A case of acute onset quadriplegia: A lesson for physicians

Indira Sahu¹, Mukesh Kumar², Manish Ruhela³, Rakesh K. Ola³

¹Department of Pathology, SMS Medical College and Hospital, Jaipur, Rajasthan, ²Department of Anaesthesia, SK Medical College and Hospital, Sikar, Rajasthan, ³Department of Cardiology, Noble Care Hospital, Sikar, Rajasthan, India

ABSTRACT

A 38-year-old female patient presented with acute onset quadriplegia which was initially diagnosed as Guillain-Barre syndrome. ECG features were suggestive of hypokalemia. Blood investigations revealed severe hypokalemia. After replacement of potassium, patient’s muscle power recovered within 20 h. Thus, if the patient presented with acute onset quadriplegia possibility of hypokalemia-induced paralysis should always be kept in mind.

Keywords: Hypokalemia, Guillain Barre syndrome, quadriplegia

Introduction

Among the causes of acute onset quadriplegia, Guillain-Barre syndrome (GBS) remains one of the commonest causes. Severe hypokalemia may have a similar presentation. Hypokalemia is defined as a deficiency of potassium below 3.5 mEq/L in the plasma and increased gastrointestinal and renal losses are the common culprits. Hypokalemia can lead to symptoms ranging from muscle weakness to cardiac arrest. Hypokalemic paralysis is a relatively uncommon yet potentially life-threatening condition that, if correctly diagnosed and treated on time, can be completely reversed and life-saving.

In hypokalemic paralysis, the patient presents muscular weakness and low serum potassium. Herein, we report a case of a young female of hypokalemic paralysis who predominantly presented acute quadriplegia.

Case Report

A 38-year-old female, known case of cholelithiasis presented with abdominal pain and recurrent episodes of vomiting from the last 3 days. She was taken to a nearby nursing home where she received glucose-containing intravenous fluid. After starting intravenous fluid, the patient suddenly started complaining of weakness in all four limbs. It started in toes, progressed upwards, and involved upper limbs for over 2 h. The primary physician diagnosed as a case of GBS and referred to a higher center for further management. During transportation, the patient developed an episode of palpitation and syncope.

On examination at our center, she was conscious and oriented with blood pressure 108/70 mmHg, pulse rate 70 bpm, respiratory rate 18 breaths/min, and oxygen saturation 98% on room air. Chest auscultation was clear and on neurological examination there was hypotonia in all four limbs with muscle power 1/5. Deep tendon reflexes (DTR) were absent. There was no sensory deficit or cranial nerve abnormality. Pupils were normal in size and reactive to light. Her abdomen was distended with right hypochondrial tenderness with absence of bowel sounds.

Address for correspondence: Dr. Manish Ruhela, Department of Cardiology, Noble Care Hospital, Sikar, Rajasthan, India. E-mail: dr.manishruhela@gmail.com

Received: 19-05-2020
Revised: 19-06-2020
Accepted: 24-08-2020
Published: 30-10-2020

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.
Electrocardiogram (ECG) at presentation showed PR interval prolongation, ST depression in precordial leads, T-wave flattening, U waves in precordial leads with prolonged QT interval [Figure 1], indicating the possibility of severe hypokalemia. Blood investigations for serum electrolytes, complete blood count (CBC), liver function test (LFT), renal function test (RFT), and thyroid profile were sent. During the evaluation, she developed an episode of hemodynamically unstable ventricular tachycardia [Figure 2] which was cardioverted with Direct Current (DC) shock immediately. She was shifted to the intensive care unit for further management. Serum sodium was normal, whereas serum potassium was low 0.9 mEq/L for which replacement was started as per protocol. The serum magnesium level was normal. She remained hemodynamically stable. Ultrasonography (USG) abdomen showed acute cholecystitis with cholelithiasis. Potassium chloride supplementation continued overnight under ECG monitoring. With the improvement in potassium levels, ECG changes reverted to normal [Figure 3], muscle power improved, and reflexes returned. Serum potassium was repeated on the next day which was 2.1 mEq/L.

On the 3rd day, she was referred to a surgeon at normal serum potassium level and normal muscle power with the advice of management for gall bladder disease.

**Discussion**

Muscular weakness is a common presentation of various neurological and non-neurological conditions. Differential diagnosis of acute onset of weakness includes neurologic, metabolic, and infectious causes. The most frequent cause of acute flaccid paralysis worldwide is GBS. In this case, the presence of ascending motor weakness with lost DTR but no sensory impairment led to an initial diagnosis of GBS but as paralysis progressed to upper limbs in just 2 h, one should raise a doubt about the diagnosis. Initial laboratory investigation revealed that the patient had severe hypokalemia which quickly recovered after potassium supplementation. Thus the diagnosis was made for hypokalemia-induced ascending muscle weakness in this patient. The possible differential diagnosis for acute hypokalemic paralysis could be familial or primary hypokalemic periodic paralysis along with secondary causes i.e., gastrointestinal losses, renal disorders, and thyroid disorders.

The cause of hypokalemia, in this case, was most likely due to gastrointestinal losses as the patient had a definitive history of potassium loss due to recurrent episodes of vomiting. Familial hypokalemic periodic paralysis is an autosomal dominant condition that manifests as sudden recurrent episodes of painless weakness. Our patient did not have a history of similar episodes of weakness. The thyroid profile and renal functions were within normal limits.

**Conclusion**

This type of presentation of hypokalemic paralysis is not very common. This diagnosis usually does not come to the mind of physicians and intensivists. Therefore, if features suggestive of GBS are accompanied with hypokalemia, a possibility of hypokalemia-induced paralysis should be kept in mind. Timely replacement of potassium may help to achieve early recovery with improved outcomes.

**Financial support and sponsorship**

Nil.

**Conflicts of interest**

There are no conflicts of interest.

**References**

1. Goyal A, Spertus JA, Gosch K, Venkitachalam L, Jones PG, Van
den Berghe G, et al. Serum potassium levels and mortality in acute myocardial infarction. JAMA 2012;307:157-64.

2. Soar J, Perkins GD, Abbas G, Alfonzo A, Barelli A, Bierens JJ, et al. European Resuscitation Council Guidelines for Resuscitation 2010 Section 8. Cardiac arrest in special circumstances: Electrolyte abnormalities, poisoning, drowning, accidental hypothermia, hyperthermia, asthma, anaphylaxis, cardiac surgery, trauma, pregnancy, electrocution. Resuscitation 2010;81:1400-33.

3. Ahlawat SK, Sachdev A. Hypokalaemic paralysis. Postgrad Med J 1999;75:193-7.

4. Yuki N, Hartung HP. Guillain-Barré syndrome. N Engl J Med 2012;366:2294-304.

5. van Doorn PA, Ruts L, Jacobs BC. Clinical features, pathogenesis, and treatment of Guillain-Barré syndrome. Lancet Neurol 2008;7:939-50.