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Acute Motor Axonal Neuropathy Secondary To Hypokalemia In An Operated Case Of Single System Ectopic Uretic Orifices With Mayer Rokitansky Kuster Hauser (MRKH) Syndrome With Urinary Incontinence With Left Vesicoureteral Reflux (VUR)

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Abstract

Hypokalemia is common cause of causing flaccid paralysis. Here we present a 6-year-old girl, presented with first episode of acute flaccid paralysis of bilateral lower limbs since 12 hrs. On admission to PICU, investigations were sent in which marked hypokalemia was observed. Nerve Conduction Velocity (NCV) was done suggestive of acute motor axonal neuropathy. Potassium correction improved the patient's power to 5/5. Here we present case to highlight hypokalemia can cause acute motor axonal neuropathy which is a reversible cause if treated promptly.

Keywords: Acute Motor Axonal Neuropathy, Hypokalemia, Flaccid Paralysis

Introduction

Hypokalemic myopathy is seen in many clinical conditions[1].Studies on the pathomorphological changes of muscle fiber have been seen but alterations in intramuscular nerves and motor end plates aren't mentioned[1].Findings suggest that severe hypokalemia produces alterations in intramuscular peripheral nerves, motor end plates as well as in muscle fibers[1].

These abnormalities, especially reduced motor amplitudes that usually recover with normalization of serum potassium levels and muscle strength, which can be confusing at the initial stage of the attack and may lead to misdiagnoses such as acute motor axonal neuropathy (AMAN). We present a case where our patient presented with bilateral lower limb weakness acute in onset. Serum electrolytes suggestive of hypokalemia and NCV suggestive of Motor Axonal Neuropathy affecting predominantly lower limbs, possibly due to early AMAN.

Case Report

A 6-year-old female presented at emergency department (ED) of Dy Patil hospital in November 2023 with bilateral lower limb weakness occurred acutely in the night. Vomiting since a day. No respiratory or swallowing difficulty were observed. On history taking she is a known case of single system ectopic uretic orifices with MRKH with left VUR since birth for which she was operated. She had complaint of urinary incontinence since last three months for which ureteral reimplantation was done in the colon.

On physical examination, the patient's heart rate was 130 beats per min, blood pressure was 98/54mmhg, GCS was 15/15. Cardiac examination revealed tachycardia with a regular rhythm and no murmurs. Examination of the lungs and abdomen no abnormalilty detected. There were no deformities or edema of the extremities and distal pulses were present and equal bilaterally. Neurologic exam revealed flaccid paralysis of bilateral lower limbs. Sensation was intact. Deep tendon reflexes were ¾ in

both the limbs. Cranial nerve examination was normal. Investigation was sent in which sodium was 140mmol/l, potassium 1.6mmol/l, chloride 110mmol/l suggestive of hypokalemia. ABG Ph-7.18, PCo2-22.8, Hco3-8.4, normal anion gap metabolic acidosis. Renal Function Test S. creat-0.7mg/dl, Bun-19.9mg/dl, Urea-42.5mg/dl, Uric Acid-2.7mg/dl. NCV was suggestive of Motor Axonal Neuropathy affecting predominantly lower limbs, possibly due to early AMAN. Intravenous potassium and bicarbonate correction was started, after few hours of treatment her lower limb power improved as S. potassium level got corrected. Power became 5/5 after 24hrs of correction.

Discussion

Hypokalemic paralysis is a common cause of acute flaccid paralysis caused by reduced serum potassium levels causing muscle weakness[2].It can be primary or secondary. Hypokalemic periodic paralysis (HPP), a calcium channelopathy, can be sporadic or familial[2].Secondary causes can be endocrine (hyperthyroid periodic paralysis), renal (renal tubular acidosis, Gitelman syndrome, and primary hyperaldosteronism), or hypokalemia due to gastrointestinal losses (diarrhea)[2.]

Reporting on reversibility of electrophysiological abnormalities of sensory nerve have been done[3]. On serum potassium correction, improvement in sensory action potential during paralytic attacks has been observed in a prospective study of 10 patients with HPP[4]. Due to incomplete blood nerve barrier of the dorsal root ganglia and by the decrease in the extracellular potassium leading to inactivation of the sodium potassium pump are the possible mechanisms postulated[4]. They haven't been able to describe abnormalities in motor nerve function[4].

Inexcitiblity of most muscle fibres during an acute attack is seen with slowing of muscle fibre conduction velocity is found in another study on muscle fiber conduction velocity in patients with hypokalemic weakness of various etiologies[5]. Increase threshold in the axons, consistent with hyperpolarisation has also been observed[4]. With potassium correction excitiblity abnormalities resolved in activity dependent conduction block induced by voluntary contraction[4].

Our child showed ascending paralysis involving only lower limbs almost mimicking GBS and NCV was suggestive of Motor Axonal Neuropathy affecting predominantly lower limbs. Possibility of early GBS is likely AMAN variety.

Patient had hypokalemia on admission with serum potassium of 1.6mmol/l. Potassium corrrection was given following which patients power improved. Repeat NCV couldn't be done to document the reversibility.

We are reporting an unusual manifestation of hypokalemia where NCV was suggestive of motor neuropathy, but patient's condition improved on potassium correction.

Conclusion

Hypokalemic paralysis should always be kept in mind when making a differential diagnosis for acute flaccid paralysis. In all patients, serum electrolytes, NCV to be done. Immediate treatment should be started either orally or intravenously on the diagnosis of hypokalemic paralysis.

References

1. Hashimoto S, Akai F, Semba E, Sakatani K, Hiruma S, Nagaoka M, Hukuda K, Imada A. Neuronal changes of hypokalemic myopathy. A light- and electron-microscopic study on muscle biopsy. J Neurol Sci. 1980 Jan;44(2-3):169-79. doi: 10.1016/0022-510x(80)90125-2. PMID: 7354366.
2. Sardar Z, Waheed KAF, Javed MA, Akhtar F, Bokhari SRA. Clinical and Etiological Spectrum of Hypokalemic Periodic Paralysis in a Tertiary Care Hospital in Pakistan. Cureus. 2019 Jan 19;11(1):e3921. doi: 10.7759/cureus.3921. PMID: 30931192; PMCID: PMC6426562.
3. Inshasi JS, Jose VP, van der Merwe CA, Gledhill RF. Dysfunction of sensory nerves during attacks of hypokalemic periodic paralysis. Neuromuscul Disord. 1999 Jun;9(4):227-31. doi: 10.1016/s0960-8966(98)00129-1. PMID: 10399749.
4. Sharma CM, Nath K, Parekh J. Reversible electrophysiological abnormalities in hypokalemic paralysis: Case report of two cases. Ann Indian Acad Neurol. 2014 Jan;17(1):100-2. doi: 10.4103/0972-2327.128566. PMID: 24753672; PMCID: PMC3992744.

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5. Cruz-Martínez A, Arpa J. Muscle fiber conduction velocity in situ in hypokalemic periodic paralyses. Acta Neurol Scand. 1997 Oct;96(4):229-35. doi: 10.1111/j.1600-0404.1997.tb00274.x. PMID: 9325474.

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