# Non-ketotic hyperglycaemic hemichorea: a rare complication of uncontrolled diabetes mellitus

**PL Lam *, PP Iu, Danny HY Cho**

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## Case 1

A 65-year-old man with a 1-month history of left upper and lower limb chorea was admitted to the medical ward of our institution in February 2021. Blood tests revealed new-onset diabetes mellitus with markedly elevated fasting glucose level of 28.5 mmol/L. Urinalysis for ketones was negative. Urgent non-contrast computed tomography (CT) of the brain showed subtle hyperdensity at the right putamen without mass effect or surrounding oedema (Fig 1). The patient was started on subcutaneous insulin, and upon normalisation of blood glucose level, his hemichorea subsided without additional antichorea medications. Follow-up non-contrast magnetic resonance imaging of the brain performed 2 months later revealed T1-weighted hyperintensity in the right putamen, without restricted diffusion (Fig 2). Imaging findings were in keeping with non-ketotic hyperglycaemic hemichorea (NHH). The patient was prescribed a biphasic insulin regimen upon discharge.

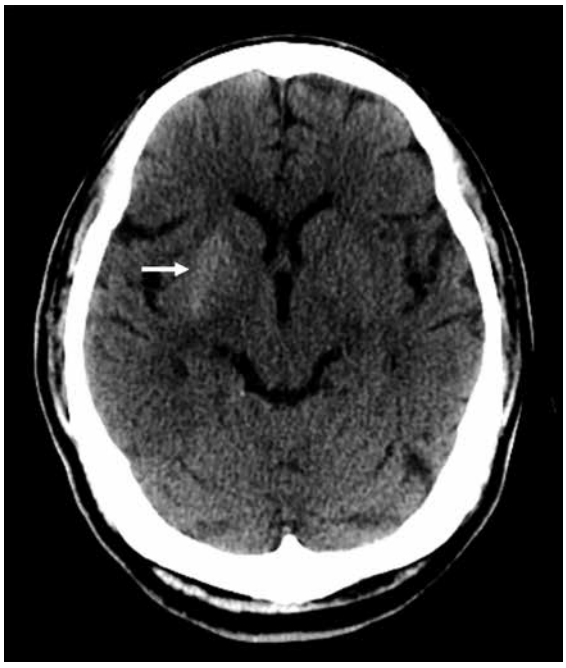

FIG 1. Case 1. Non-contrast computed tomography of the brain shows subtle hyperdensity in right putamen (arrow) without mass effect or surrounding oedema, in keeping with non-ketotic hyperglycaemic hemichorea

## Case 2

An 87-year-old man with a 2-day history of left upper limb chorea was hospitalised in April 2022. He had known diabetes mellitus but was noncompliant with oral hypoglycaemic therapy. Blood tests revealed markedly elevated random glucose level of 30.8 mmol/L. Urgent non-contrast CT of the brain showed asymmetric subtle hyperdensity in bilateral putamen and caudate nuclei, more extensive on the right, but without mass effect or surrounding oedema (Fig 3). Findings were compatible with NHH. The patient resumed metformin, with gliclazide and subcutaneous insulin injection added for optimal control. Upon normalisation of blood glucose level, his hemichorea resolved without antichorea medications.

## Discussion

Unilateral or asymmetric basal ganglia hyperdensity in brain CT of patients with focal neurological symptoms can be alarming, and intracerebral haemorrhage may be suspected. Nonetheless NHH, a rare complication of uncontrolled diabetes, should not be overlooked.

Case reports of NHH have been documented as early as 1960.[1] Previously reported cases were frequent in Asian elderly women with uncontrolled diabetes. In most patients, unilateral chorea was observed, although bilateral involvement could be present.[2-4] Of note, the aetiology of hemichorea is diverse, and other causes include infarct, haemorrhage and neoplasm. Imaging of the brain is therefore crucial.

Non-contrast CT of the brain of NHH typically shows subtle hyperdensity in contralateral putamen and/or caudate nucleus, although bilateral involvement is also seen. There will be no mass effect or perilesional oedema, and this differentiates NHH from haemorrhage or tumour.[2-4]

Similarly, magnetic resonance imaging of the brain typically shows corresponding signal changes in striatal regions contralateral to the symptomatic side. There will be T1-weighted hyperintense signal. Differentials of increased basal ganglia T1-weighted signal are diverse. They include toxin-related causes such as methanol poisoning or hepatic-related causes such as acquired hepatocerebral degeneration, but they commonly show bilateral and symmetrical involvement.[5] T2-weighted or fluid-attenuated

  

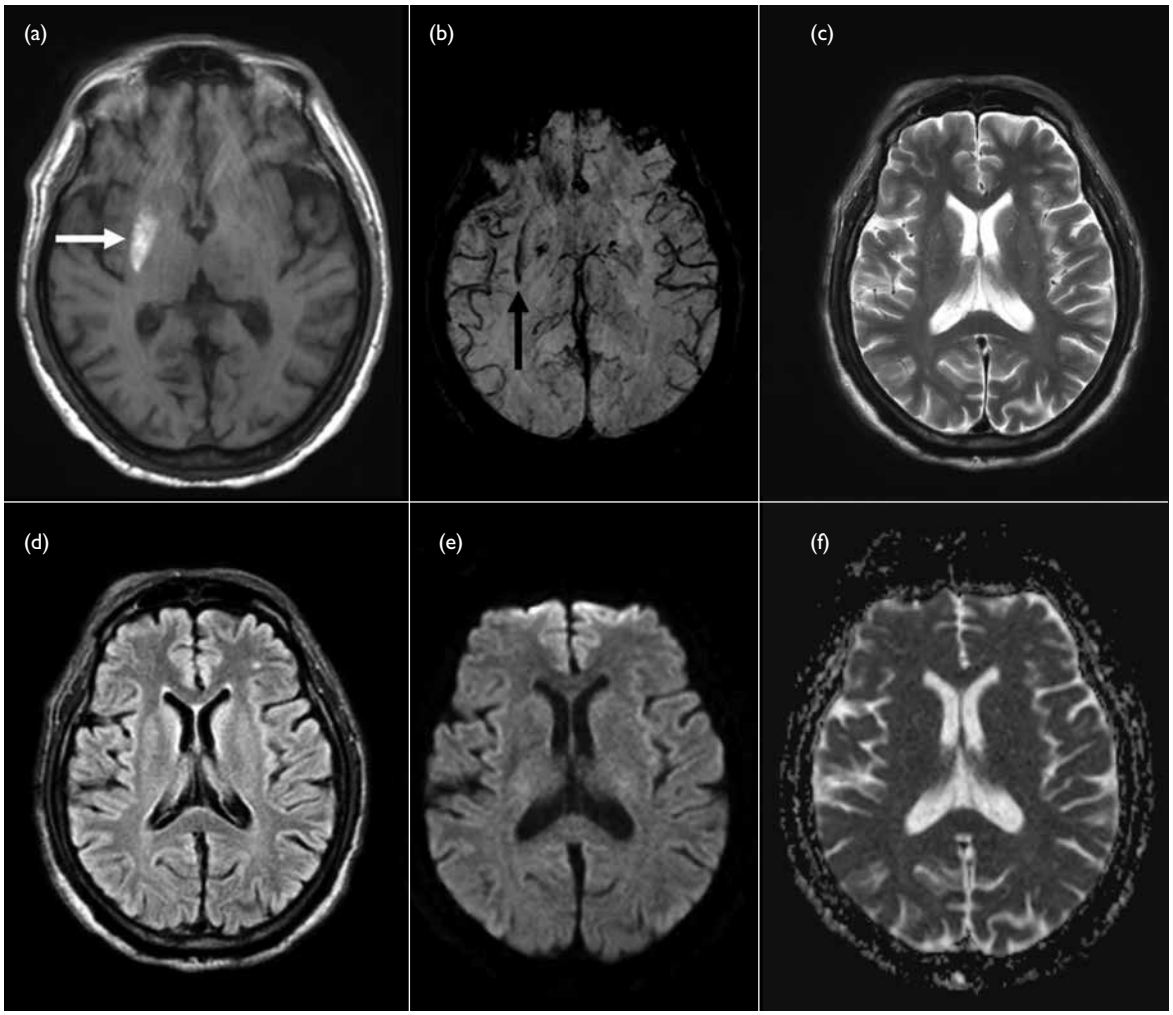


FIG 2. Case 1. Non-contrast magnetic resonance imaging of the brain with non-ketotic hyperglycaemic hemichorea. (a) T1-weighted sequence showing hyperintense signal in right putamen (white arrow). (b) Susceptibility weighted imaging showing rim of susceptibility signal in right putamen (black arrow). (c) T2-weighted sequence and (d) T2-weighted fluid-attenuated inversion recovery sequence showing symmetrical signals in bilateral basal ganglia. (e) Diffusion-weighted imaging and (f) apparent diffusion coefficient map showing no restricted diffusion

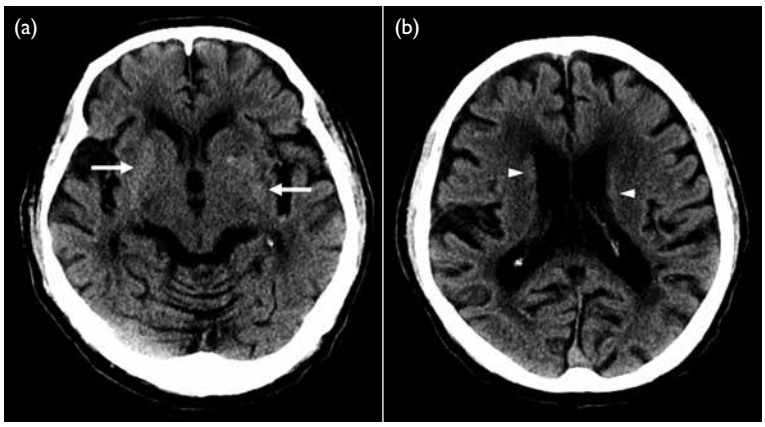


FIG 3. Case 2. Non-contrast computed tomography of the brain showing asymmetric subtle hyperdensity in bilateral putamen (a) [arrows] and bilateral caudate nuclei (b) [arrowheads], more extensive on the right, without mass effect or surrounding oedema, in keeping with non-ketotic hyperglycaemic hemichorea

inversion recovery signals can be variable. Of note, restricted diffusion is not expected in NHH,[2-4] and this differentiates it from acute ischaemic stroke.

The pathophysiology of NHH is not fully understood. Proposed mechanisms include depleted gamma-aminobutyric acid and disrupted blood-brain barrier at the corpus striatum.[2-4] Recognising this rare complication of uncontrolled diabetes mellitus enables prompt medical intervention. Neurological symptoms of NHH usually show substantial improvement after normalisation of blood glucose level without additional intervention.[2-4]

## Author contributions

All authors contributed to the concept and design of the study, acquisition of data, analysis and interpretation of data, drafting of the manuscript, and critical revision of the manuscript for important intellectual content. All authors

 

had full access to the data, contributed to the study, approved the final version for publication, and take responsibility for its accuracy and integrity.

## Conflicts of interest

All authors have disclosed no conflicts of interest.


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## Ethics approval

The patients were treated in accordance with the Declaration of Helsinki. They provided informed consent for all treatments and procedures, and consent for publication.


**PL Lam *,** MB, BS
**PP Iu,** FRCR, FHKAM (Radiology)
**DHY Cho,** FRCR, FHKAM (Radiology)

*Department of Diagnostic and Interventional Radiology, Kwong Wah Hospital, Hong Kong SAR, China*

* Corresponding author: lpl404@ha.org.hk


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