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# Acute Onset of Quadriplegia Secondary to Hypoparathyroidism: Mimicker of AMAN Variant of GBS

Syeda Saba Zaidi<sup>1</sup>, Tahira Siddiqui<sup>1</sup> and Sarwar Jamil Siddiqui<sup>2</sup>

## ABSTRACT

Acute onset of quadriplegia is a rare phenomenon seen with hypocalcemia due to hypoparathyroidism. We describe a 33-year gentleman who presented with weakness of all four limbs and areflexia. ECG showed QT abnormality. Nerve conduction study revealed normal sensory and significantly low motor CMAP amplitudes in both the upper and lower extremities. This nerve conduction study can be seen in acute motor neuropathy of various etiologies, among which GBS is the most worrisome. Our patient found to have low calcium and parathyroid hormone level. His symptoms improved after calcium replacement. Occurrence of quadriplegia in hypoparathyroidism, and its improvement after correction of calcium, suggests crucial role of calcium in neuromuscular transmission. One should suspect electrolyte imbalance, like hypocalcemia in patients presenting with nerve conduction features of AMAN variant of GBS.

**Key Words:** Hypocalcemia. Quadriplegia. Hypoparathyroidism. Nerve conduction study. Acute motor neuropathy.

## INTRODUCTION

Primary hypoparathyroidism is defined as inadequate parathyroid hormone activity, due to which serum calcium concentration falls below reference range. Causes of primary hypoparathyroidism can be acquired or hereditary. On the other hand, secondary hypoparathyroidism is a state in which parathyroid hormone levels are low due to primary pathologies that lead to hypercalcemia. Common clinical characteristics of hypoparathyroidism include muscle spasm, paresthesias, perioral numbness, tetany and seizures. Other neurological manifestations are depression, psychosis, movement disorders like chorea, athetosis, bradykinesia, hemiballism and peripheral neuropathy.<sup>1,2</sup>

We describe a case of hypocalcemia due to idiopathic primary hypoparathyroidism presented with acute onset of quadriplegia and sequential changes in nerve conduction study. To our knowledge, this is the first case report which elaborates the significance of low motor CMAP amplitude changes in hypocalcemic weakness.

## CASE REPORT

A 33-year gentleman, who was in a good health a day before presentation, suddenly developed stiffness in the lower limbs, followed by heaviness and a tendency to fall. Over the next 24 hours, his weakness progressed to an extent that he was not able to lift his legs off the bed and had difficulty in holding things in his hands.

On examination, he was alert and oriented. Visual fields, optic discs and extraocular muscles were normal. There was no facial paralysis; gag reflex was intact and neck flexion was strong. Bulk was normal, tone was decreased in all four limbs, and power grade was 0/5 in all limbs (MRC scale), except in right upper limb where it was 1/5. Reflexes could not be elicited even on reinforcement. Plantars were equivocal. Joint position, vibration, touch, position were intact.

Investigations showed normal hemoglobin, leucocyte count, blood glucose, urea, serum creatinine, electrolytes, albumin, and liver function. Serum calcium level was 5.6 mg/dl (8.5-10.2 mg/dl) and phosphate level was 2.6 mg/dl (2.5-4.5 mg/dl). Serum magnesium was 1.5 mg/dl (1.7-2.2 mg/dl). Serum PTH was 5.98 pg/ml (16-87 pg/ml). Vitamin D level was found to be 20.6 ng/ml (< 30 ng/ml). Electrocardiogram revealed QTc of 0.54 s.

Electrophysiological study was done. Motor study in upper extremities show low CMAP amplitudes of median nerve on both sides, with normal distal latencies and conduction velocities. Right ulnar nerve showed low CMAP amplitude, normal distal latencies and conduction velocities. In lower extremities, Peroneal and tibial motor studies showed low CMAP amplitudes, normal distal latencies and conduction velocities. Absent H reflex was consistent with polyneuropathy. Although peroneal F responses were absent, this finding was of unclear significance because peroneal F responses are difficult to obtain in some normal individuals (Table I). Sensory nerve conduction studies were normal.

A diagnosis of hypocalcemia due to hypoparathyroidism leading to significant motor CMAP amplitude changes in both upper and lower extremities was made. Patient received 10 ml of calcium gluconate IV (90 mg elemental calcium). His symptoms started to improve after 24 hours. He was kept on oral combination of Calciferol:100IU

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**Table I:** Motor nerve conduction study.

Nerve-muscle	Stim-site	Latency		Dist	Amplitudes		NCV		F-lat		Duration	
		R	L		R	L	R	L	R	L	R	L
Median (APB)	WRIST	3.3	3.4	7	1.8	0.5	24.5	NR	24.5	NR	4.1	5.6
Median (APB)	ELBOW	7.2	6.8	23	1.1	0.5	59	68			4.7	5.6
ULNAR (ADQ)	WRIST	3.3	2.3	7	1.3	4			26.9	24.5	6.7	6.8
ULNAR (ADQ)	D. ELBOW	6.5	5.4	19	1.3	3.4	59	61			7.6	7
ULNAR (ADQ)	P. ELBOW	8.2	6.9	10	1.2	3.7	59	67			7.6	7.3
Post TIBIAL (AH)	ANKLE	6.4	6.6	9	0.7	0.9			47.2	49.4	14.7	10.9
Post TIBIAL (AH)	KNEE	15.4	15.3	40	0.6	0.8	44	46			13.5	10.2
PERONEAL (EDB)	ANKLE	3.7	5.7	8	0.5	0.5			NR	NR	10.6	8.9
PERONEAL (EDB)	D. KNEE	10.6	12.1	37	0.5	0.4	54	58			11	11.9
PERONEAL (EDB)	P. KNEE	12.2	13.6	9	0.4	0.5	56	60			11	11.6
PERONEAL (TA)	DFH	2.7	2	10	0.2	0.2					10.1	9.4
PERONEAL (TA)	PFH	4	3.2	9	0.2	0.2	69	75			9.3	8
H REFLEX	POP. FOSSA	NR	NR									

and Calcium: 400mg, thrice daily. He started walking in next few days with support and discharged home with outpatient physiotherapy advice.

### DISCUSSION

Hypoparathyroidism is a condition of parathyroid hormone deficiency. Primary hypoparathyroidism is defined as inadequate parathyroid hormone activity, due to which serum calcium concentration falls below reference range.

While on the other hand, secondary hypoparathyroidism is a state in which parathyroid hormone levels are low, due to primary pathologies that lead to hypercalcemia.

Primary hypoparathyroidism can occur due to acquired or inherited causes. Common causes of acquired hypoparathyroidism include surgical removal of parathyroid adenoma and autoimmune parathyroid disease. Other rare causes include radioactive iodine treatment, sarcoidosis, hemosiderosis, hemochromatosis, and parathyroid gland infiltration by metastatic deposits. Inherited hypoparathyroidism occur with other developmental anomalies such as failure of adrenal glands, thyroid gland, ovaries, thymus associated with oral candidiasis, baldness and vitiligo.

Literature review revealed five cases on effect of low calcium on membrane potential and subsequent changes in nerve conduction studies. To our knowledge, this case is the first of its kind in published literature.

Gomez reported a 25-year gentleman, known case of hypoparathyroidism.<sup>3</sup> At the age of 20 years, patient developed weakness of limbs with depressed tendon reflexes. On nerve conduction studies, found to have decreased nerve conduction velocities of median motor and ulnar nerves. Nerve biopsy showed axonal degeneration.

Dionisi described a 15-month child with distal sensorimotor peripheral neuropathy and hypoparathyroidism, which was recovered after treatment with vitamin D over five months.<sup>4</sup>

Gay and Grimes described a 68-year man with large fiber sensory neuropathy and hypoparathyroidism.<sup>2</sup> It also improved after vitamin D and calcium therapy for 6 weeks.

Goswami reported another case of idiopathic hypoparathyroidism whose nerve conduction studies revealed axonal sensorimotor neuropathy.<sup>5</sup> He received calcium and vitamin D for two years. There was slowly progressive betterment in neuropathy both clinically and on electrophysiologically. The occurrence of peripheral neuropathy in conditions associated with hypocalcemia such as hypoparathyroidism and osteomalacia and its reversibility after normalisation of calcium and vitamin D suggests its crucial role in the functioning of the peripheral axons.

Decreasing concentration of calcium reversibly increased inward sodium currents, moderate depolarization and increased nerve excitability, this results in failure of excitation of muscle fibers by supramaximal stimulation of peripheral nerves, thus resulting in decreased CMAP of tested nerves. This mechanism is similar to the changes seen in hypokalemic flaccid weakness.<sup>6</sup>

Hypoparathyroidism was diagnosed in our patient on the basis of low serum calcium, normal serum phosphate, and very low serum PTH. Nerve conduction studies showed severely reduced motor CMAP amplitude, a pattern similar to pure motor axonal neuropathy, which can be seen in AMAN variant of GBS or porphyria. Patient improved markedly after calcium and vitamin D replacement.

Appropriate diagnosis always requires a combination of careful history, examination and accurate interpretation of diagnostic testing. Failure to recognize the easily reversible causes of acute quadriplegia can lead to erroneous diagnosis, inappropriate treatment, and significant morbidity, related to treatment complications. This case report highlights the fact that nerve conduction studies can be misleading in patients presenting with flaccid quadriplegia. One should suspect electrolyte

imbalance, like hypocalcemia, in patients presenting with nerve conduction study of AMAN variant of GBS.

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