Parkinsonism and Related Disorders 64 (2019) 332–333

Contents lists available at ScienceDirect

Parkinsonism and Related Disorders

journal homepage: www.elsevier.com/locate/parkreldis

Correspondence

A need to apply unified criteria to choreoballism associated with glycemic derangement

ARTICLE INFO

Keywords:
Diabetes mellitus
Hyperglycemia
Chorea
Ballism
Choreoballism
Brain magnetic resonance imaging

Dear editor,

We read the interesting study by Ryan et al. about patients with hyperglycemic chorea/ballism [1]. The authors recruited patients using temporal and laboratory criteria: chorea/ballism within 30 days of hyperglycemia (blood glucose level [BGL] > 300 mg/dL).

In historic cases of chorea and glycemic derangement, both BGLs and blood glycated hemoglobin (HbA1c) levels were variable [2,3]. There were cases with hypoglycemia or euglycemia at the time of presentation, whose clinical features did not differ from those of hyperglycemia [2,3]. It could be due to failure in measuring BGLs at the time of symptom onset, rather than at the time of presentation. However, since chorea was commonly persistent despite meticulous BGL control, factors other than BGL/HbA1c could be the culprit, which needs further studies [2].

In the study by Ryan et al. the authors stated that they identified patients in whom chorea subacutely developed in association with a discreet episode of marked hyperglycemia. However, BGLs at the time of admission were still higher than 300mg/d, which suggest continuation of increased BGLs rather than discrete episodes of hyperglycemia. Moreover, in Case 5, chorea developed one month after the hyperglycemic episode, raising concerns about the temporal correlation between specific glycemic derangement and chorea.

Thus, either the BGL or the temporal correlation is an insufficient criterion for investigating relationships between the 'episode' of glycemic derangement and chorea. Because BGLs vary and clinical features are often mixed with chorea and ballism, a new term 'glycemic choreoballism (gCB)' can be adopted to encompass the clinical and laboratory variabilities [2].

Since even pathologic studies have had difficulties capturing consistent features of gCB, tagging gCB with a reliable 'marker' is a pragmatic measure to identify appropriate cases [2]. We noticed hyperintensities on T1-weighted brain magnetic resonance imaging (MRI) which were consistently identified in 97.2% of CB cases associated with diabetes mellitus (DM), but the other MRI sequences were variable [2]. T1-weighted images are readily available in routine MRIs, and the list of disorders with T1 hyperintensities on the putamen is limited and rarely associated with CB [4]. Furthermore, a previous study showed the resolution of T1 hyperintensities in the follow-up MRIs along with the disappearance of gCB, which were done between 2 and 18 months after the initial MRIs [3]. Thus, T1 hyperintensities provide an eligible biological marker of gCB. In the study of Ryan et al. they did not present the timing of MRI acquisition. However, since MRIs might be taken near the onset of gCB, delays in MRI acquisition are less likely to make a contribution to the low frequency of T1 hyperintensities (50%) [1].

These considerations lead us to propose operational criteria for gCB as follows: (1) the diagnosis of DM before or at the time of presentation, (2) the presence of CB, and (3) hyperintensities of the putamen on T1-weighted brain MRIs. The criteria are readily applicable to a recent neuroimaging study, in which onset time of gCB was uncertain, BGLs were variable and not fully available, T2*-based sequences were inconsistent, but T1-weighted brain MRIs showed consistent hyperintensities [5]. Given the limited understandings about gCB, it is necessary to select a homogeneous set of patients by unified criteria containing the most reliable marker, which is essential for deciphering the enigma of gCB. Further prospective studies are necessary to validate these criteria in large groups of subjects.

Financial disclosure/conflict of interest

None.

Funding sources

None.

Acknowledgements

None.

References

[1] C. Ryan, J.E. Ahlskog, R. Savica, Hyperglycemic chorea/ballism ascertained over 15 years at a referral medical center, Park. Relat. Disord. 48 (2018) 97–100.
[2] D. Lee, Y.N. Kwon, S.H. Shon, J.H. Lee, T.B. Ahn, Glycemic and vascular choreoballism as main causes of secondary choreoballism involving the putamen, Park.

https://doi.org/10.1016/j.parkreldis.2018.06.009
Received 1 April 2018; Received in revised form 15 May 2018; Accepted 3 June 2018

Relat. Disord. 30 (2016) 29–35.
[3] S.H. Oh, K.Y. Lee, J.H. Im, M.S. Lee, Chorea associated with non-ketotic hyperglycemia and hyperintensity basal ganglia lesion on T1-weighted brain MRI study: a meta-analysis of 53 cases including four present cases, J. Neurol. Sci. 200 (2002) 57–62.
[4] A.G. Osborn, Osborn's Brain: Imaging, Pathology, and Anatomy. Phliadelphia, Pa, Lippincott Williams & Wilkins, 2012.
[5] F. Yu, A. Steven, L. Birnbaum, W. Altmeyer, T2*-based MR imaging of hyperglycemia-induced hemichorea-hemiballism, J. Neuroradiol. 44 (2017) 24–30.

Chaewon Shin, Tae-Beom Ahn*
Department of Neurology, Kyung Hee University Hospital, Kyung Hee University College of Medicine, Seoul, South Korea
E-mail address: taebeom.ahn@khu.ac.kr

* Corresponding author. Department of Neurology, Kyung Hee University Hospital, 23 Kyungheedae-ro, Dongdaemun-gu, Seoul, 130-872, South Korea.