Visual Vignette

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Case Presentation: A 42-year-old woman with type 1 diabetes mellitus of 9 years' duration with fluctuating glycemic control was brought to our institution for medical assistance because of a history of unattended unconscious state lasting for about 15 hours overnight. She had no history of preceding autonomic symptoms, febrile illness, seizures, insulin overdose, poisoning, or head trauma. On examination, the patient was normotensive, normothermic, and comatose with no focal neurologic deficits. On admission, the plasma glucose level was 26 mg/dL, and urine ketones were absent. Renal and hepatic functions were normal. Arterial blood gas analysis showed normal results. Cerebrospinal fluid examination revealed normal findings. Electroencephalographic features (diffuse theta waves with occasional frontal spikes) were suggestive of metabolic encephalopathy. The serum cortisol value during hypoglycemia was 26.2 μ/dL (reference range, >18). She remained in a vegetative state despite restoration of euglycemia during the next 5 days. Magnetic resonance imaging of the brain without contrast enhancement, performed about 24 hours after the onset of hypoglycemia, showed hyperintensities in the T2 and fluid-attenuated inversion recovery sequences bilaterally in the caudate nucleus, putamen, and medial temporal lobes and diffuse cortical atrophy (Fig. 1). A few small focal hyperintensities were noted in the periventricular white matter. The thalamus and cerebellum were characteristically spared (Fig. 2). **What is the diagnosis?**

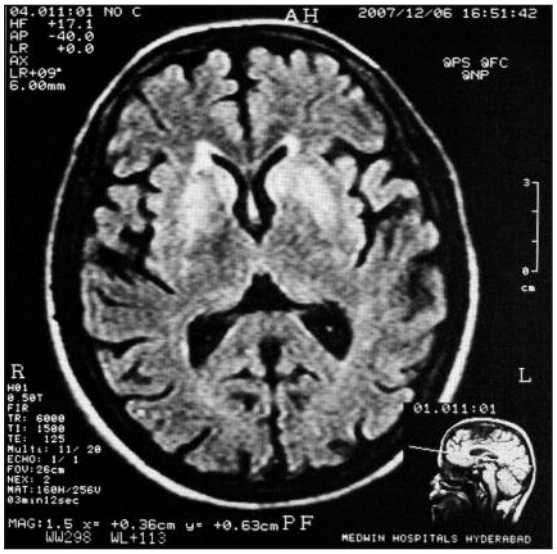

Fig. 1

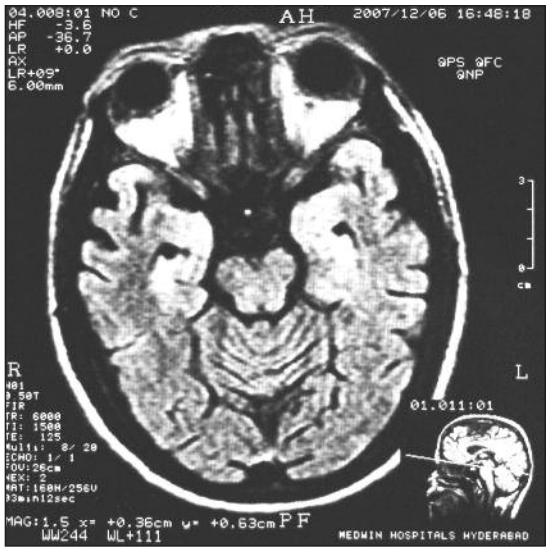

Fig. 2

Answer: The clinical profile documented prolonged hypoglycemia with the exclusion of hypoxemic-ischemic and encephalitic insult, which suggests the diagnosis of hypoglycemic brain injury. Blunting of the glucagon response, failure of autonomic regulation, and suboptimal counterregulatory hormone response possibly contributed to severe prolonged neuroglycopenia. She demonstrated no neurologic recovery, progressively deteriorated with nosocomial pneumonia and sepsis, and succumbed to her illness after 2 weeks of hospitalization.

The selective vulnerability of the basal ganglia, the hippocampus (medial temporal lobe), and the cerebral cortex to hypoglycemia has been documented on magnetic resonance images of the brain in a few earlier reports in the literature (1). A variable combination of selective neuronal death, cellular degeneration, astrocytic glial cell proliferation, and lipid accumulation is suspected to lead to the characteristic neuroimaging features (2). The thalamus and cerebellar cortex are invariably involved in hypoxemic-ischemic brain injury but spared in hypoglycemic brain damage.

REFERENCES

1. **Fujioka M, Okuchi K, Hiramatsu KI, Sakaki T, Sakaguchi S, Ishii Y.** Specific changes in human brain after hypoglycemic injury. *Stroke*. 1997;28:584-587.
2. **Auer RN, Hugh J, Cosgrove E, Curry B.** Neuropathological findings in three cases of profound hypoglycemia. *Clin Neuropathol*. 1989;8:63-68.