Finger Drop Following a Potassium Drop

Hypokalemic paralysis can occur primarily as a result of an enhanced shift of potassium ions into cells or secondarily from excessive potassium loss. It typically involves proximal muscles in a symmetric fashion. We describe a patient with asymmetric distal weakness that turned out to be due to hypokalemia and responded to potassium replacement.

A 65 year old gentleman woke up one morning to find that he could not extend 2nd, 3rd and 4th fingers of his left hand. The next day he experienced heaviness in his thighs and later fatigue and deep aches. Examination revealed grade 2 power on medical research council (MRC) scale in the extensors of 2nd, 3rd and 4th digits. Both lower limbs revealed MRC grade 4 power at hip while there was no weakness in any other muscle. Deep tendon reflexes were normal and symmetrical with flexor plantar responses. There was no sensory loss anywhere in the body. There was no facial, bulbar or respiratory weakness. Considering the mildness of the weakness, no definite treatment was offered. The next day the patient found it difficult to get out of his bed, needed support to walk and developed weakness in both the upper limbs proximally. The deep tendon reflexes continued to be normal. Nerve conduction studies suggested reduced compound motor action potentials with preserved distal latencies and conduction velocities in various motor nerves. Sensory nerve action potentials were normal from all sensory nerves.

Serum potassium levels were found to be 1.3 mmol/L. The EKG showed flattened T waves and U waves. The patient reported taking hydrochlorothiazide for previous 3 weeks. He had also been taking levothyroxine sodium for his hypothyroidism for the last ten years. Thyroid function tests were all reported normal. There was no family history of paralysis or hypokalemia. Hydrochlorothiazide was immediately discontinued. Potassium was replaced initially through intravenous infusion and later orally. The patient showed dramatic clinical improvement within 24 hours. Nerve conduction studies, repeated 48 hours later were found to be normal. Later the patient was discharged with the advice to avoid diuretics in future.

Hypokalemic paralysis is classically known to involve proximal musculature symmetrically before spreading to the distal muscles. This patient had the unusual presenting symptoms of asymmetric distal focal weakness. The subsequent clinical course suggested classical symmetric proximal muscle involvement consistent with hypokalemia while the preservation of deep tendon reflexes and absence of facial weakness were features against the initial suspicion of Guillain-Barre syndrome. It is difficult to explain the focal onset in a generalized metabolic disorder such as hypokalemic paralysis. Paralysis is caused by membrane depolarization triggering sodium channel inactivation, which renders the membrane inexcitable.1,2 In this context Cruz-Martinez, et al. found inexcitability of most muscle fibers during an acute attack.3 Activity dependent conduction block was induced by voluntary contraction, excitability abnormalities resolving with potassium replacement. The observation of muscle fiber conduction block may be compatible with our clinical finding of a focal onset of this generalized disease.

References

