OPEN ACCESS Freely available online

Tremor and Other Hyperkinetic Movements

In Response To:

Roy U, Das SK, Mukherjee A, et al. Irreversible hemichorea–hemiballism in a case of nonketotic hyperglycemia presenting as the initial manifestation of diabetes mellitus. Tremor Other Hyperkinet Mov. 2016; 6. doi: 10.7916/D8QZ2B3F

Cosentino C, Torres L, Nuñez Y, et al. Hemichorea/hemiballism associated with hyperglycemia: report of twenty cases. Tremor Other Hyperkinet Mov. 2016; 6. doi: 10.7916/D8DN454P

Letters

Glycemic Choreoballism

Dokyung Lee[1] **& Tae-Beom Ahn**[1*]

[1] Department of Neurology, College of Medicine, Kyung Hee University, Seoul, Republic of Korea

Keywords: Glycemic choreoballism, diagnostic criteria

Citation: Lee D, Ahn T. Glycemic choreoballism. Tremor Other Hyperkinet Mov. 2016; 6. doi: 10.7916/D8QJ7HNF

*To whom correspondence should be addressed. E-mail: ricash@hanmail.net

Editor: Elan D. Louis, Yale University, USA

Received: September 15, 2016 **Accepted:** October 29, 2016 **Published:** December 8, 2016

Funding: None.

Financial Disclosures: None.

Conflict of Interest: The authors report no conflict of interest.

Ethics Statement: Not applicable for this category of article.

Dear Editor,

We recently read two papers about glycemic choreoballism (gCB) with great interest.[1,2] In a case series, the authors described variable blood sugar levels (BSLs) at choreoballism onset and neuroimaging abnormalities in 18 of 19 patients.[1] Although BSL was commonly elevated in gCB, there were some cases with hypoglycemia or euglycemia.[3,4] Glycosylated hemoglobin (HbA1c) levels also varied. Thus, elevated BSL or HbA1c may not be an essential feature of gCB. Although putamimal involvement was always demonstrated in cases with gCB, brain computed tomography and T2-weighted magnetic resonance imaging (MRI) showed heterogeneous signals. Abnormal, high signal intensity in the putamen on T1-weighted MRI is the most consistent feature of gCB.[3]

In their case report, the authors reported persistent gCB for 6 months.[2] The clinical outcome of gCB varies from rapid recovery to prolongation over years. In our study, gCB persisted for longer than 1 and 12 months in 37.2% and 14.2% of cases, respectively.[3]

The study of gCB has been limited by various factors such as variable clinical presentation and inconsistent datasets, including diagnostic work-ups. Thus, we suggest the following operational diagnostic criteria for gCB: diagnosis of DM, presence of CB, and hyperintensities on T1-weighted MRI.[3] Adherence to these diagnostic criteria is important for case selection, which is fundamental for understanding disease pathophysiology and choosing treatment strategies.

Appropriate gCB management also remains unclear. Meticulous BSL control is essential but not always successful. Current medical treatments including dopamine receptor blocking agents (DRBAs) are ineffective in many cases. Moreover, DRBA use can be complicated with parkinsonism, especially in older patients with or without subclinical neurodegeneration, evidenced by abnormalities on dopamine transporter scans.[5,6] The abolition of gCB by focal lesions such as thalamic infarctions may suggest surgical treatment as an option for chronic, refractory cases.[6]

Acknowledgements

This research was financially supported by the Ministry of Trade, Industry and Energy (MOTIE) and Korea Institute for Advancement of Technology (KIAT) through the International Cooperative R&D program.

References

1. Cosentino C, Torres L, Nuñez Y, et al. Hemichorea/hemiballism associated with hyperglycemia: report of twenty cases. *Tremor Other Hyperkinet Mov* 2016;6. doi: 10.7916/D8DN454P.

2. Roy U, Das SK, Mukherjee A, et al. Irreversible hemichorea–hemiballism in a case of nonketotic hyperglycemia presenting as the initial manifestation of diabetes mellitus. *Tremor Other Hyperkinet Mov* 2016;6. doi: 10.7916/D8QZ2B3F.

3. Lee D, Kwon YN, Shon SH, Lee JH, Ahn TB. Glycemic and vascular choreoballism as main causes of secondary choreoballism involving the putamen. *Parkinsonism Relat Disord* 2016;21:29–35. doi: 10.1016/j.parkreldis.2016.06.016.

4. Lee D, Lee D, Ahn TB, Hong IK, Kim DY. Recurrent hemichorea after a hypoglycemic episode. *Parkinsonism Relat Disord* 2014;20:676–677. doi: 10.1016/j.parkreldis.2014.03.006.

5. Teodoro T, Lobo PP, Ferreira J, et al. Delayed Parkinsonism after acute chorea due to non-ketotic hyperglycemia. *J Neurol Sci* 2015;354:116–117. doi: 10.1016/j.jns.2015.04.039.

6. Lee D, Ahn TB, Hong IK. Abolition of hyperglycaemic hemichorea and recurrence after medical illness. *Can J Neurol Sci* 2016;43:745–746. doi: 10.1017/cjn.2016.265.