Commentary

Acute neuromuscular paralysis is one of the common neurological emergencies. Guillain–Barré syndrome (GBS) and hypokalemic paralysis are the most common differentials of acute flaccid quadriparesis and therapeutic interventions greatly differ in both. GBS, also known as acute inflammatory demyelinating polyneuropathy (AIDP), is an autoimmune disorder. The onset can be sudden and unexpected and is primarily characterized by progressive limb weakness with areflexia. Recognition of this disorder is important because early detection of respiratory failure may limit complications and early therapy may limit nerve fiber loss and the extent of ultimate disability.[1] Several disorders, such as acquired hypokalemia, myasthenia gravis, periodic paralysis, and polymyositis, have symptoms similar to those found in GBS, making the diagnosis challenging. Electrolyte disturbances are not a part of the syndrome; however, autonomic dysfunction in GBS can lead to cardiac disturbances that may be aggravated by the coexistence of dyselectrolytemia such as hypokalemia and hypomagnesaemia.

There is a plethora of causes of low serum potassium, caused by acquired or inherited metabolic disorders of muscle ion channels or renal tubules. All organs can be affected by hypokalemia, resulting in a variety of clinical manifestations. The critical effect of potassium on neuromuscular conduction, particularly in the cardiac muscle, accounts for the major fatalities that accompany hypokalemia. Hypokalemic periodic paralysis is one of the many disorders that mimic GBS. It is an occurrence of low serum potassium during attacks of paralysis, primarily caused by an enhanced shift of potassium (K^+) into cells. The hypokalemic paralyses represent a heterogeneous group of disorders with a final common pathway presenting as acute weakness and hypokalemia. Most cases are familial; however, sporadic cases associated with diverse underlying etiologies including thyrotoxic periodic paralysis, barium poisoning, renal tubular acidosis, primary hyperaldosteronism and gastrointestinal potassium losses are also reported.[2] The approach to the patient with hypokalemic paralysis includes a vigorous search for the underlying etiology and potassium replacement therapy.

There have been a number of published reports of hypokalaemic weakness resembling GBS and these cases reinforce the need for awareness of the effects of electrolytes, particularly potassium, calcium, and magnesium, in both the clinical and electrophysiological assessment of patients with acute flaccid paralysis.[1] The most striking electrophysiological abnormality that is a severe and generalized reduction in CMAP amplitudes; in association with modest and mainly distal slowing of motor nerve conduction and some F wave latency prolongation seen in GBS patients is also compatible with the diagnosis of hypokalemic paralysis. But, the rapid recovery of CMAP amplitudes in association with the resolution of hypokalemia with improvement of muscle weakness, suggests that the low potassium is responsible for the clinical and electrophysiological abnormalities. A study was conducted by Shah *et al*.[3] in order to differentiate GBS from hypokalemic periodic paralysis on the basis of clinical features and simple laboratory investigations. They showed that 66.7% patients were diagnosed as GBS and all had serum potassium level between 3.9 and 4.6 mmol/l (mean level 4.72 mmol/l) at the time of presentation, while 15.1% had periodic paralysis and 12.1% were diagnosed as hypokalemic paralysis with serum potassium level of 1.5 to 2.5 mmol/l (mean 2.06 mmol/l) at the time of admission. They concluded that positive family history, similar episodes of weakness in the past, low serum potassium during the episodes of weakness, and quick recovery in 24-72 h with correction of electrolyte disturbance helps to differentiate between the two clinical entities.

The authors of "*Uncommon dyselectrolytemia complicating Guillain–Barré Syndrome*"[4] in this issue described an interesting case with rare association of GBS and Gitelman syndrome (GS) and they have made a great effort in correlating the same. The case highlights complex association of dsyelectrolytemia including hypomagnesemia and hypokalemia in a patient with GBS, which can be perplexing and pose diagnostic as well as therapeutic challenges. In their patient, the coexistence of hypokalemia, hypomagnesemia, and hypocalciuria occurred as an inherited renal tubular disorder akin to Gitelman syndrome.[5] It is an autosomal recessive renal tubular disorder due to mutations in the solute carrier family 12, member 3 gene SLC12A3 which encodes the thiazide sensitive sodium chloride cotransporter.[6] Diagnosis of GS is based on biochemical abnormalities characterized by hypokalemia, hypomagnesemia, hypocalciuria, and metabolic alkalosis. The diagnosis is confirmed by genetic studies. Patients with isolated Gitelman syndrome usually present at later age group with cramps and fatigue or may be asymptomatic and come to notice once biochemical parameters are assessed for some other disorder. Combination of GBS and comorbid dyseletrolytemia could be life threatening and it is challenging to treat complex dyselectrolytemia, especially when they are part of some metabolic disorder. The association of two syndromes which have same presentation with different therapeutic interventions is very rare and separation of the two is extremely important for good clinical recovery.

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