Letter to Editor

Methylprednisolone induced hypokalemic paralysis mimicking relapse of Multiple Sclerosis

Rajesh Verma[1], Ritesh Shahu[2]

From, [1]Professor, Neurology. King George Medical University, Lucknow, UP, India. [2]Senior Resident, Neurology, King George Medical University, Lucknow, UP, India

Correspondence to: Dr. Rajesh Verma, Department of Neurology, King George Medical University, Lucknow, UP, India. E-mail: drrajeshverma32@yahoo.com

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A 30 year old woman, diagnosed case of multiple sclerosis on immunosuppressive therapy, admitted in neurology ward with acute onset quadriparesis with limb paresthesia without visual and bladder or bowel impairment. The present event was not exacerbated by fever or high temperature. There was no suggestion of concomitant infection. There was no history of cognitive dysfunction. She became dependent on family member for routine activities. Past history revealed two episodes of neurological disability with full recovery in last one and half years.

The neurological examination on day of admission revealed power of grade 4/5 in upper limbs and 3/5 in both lower limbs based on MRC scale. The deep tendon reflexes were brisk and plantars showed bilateral extensor response. The spasticity was evident in both upper and lower limbs. Magnetic Resonance imaging (MRI) brain showed increase in demyelinating lesion load suggested acute relapse phase of MS [figure1]. She was started on intravenous methylprednisolone therapy in a dose of 1000 mg/day. After one day of therapy, she developed deterioration of weakness and became bedbound. On examination, she was afebrile and vitals were stable. Higher mental function and cranial nerve examination were normal. Motor system examination revealed hypotonia in all four limbs with sluggish deep tendon reflexes. Power was grade 3/5 in both upper limbs and 2/5 in both lower limbs according to Medical research council (MRC) grading. Sensory examination was normal. Cerebellar examination was unremarkable.

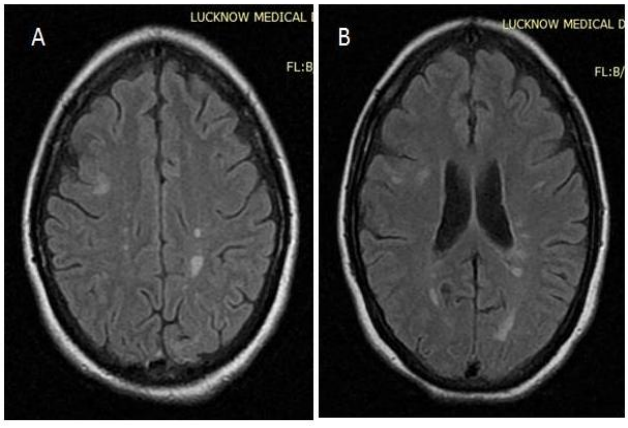

Figure 1: Magnetic resonance imaging brain axial T2 flair image showing multiple white matter hyper intensities involving periventricular and subcortical area suggestive of demyelination

Routine investigations like hemogram, blood sugar, renal and liver function test were normal except serum potassium level was found to be low (2.2mmol/L). Thyroid hormone level including thyroid peroxidase and thyroglobulin antibodies were normal. Creatine phosphokinase (CPK) level and nerve conduction study were normal. Patient was supplemented with oral potassium chloride. Next day, she showed improvement in limb weakness with return of normal tone. Repeat serum potassium level was within normal range (4.5 mmol/L).

Multiple sclerosis is demyelinating disease of central nervous system and usually presents as a relapsing-remitting course and many atypical forms. Generally acute relapse of multiple sclerosis presents with occurrence of new or worsening of existing neurological deficit [1]. High dose methylprednisolone therapy is recommended during acute relapse of multiple sclerosis. Development of hypokalemia leading to worsening of weakness and hypotonia is a potential early complication of methylprednisolone therapy. Possible hypothesis being increased Na/K-ATPase pump activity causing a rapid shift of potassium from the extracellular into the intracellular compartment [2, 3].

Differential diagnosis of flaccid paralysis in such cases should be considered are thyrotoxic periodic paralysis [4], steroid induced myopathy, acute inflammatory polyneuropathy. Thyrotoxic periodic paralysis can rarely present as acute muscle paralysis and prevalence is more common in Asian males. The triggering factors for thyrotoxic periodic paralysis include high carbohydrate diet, strenuous exercise and steroid therapy. Our patient had no evidence of thyrotoxicosis [5]. So while treating the acute relapse of multiple sclerosis, primary physician should be aware of hypokalemia as a potential complication of high dose corticosteroid therapy and hypokalemic paralysis should be considered if flaccid weakness develops during the corticosteroid therapy. Immediate potassium replacement is required to improve the motor weakness.

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