Variations of permeability between such areas are presumably important for the distribution of lesions caused by various toxic, immunological, or infectious agents, as with GBS.

Knowledge of the microscopic anatomy of the peripheral nervous system is essential for an adequate understanding of the pathogenic relevance of early pathological events in GBS.[6] Spinal roots traverse the subarachnoid space covered by a lax multicellular root sheath derived from the arachnoid and penetrate the dura at the subarachnoid angle. At the subarachnoid angle, where motor and sensory roots join to form the spinal nerve, dura mater is in continuity with epineurium, whereas the arachnoid turns into perineurium. Therefore, intrathecal nerve roots are covered by an elastic root sheath, whereas spinal nerves and more distant nerve trunks out to their preterminal segments possess epi-perineurium that is relatively inelastic. Conceivably, initial inflammatory edema may be accommodated in intrathecal nerve roots that enlarge in size but without resulting in a significant increase in endoneurial fluid pressure (EFP). Conversely, in nerve trunks surrounded by epi-perineurium, such edema may cause a critical elevation of EFP that constricts transperineurial vessels by stretching the perineurium beyond the compliance limits, leading to ischemic conduction failure, and eventually to Wallerian-like degeneration.[7] Although this phenomenon may occur in any segment of peripheral nerve trunks, MRI/STIR, ultrasound, and pathological studies indicate that spinal nerves are the hotspot in early GBS, thus explaining the high prevalence of electrophysiological changes pointing to pathology in proximal nerve segments (alteration of F waves as in the current patient).[2] In any case, inflammatory edema is also a histological feature of intermediate and preterminal nerve segments, a potential cause of partial conduction block, nerve inexcitability, or reversible conduction failure on serial studies.

In short, the MRI/STIR study in the report by Oguz-Akarsu et al[1] of cervical and lumbar spines in an early GBS patient illustrates that is a useful imaging technique for detecting the presence of edema in the spinal nerves and their ventral rami.

KEYWORDS

COVID-19 infection, endoneurial edema, Guillain-Barré syndrome, MRI, spinal nerve, STIR

CONFLICT OF INTEREST

The authors have no conflicts of interest to declare.

ETHICAL PUBLICATION STATEMENT

We confirmed that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

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Reply: "Spinal nerve pathology in Guillain–Barré syndrome associated with COVID-19 infection"

See article on pages E74–E75 in this issue.

We are pleased to see the correspondence from Berciano and Gallardo with regard to our paper published in *Muscle & Nerve*.[1,2] We would like to thank them for their interest and positive comments. They clearly mentioned the pathophysiologic mechanisms of

Guillain–Barré syndrome (GBS) in spinal nerves and we read their previous studies with great interest.[3]

Gallardo et al showed involvement of spinal nerves with electrophysiologic, ultrasonographic, and pathologic findings in patients with early GBS.[3] Correspondingly, magnetic resonance imaging (MRI) of our patient using short-tau inversion recovery (STIR) sequences showed edema of the spinal nerves and corresponding rami, demonstrating proximal demyelination.

MRI using post-contrast T1 sequences helps to demonstrate the topography of nerve root enhancement in GBS, but it is not applicable to patients with kidney failure or contrast allergies.[4] We think that adding coronal STIR sequences to the imaging protocol is a useful method for the detection inflammatory edema of nerve roots and plexus. In STIR sequences, all structures with short T1 relaxation times are suppressed, whereas structures with high water content show a bright signal on a dark background and demonstrate the swollen spinal nerves and corresponding rami. The coronal plane is the most valuable plane in STIR images, because it demonstrates anatomic structures including proximal nerve segments in a familiar perspective.[5]

Late-response alterations (F wave and H reflex) are the most common early electrophysiologic findings supporting proximal demyelination in early GBS.[6,7] In our case, the increased chronodispersion and decreased persistence of F waves in the median and ulnar nerves can be caused by demyelination in any segment of the peripheral nerve, because nerve conduction studies revealed conduction blocks of median and ulnar nerves in the wrist-elbow segment, and STIR images showed involvement of proximal nerve segments.

More investigations with electrophysiologic, radiologic, and pathologic studies will help further our understanding of the pathophysiologic mechanisms of GBS and elucidate therapeutic strategies.

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None of the authors has any conflict of interest to disclose.

ETHICAL PUBLICATION STATEMENT

We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

ABBREVIATIONS

GBS, Guillain–Barré syndrome; MRI, magnetic resonance imaging; STIR, short-tau inversion recovery.

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Upper trunk brachial plexopathy as a consequence of prone positioning due to SARS-CoV-2 acute respiratory distress syndrome

First reported in December 2019 in Wuhan, China, novel coronavirus (SARS-CoV-2) has become a major challenge for health-care systems around the world. It is estimated that about 17% of patients develop severe pneumonia, with a high incidence of acute respiratory distress syndrome (ARDS). Among the treatments for managing ARDS, prone positioning is used to improve ventilation.[1] There are a few reports of neuropathies or brachial plexopathies associated with prone positioning.[2-4] We present the case of a man who developed a right brachial plexopathy after prone positioning when being treated for SARS-CoV-2 ARDS.