

The radiological findings of hypoglycemic encephalopathy

A case report with high b value DWI analysis

Shan Ren, MD^a, Zhigang Chen, MD^a, Ming Liu, MD^b, Zhiquan Wang, MD^{b,*}

Abstract

Rationale: Hypoglycemic encephalopathy is a metabolic encephalopathy. Clinical risk is mixed with acute cerebrovascular disease, so it is critical to identify and make the correct diagnosis of the disease as early as possible.

Patient concerns: Here, we report a case of a 51-year-old male patient with hypoglycemic encephalopathy, who presented confusion and unconsciousness for 1 day.

Diagnoses: In addition to blood-related indicators and medical histories, magnetic resonance imaging (MRI), especially diffusion-weighted imaging (DWI), can be valuable to the diagnosis of hypoglycemic encephalopathy, which showed diffuse high-signal intensity in the cerebral cortex, and also the hippocampus, head of the caudate nucleus, the lentiform nucleus, and corpus callosum.

Interventions: Intravenous glucose injection and drip was performed repeatedly. The blood glucose levels were gradually corrected, and the resulting blood glucose was 6.5 mmol/L.

Outcomes: The prognosis depends on the degree of hypoglycemia, duration, and condition of the organism. Due to the long duration of hypoglycemia, unfortunately, the patient died.

Lessons: It is critical to diagnose hypoglycemic encephalopathy as early as possible. MRI reveals diffuse abnormal intensity in the cortex and basal ganglia region. DWI using high b values provides important information for diagnosis.

Abbreviations: CJD = Creutzfeldt-Jakob disease, CT = computed tomography, DWI = diffusion-weighted imaging, FLAIR = fluid attenuation inversion recovery, MRA = magnetic resonance angiography, MRI = magnetic resonance imaging, T = temperature, T1WI = T1-weighted image, T2WI = T2-weighted image, WBC = white blood cell.

Keywords: diffusion-weighted imaging, hypoglycemic encephalopathy, magnetic resonance imaging

1. Introduction

Hypoglycemic encephalopathy is a metabolic encephalopathy due to extremely low blood glucose. Such patients often suffer from the disease suddenly, which is initially characterized by multiple symptoms such as lags in response, confusion, mental and behavior disorders, and adverse physical activity. Its attack form is varied; it can last a period of time, or symptoms may be alleviated after eating. If the treatment is not administered in time,

the disease will further develop into drowsiness, lethargy, and even a coma. Clinical risk is mixed with acute cerebrovascular disease, so it is necessary to identify and make the correct diagnosis as early as possible. In addition to the blood index and past medical history, the imaging examination can also provide important information. Here, we reported a case of hypoglycemic encephalopathy including the clinical data and imaging information, which provided new evidence of the disease diagnosis.

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^a Department of Neurology, ^b Department of Radiology, Dongfang Hospital, Beijing University of Chinese Medicine, Beijing, China.

* Correspondence: Zhiquan Wang, Department of Radiology, Dongfang Hospital, Beijing University of Chinese Medicine, Fengtai District, Beijing 100078, China (e-mail: wangzhiquan@126.com).

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2. Case report

A 51-year-old male patient was admitted to emergency room on June 17, 2014, because of confusion and unconsciousness for 1 day. The patient had been diabetic for the past 9 years. However, he did not comply with systematic drug usage and blood glucose monitoring. The patient's blood glucose was too low to measure. Considering hypoglycemic coma, 20 mL of 50% glucose saline was intravenously injected. However, the patient did not show a significant improvement. In the emergency room of our hospital, laboratory tests were as follows: blood glucose 4.7 mmol/L; blood gas analysis pH 7.30; PaO₂ 89.2 mm Hg; PaCO₂ 56.5 mm Hg; K 2.70 mmol/L; white blood cell (WBC) count 15.54 × 10⁹/L; percentage of neutrophils 85.5%; and hemoglobin content 161 g/L. Furthermore, chest x-ray showed blurring of 2 lung textures. No specific abnormality was found on cerebral computed tomography (CT). Glasgow Coma Scale scores were 3 points. Considering hypoglycemic coma and aspiration pneumonia, intravenous glucose injection was repeatedly performed. In

addition, anti-infection, eliminating phlegm, stomach mucosa protection, potassium supplement, and other supportive treatments were provided. The next day, the patient was still in a coma and presented slow breathing and throat phlegm. An urgent blood gas and biochemical check showed the following: blood glucose: 1.03 mmol/L; pH 7.504; PaCO₂ 33.7 mm Hg; PaO₂ 142 mm Hg; SO₂ 99.7%; and K 3.29 mmol/L. Then, the patient received endotracheal intubation, auxiliary breathing machine assistance, midazolam intravenous pumping for calmness, and gastrointestinal decompression. Intravenous glucose injection and drip were performed repeatedly. The glucose blood levels were gradually corrected, and the resulting blood glucose level was 6.5 mmol/L. The patient was then moved into the neurology department for further treatment. Physical examination showed the following: heart rate (HR): 92 times/min, temperature (T): 38.0°C. No dry or moist rales were found via 2 lung auscultation.

Rhythm of the heart was regular. There was no pathological murmur in the cardiac auscultation. The abdomen was soft, and the muscles were not tense. The liver and spleen were not palpable. There was no edema in both legs. Neurological examination showed the following: bilateral pupils were equal and round, diameter of 3 mm; there was light reflex; limb muscle strength was level 0; muscle tension and physiological reflex were normal; and bilateral Babinski sign and Chaddock sign showed positive performances. Cerebral magnetic resonance imaging (MRI) examination showed the following: diffusion-weighted imaging (DWI) showed diffuse high-signal intensity in the cortex of the bilateral frontal, temporal, parietal, and occipital lobe, and also the hippocampus, head of the caudate nucleus, the lentiform nucleus, and corpus callosum (b value including 1000 and 2000 s/mm²) (Fig. 1A and B); we also found the slightly low signal intensity on T1-weighted image (T1WI) (Fig. 1C) and slightly

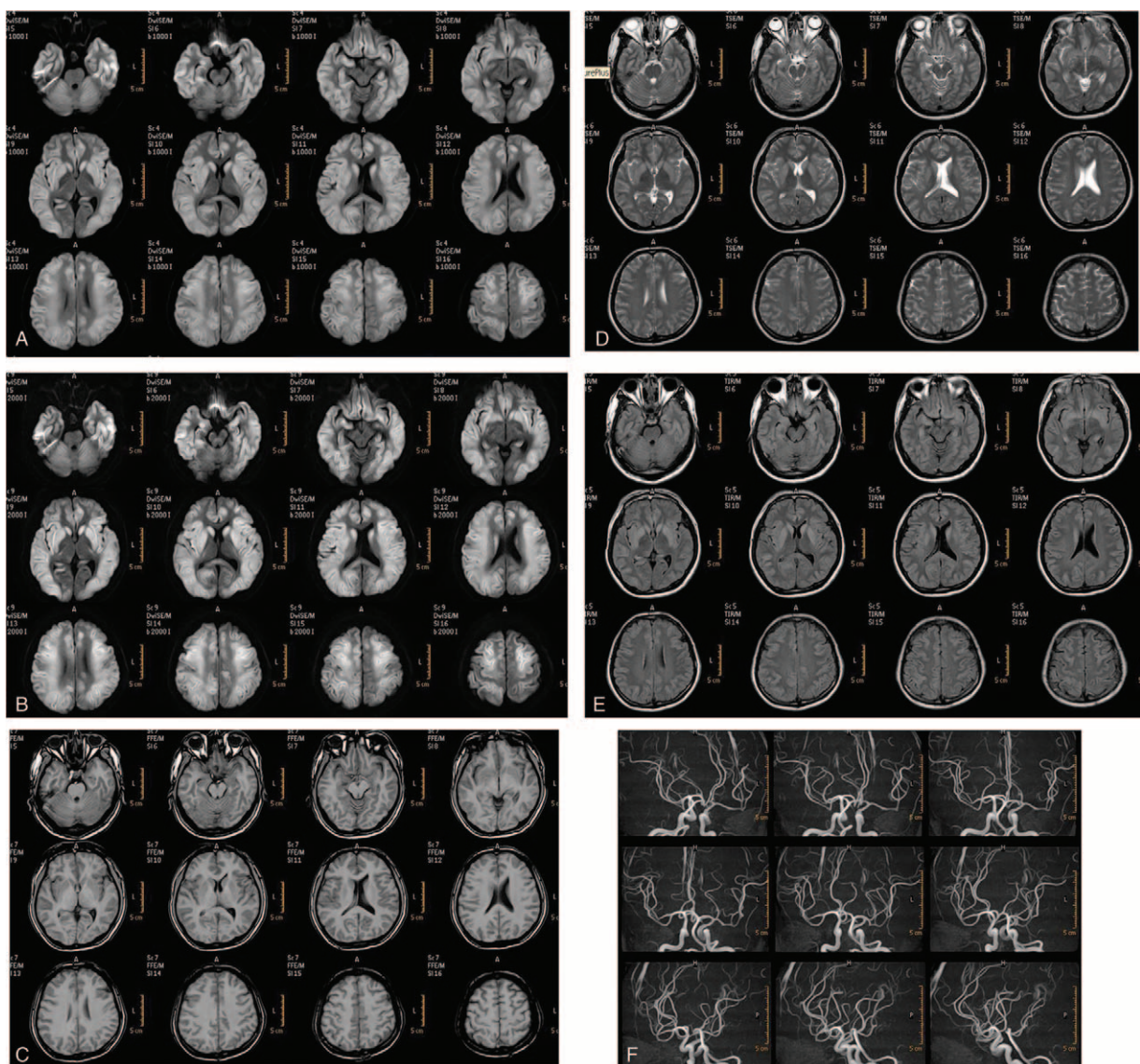


Figure 1. Diffusion-weighted imaging (DWI) showed diffuse high signal intensity in the cortex of the bilateral frontal, temporal, parietal, and occipital lobe, and also the hippocampus, head of the caudate nucleus, the lentiform nucleus, and corpus callosum (b value = 1000 s/mm²) (A). Lesions were more clearly shown by higher b values (b value = 2000 s/mm²) (B). Lesions showed the slightly low signal intensity on T1WI (C). Lesions showed the slightly high signal intensity on T2WI (D) and FLAIR (E). No obvious blood vessel stenosis was found on MRA (F). FLAIR = fluid attenuation inversion recovery, MRA = magnetic resonance angiography, MRI = magnetic resonance imaging, T1WI = T1-weighted image, T2WI = T2-weighted image.

high signal intensity on T2-weighted image (T2WI) (Fig. 1D) and fluid attenuation inversion recovery (FLAIR) in the same regions (Fig. 1E). No obvious blood vessel stenosis was found on magnetic resonance angiography (MRA) image (Fig. 1F). After our positive treatment, the patient's blood glucose was corrected to above 6 mmol/L. However, because the hypoglycemia lasted a long time, it resulted in irreversible brain tissue damage, and the patient ultimately died.

3. Discussion

When blood glucose is lower than 2.9 mmol/L, it causes brain symptoms such as hypoglycemic encephalopathy. The clinical manifestations of hypoglycemia are complex and associated with the extent, speed, duration, and responsiveness of blood glucose levels. The prognosis depends on the degree of hypoglycemia, its duration, and the condition of the organism.^[1]

Mild hypoglycemia is characterized by recurrent paroxysmal sweating, pale complexion, and syncope, whereas moderate hypoglycemia patients present hunger, weak, easy excitement, sweating, heart palpitations, sympathetic excitement symptoms such as anxiety, and tremor. If symptoms persist, lethargy and coma can occur. With the development of the disease, the patients with hypoglycemic encephalopathy appeared to have focal neurologic symptoms including hemiplegia, hemianopsia, aphasia, and the onset of convulsion. Patients with hypoglycemia that lasts too long will present hyperactivity, sucking and or strong reflex, increased muscle tone, myoclonia, myotonia, and even stiffness and brain cortex.

The oxidation of glucose is the main source of energy for the nervous system. The energy released after oxidation of glucose is stored in adenosine triphosphate and phosphocreatine. When the nervous system needs energy, it will be released by adenosine triphosphate. Once low blood glucose levels are shown to reduce the amount of energy in the brain, it can cause the brain's energy metabolic disorders, which can lead to severe brain dysfunction. The main pathological changes of hypoglycemic encephalopathy are extensive denaturation and necrosis of the neurons due to lack of energy, accompanied by a large number of infiltrating glial cells.

In the nervous system, the cerebral cortex, hippocampus, cerebellum, caudate nucleus, and pallidum parts of the brain present higher energy consumption, followed by the thalamus, hypothalamus, brain stem, and cranial nerve nuclei. As a result, the cerebral cortex, hippocampus, cerebellum, caudate nucleus, and pallidum are extremely sensitive to low blood glucose; when blood glucose level is low, these areas are affected and significant damage is caused in the first place. The lesion regions of the hypoglycemic encephalopathy are highly selective.^[2]

Cerebral MRI, especially DWI, can provide valuable information and play an important role for the diagnosis of hypoglycemic encephalopathy.^[3] In DWI, the applied diffusion-sensitive gradient field parameter is called b value. The b value has a large effect on DWI, which is an important parameter in the DWI. The clinical b value is often 1000 s/mm².^[4,5] The lesions were

found on the T1WI, T2WI, and FLAIR of this patient's MRI, but the lesions were not obvious. On DWI, the lesions can be clearly shown and were more easily discernible after the b value was properly raised, which was valuable for the early diagnosis of the disease.

It is worth noting that such diseases are easily misdiagnosed as toxic and metabolic diseases, cerebrovascular disease, degenerative conditions, some inflammatory and neoplasms, which should be identified in several ways.^[3,6,7] First, clinical history is very important. There was no objective evidence such as hypotension, hypertension, acidosis, drug intoxication, infection, and so on. However, examination found the extremely decreased blood glucose. Second, abnormal MR images did not conform to vascular distributions, and MRA showed no abnormalities, which can exclude the cerebrovascular disease. In addition, abnormal MR images were global in appearance involving the diffuse cortex, which indicated Creutzfeldt-Jakob disease (CJD) apart from hypoglycemia, which helped narrow the differential diagnosis. MR images reveal bilaterally symmetric areas and restricted diffusion in the medial thalamus, indicating "hockey stick sign," which can be used for diagnosis of CJD. Finally, cerebral infections and tumors may show perilesional edema or infiltration outside the basal ganglia or multifocal disease elsewhere in the brain and meninges. Taken together, combined with the clinical history and the key signs, MRI can help to differentiate these diseases.

4. Conclusions

It is critical to diagnose hypoglycemic encephalopathy as early as possible. MRI reveals diffuse abnormal intensity in the cortex and basal ganglia region. DWI using high b values provides important information for diagnosis.

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References

- [1] WHO. Diabetes mellitus: report of a study group. World Health Organ Tech Rep 1985;727:1.
- [2] Report and recommendations of San Antonio Conference on diabetic neuropathy. *Ann Neurol* 1988;24:99-104.
- [3] Aoki T, Sato T, Hasegawa K, et al. Reversible hyperintensity lesion on diffusion-weighted MRI in hypoglycemic coma. *Neurology* 2004;63:392-3.
- [4] Abe O, Aoki S, Shirouzu I, et al. MR imaging of ischemic penumbra. *Eur J Radiol* 2003;46:67-8.
- [5] Lee B-W, Jin ES, Hwang H-S, et al. A case of hypoglycemic brain injuries with cortical laminar necrosis. *J Korean Med Sci* 2010;25:961-5.
- [6] Kao SL, Chan CL, Tan B, et al. An unusual outbreak of hypoglycemia. *N Engl J Med* 2009;360:734-6.
- [7] Lim CC, Gan R, Chan CL, et al. Severe hypoglycemia associated with an illegal sexual enhancement product adulterated with glibenclamide: MR imaging findings. *Radiology* 2009;250:193-201.