Painless Legs and Moving Toes in Chronic Inflammatory Demyelinating Polyradiculoneuropathy

Sir

Painless legs and moving toes (PoLMT) are a subtype of painful legs and moving toes (PLMT) wherein the patients experience involuntary repetitive, non-rhythmic movements of the toes or fingers that occur in the absence of pain.[1] PLMT are more common and frequent in peripheral neuropathy.[2] There are very few reported cases of PoLMT in the literature and known to occur in spinal cord compression, parasagittal meninigioma, Wilson disease, and sacral Tarlov cyst. PoLMT in chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) has not been reported so far. Hereby, we report a 56-year-old lady who was diagnosed as CIDP based on clinical and electrophysiological criteria and had involuntary, non-rhythmic painless movements of toes and feet suggestive of PoLMT.

A 56-year-old lady presented with a history of burning paraesthesia in both lower limbs of 3 months duration, inability to grip footwears of 2 months, and difficulty in getting up from the squatting/sitting position with imbalance of 1 month duration. Symptoms of burning and tingling paraesthesia started in both distal lower limbs and ascended till mid-leg. One month later, she noticed difficulty in gripping footwears and used to slip with awareness, followed by progressive difficulty in getting up from squatting, climbing stairs. She had imbalance while walking, which was worse during night hours. She had painless involuntary movements of bilateral toes and foot of 1 month duration, which was non-progressive. There were no upper limb symptoms and craniobulbar symptoms. She did not have systemic symptoms. Systemic examination was unremarkable except dark pigmentation of distal lower limbs. Neurological examination showed normal cognition, speech, and cranial nerves. Motor examination showed normal tone and power in upper limbs, hypotonia in lower

limbs, grade 4-/5 power in proximal lower limb muscles, ankle dorsiflexors and plantiflexors, weak toe grip, and small muscles of the feet and generalized areflexia. Sensory examination showed reduced touch and pain sensation till mid-leg with absent vibration till anterior superior iliac spine and impaired joint position sensations in toes. There were no cerebellar signs, and plantar responses were mute. She had bilateral foot and toe involuntary, painless, repetitive, non-rhythmic, and non-suppressible movements, which are present both in eye closure and open, suggestive of PoLMT [Video 1]. Complete blood count, renal, hepatic, and thyroid function tests were normal. Nerve conduction studies showed reduced nerve conduction velocities in common peroneal and posterior tibial nerve with prolonged distal motor latency and reduced compound motor action potentials and absent sensory nerve action potentials in sural and superficial peroneal nerves, suggestive of demyelinating polyneuropathy in both upper and lower limbs, satisfying the 2021 European Academy of Neurology (EAN)/Peripheral Nerve Society (PNS) criteria. Serum protein electrophoresis was normal. The patient received 1 g of intravenous methylprednisolone for 5 days and had no significant improvement and subsequently received 5 cycles of large volume plasmapheresis. At 1-month follow-up, there was improvement in imbalance with ability to walk with mild support and reduced PoLMT. She was on azathioprine with a tapering dose of oral steroids. At 6-months follow-up, the patient was able to self-ambulate with no PoLMT.

The movements in PoLMT are spontaneous and purposeless and consist of complex sequences of flexion, extension, abduction, and adduction. The exact pathogenesis of PoLMT is unclear, but alterations in afferent sensory information with subsequent reorganization of segmental or suprasegmental efferent motor activity may explain the movements. Walters *et al*.[1] (1993) reported a case of PoLMT without any apparent etiology. Dziewas *et al*. (2003) reported PoLMT in mother and daughter with no apparent trigger and probable hereditary basis of PoLMT.[3] A few cases of PoLMT were reported following ischemic stroke, spinal cord compression, sacral Tarlov cyst, and parasagittal meningioma.[4-7] The associated symptom of PoLMT in CIDP has not been reported. We postulate it to be secondary to the impaired/mismatch sensory information from the extremities due to demyelination and aberrant efferent segmental motor activity causing involuntary movements. The involuntary movements reported in CIDP are the tremors and myoclonus. The tremors in CIDP are postural and kinetic upper limb tremors, which may be refractory to the treatment.[8]

This case adds PoLMT to the list of involuntary movements in CIDP apart from the more common postural and kinetic tremors of upper limbs and less common myoclonus. It is important to identify these involuntary movements in patients with CIDP.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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