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Teriflunomide Cannot Be Held Responsible for Median Nerve Damage until Other Pathologies are off the Table

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LETTER TO THE EDITOR

We were interested to read the article by Etemadifar *et al*. on a cross-sectional study on the prevalence of carpal tunnel syndrome (CTS) or distal median nerve dysfunction (DMND) in patients with multiple sclerosis (MS) treated with teriflunomide [Genova, A. *et al*., 2024]. Of the 52 patients included, 5.8% had CTS, but 52% had DMND [Genova, A. *et al*., 2024]. DMND was correlated with right side, duration of MS and age [Genova, A. *et al*., 2024]. The study is appealing, but some points should be discussed.

The first point is that CTS is multicausal, and until all known causes of CTS have been thoroughly ruled out, it should not be attributed to teriflunomide [Genova, A. *et al*., 2024]. Common causes of CTS that need to be ruled out include frequent repetitive movements of the hands, extension or flexion of the wrist over a long period of time, rheumatoid arthritis, hormonal imbalances (e.g. thyroid dysfunction, diabetes, menopause, pregnancy), metabolic disorders, anatomical changes to the carpal tunnel (e.g. change in bone or joint shape over time) and injuries to the wrist (e.g. strain, sprain, dislocation, fracture) [Genova, A. *et al*., 2020]. A strong argument against a causal relationship is the fact that CTS due to teriflunomide has not been reported so far. A second argument against teriflunomide as a cause of CTS is that no dose dependence has been reported. There did not appear to be an increase in symptoms and instrumental abnormalities with increasing dose.

The second point is that only the median nerve was examined [Genova, A. *et al*., 2024]. If one assumes that teriflunomide is neurotoxic and causes peripheral nerve damage, it would have been imperative to examine motor and sensory nerves other than the median nerve. The examination of other nerves is also crucial for assessing whether a patient really suffers from CTS or rather from a polyneuropathy including the median nerve.

The third point is that no control group was studied. To assess whether MS patients not taking teriflunomide have different or similar results, it would have been crucial to study healthy and diseased control subjects who are the same in terms of age and gender.

The fourth point is that the reproducibility of the electrophysiological results was not investigated. To assess whether the electrophysiological results are consistent and reliable, it would have been important to retest all included patients.

The fifth point is that comorbidities were not included in the analysis. Before attributing DMND to teriflunomide, it is important to exclude diabetes, alcoholism, renal insufficiency, previous chemotherapy, vitamin deficiency, vasculitis and immune neuropathies as causes of DMND [Horlings, C. G. C. *et al*., 2020].

In summary, this interesting study has limitations that put the results and their interpretation into perspective. Addressing these limitations could strengthen the conclusions and reinforce the message of the study. All open questions need to be addressed before readers can uncritically accept the study's conclusions. Before attributing CTS or DMND in MS patients to teriflunomide, all other causes of median nerve damage must be thoroughly ruled out.

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