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Pseudoâ€“Brain Death After Neurotoxic Snakebite: A Case Report

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1. Abstract

Snakebite envenomation remains a major public health problem in tropical regions [1,4] and can rarely present with severe neuroparalysis mimicking brain death. Neurotoxic bites, particularly from elapid snakes, contain α- and β-neurotoxins that disrupt neuromuscular transmission, leading to descending paralysis, bulbar involvement, and respiratory failure. In severe cases, paralysis may cause absent brainstem reflexes, a phenomenon referred to as pseudo–brain death [7-9]. Such presentations are diagnostically challenging, especially when patient history is unclear and neuroimaging, EEG, and CSF analysis are normal. We report a case of a 34-year-old male presenting with unexplained coma and absent brainstem reflexes, who showed complete neurological recovery following empirical administration of anti-snake venom (ASV) and supportive care. This case highlights the importance of high clinical suspicion for neurotoxic envenomation in patients with unexplained coma, even in the absence of local signs or clear history.

2. Introduction

Snakebite poisoning remains a major health problem, especially in tropical regions. The World Health Organization considers snakebite a neglected tropical disease, particularly affecting rural populations [1]. It contributes to mortality of more than 100,000 people and morbidity of more than 400,000 people every year. Venomous snakes mainly belong to the families Elapidae (neurotoxic), Viperidae (hemotoxic), and Hydrophiidae (myotoxic) [2]. The family Elapidae includes cobras, kraits, and coral snakes, and members of this family predominantly possess neurotoxic venom that interferes with neuromuscular transmission, resulting in descending paralysis, beginning with ptosis and progressing to bulbar and respiratory muscle weakness. In severe cases, profound neuroparalysis may mimic brain death, with absent brainstem reflexes and unresponsiveness. Early recognition and prompt administration of anti-snake venom along with supportive care can lead to complete neurological recovery, even in brain-death-like presentations [3]. Here, we present a case of a 34-year-old male presenting in a state of pseudo-coma with no clear history. He was given anti-snake venom empirically and showed dramatic improvement thereafter.

3. Case Report

A 34-year-old male was brought to the emergency department unconscious and cyanosed. On arrival, his vital parameters were blood pressure 180/96 mmHg, pulse rate 131 beats per minute, oxygen saturation 30% on room air, and Glasgow Coma Scale (GCS) score of E1M1V1. He was immediately intubated and placed on mechanical ventilation. He was accompanied by a friend who was unaware of the exact sequence of events. According to the friend, the patient had been complaining of uneasiness since morning, along with recurrent episodes of presyncope. The patient was conscious prior to arrival at the hospital but became unconscious and unresponsive while being transported, just outside the hospital premises.

Electrocardiography revealed sinus tachycardia without any significant ST-T changes. Transthoracic echocardiography was within normal limits. Non-contrast computed tomography (NCCT) of the head was unremarkable, and subsequent magnetic resonance imaging (MRI) of the brain also revealed no abnormalities. The patient was started on antibiotics for aspiration pneumonitis. Despite supportive treatment, there was no neurological improvement, and the patient remained unresponsive with absent brainstem reflexes. Electroencephalography (EEG) and cerebrospinal fluid (CSF) analysis were within normal limits.

Despite detailed discussions with relatives and attendants, no significant history could be obtained, as the patient lived alone and was reportedly well the previous night. His GCS remained unchanged. After reviewing the literature on conditions that can mimic brain death, a decision was made on day 2 to empirically initiate anti-snake venom (ASV) therapy. Following administration of 30 vials of ASV, the patient began to show flickering movements of the eyes and tongue and started responding to commands with tongue movements. By day 4, after completion of ASV therapy, there was gradual improvement in distal muscle strength, followed by proximal muscle strength in both upper and lower limbs.

The patient could not provide specific details regarding the event and only reported that something had bitten him, though he could not recall what it was. He did not see a snake, and no fang marks were identified. He was subsequently extubated and discharged

home in stable condition.

4. Discussion

Snakebite envenomation continues to be a major public health problem in tropical and subtropical regions, particularly in South Asia, where it disproportionately affects rural populations and often presents with incomplete or unreliable histories [4]. India accounts for a substantial proportion of global snakebite-related mortality and morbidity, largely due to delayed recognition, limited access to healthcare, and the wide distribution of highly venomous species [5].

Neurotoxic envenomation is mostly caused by snakes belonging to the family Elapidae, which includes cobras (Naja species) and kraits (Bungarus species) [6]. The venom of elapid snakes contains low-molecular-weight neurotoxins, primarily α-neurotoxins and β-neurotoxins, which interfere with neuromuscular transmission at both postsynaptic nicotinic acetylcholine receptors and presynaptic nerve terminals [7]. This results in a characteristic descending paralysis that typically begins with cranial nerve involvement, manifesting as ptosis, ophthalmoplegia, and facial weakness, and may progress to bulbar dysfunction, respiratory muscle paralysis, and quadriplegia [8]. In severe envenomation, neuroparalysis may be so profound that patients appear deeply comatose, with absent pupillary, corneal, oculocephalic, and gag reflexes, thereby closely mimicking brain death [9]. Importantly, cerebral cortical function remains intact in such cases, and the apparent loss of brainstem reflexes is due to peripheral neuromuscular blockade rather than irreversible brain injury. This phenomenon, often referred to as "pseudo–brain death," has been reported particularly following krait envenomation, which is notorious for minimal local signs, nocturnal bites, and the absence of fang marks [10].

The diagnostic challenge is further compounded when neuroimaging, electroencephalography, and cerebrospinal fluid analysis are normal, as observed in the present case. In such scenarios, clinicians must carefully consider reversible causes of coma and absent brain-stem reflexes before making prognostic conclusions or initiating end-of-life discussions. Toxic, metabolic, and neuromuscular causes—including snake envenomation, botulism, organophosphate poisoning, and severe Guillain–Barré syndrome—are well-recognized mimickers of brain death [11].

Empirical administration of anti-snake venom (ASV) plays a critical role in suspected neurotoxic envenomation, particularly in endemic regions and in cases of unexplained acute neuroparalysis. Several case reports and series have demonstrated complete neurological recovery following timely ASV administration, even in patients presenting with clinical features indistinguishable from brain death [12–14].

This case highlights the need for high suspicion of neurotoxic snakebite in unexplained coma and early empirical ASV therapy for effective recovery.

5. Conclusion

Neurotoxic snake envenomation can rarely present with severe neuroparalysis mimicking brain death, especially in the absence of a clear history or local signs of envenomation. A high index of clinical suspicion and timely empirical administration of anti-snake venom, along with appropriate supportive care, can result in complete neurological recovery.

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