Int. j. adv. multidisc. res. stud. 2026; 6(6):1251-1252

Received: 08-11-2026
Accepted: 18-11-2026

International Journal of Advanced Multidisciplinary Research and Studies

ISSN: 2583-049X

Letter to the Editor

Serum and CSF Glucose cannot Serve as Biomarkers for the Severity of GBS Unless all Influencing Factors are Analysed

[1] Sinda Zarrouk, [2] Josef Finsterer

[1] University of Tunis El Manar and Genomics Platform, Pasteur Institute of Tunis, Tunisia
[2] Department of Neurology, Neurology & Neurophysiology Center, Vienna, Austria

Corresponding Author: **Josef Finsterer**

Letter to the Editor

We read with interest the article by Talukder *et al*. on a cross-sectional study investigating the relationship between the severity of Guillain-Barré syndrome (GBS), as measured by the Hughes Functional Scale, and serum levels of fasting blood glucose (FPG), cerebrospinal fluid glucose (CSF-G), and HbA1c in 61 GBS patients [1]. 64% of the patients were ambulatory (disability level 1–3), and 36% were non-ambulatory (disability level >3) [1]. FPG and CSF-G were higher in the non-ambulatory group than in the ambulatory group [1]. It was concluded that non-ambulatory GBS patients had higher FPG/CSF-G levels than non-ambulatory patients [1]. The study is interesting, but some points require further discussion.

First, several factors influencing FPG/CSF-G levels were not considered in the analysis. FPG/CSF-G levels depend not only on diet but also on physical activity, stress, comorbidities, medications, hormonal changes, sleep quality, lifestyle, and the environment. Simple carbohydrate intake increases FPG/CSF-G levels, while a diet high in fat, protein, and fiber slows glucose uptake (patients with severe GBS are more likely to receive glucose infusions than outpatients). Alcohol can either increase or decrease FPG/CSF-G levels. Regular exercise improves insulin sensitivity and lowers blood glucose, but intense or poorly timed activity (e.g., without adequate carbohydrate intake) can cause drastic fluctuations. Emotional and physical stress leads to the release of cortisol and adrenaline, which increases FPG/CSF-G levels (non-ambulatory patients with GBS are likely to experience greater stress than ambulatory patients). Medications such as steroids, decongestants, or antipsychotics can significantly increase FPG/CSF-G. Sleep deprivation increases insulin resistance. FPG/CSF-G levels may rise in the morning due to hormonal fluctuations, and menstruation can also cause fluctuations. Dehydration can concentrate blood glucose, and extreme temperatures can impair insulin uptake and action [2]. Until these factors influencing FPG/CSF-G levels are included in the analysis, the conclusions remain unreliable.

Secondly, the positive correlation between FPG/CSF-G can be disrupted by various factors that affect the blood-brain barrier (BBB). Conditions that increase BBB permeability through activation of matrix metalloproteinases (MMPs) or release of vascular endothelial growth factor include neurodegenerative diseases (e.g., dementia, Parkinson's disease, multiple sclerosis, amyotrophic lateral sclerosis), ischemic stroke, traumatic brain injury, bacterial meningitis, sepsis, and chronic inflammation. Factors that weaken the BBB include medications (e.g., steroids, MMP inhibitors, antioxidants, free radical scavengers, angiotensins, magnesium, and adrenergic substances), biological factors (e.g., angiopoietin, annexin, TGF-β blockers, retinoic acid), lifestyle factors (e.g., diet, BMI, sleep), and factors that reduce adhesion (e.g., downregulation of tight junction proteins, suppression of HIF-1, inhibition of the RhoA/ROCK pathway) [3]. These factors must be considered when measuring CSF-G concentration.

Third, the factors that triggered GBS in the included patients were not reported [1]. Generally, GBS can be triggered by infections, vaccinations, sepsis, immunological disorders, or trauma [4]. The most common bacterial triggers of GBS include Campylobacter jejuni, Haemophilus influenzae, Mycoplasma pneumoniae, and E. coli. Viral triggers include VZV, HIV, Zika, EBV, influenza, SARS-CoV-2, HAV, HBV, HCV, HEV, rubella, West Nile virus, Japanese nephritis, and chikungunya [4]. Triggering factors should be included in the analysis, as they can determine the severity of GBS.

As long as not all factors influencing FPG/CSF-G are included in the analysis, and as long as not all GBS-triggering factors are included, FPG/CSF-G cannot be recommended as reliable biomarkers for the severity of GBS.

Declarations
Ethical approval: Not applicable.

Consent to participation: Not applicable.

Consent for publication: Not applicable.

Funding: None received.

Availability of data and material: All data are available from the corresponding author.

Completing interests: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Author contribution: JF was responsible for the design and conception, discussed available data with coauthors, wrote the first draft, and gave final approval. SZ: Contributed to literature search, discussion, correction, and final approval.

Acknowledgements: None.

Keywords: Fasting Blood Glucose, Cerebrospinal Fluid, Guillain-Baree Syndrome, Outcome, Biomarkers

References
1. Talukder MRK, Sadat MN, Jahan A, Akhter H, Roy S. Association of Plasma and Cerebrospinal Fluid Glucose Level with Clinical Severity in Patients with Guillain-Barre Syndrome. Mymensingh Med J. 2026; 35(2):377-383.
2. Good to Know: Factors Affecting Blood Glucose. Clin Diabetes. 2018; 36(2):202. Doi: 10.2337/cd18-0012
3. Almutairi MM, Gong C, Xu YG, Chang Y, Shi H. Factors controlling permeability of the blood-brain barrier. Cell Mol Life Sci. 2016; 73(1):57-77. Doi: 10.1007/s00018-015-2050-8
4. Finsterer J. Triggers of Guillain-Barré Syndrome: *Campylobacter jejuni* Predominates. Int J Mol Sci. 2022; 23(22):14222. Doi: 10.3390/ijms232214222