



Uncommon Presentation of Snake Bite

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Abstract

A 26 years male patient was presented to us two days after snakebite on forehead. Few hours subsequently to bite he developed neuromuscular weakness with difficulty in breathing requiring mechanical ventilatory support. He had bilateral total areflexia, bilateral mute planters reflex with hypotonia of all extremities and ptosis without any sensory involvement. His biochemical, Hematological and Electrophysiological features are suggestive of Gullain Barre syndrome following snakebite. This is a very unusual complication following snakebite and has therapeutic and prognostic significance.

Keywords- Gullain- Barre syndrome, Snakebite, Neuropathy.

Introduction

Snakebite is common but underestimated cause of accidental death and medical emergency in Indian particularly in rural areas. It is world's most affected region by snakebite, due to its high population density, widespread agricultural activities, numerous venomous snake species and lack of appropriate information regarding primary treatment (first aid) in general population.

While WHO estimated 83000 snake bite per annum with 11000 deaths¹, Million death study, 2011 conducted by Registrar General of India showed approximately 46000 deaths per annum in India².

There are more than 250 types of species of snake in India but roughly only 50 are poisonous³. The major families of poisonous snakes in india are-

A. Elapidae:- It include common cobra (*Naja naja*), king cobra (*Ophiophagus hannah*) and common krait (*Bungarus caeruleus*).

B. Viperidae:- It include saw-scaled viper (*Echis carinatus*), Russell's viper (*Dabiola russeli*) and Pit viper.

C. Hydropidae:- Sea snake

Snake venom is a complex mixture of toxic proteins and a cocktail of many components containing enzymes, aminoacids, lipids, metals and biogenic amines which are mainly hematotoxic, neurotoxic, myotoxic cytoyitic and nacrotoxic in nature. They cause local tissue damage ranging from pain, swelling of the bitten limb, muscle necrosis, abnormal blood clotting, spontaneous systemic bleeding, neurotoxicity leading to ptosis, ophthalmoplegia, limb weakness and respiratory muscles paralysis, cardiac involvement causing arrhythmias, hypotension and renal toxicity. There are only few cases of GBS following snakebite has been reported in literature. We are presenting a case of snake bite mimicking GBS.

Case

A 26 years old male patient admitted to our tertiary care hospital with history of snakebite on forehead while he was sleeping on ground. Few hours subsequently to bite he developed neuromuscular weakness with difficulty in breathing. He was immediately admitted to a local hospital where he was intubated, Put on ventilator support and managed symptomatically. There was no antecedent history of viral or bacterial infection of respiratory or gastrointestinal tract or

recent vaccination. He was subsequently transferred to our tertiary care hospital.

On arrival at our hospital he was in altered level of consciousness. There was superficial tiny abrasion over the forehead. He had Endotracheal tube in situ with shallow and fast breathing, tachycardia and muscle fasciculation. Neurologically he had bilateral total areflexia, bilateral mute planters with hypotonia of all extremities and ptosis. Other systemic examinations were essentially unremarkable. Bedside 20 minute coagulation test (20WBCT) was positive (Blood did not clot in plain vial in 20 minutes). Based on classical neurotoxic signs and haemostatic abnormality, anti snake venom (ASV) was given.

All routine Biochemical, haematological, Electrolyte and radiological test were normal.

Initially he needed controlled mode of ventilation from which he was successfully weaned from ventilator and extubated on 4th day of admission. He also slowly started showing neurological improvement in his sensorium, muscle power, reflex and ptosis. He denied any sensory abnormality.

Nerve conduction velocity (NCV) test was evident of pure motor axonal neuropathy. CSF examination was evident of Albumino-cytological dissociation (cell count- 2 lymphocyte/mm³, protein 142 mg/dl, sugar-98 mg/dl, ADA-2.8 unit/dl). Bedside 20 min coagulation test was also normalized. Though NCV and CSF picture indicate possibility of Pure axonal variant of GBS (AMAN variant) but our patient showed recovery after ASV administration and he improved fast without treating him with Plasmapheresis or IVIG

After 10 days of hospitalization he was discharge from hospital with almost total recovery except fine tremors of hands and persistent tachycardia. He became ambulatory.

Discussion

Snakebite is an important cause of acute medical emergency in India. Vipridie (vipers group) are mainly hematotoxic while Elipidie (Cobra and krait) are mainly neurotoxic. Various complications are related to venom toxins affecting the coagulation cascade, or neuromuscular transmission or both. A whole blood clotting time (20 WBCT) test is very useful to detect coagulation abnormality. Venom of viper contains metalloproteinases, serine proteases and C-type lentins having anticoagulant or procoagulant activity, and either agonists or antagonists of platelet aggregation causing bleeding and ischemic or hemorrhagic strokes. Elipidie venom has Phospholipase A₂, β -bungarotoxin and proteins that are potent neurotoxins affecting the neuromuscular transmission at either presynaptic or postsynaptic levels to inhibit peripheral nerve impulse causing muscle weakness⁴. Krait is most poisonous snake and its venom is 10 times more poisonous than cobra. Cobra venom is cardiotoxic, neurotoxic, hematotoxic and cytotoxic.

The common neurological manifestations are altered sensorium, ptosis, ophthalmoplegia, limb weakness, respiratory muscle weakness, palatal and neck muscle weakness, generalized flaccid paralysis, areflexia and delayed sensory neuropathy⁵. Some rare complications include

stroke (ischemic or hemorrhagic), Encephalopathy, GBS and cortical blindness⁶.

There are only few cases of GBS following snakebite has been reported in literature. GBS occurs due to cross reactivity between GM2 ganglioside and glycosidic epitopes of venom proteins.

Usually the neurological symptoms appears within six hours after the bite. Our patient also presented within six hours of bite with difficulty in breathing, ptosis, and neuromuscular weakness to local hospital. His clinical, biochemical, electrophysiological features and presence of albuminocytological dissociation were suggestive of GBS following snakebite. *Chuang et al*⁷ reported a patient who developed axonal GBS following Krait bite. This patient presented with symmetric paresis with sensory involvement in all limbs with cranial nerve involvement and mildly raised CSF protein at 4 weeks after snakebite. Electrophysiological study showed sensory and motor polyneuropathy. *Shrivastava et al*⁸ reported a case of 40 years old farmer presented with nonhealing ulcer on left ankle and tingling sensation after 5 weeks of snakebite. He had bilateral symmetrical hypotonia and areflexia with albumin-cytological dissociation with sensory and motor demyelinating neuropathy with secondary axonal degeneration suggestive of GBS and treated with plasmapheresis.

Conclusion

Guillian Barre Syndrome can occur after snakebite but due to poor reporting, actual incidence might be much higher. This cause significant morbidity in terms of prolong

hospitalization, high dependency on mechanical ventilator and slow recovery. In our patient, clinical, biochemical, and electrophysiological feature of GBS with absence of any other antecedent event, we considered the GBS following snakebite. This group of patient may require plasmapheresis or Intravenous Immunoglobulin (IVIG) with antsnake venom and other supportive care for complete and faster recovery.

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