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LETTER TO THE EDITOR

Author's response to letter to the editor: "Needle EMG of the vagus-innervated striated larynx muscles cannot indicate laryngeal sensory neuropathy"

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In response to the letter from Finsterer and Scorza [1], we would like to thank them for their comments and for their careful neurological and neurophysiological analysis of the patient's history, published in the article "Laryngeal sensory neuropathy caused by covid-19: findings using LEMG" [2]. We agree that laryngeal electromyography (LEMG) of the vagus-innervated muscles of the larynx cannot indicate laryngeal sensory neuropathy (LSN). As we wrote in the article, LEMG assesses the function of the motor fibres, not the sensory fibres of the nerve. However, in the case of viral lesion, there is usually non-specific damage to the fibres running through the nerve. Therefore, in the context of post-infectious damage to branches of the vagus nerve, it is possible to have mixed damage to both sensory and motor fibres (as we observed in our patients). As reported in the literature, mixed damage to sensory and motor fibres is often observed in pathological processes [3]. Observations by other authors suggest that 75% of patients with LSN have evidence of motor neuropathy [4]. In the patients described in our article, despite the predominance of sensory symptoms, we found dysfunction of motor fibres of the superior laryngeal nerves. The authors of the commentary write that the EMG neurogenic pattern is indicative of motor but not sensory neuropathy [1], which is of course true, but it should be added that, as we stated above, motor neuropathy very often coexists with sensory neuropathy, and these are not mutually exclusive conditions.

As mentioned in the introduction of the commented article, the diagnosis of LSN is difficult and requires many exclusions. Therefore, the title of our article refers to EMG findings in LSN. In our opinion, the title does not suggest the use of EMG as a diagnostic criterion, but rather a description of findings that may correspond to the patient's symptoms. Regarding the questions about the differential diagnosis of the patients, as we described in the methodology section, the patients referred to us had already been diagnosed by internists, neurologists, cardiologists, and pulmonologists. None of the patients presented with thyroid dysfunction, spasmodic dysphonia, or sicca syndrome. In the absence of a diagnosis to explain the laryngeal complaints, patients were referred by other specialists to an ENT/phoniatrician. We did not question the results of the differential diagnoses that had previously been made by various specialists. All patients had undergone cerebral MRI with contrast during neurological diagnosis at other centres. Due to the absence of other signs of the nervous system dysfunction and symptoms suggestive of laryngeal disorder, examination of the cerebrospinal fluid (CSF) was not considered. The authors support the previous decision of the neurologists that there were no medical indications and ethical concerns regarding the need for CSF examination in the patients presented in the article.

Regarding patient 5, this individual was not a chronic user of pregabalin. Due to an episode of neuropathic V nerve pain years ago, the neurologist had recommended trying pregabalin for laryngeal symptoms prior to presenting to our hospital. The medication was discontinued due to lack of improvement.

Regarding the question about patient 1 with an EMG recording showing vocal fold muscle weakness, myopathy was ruled out by neurological examination. It should be noted that other conditions associated with vocal fold

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muscle weakness, such as hypofunctional dysphonia, also show EMG changes of this type, which are not necessarily indicative of myopathy. The type of EMG recording does not indicate the cause of the disease, but only the type of damage to the motor unit. All patients associated the sudden onset of symptoms with COVID-19 infection. We and clinicians from other disciplines were unable to find any other cause for the disorders. We included in the methodology the exclusion of asthma exacerbation at the time of diagnosis in the phoniatrics centre. This was in accordance with the literature review of LSN differential diagnosis. Infection-induced asthma exacerbation was noted 1.5 years before the diagnostics in our department in our department. On admission, the patient had well-controlled asthma according to the pulmonologist. Other specialists had ruled out neurological, cardiovascular, and pulmonary diseases as possible causes of symptoms. This was the reason for the referral to our centre and the detailed diagnosis of the voice.

Over time, the patients do not report any new symptoms and their LSN complaints are decreasing. When preparing to publish this Letter to the Editor, we contacted patients and asked them to report the severity of their symptoms. Three patients reported the disappearance of their symptoms, one patient reported periodic symptoms of low severity, and two patients reported no change in their symptoms.

In our work, the patients were not tested with the depression scales. Depression scales are not the standard questionnaires we use to assess patients with voice disorders. We look at the interview (where we ask about other medical care received). We also ask the patients to complete the Voice Handicap Index (VHI) questionnaire. However, all the patients described in the article were diagnosed by us as part of a multidisciplinary diagnostic inpatient assessment, which includes a psychological assessment as well as ENT/phoniatric and logopaedic assessment. None of the discussed patients were found by the psychologist to be at risk of depression.

We hope that we have provided an exhaustive response to the issues raised by the authors of the letter to the editor [1]. We would like to thank the specialists in neurology and neurophysiology for their comments and insightful analysis of the article and for highlighting the relevant issues from their point of view. We are specialists in otolaryngology, audiology, and phoniatrics. Our interest and attention in the article is mainly focused on aspects of our specialties and the results of our diagnoses. In our opinion, since various differential diagnoses ruled out any other cause of the laryngeal dysfunction manifested as hoarseness and sensory disturbances that began shortly after COVID-19, it is reasonable to suspect SARS-CoV-2 infection as the cause of LSN.

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Data availability Data available on request from the authors.

Declarations

Conflict of interest We assure that no conflict of interest, real or perceived, exists.

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