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Case Report

A Case Report on Neurotoxicity in Snakebite with Alleged History of Chest Pain and Aphasia

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ABSTRACT

Snake bites are accepted medical emergencies in several elements of the globe, particularly in rural areas. Asian country is calculable to own the very best snake bite mortality within the world. A seventy year recent feminine was delivered to the emergency department with alleged history of pain, encephalopathy and unintelligible speech of unexpected onset since 6 am. She was apparently traditional the previous night. Next day morning once she awakened, she had slurring of speech with no weakness of limbs. Elaborate medical specialty examination unconcealed GCS of E4VTM1 with depressed brain stem reflexes, pupil was a pair of 5 metric linear unit reacting to lightweight, doll's eye absent, cough and unconditioned reflex were absent.

Keywords: Intensive care unit; Computed tomography; Cardiovascular; Neurotoxicity; Neuroparalytic

INTRODUCTION

Snake bites are well known medical emergencies in many parts of the world, especially in rural areas. India is estimated to have the highest snake bite mortality in the world. World Health Organisation (WHO) estimated the number of bites to be 83,000 per annum with 11,000 deaths [1]. In India, the common species of snakes seen are *elapidae* which includes common cobra, king cobra and krait, *viperidae* which includes Russell's viper, pit viper and saw-scaled viper and *hydrophidae* (the sea snakes) [2].

CASE PRESENTATION

A 70 year old female was brought to the emergency department with alleged history of chest pain, aphasia. Slurred speech of sudden onset since 6 am. She was apparently normal the previous night. The next day morning when she woke up, she had slurring of speech with no weakness of limbs. She was initially taken to nearby government hospital where some injections given. On arrival at emergency department, she was conscious, responding to simple commands with Glasgow Coma Scale (GCS) E4V1M6 with blood pressure of 150/90 mmHg, PR-86/min, saturation was 54% in room air and was intubated. Her pre-intubation ABG revealed type 2 respiratory failure and post-intubation ABG showed improvement. She was admitted in ICU.

Detailed neurological examination revealed GCS of E4VTM1 with depressed brain stem reflexes, pupil was 2.5 mm reacting to light, doll's eye absent, cough and gag reflex were absent. She had generalised hypotonia with absent deep tendon reflexes and bilateral plantar was mute. She had normal vesicular breath sounds in all chest areas. Cardiovascular examination revealed ejection click at mitral area and abdominal examination was normal as shown in Table 1.

Patient	Signs and symptoms	Investigations
70 year old female	Alleged history of chest pain, aphasia	Neurological examination revealed GCS of E4VTM1 with depressed brain stem reflexes, pupil was 2.5 mm reacting to light, doll's eye absent, cough and gag reflex were absent.
	Slurring of speech with no weakness of limbs	CT brain done which showed age-related cerebral atrophy. MRI brain done which revealed small vessel ischemic changes.

Table 1: Signs, symptoms and investigations on a patient with neurotoxicity in snake bite.

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Differential diagnosis of cerebrovascular accident or hypokalemic paralysis or acute inflammatory demyelinating polyneuropathy or neurotoxic snake bite or acute myasthenia syndrome was considered.

Her baseline investigations showed normal hemogram and biochemical parameters. Hence hypokalemic paralysis was ruled out. CT brain done which showed age-related cerebral atrophy. MRI brain done which revealed small vessel ischemic changes. Hence cerebrovascular accident was ruled out. CSF analysis was normal. Hence acute inflammatory demyelinating polyneuropathy was ruled out. She was started on anti-snake venom in view of neurotoxic snake bite as elapid bite would mimic brain death. Her neurological examination of motor system and ophthalmoplegia showed improvement within 24 hours. She was gradually weaned off ventilator and extubated after 72 hours.

Acetylcholine receptor antibodies were normal. Nerve conduction study of both ulnar nerves revealed symmetrical motor axonal neuropathy. Repetitive nerve stimulation done which was normal, hence myasthenia was ruled out. She was discharged after 10 days with full improvement in clinical status.

DISCUSSION

Early Morning Neuroparalytic Syndrome (EMNS) or locked-in syndrome is a rare presentation of elapid bite that is commonly seen among farmers and slum dwellers that sleep out in open environment [3]. These patients are brought to the hospital with a history of ptosis and paralysis with no bite marks or local skin changes on the body. This is characteristic of the nocturnally active kraits whose bites are generally painless with minimal to no skin changes. It found that 60%-70% of snakebites occurred while the patients were asleep with 17% of these having undetectable bite marks on the body [4].

There have been various case reports on neurological manifestations mimicking brain death in krait bite "Is the patient brain dead". Snake bite mimicking brain death early morning neuroparalytic syndrome, Suppression of brain stem reflexes in snake bite two cases of locked in syndrome in snake bite reported and two cases of Early Morning Neuroparalytic Syndrome (EMNS) in the Tropics-masquerading as brain death [3-9].

Elapid neurotoxicosis acts at the peripheral neuromuscular junctions either post-synoptically or pre-synoptically causing paralysis. This paralysis is first detected as bilateral ptosis and external ophthalmoplegia [10], gradually progressing to involve muscles of palate, jaw, tongue, larynx, neck and muscles of deglutition usually but strictly not in that order [8]. The proximal muscles of the limbs are involved earlier than the distal, and there can be complete quadriplegia and locked-in state [11]. Patients with acute respiratory failure are categorized as severe envenomation. This generalized flaccid paralysis may occur with consciousness provided the patient is not in circulatory failure. The pre-paralytic symptoms may include numbness and paranesthesia. The neurotoxic effects spontaneously resolve over several days, median time of onset for recovery for respiratory failure is 2 days or in response to anti venom and anticholinesterase [1,2,11].

CONCLUSION

A differential diagnosis for an acute onset flaccid paralysis should include early morning neuroparalytic syndrome in snake bite endemic areas who sleep out in open environment and anti-snake venom should be started immediately even in absence of snakebite marks. Medical diagnosis of neural structure accident or hypokalemic paralysis or acute inflammatory demyelinating polyneuropathy or neurotoxic snake bite or acute carcinomatous myopathy was thought of her baseline investigations showed traditional hemogram and organic chemistry parameters. She was started on anti-snake venom seeable of toxin snake bite as snake bite would mimic death. Her medical specialty examination of motor system and palsy showed improvement among twenty four hours. She was step by step weaned off ventilator and extubated seventy two hours. A medical diagnosis for AN acute onset neurological disorder ought to embrace early morning neuroparalytic syndrome in snake bite endemic areas United Nations agency travel back and forth in open setting and anti-snake venom ought to be started right away even in absence of bite marks.

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