

Letters to the Editor



Neuropathic Pain in Early Guillain-Barré Syndrome

TO THE EDITOR:

I read with great interest the systematic review of neuropathic pain (NP) in acute/subacute neuropathies (APN) by Artemiadis and Zis (1). Their study indicates that out of the total of 2,341 patients, 1,139 of them were diagnosed with NP (pooled incidence of NP 48.7%). In Guillain-Barré syndrome (GBS), the commonest cause of acute APN, the pooled estimated incidence of NP was 34.8%. As stated by the authors the types of pain in GBS include peripheral NP, radicular pain, meningism, headache, muscular pain secondary to bad posture, visceral pain, and arthralgias. In some GBS cases pain may precede or coincide with weakness. Worthy of note is the fact that NP occurred in about half the patients showing pure motor GBS (see their Table 1), certainly an unexpected feature in a motor disorder. Furthermore, in the original description of acute motor axonal neuropathy (AMAN), McKhann and colleagues wrote: "many patients had neck and back stiffness and pain; one father said his son seemed as though he had a rod up his spine" (2). Pain in early GBS, conventionally up to 10 days after symptomatic onset, is usually defined with the characteristics of nerve trunk pain: throbbing, excruciating neck, interscapular or lumbar pain, and sometimes associated with sciatica, and neck and back stiffness (3,4). I wish to make a brief comment on the pathophysiology of such nerve trunk pain.

In early GBS, pathological changes, consisting of inflammatory edema, predominate in the region where motor and end sensory roots join to form the spinal nerve (4,5). This lesional predominance has been correlated with less efficient radicular blood-nerve interface (6). Spinal roots traverse the subarachnoid by an elastic multicellular root sheath derived from the arachnoid and penetrate the subarachnoid angle. External to this angle, the newly formed spinal nerves possess epi-perineurium and endoneurium as in the peripheral nerve trunks (7). The absence of epi-perineurium in the spinal roots probably prevents their having an increase of endoneurial fluid pressure (EFP) and isch-

emic injury despite inflammatory changes. Perineurium is relatively inelastic and has only a limited ability to expand (4). Small increases of EFP, caused by endoneurial inflammation, can be accommodated, but any increase beyond these limits, presumably occurring in the early stages of severe forms of GBS, will produce an increase in EFP leading to endoneurial ischemia with the corresponding conduction failure.

Our nerve ultrasonographic and autopsy studies in early GBS, including acute inflammatory demyelinating polyneuropathy (AIDP), AMAN, and acute motor sensory axonal neuropathy, have revealed that changes essentially rely on anterior roots and ventral rami of the spinal nerves (4,8,9). The spinal nerve emerges through the vertebral foramen and divides into a dorsal and ventral ramus (10). The dorsal rami of the spinal nerves are directed backwards, and with the exceptions of those of the first cervical, the fourth and the fifth sacral, and the coccygeal, divide into medial and lateral branches for the supply of the muscles and skin of the posterior part of the neck and trunk. Conceivably, early GBS changes in the dorsal rami have to be similar to those of the ventral rami, though this remains speculative given that the dorsal rami are not amenable to imaging or electrophysiological techniques, and pathological studies are lacking (4). In any case, early endoneurial inflammatory edema, located in the anterior spinal roots at the vertebral foramina entrance, the ventral rami of the spinal nerves or both, could involve abutting dorsal rami, thus causing nerve trunk pain referred to their innervation territories, from neck to buttocks, eventually accompanied by neck and back stiffness.

José Berciano, MD, PhD
 Professor Emeritus
 Service of Neurology
 University Hospital
 "Marqués de Valdecilla (IDIVAL)"
 University of Cantabria

“Centro de Investigación Biomédica en Red de Enfermedades Neurodegenerativas (CIBERNED)”
University of Cantabria
EUE Building, 4th Floor

Avda Valdecilla s/n, 39008
Santander, Spain
E-mail: jaberciano@humv.es

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