Short Communications

Hypoglycemic Hemiplegia: Two Cases and a Clinical Review

John W. Foster, MD and Robert G. Hart, MD

Hypoglycemic hemiplegia mimics cerebrovascular disease. Two patients are reported who experienced multiple attacks of transient hemiplegia associated with hypoglycemia and who were initially diagnosed as having transient ischemic attacks. In both, angiography was normal and the attacks resolved with reduction of insulin dose. Recognition of hypoglycemia as the cause of transient hemiplegia is important, often obviating the need for cerebrovascular evaluation. (Stroke 1987;18:944-946)

Transient hemiplegia with preservation of alertness is an underrecognized manifestation of hypoglycemia. When hypoglycemia is accompanied by symptoms of adrenergic discharge, such as tachycardia and profound sweating, in combination with altered mental status, the diagnosis is straightforward. Yet in certain patients, hypoglycemia presents with isolated, focal neurologic signs. In these patients, the diagnosis is not readily apparent, leading to unnecessary evaluation for cerebrovascular disease. Two such cases are reported.

Reports of Cases

Case 1

A 64-year-old Mexican-American woman with insulin-dependent diabetes (45 units NPH insulin/daily) presented with the sudden onset of right hemiparesis, right hemisensory loss, and inability to speak. She drank orange juice at home and was slightly improved by the time she arrived in the emergency room. On initial examination, she was noted to be awake and alert, with a right hemiparesis and dysphasia. Her blood glucose was 70 mg%; a solution of 50% dextrose in water was given with subsequent complete resolution of her neurologic deficit. Further laboratory evaluation was unremarkable. A diagnosis of transient ischemic attack (TIA) was made. She underwent cranial computed tomography (CT) and four-vessel cerebral angiography, both of which were entirely normal. She had no further episodes while in the hospital and was treated with aspirin.

Three weeks after hospital discharge, she again became hemiparetic on the right and mute. She was given i.v. dextrose with prompt recovery; a serum sample obtained before dextrose administration revealed a serum glucose of 30 mg%. She reported that she had experienced 3 other similar episodes since hospital discharge, all occurring at approximately 9 AM, and all responding rapidly to glucose ingestion. Her daily insulin dosage was decreased to 20 units/day. She experienced no further episodes over the subsequent 6 months.

Case 2

A 62-year-old man with "brittle" insulin-dependent diabetes, chronic hypertension, and diabetic nephropathy experienced generalized weakness and diaphoresis, similar to his many previous hypoglycemic reactions. Shortly thereafter, he was noted by his family to have right-sided weakness and abnormal speech. Oral glucose was administered, and all symptoms and signs had resolved by arrival at an emergency room 2 hours later. He was admitted for evaluation of presumed TIA.

At the same time on the next day, he again developed abnormal speech and right hemiparesis. Serum glucose was 29 mg%. Symptoms resolved within 15 minutes of i.v. dextrose infusion. A CT scan and selective left carotid angiogram were normal. During the remainder of his hospitalization, several episodes of hypoglycemia occurred, with serum glucose values as low as 39 mg%. No further episodes of hemiparesis occurred, with generalized weakness and diaphoresis accompanying subsequent hypoglycemic attacks. Ten weeks later, he experienced a left thalamic hemorrhage.

Discussion

Hypoglycemic hemiplegia (HH) is important to recognize as it is often confused with cerebrovascular disease. Both of our recent patients were initially evaluated for TIA before HH was diagnosed.

Transient hemiparesis associated with insulin-induced hypoglycemia has been recognized for more than 50 years. In a group of 125 consecutive patients admitted to Harlem Hospital over a 12-month period with symptomatic hypoglycemia, 3 (2%) experienced transient hemiparesis. HH is probably under-
recognized. It is our impression that most physicians who are aware of this entity and who care for diabetic patients have seen patients with suspected or definite HH.  

Patients with HH experience transient hemiparesis with retained alertness during periods of hypoglycemia, which resolves with correction of hypoglycemia. Surprisingly, the usual manifestations of hypoglycemia (e.g., hunger, diaphoresis, generalized weakness, and dizziness) are usually absent during attacks of HH. Many patients experience recurrent HH interspersed with nonfocal, typical hypoglycemic manifestations during other episodes of hypoglycemia. Hypoglycemia purposely induced by insulin infusion, undertaken in an attempt to reproduce HH, has unpredictably resulted in hemiplegia. 

Based on 29 well-documented cases from the literature, the clinical spectrum of HH is outlined in Table 1. HH usually occurs in diabetic patients receiving insulin (72%) or oral hypoglycemic agents (14%), but HH also occurs in nondiabetic patients with other causes of hypoglycemia (14%). The mean serum glucose during HH is 35 mg%. On average, most patients experience 3.5 attacks before HH is recognized and treated. Attacks of hemiparesis alternate, involving both the left and right sides, in 29% of cases. Curiously, right hemiparesis is far more common than left hemiparesis (72 vs. 28%) and is often accompanied by aphasia. The nature of the paralysis is not distinctive and may be either flaccid, or, less often, spastic. The duration of HH is, in general, somewhat longer than the usual duration of TIA due to cerebrovascular disease, although there is considerable overlap. Brain hemorrhage following HH has not previously been reported and may have been unrelated in our patient.

The mechanism underlying hypoglycemia-induced hemiplegia is unclear. Current hypotheses include cerebr.al vasospasm, selective neuronal vulnerability, and underlying cerebrovascular disease. The role of cerebrovascular disease gained prominence after Porto14 reported a patient with left-sided weakness and dizziness) are usually absent during attacks of HH. The nature of the paralysis is not distinctive and may be either flaccid, or, less often, spastic. The duration of HH is, in general, somewhat longer than the usual duration of TIA due to cerebrovascular disease, although there is considerable overlap. Brain hemorrhage following HH has not previously been reported and may have been unrelated in our patient.

The mechanism underlying hypoglycemia-induced hemiplegia is unclear. Current hypotheses include cerebral vasospasm, selective neuronal vulnerability, and underlying cerebrovascular disease. The role of cerebrovascular disease gained prominence after Porto14 reported a patient with left-sided weakness and speech disturbance in whom correction of right carotid stenosis apparently prevented subsequent hypoglycemic episodes from producing focal deficits. In animals, ligation of the middle cerebral artery produces contralateral hemiparesis when hypoglycemia is artificially produced, which resolves when glucose is re- turned to normal. In our patients, however, angiography revealed no underlying abnormality. In a series of 16 patients with HH, Wallis et al noted underlying brain disease in only 1 patient. Additionally, HH has been reported in patients with low likelihood of underlying cerebrovascular disease, including young patients with insulinomas, children, and patients receIVING insulin shock therapy. Different regions of the nervous system demonstrate variable response to systemic insults such as anoxia, intoxicants, and metabolic disorders. This regional variability has been termed selective vulnerability and has been attributed to local differences in cellular metabolism and vascular supply. The concept of selective neuronal vulnerability has been demonstrated in both clinical and anatomic studies, but this does not readily explain the lateralized and often alternating nature of the deficit in HH.

In diabetic patients receiving either insulin or oral hypoglycemic agents, the presence of focal or general neurologic symptoms requires the consideration of hypoglycemia as the offending agent. Particularly in diabetic patients with autonomic neuropathy, blunting of the epinephrine response to hypoglycemia may result in isolated neuroglycopenic symptoms. In view of the infrequent association with underlying cerebrovascular disease, hemiparesis that is clearly associated with hypoglycemia probably does not warrant angiographic evaluation.

References


Table 1. Hypoglycemic Hemiplegia: Clinical Spectrum in 29 Patients

| Age: mean 47 yrs, range 17-73 yrs; 45% < 40 yrs; 55% ≥ 40 yrs | Sex: 52% men, 48% women |
| Cause of hypoglycemia: | Insulin 72% |
| Oral hypoglycemics | 14% |
| Other | 14% |
| Insulinoma | 2 individuals |
| Fibrosarcoma | 1 individual |
| Alcohol | 1 individual |
| Serum glucose: mean 35 mg%, range 17-40 mg%; 18% < 20 mg%, 40% 20-30 mg%, 42% 31-40 mg% |
| Paralysis: 72% right-sided, 28% left-sided |
| Recurrent attacks: 78% |
| Angiography results: stenosis in 9% (1 of 11) |

Patients described in Refs. 3-11, 13-15.

Key Words • hypoglycemia • insulin reaction • transient ischemic attack • cerebrovascular disease
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