]. Barkovich et al.[2], reviewed the imaging findings of five patients with transient neonatal hypoglycemia. The most common feature was diffuse cortical and subcortical white matter damage, with the parietal and occipital lobes being affected more severely. Globus pallidus injury was seen only in one patient, who had the most severe cortical injury. The diffusion-weighted image (DWI) is a neuroimaging technique that evaluates the water movement, detecting the tissue injury as in acute cerebral damages. Singhal et al.[5] demonstrated the DWI features in three patients who suffered different conditions of anoxic brain injury. All the cases showed severe diffuse lesions in the gray matter of the parietal and occipital lobes, as well as in the striatum, thalamus and hippocampus. To our knowledge, the DWI find-

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ings in patients with brain lesions secondary to severe hypoglycemia have not been reported.

We present the imaging findings of a patient with diffuse brain injury secondary to intoxication with oral hypoglycemics, emphasizing the DWI features.

CASE

A previously healthy nine years old boy was admitted to the hospital with generalized tonic-clonic convulsions and decreased consciousness. His grandmother informed that he had cough and nasal congestion in the two days before, and she gave him some analgesics. At the physical examination he was comatose and showed left ocular deviation. Laboratory investigation demonstrated blood glucose level of 21 mg/dL. Immediately it was administered a bolus of 10% glucose followed by rapid drip infusion. The glucose levels increased to 44 mg/dL after six hours, when he was admitted at the Intensive Care Unit (ICU) and started anti-convulsive drugs (hydantol and benzodiazepines). Another laboratory results were unremarkable. At that time, head CT scan was normal.

At ICU the glucose blood levels were progressively corrected. In the following days, the electroencephalogram showed diffuse slowly of base activity and outbreaks of generalized irritative activities in brain hemispheres. The patient underwent head CT scan, which demonstrated multiple hypodense subcortical areas in the temporal, parietal and occipital lobes. The MRI showed cortical and subcortical areas of high signal on T2-weighted images and discrete low signal on T1-weighted images at the same regions demonstrated in the CT scan (Fig A-B). These areas demonstrated high signal on the DWI and low signal on apparent diffusion coefficient (ADC) maps, suggesting an acute ischemic event.

The grandmother was asked to show the analgesics that the child had ingested, and, actually, they are tablets of oral hypoglycemics that she was taking for diabetics. The patient progressively recovered the consciousness level and discharged the hospital two months later with mild cognition deficit and motor deficiency and some episodes of seizures. On the follow-up, four months later, the patient was showing hyporeflexia in the lower extremity, stereognosis and mild motor incoordination. At this time, the parents agreed with the publication of the case and signed the consent.

DISCUSSION

Hypoglycemia, as a cause of diffuse brain injury, can determine characteristic patterns of damage according to the duration of the event, and patient's age. Maladaptative metabolic adjustments during the transition intrauterine to extra uterine life, intra-

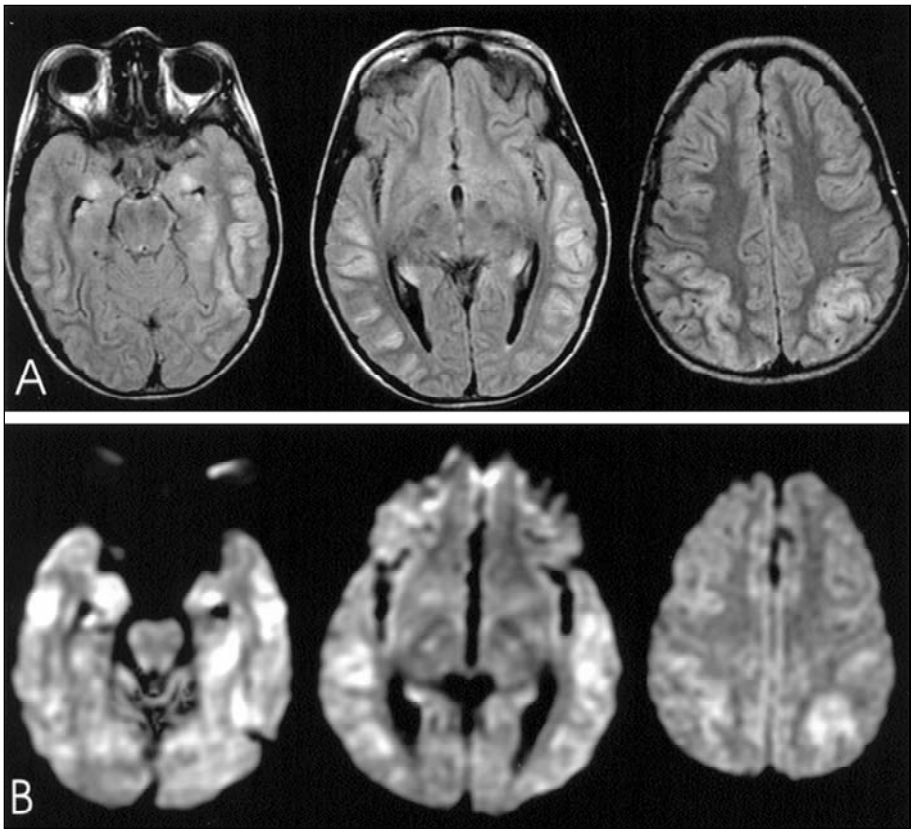

Fig. (A) FLAIR images demonstrating diffuse high signal in the cortical and subcortical regions of the temporal, parietal and occipital lobes. (B) DWI also show high signal in the same areas demonstrated in the FLAIR images.

uterine under nutrition, secondarily to specific primary enzymatic or metabolic endocrine abnormalities are the main causes of hypoglycemia in neonates[2]. Exogenous intoxication, as seen in this case, and hypoxic ischemic disorders are the main causes of hypoglycemia in another ages.

Neuroimaging studies emphasizing the distribution of brain injury in patients with hypoglycemia may evidence a disproportionate involvement of the parietal and occipital lobes[1-6]. Spart et al.[1] showed the CT and MRI findings of one patient with severe hypoglycemia and found progressive parenchymal loss with predominant occipital involvement. Abnormally high intensity in periventricular deep white matter of parieto-occipital lobes on T2-weighted images and hypointensity on T1-weighted were considered characteristic patterns of hypoglycemic lesions[7].

The diffusion-weighted MRI is based on the free movement of the water molecules along random pathways (Brownian motion). Usually, water protons diffuse within a tissue and apparent rate and direction of this diffusion will reflect the barriers that these protons undergo during its translational path length. The diffusion technique uses a pair of gradient pulses to diphase and subsequently rephase protons. Protons experiencing slow or hindered diffusion will largely rephase and it will appear as retention of high signal intensity. Nowadays, there are many applications of DWI in neuroimaging[8,9]. Besides, helping to differentiate acute from chronic infarction in patients with cerebral ischemia still is the main application of the technique[5,6].

In this case, initial head CT scan was normal. Subsequently, the CT scan showed multiple hypodense subcortical areas in the temporal, parietal and occipital lobes. In addition, the MRI demonstrated cortical and subcortical areas of high signal on T2-weighted images and discrete low signal on T1-weighted images at the same regions demonstrated in the CT scan. In these regions the DWI demonstrated high signal and ACD maps low signal, suggesting acute ischemia.

In conclusion, patients with severe hypoglycemia secondary to oral intoxication may present a pattern of diffuse brain damage, showing in the MRI cortical and subcortical temporo-parieto-occipital areas of low signal on T1- and high signal on T2-weighted images. In addition, the DWI may show signals of restriction of the water movement in these areas, allowing the early diagnosis of this severe cause of brain injury.

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