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Letter to the Editor

Axonal Motor Neuropathy in a Patient with Unintentional Weight Loss Should Not Remain Unexplained

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Letter to the Editor

We read with interest the article by Kheirbeck *et al.* regarding a 47-year-old man with a 13-year history of Sjögren's syndrome who developed a slowly progressive axonal motor neuropathy that manifested as progressive tetraparesis over the last five years ^[1]. Despite treatment with cyclophosphamide, plasma exchange, glucocorticoids, and rituximab, the patient showed no significant clinical improvement ^[1]. The study is noteworthy, yet several points warrant discussion.

A first point is that the cause of the axonal neuropathy was not identified ^[1]. Given that the symptoms of axonal motor neuropathy had begun five years earlier and the patient had unintentionally lost 14 kilograms of weight in the three years prior to presentation, it is crucial to consider a neoplastic or paraneoplastic neuropathy. Was the patient tested for other antibodies associated with paraneoplastic neuropathies in addition to anti-Hu, -Yo, -Ri, -amphiphysin, and -CV2? These antibodies include ANNA1, CRMP5, anti-amphiphysin, anti-ganglionic acetylcholine receptor antibodies, SOX1, PCA2, and MAP1b ^[2]. Was malignancy ruled out via computed tomography (CT) of the thorax and abdomen, gastroscopy, colonoscopy, and fluorodeoxyglucose (FDG) positron emission tomography (PET)? What was the cause of the unintentional weight loss?

A second point is that hereditary neuropathy was not considered or ruled out ^[1]. Was the family history positive for neuropathies? Hereditary neuropathies manifesting as purely motor and axonal lesions include distal hereditary motor neuropathy, SORD-associated neuropathy, HINT1-associated axonal neuropathy, and motor-predominant CMT2 subtypes ^[3].

A third point is that no cerebrospinal fluid (CSF) analysis was performed on the patient ^[1]. CSF analysis would have been crucial to rule out polyradiculitis, multifocal motor neuropathy, intoxication, and neuroborreliosis.

A fourth point is that no information was provided regarding comorbidities or concomitant medications ^[1]. Since various medications can cause neuropathy, knowledge of current medication is of critical importance. Medications known to trigger neuropathies include chemotherapeutic agents (platinum derivatives, taxanes, vinca alkaloids), antibiotics (metronidazole, nitrofurantoin), amiodarone, nucleoside analogues (reverse transcriptase inhibitors), phenytoin, carbamazepine, statins, thalidomide, and dapsone ^[4]; therefore, it is essential to know which medications the patient was taking regularly. What medications did he receive for Sjögren's syndrome?

A fifth point is that there was no report on whether the patient had previously had a SARS-CoV-2 infection ^[1]. It is known that SARS-CoV-2 infections can be associated with neuropathy ^[5].

Finally, the question arises as to what is meant by "predominantly motor." Were there also sensory disturbances or abnormalities in sensory nerve conduction velocity studies? Was the autonomic nervous system also affected?

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