

Diabetic Patient with Involuntary Movements

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A 60-year-old diabetic patient came to the emergency department with complaints of sudden-onset of involuntary movements of the right side of the body. The patient was diagnosed to have diabetes mellitus (type II) five years back and she had been taking oral hypoglycaemic drugs irregularly. Her random blood sugar level was 492 mg%, with a serum osmolality of 297mOsm/kg. Her urine sample showed no ketones. Other routine blood investigations including the sodium, potassium, calcium, magnesium, and phosphate concentrations were within the normal limits. Her liver and renal function tests gave normal results. There was no history of any drug intake or similar episodes in the past. There was no other significant personal or family history. Unenhanced computed tomography (CT) scan of the brain was done on admission, which revealed hyperdensity in the bilateral putamina and the caudate nuclei, more on the left side [Table/Fig 1]. No other significant intracranial abnormality was seen.

A diagnosis of diabetic non-ketotic hyperglycaemia (DNKHG) was made. The patient was started on insulin injections and supportive therapy for the involuntary movements. Marked symptomatic improvement was noted after the restoration of the blood glucose levels. The follow up CT scan showed a significant reduction of the bilateral basal ganglia hyperdensity [Table/Fig 2].



[Table/Fig-1]: Nonenhanced axial CT scan of brain taken during patient's admission shows hyperdensity in bilateral putamina (white arrows) and caudate nuclei (black arrows), more on left side.



[Table/Fig-2]: Follow up CT scan shows significant reduction in bilateral basal ganglia hyperdensity.

DNKHG can present clinically as chorea-ballismus, unilateral or bilateral. Radiologically, changes are seen in the contralateral basal ganglia. The characteristic imaging findings include hyperdense basal ganglia on the CT scan, with no post contrast enhancement. Magnetic resonance imaging (MRI) reveals hyperintensity on the corresponding T1 weighted images and hypo or hyperintensity on the T2 weighted/ FLAIR images [1-5]. The exact underlying mechanism responsible these radiological changes has been debated by many authors and it still remains unclear. Various proposed causes include ischaemia, petechial haemorrhage/ microcalcifications, hyperviscosity, myelin destruction or acute basal ganglia dysfunction [1,2,4,5].

As this condition is easily treatable, an early recognition of the imaging findings is important in patients who present with involuntary movements and hyperglycaemia.

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