Motor Aphasia due to Prolonged Hypoglycaemic Coma in a Patient with Insulin-dependent Diabetes Mellitus

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A 45-year-old insulin-dependent diabetic man was in a hypoglycaemic coma for one month but recovered after continuous infusion of glucose and insulin. An isolated neurological deficit, motor aphasia, persisted after recovery from the coma. Repeated computerized tomography did not demonstrate any abnormal findings attributable to coma or aphasia. Precise follow-up examinations of aphasia showed improvement of Broca type motor aphasia to transcortical motor aphasia. Hypoglycaemic aphasia in a patient after recovery from prolonged coma is rare and its clinical course and pathogenesis are discussed with reference to the available literature.

KEY WORDS: Aphasia; Diabetes mellitus; Hypoglycaemia; Coma

INTRODUCTION

The interval from the onset of hypoglycaemic coma to the start of treatment and the overall duration of coma are poor prognostic factors for ultimate outcome. Prolonged coma has been found to cause diverse and severe brain injury at autopsy. However, hypoglycaemia presents with neurological dysfunction and, rarely, with aphasia alone, emphasizing the importance of differential diagnosis from cerebral vascular diseases. Computerized tomography (CT) and angiography demonstrate no abnormal findings attributable to hypoglycaemic focal deficit, in contrast to stroke. A diabetic patient is described who recovered from a deep coma which lasted for 1 month which was caused by hypogly-
caemia, following which he developed an isolated neurological deficit of motor aphasia.

**CASE REPORT**

A 45-year-old right-handed man had been diagnosed with insulin-dependent diabetes mellitus 8 years previously and treated with isophane insulin 30 units every morning. In July 1994, he was discovered comatose in bed in his room at 17.00 h with a used insulin syringe beside him and was quickly transported to our hospital. Medical records indicated that he had been referred to our hospital 3 years previously, but he subsequently seldom attended for medical checks, despite being sent reminders. His records showed that he experienced hypoglycaemic symptoms before lunch once or twice a year. Serum glucose levels after breakfast were 11.1–19.4 mmol/l and the serum glycohaemoglobin A1c levels were 9–12%.

At the time of his current admission, the capillary glucose level was below 1.11 mmol/l (20 mg/dl) but other routine laboratory tests were normal. Normal deep tendon reflexes and flexor plantar response were present despite the fact that he was in a coma. Meningeal signs were not recognized. Plain- and contrast-enhanced CT showed no abnormal findings. Fundoscopy showed diabetic retinopathy of Scott I and ultrasonography demonstrated the presence of a fatty liver.

An intravenous injection of 60 ml of 50% glucose was given followed by an intravenous drip infusion of 10% glucose with electrolytes, but the patient remained comatose with serum glucose levels of 2.55, 4.00 and 2.55 mmol/l at 19:00, 21:00 and 23:00 h, respectively. On the following day, however, both the light reflex and the ciliary reflex reappeared. Serum glucose levels were 3.55, 4.44, 20.3, 10.1 and 24.7 mmol/l at 5:00, 9:00, 13:00, 17:00 and 23:00 h, respectively. On day 3, insulin with intravenous hyperalimentation, including 17% glucose (1400 kcal/day), was started under the strict monitoring of serum glucose concentrations. On day 5 he began to move his extremities and respond to his name. On day 10, the intravenous infusion of 17% glucose was increased to 25% glucose (2060 kcal/day). He began to say a few unclear words in a very low voice with his eyes still closed. On day 15, he sometimes turned his head towards sounds or speech. His eyes were open but he said nothing.

One month after the onset of the hypoglycaemic coma, he had come out of the coma and could understand simple instructions to eat a small meal in bed with assistance. No neurological focal deficits other than motor aphasia were recognized, and there were no abnormal findings on CT. His clinical recovery of speech, as determined by standard language tests for aphasia, is summarized in Figure 1. The motor aphasia of Broca type progressed to transcortical motor aphasia.

On day 38 in hospital, the intravenous drip was discontinued because he had recovered sufficiently to eat an adequate meal by himself. He was given 16–24 units of isophane insulin every morning without any sign of hypoglycaemia.

A year after the onset, poor spontaneous speech persisted. He was on isophane insulin, 16 units daily, which gave a serum glucose profile, including that at midnight, of 8.33–13.9 mmol/l. One and a half years after the event, although poor spontaneous speech persisted, he was able to read newspapers and to travel alone by public transport.

**DISCUSSION**

We described a diabetic patient who developed motor aphasia following a hypoglycaemic coma which lasted for 1 month. His
Neurological focal deficits caused by hypoglycaemic coma have been reported rarely in patients with diabetes mellitus; hemiparesis and seizure are common, but aphasia is rare. Neuronal death has been reported in the cerebral cortex, hippocampus and putamen in patients who died of severe hypoglycaemia. In most cases reported previously, CT and angiography performed during hypoglycaemic coma showed no abnormal findings. In contrast, Koppel and Daras described a patient with diabetes mellitus in whom non-enhanced hypodensity was recognized in the left internal capsule on CT during coma followed by aphasia and right hemiplegia. However, the symptoms disappeared immediately after the injection of glucose solution. In a patient with hypoglycaemic coma accompanied by aphasia and right hemiplegia lasting 24 h, Meer et al. showed that there were abnormal findings in
the brain region in the right caudate nucleus and lenticular nucleus on enhanced CT. However, the brain regions described in those reports could not explain the cause of aphasia fully. Perros et al. demonstrated a high MRI signal in the pons during pontine dysfunction resulting from hypoglycaemia in diabetic patients. As such an abnormal sign was not detected on CT, they emphasized the usefulness of MRI. However, the involvement of brain regions on MRI in the occurrence of aphasia currently remains controversial.

Focal deficits in hypoglycaemia are reportedly caused by underlying vascular lesions, partial vessel occlusion, vasospasm induced by hypoglycaemia, and selective neuronal vulnerability. Results from various investigations including angiography, CT and single photon emission tomography (PET) and MRI were inconclusive for explaining the pathogenesis of focal signs in hypoglycaemia. Auer and co-workers proposed that a cerebrospinal fluid (CSF)-borne toxin produced endogenously by the excitatory amino acids, aspartate and glutamate, affects calcium channels and that neuronal death involves that these neurotoxins trigger hyperexcitation, culminating in cell membrane rupture rather than in internal catabolic death of the neuron by hypoglycaemia. From this hypothesis, it is assumed that transient cerebral lesions caused by hypoglycaemia might be explained, in part, by cell dysfunction such as membrane or calcium channel disorder rather than by the cell necrosis-like hypodensity region on CT observed in cerebral infarction.

REFERENCES


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