CASE REPORT

Delayed Neurotoxic Paralysis with Confounding Hypokalaemia

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Abstract

Snakebite is a major health issue in India and developing nations across the globe. Common krait bites are associated with delayed paralysis and locked-in syndrome. Locked-in syndrome has been documented among patients of snakebite. Our case is unique due to the confounding effect of hypokalaemia which may confuse the physician as hypokalaemic periodic paralysis (HPP), a rare genetic condition which can also be precipitated by a stressful event such as snakebite. To the best of our knowledge, we could not associate the presence of hypokalaemia in patients of neurotoxic snakebite. As krait bite are often not associated with the history of having witnessed, a snake due to its nocturnal habit and painless bite, such hypokalaemia should not lead the physician to misdiagnose this snakebite as severe HPP. We urge our fellow researchers to look out for similar findings in their patients.

Key messages: Locked-in syndrome in snakebites can be due to the effect of the venom in blood onto the ventral pons. To the best of our knowledge, we could not associate the presence of hypokalaemia in patients of neurotoxic snakebite. We presume this hypokalaemia to be nutritional in origin. As krait bites are often not associated with the history of having witnessed, a snake due to its nocturnal habit and painless bite, such hypokalaemia should not lead the physician to misdiagnose this snakebite as severe HPP.

Keywords: Case report, Hypokalaemia, Neurotoxic, Snake envenomation.

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Introduction

Snakebite is a major health issue in India. Snakebite deaths are often underestimated due to a high 'out of hospital' deaths that go unreported.[1] The Million Deaths Study identified Uttar Pradesh state as having the highest annual snakebite death.[2] During the year 2020–2021, the Department of Emergency Medicine, King George's Medical University (KGMU) treated 287 patients of snakebite. Of these, 212 presented with neurotoxic symptoms of envenomation. The ratio of Krait:Cobra bites was 3:1. About 30% of neurotoxic bites were of unknown origin, that is, the patient was unable to identify species of snake or the patient did not know what bit him. The common krait (*Bungarus caeruleus*) (Fig. 1) is a venomous elapid serpent that belongs to the Genus *Bungarus*. The genus *Bungarus* has 16 species of snakes, of which 8 are native to India. Common krait along with Indian Cobra (*Naja naja*) produces neurological signs of snakebite like ptosis, diplopia, cranial nerve paralysis, descending paralysis, hypoxia due to intercostal involvement, and paradoxical respiration. The common krait is also notorious for biting people after entering the houses at night in search of prey. The bite is often without fang marks and does not cause enough pain to awaken the patient thus leading to the commonly associated term of 'sleep paralysis'.[3] This absence of history may pose a serious problem for the clinician in deciding the future course of action for the patient. We report to you a case where we documented severe hypokalaemia in patient of krait envenomation.

Case Description

A 20-year-old man presented to the emergency in the morning with chief complaints of altered sensorium and breathlessness. The patient was found on the floor by his parents who described him groaning with generalised pain, irritable to touch, and not making any meaningful response to their verbal queries. The patient was not on any medication and had no history of past illnesses. The patient and his family slept on the floor of a hut with minimal belongings and were farmers by occupation.

The patient's family gave no history of alcohol use, poison consumption and drug dependence. There was no history of fever, headache, and cough in the past. Upon measuring the vitals, heart rate was 100/min, blood pressure was 108/80 mm Hg with saturation of 98% on room air, respiratory rate 32/min, blood sugar level 98 mg/dL. The patient was E2V4M5 on the Glasgow coma scale (GCS). Nuchal rigidity was absent. Brudzinski's sign, Kernig's sign were absent and Jolt accentuation could not be performed. Routine Blood work was collected, and the patient was scheduled for a non-contrast computed tomography (NCCT) of head with a bedside chest X-ray.

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Fig. 1: The common Krait Picture courtesy – Mr Dinesh Pawar

Fig. 2: Picture of snake as taken by the relative

An accompanying cousin of the patient was sent to the house of the patient to look out for any bottle of pesticide as they expressed uncertainty regarding intake of poison. Zinc phosphide commercially available as 'Rat Kill' and aluminium phosphide, 'Celphos' are commonly consumed among farmers as a means to self-harm. After consumption of these poisons, the patients present with similar signs and symptoms of altered sensorium and breathlessness.

After 2 hours of the initial presentation, on reassessment, the GCS was E1V2M4. The respiratory rate was 40/min, and the patient was maintaining a saturation of 96% on oxygen delivered at 12 L/minute though a non-rebreathing mask with reservoir bag. An arterial blood gas analysis (ABG) was performed which suggested a pH of 7.32, paO_2 of 98 mm Hg on oxygen support, $paCO_2$ of 36 mm Hg, lactate 2.1 mmol/L, bicarbonate 18 mmol/L, anion gap 14 mmol/L. Blood sugar level repeated was 102 mg/dL. Urine ketone test performed by dip stick was negative. Complete blood count showed haemoglobin levels of 12.8 gm/dL and total leucocyte count was 6500. Renal function and liver chemistries revealed serum creatinine of 0.65 mg/dL, serum urea 36.9, serum bilirubin 0.48 mg/dL, and serum alkaline phosphatase 114 IU/L. Serum electrolytes revealed the values of sodium: 136.7 mmol/L, potassium: 1.2 mmol/L, and ionic calcium: 4.8 mg/dL.

The patient was intubated in view of rapidly deteriorating GCS, laboured breathing, and metabolic acidosis. Bedside chest X-ray revealed clear lung fields which supported good air entry heard on auscultation. The pupils of the patient were sluggishly reacting to light. Serum potassium levels were low, and this raised doubts about hypokalaemic periodic paralysis (HPP) in the patient. The patient was administered potassium chloride with mannitol 10 mEq per hour via infusion through a central venous catheter. Non-contrast computed tomography Head appointment of the patient was cancelled as the patient required a continuous cardiac monitoring and repeated serum potassium sampling. Phosphide poisoning was the provisional diagnosis.

The patient's cousin who was sent to look for a poison container in the house called back to say that he found a snake in the house. He described the snake as black with multiple white bands and sent a picture of the snake on WhatsApp (Fig. 2). The patient was now examined head to toe and on log rolling to look for any fang marks. None were found on the body after a thorough inspection. Neurotoxic snakebite was now the diagnosis with the species identified as common krait (*Bungarus caeruleus*). The patient was administered 10 vials of polyvalent anti-snake venom (ASV). Potassium chloride infusion was stopped during the administration of ASV and the patient was shifted to oral potassium chloride given at 60 mEv every 3 hours till serum potassium was normal.

Neurotoxic envenomation presents with ptosis as an early feature followed by cranial nerve paralysis and involving the diaphragm late. In the presenting patient, this sequence was not reported as patient presented late and the bite was nocturnal. Most snake envenomation in India occur from June to September and this patient reported to us in April. Another 15 vials of ASV were administered after 3 hours of previous administration and injection calcium gluconate was given 10 mL QID.

On day 2 of mechanical ventilation, GCS was E1VTM1 and both pupils were fixed and dilated. On day 11, the patient started regaining motor function which was first noted in the form of vertical eye movements in response to leading questions asked. This was followed by further recovery on the next day with ability to move neck and the regaining of muscle power innervated by cranial nerves. On day 15, the patient was extubated and placed on non-invasive ventilation (NIV). He reported that he was aware of the surroundings while he was in coma and could recall the conversations he heard. Upon discharge on day 22, final diagnosis was locked-in syndrome due to krait bite with hypokalaemia.

Discussion

Common krait bites are associated with delayed paralysis and locked-in syndrome. Locked-in syndrome has been documented among patients of neurotoxic snakebite. Our case unique due to the confounding effect of hypokalaemia seen which may confuse the physician as severe HPP, a rare genetic condition that can also be precipitated by a stressful event such as snakebite. The key to successful management of such a patient would be thorough evaluation, monitoring and aggressive treatment of hypokalaemia when it occurs.[4] In our patient, after the correction of potassium and administration of ASV, calcium gluconate, the neuroparalysis did not resolve as expected. Delayed descending paralysis, internal ophthalmoplegia and areflexia should not give the impression of brain death as the patient gains eye movement and recovers

other motor functions gradually.[5] Atropine and neostigmine administration is helpful in cobras but do not reverse paralysis in krait envenomation as krait venom affects presynaptic fibres and cobra venom affects postsynaptic fibres.[6,7] Injection of calcium gluconate 10 mL given 6 hourly alleviates paralysis features and should be administered if ASV is unavailable. It can also be given as adjunctive therapy for faster recovery from paralysis. Locked-in syndrome is a neurological entity where a patient is unable to communicate due to motor paralysis despite being aware of the surroundings. It is also known as 'pseudo-coma', 'De-differentiated state', 'Monte Cristo syndrome' and 'Ventral Pontine syndrome' in association with infarction of ventral pons where it was first described.[8] Locked-in syndrome has been further classified as classical (preserved consciousness and vertical eye movement in a patient with quadriplegia and anarthria), complete (preserved consciousness with no form of communication) and incomplete (preserved consciousness with some form of communication other than vertical eye movement).[9] The causes of locked-in syndrome beside krait envenomation include pontine haemorrhage, vertebro-basilar axis dissection, central pontine myelinolysis, dysmyelination in ventral pons due to multiple sclerosis, brainstem encephalitis, diazepam, and heroin abuse.[8,10]

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