



## LOCKED IN SYNDROME DUE TO NEUROTOXIC SNAKE BITE

### Medical Science

**Dr. Moses. P. Moorthy**

The Tamilnadu Dr Mgr Medical University, Chennai, India

**Prof. A.V. Srinivasan\***

The Tamilnadu Dr Mgr Medical University, Chennai, India\*corresponding Author

**Prof. K. Bhanu**

The Tamilnadu Dr Mgr Medical University, Chennai, India

### KEYWORDS

#### Objective:

To Document a case of complete locked in syndrome due to neurotoxic snake bite in the tropical region.

#### Background:

Neurotoxic snake bite is common in tropical region. Locked in syndrome due to neurotoxic snake bite, with complete recovery after treatment is not been described.

#### Methods:

A case report from tertiary medical center.

#### Results:

Female aged 32 years lost her vision at the age of 7years due to acute CNS infections sequel, had snake bite in her kitchen, following which she developed giddiness, difficulty in breathing and speech, brought to nearby hospital, she was intubated and referred to tertiary care center. On arrival she was unconscious, had bilateral ptosis, complete ophthalmoplegia, pupil 3mm with sluggish reaction to light, quadriparesis with absent spontaneous breathing. She had bite mark with cellulites in the right thumb. Investigation revealed whole blood clotting time of <20 minutes, other blood parameters were normal, ECG showed features of toxic myocarditis in the form of ST, T wave changes with echo cardiogram finding of global hypokinesia with ejection fraction of 40%, patient was treated with adequate dose of anti-snake venom, neostigmine and ventilator support, patient become conscious, communicative on third day. Patient gave history that she was able to understand the surroundings but not able to communicate with the surroundings. Patient was extubated on 6th day, discharged on 10th day in completely self ambulant state, neuro electrophysiological evaluation, cardiac function were normal at the time of discharge.

#### DISCUSSION

Snake envenomation produces neurotoxicity either by presynaptic or post synaptic inhibition at the neuromuscular junction. Clinically manifests as ptosis, ophthalmoparesis, bulbar weakness and respiratory failure.

Krait venom contains Bangarotoxin-A, Bangarotoxin-B. Bangarotoxin-A produces post synaptic inhibition. Bangarotoxin-B produces presynaptic inhibition. Neither antivenom nor neostigmine is impressive in krait envenomation. Assisted ventilation and supportive care brings full recovery within few days.

Cobra venom contains neurotoxic polypeptide which produces post synaptic inhibition at the neuromuscular junction. Patients will have myasthenic type of decrement in RNS during initial period. Patient improve well with the support of Antivenom, neostigmine and ventilatory support.

Viper group of snake bite produces neurotoxicity by presynaptic inhibition. The venom contains phospholipase A2. Patient usually improve with antivenom, rarely needs assisted ventilation. They do not show decrement pattern in Repetitive Nerve Stimulation.

Neurotoxic snake bites improves well with antivenom, neostigmine and adequate ventilatory support. If we provide adequate care at primary care level with ventilator support we can reduce the mortality due to neurotoxic snake bite.

With effective treatment ocular muscles recover in 2-4 days. Full recovery of motor function in 3-7 days.

Repetitive nerve stimulation was done on 10th day in right abductor pollicis brevis, right deltoid and right orbicularis oculi, which did not show significant decremental pattern on 10th day since the patient has recover completely.

This patient was admitted with profound quadriparesis, bulbar weakness, and respiratory muscle weakness, which required assisted ventilation. With mechanical ventilation, anti snake venom, neostigmine, patient recovered well on 10th day. Without any residual deficit. Electrophysiological evaluation done after recovery were normal.

#### Conclusion:

Adequate anti-snake venom, neostigmine and ventilator support in due time helps in complete recovery of locked in syndrome of neurotoxic snake bite.

#### Reference:

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