

**Hypoglycemic Hemiparesis:
The Stroke Masquerader**

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To the Editor:

The classic presentation of hypoglycemia involves Whipple's triad of signs and symptoms of hypoglycemia, low plasma glucose, and resolution of symptoms with correction of glucose derangement.¹ Findings in hypoglycemia are classified as either neuroglycopenic or autonomic.² Whereas some are synonymous with hypoglycemia and allow prompt recognition and treatment, there are other manifestations of hypoglycemia that are relatively uncommon, such as hemiparesis, which can be mistakenly attributed to other etiologies.

A 63-year-old female diabetic smoker with hypertension presented to the hospital with right-sided weakness that started acutely that morning. She reported having 3 similar transient episodes over the last month. She affirmed compliance with her medications, which included glyburide, metformin, hydrochlorothiazide, triamterene, and lisinopril, and described anorexia with poor oral intake for several weeks. Emergency medical technician evaluation identified a blood glucose level of 42 mg/dL, which, upon correction, resulted in immediate resolution of her hemiparesis. At emergency department arrival, the patient had stable vital signs with an unremarkable physical examination including normal strength, sensation, reflexes, cranial nerves, and higher cognitive functions. Laboratory investigations revealed acute elevations of creatinine 5.1 mg/dL and blood urea nitrogen 38 mg/dL, with a hemoglobin A1c 4.8% and normal lipid profile. The reported hemiparesis triggered the hospital's stroke protocol, initiating neurology consultation in the emergency room. A noncontrast computed tomography scan of the head showed no acute abnormality, and magnetic resonance imaging/magnetic resonance angiography of the brain and transthoracic echocardiography performed the next day also showed no abnormality. After admission, glyburide and metformin were discontinued given the suspicion that the hypoglycemia was precipitated by impaired sulfonylurea

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E-mail address: nnannapa@med.wayne.edu

clearance in the setting of acute renal insufficiency from poor oral intake combined with diuretic use and was leading to the hemiparesis. No further episodes occurred during her hospitalization, and her renal failure resolved with intravenous fluid resuscitation and withholding of the lisinopril and diuretics. Endocrinology consultants recommended against prescribing oral hypoglycemics upon discharge given her previous episodes of hypoglycemia and euglycemia during hospitalization while off any therapy.

Hemiparesis is a known, yet uncommon, presentation of hypoglycemia that can be mistakenly attributed to other causes. The association between hypoglycemia and hemiparesis has been described as far back as 1928.³ More recent investigations have noted a predilection for right-sided symptoms⁴ and a correlation with transient abnormal radiographic findings within the internal capsule and splenium of the corpus callosum.³ At present, the pathophysiology behind hypoglycemia-induced hemiparesis is unclear, but hypotheses include cerebral vasospasm, asymmetric cerebral blood flow, and selective neuronal vulnerability.³ Should a patient present with acute hemiparesis, prompt evaluation of plasma glucose and review of their diabetic history will allow for rapid identification of hypoglycemia as a potential cause. Caution must be given, however, to simply attributing the neurological deficit to hypoglycemia and disregarding a cerebrovascular event due to the significantly higher prevalence of the latter in patients with significant risk factors for such an event.

Naveen Nannapaneni, MD^a

Alaa Elkhider, MD^a

Joel Steinberg, MD^{a,b}

^aDepartment of Internal Medicine

^bDivision of Geriatrics

Wayne State University/Detroit Medical Center

Detroit, Mich

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