



Spinal Cord Lesions and Movement Disorders

Dear Editor,

We read the article entitled, “Tremors as an Atypical Presentation of Cervical Myelopathy” on the esteemed “Journal of Medical Sciences” with great interest. Goh *et al.* reported a case of an elderly male who presented with tremors, unsteady gait, and loss of dexterity. A cervical magnetic resonance imaging done showed large disc herniation at the C3–C4 level. Later, anterior cervical discectomy and fusion of the region were performed, and the patient had full recovery.¹

Here, we address some topics that, together with the study of Goh *et al.*, could lead to a better comprehension of spinal cord lesions and movement disorders.

First, one possible pathophysiological explanation for the case presented by Goh *et al.* could be the fact that the lesion in the cervical region probably affected only some motor neurons.² More specifically, a few numbers of neurons were possibly damaged without the involvement of an entire fascicle. The clinical neurological examination results with the presence of upper motor neurons signs with normal strength described by Goh *et al.* can support this hypothesis. Furthermore, this presentation is commonly seen in reference spinal disorder centers; however, as Goh *et al.* stated, it is probably underreported.³

Second, their report lacks information about electrodiagnostic studies, which could have collaborated with the explanation of a pathway involved with the presenting clinical manifestations. Electromyography and electroencephalogram are essential for a better characterization of movement disorders, secondary to spinal cord lesions.

Third, the author mentions myoclonus and pseudoathetosis occurring secondary to myelopathy. These are well-known conditions secondary to spinal cord lesions, which are explained by an abnormal neuronal pathway. They could be primary or secondary to traumatic/vascular lesions in the spinal cord. These pathological pathways can be visualized by diffusion tensor imaging and tractography of the spinal cord.⁴ It is worth mentioning that tremor is easily mistaken for myoclonus in the clinical practice, especially when movement disorders’ specialists are not available for consultation.

Fourth, there are, in the literature, several hypotheses to explain the essential tremor caused by cervical lesions. One of them is based on the knowledge of pathways to the thalamus, which were investigated after individuals with lesions localized in the brainstem presenting tremor in the absence of upper motor neuron signs.⁵ Interestingly, the oscillating central

network of tremor involves mainly four structures: cortical motor areas, thalamus, brainstem, and muscle. It is noteworthy that physiological studies about these four structures came to very distinct conclusions on which area was the main responsible for the central control of tremor; all of these areas have already been reported individually as the culprit because researchers observed metabolic and electrical activity in each of those regions separately. Thus, it is possible that each network component may act as a dynamically changing oscillator on its own.⁶

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Conflicts of interest

There are no conflicts of interest.

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