Section: Internal Medicine

Case Report

ACUTE FLACCID PARALYSIS: HYPERKALEMIA THE UNLIKELY CAUSE

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Abstract

Acute flaccid paralysis is considered as a medical emergency condition requiring urgent intervention. Various causes have been implicated most of which involves the neuro-muscular component. Metabolic causes have also been reported as a cause of acute flaccid paralysis most of which includes alteration in thyroid profile and reduction in serum potassium and magnesium levels. Hyperkalaemia as a cause of acute flaccid paralysis has not been reported as a common cause and only few cases have been reported. Metabolic abnormalities as a cause are very important to identify as they can be easily corrected and leads to complete reversal of the paralysis. We report here one such case with hyperkalaemia secondary to Diabetic kidney disease presenting as acute flaccid paralysis with complete recovery following normalization of serum potassium levels.

INTRODUCTION

Abnormalities of potassium is a common entity encountered in medical practice with varying manifestations.[1] The abnormalities need urgent correction to prevent fatal conditions especially cardiac arrhythmias which can lead to sudden death. Neurological manifestations such as muscle weakness and paralysis are commonly associated with reduction in potassium levels. However, there have been reported cases of hyperkalemia with neurological presentations.[2,3] The hyperkalemic paralysis due to Sodium channelopathy is a known entity. However, secondary hyperkalemia with acute flaccid paralysis remains scarcely reported.[4-7]

CASE PRESENTATION

A 54-year-old male presented with acute onset weakness of all four limbs progressed within eight hours of onset. There was no history suggestive of respiratory distress, bowel and bladder involvement, trauma or preceding infection. He was a diagnosed case of hypertension and Diabetic kidney disease on medical management. His physical examination revealed a blood pressure of 160/100 mm Hg and pulse rate of 106 bpm The neurological examination revealed flaccid paralysis of all four limbs with power of 1/5 (MRC grade) along with absent reflexes and mute plantar reflex. His sensory system examination and other system examinations were found to be normal. Given acute flaccid paralysis, the patient underwent an MRI of the brain and spinal cord as well as nerve conduction studies of all four limbs which were found to be normal. His metabolic profile revealed metabolic acidosis (pH – 7.212, HCO3 – 16.9). anemia with haemoglobin of 7.6 g/dl, Serum creatinine of 8.2 mg/dl, and hyperkalemia with Potassium of 8.2 meq/l. The rest of the laboratory investigations were unremarkable. ECG showed prolonged PR interval with widened QRS complex and tall T waves, a complete LBBB pattern - suggestive of severe hyperkalemia [Figure 1]. The patient was initiated on medical management for hyperkalemia with administration of regular insulin, calcium gluconate, bicarbonate, and beta-2 agonist nebulization. However, there wasn't any significant improvement in the potassium correction or the paralysis. The patient was then taken up for hemodialysis. Following one session of haemodialysis his potassium levels improved to 6.2 meq/l and power in the limbs improved to 3/5 (MRC grade). He was taken up for another session of haemodialysis the next day following which his

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potassium level had corrected to 4.2 meq/l with complete recovery of power in all four limbs Power 5/5 (MRC grade). His ECG abnormalities also normalized [Figure 2].

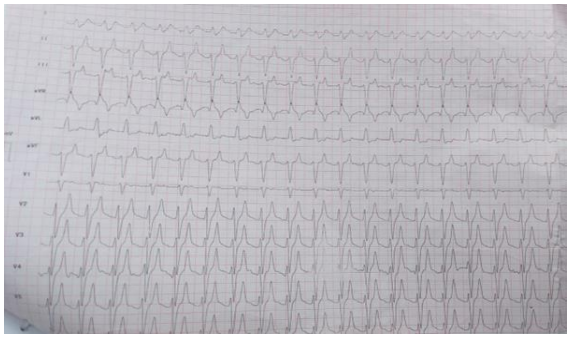


Figure 1: ECG at presentation suggestive of a complete LBBB pattern

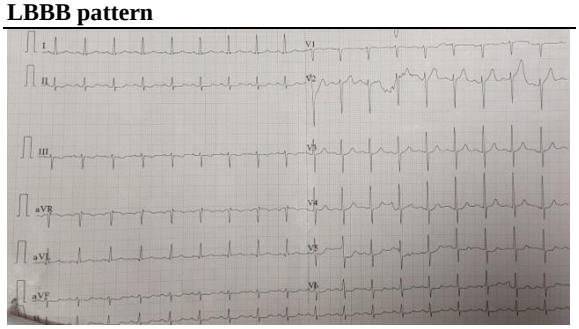
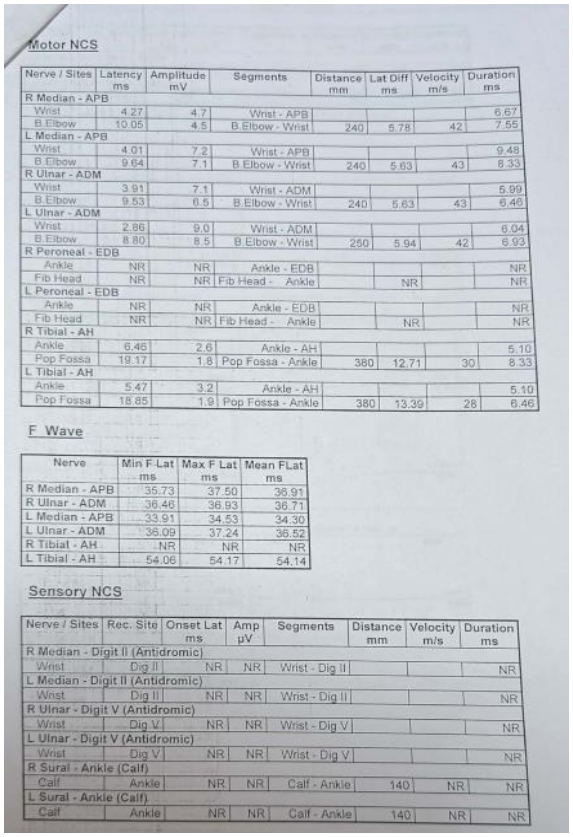


Figure 2: ECG after dialysis suggestive of Normal sinus rhythm with no abnormalities



Motor NCS

Nerve / Sites	Latency ms	Amplitude mV	Segments	Distance mm	Lat Diff ms	Velocity m/s	Duration ms
R Median - APB							
Wrist	4.27	4.7	Wrist - APB				6.67
B.Elbow	10.05	4.5	B.Elbow - Wrist	240	5.78	42	7.55
L Median - APB							
Wrist	4.01	7.2	Wrist - APB				9.48
B.Elbow	9.64	7.1	B.Elbow - Wrist	240	5.63	43	8.33
R Ulnar - ADM							
Wrist	3.91	7.1	Wrist - ADM				5.99
B.Elbow	9.53	6.5	B.Elbow - Wrist	240	5.63	43	6.46
L Ulnar - ADM							
Wrist	2.86	9.0	Wrist - ADM				6.04
B.Elbow	8.80	8.5	B.Elbow - Wrist	250	5.94	42	6.93
R Peroneal - EDB							
Ankle	NR	NR	Ankle - EDB				NR
Fib Head	NR	NR	Fib Head - Ankle		NR		NR
L Peroneal - EDB							
Ankle	NR	NR	Ankle - EDB				NR
Fib Head	NR	NR	Fib Head - Ankle		NR		NR
R Tibial - AH							
Ankle	6.46	2.6	Ankle - AH				5.10
Pop Fossa	19.17	1.8	Pop Fossa - Ankle	380	12.71	30	8.33
L Tibial - AH							
Ankle	5.47	3.2	Ankle - AH				5.10
Pop Fossa	18.85	1.9	Pop Fossa - Ankle	380	13.39	28	6.46

F Wave

Nerve	Min F Lat ms	Max F Lat ms	Mean FLat ms
R Median - APB	35.73	37.50	36.91
R Ulnar - ADM	36.46	36.93	36.71
L Median - APB	33.91	34.53	34.30
L Ulnar - ADM	36.09	37.24	36.52
R Tibial - AH	NR	NR	NR
L Tibial - AH	54.06	54.17	54.14

Sensory NCS

Nerve / Sites	Rec. Site	Onset Lat ms	Amp µV	Segments	Distance mm	Velocity m/s	Duration ms
R Median - Digit II (Antidromic)							
Wrist	Dig II	NR	NR	Wrist - Dig II			NR
L Median - Digit II (Antidromic)							
Wrist	Dig II	NR	NR	Wrist - Dig II			NR
R Ulnar - Digit V (Antidromic)							
Wrist	Dig V	NR	NR	Wrist - Dig V			NR
L Ulnar - Digit V (Antidromic)							
Wrist	Dig V	NR	NR	Wrist - Dig V			NR
R Sural - Ankle (Calf)							
Calf	Ankle	NR	NR	Calf - Ankle	140	NR	NR
L Sural - Ankle (Calf)							
Calf	Ankle	NR	NR	Calf - Ankle	140	NR	NR

Figure 3: Nerve conduction studies depicting Sensorimotor polyneuropathy involving both upper and lower limbs.

DISCUSSION

Hyperkalemia is a common clinical entity and is defined when the serum potassium level exceeds 5 meq/l. Mild increase in its level is usually asymptomatic, however, a higher increase in its level is associated with various clinical manifestations including cardiac arrhythmias and muscle weakness. Various mechanisms causing hyperkalaemia include either reduced excretion from the body or increased intake. Acute and chronic kidney diseases, reduced mineralocorticoid action in hypoaldosteronism, various drugs like beta-blockers, anti-inflammatory drugs, and calcineurin inhibitors are commonly implicated causes.[4]

Muscle weakness associated with hyperkalemia has been reported commonly, however, its presentation as acute flaccid paralysis is scarcely reported. The clinical presentation in such cases involves paralysis with absent reflexes without any sensory or cranial nerve involvement. The pathophysiology of paralysis involves its effect on the nerves and the muscles. Reduction in membrane potential after a change in the ratio of intracellular and extracellular potassium levels leads to partial depolarization of cell membrane and an increase in conduction velocity. This persistent increase eventually leads to reduction in membrane excitability through the inactivation of voltage-gated sodium channels manifesting as paralysis of muscles.[5-7]

The cause of hyperkalemia in our case was attributed to Diabetic kidney disease due to a pre-existing illness. Apart from the secondary causes, a primary cause has been described which is known as hyperkalemia periodic paralysis. It is caused by a genetic mutation involving the SCN4A protein of the sodium channel in the skeletal muscle. The clinical presentation in this entity involves repeated episodes of paralysis and usually begins in the first decade of life.[8,9]

Evaluation of acute flaccid paralysis involves ruling out various causes like acute myelitis, Guillain-barre syndrome, and myasthenia presenting as a crisis. Multiple modalities including imaging of brain/spine, nerve conduction studies, electromyography etc are performed to rule out various causes. Numerous earlier studies have noted nerve conductions in hyperkalemic paralysis revealing features of demyelination.[10] Although Our patient had markedly lower amplitudes with slowed conduction velocities in both lower and upper limbs [Figure 3], suggestive of axonal distal sensorimotor poly neuropathy which was attributed to long-standing diabetes and chronic kidney disease. Once a diagnosis of hyperkalemia-induced paralysis is confirmed, the goal of the management is prompt correction of potassium levels. It may involve medical management which includes administration of insulin, calcium gluconate, bicarbonate, beta 2-agonist nebulization as well as hemodialysis. In our case there was rapid correction of potassium levels

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and reversal of ECG changes and correction of paralysis following hemodialysis.

CONCLUSION

Hyperkalemia leading to acute flaccid paralysis is not commonly reported. It may lead to life threatening condition as it can lead to paralysis of respiratory muscles as well as cause cardiac arrythmias. Early identification of it as an implicating cause and prompt management can lead to complete recovery of paralysis and prevent mortality.

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