Reversible decerebrate posturing after profound and prolonged hypoglycemia

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■ Decerebrate rigidity is one of several reversible neurological abnormalities which have been observed in the setting of metabolic coma. We present the case of a patient who recovered fully from prolonged decerebrate rigidity associated with hypoglycemic coma. This case emphasizes the possibility of recovery from severe, prolonged hypoglycemia.

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HYPOGLYCEMIA may cause a number of changes in brain function, some of which are reversible. We describe the case of a patient who recovered after exhibiting decerebrate posturing for six hours during hypoglycemia from a massive insulin overdose.

CASE REPORT

A 32-year-old man with poorly controlled type I diabetes mellitus of 19 years' duration complicated by end stage renal disease and peripheral and autonomic neuropathy, was found unresponsive in his home. He had a history of frequent hypoglycemic episodes, including several in the five days before presentation.

On initial presentation to a local hospital emergency room, the patient was bradycardic (40 beats/min) and hypotensive. He was treated with intravenous atropine within three minutes of arrival, after which he assumed a decerebrate posture. Noteworthy laboratory results included: serum glucose 8 mg/dL, potassium 7.7 mEq/L, sodium 144 mEq/L, BUN 146 mg/dl, bilirubin 0.5 mg/dl, and serum pH 6.98. With initial correction of acidemia and hypoglycemia (3 ampules of 50% dextrose over one hour), serum glucose rose to 224 mg/dl and decerebrate posturing ceased. One hour later, however, unresponsiveness with decerebrate posturing recurred. Serum glucose was found to be less than 20 mg/dl, and he was transferred to the Cleveland Clinic Hospital.

On arrival, about 6 hours after the initial presentation, he was unresponsive and diaphoretic with roving eye movements. His temperature was 38.6°C (axillary), blood pressure was 132/80, heart rate was 160 and regular, and respirations were 32. Sternal pressure elicited decorticate posturing. Bilateral corneal reflexes were present, and the patient moved all extremities spontaneously. Deep tendon reflexes were absent, but plantar reflexes were extensor bilaterally.

Two hours after admission, the patient had a brief tonic-clonic seizure. Serum glucose was 26 mg/dL at the onset of the seizure. A residual decerebrate posture remained for the next 6 hours, during which time his serum glucose level remained less than 60 mg/dL in spite of 100 mg of intravenous methylprednisolone,

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and a total of 800 mL of 50% dextrose solution.

When the serum glucose level rose to 238 mg/L and was maintained at roughly this level, decerebrate posturing disappeared, and the patient's level of consciousness progressively improved. Over the next 3 days, serum glucose levels ranged from 130 mg/dL to 250 mg/dL, and his mental status returned to a reported baseline of verbal coherence, alertness, answering yes or no questions, and following simple commands. Decerebrate posturing did not return once the hypoglycemia was corrected.

This patient's prior episodes of sustained hypoglycemia, and the severe degree and long duration of the current hypoglycemic episode were consistent with an insulin overdose. This was confirmed by a serum insulin concentration of greater than 11,000 μU/mL (normal fasting concentration 4 to 24 μU/mL) in a sample obtained 12 hours after his initial presentation to the local hospital. At 96 hours after presentation, the level was 79.7 μU/mL, and at 108 hours, it was 65.0 μU/mL. Assays for plasma protein binding of insulin ("insulin antibodies") showed <5% binding, which is considered to be a normal value and of no clinical significance in patients with diabetes mellitus.

DISCUSSION

The current report demonstrates two uncommon but noteworthy clinical points: (1) decerebrate posturing in hypoglycemia may be transient and reversible with correction of hypoglycemia, and (2) neurologic recovery is possible despite severe insulin overdose with prolonged hypoglycemia.

Abnormal posturing was first observed in experimental animals following intercollicular section of the brain stem (decerebrate)[1,2] and removal of all cortex and white matter above the level of the basal ganglia (decorticate). These two postures have been consistently produced by specific anatomic lesions in experimental animals, and it has been inferred that analogous anatomic lesions are responsible for these postures in humans. Indeed, clinical decortication and decerebration most often result from a structural lesion (usually trauma). However, a variety of metabolic and other causes have been reported, including anoxia,[3] infections,[4] hepatic coma,[5,6] the syndrome of inappropriate secretion of antidiuretic hormone (SIADH),[7] and hypoglycemia.[8,9] A few of these non-structural causes have been associated with reversible posturing.

In our patient, decerebrate rigidity coincided with hypoglycemia. During the first 6 hours, decerebration rapidly resolved when hypoglycemia was corrected and recurred when hypoglycemia returned.

Two previous cases of reversible decerebrate posturing secondary to hypoglycemia have been reported. Seibert[8] described the remission of decerebrate posturing after administration of intravenous glucose in two patients, one of whom was a man with adult-onset diabetes who presented in coma with decerebrate posturing in response to painful stimuli and a serum glucose of 35 mg/dL. He became alert and conversant following 50 mL of 50% dextrose. (The other patient was a 59-year-old male alcoholic with neither diabetes nor insulinoma and without documented hypoglycemia, who presented with hypothermia, tachycardia, diaphoresis, and coma with decerebrate posturing, as well as anisocoria and bilateral extensor plantar reflexes. He awoke and became neurologically normal immediately after receiving 25 g of dextrose.)

The second case, described by Ogunyemi and Olowoyeye,[9] concerned a 65-year-old man with hypoglycemia caused by religious fasting and diarrhea. He became comatose with decorticate posturing on the right side in response to painful stimuli on either side of the body. The serum glucose was 8 mg/dL. An intravenous bolus of 100 mL of 50% dextrose solution resulted in resolution of the decorticate posture and return of consciousness within 90 minutes.

Our patient's course differs from the previously described experience, principally in the duration of the decerebrate posturing. In each previous case, the comatose patient demonstrated decerebrate posturing in response to painful stimuli, but decerebration was rapidly reversed and consciousness restored by a single dose of dextrose. In the current case, posturing occurred intermittently for 6 hours after admission, then constantly for 6 hours, and the presence of decerebrate rigidity coincided with hypoglycemia.

It is known that hypoglycemia can cause seizures, and Seibert[8] postulated hypoglycemia-induced tonic seizure activity as a mechanism for the decerebrate rigidity seen in his patients. Our patient, however, clearly demonstrated adduction, extension, and pronation of the arms, a posture very different from the abduction and flexion of the arms seen in global tonic seizures.[10] Furthermore, this patient's posture was made more prominent by noxious stimuli, which is typical of decerebrate rigidity[11] but not of seizures. Haines[12] describes five cases of decerebrate posturing which were misinterpreted as seizures, and gives an excellent account of the manner and importance of differentiating between the two.

Our patient's return to baseline neurologic status after a total of more than 6 hours of decerebration was unexpected. Arem and Zoghbi[13] indicate that full neurologic recovery after prolonged hypoglycemic coma without posturing (up to 6 days) is not unusual, although there is no account of recovery from such prolonged hypoglycemic decerebration. Using a pooled analysis of eight of their own patients and 38 cases from the literature, Arem and Zoghbi found that neither the magnitude of the insulin dose nor the severity of the resulting hypoglycemia had any effect on the clinical outcome in intentional insulin overdose. The most important factor was delay in initiation of treatment.[13] That the human brain can withstand long periods of metabolic insult severe enough to cause decerebration is shown by the reports of Conomy and Swash[5] in which two patients with hepatic coma demonstrated decerebrate rigidity that lasted about 2 days and resolved as the coma cleared, without neurological sequelae.

The total amount of insulin given to this patient is unknown. That the insulin dose was especially large can be inferred from the serum insulin level: 12 hours after admission it exceeded 11,000 μU/mL, which is among the highest values ever reported. However, confounding factors make it difficult to estimate the size of the dose. When injected intravenously, insulin has a plasma half-life of about 10 minutes,[14] but the principal determinant of serum levels is the rate of release from the injection site which, in turn, is influenced by the type of insulin used. An added factor in this case was the presence of complete renal failure, a variable not discussed in most of the earlier reports of insulin overdose. Insulin is degraded mainly in the liver and kidneys, and renal failure causes a significant lengthening of the half-life of insulin (the liver normally operates close to its capacity to metabolize insulin and cannot compensate for loss of kidney function).[14]

The current case demonstrates that decerebrate posturing in hypoglycemia may be transient and reversible with correction of hypoglycemia, and that neurologic recovery may be possible despite severe insulin overdose with resulting prolonged hypoglycemia.

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