Dear Sir,

The article by Hirahara et al. describes retrospective observations on 16 patients with nonsystemic vasculitic neuropathy (NSVN) [1]. NSVN is a rare disorder with only eight series reporting clinical information on more than ten patients [2, 3]. A Peripheral Nerve Society (PNS) task force published a guideline on NSVN in 2010 [2]. As members of this task force, we have questions about this study.

How did the authors distinguish between ‘polyneuropathy’ and ‘mononeuritis multiplex?’ How were diffuse polyneuropathies with asymmetric features categorized?

How many patients had pain? In table 1, only three had ‘myalgias’, but another three presented with lower limb pain.

Were electrodagnostic findings axonal or demyelinating?

Patient 13 had an erythrocyte sedimentation rate of 115 mm/h, exceeding the PNS guideline cutoff of 100 mm/h for NSVN. This patient should be presumed to have a systemic vasculitic neuropathy.

In table 1, the ‘diameter of artery with angiitis’ ranges from 75–600 μm (mean 270 μm). In five patients, affected arteries were 400–600 μm. For the 11 patients with ‘probable’ vasculitis, that is, no vessel exhibiting inflammatory vessel-wall damage, there would be no pathologic basis on which to ascertain the size of angiitis-affected vessels [2]. How did the authors determine the size of the involved vessels in these patients? Moreover, the tabulated vessel sizes are unexpectedly large. Almost all vessels in sural nerve biopsies are ≤300 μm [4]. In the two previous analyses of affected vessel size in NSVN, mean diameters were 98 ± 87 μm and 80 ± 31 μm [5, 6]. Can the authors explain these inconsistencies?

What was the duration between the ‘initial’ and ‘final’ modified Rankin scale (mRS) assessment for each patient?

The final mRS for patients 6, 7, 8, and 14 is inconsistent between tables 1 and 2. According to table 1, three of sixteen patients improved; according to table 2, five of twelve patients improved. Both response rates are lower than those reported in past studies, where most patients improved with immunosuppressive therapy [7–9]. This cohort might have done worse due to the reliance on corticosteroid monotherapy and under-treatment with the gold standard for severe small-vessel vasculitis, cyclophosphamide.

The title of this paper is misleading. The gait disturbance is likely due not only to foot drop but also sensory loss and planter flexion weakness. Was the foot drop bilateral or unilateral? Foot drop is an indicator of the severity of a neuropathy; its refractoriness is usually relative and not absolute.

We appreciate the authors’ interest in this underreported disorder and applaud their pioneering work in identifying prognostic factors for NSVN.
References


