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Commentary

Commentary to: The Diagnostic Role of Adding the Hoffman Reflex for L5 Radiculopathy in the Electrodiagnostic Laboratory: A Cross-sectional Study

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To the editor:

Kara et al.'s article[1] is excellent, but some points need discussion.

A first point is that it remained unclear how it could be excluded that a positive EMG was due to causes other than L5 or S1 radiculopathy. Although patients with diabetes, polyneuropathy, rheumatic disease, malignancy, lumbosacral spine surgery, vertebrostenosis, spondylolisthesis, other radiculopathy, central nervous system disease, and myopathy were excluded, this does not exclude the possibility that the patients had other causes of positive EMG. A negative history with regard to the exclusion criteria does not protect against subclinical or mildly manifesting muscle disease caused by hypovitaminosis, endocrine disorder, immunological disease, paraneoplasia, or intoxication.

Second, it is unclear how CNS disease could be excluded. We should know whether all included patients actually underwent brain MRI as well as cervical and thoracic spine MRI before inclusion. Have cerebral and spinal cord diseases been ruled out based solely on the medical history? Even if imaging has been done and was normal, this does not rule out an infectious disease that may not be visible on imaging. How could plexopathy be ruled out as the cause of the delayed H-reflex response? What medications did the included patients take regularly?

A third point is that radix L5 also contains motor fibers that partially innervate the soleus muscle, and conversely, radix S1 also contains motor fibers that supply the long peroneal and anterior tibial muscles. This co-innervation can lead to inconclusive results.

A fourth point is that the relationship between the ASR and the H-reflex of the soleus muscle has not been analysed. In how many patients was the H-reflex absent but the ASR was normal and vice versa? How many patients with L5 radiculopathy had weakness for foot extension and how many with S1 radiculopathy had muscle weakness for foot flexion?

A fifth point is that MRI findings are often inconsistent with clinical or electrophysiological findings[2,3]. We should know how many had L5 radiculopathy on imaging but S1 radiculopathy on clinical or electrophysiological examination and vice versa. It should also be explained why half of the patients in the L5 and S1 radiculopathy groups were EMG negative. Since one inclusion criterion was radiculopathy for at least three months[1], one can assume that the EMG is positive in all patients with L5 or S1 radiculopathy. Is it conceivable that disc herniation was a false positive diagnosis in patients with negative EMG?

A sixth point is that the contralateral, unaffected limb was used as a control[1]. Because the clinically unaffected side of L5 or S1 radiculopathy may be subclinically affected on imaging, it is recommended to use controls who do not show disc herniation either clinically or on imaging.

A seventh point is that the number of patients with EMG-positive and EMG-negative L5 or S1 radiculopathy was too small to make a statistical comparison between these groups reliable.

A final point is that it is unclear why patients over 65 were excluded[1]. If patients >65 met the inclusion and exclusion criteria, why should they not be included in the study?

In conclusion, L5 or S1 radiculopathy should be diagnosed using clinical assessment plus MRI of the lumbar spine. Radiculopathy is unlikely in patients without radicular pain, negative EMG, and absence of muscle weakness. There is no need to diagnose radiculopathy using the H-reflex.

The author has no conflict of interest.

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