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Case Report Open Access

Sea Snake Bites Resulted in a Mimic of Brain Death

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Abstract

A 69 year old man with deep coma presented after an alleged history of sea snake bite. Patient presented with irresponsiveness to painful stimuli, complete ptosis, internal and external ophthalmoplegia. He was given continuous ventilator support despite signs of brain stem dysfunction. On admission day 20, the patient was weaned off ventilator and discharged from the hospital. Whenever treating patients who were suspected with a history of snake bite, an emergency physician should always consider severe envenomation in the differential diagnosis of acute neuroparalytic syndrome, which may avert the unfortunate misdiagnoses of brain death.

Keywords: Brain death; Snake bites; Snake envenomation; Ophthalmoplegia

Introduction

Brain death is defined as the function loss of cerebrum and brain stem, resulting in coma, no spontaneous respiration and irresponsiveness of all brain stem reflexes [1]. However, a coma with symptoms and signs largely consistent with brain death may also occur following a fatal sea snake envenomation. This mimic of brain death presented in emergency room could pose a dilemma to emergency physicians regarding continuation of therapy [2].

Case Presentation

A 69 year old man with comatose presented to our emergency department from a local community hospital. He was allegedly bitten by a sea snake (later confirmed by the patient after his recovery) on his left inner ankle while walking along the shore of China yellow sea in the morning. He passed out during his walking back home, and was urgently taken to a community hospital. He was intubated, put on ventilator support, and was given one dose of anti snake venom therapy (unknown dose) before referring to our hospital along with a provisional diagnosis of sea snake bite with suspected brain death.

On examination, the pulse was 100 beats per minutes, the blood pressure was 100/70 mm Hg, oxygen saturation (SaO_2) was 70% and body temperature was 98.1°F. Patient presented with deep coma, irresponsiveness to painful stimuli, complete ptosis, internal and external ophthalmoplegia. There was no deep tendon and plantar reflexes after given the eliciting stimulus. The Glasgow Coma Scale Score was E1VTM1. He had bilateral fixed dilated pupils measuring about 6 mm in diameter, which were not reacting to the light stimulus. His breathing was quick and shallow with apnea. He had grade 0 power in all four limbs. Three clean and typical fang marks were found on his left inner ankle with no signs of bleeding or swelling. He became anuric upon catheterization.

Under endotracheal intubation and some spontaneous respiratory effort, the patient was being ventilated via a mechanical ventilator on a synchronized intermittent mandatory ventilation (SIMV) mode. He was treated with polyvalent anti snake venom therapy at a dose of 100 ml stat, followed by intravenous (IV) injection of Furosemide 20 mg and Dexamethasone 10 mg, and infection prophylaxis together with supportive cares. Over a period of 48 hours, both pupils have constricted back to normal with voluntary eye movements, and the oxygen saturation improved. However, he still had grade 0 power in his four limbs, and showed no spontaneous respiratory movements and no responses on tracheal suctioning. The patient went onto full ventilator support along with continuous supportive cares. During the next 4 days, the patient presented gradual neurologic recovery with a response to verbal commands by moving his limbs and raising the eyebrows. At 10 days after sea snake bites, the patient presented with acute complication of gastrointestinal stress ulcer bleeding, and was treated with medicine to protect the gastric mucosa, inhibit gastric acid secretion along with supportive therapy. He was weaned off the ventilator and extubated after 15 days of ventilation. The patient was breathing well and had complete neurological recovery, and discharged on the 20th day of admission in a stable condition.

Discussion

Snake toxins vary greatly in their functions, among which two broad classes of toxins are neurotoxins (mostly found in elapids) and hemotoxins (mostly found in viperids) [3,4]. Polypeptide neurotoxins in snake venoms can cause muscle paralysis by binding to the postsynaptic portion at the neuromuscular junction to produce a competitive or noncompetitive nicotinic acetylcholine receptor blockade, or affecting the mode of neurotransmitter release at the presynaptic motor nerve endings to cause irreversible loss of functions; hence, clinical recovery occurs slowly and only with the formation of a new neuromuscular junctions [5-7].

Usually, little or no pain is involved in sea snake bites, and it is rare for any local signs to be presented after envenoming [8]. However, a

delayed onset of severe envenomation resulting in respiratory muscle weakness might occur due to the absorption of neurotoxin into wound bed, which apparently is a major component of elapids and some sea snake venoms [5,9,10]. Neuromuscular paralysis is the predominant toxic effect of sea snake venom, which can cause a rapid development of respiratory failure (as in the present case). Together with the sequelae of cardiac arrest and renal failure, respiratory paralysis will more likely result in the hypoxic effect on the brain that eventually lead to a mimic of brain death [5]. Brain death implies the irreversible function loss of cerebrum and brain stem, which clinically presented as the absence of pupillary light, corneal, oculocephalic, oculopharyngeal, oculovestibular, and respiratory reflexes [5]. Absent cerebral functions are manifested by occurrence of deep coma and both internal and external ophthalmoplegia as seen in this case, which would misdiagnose the brain death in many ways [2]. This mimic of brain death can falsely guide an emergency physician to consider withdrawing the ventilator support, which has become mostly trustful to efficiently wear off the effects of venom even without assistance of anti-snake venom (ASV) [5,10]. Although this option of solely giving mechanical ventilation support is only acceptable to cases where there is no ASV or dilemma in diagnosis.

In conclusion, it is vitally important that severe snake envenomation shall always be considered in the differential diagnosis of acute neuroparalytic syndrome when an emergency physician is accepting comatose patients, especially if associated with a known suspect of sea snake bites in a coastal area. This care will avert the unfortunate misdiagnoses of brain death, which often pose a dilemma to the emergency physicians regarding continuation of therapy.

References

1. Wijdicks EF (2001) The diagnosis of brain death. N Engl J Med 344: 1215-1221.
2. John J, Gane BD, Plakkal N, Aghoram R, Sampath S (2008) Snake bite mimicking brain death. Cases J 1: 16.
3. Sung CK, Hah MC (1981) A case of extraocular muscle paresis by viper bite. J Korean Ophthalmol Soc 22: 261-263.
4. Alirol E, Sharma SK, Bawaskar HS, Kuch U, Chappuis F (2010) Snake bite in South Asia: a review. PLoS Negl Trop Dis 4: e603.
5. Sodhi R, Khanduri S, Nandha H, Bhasin D, Mandal AK (2012) Brain death--think twice before labeling a patient. Am J Emerg Med 30: 1321.
6. Lee SW, Jung IC, Yoon YH, Hong SH, Han KS, et al. (2004) Anticholinesterase therapy for patients with ophthalmoplegia following snake bites: report of two cases. J Korean Med Sci 19: 631-633.
7. Vithanage KK, Thirumavalavan K (2012) A case of a sea snake bite resulting in fatal envenoming. Ceylon Med J 57: 174-175.
8. Senanayake MP, Ariaratnam CA, Abeywickrema S, Belligaswatte A (2005) Two Sri Lankan cases of identified sea snake bites, without envenoming. Toxicon 45: 861-863.
9. Bhattacharya P, Chakraborty A (2007) Neurotoxic snake bite with respiratory failure. Indian J Crit Care Med 11: 161-164.
10. Bomb BS, Roy S, Kumawat DC, Bharjatya M (1996) Do we need anti snake venom (ASV) for management of elapid ophitoxaemia. J Assoc Physicians India 44: 31-33.