

SHORT REPORT

Hypoglycaemic hemiplegia: a repeat SPECT study

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Abstract

During a hypoglycaemic right hemiplegia induced by a deliberate overdose of oral hypoglycaemics, brain CT and angiography revealed no abnormalities. SPECTs made one day and six days later showed relative hypoperfusion in the left hemisphere. Repeat SPECT study suggested that the left hemisphere was more vulnerable than the right in the cerebral blood perfusion. This vulnerability might provoke the right hemiplegia in a critical condition, such as severe hypoglycaemia.

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Hypoglycaemia usually causes diffuse brain dysfunction. Occasionally, however, focal neurological signs and symptoms such as hypoglycaemic hemiplegia or movement disorders may masquerade as cerebrovascular disease. We describe a patient with hypoglycaemia-induced right sided hemiplegia, and discuss the mechanism of hypoglycaemic hemiplegia.

Case report

A right handed 31 year old woman took 30 75 mg tablets of Glibenclamide in an attempted suicide. The next day, she became somnolent and could not speak. That evening she was admitted as an emergency.

On admission, when we were unaware of her attempted suicide, her blood pressure was 128/80, and heart rate was 76/min and regular. Body temperature was 34.4°C. Respiration was 18/min and regular. Her

family and past history were unremarkable. She was confused mute and unable to answer questions. She had a right hemiplegia which included her face. Her right arm showed flaccid paralysis, and did not respond to any painful stimuli. Her right leg moved slightly in response to extensive painful stimuli. Her left extremities were intact, and the patient strongly extended and flexed the left leg and arm at the elbow and knee joints in her confusional state. Deep tendon reflexes were increased bilaterally, and the right plantar response was extensor.

Brain CT and left carotid angiography revealed no abnormalities including the venous phase. Later, after angiography, her blood glucose investigation taken on admission was reported to be 21 mg/dl. Immediate infusion of 50% glucose 60 ml resulted in a remarkable improvement. She regained consciousness, and the right hemiplegia and Babinski's sign promptly disappeared. She was aware of her situation and was able to answer questions. The patient's mutism and aphasia also improved after correction of the hypoglycaemia.

The following day, single photon emission CT (SPECT), tracer: ^{99m}Tc-hexamethyl (HM)-propyleneamine oxime (PAO), was performed when her blood glucose was 119 mg/dl. SPECT showed marked hypoperfusion on the left fronto-parietal lobe although blood glucose was normal and she was considered neurologically normal (fig 1A, B). MRI the next day showed no abnormalities.

Laboratory examination on admission showed the following: red blood cells (RBCs) $330 \times 10^4/\text{mm}^3$, haemoglobin 11.2 g/dl, haema-

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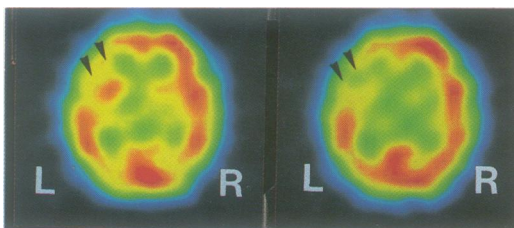


Figure 1AB SPECT (tracer: ^{99m}Tc-hexamethyl-propyleneamine oxime [PAO]) on the day after admission (the simultaneous blood glucose concentration: 119 mg/dl) revealed the hypoperfusion (arrow heads) of the left hemisphere.

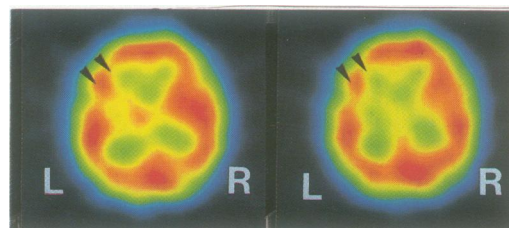


Figure 2AB SPECT on the seventh hospital day (the simultaneous blood glucose concentration: 108mg/dl) showed the perfusion improved in the left hemisphere, but relative hypoperfusion compared with the right hemisphere had remained (arrow heads).

tocrit 33.0%, white blood cells (WBCs) $13,800/\text{mm}^3$, platelets $32.6 \times 10^4/\text{mm}^3$. Blood chemistry showed normal findings. Coagulation tests were all within normal range.

On day 7 a repeat SPECT study was performed on her simultaneous blood glucose 108mg/dl. It showed an improved cerebral blood perfusion in the left hemisphere, but relative hypoperfusion compared with the right hemisphere had remained (fig 2A, B). On day 8 she was discharged without any neurological sequelae.

Discussion

It is important to recognise hypoglycaemic hemiplegia as it is often confused with cerebrovascular disease. Our patient was initially evaluated for juvenile ischaemic cerebrovascular disease before hypoglycaemic hemiplegia was diagnosed.

We evaluated previously reported 56 hypoglycaemic hemiplegia cases.¹⁻¹¹ Hypoglycaemic hemiplegia usually occurs in diabetic patients receiving insulin (63%) or oral hypoglycaemics (11%), but hypoglycaemic hemiplegia also occurs in nondiabetic patients with other causes of hypoglycaemia (26%), such as insulinoma, insulin autoimmune syndrome. Our case took an overdose of hypoglycaemics in a suicide attempt, and a nondiabetic healthy case with hypoglycaemic hemiplegia has not previously been reported. In the previously reported cases, the mean serum glucose during a hypoglycaemic hemiplegia attack was 36.5 mg/dl. Angiography was performed in 16 cases. Stenosis of the initial or siphon part of the internal carotid artery was present in 3 of 16 (19%).^{3,8,9} The other 13 cases including our case showed normal findings.^{1,4-6,10,11}

The cerebral angiography in our case was carried out at the same time as the hypoglycaemia. In other reported cases, angiographies were performed after recovery to normoglycaemia. CT findings were normal in 89% (16 of 18). Hemiparesis is far more common on the right side than on the left (64 vs 27%, $p < 0.05$) and is often accompanied by aphasia. Attacks of hemiparesis may, involve both the left and right sides, in 9% (5 of 56).^{5,7}

The mechanism underlying hypoglycaemia-induced hemiplegia remains unknown. Current hypotheses include cerebral vasospasm,¹ selective neuronal vulnerability,² and underlying cerebrovascular disease.³

The role of vasospasm has not been proven

by angiography. In our case the carotid angiogram carried out at the same time as the hypoglycaemic attack revealed normal findings without vasospastic appearances. Although the findings of vasospasm has not been previously reported, the resolution of angiography is such that it does not image small arteries less than about 500 μ .

The role of cerebrovascular disease gained prominence after Portnoy reported a patient with left sided weakness and speech disturbance in whom correction of right carotid stenosis apparently prevented subsequent hypoglycaemic episodes from producing focal deficits.³ However, in most of the previously reported angiography results in hypoglycaemic hemiplegia were normal.

SPECT study was initially performed in hypoglycaemic hemiplegia in 1990.¹⁰ In that reported case, SPECT showed normal findings with neither hypoperfusion nor laterality.

In our case, a repeat SPECT study revealed persistent relative hypoperfusion in the left hemisphere compared with the right. Although the angiography showed normal findings, repeat SPECT suggested that the left hemisphere perfusion was weaker than the right. This weakness may provoke a right hemiplegia in a critical condition, such as, severe hypoglycaemia.

The patient's body temperature was 34.4°C on admission. This hypothermia might be correlated with the hypoglycaemia-induced hypometabolism in the patient.

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