



Received: 26-04-2026  
Accepted: 06-05-2026

ISSN: 2583-049X

Letter to the Editor

## **Before CIDP is Associated with Malignancy, A Causal Relationship should be Proven**

**Josef Finsterer**

Department of Neurology, Neurology & Neurophysiology Center, Vienna, Austria

DOI: <https://doi.org/10.62225/2583049X.2026.6.3.6303>

Corresponding Author: **Josef Finsterer**

### **Letter to the Editor**

We read with interest the article by Nekrasova *et al.* on a retrospective study of the incidence of malignancies in 61 patients with chronic inflammatory demyelinating polyneuropathy (CIDP) and its subtypes, as well as the differences between patients with and without malignancies in terms of clinical, biochemical, and electrophysiological characteristics [1]. The mean INCAT (Inflammatory Neuropathy and Treatment) score was 2.29 in CIDP patients with malignancies (CIDP-m) and 2.21 in CIDP patients without malignancies (CIDP-nm) [1]. In CIDP-nm, the distal latency of the peroneal nerve correlated negatively with the INCAT score, the cerebrospinal fluid (CSF) values correlated positively with the distal latency in the peroneal nerve and ulnar nerve, and CSF values correlated negatively with the nerve conduction velocity (NCV) of the ulnar and peroneal nerves [1]. It was concluded that CIDP-m tends to have a more aggressive disease course than CIDP-nm [1]. The study is interesting, but some points should be discussed.

The first point concerns the causal relationship between malignancy and CIDP. Since malignancy was diagnosed later than CIDP in all patients, it is conceivable that, at least in some patients, there is no causal relationship between the two. The probability of a causal relationship may depend on the latency period between the time of CIDP diagnosis and the time of malignancy diagnosis. This parameter should be included in the analysis.

The second point concerns the discrepancy between the statement in the “Methodology” section that nodal and paranodal antibodies were determined and the statement in the “Exclusion Criteria” section that patients with nodopathy were excluded. It is not clear why contactin and neurofascin antibodies were determined, but patients who tested positive for these antibodies were excluded. What is the point of determining these antibodies if these patients are excluded from the study anyway?

The third point concerns the discrepancy between the exclusion criteria (IgM paraproteinemia) and the findings that one patient had lambda-type monoclonal gammopathy of undetermined significance (MGUS) [1]. Why was this patient with MGUS not excluded from the study?

The fourth point is that the number of CIDP-m patients was too small ( $n = 14$ ) to draw general conclusions. This view is supported by the fact that no statistically significant differences between the CIDP-m and CIDP-nm groups could be calculated. Larger and more homogeneous cohorts are needed to establish a link between the severity of CIDP and malignancies.

The fifth point is that the electrophysiological parameters in CIDP-m patients may also depend heavily on the type of chemotherapy that patients with malignancies have received. Since some chemotherapies can be neurotoxic, it is conceivable that, at least in some patients, the electrophysiological findings were due to toxic neuropathy rather than CIDP. Were the electrophysiological examinations repeated after the diagnosis and treatment of the malignancy? Was there a difference between the study results at the time of CIDP diagnosis and at the time of diagnosis and treatment of the malignancy?

The sixth point is that comorbidities and concomitant medications were not included in the study. Since numerous diseases can be associated with neuropathy (e.g., diabetes, renal insufficiency, alcoholism) or intoxication from concomitant medications (e.g., phenytoin, disulfiram, proteasome inhibitors, immune checkpoint inhibitors, amiodarone, hydralazine, perhexiline, colchicine, metronidazole, quinolones, isoniazid, dapsone, tacrolimus, cyclosporine) [2], it is essential to include concomitant medications and comorbidities in the analysis.

Finally, the INCAT score only evaluates motor functions [3]. If the CIDP was predominantly sensory, the score may be low or zero despite significant sensory involvement. The lack of an assessment of sensory deficits could therefore lead to incorrect results and interpretations. In addition to the INCAT, the INCAT Sensory score should have been applied [4].

Overall, before attributing CIDP to a malignancy, a causal link should be established and all possible differential causes of

neuropathy should be ruled out.

**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. SM: contributed to literature search, discussion, correction, and final approval.

**Acknowledgements:** None.

**Keywords:** CIDP, Electrophysiology, Malignancy, Nodopathies, INCAT Score

---

**References**

1. Nekrasova P, Koszewicz M, Wiczorek M, Kłębek-Targowska P, Budrewicz S, Dziadkowiak E. Clinical, serological, and electrophysiological differences in chronic inflammatory demyelinating polyradiculoneuropathy patients with and without malignancy - real-life evidence. *Neurol Neurochir Pol*, Nov 25, 2025. Doi: 10.5603/pjnns.108522
2. Jones MR, Urits I, Wolf J, Corrigan D, Colburn L, Peterson E, *et al.* Drug-Induced Peripheral Neuropathy: A Narrative Review. *Curr Clin Pharmacol*. 2020; 15(1):38-48. Doi: 10.2174/1574884714666190121154813
3. Breiner A, Barnett C, Bril V. INCAT disability score: A critical analysis of its measurement properties. *Muscle Nerve*, Aug 2014; 50(2):164-169. Doi: 10.1002/mus.24207
4. Draak TH, Vanhoutte EK, Van Nes SI, Gorson KC, Van Der Pol WL, Notermans NC, *et al.* PeriNomS Study Group. Comparing the NIS vs. MRC and INCAT sensory scale through Rasch analyses. *J Peripher Nerv Syst*, Sep 2015; 20(3):277-288. Doi: 10.1111/jns.12127