Snake Bite Complicated By Bacterial Meningitis

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ABSTRACT

Background: Neurotoxic snake bite envenomation is a common life threatening medical emergency in India, especially in the rural areas.

Case Presentation: We report a case of a neurotoxic snake envenomation presenting with abdominal pain and neuromuscular paralysis, which developed bacterial meningitis during her stay in the hospital. Possibly, secondary to the snake bite, a very rare incidence.

Conclusion: Our case is a step towards the direction pointing to the rare possibility of bacterial meningitis complicating snake bites which needs further research.

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Implication for health policy/practice/research/medical education:
Snake Bite Complication


1. Introduction:
Every year more than 2,00,000 snake bites are reported in India, out of which estimated 35,000 to 50,000 people die (1). Majority of the snake bite deaths go unreported due to the presence of traditional healers and deaths occurring on the way to the hospital. Around 52 species of poisonous snakes are found in India (2) of which the most commonly encountered families are:

(1) Common cobra- Naja
(2) King cobra- Ophiophagushannah
(3) Russell’s viper- Daboia russelli

(4) Common krait- Bungarus caeruleus
(5) Saw scaled viper- Echiscarinatae

Common Krait’s venom is considered to be 10 times more poisonous than the cobra venom. Neurological manifestations seen in patients with krait bite range from medical emergencies like respiratory failure secondary to respiratory muscle paralysis, ptosis, ophthalmoplegia to delayed manifestations like peripheral neuropathies, optic neuritis and cortical blindness (3). Rare cases of acute disseminated encephalomyelitis (4) and Guillain-Barresyndrome (5) also have been reported. After a thorough review of the available literature we could not find any instance of snake bite complicated by bacterial meningitis and hence would like to report our very interesting experience.
2. Case Presentation:
A 15-year-old female was brought to the emergency department of Krishna Institute of Medical Sciences [KIMS], Karad at around 5.30 pm with the complaints of snake bite on the calf of her left leg 12 to 15 hours ago and pain in abdomen since two hours. The killed snake brought was identified to be a krait. Patient had ignored the bite initially as there had been no local bleeding or paraesthesia but then she started having abdominal pain, heaviness of her eyelids and blurring of vision. There was no history of headache, vomiting, any drug intake or addiction.

On general examination, patient was conscious and cooperative but appeared drowsy. She had a pulse of 80 per minute, blood pressure of 120/80 mmHg, respiratory rate of 16 per min along with ptosis. There was no pallor, icterus, cyanosis or lymphadenopathy. Local examination of the left calf muscle revealed the fang marks but there was no local bleeding, swelling or tenderness. On systemic examination, she had diffuse tenderness per abdomen. Neurological examination revealed mild quadriaparesis, brisk deep tendon reflexes, neck stiffness; a positive Kernig's sign and an extensor left plantar response. She had no external/internal ophthalmoplegia/papilledema. Cardiac and respiratory system examination was within normal limits.

The blood sample taken for the 20-minute clotting test was positive and hence her treatment was started with 150 units of polyvalent antisnake venom. Atropine and neostigmine were given for 3 cycles every 15 minutes. Her CT-Brain with contrast revealed meningeal enhancement and cerebral oedema. Her CSF examination showed a total count of 280 cells, of which polymorphs were 80% and lymphocytes were 20%, elevated proteins-67 mg/dl and reduced sugar levels of 30 mg/dl. CSF culture was sterile.

Other investigations showed haemoglobin-13.7 mg/dl, WBC count-11,600 with predominantly polymorphs-78%, platelet count-2.70 lakhs. Her bleeding time, clotting time, prothrombin time, INR was normal. Even plasma glucose, blood urea, serum creatinine, electrolytes including serum sodium, serum potassium, arterial blood gases and electrocardiogram were within normal limits. Ophthalmology consultation sought did not reveal any abnormality.

Considering acute bacterial meningitis, she was given intravenous broad spectrum antibiotic coverage with injection ceftriaxone 2 grams every 12th hourly and injection vancomycin 1 gm every 12th hour for a total of 10 days. She was given mannitol in view of her cerebral oedema. During her hospital stay of 15 days she improved gradually and completely without any focal neurologic deficit. Fortunately, she never required any ventilatory support.

3. Discussion:
Krait bites commonly occur at night. Those sleeping on the floor are at a greater risk and they mostly do not show any local tissue reaction (6). Many a times the fang marks also may not be seen due to the very sharp fangs of krait. Its venom stimulates the autonomic nervous system as early as within 20-30 minutes of the bite leading to colicky abdominal pain which is the most common presenting compliant of occult krait bites. Bradycardia, sweating, vomiting, raised blood pressure may also be seen. Within 30mins to 18hrs the presynaptic acetylcholine receptors are attacked by the venom (2). Common neurological symptoms in decreasing order of frequency include ptosis (85.7%), ophthalmoplegia (75%), limb weakness (26.8%), respiratory failure (17.9%), palatal weakness (10.7%) and neck muscle weakness (7.1%) (7).

Goldstein et al found that the oral flora of snakes comprises a wide range of aerobic and anaerobic micro-organisms, especially the faecal Gram-negative rods, because their prey usually defecates while being ingested (8). Kraits are known to be
cannibals also. Culture of fangs, fang sheaths, and venom of various snakes such as bothrops, vipers, rattlesnakes and naja naja, have shown heavy colonization with many bacteria, including members of Enterobacteriaceae including *Morganella* spp. and *Escherichia coli*, Group D streptococci, *Aeromonas* spp., and anaerobes such as *Clostridium* spp (8-10).

Atul Garg *et al* studied wound infections secondary to snake bite and found that *Staphylococcus aureus* (32%) was the most common isolate followed by *Escherichia coli* (15%); monomicrobial infections were more frequent than polymicrobial infections. The majority of the isolates were antibiotic sensitive. The major limitation of this study is that anaerobic culture was not performed and only aerobic organisms were cultured (11).

Ka Keung Lam *et al* did a cross-sectional survey of snakes’ oral bacterial flora from Hong Kong, SAR, China and found 406 bacterial isolates of 72 different species were cultured: these included Gram negative and positive bacterial species and also anaerobic bacterial species. With the exception of the white-lipped pit viper (*Cryptelytrops albolabris*), venomous snakes harboured more pathogenic bacteria and total bacteria species compared to the non-medically important species. Of the venomous snakes, the Chinese cobra (*Naja atra*) harboured the largest number of bacterial species (12).

Bacterial meningitis is an acute infection within the subarachnoid space. Once the bacteria enter the bloodstream, they are able to avoid phagocytosis by neutrophils and classic complement mediated bactericidal activity. They reach the choroid plexus infect the choroid plexus epithelial cells and gain access to CSF (13).

Although there is no direct evidence to prove the correlation between snake bite and bacterial meningitis, our patient had a punctured wound on the calf muscle following which, hypothetically proposing, the oral microbial flora of the biting snake must have had a direct vascular access which in turn must have led to the bacterial meningitis as discussed above.

*Staphylococcus aureus* and coagulase negative staphylococci are important causes of meningitis that occur following invasive procedures (13). Snake bite puncture wound can be considered similarly invasive, of course to a lesser extent. *Staphylococcus aureus* and coagulase negative staphylococci occurred to be the most common amongst the cultured aerobic bacteria found in wound infections secondary to snake bite (11, 12). Vancomycin is supposed to be the drug of choice for staphylococcal meningitis (13). We treated our patient with the same and witnessed complete recovery. The cause for meningitis in patients post snake bite and its treatment remains a very promising area for research.

4. Conclusion:
The patient had neurotoxic snakebite envenomation. The clinical profile of acute bacterial meningitis, temporal relation to the snake bite, absence of other causes like sinusitis, chronic and debilitating diseases or any invasive surgical procedure and complete recovery following treatment with antisnakevenom, neostigmine, atropine and broad spectrum antibiotics strongly suggest meningeal involvement secondary to snake bite. Our case is a step towards the direction pointing to the rare possibility of bacterial meningitis complicating snake bites which needs further research. This phenomenon should be kept in mind and thoroughly investigated if the clinical presentation so suggests. As, *YOUR EYES SEE ONLY WHAT YOUR MIND KNOWS.*

References
3. Bawaskar HS, Bawaskar PH, Envenoming by the common krait