



Postmenopausal Female with Unilateral Involuntary Movements *Tek Yanlı İstemsiz Hareketleri Olan Postmenapozal Hasta*

• Venkatraman Indiran, • Amish Aggarwal, • T. Ramachandra Prasad

Department of Radiodiagnosis, Sree Balaji Medical College and Hospital, Tamil Nadu, India

Keywords: Hemiballismus, hemichorea, hyperglycemia
Anahtar Kelimeler: Hemiballismus, hemikore, hiperglisemi

Dear Editor,

A 52-year-old postmenopausal female was brought to our hospital with a history of gradually progressing involuntary movements of the right upper and lower limb for the last ten days. The movements did not involve the head, neck, left upper or lower limb. There was no loss of consciousness or altered sensorium. There was no limb weakness, sensory deficit or slurring of speech. There was no fever or neck stiffness. She was a known diabetic on oral medications for the last two years. Her fasting blood sugar was 143 mg/dL, and postprandial blood sugar was 184 mg/dL. Her glycosylated hemoglobin (HbA1c) was 10.3%. A urine specimen was negative for ketones. Her calcium level was 8.5 mg/dL (normal, 8.4-10.2 mg/dL). On examination, chorea involving the right arm, forearm, hand, leg, and foot was seen. Tone and power of the limb muscles were normal. Deep tendon reflexes were normal with bilateral plantar flexor response. Brain magnetic resonance imaging (MRI) of the patient showed T1 hyperintense signal and mild T2 and gradient recalled hypointense echo signal in the left basal ganglia region (Figure 1, 2). Diagnosis of hemiballism-hemichorea caused by nonketotic hyperglycemia (NKH) was made. This patient was treated with glimepiride (1 mg bid), metformin (500 mg bid), trihexyphenidyl (2 mg tid) and lorazepam (0.5 mg bid). The involuntary movements subsided gradually over two days, following which she was discharged.

Bedwell reported the first case of hemiballism-hemichorea due to nonketotic hyperglycemia in 1960 (1). NKH, usually observed in patients with type 2 diabetes mellitus, has been associated with different neurologic manifestations such

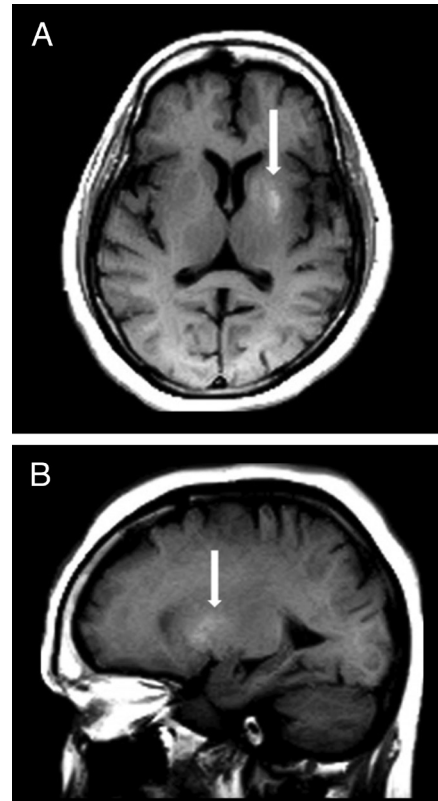


Figure 1. Axial (A) and sagittal (B) T1-weighted images showing hyperintense signal in the left basal ganglia region (white arrows)

Address for Correspondence/Yazışma Adresi: Venkatraman Indiran MD, Department of Radiodiagnosis, Sree Balaji Medical College and Hospital, Tamil Nadu, India
Phone: +04422653429 E-mail: ivraman31@gmail.com ORCID ID: orcid.org/0000-0001-5296-0175

Received/Geliş Tarihi: 05.10.2017 **Accepted/Kabul Tarihi:** 30.11.2017

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Turkish Journal of Neurology published by Galenos Publishing House.

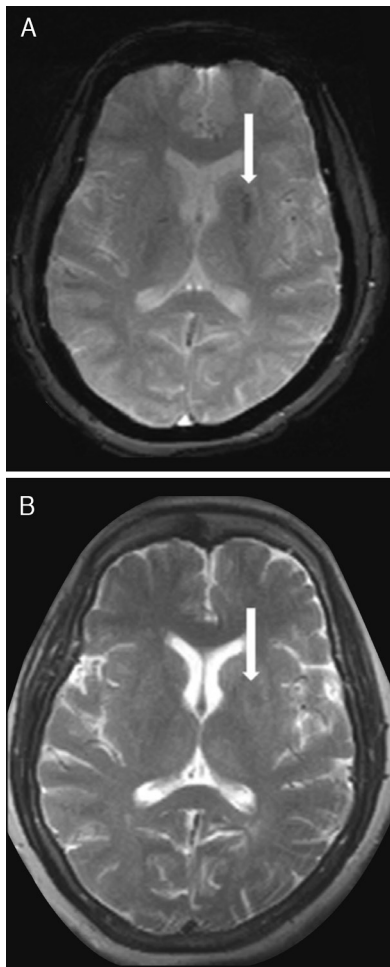


Figure 2. Axial gradient recalled echo image (A) and T2 weighted image (B) showing mild hypointense signal in the left basal ganglia region (white arrows)

as delirium, seizures, hemiballism-hemichorea, dysphagia, hemianopsia, hemiparesis, and hemisensory loss (2). Depletion of the inhibitory neurotransmitter gamma-aminobutyric acid, which is metabolized in the brain as an energy source in NKH, in the basal ganglia is considered the probable cause for hemiballism-hemichorea (3).

Patients with NKH with hemiballism-hemichorea show hyperdensity on CT and hyperintense signal on T1-weighted MRI

in the contralateral basal ganglia (4). Some patients may show subcortical T2 hypointensity (3). The basal ganglia signal change may be unilateral or bilateral. Possible hypotheses for putaminal T1 hyperintensity include a protein hydration layer in the swollen gemistocytes, putaminal petechial hemorrhage, demyelination, transient ischemic changes, and localized Wallerian degeneration (5). Differential diagnoses for bilateral (sometimes unilateral) basal ganglia T1 hyperintensity include manganese toxicity in prolonged parenteral nutrition, chronic liver disease, hypoxic-ischemic changes, disorders of calcium metabolism such as hypo- or hyperparathyroidism, Fahr disease, neurofibromatosis, and Wilson disease (5).

Knowledge of the neurologic and radiologic abnormalities seen with NKH is important for emergency physicians because prompt correction of the underlying hyperglycemia usually leads to quick improvement.

Ethics

Informed Consent: Consent form was filled out by all participants.

Peer-review: Internally peer-reviewed.

Authorship Contributions

Concept: VI., Design: VI., T.R.P., Data Collection or Processing: VI., A.A., Analysis or Interpretation: VI., T.R.P., Literature Search: VI., T.R.P., Writing: VI., A.A.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

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