Sarcouncil Journal of Medicine and Surgery

ISSN(Online): 2945-3534

Volume- 03| Issue- 06| 2024

Letter to the Editor **Received:** 20-05-2024 | **Accepted:** 10-06-2024 | **Published:** 29-06-2024

Postictal Hyperglycemia is not suitable as a Biomarker of Mitochondrial Disease

Josef Finsterer
MD, PhD, Neurology & Neurophysiology Center, Vienna, Austria, ORCID: 0000-0003-2839-7305

Keywords: MELAS, mtdna, mitochondrial, epilepsy, diabetes.

LETTER TO THE EDITOR

We read with interest the article by Liao *et al*. about two epilepsy patients carrying the m.3243A>G variant who presented with postictal hyperglycemia in the absence of a history of diabetes, normal HbA1c values, normal ketone bodies, and absence of an infection [Liao, N. Y. *et al*., 2024]. It was concluded that transient postictal hyperglycemia may be a red flag to consider the diagnosis of mitochondrial encephalopathy, lactic acidosis, and stroke-like episode (MELAS) syndrome [Liao, N. Y. *et al*., 2024]. The study is appealing but raises concerns that need to be discussed.

We disagree with the statement that postictal hyperglycemia can be an indicator for MELAS. Though postictal hyperglycemia is a rare constellation, it is not only rare in non-MELAS patients but also rare in MELAS patients. When searching the relevant medical databases, not a single hit could be achieved with the search terms "postictal hyperglycemia" combined with "MELAS". Therefore, we strongly disagree that post-ictal hyperglycemia suggests MELAS.

There are several reasons why the two patients presented with post-ictal hyperglycemia. First, both of them could have subclinical diabetes. Missing in this respect are the results of the oral glucose tolerance test (oGTT). To definitively exclude pre-diabetes in the two index patents, it is crucial that the results of the oGTT are presented. Diabetes is not infrequent in patients carrying the m.3243A>G variant [Yee, M. L. *et al*., 2018]. Therefore, the stress of the seizure may have unmasked subclinical diabetes. An argument in favour of diabetes in the two index patient is the family history of patient 1 which was positive for diabetes [Liao, N. Y. *et al*., 2024]. Second, nothing is reported about the food habits of the two index patients. As long as we do not know anything about what the two patients had been eaten shortly before hospitalisation, we cannot exclude that transient hyperglycemia resulted from their nutritional habits. Missing is the current medication of both patients. Knowing the current medication is crucial as some drugs have a hyperglycemic potential, such as steroids, glucagon, or diazoxide [3]. Which compounds were administered to stop the seizures?

WE do not agree that patients with a mitochondrial disorders in general have pancreas insufficiency [Liao, N. Y. *et al*., 2024]. Some patients with a mitochondrial disorder can indeed develop pancreatitis [Finsterer, J, 2007], which may secondarily lead to beta-cell insufficiency. However, the frequency of mitochondrial pancreatitis is low. Were there any indications in the index patients that they elevated amylase or lipase or a previous history of pancreatitis?

Overall, the interesting study has some limitations and inconsistencies which challenge the results and their interpretation. Addressing these issues would strengthen the conclusions and could increase the status of the study.

REFERENCES

1. Liao, N. Y., Lin, L. Y., Chen, C., Liao, Y. C. & Lee, Y. C. "Transient Postictal Hyperglycemia as a Diagnostic Clue of Mitochondrial Encephalomyopathy, Lactic Acidosis, and Stroke-like Episodes." *Acta Neurol Taiwan, 31.2* (2022): 79-83.
2. Yee, M. L., Wong, R., Datta, M., Fazlo, T. N., Ebrahim, M. M., McNamara, E. C., De Jong, G. & Gilfillan, C. "Mitochondrial disease: an uncommon but important cause of diabetes mellitus." *Endocrinol Diabetes Metab Case Rep* (2018): 18-0091.
3. "Diabetes: Oxidative Stress and Dietary Antioxidants." *Academic Press* (2014): 173-182.
4. Finsterer, J. "Pancreatitis as a manifestation of mitochondrial disorder." *Am J Med Genet A, 143A.6* (2007): 632-633.

***Corresponding Author:** Josef Finsterer

DOI- https://doi.org/10.5281/zenodo.12600799

Source of support: Nil; **Conflict of interest:** Nil.

Cite this article as:
Finsterer, J. "Postictal Hyperglycemia is not suitable as a Biomarker of Mitochondrial Disease." *Sarcouncil Journal of Medicine and Surgery* 3.6 (2024): pp 97-98.

Publisher: SARC Publisher