Case Report

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Severe Hypokalemia Mimicking Guillain-Barr'e Syndrome in Young Female

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Abstract

In all emergency departments, acute neuromuscular weakness, which is linked to respiratory failure, is a common neurological emergency. Guillain-Barr´e Syndrome (GBS) remains the leading cause of acute ascending quadriplegia presents with ascending muscle weakness associated with paresthesia and loss of deep tendon reflexes and usually preceeded by diarrheal illness or upper airway infection. Here we report 20-year-old female presented with rapidly progressing, ascending weakness of all four extremities for 24 hours duration with subsequently complicated by respiratory paralysis due to severe hypokalemia.

Keywords: Guillain-Barr´e Syndrome; Hypokalemia; Acute Neuromuscular weakness; Quadriparesis; Emergency.

INTRODUCTION

Acute neuromuscular weakness is a common neurological emergency. In a clinical setting, muscle weakness is and progression to maximum severity within several days to weeks (less than 4 weeks).[1,2] Acute neuromuscular weakness Immediate and careful evaluation to determine the etiology is crucial as the accurate diagnosis has significant implications on management and prognosis.

The differential diagnosis includes neurological problems, metabolic disorders and infectious disease.[3] GBS is an autoimmune polyradiculoneuropathy in which involves peripheral nerve myelin sheath and it is secondary to minor infectious illness like acute gastroenteritis, upper respiratory infection, and some vaccinations.[4] Guillain-Barr´e Syndrome (GBS) remains the leading cause of acute ascending quadriplegia presents with ascending muscle weakness associated with paresthesia and loss of deep tendon reflexes.[5] The diagnosis of symptoms similar to those found in GBS can be challenging, especially when accompanied by acute hypokalemia-induced areflexic weakness. The reason for acute ascending areflexic muscle weakness is potentially treatable

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and reversible.[6] A case of severe hypokalemia was reported here that progressed rapidly, the symptoms of increasing weakness in all four extremities and increasing difficulty breathing are similar to Guillain-Barré Syndrome.

CASE PRESENTATION

A 20-year-old female was brought to Emergency Department by her family with Complaint of sudden onset of rapidly progressing, ascending weakness of all four extremities for 24 hours duration. There is history of strict dieting since 1 month. No history of similar condition in past or in her family, no history of trauma, any history of recent vaccination, flu symptoms. On examination, the patient was fully conscious and oriented but in acute respiratory distress with SpO_2 - 92% with oxygen by face mask.

Neurological examination revealed hypotonia in all four limbs (LL > UL) with muscle power 1/5 (LL) and 1/5 (UL) with absent DTR. There was no sensory deficit and cranial nerve abnormality. Supportive management was started after a provisional diagnosis of GBS was made. With in few hours, the patient developed severe respiratory distress & was put on NIV support following which she had an episode of VT. Rhythm was reverted to Junctional bradycardia with HR-35-50/min, after which patient was intubated in view of airway protection.

Arterial Blood Gas, electrolytes and other initial investigation were ordered, which revealed severe metabolic acidosis with hypokalemia. The diagnosis of hypokalemia induced paralysis was taken into account and the management of hypokalemia was initiated through correction of metabolic acidosis. Following the ICU admission, the patient was given parenteral KCl, sodium bicarbonate infusion for the correction metabolic acidosis.

The patient's conditions were reassessed due to improvement in potassium levels, leading to a significant improvement in muscle strength and respiratory condition. patient was successfully extubated. She was able to breathe normally herself, moving all the four limbs. Plans for more testing, which included nerve conduction studies, spinal MRI, and CSF analysis, were put on hold. After stay in the ward the patients' cause of hypokalemia is concluded as refractory hypokalemia and safely discharge.

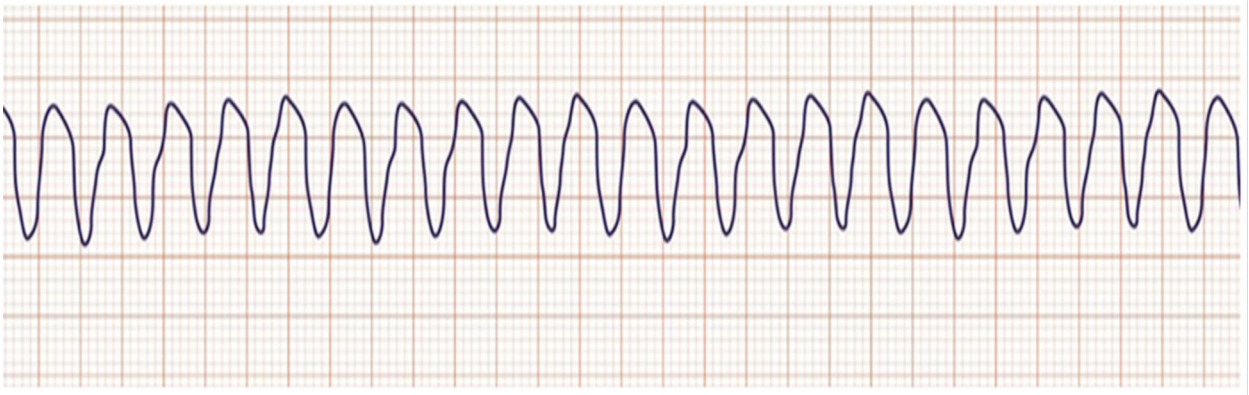

Table 1: Summarizing of Laboratory

Blood Investigation	At Admission	After 24 hrs	On Discharge
WBC	7.5	15.6	11
HGB	10.9	9.3	9.1
Potassium	1.82	2.7	4.1
Sodium	137	145	140
Magnesium	9.3	1.53	2.6
Glucose	193	145	99
pH	7.1	7.3	7.42
PcO_2	35.5	35	32
HCO_3-	10.5	13.9	20

DISCUSSION

Acute neuromuscular paralysis is one of the common neurological emergencies of which Guillain Barre Syndrome (GBS) remains the leading cause In this patient, the clinical presentations and physical findings is similar with Guillain Barre Syndrome, although more typically weakness progresses over several days in GBS rather than the rapid involvement of upper extremity muscles and respiratory muscles. Further more, in this after correction potassium level. Hypokalemia is a well-known cause of muscle weakness and mimics features with GBS which is many times overlooked or missed in differential diagnosis and extensive review of literature revealed only three case reports in which the patients had presented.[7]

The transcellular distribution of potassium can be altered by hypokalemia, resulting in a wide range of conditions. Potassium is actually depleted because of renal or extra renal-related losses, decreased intake, and thyrotoxic periodic paralysis. while the most reported cases were associated in with familial periodic paralysis.[3] Acute hypokalemic paralysis is a clinical condition presented with acute ascending, areflexic extremities and weakness. Complete muscular paralysis can result when there is severe hypokalemia, which may affect the respiratory, bulbar, and cranial muscles, as well as death through respiratory failure and arrhythmia.[3]

In conclusion, the clinical presentations and physical findings of patient we saw were consistent to those of GBS and provisional diagnosis of GBS was made until routine electrolyte result was obtained with severe hypokalemia and diagnosis reconsidered to hypokalemia induced paralysis. To ensure quick recovery and a good prognosis, hypokalemia should be treated aggressively. The association between severe hypokalemia and ascending paralysis needs to be measured through more research. Likewise, it is imperative to assess the frequency and long-term consequence of ascending paralysis caused by severe hypokalemia.

CONCULSION

Hypokalemia is a well-known cause of muscle weakness and mimics features with GBS which many times missed in differential diagnosis.

In summary, GBS was confirmed by the clinical presentations and physical findings of the patient. The diagnosis was changed to hypokalemia induced paralysis after routine electrolyte results were obtained with severe hypokalemia.

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