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TWO CASES OF HYPERKALEMIA PRESENTING AS ACUTE DEMYELINATING POLYNEUROPATHY: CLINICAL AND ELECTROPHYSIOLOGICAL REVERSIBILITY WITH IN 72 HOURS WITH POTASSIUM CORRECTION.

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ABSTRACT

GullianBarre syndrome (GBS) is the most common cause of acute flaccid paralysis.Hypokalemia can present with flaccid paralysis and nerve conduction studies show axonal neuropathy.Here we present two cases who were initially admitted with suspicion of GBS but later on baseline investigations showed very high serumpotassium and in both cases nerve conduction showed acute demyelinating polyneuropathy.They were admitted in high dependency units and urgent dialysis was done.In first case NCS were repeated 72 hours of correction of hyperkalemia and they showed significant improvement. In second patient NCSwere repeated after 24 hours and they showed mild improvement in all parameters.

Background

GullianBarre Syndrome is the most common cause of acute flaccid quariparesis.One of the important differential of GBS is hypokalemia which is not only clinically similar but also gives similar changes in nerve conduction studies.We are presenting two cases of hyperkalemia which were initially admitted with suspicion of GBS but laboratory tests showed hyperkalemia.When we did nerve conductionstudies, changes were very similar to GBS.Moreover, electrophysiological changes improved with treatment of hyperkalemia.

Case 1

70 years old patient presented in the neurology outpatient clinic withprogressive weakness of all four limbs for the last 1 week.It was preceded by a diarrheal illness that settled spontaneously. Weakness started in the legs, and for last 2 days it involved the arms as well.She was having mild numbness of arms as well. She had normal sphincter control and did not have any difficulty in breathing or swallowing. The rest of her neurological inquiries likeconsciousness,vision,hearing, speech,comprehension were normal.Systemic inquiries were normal. She was havingdiabetes mellitus and hypertension.On examination, she was well oriented with normal vital signs. Her cranial nerves examination was normal. Power was MRC Grade 4/5 in all four limbs.Her reflexes were diminished and planters were flexor bilaterally. There was stockings type sensory loss in the legs.

Table 1. Baseline investigation (Case1)

CBC	**WBC**	**HB**	**PLATLETS**	
	21500	11.30	306K	
LFTs	AST	ALP	ALP	Billirubin
	16	15	54	0.66
RFTs	Serum Urea	Serum Creatinine		
	57	2.98		
Serum Electrolytes	Serum Sodium	Serum Potassium	Serum chloride	
	113	7.2	91	

She was admitted in the High Dependency area, she developed bradycardia,breathing difficulty and was intubated and put on ventilator.Immediate nephrology consult was sought and hemodialysis started. Nerve conduction studieswere done (Fig 2a) which showed acute demyelinating neuropathy probably superimposed on background diabetic axonal neuropathy. Repeat NCS done 72 hours later showed marked improvement (Fig 2b).

Next day the patient improved , she was extubated. Serum potassium and sodium repeated after dialysis was 4.4 and 131 respectively.She was clinically improving and started to walk with support.

Table 2(a): Nerve Conduction Studies on admission

Nerve Tested Right side (MOTOR)	**Site**	**Latency (ms)**	**Amplitude (mV)**	**Conduction Velocity (m/s)**	**F-Wave (ms)**
Median	Wrist	6.40	4.90	28	45.7
	Elbow	14.60	4.10		
Ulnar	Wrist	5.0	6.20	28	48.2
	Below elbow	11.30	5.60		
	Above elbow	14.90	5.40		
Tibial	Ankle	9.50	2.10	32	82.40
	knee	21.2	2.10		
Peroneal(TA)	Below Knee	6.60	3.0	36	
	Above knee				
SENSORY			(μV)		
Median		7.40	12	23	
Ulnar		7.00	15	21	
Radial		4.60	15	30	
Sural			NR		
Superficial peroneal			NR		

Table 2(b): Nerve Conduction Studies after 72 hours

Nerve Tested Right side **(MOTOR)**	Site	Latency (ms)	Amplitude (mV)	Conduction Velocity (m/s)	F-Wave (ms)
Median	Wrist	4.9	3.5	43	31.8
	Elbow	9.5	3.1		
Ulnar	Wrist	3.4	6.1	50	31.3
	Below elbow	7.6	5.8		
	Above elbow	9.5	5.7		
Tibial	Ankle	4.7	6.5	33	56.2
	knee	15.5	7.3		
Peroneal(TA)	Below Knee	3.3	4.6	67	51.9
	Above knee	4.5	4.5		
SENSORY			(μV)		
Median		4.3	6	41	
Ulnar		3.7	5	37	
Radial		2.1	8	63	
Sural			NR		
Superficial peroneal			NR		

Case 2

Forty three years old male, known diabetic for the last 20 years. He suffered from end stage renal disease and was on regular hemodialysis when he missed his scheduled dialysis. He presented in emergency department with difficulty in getting up from chair.He was well before that and was able to walk but for last 3 days he developed weakness of limbs and was unable to get up from chair. He also had a history of gastroenteritis 2 days before onset of weakness.He was had a family history of diabetes mellitus,hypertension and chronic kidney disease.Drug history includes using insulin, tab Carvedilol 6.25 mg 1+1,tabHydralazine 1+1,tab Aspirin 75 QD,Calcium and vitamin D supplements.His general physical and systemic examination was normal. Power on MRC scale was 5/5 in upper limbs, it was 3/5 in lower limbs, with absent reflexes and flexor plantars. Cranial nerve,sensory system and cerebellar examination were normal. The patient was admitted in High Depenency unit and dialysis started. Patient started to improve as his serum potassium was corrected.

Table 3: Baseline investigationCase 2

CBC	**HB** 10.40	**WBC** 8200	**Platlets** 330,000
RFTs	Urea 67	Creatinine 7.86	
Serum	Sodium	potassium	chloride
Electrolytes			
	128	6.3	95

Discussion

GullianBarre syndrome is a post-infectious polyneuropathy which presents as acuteflaccidquariparesis.Low serum potassium level can present with weakness mimicking GBS1. Hypokalemia is relatively a common disorder causing acute flaccid weakness of limbs. In patients presenting with acute flaccid quadriparesis, we should check serum potassium before making diagnosingGullianBarre Syndrome.Hyperkalemia can also causeweakness of limbsand is rare cause of weakness mimicking GBS.It is an ascending type of weakness that ascends upward from legs to arms and trunk.Cranial nerves ,sensations and sphincters remain intact and respiratory failure is rare2. Primary hyperkalemic paralysis which is a genetic

Table 4(a): Nerve Conduction Studies of 2nd patient on admission

Nerve Tested Right side **(MOTOR)**	Site	Latency (ms)	Amplit-ude (mV)	Conduct-ion Velocity (m/s)	F-Wave (ms)
Median	Wrist	6.2	4.5	36	39.6
	Elbow	12.3	3.6		
Ulnar	Wrist	4.3	1.3	36	NR
	Below elbow	10.9	0.3		
	Above elbow	14.0	0.3		
Tibial	Ankle	6.4	0.2		NR
	knee				
Peroneal(TA)	Below Knee	6.60	3.0	43	NR
	Above knee				
SENSORY			(μV)		
Median			NR		
Ulnar			NR		
Radial		2.5	7	48	
Sural			NR		
Superficial peroneal			NR		

Table 4(b): Nerve Conduction Studies of 2nd patient after 24 hours

Nerve Tested Right side **(MOTOR)**	Site	Latency (ms)	Amplit-ude (mV)	Conduct-ion Velocity (m/s)	F-Wave (ms)
Median	Wrist	6.1	5.7	35	38.5
	Elbow	12.4	4.4		
Ulnar	Wrist	3.1	1.6	34	NR
	Below elbow	9.5	0.9		
	Above elbow	13.4	0.8		
Tibial	Ankle	5.0	2.5	32	NR
	knee	18.0	1.7		
Peroneal(TA)	Below Knee	4.1	2.9	59	NR
	Above knee	5.8	2.9		
SENSORY			(μV)		
Median			NR		
Ulnar			NR		
Radial		2.7	7	45	
Sural			NR		
Superficial peroneal			NR		

disease of sodium channel is common and usually presents in the first decade of life but secondary hyperkalemia leading to acute flaccid quadriparesis is anuncommon phenomenon3,4.Here we have presented two cases ofacute flaccid weaknesswho were initially admitted with a suspicion of GBS but later on they were found to have hyperkalemia. Electrophysiological studies of bothcases showed prolonged distal latencies and decreased conduction velocities on presentation .In both patients,immediate dialysis was done to correct serum potassium and they improved.In first patient we repeated studies after 72 hours and they were significantly improved and some axonal neuropathic features were present because of underlying diabetic axonal polyneuropathy.In second patient studies repeated after 24 hours and they showed mild improvement in all parameters. The proposed mechanism of hyperkalemia induced paralysis is either effect of high potassium on muscle fibers and cell membranes orit might be associated with neuropathy by high serum potassium level5,6.High potassium also inactivates sodium channel and it would cause impairment of action potential generation and lead to reduced amplitudes of sensorimotor nerves7.Impairment of nerve conduction leads to prolonged latencies ,conduction blocks and decreased conduction velocities8.These changes are reversible as evident in our cases and they should be done after three days of correction of poatssium as seen in our first case where most of nerve conduction changes have improvedsignificantly. The changes seen in both cases are predominantly demylinating with prolonged latencies and decreased conduction velocities and some reduction in amplitudes of combined muscle action potentials.Hyperkalemia induced weakness is very similar to GBS and electrophysiological studies may show changes similar to it.Sensory system usually remains normal in both hyperkalemic paralysis and GBS9,autonomic dysfunction is a feature of GBS and not associated with hyperkalemia, however arrhythmias may occur due to hyperkalemia10. We suggest that Serum potassium must be checked before diagnosing GBS as treatment is completely different.

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Wasim Tariq Mallick: Study concept and design, protocol writing, data collection, data analysis, manuscript writing, manuscript review
Arsalan Ahmed: Study concept and design, data analysis, manuscript writing, manuscript review