Early Morning Neuroparalytic Syndrome

Mohd Haneef

Koyili Hospital, Kannur, Kerala.*

ABSTRACT

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Snake bite is a common problem seen in the rural parts of India and is associated with a high rate of mortality due to their related toxicity. Neurotoxicity from the elapid bite may manifest as the early morning neuroparalytic syndrome (EMNS) or even as Locked in syndrome. Determining the types of toxicity in the patient is based on cl inical signs and laboratory findings or by direct examination of the snake. Patients presenting as EMNS do not have bite marks on their body and hence the diagnosis may be complicated. A 14 year old girl presented with a tingling sensation over her body who later developed respiratory failure is reported here. She was treated with a standard regimen of anti-snake venom (ASV) and atropine – neostigmine along with other supportive measures. She made a remarkable recovery with the above treatment and hence it is essential to consider a possibility of elapid envonomation in these patients presenting with acute neuroparalysis in snake bite endemic areas.

Keywords: Early morning neuroparalytic syndrome, Elapid bite

*See End Note for complete author details

INTRODUCTION

Snake bite envenomation is a common occurrence in India and is seen especially in the rural areas of our country. There are a total of about 2500 to 3000 species of snakes distributed worldwide and of which 500 are venomous.[1] In India, the common species of snakes seen are the 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]

It is estimated that there could be over five million cases of envenomation per year with about 100,000 developing complications.[3] Early morning neuroparalytic syndrome is a rare presentation of elapid bite. A few cases are reported from northern part of the country.[4] The absence of bite marks with no specific history of a snake bite makes diagnosis and management complex.

CLINICAL HISTORY

A 14 year old girl presented with abnormal sensations all over her body when she got up from sleep early in the morning and reported to the hospital at 4 am. She had no other significant past medical history. On examination, her vitals were stable with a normal systemic examination. She was kept under observation for further evaluation.

The young girl underwent a sequence of clinical tests such as complete hemogram (White blood cells – total and differential, erythrocyte sedimentation rate, platelets, hemoglobin), serum electrolytes (sodium and potassium), renal function test, liver function test, ECG and chest x-ray. The results of these tests reported normal.

She subsequently developed acute paralysis with respiratory failure 3 hours later on arrival to the hospital. She was intubated and put on continuous mechanical ventilation. At this point, she had partial ptosis (figure 1), dilated pupils, complete external ophthalmoplegia and flaccid quadriparesis with a flexor plantar response. Based on the clinical picture, a differential diagnosis of hypokalaemic paralysis, myasthenic crisis, drug over-dosage, Miller Fischer syndrome and EMNS were considered. Her cerebrospinal fluid examination was unremarkable and EEG did not show any epileptiform activity. Through exclusion,

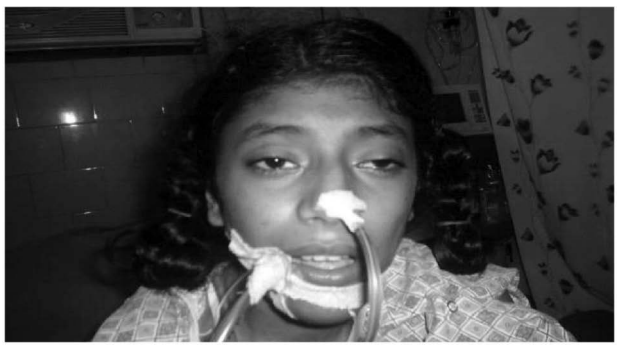

Figure 1. **Patient on ventilator – showing bilateral ptosis**

Corresponding Author:
Dr. Mohd Haneef, MD, PGDHS, Consultant Physician / Diabetologist, Koyili Hospital, Kannur, Kerala.
Mobile : 94472 36625, E-mail : dr.mhaneef@gmail.com

the most probable diagnosis for the symptoms was elapid envenomation hence she was started on anti snake venom (ASV) and atropine-neostigmine with a working diagnosis of EMNS. Within 3 hours, she began to show spontaneous respiratory effort and she required mechanical ventilation for another 12 hours. This recovery with ASV suggests the possibility of elapid envenomation. The ptosis and ophthalmoplegia resolved over the next two days and she was discharged after four days from the day of admission. After a week's follow up, she showed no signs of focal neurological deficits.

DISCUSSION

Early morning neuroparalytic syndrome is a rare presentation of the elapid bite.[4] It is commonly seen among farmers and slum dwellers that sleep out in the open environment.[4,5] These patients are brought to the hospital with a history of ptosis and paralysis with no bite marks on the body. Kraits are nocturnally active snakes and their bites are generally painless. Victims may not feel or recognize the bite. It seldom produces skin changes. Several cases of EMNS after krait bite have been reported.[6] In various studies 60-70% of the cases of snake bite occurred when the patients were asleep. The site of the bites was undetectable in 17% of the cases in a prospective study.[6] Elapid neurotoxicosis acts at the peripheral neuromuscular junctions either post-synaptically by binding competitively at acetylcholine receptors or pre-synaptically by preventing the release of acetylcholine from the nerve terminals.[6,7]

Paralysis is first detected as bilateral ptosis and external ophthalmoplegia progressing to involve the muscles of the palate, jaw, tongue, neck and deglutition. Generalized flaccid paralysis may result with consciousness provided the patient is not in circulatory failure.

Pre- paralytic symptoms include paresthesia and numbness. The young girl reported her initially presented herself with the same symptoms. However, neurological signs became evident only after she developed acute flaccid paralysis with respiratory failure.

Neurotoxic effects are completely reversible spontaneously over several days or weeks, or in response to antivenom and anticholinesterase. This was observed in this case where the girl responded dramatically to the treatment as mentioned by Dutta et al.[7]

CONCLUSION

A differential diagnosis for an acute onset of flaccid paralysis should include EMNS in snake bite endemic areas and ASV should be started immediately even in the absence of snake bite marks.

END NOTE

Author Information

Dr. Mohd Haneef, MD, PGDHS,
Consultant Physician / Diabetologist
Koyili Hospital, Kannur, Kerala.

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