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DIABETIC STRIATOPATHY

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INTRODUCTION:

Diabetic striatopathy (DS) is a rare medical condition resulting from poorly controlled diabetes mellitus (DM), characterized by non-ketotic hyperglycemia associated with choreo-ballistic movement and/ or reversible characteristic basal ganglia abnormalities on brain imaging. The prevalence has been reported to be 1 in 100,000 and it is more prevalent in older diabetic Asian women with poor glycaemic control[1]. We would like to report a case presented to our emergency department (ED) for its rarity.

CASE DESCRIPTION:

The patient is a 69-year-old housewife with underlying DM and hypertension whom defaulted her follow-up. She presented to ED with the first seizure episode of her life with the semiology of bilateral upper and lower limbs stiffening and up-rolling of eyeballs. She was noted to be unwell by her family for the past 7 days with lethargy. Upon arrival to ED, she was hypertensive with a Glasgow Coma Scale (GCS) of E1V1M1. She developed another episode of seizure during physical examination which was aborted with intravenous (IV) diazepam and subsequently loaded with IV phenytoin. Her blood sugar was noted to be high (33 mmol/L) and serum ketone was not raised. Computed Tomography (CT) scan of her brain revealed hyperdensity over left basal ganglia which was initially mistaken as a bleed. She was admitted for glycaemic control and she was seizure-free since then and discharged well after 4 days.

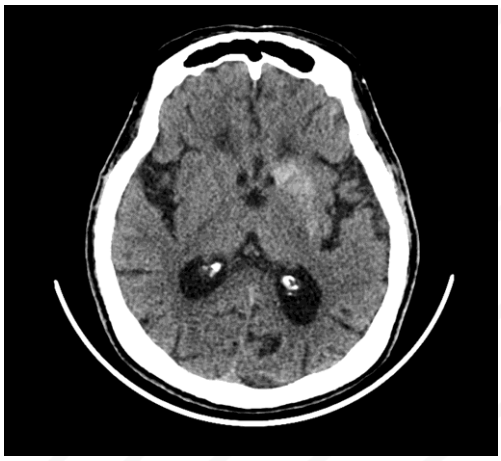

Figure 1. Plain CT brain of the patient showed hyperdensity over left basal ganglia

CONCLUSION:

Diabetic striatopathy is a rare complication of DM, due to a combination of different pathogenesis. It carries good prognosis if detected early, and the lesions are reversible with good glycemic control.

DISCUSSION:

Although the majority of DS manifested with unilateral dyskinesia, other rare presentations that were reported include altered consciousness, seizure, limb weakness, dysarthria and dysphagia[2]. Our patient presented with seizure without any dyskinesia. The pathophysiology is unclear, but several hypotheses to explain the imaging abnormalities include petechial haemorrhage, mineral deposition, myelin destruction and infarction with astrocytosis[2]. Glycaemic control with proper hydration to correct the underlying metabolic imbalance remain the mainstay of treatment[2, 3].

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