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Case Report

Radiology Section

Magnetic Resonance Imaging Characteristics of Persistent Vegetative State Due to Prolonged Hypoglycemia

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ABSTRACT

Hypoglycemia is the sudden decrease in serum glucose level <50mg/dL. Neurological manifestations complicating profound and prolonged hypoglycemia range from reversible focal deficits and transient encephalopathy to irreversible coma. Here, we report magnetic resonance imaging characteristics of a patient with prolonged hypoglylicemia.

A 47-year-old woman with a history of insulin dependent diabetes mellitus has been brought to the emergency room by her relatives. She used mistakenly overdose insulin injection and probably stayed 11 hours with low level blood glucose. The initial blood sugar level was 39.6 mg/dL at the emergency department visit, which was recovered urgently by 50% dextrose. MR imaging revealed high intensities at the bilateral posterior parietal cortices, corona radiata and hippocampus, but not in the basal ganglia. Seventy-two hour after admission, confluent lesions in the posterior parietal, temporal, frontal cortices and splenium of corpus callosum were more prominent on DWI and FLAIR, and did not match typical arterial territories. None of the lesions were enhanced on contrast-enhanced T1-weighted images.

The prognosis or neurologic sequelae of hypoglycemic encephalopathy may depend on the severity and duration of hypoglycemia and persistent, diffuse involvement of the cerebral cortex, basal ganglia, or hippocampus on the following MR imaging. MR imaging findings in hypoglycemic vegetative state can be helpful in the differential diagnosis distinguishing from other neurologic conditions.

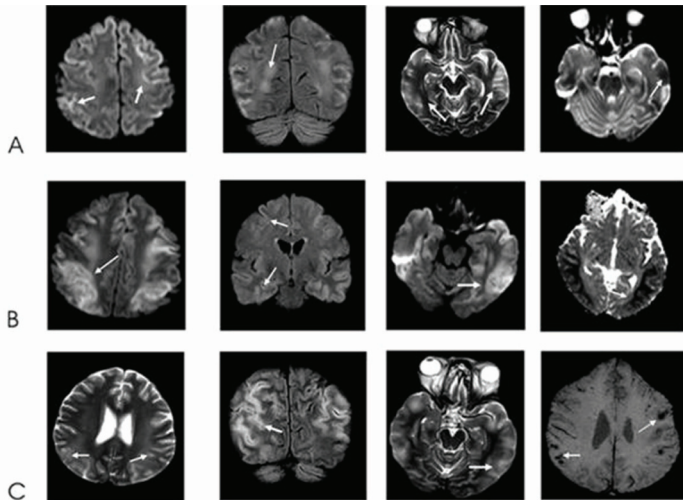
Keywords: Hypoglycemia, Hypoglycemic brain injury, MR imaging, Susceptibility weighted imaging

CASE REPORT

A 47-year-old woman with a history of insulin dependent diabetes mellitus has been brought to the emergency room by her relatives. Her family noticed that she had been unconscious when they found her in the bedroom. She used mistakenly overdose insulin injection and probably stayed 11 h with low level blood glucose. The initial blood sugar level was 39.6 mg/dL at the emergency department visit, which was recovered urgently by 50% dextrose. Following rapid normalization of blood glucose to 130mg/dL, there was no clinical improvement. There was no evidence of stroke, seizure, drug toxicity, viral infection, hypotension, hypothermia, acidosis, and cardiac, renal, hepatic disease. Vital parameters were normal. She remained comatose during the hospitalization, exhibiting no response to pain stimulus (Glasgow Coma Scale score was 3). The initial neurologic symptoms and mental status of the patient did not improve, and persistent vegetative state was still present after three months of admission. She underwent MR imaging within 12 h of the time of hospital admission. MR imaging was performed using a 3-Tesla MR imaging system (Magnetom Vision, Siemens Medical systems, Erlangen, Germany), included T1- and T2- weighted, fluid attenuated inversion recovery (FLAIR), diffusion-weighted (DWI) with apparent diffusion coefficient (ADC), susceptibility weighted imaging (SWI) sequences. DWI revealed high intensities (4200/95, b-value 1000 seconds/mm2) at the bilateral posterior parietal cortex, corona radiata and hippocampus, but not in the basal ganglia. Seventy-two hour after admission, confluent lesions in the posterior parietal, temporal, frontal cortex and splenium of corpus callosum were more prominent on DWI (decreased ADC of the splenium of the corpus callosum [350x10-3 mm^2/s] compared with the genu [750x10-3 mm^2/s]) and FLAIR, and did not match typical arterial territories. None of the lesions were enhanced on contrast-enhanced T1-weighted images. After three weeks of admission, SWI showed linear and patchy noncontiguous microhemorrhage in the subcortical regions, while DWI findings seen in the second MRI regressed [Table/Fig-1].

DISCUSSION

Neuropathologic studies have demonstrated that the cerebral cortex, hippocampus, and basal ganglia are commonly affected sites in severe hypoglycemia. However, cerebellum and the brain stem are usually spared, while the occipital cortex, dorsofrontal cortex, and hippocampus may be more resistant to prolonged hypoglycemia [1,2]. MR imaging of hypoglycemic brain injury reported that DWI is useful in evaluating severe hypoglycemic encephalopathy. Reversible diffusion restriction has been found in most cases [3]. The reversibility of the lesions suggested cytotoxic oedema at the cerebral cortex, hippocampus, splenium, and white matter. Severe



[Table/Fig-1]: A. First MRI. DWI on admission shows bilaterally asymmetric confluent hyperintense lesions in the frontal, parietal, insular, temporal, occipital cortices, and corona radiata with reduced ADC values (arrows). These lesions are bilateral and symmetric B. Second MRI. DWI obtained 3 days after symptom onset shows increased signal intensity frontal, parietal, insular, temporal cortices with with prominent reduced ADC values. C. Third MRI. DWI 22 days after glucose infusion showing regression of the hyperintensity lesions with normalization of ADC values. SWI revealed patchy noncontiguous microhemorrhage in the subcortical areas

hypoglycemia induces some kinds of neurochemical changes. First, neuroactive amino acids (aspartate) release into the extracellular space and causes selective neuronal necrosis, especially in the cerebral cortex, caudoputamen, and hippocampus. Second, excitotoxic oedema appear due to increased extracellular glutamate. In contrast to cytotoxic oedema, excitotoxic oedema does not imply neuronal damage. Because glutamate induces oedema of glial cells and myelinic sheaths might protect axons from intracellular oedema and irreversible damage [4]. The small pathcy microhemorrhages on SWI is probably a "nonvascular" lesion, the hypoglycemic lesion is aggravated by acidosis and transformed into infarction accompanied by perivascular erythrocytes in the subcortical regions. The MR imaging findings of our patient with persistent vegetative state were in accord with the proposed mechanisms.

CONCLUSION

The degree of neurologic sequelae due to hypoglycemic encephalopathy may depend on the severity and duration of hypoglycemia and also persistent, diffuse involvement of the cerebral cortex, basal ganglia, or hippocampus on the following MR imaging. MR imaging findings in hypoglycemic vegetative state can be helpful in the differential diagnosis distinguishing from other neurologic conditions.

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