


CT hyperdense lesion after head trauma: is it traumatic?

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Accepted 8 May 2022

DESCRIPTION

A woman in her 70s presented in our emergency department after a car accident with reported loss of consciousness on scene after the trauma. In our initial examination, she presented with retrograde amnesia to the event, elevated BP (194/108 mm Hg), tachycardia (109 beats per minute) and normal neurological examination. Laboratorial tests showed hyperglycaemia of 428 mg/dL, glycosuria and ketonuria. She had a medical history of hypertension, insulin-dependent diabetes mellitus, dyslipidaemia and multinodular goitre. Cataract surgery accounted for the only previous surgical procedure. She denied any pharmacological allergies. She had been prescribed glargine and glulisine insulin, but due to non-compliance, her glucose and glycated Hb levels were not controlled.

We ordered a head CT as part of our trauma protocol and found a noticeable hyperdense image in the right lenticular and caudate nuclei, confined to the grey matter, without enhancement after contrast (figure 1). To the untrained observer, this could be interpreted as a haemorrhage secondary to traumatic brain event. Yet, this finding is compatible with sequelae of severe hyperglycaemic profile. The lesion was found to have around 40–45 Hounsfield units. The most consistent and common imaging features of hyperglycaemic changes are CT hyperdensities or hyperintense basal ganglia signal on MRI T1 weighted images, without surrounding oedema or mass effect.¹ Haemorrhagic CT lesions following blunt trauma are usually in subdural, subarachnoid or intraparenchymal locations, and less commonly epidural, having Hounsfield units values of around 65–80. Thus, traumatic haemorrhage was ruled out. Professionals dealing with traumatised patients should be aware of this finding.

The patient was referred to immediate optimisation of antidiabetic therapy on the same day at the Emergency Department and afterwards continued long-term diabetes specialised consultations. As a result, glycated Hb levels decreased from 14.6% to 7.8%. Regarding neurological manifestations, she remains asymptomatic with no signs of either hemichorea or hemiballismus. A comparison head CT was performed after stabilisation of glycated Hb levels, 18 months after the accident, and the hyperdense image was no longer detected, while encephalomalacia was observed instead (figure 2).

Cases reported in the literature with basal ganglia CT hyperdensities usually describe patients with severe ketotic or non-ketotic hyperglycaemic and contralateral hemiballismus and/or hemichorea.^{2–4} There are also other cases in which the patient

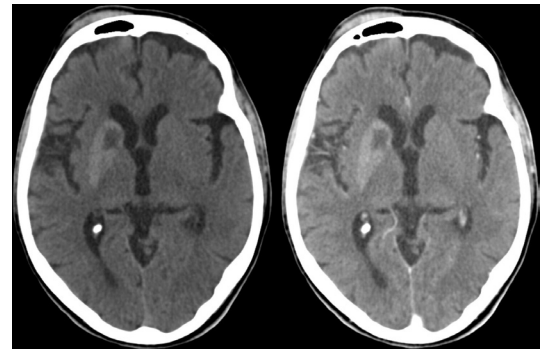


Figure 1 CT images showing a hyperdense lesion in the right lenticular and caudate nuclei, confined to the grey matter, without enhancement after contrast. Image in the left: before contrast administration. Image in the right: after contrast.

presents atypically, with loss of consciousness.⁵ The pathophysiological mechanism underlying these imaging abnormalities is not yet clear. Some investigators postulate it is due to hyperglycaemia-induced hyperviscosity and vasogenic oedema,⁶ other describe hyperviscosity-induced partial ischaemic injury,⁷ while others hypothesise an excess



Figure 2 CT image 18 months after the accident, showing absence of basal ganglia hyperdensity found at the initial admission.



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To cite: Gomes J, Germanova L, Monteiro AR, et al. *BMJ Case Rep* 2022;**15**:e249586. doi:10.1136/bcr-2022-249586

Images in...

formation of gemistocytes, reactive swollen forms of astrocytes, which result from ischaemic changes and metabolic derangement.⁸ Movement disorders may as well be a manifestation of depletion of gamma-aminobutyric acid and acetylcholine neurotransmitters in the basal ganglia, mainly the putamen, as brain reverts to anaerobic pathways in hyperglycaemic states, depressing Krebs cycle.¹ No consensus has been reached so far. In this case, our patient did not present any movement or neurological disorder prior or after the accident.

The basal ganglia are highly metabolically active, and one should hypothesise other possible non-traumatic aetiologies for CT hyperdensities. Most of these are bilateral and examples are Wilson's disease, neurodegenerative disease, poisoning, Tay-Sachs disease, drug consumption or ischaemic events. Regarding haemorrhage and tumour, which also present with unilateral abnormality, differentiation can be made as the hyperdense area in hyperglycaemia has lower Hounsfield units values and conforms to the shape of basal ganglia sparing the internal capsule.⁹

Concerning the disappearance of these imaging findings, the cases reported in the literature do not mention a time interval

in which it occurs. In our case, the hyperdense lesion evolved to an encephalomalacia area, which can hypothetically be related to a stroke (one of the possible pathophysiological mechanisms previously described). As a principle, follow-up with internal medicine or neurology consultations is advised, and neuropathological processes should be studied accordingly, outside the acute setting.

Contributors Dr JG—main author. On duty surgeon who observed the patient on the ED. Dr LG—literature review. Article writing. Dr ARM—article writing. Dr MT—images edition.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent for publication Consent obtained directly from patient(s).

Provenance and peer review Not commissioned; externally peer reviewed.

Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

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Learning points

- ▶ Hyperdense lesions on head CT after trauma are usually a result of intracranial bleeding, namely cerebral contusion, epidural, subdural or subarachnoid haemorrhage.
- ▶ When these are observed in the basal ganglia, intracranial haemorrhage might not be correct diagnosis. Other pathologies, mostly metabolic, such as sustained severe hyperglycaemia, should be ruled out.
- ▶ Basal ganglia CT/MRI hyperdense images with movement disorders (hemiballismus and/or hemichorea) are rare in diabetic patients, but they can be present in those with very poor glycaemic control.
- ▶ We report a case of an asymptomatic diabetic patient with the above-mentioned abnormal CT finding which disappeared in a control CT 18 months afterwards, when glycaemic values were already under control.

REFERENCES

- 1 Jagota P, Bhidayasiri R, Lang AE. Movement disorders in patients with diabetes mellitus. *J Neurol Sci* 2012;314:5–11.
- 2 Ilic A, Semnic M, Stefanovic D, et al. Chorea hyperglycemia basal ganglia syndrome—a rare case of bilateral Chorea-Ballismus in acute non-ketotic hyperglycemia. *Ann Indian Acad Neurol* 2021;24:824–6.
- 3 Marques JS, Monteiro N, Nunes A. Hyperglycaemic hemichorea. *Eur J Case Rep Intern Med* 2018;5.
- 4 Bathla G, Policeni B, Agarwal A. Neuroimaging in patients with abnormal blood glucose levels. *AJNR Am J Neuroradiol* 2014;35:833–40.
- 5 Choi JY, Park JM, Kim KH, et al. Radiographic basal ganglia abnormalities secondary to nonketotic hyperglycemia with unusual clinical features. *Clin Exp Emerg Med* 2016;3:252–5.
- 6 Chu K, Kang D-W, Kim D-E, et al. Diffusion-weighted and gradient echo magnetic resonance findings of hemichorea-hemiballismus associated with diabetic hyperglycemia: a hyperviscosity syndrome? *Arch Neurol* 2002;59:448–52.
- 7 Ohara S, Nakagawa S, Tabata K, et al. Hemiballismus with hyperglycemia and striatal T1-MRI hyperintensity: an autopsy report. *Movement Disorders* 2001;16:521–5.
- 8 Shan DE, Ho DM, Chang C, et al. Hemichorea-hemiballismus: an explanation for Mr signal changes. *AJNR Am J Neuroradiol* 1998;19:863–70.
- 9 Johari B, Hanafiah M, Shahizon AMM, et al. Unilateral striatal CT and MRI changes secondary to non-ketotic hyperglycaemia. *BMJ Case Rep* 2014;2014:bcr2014204053.

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