*Case Report*

**Received on** 21.12.2016, **Accepted on** 24.12.2016

Indian Journal of Emergency Medicine
Volume 2 Number 2, July - December 2016
DOI: http://dx.doi.org/10.21088/ijem.2395.311X.2216.10

# Guillian-Barre Syndrome with Hypokalemia: A challenge for Emergency Physician

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**Abstract**

Acute flaccid paralysis is one of the common presentation in Emergency and Gullian-Barre Syndrome and Periodic Hypokalemic Paralysis being important differriantial diagnosis with separate management. But the combination of both these conditions can be a diagnostic challenge for Emergency Physician. We report a case of 20 year old male who presented with such an association of Hypokalemia along with Guillian Barre Syndrome highliting the importance of the need to rule out various differential diagnosis during management of acute flaccid paralysis.

**Keywords:** Guillian Barre Syndrome; Hypokalemia; Hypokalemic Periodic Palsy; Nerve Conduction Studies.

## Introduction

Acute flaccid paralysis is a clinical syndrome characterized by rapid onset of weakness of limbs including weakness of respiratory and swallowing progressing to its severity within days to weeks in absence of spasticity or other signs of disordered CNS motor tracts such as hyperreflexia, clonus or extensor plantar response.

Hypokalemic paralysis and Acute Inflammatory Demyelinating Polyneuropathy (AIDP) are among common differential diagnosis of neuromuscular paralysis with different specific therapeutic interventions[1,6]. While AIDP is an autoimmune process that is characterized by progressive areflexic weakness and mild sensory changes.

Hypokalemic paralysis is characterized by muscle weaknes with a matching fall in potassium levels in blood [4]. Simultaneous presence of hypokalemia and AIDP at the time of presentation can cause both diagnostic and therapeutic dilemma [1,3]. Here we wish to report a case of a 20 year old male presenting with acute weakness of limbs and discuss the possible consequences and therapeutic considerations in management of rare association of hypokalemia and AIDP.

## Case Presentation

A 20 year old male patient with no prior known co-morbidities presented to our Emergency Department with complains of inability to walk or stand along with difficulty in lifting his upper limbs. He was apparently alright when he suddenly complained of pain in his upper and lower limbs followed by tingling sensation and weakness of upper limbs prior to his arrival in ER. The weakness in his upper limbs was gradually progressive and he was unable to raise his arms above his shoulder. When he woke up in evening after his afternoon sleep , he realized he was unable to walk or stand. He was rushed to a nearby hospital from where he was referred to higher centre for further evaluation and management. There was history of mild fever one month back which subsided within 2-3 days with use of only antipyretics requiring no antibiotics. There was no history of recent vomiting , diarrhoea or any urinary symptoms. There is no history of intake of any diuretics.

On arrival to ER, patient was conscious , awake and oriented about time , place and person. He was afebrile on presentation with heart rate of 48 beats per minute with respiratory rate of 14-16 per minute no signs of respiratory distress or use of accessory

muscle. Oxygen saturation was 100% on room air and his BP was recorded at 130/70 mm Hg in his right arm. His capillary blood sugar was 113 mg/dL. On clinical examination of his nervous system , he was found to have symmetrical muscle weakness ( B/L upper limb 3/5, B/L lower limb 3/5 by Medical Research Council (MRC) grading ). There was mild weakness in the intrinsic hand muscles causing slight weakness in his grasping. His sensations were intact. Cranial nerve examination was normal. His deep tendon reflexes ( knee, ankle, brachioradialis, triceps) were intact but sluggish.

His ECG had flat T waves with bradycardia suggestive of hypokalemia. His initial serum potassium level was 1.8 mEq/ L , Sodium level was 152 mEq/L ,magnesium 2.6 mEq/L. His arterial blood gas showed mild metabolic acidosis with pH 3.02 with low bicarbonate of 16.8 [Table 1-3]. In view of his low potassium level along with limb weakness and ECG changes, potassium correction was started with potassium chloride infusion through central line. His MRI spine was done to rule out any spinal pathologies which was suggestive of disc dessication at L2-3 and L 3-4 without any significant disc herniation . In spite of potassium correction and normalisation of potassium levels his muscle weakness gradually worsened ( upper limb 2/5 , lower limb 2/5) . Nerve conduction test was ordered [Figure 2]to rule out other pathologies which suggested axonal and Demyelinating neuropathy with low amplitude and F- latency absence characteristic of AIDP for which intravenous immunoglobulin was started. CSF study was done which showed normal protein and cells count with no albumino-cytological dissociation. Drastic improvement was seen in muscle power after IVIG administration. On 2nd day of admission potassium, potassium level declined despite continuing parenteral potassium supplements but muscle power continued to improve to 4/5.

Repeated blood gases suggested persistent mild metabolic acidosis with pH between 7.302 to 7.323 and lower side bicarbonate between 16.8 to 18.6 mmol/L. Urinalysis showed acidic urine with pH 6.5 with normal urine potassium level. His abdominal-pelvic ultrasound showed probable bilateral renal papillary necrosis. His serum cortisol and thyroid hormones were normal. Renal tubular acidosis was suspected as the cause for the persistent biochemical abnormalities. Sodium bicarbonate and potassium supplements were continued.

Patient regained his muscle power to 5/5 by 5th day of his admission and was able to walk without any assistance. He was discharged with oral bicarbonate supplements. He was advised for a nephrologist follow up to find out the cause for hypokalemia. There has been no subsequent follow up by the patient.

epoc BGEM Blood Test

Patient ID: 3041
Date & Time: 17-Aug-16 09:05:49

| | | | |
|---|---|---|---|
| Results: Gases+ | | | |
| pH | 7.302 | | Low |
| pCO2 | 34.0 | mmHg | Low |
| pO2 | 113.6 | mmHg | High |
| cHCO3- | 16.8 | mmol/L | Low |
| BE(ecf) | -9.6 | mmol/L | Low |
| cSO2 | 98.0 | % | |
| Results: Chem+ | | | |
| Na+ | 152 | mmol/L | High |
| K+ | 1.8 | mmol/L | Low |
| Ca++ | 0.59 | mmol/L | Low |
| cTCO2 | 17.9 | mmol/L | Low |
| Hct | 31 | % | Low |
| cHgb | 10.4 | g/dL | Low |
| BE(b) | -8.7 | mmol/L | Low |
| Results: Meta+ | | | |
| Glu | 105 | mg/dL | High |
| Lac | 0.91 | mmol/L | |
| Reference Ranges | | | |
| pH | 7.350 - 7.450 | | |
| pCO2 | 35.0 - 48.0 | mmHg | |
| pO2 | 83.0 - 108.0 | mmHg | |
| cHCO3- | 21.0 - 28.0 | mmol/L | |
| BE(ecf) | -2.0 - 3.0 | mmol/L | |
| Na+ | 138 - 146 | mmol/L | |
| K+ | 3.5 - 4.5 | mmol/L | |
| Ca++ | 1.15 - 1.33 | mmol/L | |
| cTCO2 | 22.0 - 29.0 | mmol/L | |
| Hct | 38 - 51 | % | |
| cHgb | 12.0 - 17.0 | g/dL | |
| BE(b) | -2.0 - 3.0 | mmol/L | |
| Glu | 74 - 100 | mg/dL | |

**Figure 1:**

## Discussion

AIDP and hypokalemia are among the most commonly encountered causes of acute neuromuscular paralysis [1].

Acute Inflammatory Demyelinating Polyneuropathy is an inflammatory neuropathy characterized by progressive areflexic weakness and mild sensory changes[1,6,7]. It encompasses groups of heterogeneous disorders due to pathogenic immune mediated hematogenous leukocyte infiltration of peripheral nerves, nerve roots or both with resultant demyelination or axonal degeneration or both. It is

**Table 1:** Moter Nerve Conduction

| Nerve and Site | Latency | Amplitude | Segment | Latency Difference | Distance | Conduction Velocity |
|---|---|---|---|---|---|---|
| **Tibial** | | | | | | |
| Ankle | 5.5 ms | 0.8 mV | Abductor hallucis-Ankle | 5.5 ms | mm | m/s |
| Popliteal fossa | 14.0ms | 0.8 mV | Ankle-Popliteal fossa | 8.5 ms | 430 mm | 51 m/s |
| **Peroneal. L** | | | | | | |
| Ankle | ms | mV | Extenson digitorum brevis-Ankle | ms | mm | m/s |
| Fibula(head) | ms | mV | Ankle-Fibula (head) | ms | mm | m/s |
| **Tibial. R** | | | | | | |
| Ankle | 6.5 ms | 1.0 mV | Abductor hallucis-Ankle | 6.5 ms | mm | m/s |
| Popliteal fossa | 15.3 ms | 0.8 mV | Ankle-Popliteal fossa | 8.8 ms | 430 mm | 49 m/s |
| **Peroneal. R** | | | | | | |
| Ankle | ms | mV | Extenson digitorum brevis-Ankle | ms | mm | m/s |
| Fibula (head) | ms | mV | Ankle-Fibula (head) | ms | mm | m/s |
| **Median. R** | | | | | | |
| Wrist | 3.7 ms | 1.1 mV | Abductor pollicis brevis-Wrist | 3.7 ms | mm | m/s |
| Elbow | 8.3 ms | 1.1 mV | Wrist-Elbow | 4.6 ms | 230 mm | 50 m/s |
| **Ulnar.R** | | | | | | |
| Wrist | 2.2 ms | 0.6 mV | Abductor digiti minimi (manus)-Wrist | 2.2ms | mm | m/s |
| Below elbow | 7.1 ms | 0.5 mV | Wrist-Below elbow | 4.9 ms | 250 mm | 51 m/s |
| **Median. L** | | | | | | |
| Wrist | 3.3 ms | 1.0 mV | Abductor pollicis brevis-Wrist | 3.3ms | mm | m/s |
| Elbow | 7.4 ms | 0.8 mV | Wrist-Elbow | 4.1 ms | 230 mm | 56 m/s |
| **Ulnar. L** | | | | | | |
| Wrist | 4.4 ms | 0.4 mV | Abductor digiti minimi (manus)-Wrist | 4.4 ms | mm | m/s |
| Below elbow | 9.2 ms | 0.6 mV | Wrist-Below elbow | 4.8 ms | 250 mm | 52 m/s |

**Table 2:** F-Wave Studies

| Nerve | M-Latency | F-Latency |
|---|---|---|
| Tibial. L | 3.9 | |
| Peroneal.L | | |
| Tibial.R | 5.9 | 46.3 |
| Peroneal.R | 5.2 | |
| Median.R | 3.9 | |
| Ulnar.R | 2.7 | |
| Median.L | 3.1 | 70.6 |
| Ulnar.L | 4.4 | |

believed to be caused by an immunological attack that is directed against myelin components resulting in segmental demyelination.

Blood tests have little role in diagnosis of AIDP. Albumino-cytological dissociation in CSF is observed classically in AIDP but is not specific to it. Electrodiagnostic testing is always necessary to confirm the diagnosis of AIDP. Nerve conduction studies can document demyelination , the hallmark of AIDP. The electrodiagnostic criteria provided in Cornblath includes :

- Reduced conduction velocity
- Conduction block or Abnormal dispersion
- Prolonged distal latencies
- Prolonged F-waves

Patients with 3 of 4 NCS Criteria have a clear primary Demyelinating neuropathy but patients who meet fewer than 3 criteria still may have AIDP but it may be necessary to exclude alternative diagnosis. MRI spine may be used to rule out spinal cord or nerve root processes that mimic AIDP.

**Table 3:** Sensory Nerve Conduction

| Nerve and Site | Onset Latency | Peak Latency | Amplitude | Segment | Latency Difference | Distance | Conduction Velocity |
|---|---|---|---|---|---|---|---|
| **Sural. R** | | | | | | | |
| Lower leg | 1.3 ms | 2.7 ms | 45 μV | Ankle-Lower leg | 1.3 ms | 90mm | 67 m/s |
| **Sural. L** | | | | | | | |
| Lower leg | 1.8 ms | 3.4ms | 24μV | Ankle-Lower leg | 1.8ms | 90mm | 50 m/s |
| **Median. R** | | | | | | | |
| Wrist | 2.4 ms | 4.6 ms | 95 μV | Digit II (index finger)-Wrist | 2.4 ms | 150 mm | 63 m/s |
| **Median. L** | | | | | | | |
| Wrist | 2.4ms | 4.7 ms | 101 μV | Digit II (index finger)-Wrist | 2.4ms | 150 mm | 63 m/s |
| **Ulnar. L** | | | | | | | |
| Wrist | 2.3 ms | 3.0 ms | 77 μV | Digit V (Little finger)-Wrist | 2.3 ms | 130 mm | 57 m/s |
| **Ulnar. R** | | | | | | | |
| Wrist | 2.2 ms | 3.6 ms | 66 μV | Digit V (Little finger)-Wrist | 2.2 ms | 130 mm | 59 m/s |

Periodic paralysis (PP) is a group of heterogeneous muscle disorders characterized by episodes of flaccid muscle weakness occurring at irregular intervals[2,4]. A clinically useful classification of primary PP includes hypokalemia, hyperkalemia and paramyotinic forms. The physiological basis of flaccid weakness is inexcitability of sarcolemma. Alteration of serum potassium levels is not the defect in primary PP, the altered potassium metabolism is a result of PP. The weakness is generalized but may be localised. Cranial musculature and respiratory muscles are generally spared. Stretch reflexes may be either absent or diminished during attacks.

Severe cases of hypokalemic periodic paralysis are generally seen in early childhood and mild cases may present as late as the third decade. Weakness may range from slight transient weakness of an isolated muscle group to severe generalized weakness. Severe attacks begin in the morning often with strenous exercise or a high carbohydrate meal on preceding meals. Patients wake up with severe symmetrical weakness. Serum potassium level decreases during attacks. The compound muscle action potential amplitude declines during the paralytic attacks. Sensory nerve conduction study findings are normal in most patients with periodic paralysis.

Hypokalemia can result from excessive loss of potassium in urine or from the gut, poor intake, increased translocation into cells or inherited tubular disorders.

Our patient had hypokalemia with mild metabolic acidosis and low plasma bicarbonate when he was admitted with quadriparesis. He had no predisposing acquired causes known to cause hypokalemia. During the course of hospital stay patient had recurrence of hypokalemia despite potassium supplementation suggestive of presence of underlying metabolic dysfunction. Hypokalemia with metabolic acidosis and low serum bicarbonate levels raises the suspicion for renal tubular acidosis which is a group of transport defects secondary to reduced proximal tubular reabsorption of bicarbonate, distal secretion of protons or both resulting in impaired capacity for net acid excretion and persistent hypercholesterolemic metabolic acidosis.

Improvement in muscle power in our patient in spite of fluctuating potassium levels after IVIG infusion along with corrorabating nerve conduction studies indicate AIDP as being primary pathology responsible for the quadriparesis. Presence of simultaneous hypokalemia along with other biochemical abnormalities along with echogenic debris in abdomen ultrasonography mandates further nephrologist evaluation which could not be done due to non-follow up by the patient after discharge.

## Conclusion

Acute neuromuscular paralysis caused due to either hypokalemia or AIDP manifesting as isolated pathologies are generally quite straightforward in diagnosis and management.But the combination of both these conditions complicates the diagnosis and can perturb a clinician facing such situation. Emergency physician should keep high suscpicion and should have a broader diffrential diagnosis during management of acute flaccid paralysis.

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