Barium Poisoning Mimicking Guillain-Barre Syndrome

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Abstract
Barium poisoning is an important cause of areflexic quadriplegia associated with hypokalemia. Presenting features may mimic Guillain-Barre syndrome. A high index of suspicion of barium poisoning in two patients who presented with features resembling Guillain-Barre syndrome, led to timely management and early recovery in both the cases.

INTRODUCTION
The most common cause of acute or subacute areflexic paralysis is acute inflammatory polyradiculo-neuropathy or Guillain-Barre syndrome.1 When areflexic quadriplegia is associated with hypokalemia, barium poisoning should be considered in the differential diagnosis. Two case reports of areflexic quadriplegia due to barium poisoning mimicking Guillain-Barre syndrome are presented.

CASE REPORT

Case 1
A 26 years male was brought to the emergency department with history of vomiting and pain abdomen for 1 day and weakness of all four limbs since 5 hours. The patient was conscious and haemodynamically stable. However, he was tachypnoeic (respiratory rate-36 min) and O2 saturation on room air was 89%. Neurological examination revealed a flaccid quadripareisis, involvement of intercostals muscles and significant hyporeflexia. Examination of the sensory system, cranial nerves and spine revealed no abnormalities. There was history of having consumed food at a marriage party one day prior to admission.

Due to the ascending nature of motor involvement, a provisional diagnosis of Guillain-Barre syndrome was made. The patient was intubated, and transferred to the Intensive Care Unit (ICU) for ventilation and further management. Within 2 hours of admission to the ICU, he developed quadriplegia and absence of spontaneous respiration. He was able to respond to verbal commands by blinking of the eyes only. Deep tendon and bilateral plantar reflexes were not elicitable. After four hours, the patient had an episode of ventricular tachycardia which was managed with injection lignocaine followed by two DC shocks (100 J).

Biochemical investigations were within normal limits except for serum potassium which was 2.7meq/L. Repeat serum potassium continued to be low (1.37 / 1.8 meq/L), and correction was started with supplemental potassium chloride (KCl) of 80-150 meq/day. Nerve conduction study and cerebrospinal fluid examination were within normal limits. Presence of persistent hypokalemia along with a normal nerve conduction study made us suspicious, and further enquiry from the patient revealed a history of suicidal ingestion of a white powder (barium carbonate) 1 day prior to admission. The patient started regaining motor power from the fifth day onwards, and was extubated on the 8th day.

Case 2
A 32 years male was admitted with history of diarrhoea for one day and quadripareisis since a few hours prior to admission. Examination of the cardiopulmonary system was unremarkable. Deep tendon and bilateral plantar reflexes were not elicitable. Within a few hours of admission, he became tachypnoeic and started using accessory muscles of respiration. Gag and cough reflexes became absent and he developed complete quadriplegia. The patient was intubated and transferred to the ICU for ventilation and further management. A sample of blood was sent for haematological and biochemical investigations.

After four hours of admission to the ICU, the patient had a generalized seizure which was managed with diazepam and a loading dose of dilantin. At the same time, his ECG showed broad complex ventricular tachycardia (130/ minute). Since the blood pressure was stable, a bolus of injection amiodarone was given and an amiodarone infusion was started. Arterial blood gas analysis revealed severe metabolic acidosis (pH 7.1, BE-17.7, PO, 151, PCO2 45), for which sodabicarb was administered. All investigations were within normal limits, except for serum potassium which was reported to be very low. KCl infusion was started and a repeat serum potassium done after 12 hours was found within normal limits. On repeated questioning from

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the relatives it was found that a few hours prior to admission, the patient had taken a cup of tea at his in-laws house, where he had gone to patch up with his wife after a fight. Subsequently, a sample of blood was sent for toxicology screening.

On the second day, the ECG became normal and amiodarone infusion was stopped. Toxicology screen detected the presence of barium in the blood sample (5.7 mg/L; normal: 0.08–0.4 mg/L) using atomic absorption spectrophotometer technique. Though the motor power started improving from the third day onwards, the patient developed a chest infection and was intubated on the ninth day.

**DISCUSSION**

A patient presenting with acute systemic weakness in the emergency department may have a wide differential diagnosis ranging from neurologic to metabolic to infectious causes. Acute hypokalemic paralysis is a rare but treatable cause of acute systemic weakness and it is essential for clinicians and intensivists to be aware of this condition, so that they can differentiate it from other causes of quadriparesis.

Barium carbonate is commonly used in North India as a rat poison. Most cases of acute toxicity occur due to ingestion of barium carbonate (mainly suicidal), food contaminated by barium carbonate or carelessness in handling rat poison. Typical manifestations of barium poisoning range from abdominal pain, nausea, vomiting and diarrhoea, to areflexic flaccid quadriplegia and respiratory paralysis which sometimes develops within a few hours. Hypertension and malignant cardiac arrhythmias are common. Central nervous system signs of poisoning include mydriasis, anxiety, headache, confusion, myoclonus, trismus and seizures. Occurrence of convulsions in our second patient may have been due to a direct effect of barium poison or due to acidosis which was noted on arterial blood gas analysis.

Hypokalemia occurs as a result of redistribution of body potassium with a shift from extracellular to the intracellular compartment. The large active and passive influx of extracellular potassium into the muscle turns off the Na⁺K⁺ ATPase pump causing depolarization and paralysis. Extracellular hypokalemia may be further exacerbated by gastrointestinal potassium losses secondary to vomiting and diarrhoea. Though the time course of development of paralysis correlates with that of hypokalemia, areflexic quadriplegia may persist despite its correction, as reported by Koch et al and noted by us in the second patient (case 2). Barium itself is responsible for membrane depolarization by causing release of acetylcholine and by competitively reducing the permeability of all membranes to potassium, with resultant intensity of neuromuscular blockade correlating directly with plasma barium concentration.

Although both our patients presented with typical features of barium poisoning, the progressive areflexic quadriplegia with intact sensation very closely resembled Guillain-Barre syndrome. Clinically, there may be some common presenting features between barium carbonate poisoning and Guillain-Barre syndrome, namely, ascending quadriplegia, areflexia, absence of sensory impairment and involvement of respiratory muscles. In Guillain-Barre syndrome, weakness may progress rapidly or over several days, reaching a peak at 3–4 weeks of onset. Disturbances of autonomic function, such as persistent tachycardia, cardiac arrhythmias and ST-T wave changes on the ECG are common, thus confusing the picture further with barium overdose. Features which may differentiate Guillain-Barre syndrome from barium carbonate poisoning include, a history of respiratory or gastrointestinal infection (in 50% of cases), cranial nerve involvement, protein cytologic dissociation in the CSF (in second week) and early conduction block in nerve conduction velocity studies (in 90% of cases).

Both our patients did not give any history of poisoning at presentation. However, the presence of quadriplegia coupled with hypokalemia, led to a high index of suspicion of barium poisoning, and subsequent history of suicidal intake by the first patient and toxicology screening in the second patient clinched the diagnosis. Though treatment of both Guillain-Barre syndrome and barium poisoning is symptomatic and supportive, early management of barium intoxication includes a gastric lavage and enteral administration of sodium sulfate to prevent digestive absorption by precipitating barium ions to insoluble barium sulphate. In addition, large amounts of intravenous potassium (upto 400 meq) may be necessary to correct the hypokalemia. Prognosis is favorable as symptoms usually fade after 24 hours of potassium supplementation and supportive treatment.

A detailed history at presentation, coupled with a high index of suspicion of barium intake in patients who present with features mimicking infective polyneuropathy along with associated hypokalemia, will help in making an early diagnosis and lead to timely management and recovery of cases, without resulting in undue morbidity and mortality.

**REFERENCES**


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