LETTER TO THE EDITOR EDİTÖRE MEKTUP

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The Hidden Cause of Presentation with Ataxia: Subacute Combined Degeneration of the Spinal Cord without Hematologic and Imaging Findings

Ataksi ile Başvurunun Gizli Nedeni: Hematolojik ve Görüntüleme Bulguları Olmadan Medulla Spinalisin Subakut Kombine Dejenerasyonu

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Dear Editor,

A 64-year-old male patient was admitted to our clinic with complaints of ataxia. There were no features in the medical, family, or nutritional history. In the physical examination, the muscle strength was full, and no sensory deficit was observed. Urine/stool was continent. The deep tendon reflexes were normoactive. The proprioceptive sensory was normal. The gait was ataxic. Romberg's test was positive. The vibration sense was decreased. At the admission examination, the Berg Balance Scale (BBS) score was 39. It was learned that the cobalamin value had been 103 pg/mL and below for approximately 9 years in past laboratory results and that the patient did not use the cobalamin. Hemoglobin and serum folic acid levels, mean corpuscular volume, and acute phase reactants were normal. Brain and spinal magnetic resonance imaging (MRI) and electroneuromyography were normal. Tissue transglutaminase IgA-IgG levels, gastric endoscopy, and biopsy were normal. We started intramuscular hydroxycobalamin and oral folic acid for the suspicion of subacute combined degeneration (SCD) of the spinal cord. A tripod was provided for safe ambulation. The physical therapy program was planned as strengthening, balance/coordination exercises, whirlpool, and exercises with computerized support devices. After 4 weeks, the patient's BBS score increased to 42, and he was able to ambulate independently.

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In spinal cord SCD, demyelination develops in the lateral and posterior columns due to cobalamin deficiency. The deep sensation is impaired in the dorsal column involvement. Lateral column involvement was accompanied by upper motor neuron findings. Romberg positivity and sensory ataxia are observed in spinocerebellar tract degeneration. Rapid diagnosis and treatment are important to ensure that neurological symptoms do not become irreversible.[1] In our case, the findings were consistent with dorsal column and spinocerebellar tract involvement.

Cobalamin deficiency usually occurs because of malnutrition, malabsorption, or as a side effect of some medications. In terms of nutritional history, the consumption of animal products is particularly important. Some gastrointestinal system pathologies can result in malabsorption.[1] In our case, cobalamin deficiency could be due to malnutrition, as we excluded others.

On MRI, hyperintensities can be observed in the dorsal columns in the early stages and lateral columns in the advanced stages of the cervical and upper thoracic spinal cord on T2 sequences.[1] However, lesions may not have been observed as in our patient.[2] Differential diagnosis includes copper deficiency, myelopathy induced by methotrexate use, and many diseases localized to the spinal cord that may cause spinal cord damage. Copper deficiency should be considered in cases where there is no response to cobalamin treatment alone. MRI is used in the differential diagnosis of other diseases localized to the spinal cord.[3] Cobalamin response and lack of history of drug use supported the diagnosis of SCD.

Folic acid-added cobalamin supplementation is used in the treatment because severe inhibition of cobalamin-dependent methionine synthase may also decrease folate levels.[4]

Clinical improvement in neurological symptoms in these patients typically takes at least 3-12 months.[1] However, the patient demonstrated early improvement in neurological symptoms due to the rehabilitation program.

SCD should be considered in patients with ataxia.

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Conflict of Interest

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