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LETTER TO THE EDITOR



# Authors response: other autoimmune disorders in chronic immune-mediated neuropathies: why trying to find out does matter

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We thank the correspondents for raising important methodological considerations regarding the interpretation of associations between autoimmune diseases and chronic immune-mediated neuropathies (CIN).

Our article mainly aimed to be a narrative literature review with the objective of synthesizing the available clinical descriptions of autoimmune comorbidities reported in patients with CIN. The objective was not to estimate prevalence or establish causality, but rather to map the spectrum of reported associations and highlight areas requiring further investigation.

To achieve this, we performed a structured literature search of the PubMed/MEDLINE and Embase databases covering publications up to 2025. Search terms combined concepts related to immune-mediated neuropathies (e.g. *CIDP*, *chronic inflammatory demyelinating polyradiculoneuropathy*, *immune-mediated neuropathy*) with terms related to systemic autoimmunity (e.g. *autoimmune disease*, *polyautoimmunity*, *autoimmune disorders (each condition separately*). Reference lists of relevant articles were also screened to identify additional reports. Studies describing autoimmune comorbidities in patients with CIN, including observational studies, cohort analyses, case series and case reports were considered. Given the heterogeneity of studies, populations, and diagnostic criteria across the available literature, the data were synthesized qualitatively rather than through meta-analysis or formal risk-of-bias scoring.

The correspondents suggest that the clustering of autoimmune diseases in CIN may reflect background population trends rather than shared pathophysiology. We agree that this is a possibility and acknowledge that population-based controlled studies remain the most appropriate approach for determining true prevalence and causal associations. Indeed, epidemiological studies have shown that autoimmune diseases frequently co-occur across different organ systems, reflecting shared genetic and immunological susceptibility rather than disease-specific mechanisms alone [1–4]. In this context, several reports have described associations between chronic immune-mediated neuropathies and systemic autoimmune disorders such as autoimmune thyroid disease, Sjögren's syndrome, or systemic lupus erythematosus [5]. While these observations do not establish causality, they support the rationale for considering CIN within a broader immunological framework.

We acknowledge that surveillance bias, referral bias, and diagnostic heterogeneity may influence the associations reported in the literature [6]. Such limitations are well recognized in studies of immune-mediated neuropathies [7]. Because our article aimed to synthesize observations across a heterogeneous body of literature, it was not feasible to apply a standardized quantitative risk-of-bias tool. We therefore emphasize that the findings should be interpreted as hypothesis-generating rather than definitive epidemiological evidence.

The correspondents also raise concern that highlighting autoimmune associations might lead to unnecessary immunotherapy. Our intention was the opposite. Recognizing systemic autoimmune context may help clinicians refine diagnostic evaluation and potentially tailor therapeutic strategies, rather than apply empiric treatment. Increasingly, the field of immune-mediated neuropathies is moving toward biomarker-based stratification and more precise disease classification, as emphasized in recent consensus and research efforts [8].

Moreover, preliminary data from our units suggest that the presence of concomitant autoimmune disease may influence therapeutic response in some patients with CIDP, through potentially higher immunoglobulin dosing requirements [9]. While these findings require confirmation in larger studies, they illustrate why further investigation of systemic immune context may be clinically relevant.

In summary, we agree that caution is warranted when interpreting associative observations and that controlled epidemiological studies are necessary to determine whether autoimmune comorbidities occur in CIN more frequently than expected by chance. Nevertheless, documenting and synthesizing these clinical observations represents an important first step toward developing the more refined classifications, biomarker-guided stratification, and diagnostic precision that the correspondents appropriately advocate.

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## Declaration of interests

The authors have no relevant affiliations or financial involvement with any organization or entity with a financial interest in or financial conflict with the subject matter or materials discussed in the manuscript. This includes employment, consultancies, honoraria, stock ownership or options, expert testimony, grants or patents received or pending, or royalties.

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