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Reversible splenium diffusion weighted MRI changes associated with hypoglycemia

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ABSTRACT

Hypoglycemia can manifest as a stroke. MRI diffusion-weighted imaging is the most useful technique in diagnosing early ischemic injury. We report two cases of transient MRI lesions of the splenium of the corpus callosum related to hypoglycemia. Clinicians must be aware of such cases to avoid misdiagnosis.

1. Introduction

Hypoglycemia can manifest with various neurologic deficits such as weakness, confusion, coma, and seizures, but also focal deficits such as hemiplegia, and these symptoms can be difficult to differentiate from stroke. MRI techniques and particularly diffusion-weighted imaging (DWI) are the most useful techniques in diagnosing early ischemic injury.

Reversible MRI splenial lesions have been associated with various disorders including seizures, antiepileptic drug toxicity and withdrawal, infectious encephalitis, high altitude cerebral edema, hemolytic and uremic syndrome (Doherty, Jayadev, Watson, Konchada, & Hallam, 2005; Sing, Gogol, Vyas, & Khandelwal, 2010). Rare reports incriminate metabolic disturbances such as hypernatremia, or hypoglycemia. We report two new cases of transient MRI lesion of the splenium of the corpus callosum related to hypoglycemia.

2. Case reports

2.1. Case 1

A 57-year-old female with a 5 year history of type 2 diabetes treated with metformin 3000 mg/day and glimepiride 4 mg / day, woke up in the night to go to the bathroom, fell and was found by her husband presenting with mutism, but there was no loss of consciousness. She understood what was said around her but was unable to say a word.

Upon arrival to the Emergency Room one and a half hours later, there was a partial regression of the language disorders, dysarthria without other neurological deficits.

We noted a blood pressure of 134/74, heart rate 95, oxygen saturation 100%, temperature 37° 5C, capillary glycemia at 5:00 am was 2.2 mmol/l; intravenous glucose was injected immediately. Blood glucose level at 5:07 am was 2 mmol/l. Biology also noted macrocytic anemia with hemoglobin at 10.7 g/dl, mean corpuscular volume 114 μm^3.Vitamin B12 assay performed after going home found a deficit—163 pg/ml (N: 211–911) probably secondary to treatment with metformin. Other blood laboratory tests including complete blood count, electrolytes, chemistries, urea, and creatinine were all within normal range.

Blood glucose increased to 7.6 mmol/l and her state improved to normal over the following few hours. There was no cognitive sequela.

The MRI performed 4 h after the onset of symptoms found on diffusion-weighted sequences a hypersignal in the splenium of the corpus callosum, with apparent diffusion coefficient (ADC) reduction at this level; this lesion was not visible in FLAIR. There were no bleeding on T2*-weighted sequences, and no stenosis of major cerebral arteries on TOF angiography. Follow-up MRI performed four weeks later showed complete resolution of the hyperintense signal observed in initial diffusion. FLAIR (Fluid attenuated inversion recovery) weighted sequences showed no visible lesion in the splenium (cf Fig. 1a, b, c, d).

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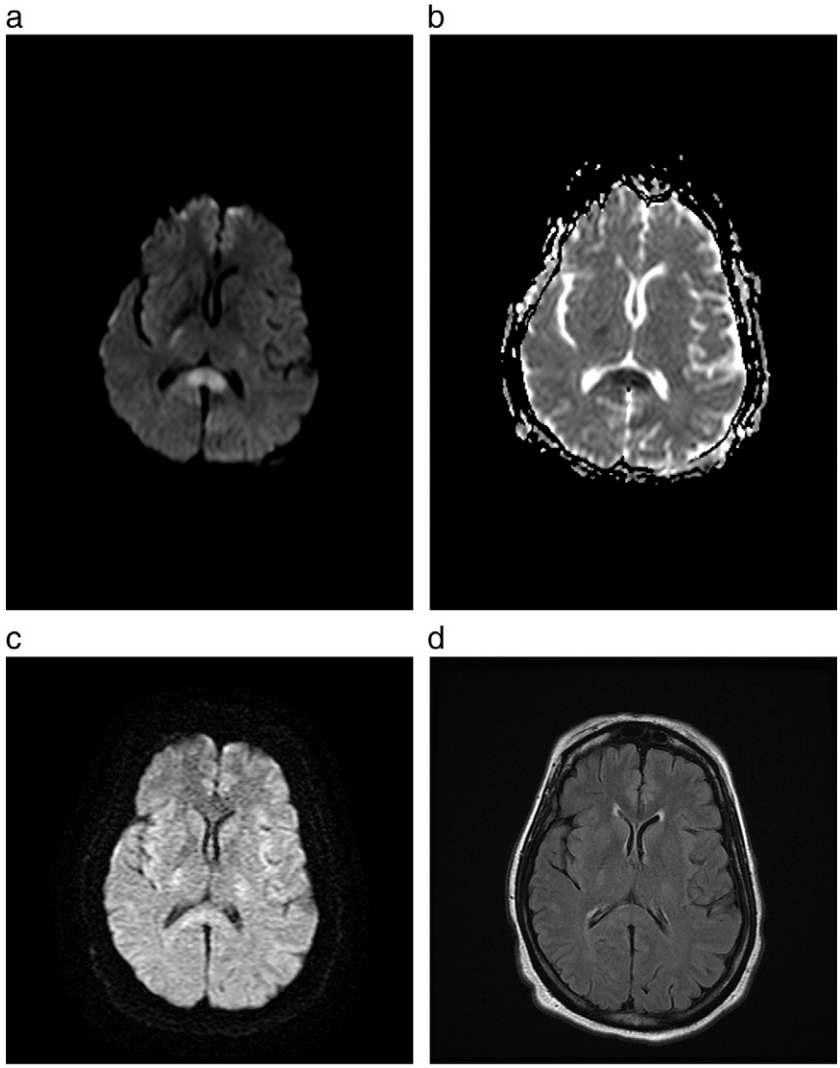

Fig. 1. (case1): a. First DWI image showing hypersignal in the splenium of the corpus callosum. b. ADC map shows reduction in the central splenium. c. Follow-up DWI MRI shows complete resolution of the splenial lesion. d. Follow-up FLAIR image also shows no residual lesion.

2.2. Case 2

A woman, 79 years of age, autonomous, with a history of type 2 diabetes, dyslipidemia, and hypertension treated with metformin 2000 mg/day, glimepiride 6 mg/day, pravastatin 40 mg/day, and valsartan 80 mg/day, was taken to the emergency room in a comatose state, disorder of consciousness lasting for 2 h with on admission a Glasgow Coma Scale score at 9 (E4 V1 M4). Initial blood glucose was 3 mmol/l and intravenous glucose was immediately undertaken. A gradual improvement in the state of consciousness was then observed over 2 days. The patient returned to her former neurological status.

Other laboratory tests were unremarkable apart from a vitamin B12 deficiency of 113 pg/ml (189–883) probably secondary to metformin without anemia or macrocytosis.

MRI performed after 24 h of onset of symptoms found on diffusion-weighted sequences a hyperintense signal in the splenium of the corpus callosum, not visible on FLAIR weighted sequences, combined with a low signal on ADC mapping. MR angiography was normal.

The MRI scan that was performed 6 weeks later showed complete resolution of splenial high signal observed on initial diffusion. FLAIR-weighted MR images showed no residual lesion in the splenium; there was no contrast enhancement after injection of gadolinium (cf Fig. 2: e, f, g).

3. Discussion

Transient splenial lesions have been reported associated with variable conditions including seizures, antiepileptic drug toxicity and withdrawal, infectious encephalitis, high altitude cerebral edema, alcohol abuse, hemolytic and uremic syndrome (Doherty et al., 2005; Sing et al., 2010) and metabolic disturbances such as hypernatremia (Doherty et al., 2005); to our knowledge, only a few cases have been reported following hypoglycemia (Böttcher et al., 2005; Doherty et al., 2005; Kim, Choi, Koh, & Lee, 2007; Lo, Tan, Umapathi, & Lim, 2006; Taguchi, Kamiyama, Kubo, & Horie, 2011; Terakawa et al., 2007). In those cases hypoglycemia was generally marked. In our two cases, hypoglycemia (mild in one case, severe in the other) appears to be the most likely cause of the reversible splenial lesion with transient encephalopathy because the clinical disturbance completely disappeared after correction of hypoglycemia. MRI abnormalities also disappeared on follow-up MRI although these were delayed and no other cause of transient splenial lesion was found. Several mechanisms have been invoked to explain reversible MRI splenial abnormalities: perturbed cellular fluid mechanisms, intramyelinic edema, and inflammatory infiltrates (Doherty et al., 2005; Kim et al., 2007; Sing et al., 2010). MRI DWI high signal with ADC reduction is usually representative of cytotoxic edema, as seen in ischemia. Patients with diabetes may present atherosclerosis that may