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Letter to the Editor

Before Attributing Flaccid Quadruparesis with Sensory Neuropathy and Posterior Column Hyperintensity to Vitamin-B12 Deficiency, All Differential Cause must be Ruled Out

Josef Finsterer
Department of Neurology, Neurology & Neurophysiology Center, Vienna, Austria

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Corresponding Author: **Josef Finsterer**

Letter to the Editor

We read with interest the article by Zolfagbari *et al*. about a 19-year-old woman who developed progressive flaccid tetraparesis after a respiratory infection, leading to suspicion of Guillain-Barré syndrome (GBS) [1]. However, further investigations revealed normal motor nerve conduction studies (NCS) but sensory neuropathy, macrocytic anemia, vitamin B12 deficiency, and hyperintensity of the dorsal columns C1 to T3 [1]. With vitamin B12 supplementation, the tetraparesis had improved at the three-month follow-up examination [1]. The study is interesting, but some points require discussion.

The first issue is that the flaccid tetraparesis remained unexplained [1]. The motor NCS were normal, ruling out motor neuropathy. Needle electromyography of several muscles was also normal. Hyperintensities of the posterior column also do not explain the tetraparesis. If the tetraparesis is nevertheless attributed to subacute combined degeneration (SCD) of the spinal cord, a disease that affects the lateral and posterior columns of the spinal cord due to demyelination, one would expect increased tendon reflexes and spasticity, but not areflexia and hypotonia [1]. Based on these considerations, it would have been essential to rule out all possible differential diagnoses of acute tetraparesis. In this regard, the results of several examinations are missing, such as magnetic resonance imaging of the brain with contrast agent, cerebrospinal fluid examinations, screening for malignancy, and exclusion of a myelin oligodendrocyte-associated disease (MOGAD, leukoencephalopathy with involvement of the brain stem and spinal cord and lactic acidosis (LBSL)), Leigh syndrome, and neuromyelitis optica spectrum disorders (NMO-SD). NCS may be normal in the early stages of GBS, which is why they must be repeated over time.

The second issue is that the cause of the vitamin B12 deficiency was not explained [1]. Was the vitamin B12 deficiency due to low intake (as in vegans or vegetarians), antibodies against parietal cells in the small intestine (pernicious anemia), Crohn's disease, impaired absorption, intestinal surgery, chronic alcoholism, or the use of metformin, certain antiepileptic drugs, or proton pump inhibitors? Did the patient regularly inhale nitrous oxide for recreational purposes?

The third point is that neuropathy due to vitamin B12 deficiency is usually sensorimotor [2]. It should be explained why only sensory fibers were affected in the index patient and why motor fibers were preserved. Were NCS performed repeatedly during the course of the disease? Did the motor fibers remain unaffected throughout the course of the disease?

The fourth point is that the patient did not fully recover with vitamin B12 supplementation [1]. We should know the long-term outcome of the patient and whether the tetraparesis persisted despite adequate vitamin B12 supplementation.

In summary, flaccid tetraparesis cannot be explained solely by vitamin B12 deficiency if the NCS of the motor nerves are normal.

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