Hypoglycemia Presenting As Basilar Artery Thrombosis

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Background and Purpose: Following our treatment of a patient with hypoglycemia-induced brain stem symptoms, we examined the possible mechanisms for hypoglycemia presenting as basilar artery disease.

Case Description: We describe a patient who had progressive brain stem symptoms due to a diet-induced hypoglycemia initially diagnosed as basilar artery thrombosis. Symptoms ceased immediately after intravenous glucose.

Conclusions: Before invasive diagnostic and thrombolytic strategies are considered, hypoglycemia as a rare but important cause of acute brain stem dysfunction must be considered in patients suspected to suffer from basilar artery thrombosis. (Stroke 1992;23:112–113)

Hypoglycemia may cause diffuse brain dysfunction, most often with signs of adrenergic stimulation such as sweating, tachycardia, trembling, and hunger, which can progressively lead to coma, sometimes with seizures.1 In these cases, diagnosis is clear and does not present any difficulties.

Occasionally, however, focal neurological signs and symptoms such as hypoglycemic hemiplegia or movement disorders may masquerade as cerebrovascular disease without altered awareness or other common manifestations of hypoglycemia.2-7

We describe a patient with hypoglycemia-induced progressive brain stem dysfunction similar to basilar artery thrombosis.

Case Report

A 32-year-old woman with chronic diarrhea had not been eating regularly for approximately 5 days because of personal problems. After 5 days of near fasting, she caught a cold and went to bed early in the evening. When she awoke at midnight, she fell three times on her way to the bathroom because of dizziness and weakness in both legs. At 4 AM she noticed dysarthria and progressive paresis of both arms.

On initial examination in our hospital at 6:15 AM, she was awake and alert and had severe bulbar dysarthria. Examination revealed a gaze-directed vertical upward nystagmus with spontaneous nystagmus to the left, a central facial palsy on the right side, and tongue dyspraxia. Motor examination revealed a spastic tetraplegia with involuntary plantar flexion of both feet and flexion of both arms to arousal stimuli, a hyperreflexic state, and clonus and extensor plantar reflexes (Babinski sign) in both legs. Examination of sensation was difficult because the patient could answer only by moving her head, but it generally revealed no severe abnormalities.

During neurological examination, dysarthria progressed and severe dysphagia occurred, accompanied by impaired respiratory movements, breathlessness, and tachypnea, without alteration of consciousness. These symptoms were attributed to progressive basilar artery occlusion, and angiography with eventual thrombolysis was planned. Computed tomography (CT) scan was normal, as was the cerebrospinal fluid, except for a reduced glucose level of 26 mg%. Blood glucose was also reduced at 40 mg%, indicating hypoglycemia.

After intravenous administration of 10 ml 20% glucose, the patient recovered within 2 minutes, and neurological deficits completely disappeared. Two and one-half hours later, common hypoglycemic symptoms such as sweating, thirst, and confusional state occurred. An immediately drawn blood glucose level was 29 mg%. Again, the symptoms disappeared after intravenous glucose.

Because of the clear correlation between neurological symptoms and glucose injection, angiography was not done. However, magnetic resonance imaging and magnetic resonance angiography, as well as transcranial and extracranial Doppler sonography, brain stem evoked potentials, somatosensory evoked potentials, and magnetic evoked potentials were...
done to exclude any predisposing factors such as vascular malformation, infarction, nerve conduction failure, or tumor. Other mechanisms like hypotension-induced dysfunction of pontine nuclei or bilateral cerebral hemisphere dysfunction were ruled out by continuous blood pressure monitoring and electroencephalography. Further laboratory investigations were performed to search for an islet cell tumor of the pancreas, glycogen storage disease, or accidental or deliberate insulin or oral antidiabetics intake, without any pathological results. Insulin level, C-peptide level, glucose tolerance test, and glucagon test were normal. Central pontine myelinolysis was excluded by normal serum sodium and osmolality.

The patient made an excellent recovery and was discharged home.

Discussion

Since the discovery of insulin in 1921, factitious hypoglycemia is a common cause for neurological and psychiatric disturbances, and it most often occurs in diabetics taking insulin or oral hypoglycemic agents. Hypoglycemia is no diagnostic problem if symptoms appear in the typical fashion: sweating, hunger, anxiety, and trembling leading to confusion, drowsiness, and coma. Diagnosis may be difficult, however, if focal neurological symptoms mimic cerebrovascular ischemia events.

Hemiparesis caused by hypoglycemia was documented by Ravid more than 60 years ago and has been occasionally described since. Less frequently, other neurological deficits like paroxysmal choreoathetosis and movement disorders with dystonic posturing of all four limbs have been reported. All these patients had normal CT scans and were either insulin-dependent diabetics, took oral hypoglycemic agents, or, in one case, had an islet cell tumor.

In our patient, hypoglycemia was induced by starvation. Thus, in the absence of a history of diabetes and without the characteristic autonomic features, her condition masqueraded as basilar artery thrombosis with progressive spastic tetraplegia, conjugate gaze disturbances, supranuclear oculomotor dysfunction, dysarthria, dysphagia, and retained alertness indicative of patchy lesions within the brain stem and the cerebellum.

The mechanisms underlying hypoglycemia-induced focal neurological symptoms are unclear. Possible pathomechanisms include cerebral vasospasm, predisposing cerebrovascular disease, and "selective neuronal vulnerability" to intoxication and metabolic dysregulation. In our patient, a selective vulnerability of neuronal networks in the brain stem and cerebellum without underlying recognizable structural disease is a logical explanation. Analogous to the well-known selective sensitivity of the hippocampus and the basal ganglia to systemic hypoxia and intoxication, it is reasonable to assume that metabolic neuronal dysfunction such as transient hypoglycemia may also affect the pyramidal tract, the basal ganglia, and the brain stem.

Auer has reviewed the literature on hypoglycemic brain damage and draws the conclusion that a metabolically derived aspartate accumulates in cerebrospinal fluid and acts as an excitotoxin. This hypothesis is based on rat experiments in which neuronal necrosis appears in brain regions exposed to subarachnoid spaces. In our patient, this mechanism might be a possible explanation for the focal neurological deficits followed by hypoglycemia, which initially affected the autonomic system in the reticular formation, possibly resulting in an inhibitory effect on catecholamine secretion.

For practical reasons, we conclude that, although this case might represent a rare neurological manifestation of hypoglycemia, it can be taken as an illustrative example of the importance of assessing blood glucose levels in all patients with acute cerebrovascular events. This is particularly important for patients who, in the absence of vascular risk factors, are eligible for selection for recently propagated emergency therapeutic strategies such as intravenous thrombolytic therapy without prior angiographic proof of thrombosis.

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References


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