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Basal ganglia T1W hyperintensity in non-ketotic hyperglycemic hemichorea

T1W hyperintenzita bazálních ganglií u neketotické hyperglykemické hemichorey

Dear Editor,

Non-ketotic hyperglycemic hemichorea (NHH), or diabetic striatopathy, is a rare complication of poorly controlled diabetes, predominantly in elderly females [1,2]. It presents as unilateral, involuntary upper limb movements in chronic hyperglycemia. NHH is linked to T1-weighted (T1W) hyperintensity in the basal ganglia, reflecting metabolic rather than structural changes. Given its overlap with neurovascular and metabolic disorders, clinical-radiologic correlation is crucial. We report two cases highlighting the importance of integrating clinical and imaging findings for prompt diagnosis, discussing differential diagnoses and the role of multidisciplinary collaboration.

A 75-year-old female with a history of arterial hypertension and diabetes mellitus presented to the emergency department with involuntary, jerking movements of the right arm, persisting for five days. According to family members, speech had slowed, but no additional active complaints were noted. Medical history included a total abdominal hysterectomy and bilateral salpingo-oophorectomy. Physical examination yielded unremarkable findings. Laboratory results showed a blood glucose level of 285 mg/dL (15.8 mmol/L) and an HbA_{1c} of 16.1%, with other parameters within normal limits. Cranial CT obtained at the time of emergency department admission (5 days after symptom onset) revealed hyperdensity in the left caudate nucleus and putamen (Fig. 1A). MRI performed on the same day showed T1W hyperintensity and slight hypointensity on T2-weighted (T2W) and fluid attenuated inversion recovery (FLAIR) images in the same regions, with no diffusion restriction observed on diffusion-weighted imaging (DWI). Susceptibility-weighted ımaging (SWI) showed no artifacts suggestive of haematoma. Findings were consistent with a diagnosis of NHH (Fig. 1 B–F). For blood glucose management, 10 units of crystalline insulin were initially administered, followed by an infusion of 30 units of crystalline insulin in 150 cm^3 saline at 30 cm^3/h. Additionally, haloperidol was administered at 3× 10 drops daily. Blood glucose levels returned to normal, with significant improvement in hyperkinetic symptoms. Insulin doses were adjusted, and discharge followed with stable glucose levels and controlled symptoms. Follow-up imaging conducted 34 days after the initial scans, including both CT and MRI on the same day, showed minimal medial regression of hyperdensity on CT and hyperintensity on T1W MRI (Fig. 1 G–H).

A 71-year-old female patient with a history of diabetes and hyperlipidemia presented to the internal medicine outpatient clinic with complaints of involuntary movements in her left arm that had persisted for one week. It was noted that the patient had been advised to initiate insulin therapy for a long time; however, despite being informed about the risks, she refused to start using insulin. Laboratory tests revealed a postprandial blood glucose level of 330 mg/dL (18.3 mmol/L), HbA_{1c} of 11.8%, cholesterol of 247 mg/dL (13.7 mmol/L), low density lipoprotein of 154 mg/dL (8.6 mmol/L), and triglycerides of 231 mg/dL (12.8 mmol/L), while other blood parameters were within normal limits. Neurology consultation was requested, and examination revealed hemiballism on the left side, with no other abnormal neurological findings. Cranial CT, performed upon presentation to the emergency department, demonstrated hyperdensity in the right caudate nucleus and lentiform nucleus (Fig. 2A). Cranial MRI, also conducted during the initial evaluation, revealed T1W hyperintensity and faint hypointensity on T2W and FLAIR images in these regions, with no diffusion restriction observed (Fig. 2 B–F).

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The patient was diagnosed with NHH due to uncontrolled diabetes and hemiballism symptoms. Following insulin therapy and blood glucose control (blood glucose level: 118 mg/dL [6.5 mmol/L]), the patient experienced significant symptom improvement. CT performed 77 days after the initial imaging demonstrated complete regression, whereas T1W MRI obtained on the same day showed no evidence of regression (Fig. 2 G–H).

The pathophysiology of diabetic NHH is multifactorial, involving metabolic, vascular, and cellular mechanisms. Hyperglycemia shifts glucose metabolism to an anaerobic pathway, depleting gamma-aminobutyric acid (GABA) and acetate in the basal ganglia. This disrupts medium-sized spiny neurons (MSNs), leading to excitatory disinhibition of

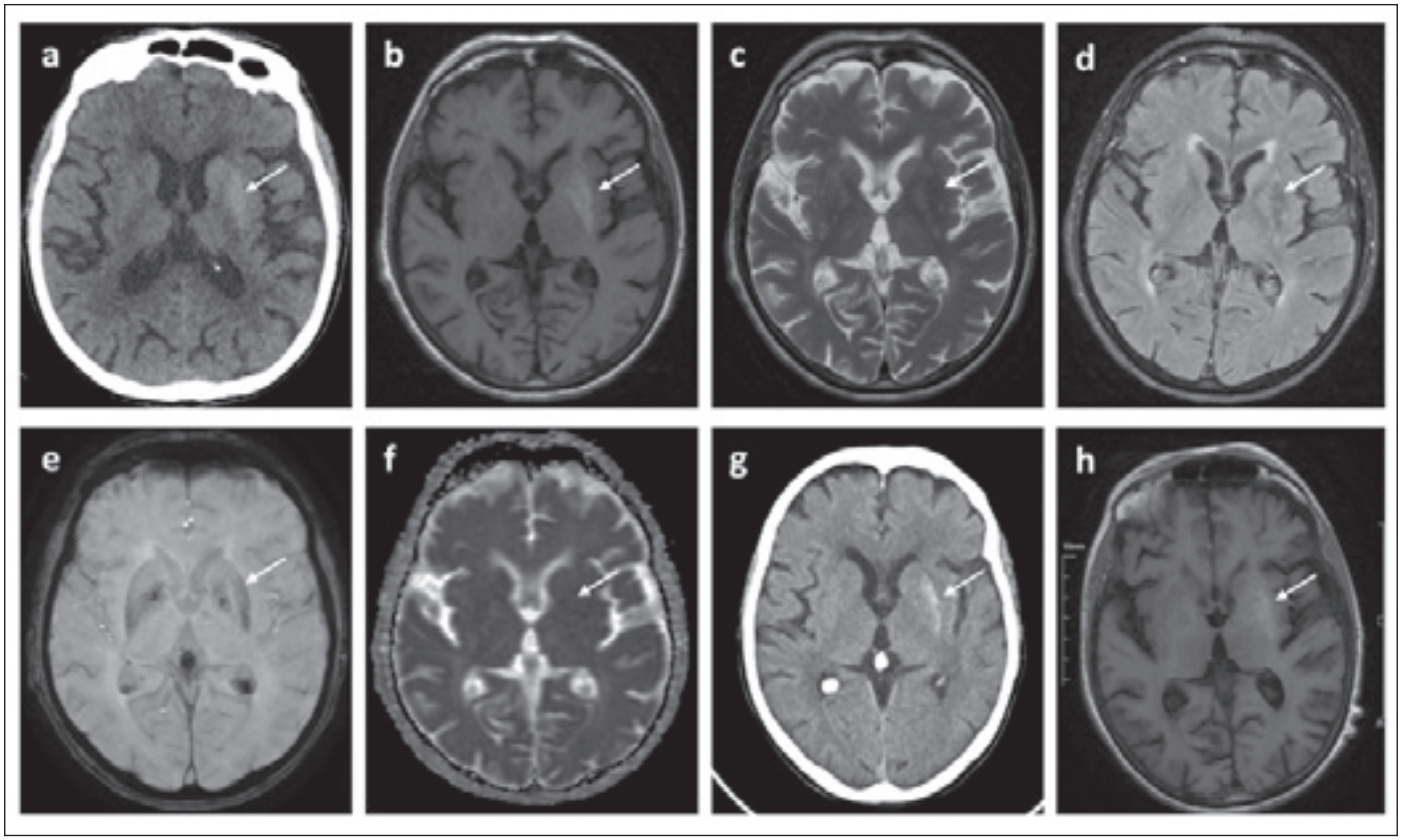

Fig. 1. Initial and follow-up imaging findings consistent with NHH in a 75-year-old female patient presenting with hemiballism in the right arm.
(A) In CT, hyperdensity is noted in the left caudate nucleus and putamen (arrow). On MRI (B), T1W imaging shows hyperintensity in the left putamen. (C) In T2W and (D) FLAIR sequences, slight hypointensity is observed relative to the contralateral side. (E) The SWI sequence shows no signal suggestive of hemorrhage, and (F) the ADC map demonstrates no signal changes indicative of ischemia. These findings in a diabetic patient support a diagnosis of NHH.

The follow-up CT scan obtained 34 days after the initial imaging (G) shows hyperdensity with minimal medial regression. Similarly, the follow-up T1W MRI obtained on the same day (H) shows hyperintensity with minimal medial regression. Lesion and follow-up changes are indicated by arrows.

ADC – apparent diffusion coefficient; FLAIR – fluid attenuated inversion recovery; NHH – non-ketotic hyperglycemic hemichorea; SWI – susceptibility-weighted imaging

Obr. 1. Vstupní a kontrolní nálezy na snímcích u 75leté pacientky s hemibalizmem v pravé paži odpovídající NHH.
(A) Na CT je patrná hyperdenzita v levém nucleus caudatus a putamen (šipka). Na MR je při (B) T1W zobrazení patrná hyperintenzita v levém putamen. (C) V T2W a (D) FLAIR sekvencích je pozorována mírná hypointenzita vzhledem ke kontralaterální straně. (E) SWI sekvence nevykazuje žádný signál svědčící o krvácení a (F) ADC mapa neprokazuje žádné změny signálu svědčící o ischemii. Tyto nálezy u diabetického pacienta podporují diagnózu NHH.

Kontrolní CT vyšetření získané 34 dní po prvním zobrazení (G) ukazuje hyperdenzitu s minimální mediální regresí. Podobně kontrolní T1W MR získaná ve stejný den (H) vykazuje hyperintenzitu s minimální mediální regresí. Léze a následné změny jsou označeny šipkami.

ADC – apparent diffusion coefficient; FLAIR – fluid attenuated inversion recovery; NHH – neketotická hyperglykemická hemichorea; SWI – susceptibilně váhované zobrazení

motor pathways and resulting in chorea or ballism. Vascular factors include striatal ischemic hypoperfusion due to diabetic vasculopathy and increased blood viscosity from hyperosmolarity and dehydration, further impairing GABAergic transmission [1]. Histopathological findings, such as astrocytosis, macrophage infiltration, microbleeding, and hemosiderin deposits, indicate inflammatory and reparative processes. The transient nature of striatal hyperintensity on T1W MRI and hyperdensity on CT suggests reversible cellular and metabolic changes rather than infarction. However, variability in clinical presentation, including delayed symptom onset after glycemic correction, highlights the need for further research into these mechanisms.

Ischemic stroke, hemorrhage, basal ganglia lesions, metabolic disorders, infections, toxins, Parkinson's disease, paraneoplastic syndrome, and dopamine antagonists can cause hemiballism. Clinical examination, imaging, and laboratory findings are key to differentiation [3].

In unilateral basal ganglia T1W hyperintensity, infarction and hemorrhage should be considered; tumors such as melanoma metastases are rare. NHH can present with unilateral or bilateral T1W hyperintensity, typically preserving the internal capsule, unlike hypertensive hematomas or other basal ganglia pathologies.

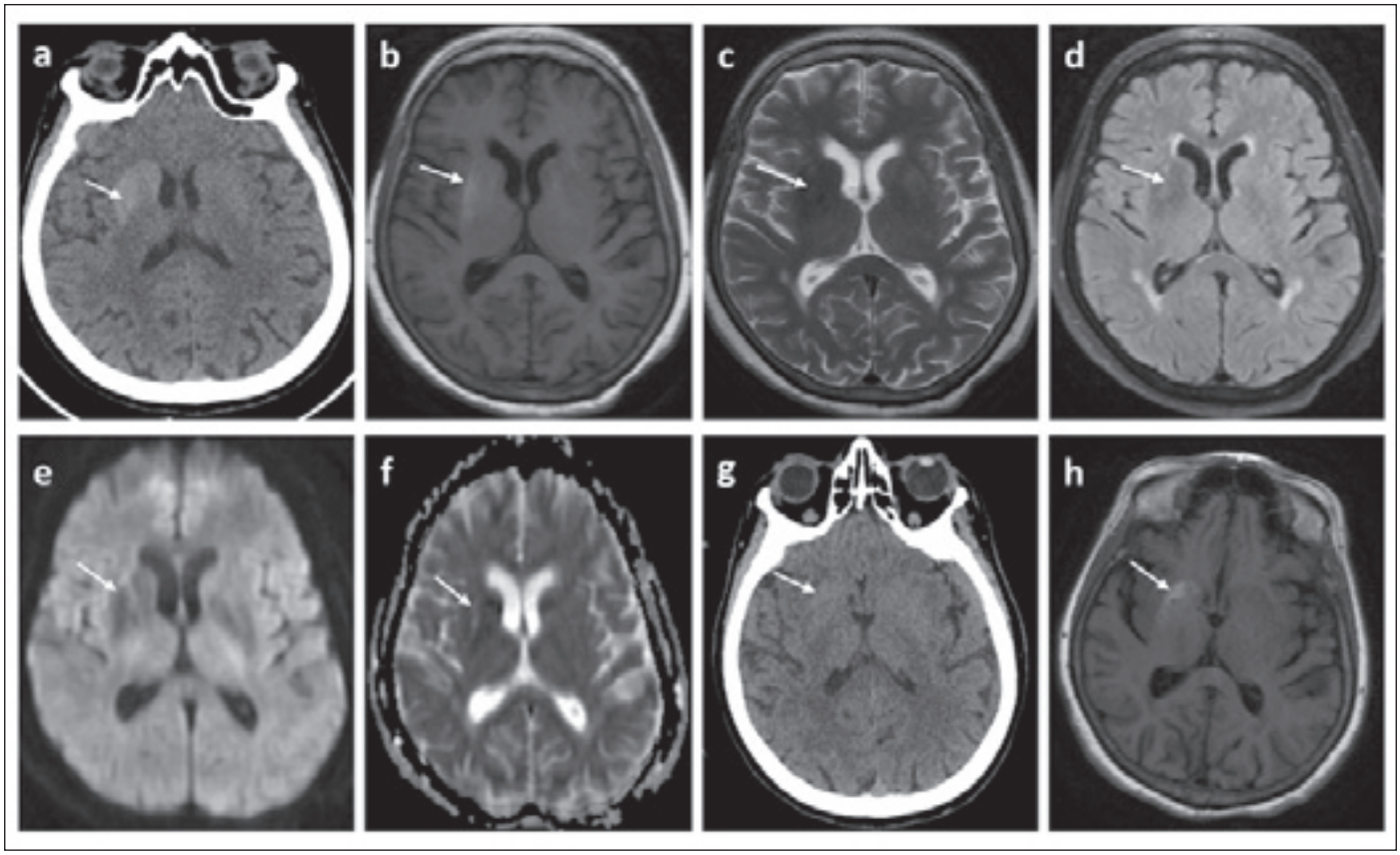

Fig. 2. Initial and follow-up imaging findings consistent with NHH in a 71-year-old female patient presenting with involuntary movements in the left arm.

(A) In CT, hyperdensity is observed in the right caudate nucleus and lentiform nucleus, with preservation of the internal capsule. On MRI (B), T1W imaging demonstrates hyperintensity in the right putamen. (C) T2W and (D) FLAIR sequences show slight hypointensity. (E) In DWI and (F) the ADC map, slight hypointensity is seen relative to the contralateral side, without evidence of diffusion restriction.

The follow-up CT scan obtained 77 days after the initial imaging (G) shows complete regression, while the follow-up T1W MRI obtained on the same day (H) shows no evidence of regression. Lesion and follow-up changes are indicated by arrows.

ADC – apparent diffusion coefficient; DWI – diffusion weighted imaging; FLAIR – fluid attenuated inversion recovery; NHH – non-ketotic hyperglycemic hemichorea

Obr. 2. Vstupní a kontrolní nálezy na snímcích u 71leté pacientky s mimovolními pohyby v levé paži odpovídající NHH.

(A) Na CT je patrná hyperdenzita v pravém nucleus caudatus a v nucleus lentiformis se zachováním capsula interna. Na MR prokazuje (B) T1W zobrazení hyperintenzitu v pravém putamen. (C) Sekvence T2W a (D) FLAIR ukazují mírnou hypointenzitu. (E) V DWI a (F) v ADC mapě je patrná mírná hypointenzita vzhledem ke kontralaterální straně, bez známek omezení difuze.

Kontrolní CT vyšetření provedené 77 dní po prvním zobrazení (G) ukazuje úplnou regresi, zatímco kontrolní T1W MR provedené ve stejný den (H) nevykazuje žádné známky regrese. Léze a následné změny jsou označeny šipkami.

ADC – apparent diffusion coefficient; DWI – difuzně vážený obraz; FLAIR – fluid attenuated inversion recovery; NHH – neketotická hyperglykemická hemichorea

Bilateral basal ganglia T1W hyperintensity may occur in calcification, genetic disorders (Fahr's syndrome), metabolic diseases (Wilson's, acquired hepatocerebral degeneration), or toxic exposure (manganese toxicity).

A thorough history should assess diabetes, liver disease, or heavy metal exposure, with serum levels of copper, ceruloplasmin, and manganese measured. CT detects calcifications, while MRI (SWI/T2*-gradient echo [GRE]) excludes hematoma [1,4]. These sequences help differentiate striatal hypointensity, suggesting petechial hemorrhage or paramagnetic accumulation (iron/manganese). However, striatal hypointensity remains controversial, as some studies show no SWI/T2*-GRE abnormalities, making it an unreliable NHH criterion. DWI findings also vary; some report restricted diffusion in the putamen, possibly reflecting acute putaminal dysfunction due to hyperglycemic or hyperosmolar effects [5,6].

In Case 2, follow-up imaging at 77 days showed complete resolution of CT hyperdensity, while T1W MRI hyperintensity persisted. This aligns with literature reports, where CT hyperdensities resolve faster than T1W abnormalities. Chua et al. found median CT resolution at 60 days, while T1W changes can persist up to 180 days [2]. This suggests that prolonged T1W visibility may reflect gliosis or residual metabolic disruption despite CT regression.

Non-ketotic hyperglycemic hemichorea is a reversible complication of uncontrolled diabetes, presenting in outpatient or emergency settings. A multidisciplinary approach ensures prompt diagnosis and treatment. Effective clinician-radiologist communica-

tion is key, as T1W hyperintensity in the basal ganglia has a broad differential. Integrating clinical and imaging findings is essential, and further research is needed to refine diagnostic criteria.

Conflict of interest

The authors declare they have no potential conflicts of interest concerning drugs, products, or services used in the study.

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