

Acute inflammatory demyelinating polyneuropathy shortly after administration of intravitreal ranibizumab: A clinical and electrophysiological annotation



Polineuropatía desmielinizante inflamatoria aguda tras la administración de ranibizumab intravítreo: un apunte clínico y electrofisiológico

Dear Editor,

We read with interest the paper by Fuerte-Hortigón et al.¹ reporting a hitherto unheard-of association between acute inflammatory demyelinating polyneuropathy (AIDP) and administration of intravitreal ranibizumab. We wish to make a comment on the authors' clinical and electrophysiological findings.

AIDP is a classical form of Guillain-Barré syndrome (GBS) presenting with acute flaccid paralysis of all 4 limbs and electrophysiological evidence of demyelination.² The reported patient developed a 3-day history of lower-limb tingling, unsteadiness, and sinus tachycardia.¹ Regrettably, no mention is made of the presence of muscle weakness. Given that early reflex loss may be a core feature of GBS,³ it would also have been advisable for the authors to include specific reference to the status of upper- and lower-limb tendon reflexes. Be that as it may, the reported clinical data are insufficient to establish a diagnosis of classic GBS.

The authors report a nerve conduction study performed on the fourth day after onset,¹ namely within the conventional period of very early GBS⁴; the observed changes are worth commenting on, and may be summarized as follows: (i) normal motor conduction velocities and distal motor latencies in the median, ulnar, tibial, and peroneal nerves, with compound motor action potential (CMAP) amplitudes always being preserved; (ii) increased F-wave latency in just one nerve, the right peroneal nerve; (iii) alterations of sensory conduction parameters consisting of reduced sensory nerve action potential (SNAP) amplitudes in the right median, left ulnar, and right and left sural nerves, with sensory conduction velocities (SCV) being preserved or minimally slowed; and (iv) SNAP of left median and right ulnar nerves "were not induced"; therefore, we assume that they were inexcitable. The authors interpret these results as fulfilling the electrodiagnostic criteria for AIDP. We entirely disagree with this interpretation, given that using optimized criteria in GBS subtypes,⁵ the only criterion of demyelination is the reported increase of F-wave latency in one nerve (right peroneal); however, the authors do not establish whether this increase is >120% of the upper limit of normal, a necessary condition for diagnosis of AIDP. Moreover,

the early SNAP attenuation with preserved SCV points to an axonal process.⁵ In very early GBS, whether demyelinating or axonal, the outstanding pathogenic event is inflammatory edema, predominantly involving nerve trunks with less efficient blood–nerve interface, particularly the spinal roots, spinal nerves, and probably pre-terminal nerve trunks.⁶ Such histopathological changes may cause a critical increase in endoneurial fluid pressure in nerve trunks possessing epiperineurium (here, spinal nerves and pre-terminal nerve trunks), leading to ischemic conduction failure manifesting as abnormal late responses, CMAP/SNAP attenuation, or even nerve inexcitability.⁴ In most cases of very early GBS, accurate subtyping requires serial electrophysiological evaluation.

In short, the reported case should be categorized as an acute predominantly sensory neuropathy whose causal relationship to intravitreal ranibizumab would require further study.¹

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Authors' contribution

Both authors contributed equally to this work.

Conflict of interest

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J. Berciano^{a,*}, A. García^{b,c}

^a *Service of Neurology, University Hospital Marqués de Valdecilla (IDIVAL), University of Cantabria (UC), Santander, Spain*

^b *Service of Clinical Neurophysiology, University Hospital Marqués de Valdecilla (IDIVAL), University of Cantabria (UC), Santander, Spain*

^c *Centro de Investigación Biomédica en Red de Enfermedades Neurodegenerativas (CIBERNED), Santander, Spain*

* Corresponding author.

E-mail address: joseberciano51@hotmail.com (J. Berciano).

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