A rare case of quadripareisis

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Abstract

Snakebite is a public health problem. Common krait is nocturnally active with painless bite. Patients exhibit neuromuscular weakness.

Keywords: Neurology, neuromuscular, neurotoxicology, venoms

Background

Snakebite is an occupational hazard causing considerable morbidity and mortality worldwide, particularly in tropical countries such as India. An estimated 50,000 Indians die due to venomous snakebite yearly, 70% of whom are males between the ages of 20 and 50 years.¹ They are hematotoxic, neurotoxic, and myotoxic. Over 2000 species of snakes are known worldwide, of which around 400 are poisonous. These snakes belong to the families Elapidae, Viperidae, Hydrophiidae, and Colubridae. Indian cobra (Naja naja) and Common Indian krait (Bungarus caeruleus) are the two important species of elapid snakes found in India and are responsible for most of the cases of neurotoxic snakebite.² Kraits of Southeast Asia are elapid snakes represented by 12 species. Common krait is nocturnally active and its bite is painless. Patients exhibit ptosis, ophthalmoptelgia, dysphagia, dyspnea, and neuromuscular weakness.³

Case Presentation

A 30-year-old married female presented with sudden onset upper abdominal pain and vomiting 7 h back followed by inability to speak, dysphagia, dyspnea, choking, drooping of eyelids, and weakness of all four limbs within half an hour. On further enquiry, attendants told that patient had gone for a festival celebration and dinner in the night in village and after half an hour of returning back, the problem occurred but other people who ate the dinner had no complaints. She had two children with history of eclampsia in last child birth.

She was unconscious with pulse 64/min, blood pressure 100/60 mmHg, and oxygen saturation 50% which was decreasing and was intubated. On further examination, bilateral proxis with complete ophthalmoptelgia on oculocephalic reflex testing, quadripareisis with power 0/5 in all muscle groups was present, plantar reflex and deep tendon reflexes were absent. Chest, cardiac, and abdominal examinations were normal. Blood and urine investigations were normal. Chest X-ray, MRI brain [Figure 1], and spine [Figure 2] were normal. Neostigmine test was negative. Tests for porphyrias were negative. High degree of suspicion of snakebite was considered but attendants gave no history and no bite mark was found at first but later, a mark on right ear [Figure 3] was seen and attendants told that it was not present before. Patient was given polyvalent antivenom urgently and after 2 days, she was conscious, ptosis decreased, muscle power increased to 1-2/5. After 4 days, patient was successfully extubated. She was whispering earlier, slowly speech became normal. After 12 days, she could walk with support, ptosis disappeared, and was discharged.

Discussion

Unexplained unresponsiveness in a previously healthy person in areas where snakebites are common should create the suspicion...
Polyvalent antisnake venom (ASV) is relatively safe, and allergic reactions after ASV injection can be prevented by premedication with adrenaline, intravenous hydrocortisone, and histamines. Therefore, suspected snake envenomation should be treated empirically with intravenous polyvalent ASV, and simultaneously should be investigated for differential diagnosis and etiology of unresponsiveness. Abdominal pain is the initial symptom of krait bite and may mimic surgical abdomen. Patient may also present with vomiting and at times diarrhea. Our patient had recurrent vomiting along with pain abdomen mimicking botulinum poisoning. This is followed by drooping of eyelids, double vision, weakness of limbs, and breathing difficulty progressing to neuromuscular paralysis. In an unknown scenario, patient gets up at night, with colicky abdominal pain and may be misdiagnosed as acute abdomen. Kraits may have painless bites and negligible local swelling.

Other differential diagnoses were human botulism, Guillain-Barre syndrome (GBS), myasthenia gravis, periodic paralyses, and porphyria. Human botulism is a rare, serious, potentially fatal disease. It is an intoxication caused by ingestion of potent neurotoxins in contaminated foods. Early symptoms are marked fatigue, weakness, and vertigo, usually followed by blurred vision, ophthalmoplegia, dry mouth, and dysphagia and speaking. Vomiting, diarrhea, constipation, and abdominal swelling may also occur. It can progress to weakness in neck and arms, after which respiratory muscles and muscles of lower body are affected. The paralysis may make breathing difficult. There is no fever and unconsciousness. The progression to complete quadriparesis and respiratory paralysis is not that fast (within hours) as happened in our case. People with GBS usually experience their most significant weakness within 2–4 weeks after symptoms begin. Recovery usually begins 2–4 weeks after weakness plateaus. Guillain-Barre syndrome (GBS) often begins with tingling and weakness starting in feet and legs and spreading to upper body parts including arms. A core clinical feature of all GBS subtypes is the characteristic disease time course. In 90% patients, this is monophasic, although up to 10% develop recurrent or relapsing GBS. The time interval between onset of neurological symptoms and nadir ranges between 12 h and 28 days and is usually followed by subsequent clinical plateau or improvement. Myasthenia gravis patients progress from mild to severe disease over weeks to months. Weakness tends to spread from ocular to facial to bulbar muscles and then to truncal and limb muscles. On the other hand, symptoms may remain limited to extraocular and eyelid muscles for years. Rarely, patients with severe generalized weakness may not have associated ocular muscle weakness. Periodic paralyses is characterized by episodes of flaccid muscle weakness occurring at irregular intervals. Most of the conditions are hereditary and more episodic than periodic. It usually recovers very fast and ophthalmoplegia and respiratory paralysis is rare. Major manifestations of acute hepatic porphyrias are neurologic including neuropsychic abdominal pain, peripheral motor neuropathy, ophthalmoplegia, and mental disturbances, with attacks often precipitated by dieting, certain drugs, and hormonal changes. Acute porphyria should be suspected in patients with
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neuro-visceral symptoms after puberty, such as abdominal pain, and when the initial clinical evaluation does not suggest another cause.

Conclusion

There should be high suspicion of snake (krait) bite in cases of sudden onset quadriplegia and respiratory failure even if the local signs of painless bite are absent. Patient's life can be saved if the possibility is timely considered and appropriate management started accordingly.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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