Hypoglycemia – Old foe with a new face – Masquerading as an acute stroke

Sir,

A 50-year-old man, known diabetic since 6 months on glibenclamide, presented to us in the emergency department with sudden onset weakness in left upper and lower limb for 3 hours. The patient was afebrile, with a regular pulse rate of 78/min and blood pressure of 130/70 mmHg. He was conscious but confused. Power in left upper limb was grade 1 and lower limb grade 3. Cranial nerves were normal on examination. The National Institute of Health Stroke Score (NIHSS) was 6 on admission. Rest of the physical examination was normal. The patient was immediately shifted to magnetic resonance imaging (MRI) for a scan. The emergency laboratory reports showed a random blood sugar of 40 mg/dl. Liver and renal function tests, lipid profile, electrolytes (calcium, sodium, potassium, phosphate), and hemogram were normal. On questioning, it was found that the patient had taken an extra tablet of glibenclamide. 100 ml of 25% dextrose solution was given. Repeat blood sugar was 77 mg/dl. The confusion improved, power normalized without any residual neurodeficit and repeat NIHSS was 0. Surprisingly, patient’s MRI-diffusion weighted imaging (DWI) and activated diffusion coefficient (ADC) was suggestive of hyperacute ischemia in posterior limb of the right internal capsule [Figures 1 and 2]. However, as it did not appear hyperintense on fluid-attenuated inversion recovery sequence, hyperacute ischemia was suspected. The magnetic resonance angiogram was normal.

Although the patient recovered fully with no residual deficit, treatment with antiplatelets and statin was started. An endocrinology consultation was made. The patient was counseled about hypoglycemic symptoms and dosage of oral hypoglycemic drugs were adjusted. On follow-up at 6 weeks, the patient was completely asymptomatic with no neurodeficit.

Hypoglycemia in diabetic patients can occur because of dietetic errors, alcohol abuse, excessive physical effort, or error in the dose of oral hypoglycemic agent (OHA), especially in elderly patients. Nonischemic causes account for around 13% of cases.[1] Hemiparesis is a rare sign of hypoglycemia as usually ischemia, and hemorrhagic events are considered the most common events for hemiparesis in an elderly in the clinical setting of risk factors. Hemiparesis secondary to hypoglycemia has been reported as early as 1928.[2] Patients experience transient hemiparesis with retained alertness during periods of hypoglycemia, which resolves with correction of hypoglycemia. Surprisingly, the manifestations of hypoglycemia (hunger, diaphoresis, generalized weakness, dizziness) are usually absent during attacks of hypoglycemia-induced hemiparesis (HH).[3]

In neonates, hypoglycemia is very common, with occipital lobes being involved more than parietal lobe. This is due to a regional deficit in expression and function of the glucose membrane transporter proteins. However, in adults, the regions involved are basal ganglia, hippocampus, insula, frontal regions, and corpus callosum. Pathogenesis of neuronal damage during hypoglycemic episodes involves cytotoxic edema, shrinkage of the extracellular space (ECS) and failure of ionic pumps of the cell membrane after energy depletion.[2] Hypoglycemic brain injury is widely studied in patients with hypoglycemic coma showing the involvement of cerebral cortex, basal ganglia, and hippocampus. In patients presenting with hemiparesis, the internal capsule and splenium were often affected, suggesting that these areas are more vulnerable.[4] Local neurodeficit may also occur after seizures. A 2–3-fold increase in glucose utilization
In patients with diabetes mellitus, receiving either insulin or OHA, the presence of focal or general neurologic symptoms requires the consideration of hypoglycemia as a possible cause. Especially in diabetic patients with autonomic neuropathy, blunting of the catecholamine response to hypoglycemia may result in isolated neuroglycopenic symptoms.

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Conflicts of interest
There are no conflicts of interest.

References

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