ORIGINAL RESEARCH PAPER
Volume - 13 | Issue - 02 | February - 2024 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

BRAIN DEATH – COULD IT BE A SNAKE BITE?

Anesthesiology

Dr. Alshipa Senior Resident, Department of Anaesthesia and Critical Care, Christian Medical College and Hospital, Ludhiana, Punjab, India

Dr. Seema Sharma House Doctor (MBBS) Department of Anaesthesia and Critical Care, Christian Medical College and Hospital, Ludhiana, Punjab, India

Dr. Samir Paul Assistant Professor, Department of Anaesthesia and Critical Care, Christian Medical College and Hospital, Ludhiana, Punjab, India

Dr. Vineeth Jaison* Professor, Department of Neurology, Christian Medical College and Hospital, Ludhiana, Punjab, India *Corresponding Author

ABSTRACT

In toxicology literature, snake bites were the second toxicology-relevant cause mimicking brain death. This is a case report of 22 year old man already on mechanical ventilation with fixed dilated pupils, absent brain stem reflexes and a GCS of E1VTM1 on arrival in casualty. The patient accomplished full neurological recovery after using a novel combination of Polyvalent Snake Antivenom (PSA) and anticholinesterases where snake bite was the diagnosis of exclusion. This case highlights a unique presentation of snake bite induced brain death mimicking. Thus, intensivist should exclude neuroparalytic effect of snakebite before considering withdrawal of ventilatory support or organ donation.

KEYWORDS

Neurotoxic Snake Bite, Brain Death, Anti-snake Venom (ASV) And Icu Management Of Snake Bite.

INTRODUCTION

Brain Death is defined as "irreversible cessation of brainstem functions." Indian law requires two consecutive bedside tests of brainstem functions to be negative to declare a patient brain dead. The pre-requisites to subject a patient to these bedside tests is to rule out reversible causes that may confound the findings. Neuroparalytic snake bite is a common emergency and a global concern. It is recognized as "neglected tropical disease" by WHO. Worldwide approximately 5 million people are bitten by snake every year with 100,000 deaths and 30,000 amputation and permanent disability. Rarely it may mimic brain death or locked-in-state with absent reflexes and complete ophthalmoplegia. We present a case report of 22 year old male of common krait (bungarus caeruleus) bite that remained in such state for over 5 days before a gradual and sustained recovery.

Case Report

A 22-year-old male who was an orphan presented to our tertiary care center ICU on mechanical ventilation with fixed dilated pupils, absent brain stem reflexes and a GCS of E1VTM1 on arrival. History given by the aunt at admission was of sudden onset early morning (3 am) occipital headache, 2 episodes of vomiting, dysphasia, up-rolling of eyes, involuntary body movement, difficulty breathing. Past medical history also revealed that he was a reformed IV drug user, HIV and HCV positive with CD4 Count 279 was receiving antiviral treatment for the last 2 years (Dolutegravir, Lamivudine, Tenofovir and Isoniazid).The patient was taken to a rural hospital, where he was intubated and referred to CMC for further management. On examination pupils fixed and dilated, corneal reflex absent, dolls eye test negative, cold caloric test negative, gag reflex absent and no spontaneous breath. He had no visible external injuries or bite/fang marks. Patient was on VCV mode with low ventilatory settings, neither on inotropic supports nor on sedation hemodynamically stable. All lab values were within normal range except for raised CPK of 400. Patient was screened for all reversible causes that mimics brain death. Toxicology screen, CSF studies were normal. MRI brain showed T2 Flair gyral hyperintensities involving left posterior temporal region and left middle cerebellar peduncle.EEG showed nonspecific neurophysiological disturbances over both cerebral hemispheres. A multidisciplinary meeting of the neurologist, Internal medicine and the Intensivist was held. A repeat detailed history revealed patient had gone to the washroom around 2:30 am followed by above mentioned symptoms and progressive weakness involving neck and proximal muscles, with breathing difficulty over a few hours. A nerve conduction study was ordered which showed absent motor responses with normal sensory action potentials in upper and lower limbs. Possibility of complete neuromuscular blockage due to toxin likely snake envenomation (Krait) was kept. Differentials kept was Guillain Barre Syndrome. Considering descending paralysis and high CPK values, Krait envenomation was high on suspicion and ASV treatment was initiated. On day sixth he showed signs of improvement, particularly in the movement of eyelids and distal upper extremities. The patient was extubated on day 15 post ASV treatment.

DISCUSSION

Snake bite remains an underestimated cause of accidental or occupational death in modern India. About 50% bites are dry bites that result in negligible envenomation. The three major families of venomous snakes are the Elapidae, the Viperidae, and the hydrophiidae. Elapidae includes Cobra and krait snake which are typically neuroparalytic, whereas envenomation by *Viperidae* species shows characteristic vasculotoxic and hemotoxic features. The most commonly found snakes in North India are from Elapidae family. Neuroparalytic snake bite is associated with higher mortality rates due to the rapid onset of respiratory paralysis and as most envenomation occur in rural areas, these patients may not reach a center capable of providing mechanical ventilation in time. Neuroparalysis in the neurotoxic snake is due to blockade of neuromuscular receptors pre- and post- synaptically in krait bite and post synaptically in cobra bite. Skin manifestation is not present in krait bite (absent fang marks or painless bite). Paralysis begins within the 1st h is heralded by ptosis, followed by external ophthalmoplegia followed by distal muscles. Recovery is slow with krait envenomation due to irreversible binding of the toxin to pre-synaptic receptors and hence clinical recovery occurs slowly and only with formation of new neuromuscular junctions and duration varies from 30 h to 6 days.

RESULT:

Snake envenomation should be considered as a potential treatable cause of acute neuroparalytic even without history based on rural background, rainy season, harvesting season and time of bite. In north India possibility of snake bite must be considered before declaring brain death. A multidisciplinary approach and reversible causes of brain death must be ruled out.

REFERENCES:

[1] Law AD, Agrawal AK, Bhalla A. Indian common krait envenomation presenting as coma and hypertension: A case report and literature review. J Emerg Trauma Shock. 2014 Apr;7(2):126-8.
[2] Thakre M.Snake bite poisoning presenting as pseudo brain death. J. Neurol. Sci. 2019;(405):343.
[3] Agarwal S, Kaeley N, Khanduri S, Kishore N. Brain dead presentation of snake bite. Indian J Crit Care Med 2018;22(7):541–543.
[4] Senthilkumaran S, Balamurugan N, Nath Jena N, Thirumalai kolundusubramanian P. Locked-in syndrome in post snake bite: can it be unlocked? Trop Doct 2018;48(3):257.