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Case Report Open Access

An Unusual Presentation of Hypokalemia

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Abstract

We report a case of severe hypokalemia presenting as Guillain Barre Syndrome (GBS). A 25 years old male presented to hospital with history of acute onset ascending areflexic paralysis. A diagnosis of GBS was made by the physicians. The patient was shifted to Intensive Care Unit for mechanical ventilation and further management. Investigations revealed severe hypokalemia for which potassium replacement was started. With correction of serum potassium levels, the patient's muscle power recovered completely within 24 hours of Intensive Care Unit stay. Thus, if features suggestive of GBS are accompanied by low serum potassium levels, a possibility of hypokalemia induced paralysis should always be kept in mind.

Keywords: Hypokalemia; Muscle weakness; Guillain barre syndrome

Introduction

Guillain Barre Syndrome (GBS) remains the commonest cause of acute ascending muscle weakness with loss of deep tendon reflexes (DTR). However, severe hypokalemia may have a similar presentation.

Case Report

A 25 years male presented with symmetrical weakness in all four limbs for last one and half days. It started in toes, progressed upwards and involved upper limbs over next four hours. There was no history of similar illness in past or in family. However, the patient had diarrhoea three days back; and vomiting and high grade fever (1010-1020 F) for last two days. He was taken to a private clinic where some intravenous fluids were infused. There was no history of trauma, diabetes or intake of any drugs prior to this illness.

On examination, the patient was conscious and oriented with blood pressure 140/90 mmHg, pulse rate 100 beats/minute with 4-5 ectopic beats every minute, respiratory rate 20 breaths/minute and oxygen saturation 98% with oxygen by face mask. Chest auscultation was clear and on neurological examination, there was hypotonia in all four limbs with muscle power 2/5 in lower limbs and 1/5 in upper limbs. Deep tendon reflexes were absent. There was no sensory deficit or cranial nerve abnormality. Pupils were normal in size and reaction to light. His abdomen was distended with absent bowel sounds but no guarding, tenderness or rigidity.

A provisional diagnosis of GBS was made by physicians and supportive management started. Within few hours, the patient started developing signs of respiratory distress. His haemogram and blood sugar were within normal range; and urine was negative for albumin, sugar and ketones. However, his renal function was deranged with blood urea 52 mg/dl and creatinine 2.4 mg/dl. The urine output was about 1 L in 24 hours. A previous ultrasound of abdomen revealed left sided contracted kidney and right sided pelviureteric junction obstruction resulting in hydronephrosis. Serum sodium was normal with a value of 145 meq/L, whereas serum potassium was low (1.9 meq/L) for which replacement was started. Serum magnesium levels could not be done due to logistic problems. The arterial blood gas analysis (ABG) showed partially compensated metabolic acidosis (pH 7.268, $HCO3^-$ 14.8 mmol/L, BE -13.9 mmol/L, pCO_2 27 mmHg) with anion gap 20 mmol/L. He was shifted to Intensive Care Unit (ICU), his trachea was intubated and mechanical ventilation started. Cerebrospinal fluid (CSF) examination was planned for the next morning. The patient remained haemodynamically stable; however, ECG showed ST depression and T-wave inversion (Figure 1). Serum potassium was repeated which revealed severe hypokalemia (2.3 meq/l). Potassium chloride supplementation continued overnight under ECG monitoring. With improvement in potassium levels, ECG changes reverted back to normal, muscle power improved, reflexes returned and trachea was extubated within 24 hours of ICU stay. The plan to conduct CSF examination was cancelled following such quick recovery. Next day, the patient was transferred to ward and was advised follow-up by nephrology and surgery departments in view of deranged renal function and hydronephrosis. The patient was discharged from the ward with the advice to get renal Doppler done. He was lost to follow up as he had come from some other state.

Discussion

Differential diagnosis of acute onset of weakness includes neurologic, metabolic and infectious causes [1]. The most frequent cause of acute flaccid paralysis worldwide is GBS; [2] however, the probable cause in the present case was metabolic i.e. hypokalemia.

GBS is an inflammatory peripheral neuropathy presenting as bilateral, symmetric, ascending weakness of limbs. A history of upper respiratory infection or diarrhoea 3 days to 6 weeks before onset is common [2]. The weakness may progress for 12 hours to 28 days before reaching plateau [3], however, in majority of patients, progression is up to 1 to 3 weeks after onset of symptoms [4]. Initial symptoms include numbness, paraesthesia, weakness or pain in limbs [2]. DTR are diminished or absent with intact sensation. Recovery may take weeks to months.

In our case, presence of ascending motor weakness with lost DTR but no sensory impairment led to an initial diagnosis of GBS. However, there were certain features which are not usually seen in typical case of GBS. Paralysis progressed to upper limbs in just four hours. In GBS,

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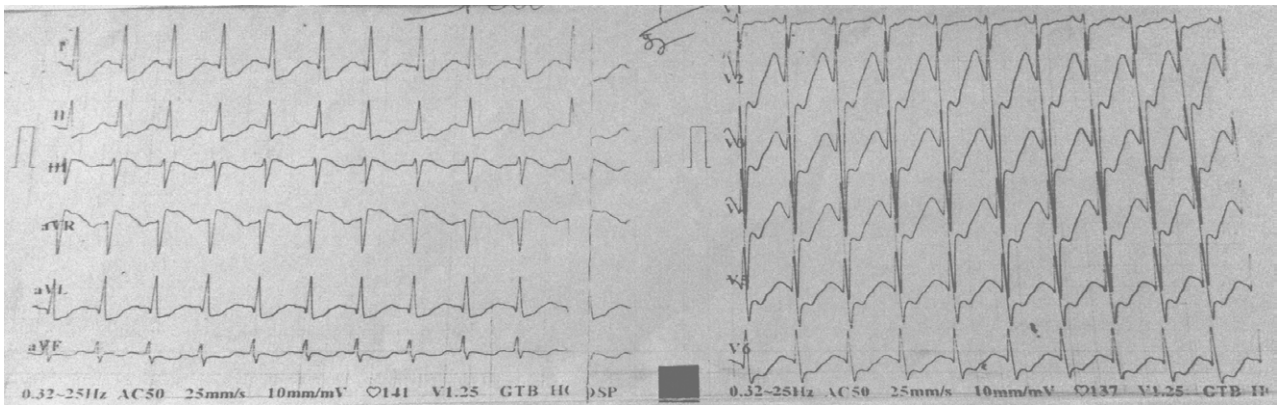

Figure 1: ECG showing features suggestive of hypokalemia.

fever is absent at the onset of neuritic symptoms and its presence should raise a doubt about the diagnosis; [5] but in our patient, fever was present simultaneously with the onset of paralysis. Moreover, recovery of the patient was very quick after potassium supplementation. Thus the diagnosis made for this patient was hypokalemia induced ascending muscle weakness with respiratory failure. CSF examination is routinely performed in cases with suspected diagnosis of GBS, where it shows raised proteins with normal white cell count [5]. Although these changes usually appear during second week of illness, CSF can be examined during first week also to have the baseline values. In our case, this investigation was planned for the next morning. However, it was decided to not do it due to marked improvement in the patient's condition by that time. The possible differential diagnosis for acute muscle weakness with hypokalemia could be gastrointestinal losses, familial hypokalemic periodic paralysis, thyrotoxic periodic paralysis, barium poisoning or renal disorders e.g. renal tubular acidosis (RTA) [1]. The cause of hypokalemia in the present case was most likely gastrointestinal losses as the patient had definitive history of potassium loss due to diarrhoea and vomiting. Hypokalemic periodic paralysis is an autosomal dominant condition that manifests as sudden recurrent episodes of painless weakness [1]. Our patient did not fit into this condition because there was no past history of similar episodes of weakness. There was no history suggestive of thyrotoxicosis or barium poisoning. The patient had metabolic acidosis and urine pH and electrolyte values were not available; however, the diagnosis of RTA was ruled out as this condition is associated with a normal anion gap and the same in our case was high.

Hypokalemia usually manifests as impairment of neuromuscular, cardiovascular or gastrointestinal system. Neuromuscular dysfunction may present as skeletal muscle weakness or even total paralysis involving respiratory, bulbar and cranial musculature in very severe cases [1]. Thus hypokalemia is a well known cause of muscle weakness and shares some features with GBS; however, it is commonly overlooked in differential diagnosis of this disease [2]. Extensive review of literature revealed only three case reports in which the patients had presented like GBS, but later the cause was found out to be hypokalemia [6-8]. Haddad et al reported a case of 33 years female who developed ascending motor weakness leading to respiratory failure after severe diarrhoea and vomiting [6]. However, this patient did not require mechanical ventilation and recovered fully after potassium supplementation. Another report described development of quadriparesis as result of severe hypokalemia and acidosis following ureterosigmoidostomy [7]. The patient improved after correction of hypokalemia and acidosis. The third report discussed two cases of barium poisoning who presented as areflexic quadriplegia resembling GBS [8].

As this type of presentation of hypokalemia is not very common, this diagnosis usually does not come to the mind of physicians and intensivists. In our case also, hypokalemia was detected in ward and potassium supplementation was started but diagnosis of hypokalemia induced muscle weakness was not considered by physicians. A diagnosis of hypokalemic paralysis should always be considered if a patient having hypokalemia presents with sudden onset, areflexic, pure motor weakness in one or more limbs, with no change in consciousness or sphincter function [1].

Conclusion

If features suggestive of GBS are accompanied by low serum potassium levels, a possibility of hypokalemia induced paralysis should be kept in mind and hypokalemia vigorously managed to achieve early recovery of patients with improved outcome.

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