

Interest of Diffusion MRI in Hypoglycemic Coma

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Article Information

Received date: Jun 08, 2017

Accepted date: Aug 18, 2017

Published date: Aug 22, 2017

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Keywords Hypoglycemia; Coma;
Diffusion; Hyperintensities

Abstract

Hypoglycemia is a frequent and severe cause of coma, especially of diabetic patients. Prognosis is related to irreversible neuronal lesions and the most important predictive factors are the severity and duration of hypoglycemia. Cerebral cortex, hippocampus and basal ganglia are the most vulnerable sites.

We report a case of hypoglycemic coma with cortical and basal ganglia hyper-intensities on diffusion weighted MRI suggesting cerebral complication, also we focused on the interest of Diffusion weighted MRI sequences in this issue through a literature review.

Introduction

Hypoglycemic coma is a quite frequent emergency situation of diabetic subject. The diagnostic is clinical and biological. However, it is not contributing to look for neurologic complications immediately, because of the frequent normality of cerebral CT-Scan. Diffusion MRI seems to be the only technique that can detect neurologic lesions specific to hypoglycemic coma.

Observation

A 38 years old woman, with 10 years history of insulin dependent diabetes, presented to the emergency with postpartum coma.

Biological tests suggested a hypoglycemic cause for coma.

Intravenous glucagon and G30 % were administered without response or change of her level of consciousness.

A brain MRI revealed bilateral and symmetric hyper-intensities on T2, FLAIR and markedly on diffusion weighted images located in frontal, parietal and occipital cerebral cortex and the caudate nuclei (Figure 1). They showed hypo and iso signal on T1 images, without mass effect on midline structures.

Venous and arterial MRI angiographies were normal.

We concluded to hypoglycemic coma based on MRI and biological results.

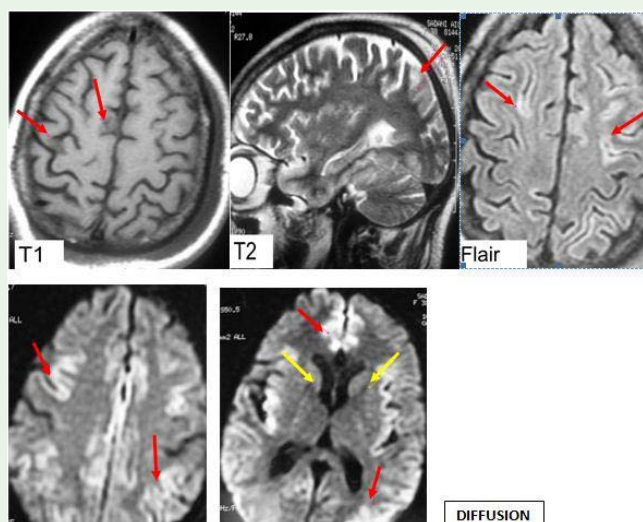


Figure 1: MRI axial T1, sagittal T2, axial FLAIR and diffusion : Cortical hyperintensities on T2, Flair, diffusion and T1 hypo-intensity (red arrows). Hyperintensities of the caudate nuclei (yellow arrows).

Discussion

Hypoglycemic coma is a very common in the course of life of diabetics, especially insulin dependent ones. It can occur for glycemia ranging between 2,3 and 2,7 mmol/l (0,41 and 0,49 g/l) [1].

Consciousness disorders during hypoglycemia are caused by glucose deprivation leading to the interruption on many neurological circuits in which Renin Angiotensin Aldosterone system.

Then, neuronal dysfunction and cell necrosis might happen, leading to irreversible changes. Brain death might happen, in 2- 4 % of cases, when hypoglycemia lasts long time and deepens [2].

Initially, clinical examination shows coma with agitation, tendon hyperreflexia and pyramidal irritation, after, as hypoglycemia lasts, coma tends to become calm and areflexic.

Neurological examination showed abolition of tendon reflexes and bilateral Babinski sign.

Brain lesions caused by hypoglycemia are unusual especially in young diabetics; however, rare cases have been reported of bilateral lesions in the basal ganglia, cerebral cortex and hippocampus.

Brain MRI is the mainstay of imaging in evaluation of neurological lesions caused by hypoglycemic coma. Diffusion weighted image is a special MRI technique that explore the movement of water through extracellular and intracellular spaces [2].

The anomalies are mostly located in the parietal, occipital and temporal lobes, the hippocampus and cerebral tonsils. Also, Cerebral cortex, subcortical and deep white matter can be affected. In addition, the basal ganglia, splenium of the corpus callosum may show lesions but classically thalami are spared [3].

MRI shows hyper-intensities on T2 weighted images and Flair associated with cortical edema and loss of grey-white matter differentiation in T1 weighted images. Often diffusion is reduced and apparent diffusion coefficient is restricted [4].

These lesions spare the cerebellum and brainstem. Evolution is marked by rapid cerebral atrophy in the areas of concern. The severity and duration of hypoglycemia are the main predictors of irreversible neuronal damage [5].

Brain CT scan is often normal or sometimes shows areas of hypo attenuating brain tissue.

In the newborn, severe hypoglycemia results in diffuse cortical and subcortical lesions located mostly in the parieto-occipital lobes.

Sequelae changes include cortical atrophy and cystic encephalomalacia, which may subsequently be responsible for occipital epilepsy. The reversibility of these lesions or hypersignals in MRI diffusion is possible and has been reported to be proportional to clinical improvement until patient regains consciousness [6].

The main differential diagnosis is ischemia, but the context of severe hypoglycemia makes the diagnosis.

Treatment consists of immediate administration of serum glucose 30 %. The prognosis is variable, depending on the extent of the lesions especially to basal nuclei.

Conclusion

The diffusion MRI is the mainstay imaging technique to set the diagnosis based on specific signs. Also, it is used for the follow-up to assess the response for treatment and evolution of cerebral lesions.

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