Case Report

Beyond the venom: Unmasking leukoencephalopathy in snakebite envenomation

Saurabh Srivastava[1], Deepak Sharma[2], Preeti Solanki[3], Mohd Laraib[4], M Sakthidhasan[5]

From [1]Professor, [2]Assistant Professor, [3]Assistant Professor, [4]Resident, Department of Medicine, Government Institute of Medical Sciences, Greater Noida, Uttar Pradesh, [5]Consultant Medicine, Department of Medicine, Keeramangalam Primary Health Centre, Pudukkottai, Tamil Nadu, India

ABSTRACT

Snakebite envenomation remains a significant public health challenge in India, yet central nervous system complications such as leukoencephalopathy are exceptionally rare. This report describes a 24-year-old farmer who presented with rapidly progressive neurological deterioration over 12 h following a snakebite. Despite standard treatment, persistent neurological deficits prompted neuroimaging, which revealed leukoencephalopathy. Comprehensive treatment involving mechanical ventilation, polyvalent anti-snake venom, corticosteroids, and neuroprotective agents facilitated a gradual recovery from an initial akinetic-mute state to functional independence. This case underscores the critical importance of early neuroimaging in unexplained acute neurological presentations within endemic regions to ensure accurate diagnosis and prompt, targeted therapy.

Key words: Anti-snake venom, Leukoencephalopathy, Neuroparalytic, Neurotoxicity, Snakebite envenomation

Snakebite envenomation represents a critical public health concern in tropical and subtropical regions, especially in India, where it significantly contributes to morbidity and mortality [1]. The neurotoxic effects of snake venom manifest through diverse clinical presentations, ranging from acute respiratory paralysis to various neurological complications. While acute manifestations such as ptosis, ophthalmoplegia, and respiratory failure are well-documented, certain neurological complications like subarachnoid hemorrhage, intracerebral hemorrhage, ischemic stroke, acute disseminated encephalomyelitis (ADEM), and leukoencephalopathy occur less frequently [2,3]. Refractoriness to standard therapeutic protocols in neurotoxic envenomation warrants investigation into atypical etiologies.

We report this rare incidence of snakebite-induced leukoencephalopathy to underscore the necessity of extended diagnostic evaluation in such clinical scenarios.

CASE PRESENTATION

A 24-year-old male farmer from a rural background presented to the emergency department during the monsoon season in a state of progressive neurological deterioration. The sequence of events began with the abrupt onset of symptoms approximately 12 h before hospital admission. The patient was in his usual state of health until the morning of presentation, when he experienced a sudden onset of severe abdominal pain just after waking up from sleep. The patient noticed a bite mark on his right foot suggestive of a snake bite. The abdominal pain was severe, colicky, diffuse, non-radiating, and accompanied by multiple episodes of nausea and vomiting. After approximately 4 h, he developed bulbar symptoms characterized by progressive difficulty in swallowing and slurred speech, and it was followed by generalized muscular weakness. Then the patient developed respiratory distress, initially manifesting as mild breathlessness that steadily progressed to severe respiratory distress. The patient had no significant medical history and denied any chronic illnesses, previous hospitalizations, or regular medications. He maintained regular working hours in agricultural fields and followed a mixed dietary pattern without any substance abuse.

On emergency department arrival, vital parameters revealed blood pressure of 144/80 mmHg, pulse rate of 130/min, which was regular in rhythm, oxygen saturation of 40% at room air, and respiratory rate of 08 breaths/min. The patient was drowsy at the time of presentation to our hospital (Glasgow coma scale-E2V3M4). The general examination revealed a patient in respiratory distress

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Correspondence to: Deepak Sharma, F-274, Sector 36, Greater Noida - 201310, Uttar Pradesh, India. E-mail: deepak.102sharma@gmail.com

with bilateral ptosis and peripheral cyanosis. Pupils were sluggishly reactive to light bilaterally. Meningeal signs were not elicitable at presentation. Plantar response was equivocal. No significant clinical findings were observed on abdominal and cardiovascular system examination. The computed tomography scan of the head showed no abnormalities.

Based on initial clinical evaluation and other circumstantial evidence, the patient was provisionally diagnosed as a case of neuroparalytic snake bite, probably due to krait envenomation, which is common in that particular geographical region during the monsoon season. The patient was then transferred to the intensive care unit for invasive mechanical ventilation and further conservative measures.

The initial investigations of the patient- hemogram, liver and kidney function tests, and urine examination were within normal range. Smear for Malarial parasites was negative. Hepatitis B surface antigen, Hepatitis C virus antibody, and enzyme-linked immunosorbent assay for human immunodeficiency virus were non-reactive. Cerebrospinal fluid (CSF) examination was normal. The CSF and serum were negative for viral markers, especially dengue, Herpes simplex, and Japanese encephalitis. Ultrasound scan of abdomen revealed no abnormalities.

The ptosis of the patient resolved along with spontaneous eye movements in the first 24–48 h of mechanical ventilation and administration of polyvalent Anti-snake venom (ASV) as per the protocols [3]. The patient did not show any significant improvement in motor response even after 72 h of the treatment (akinetic-mutism). To further search for the neurological insult, the patient then underwent contrast-enhanced magnetic resonance imaging (MRI) of the brain, which showed bilateral symmetrical T2/fluid-attenuated inversion recovery hyperintensity in the bilateral putamen and head of caudate nucleus, displaying restriction on diffusion weighted imaging (Fig. 1).

Based on clinical findings and MRI findings, we arrived at a diagnosis of snake bite-induced leukoencephalopathy. The patient was managed conservatively and initiated with an injection of methylprednisolone 500 mg once a day for 3 days, along with a combination of levodopa with carbidopa (100 mg + 25 mg) titrated according to clinical response. The other supportive measures, such as cerebral decongestants such as mannitol, enteral feeding, stress ulcer prophylaxis, and limb physiotherapy, were continued. The patient responded well to the treatment and was extubated from the ventilator on the 9th day of admission. The patient was kept on regular follow-up for 6 months, and there was significant improvement in power and other neurological functions.

DISCUSSION

The development of central nervous system pathologies following snake bite, particularly leukoencephalopathy, represents an exceptionally rare manifestation that warrants careful consideration. The MRI findings of bilateral symmetric hyperintensities in the putamen and caudate nucleus represent a characteristic pattern that differs from other causes of toxic leukoencephalopathy. Other differential diagnoses related to this clinical entity include hypoxic ischemic encephalopathy, ADEM, posterior reversible encephalopathy syndrome, and brainstem stroke.

The development of leukoencephalopathy following snake envenomation appears to involve multiple pathophysiological pathways as follows:

a. Direct neurotoxicity: Snake venom contains various neurotoxic components that can potentially cross the blood-brain barrier and can directly affect the central nervous system white matter [2]
b. Vascular injury: The vascular effects of snake venom may contribute to white matter damage through microangiopathy and endothelial dysfunction [4]
c. Immune-mediated response: The development of leukoencephalopathy may also involve immune-mediated processes, potentially triggered by either the venom itself or the administered antivenom. This mechanism is particularly relevant in cases where neurological deterioration occurs despite initial improvement with antivenom therapy [4].

The prompt administration of ASV remains the cornerstone of treatment, and it should be started

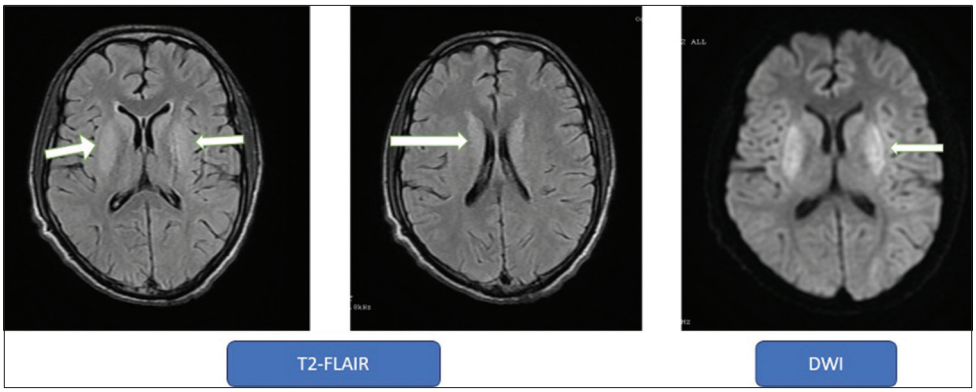

Figure 1: Magnetic resonance imaging brain showing bilateral putamen hyperintensities in T2-weighted image and fluid-attenuated inversion recovery sequences. Diffusion weighted imaging sequence showing restricted diffusion in the bilateral putamen and head of caudate nucleus

promptly in cases of snakebite. The addition of corticosteroids and other neuroprotective agents may play a crucial role in minimizing white matter damage in such patients. The transition from an akinetic-mute state to complete functional recovery suggests that aggressive management can lead to favorable outcomes despite severe initial presentation. The resolution of extrapyramidal symptoms with levodopa therapy indicates the potential reversibility of basal ganglia dysfunction in these cases [5]. Some reported cases also mentioned the role of conservative management and empirical ASV in managing such cases [6,7]. There is a possibility of seizures in such cases, particularly in pediatric age groups, and can be managed with antiepileptics along with standard treatment [8].

CONCLUSION

This case emphasizes that the diagnosis often relies on a high index of clinical suspicion, supported by characteristic clinical and neuroimaging findings. The multifaceted treatment approach employed in this case, combining antivenom therapy, neuroprotective measures, and supportive care, proved effective in achieving complete neurological recovery.

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