A 57 years male who was a follow up case of hypertension and ischemic heart disease for around 20 years presented for last six months with complaints of staggering of gait which got aggravated in darkness, numbness, tingling and paresthesias in distal part of both upper and lower extremities. Patient also complained of slippage of slippers without getting aware of this phenomenon and decline in memory. Patient was a strict vegetarian and did not undergo any gastrointestinal surgery. On examination, pulse rate was 86/min, blood pressure 126/78 mm Hg and pallor was present. Central nervous examination revealed mini mental status score of 21/30. Fundus examination revealed early optic atrophy on both sides. Motor assessment showed grade 4/5 to 4+/5 power at all the joints. There was generalized hyporeflexia to areflexia. Plantar reflex were bilaterally non-elicitable. Sensory system revealed loss of vibration sensation in both upper and lower limbs accompanied by loss of joint position sense. Romberg sign was positive. On investigations Hb-10.6 gm/dl, general blood picture revealed macrocytic anemia and hypersegmented polymorphs. Mean corpuscular volume-110, mean corpuscular hemoglobin-34.2, mean corpuscular hemoglobin concentration-31.1 and Vitamin B12 level was 115 pg/ml. Thyroid profile revealed raised TSH levels (T₃-1.40, T₄-72.85, TSH-19.66). Nerve conduction study was suggestive of axonal sensorimotor neuropathy. Magnetic resonance imaging delineated hyperintense signals in cervicothoracic posterolateral columns on T₂ and flair images. Magnetic resonance imaging of the cranium revealed generalized cerebral and cerebellar atrophy. So with this profile patient was diagnosed as case of vitamin B12 deficient myeloneuropathy with mild dementia along with hypothyroidism. Patient was given vitamin B12 I/M injections according to schedule and thyroid hormone supplementation. Patient showed dramatic recovery at follow up after four weeks. 

Apart from the well-characterized hematological changes of vitamin B12 deficiency, a variety of neurological impairments have been described. Typically, signs of peripheral neuropathy and myelopathy can be observed: distal paresthesias, impairment of vibratory and position sense, reduced ankle jerks. In more severe cases symmetrical limb weakness and other pyramidal deficits can occur. Demyelination produced by B12 deficiency can be recognized on magnetic resonance imaging especially in the cervical-dorsal region. The lesion can be shown to be posteriorly located in posterior columns of spinal cord on the axial scans. Similar MRI finding of the posterior columns in vitamin B12 deficiency has been described in recent paper. We proposed that magnetic resonance imaging of cervical-dorsal spine showing characteristic posterolateral column hyperintensities in addition to clinical and lab parameters could be diagnostic of vitamin B12 deficiency neurological syndromes.

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