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ORIGINAL ARTICLE

T2*-based MR imaging of hyperglycemia-induced hemichorea-hemiballism

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Summary
Introduction: Hyperglycemia can induce hemichorea-hemiballism, especially in elderly type II diabetics. CT and MRI findings include hyperdensity and T1-shortening in the contralateral lentiform nucleus, respectively. This study explores the associated imaging findings on T2*-based sequences.
Methods: Six patients with clinically documented hyperglycemia-induced hemichorea-hemiballism who had undergone MR imaging with a T2*-based sequence (T2* gradient echo or susceptibility-weighted imaging) were included in this retrospective case series.
Results: All six patients demonstrated T1-shortening contralateral to their hemichorea-hemiballism. T2*-based sequences demonstrated unilateral hypointense signal within the striatum in four patients. One patient had mild bilateral striatal hyperintensities, while another did not show significant signal changes.
Conclusion: It is important for the radiologist to be aware of the signal changes that can be seen on T2*-based sequences in hyperglycemia-induced hemochorea-hemiballism.

Introduction

Hemichorea-hemiballism refers to unilateral involuntary nonrhythmic movements. The most common etiology of hemichorea is infarction involving the basal ganglia, followed by hyperosmolar nonketotic hyperglycemia. The latter diagnosis is termed hyperglycemia-induced hemichorea-hemiballismus (HIHH) [1,2]. Differentiating HIHH from an infarction-related movement disorder may be difficult by clinical history or physical examination, especially because diabetes is a known risk factor for both entities. To further complicate the diagnosis, other diseases,

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such as Wilson's disease, systemic lupus erythematous, and thyrotoxicosis, may present with a similar constellation of symptoms.

The radiologist's role is paramount in arriving at the correct diagnosis. The classic finding on CT is unilateral hyperdensity within the basal ganglia contralateral to the side of the patient's clinical findings. MRI typically demonstrates T1-shortening approximately within the same region as the CT abnormalities [3]. The putamen is most often involved, although the globus pallidus, caudate, and subthalamic nucleus have also been reported [4,5]. Rarely, patients may present with bilateral clinical and imaging findings [6,7].

Although these imaging features are typical of HIHH, they may be seen in other metabolic and neurodegenerative disorders. The utilization of T2*-based MR sequences, such as T2*-gradient recalled echo (GRE) and susceptibility-weighted imaging (SWI), may offer additional clues to narrow the differential diagnosis. These sequences employ a form of imaging contrast distinct from conventional T1 and T2-weighted imaging, exploiting the dephasing effect of magnetic field inhomogeneities on transverse magnetization. To date, there have only been a handful of case reports about this topic in the literature. The purpose of our analysis is to describe the appearance of HIHH on T2*-GRE and SWI.

Methods

Our institutional review board (IRB) approved this retrospective study, and informed consent was waived. The patients included in the study were treated from 2014 to 2015. Individual medical records, including the hospital course and laboratory values, were reviewed.

Three patients (patients 2, 3, 4) underwent MR imaging on a 1.5T Toshiba Titan scanner, while two others were imaged with a 3T Philips Achieva scanner (patients 1, 5). These five patients underwent axial T1- and T2-weighted FSE, axial FLAIR, axial DWI, and coronal T2*-GRE sequences. A non-contrast CT of the head was obtained for three patients using a 256-slice Philips Brilliance iCT scanner (patients 1, 5) or a 128-slice Philips Ingenuity CT scanner (patient 3). One patient (patient 6) was imaged on a 1.5T Siemens Avanto scanner with axial T1- and T2-weighted, axial FLAIR, axial DWI, and axial SWI sequences (to include magnitude, phase, and minIP images).

Regions of interest (ROI) were drawn in the abnormal areas on the T2*-GRE/SWI images, as well as in the contralateral normal brain region(s) to serve as an internal reference. The ROIs (12 mm^2) were selected with correlation to the T1-weighted images obtained from the same acquisition. Contrast ratios (CR) of the ROIs were obtained using the following equation:

$$CR = (C_l - C_r)/C_r \quad (1)$$

where C_l represents the signal intensity within the abnormal ROI, and C_r refers to the signal intensity of the contralateral reference ROI. The CT and MR images were reviewed by two board-certified neuroradiologists (W.A. and A.S.).

Results

Patient 1

A 71-year-old diabetic man presented with right-sided choreiform movements for 2 weeks. He had a serum glucose level of 332 mg/dL, plasma osmolality of 308 mOsm/kg, and HbA1c of 15.7%. Trace ketones were present on urinalysis. NECT showed hyperdensity within the left lentiform nucleus (Fig. 1). T1-shortening was present within the left medial putamen. There was no diffusion restriction. Corresponding mild asymmetric hypointensity was seen on T2WI and T2*-GRE (CR = 0.05). His choreiform movements improved shortly after glucose control, and fully resolved within one week.

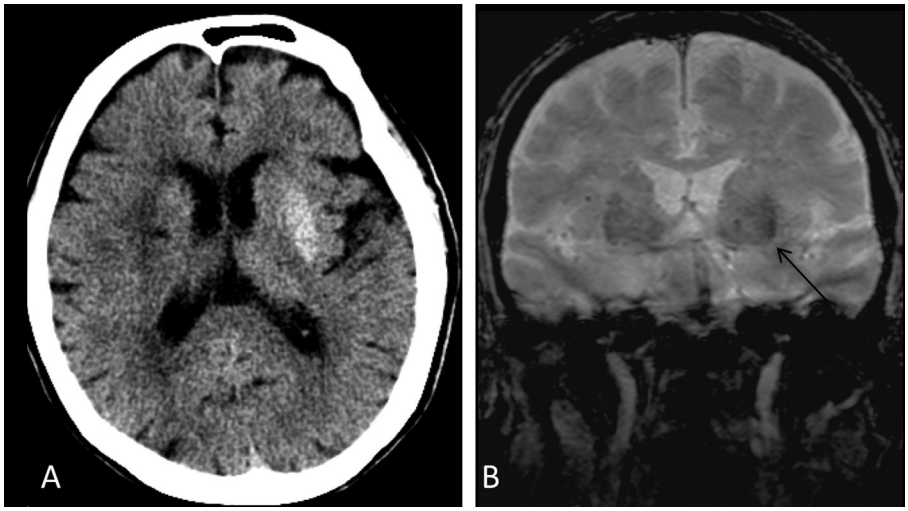

Figure 1 A 71-year old diabetic man with right-sided choreiform movements for 2 weeks. CT demonstrates hyperdensity within the left lentiform nucleus (A). There is mild hypointensity along the lateral margins of the left putamen on coronal T2*-GRE (B).