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Letter to the Editor

Does B12 deficiency lead to syringomyelia?

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A 20-year-old female was admitted to our clinic with the complaint of gait disturbance, which started three months ago and slowly progressed. Her medical history was non-specific. Her initial complaints began with numbness in her right foot and, then, under the left knee. Over time, activities such as sitting and standing up and climbing stairs became more difficult, and urinary incontinence started. Her physical examination revealed an ataxic gait pattern. Bilateral lower extremity strength was 4/5. Bilateral patellar reflexes were hyperactive. Bilateral Achilles reflexes were hypoactive. The plantar reflex was unresponsive at right and flexor at left. The superficial sensation was normal at lower extremities; however, there was a loss of deep sensation. The Romberg sign was positive. Laboratory test revealed that hemoglobin was 11 g/dL (reference: 13.6 to 17.2), mean corpuscular volume was 124 fL (reference: 80.4-95.9), serum vitamin B12 level was 80 pg/mL (reference: 190911), and liver and kidney function tests, electrolyte levels, blood glucose, glycosylated hemoglobin, C-reactive protein, and erythrocyte sedimentation rate were within normal limits. Nerve conduction studies showed that bilateral common peroneal, tibial, ulnar, and median nerve sensory and motor conduction were within normal limits. On spinal magnetic resonance imaging (MRI), a syrinx cavity was detected at the anterior spinal cord, which was 1-mm in thickness at it's the widest location (Figure 1a). The patient was consulted to the neurology department. Considering the clinical and the laboratory findings, she was diagnosed with subacute combined degeneration (SCD). Intramuscular hydroxocobalamin was given intramuscularly, every day for the first week, every week for the next month, and then every month for the next four months. In addition to the medical treatment, physical therapy as 20 sessions of lower extremity strengthening, balance and coordination exercises, and gait training were applied. At the first-month control, B12 level was 970 pg/mL. Spinal MRI at eight weeks was within normal limits. The syrinx cavity, which was observed at anterior previously, disappeared (Figure 1b). The Berg balance scale score increased from 25 to 55. She was discharged with a cure without needing a supportive device.

Neurological findings may include clinical pictures such as neuropathy, myelopathy, dementia, and optic neuropathy. Subacute combined degeneration, which is a rare form of myelopathy presenting with B12 deficiency, is characterized by the demyelination of the lateral and posterior cords of the spinal cord.[1] In the literature, syrinx cavities with 1 to 2-mm diameter are usually accepted as normal and follow-up is recommended for those that are above 3 mm in diameter.[2]

The patient was treated for B12 deficiency, and a rehabilitation program was applied for gait disturbance. Spinal MRI at eight weeks after the treatment was totally normal (Figure 1b). At her control visit after one year, she was ambulated with a normal gait pattern. No sensory or motor deficit was detected.

An interesting finding was that syrinx cavity regressed in our case together with symptoms after B12 treatment. No case is available in the literature

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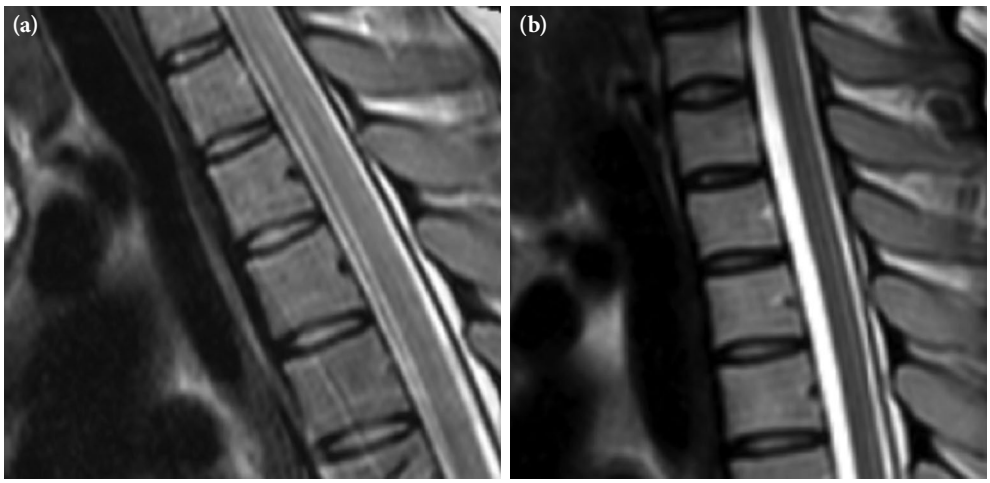

Figure 1. (a) T2 sequence of magnetic resonance imaging. A syrinx cavity in thoracic vertebra (before treatment). **(b)** T2 sequence of magnetic resonance imaging. Thoracic vertebra (after treatment).

presenting with a syrinx cavity and B12 deficiency. The cause of the syrinx cavity of the patient could not be identified. In the light of current knowledge, it is not possible to predict whether B12 deficiency causes a syrinx cavity or not. The lesions that are formed in medulla spinalis due to SCD usually disappear after six or eight weeks.[3] In our case, the syrinx cavity disappeared on MRI after eight weeks. This finding suggests that B12 deficiency may cause asymptomatic syrinx cavity.

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