Neurologia i Neurochirurgia Polska
Polish Journal of Neurology and Neurosurgery
2023, Volume 57, no. 4, pages: 403–404
DOI: 10.5603/PJNNS.a2023.0045

ISSN: 0028-3843, e-ISSN: 1897-4260

Response to 'Before blaming levodopa/carbidopa intestinal gel for demyelinating polyneuropathy, all differential aetiologies must be ruled out'

Radosław Piekarski[1, 2], Anna Roszmann[1, 2], Jarosław Dulski[1, 2], Jarosław Sławek[1, 2]

[1]Department of Neurology & Stroke, St. Adalbert Hospital, Gdansk, Poland
[2]Division of Neurological and Psychiatric Nursing, Faculty of Health Sciences, Medical University of Gdansk, Gdansk, Poland

To the Editors

We thank the author of the Letter to the Editors entitled 'Before blaming levodopa/carbidopa intestinal gel for demyelinating polyneuropathy, all differential aetiologies must be ruled out' [1] concerning our article [2]. The author points out the need for thorough differential diagnosis in all Parkinson's disease (PD) patients treated with LCIG (levodopa/carbidopa intestinal gel), before identifying this medication as causing polyneuropathy (PNP).

We do agree that before making the diagnosis of Guillain–Barré syndrome (GBS)/chronic inflammatory demyelinating polyneuropathy (CIDP), all the differential diagnoses listed in this Letter to the Editors [1] should be excluded. This is crucial especially when considering the introduction of intravenous immunoglobulins (IVIG) treatment. Such a decision should always be made with great caution because the treatment itself is indicated only in a limited number of illnesses, is limited in its access, and generates high therapy costs. The authors of the referred papers on single cases usually make the diagnosis of GBS/CIDP-like neuropathies, which might suggest that the differential diagnostic was in fact performed. However, data from referred papers on the performed differential diagnosis is scarce and — as mentioned in this Letter to the Editors [1] — may be a limitation of this review. Nevertheless, we have made only a review of the existing literature and we have presented already published data.

The problem of GBS or CIDP following an infection or vaccination against SARS-CoV-2 might also be another limitation of the discussed review. However, despite the article being published in the SARS-CoV-2 pandemic period, all of the patients (including our patient) were diagnosed several years before the pandemic began.

Regarding our case report presented in the 'clinical vignette' with the pre-existing nerve damage, we were aware of this condition. However it was asymptomatic. Our patient did not suffer from any other illnesses except for PD and PD-related mild dementia, and only rivastygmine treatment was introduced shortly before the PNP diagnosis. Furthermore, no unequivocal conclusion on whether the LCIG caused the PNP was made in this particular case.

Patients with long-lasting Parkinson's disease (PD) are usually treated with high levodopa (LD) doses. It has been proven in previous papers [3] that PNP in PD may be related to high homocysteine levels and its neural toxicity may be triggered by LD therapy. This is usually of the axonal type, but in this paper [2] we discussed the far less common demyelinating type. As PNP due to LCIG therapy is common, it is easy to miss such a potentially treatable cause. We advise physicians to look carefully at the type of neuropathy and not to exclude patients from something that is usually a 'last chance' treatment of choice in advanced PD.

Our aim was to emphasise the possible demyelinating form on neuropathy; however, we agree that there was uncertainty as to whether this was LCIG-related or just a coincidence.

Again, the fact of limited access to detailed clinical data, i.e. that our paper was based on the available historical cases, was discussed at length in our paper. No unequivocal conclusions should be made upon such data, and no such conclusions were presented by us. We concluded that all patients starting LCIG therapy should be carefully examined (both clinically and electrophysiologically) to detect those at risk (with initially mild symptoms) of developing any type of neuropathy. Those patients should be thoroughly monitored during therapy. Indeed, this is routine procedure at our centre.

Address for correspondence: Radosław Piekarski, Department of Neurology & Stroke, St. Adalbert Hospital, Al. Jana Pawła II 50, 80–462 Gdansk, Poland; e-mail: piekarski.md@gmail.com

Received: 05.07.2023 Accepted: 05.07.2023 Early publication date: 19.07.2023

Conflicts of interest: None.
Funding: None.

References

1. Finsterer J. Before blaming levodopa/carbidopa intestinal gel for demyelinating polyneuropathy, all differential aetiologies must be ruled out. Neurol Neurochir Pol. 2023 [Ahead of print], doi: 10.5603/PJNNS.a2023.0043.
2. Piekarski R, Roszmann A, Dulski J, et al. Acute/subacutae demyelinating polyneuropathy in Parkinson's disease patients on levodopa-carbidopa intestinal gel therapy: systematic review with new case report. Neurol Neurochir Pol. 2023; 57(2): 169–176, doi: 10.5603/PJNNS.a2023.0001, indexed in Pubmed: 36628506.
3. Szadejko K, Dziewiatowski K, Szabat K, et al. Polyneuropathy in levodopa-treated Parkinson's patients. J Neurol Sci. 2016; 371: 36–41, doi: 10.1016/j.jns.2016.09.061, indexed in Pubmed: 27871444.