CASE REPORT

Hypoglycaemic haemiparesis

Othman Kirresh,¹ Achmed Kamara,² Samad Samadian³¹Department of Medicine, St Georges Hospital, London, UK²Department of Geriatrics, St Hellier Hospital, Surrey, UK³St Hellier Hospital, Surrey, UK**Correspondence to**Dr Othman Kirresh,
othmankirresh@doctors.org.uk**SUMMARY**

Hypoglycaemic haemiparesis (HH) is an uncommon but important presentation to the emergency department, and it often mimics stroke and is therefore frequently misdiagnosed by clinicians. The mechanism of haemiparesis is not fully understood. This case outlines a diabetic elderly woman, who had been having frequent hypoglycaemic episodes and presented to paramedics with hypoglycaemia associated with a right-sided haemiparesis. She was immediately transferred to the local stroke centre after presenting to the emergency department. CT and MRI did not fit in with her presenting neurology. Her weakness resolved, after normoglycaemia was achieved with dextrose infusion; however, she was reported to be more sleepy and drowsy than usual. After extensive and costly investigations during her prolonged inpatient stay, her unifying diagnosis was an HH which triggered of a hypoactive delirium.

BACKGROUND

Hypoglycaemic haemiparesis (HH) is an uncommon presentation of hypoglycaemia that is frequently misdiagnosed as stroke. A high index of suspicion when a patient presents with impaired mental state or clinical features of stroke, especially in patients with diabetes mellitus, and a low threshold for checking capillary $\pm$ serum glucose will facilitate accurate diagnosis of HH. If diagnosed early, HH is rapidly reversed with intravenous glucose infusion, avoiding permanent neurological damage, coma or death. This would also avoid performing expensive and time-consuming investigations that may not be cost effective and in some cases put patients at further risk.

CASE PRESENTATION

Paramedics attended to a 75-year-old woman with drowsiness and slurred speech. Capillary blood glucose was 1.9 and intramuscular glucagon (1 mg) was administered. The medical history included type 2 diabetes mellitus, hypertension, chronic kidney disease and treated spinal tuberculosis (TB). Relatives reported three episodes of hypoglycaemia in the preceding 2 days. Diabetic medications included metformin (500 mg twice daily) and gliclazide (80 mg twice daily). On arrival to the local emergency department triage assessment noted right-sided haemiparesis. The patient was immediately transferred to the regional stroke centre. Further assessment additionally detected dysphasia, dysarthria and right-sided positive Babinski's reflex. Cranial nerves examination was normal.

Dextrose intravenous infusions stabilised the blood sugar level and gliclazide initially reduced to 40 mg twice daily. High-dose aspirin (300 mg once daily) was started and the patient transferred to her

local hospital for ongoing care. The subsequent inpatient stay was complicated by fluctuating Glasgow Coma Score (10–15). The gliclazide was later stopped and sitagliptin started after endocrinology review. Further review by a local stroke consultant found no evidence of a stroke.

Following a neurologist review an EEG was conducted, which was normal, serum metabolic screen, including ammonia and autoimmune profiles were normal. Lumbar puncture was performed and cerebrospinal fluid analysis was normal (including staining for *Mycobacterium* TB. Overnight oximetry showed no significant oxygen desaturation events.

INVESTIGATIONS

Routine serum blood tests were unremarkable. The creatine was at baseline (160 μ mol/L (NR 60–100). Serum glucose was 3.9 mmol/L. ECG and plain chest radiograph were normal. Immediate CT scan of the brain demonstrated chronic small vessel ischaemia but no acute intracranial haemorrhage or large-vessel infarction. Carotid arterial Doppler ultrasonography showed no significant stenosis. Transthoracic echocardiography revealed moderately impaired left ventricular (LV) systolic function, but normal LV size and wall thickness. Diffusion-weighted imaging (DWI)–MRI scan of the brain 2 days later demonstrated acute anterior–superior right internal capsule infarct but no compatible area of infarct to account for the right-sided haemiparesis (figure 1).

Figure 1 Diffusion-weighted imaging (DWI)–MRI of the brain (2 days after admission) findings. There is an acute lacunar-type infarct (arrowed) anteriorly in the superior portion of the right internal capsule and some artefact anteriorly on the DWI sequence.

To cite: Kirresh O, Kamara A, Samadian S. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2013-008893

DIFFERENTIAL DIAGNOSIS

- ▶ Transient ischaemic attack
- ▶ Stroke
- ▶ Migraine (atypical)—Have they had migraines before? Family history?
- ▶ Hypoglycaemia
- ▶ Partial (focal) seizure
- ▶ Peripheral nerve lesion—generally associated with pain and history of cause of nerve
- ▶ Entrapment

TREATMENT

- ▶ Intramuscular glucagon
- ▶ Intravenous dextrose

OUTCOME AND FOLLOW-UP

A unifying diagnosis of HH with associated hypoactive delirium was made. The patient made a full recovery and was discharged home with a package of care.

DISCUSSION

'A stroke is never a stroke until it has received 50 of D50'. Hypoglycaemia should not be missed when treating a patient who is suspected to have had a stroke. Haemiparesis is a classic stroke feature, often mimicked by clinical entities, such as hemiplegic migraine, severe electrolyte imbalance, ictal/postictal phenomena and intracranial tumours/infections. In only about 2% of haemiparesis, hypoglycaemia is the attributed cause.^{1 2}

Blood glucose <2.2 mmol/L, is a recognised cause of metabolic encephalopathy. It commonly presents with autonomic manifestations attributed to increased sympathetic drive, for example, sweating, tachycardia, hunger, anxiety, irritability, etc. Alternatively, it can cause neuroglycopenic symptoms, including poor concentration, perioral tingling, personality change, seizures and focal neurological signs such as HH. According to one retrospective chart review of 59 patients, the most common neuroglycopenic symptoms are confusion (83%) and personality change (64%), while the most common autonomic symptoms are diaphoresis (69%) and tremor (24%).³ These symptoms are reversed on correction of serum glucose levels. The precise mechanism of the neuroglycopenic features is unknown, although several theories have been proposed.^{1 2}

Pathophysiology of HH

Early animal studies have shown a progressive decrease in high energy phosphate level concentrations in rat brains correlate proportionately to the severity of hypoglycaemia.⁴ Other studies suggest reduced acetylcholine levels could explain the generalised dysfunction (stupor) but does not explain the focal symptoms. Further studies on rats have suggested neuroglycopenic symptoms in HH may be multifactorial, in part due to selective loss of vulnerable cerebral neurons that develop impaired vascular autoregulation.^{5 6} A further review reported 12/16 patients with HH had right-sided weakness. All patients had resolution of their neurological deficits within 15 min of intravenous glucose infusion. The theory of selective neuronal vulnerability may explain the preponderance of right-sided haemiparesis in HH, as in the case presented.⁷

Why is HH more common in the elderly?

In the elderly, neuroglycopenic features of hypoglycaemia dominate and personality change might be the only manifestation, making diagnosis challenging. This could be explained by their

higher prevalence of intracerebral atherosclerosis, arteriosclerosis and arterial stenosis. During normoglycaemic states glucose delivery to intracerebral neurones is not compromised. When hypoglycaemia develops glucose delivery is impaired, deleteriously impacted by the increased sympathetic drive causing vaso-spasm in selective areas of the brain, it results in ischaemia and neuroglycopenic symptoms.⁸

Neuroimaging in diagnosing HH

Hypoglycaemic brain injury usually manifests as diffuse abnormalities. Unlike ischaemic stroke, MRI abnormalities in HH are atypical, diffuse and bilateral, and can affect the cortex, internal capsule, hippocampus and basal ganglia. Case reports demonstrated on imaging that changes were apparent on the contralateral side or both sides of the internal capsule in 13 (65%) of 22 cases, and in the splenium of the corpus callosum in 6 cases (30%).⁹ These changes usually reverse after clinical improvement.¹⁰

MRI techniques such as DWI are useful in diagnosing early ischaemic injury. One study demonstrated reversible abnormalities in DWI–MRI scans of the brain in 10 patients after glucose infusion was administered.¹¹

Learning points

- ▶ Always remember to check the blood sugar of any patient presenting with any neurological deficit.
- ▶ The elderly can present atypically.
- ▶ Hypoglycaemia can trigger of a delirium in the elderly.
- ▶ Do not forget to always use a systematic approach, based on airway, breathing, circulation, disability (blood glucose) and exposure/examinations (ie, the ABCDEs) to assess and treat the acutely ill patient.

Patient consent Obtained.

Competing interests None.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Longmore M, Wilkinson I, Davidson E, et al. *Oxford handbook of clinical medicine*. Oxford: OUP, 2010:206–7.
- 2 Ratcheson RA, Blank AC, Ferrendelli JA. Regionally selective metabolic effects of hypoglycemia in brain. *J Neurochem* 1981;36:1952–8.
- 3 Dizon AM, Kowalyk S, Hoogwerf BJ. Neuroglycopenic and other symptoms in patients with insulinomas. *Am J Med* 1999;106:307–10.
- 4 Wong KL, Tyce GM. Glucose and amino acid metabolism in rat brain during sustained hypoglycaemia. *Neurochem Res* 1983;8:401–15.
- 5 Holstein A, Egberts EH. CNS symptoms caused by hypoglycemia: frequent misdiagnosis: "stroke". Pitfall of the neuroglycopenia syndrome. *MMW Fortschr Med* 1999;141:42–4.
- 6 Kaya M, Kucuk M, Kalayci RB, et al. Magnesium sulphate attenuates increased blood-brain barrier permeability during insulin-induced hypoglycaemia in rats. *Can J Physiol Pharmacol* 2001;79:793–8.
- 7 Wallis WE, Donaldson I, Scott RS, et al. Hypoglycemia masquerading as cerebrovascular disease (hypoglycemic hemiplegia). *Ann Neurol* 1985;18:510–12.
- 8 Wattoo MA, Liu HH. Alternating transient dense hemiplegia due to episodes of hypoglycemia. *West J Med* 1999;170:170–1.
- 9 Hasegawa Y, Formato JE, Latour LL, et al. Severe transient hypoglycemia causes reversible change in the apparent diffusion coefficient of water. *Stroke* 1996;27:1648–55; discussion 1655–6.
- 10 Fujioka M, Okuchi K, Hiramatsu KI, et al. Specific changes in human brain after hypoglycemic injury. *Stroke* 1997;28:584–7.
- 11 Sato AT, Hasegawa K, Ishizaki R, et al. Reversible hyperintensity lesion on diffusion-weighted MRI in hypoglycemic coma. *Neurology* 2004;63:392–3.

Copyright 2013 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow