

Reversible Diffusion-Weighted Imaging Changes in the Splenium of the Corpus Callosum and Internal Capsule Associated With Hypoglycemia

—Case Report—

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Abstract

A 63-year-old man presented with hypoglycemia-induced hemiparesis manifesting as diffusion-weighted magnetic resonance (MR) imaging changes in the splenium of the corpus callosum and internal capsule which disappeared after glucose administration. Clinicians should be aware that hypoglycemia can cause reversible splenium abnormalities on MR imaging, although the underlying mechanism still remains unclear, as this may be helpful in the differential diagnosis of hypoglycemia-induced hemiparesis and stroke.

Key words: hypoglycemia, hypoglycemia-induced hemiparesis, diffusion-weighted magnetic resonance imaging, splenium

Introduction

Hypoglycemia is known to manifest as hemiparesis mimicking stroke. Diffusion-weighted magnetic resonance (MR) imaging is a useful method to detect early brain injury in the acute stage of ischemia, but is less well known in hypoglycemia-induced hemiparesis.^{1,2,5,13,17} We report a case of hypoglycemia manifesting as hemiparesis in which diffusion-weighted MR imaging demonstrated completely reversible signal changes in both the splenium of the corpus callosum and the posterior limb of the left internal capsule in the course of recovery.

Case Report

A 63-year-old male with a history of diabetes mellitus, which had been controlled by subcutaneous insulin (22 U daily), noticed right hemiparesis on waking in the morning and was transferred by ambulance to the emergency department. He had no vascular risk factors except for diabetes mellitus. On admission, physical examination found hyperten-

sion (blood pressure 182/70 mmHg), but no other abnormal factors. Neurological examination revealed slight clouding of consciousness and moderate right hemiparesis (manual muscle testing 2/5). Diffusion-weighted MR imaging obtained immediately after admission revealed hyperintense areas in the left internal capsule and the splenium of the corpus callosum with reduced apparent diffusion coefficient (ADC) (Fig. 1). Fluid-attenuated inversion recovery and T₂-weighted MR imaging found no abnormalities. MR angiography excluded arterial occlusion and stenosis. Laboratory examinations after MR imaging were within normal limits except for much lower plasma glucose level of 24 mg/dl.

After intravenous administration of glucose, the patient showed drastic recovery of the right hemiparesis and disturbed consciousness, and his neurological condition had returned to normal by the following day. Follow-up diffusion-weighted MR imaging obtained approximately 28 hours after the first MR imaging demonstrated normalization of both signal intensity and ADC abnormalities (Fig. 2).

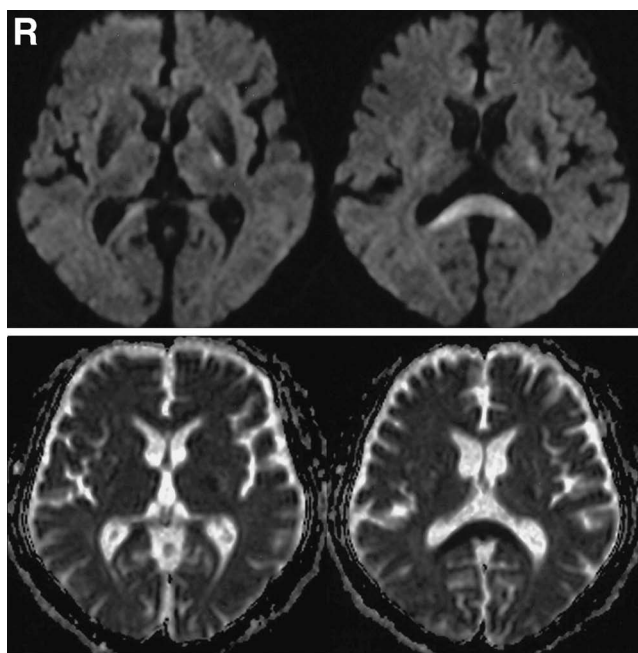


Fig. 1 Diffusion-weighted magnetic resonance images (upper row) on admission showing hyperintense areas in the splenium of the corpus callosum and the left internal capsule, and apparent diffusion coefficient (ADC) maps (lower row) showing corresponding reduced ADC.

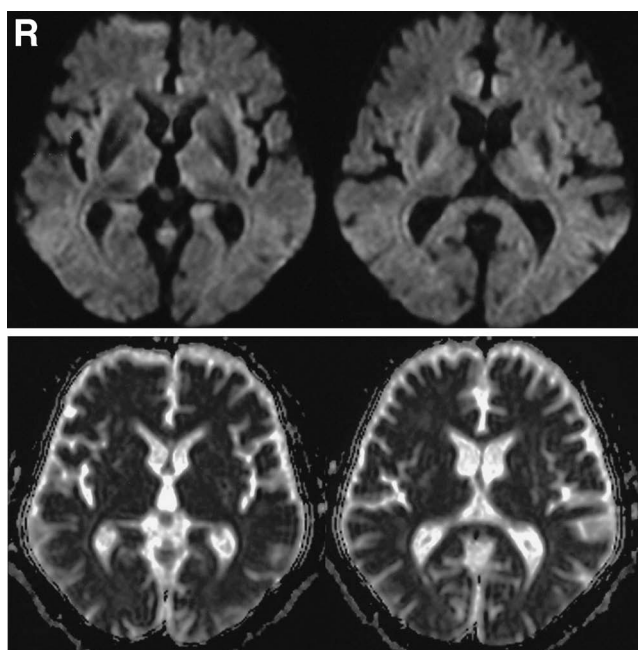


Fig. 2 Follow-up diffusion-weighted magnetic resonance images (upper row) and apparent diffusion coefficient maps (lower row) obtained 28 hours later showing normalization of abnormalities.

Discussion

Diffusion-weighted MR imaging characterized the present case of hypoglycemia as areas of abnormal signal intensity in the splenium of the corpus callosum and the posterior limb of the internal capsule which did not conform to the vascular distributions. Further, repeat diffusion-weighted MR imaging showed these areas had completely disappeared in parallel with the neurological recovery associated with glucose administration. Therefore, we attributed the clinical symptoms and MR imaging findings to hypoglycemia.

MR imaging has previously detected lesions associated with hypoglycemia in the cerebral cortex, basal ganglia, and hippocampus,^{4,7,8,10,15,18)} mostly with poor outcome resulting in death or persistent vegetative state. These regions are known to be the most vulnerable to hypoglycemic insult. Animal studies have suggested that this selective vulnerability involves differences in the regional brain glucose content,¹⁶⁾ glucose influx,¹⁴⁾ amino acid distribution,³⁾ or selective inhibition of cerebral protein synthesis.¹¹⁾ An animal study of insulin-induced hypoglycemia showed diffuse reduction of ADC after the onset of cerebral isoelectricity in rat brains and normalization of most abnormalities by 10 minutes after glucose infusion. Asymmetrical changes in ADC were also detected in 40% of the experimental animals before the onset of generalized isoelectricity.⁹⁾ This reversibility and asymmetry may help to explain the clinical presentation in our case. However, these animal studies cannot explain the selectively disturbance of the splenium and internal capsule in transient hypoglycemia-induced hemiparesis, because these regions are unlikely to have low regional glucose content and glucose influx or accelerated regional glucose metabolism compared with other surrounding areas such as the cerebral cortex or basal ganglia.

The splenium may be another area of selective vulnerability to hypoglycemia,¹²⁾ although little is known about the involvement of the splenium in the hypoglycemic condition.^{2,6,13,15)} Evaluation of nine patients with midline symmetric MR imaging signal changes in the splenium found various related causes, and most diffusion-weighted MR imaging intensity and ADC abnormalities were reversible.⁶⁾ Seven of the nine patients had intensity changes in the posterior limbs of the internal capsule as well as the splenium, as in the present case. These findings imply that splenium abnormality is not specific but is indicative of hypoglycemia, and that a combination of splenium and internal capsule abnormalities as seen in our case are not incidental. We speculate

that the underlying pathogenic process which induced the reversible signal changes in the splenium, and possibly also in the internal capsule, is shared with other pathological conditions, and is different to the mechanism of injury to the cerebral cortex, basal ganglia, and hippocampus observed in prolonged hypoglycemic coma. Identification of this common mechanism may help to understand how hypoglycemia affects and injures the human brain.

Clinicians should be aware of the reversible diffusion-weighted MR imaging signal changes in the splenium of the corpus callosum and internal capsule associated with transient hypoglycemia-induced hemiparesis, which may mimic stroke clinically. Such reversible abnormal signals in the splenium are suggestive, although not specific, of hypoglycemia and are helpful in the differential diagnosis from stroke.

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