AJNS

LETTER TO EDITOR

Bilateral diabetic striatopathy

Sir,

A 79-year-old man with a history of type 2 diabetes mellitus (DM) presented with altered sensorium and bilateral chorea-ballismus. Neurological examination was otherwise unremarkable. The blood sugar level during fasting was 540 mg/dL and serum osmolarity was 360mOsm/L with absent ketones. Plain Computed Tomography (CT) scan of the brain revealed bilateral hyperdense basal ganglia [Figure 1a]. Magnetic Resonance Imaging (MRI) revealed hyperintensity in both the basal ganglia on T1W and T2W images [Figure 1b and c]. An area of hypointensity in the left globus pallidus corresponded to petechial hemorrhage on the gradient sequence [Figure 1d]. The chorea resolved within 2 days of euglycemia on insulin therapy. On 6 months of follow-up, MR images showed decreased signal intensity in both the basal ganglia.

The term "diabetic striatopathy" is characterized by the presence of a high signal on MRI confined to the striatum with contralateral movement disorder. It is commonly associated with type 2 DM

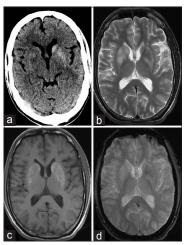


Figure 1: Axial plain CT scan of the brain shows bilateral hyperdense basal ganglia (left > right). (a) Axial T2- and T1-weighted MRI images; (b and c, respectively) Reveal increased signal intensity in the basal ganglia bilaterally. An area of decreased signal intensity seen in the left globus pallidus on T2W and GRE (gradient echo sequence) (d) Images corresponds to petechial hemorrhages

and rarely seen in type 1 DM. Most patients at presentation have a clinical picture consistent with a diagnosis of non-ketotic hyperglycemia^[1]. Rarely, patients have bilateral lesions with bilateral chorea. Although the actual pathophysiology is unknown, the underlying chronic focal cerebrovascular disease in DM may be responsible for an acute blood–brain barrier dysfunction. Moreover, the decrease in striatal blood flow causes depletion of gamma-aminobutyric acid (GABA) with resultant dyskinesia^[2]. Striatal hyperintensity on CT and MRI images in the acute stage of non-ketotic hyperglycemia helps in the early diagnosis and initiation of treatment^[3]. The chorea-ballismus is completely reversible. Neuroimaging findings may return to normal or persist after clinical recovery ^[4].

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References

- Abe Y, Yamamoto T, Soeda T, Kumagai T, Tanno Y, Kubo J, et al. Diabetic striatal disease: clinical presentation, neuroimaging, and pathology. Intern Med 2009;48:1135-41.
- Lai PH, Tien RD, Chang MH, Teng MM, Yang CF, Pan HB, et al. Chorea-ballismus with nonketotic hyperglycemia in primary diabetes mellitus. AJNR Am J Neuroradiol 1996;17:1057-64.
- Lin JJ, Lin GY, Shih C, Shen WC. Presentation of striatal hyperintensity on T1-weighted MRI in patients with hemiballism-hemichorea caused by non-ketotic hyperglycemia: Report of seven new cases and a review of literature. J Neurol 2001;248:750-5.
- Battisti C, Forte F, Rubenni E, Dotti MT, Bartali A, Gennari P, et al. Two cases of hemichorea-hemiballism with nonketotic hyperglycemia: A new point of view. Neurol Sci 2009;30:179-83.

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