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Guillain-Barré Syndrome: An Unusual Complication Following Snake Bite.

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ABSTRACT

A sixteen year old female alleged to have been bitten by snake presented with chief complaints of mild pain on right hand. She had been bitten by a snake at home at 2:00am. She had received anti-snake venom on the night of the bite and after 1 week she was discharged. The same patient presented to the hospital 2 weeks later with breathing difficulty. She had clinical, biochemical, and electrophysiological features of Guillain-Barré syndrome, with motor and sensory neuropathy—primarily suggestive of demyelination with axonal degeneration. After short term rehabilitation, patient had good recovery.

Keywords: Guillain-Barré syndrome, neuropathy, Krait snake bite, Antisnake venom

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INTRODUCTION

Guillain-Barré syndrome (GBS) also known as acute inflammatory or post –infective demyelinating polyneuropathy which develops 1-4weeks after respiratory infection or diarrhea in 70% of patients³ but can follow surgery or immunization. So far there are been only 2 case reports of GBS following snake bite in literature, first case in China in 1996 and second in India in 2008. We want to report a similar case which we have treated.

Case History

A sixteen year old female alleged to have been bitten by snake (krait) presented with chief complaints of mild pain on the right hand. On examination in RICU, patient was conscious, mild ptosis present and afebrile. Pulse rate 74/min, Blood pressure 90/60 mmHg. CVS and RS were clinically normal.

She was administered 100ml of anti- snake venom in 100 ml of 0.9% saline (loading dose) along with Neostigmine 1mg+glycopyrolate 0.2mg/hr infusion with antibiotic coverage.

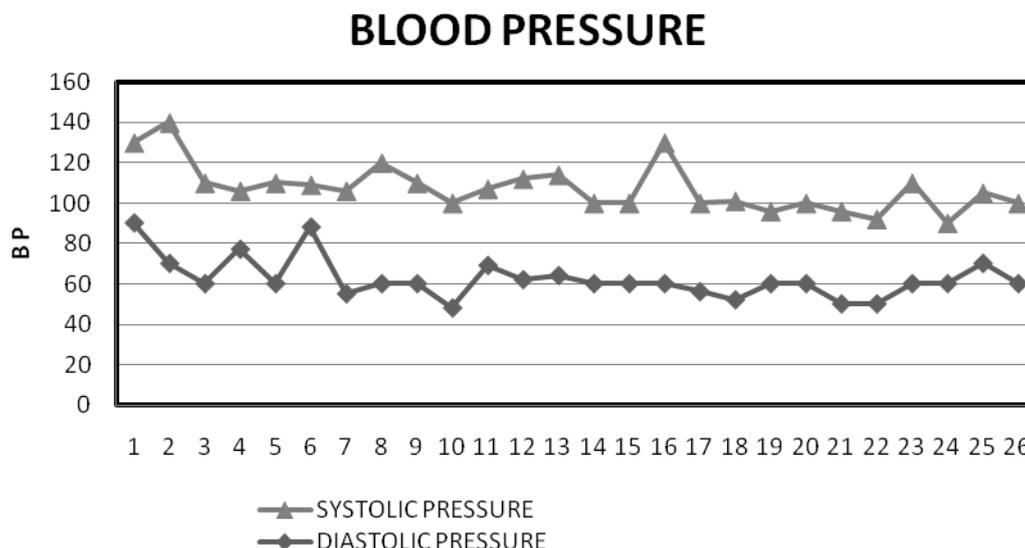
After 1 hr patient became unconscious and not responding to deep painful stimuli, Glasgow Coma Scale – 3. Patient was intubated and mechanically ventilated. Her vitals were stable. Her blood picture revealed Hemoglobin 10.8gm/dl TLC 15,200cells/mm³, platelets normal, blood sugar 60mg/dl, blood urea 36mg/dl and serum creatinine 0.9mg/dl, coagulation profile, ECG and ABG were within normal limits.

She regained consciousness on 4th day and there was progressive improvement in muscle power. She was weaned of ventilator and her further course in the hospital was uneventful. She was discharged on the 7th day.

The same patient presented to the hospital 2weeks later with breathing difficulty. On examination her higher functions and cranial nerves are normal. Patient had dysesthetic pain in all 4 limbs (more in lower limbs than upper limbs) and proprioception is lost. There was quadriparesis (upper limb-proximal 1/5, distal 3/5 and lower limb proximal 0/5 and distal 1/5 on both the right and left side) with loss of DTR (deep tendon reflexes). Her vitals were stable and cardiovascular and respiratory systems were normal. Patient was readmitted in the RICU and was intubated and mechanically ventilated. Her total blood count and blood biochemistry, CT brain and MRI were normal.

Her management consisted of Nutritional support (TPN), endotracheal tube care, Bowel and bladder care, Physiotherapy, Antibiotics, Sedation and Psychological counseling.

GRAPH 1



On 4th day, CSF analysis revealed proteins 104mg/dl, sugar 85mg/dl and 4cells/mm³ (lymphocytes). Nerve conduction study revealed demyelination with secondary axonal degeneration. Complete blood count, comprehensive serum biochemical analysis were within normal limits. The CSF analysis and nerve conduction studies confirmed our suspicion of Guillain-Barré syndrome. Plasmapheresis being the treatment of choice was carried out (a total of 7 sessions over 4 weeks). Inj. Methylprednisolone 1gm in 100ml Normal Saline IV for 3 days was given. Her supportive care was continued. After the third Plasmapheresis, the patient noticed increased strength. She was slowly weaned off ventilator. Her muscle power improved to 3/5 and she was shifted to the ward after 2 weeks and later on discharged after 2 weeks.

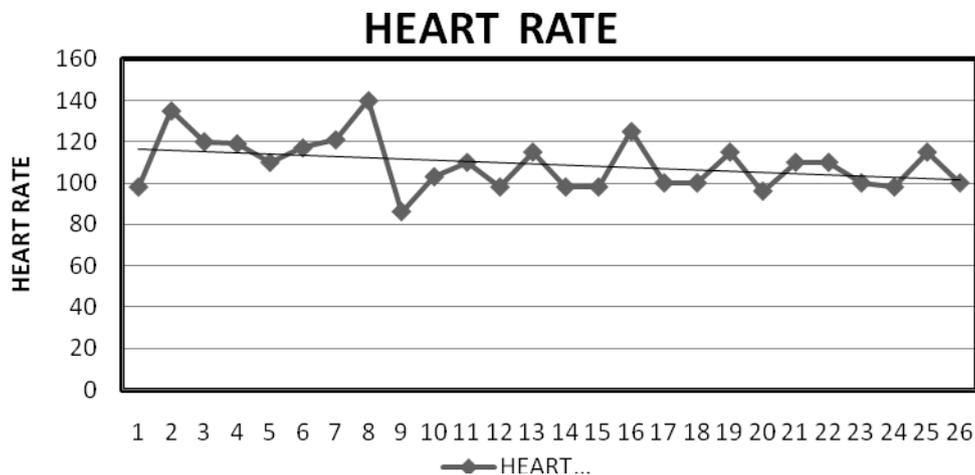
Graph 1 and 2 shows, the blood pressure and heart rate values in the 4 weeks of her hospital stay. There has been change in hemodynamic parameters with change in position during care of the patient reflecting the autonomic neuropathy associated with Guillain Barre Syndrome.

DISCUSSION

Common krait (*Bungarus Caerulus*) is a neurotoxic snake which is a common cause of envenomation in farmlands. The untreated mortality rate is 70-80% in India [1]. The common cause of death is respiratory paralysis due to presynaptic and postsynaptic binding of alpha-bungarotoxin and beta -bungarotoxin respectively [2]. In this patient muscle weakness recurred after 2 weeks after snake bite with neither evidence of cerebral oedema or hemorrhage in CT brain nor any other initiating causes of Guillain -Barre syndrome such as viral infections , causing respiratory tract infections or diarrhea , immunization or surgery [3].

The cytobiological dissociation on CSF analysis [4] and the demyelination with axonal degeneration pattern on nerve conduction studies made us think inlines of Guillain Barre Syndrome. The other causes for these symptoms could be critical illness neuropathy [5], but absence of sepsis, elevated CSF protein content and remarkable response to plasmapheresis are few points against this. Patient also had autonomic dysfunction as evidenced by alterations in heart rate and blood pressure on change of positioning during care of patient which further supports GBS.

GRAPH 2



As patient had no antecedent viral infections causing episodes of diarrhea or surgeries or any other previous similar episodes we came to a suspicion snake bite or ASV /TT could be a cause of GBS. So far there are been only 2 case reports of GBS following snake bite in literature. In the case reported by Chuang *et al* [6], the patient needed ventilatory support and regained consciousness after 8 days. At 3 weeks after admission, patient was weaned and total of 5 sessions of plasmapheresis were needed. In the second case reported by Abhishek Srivastava *et al*, patient was bitten by snake 6 weeks back, no Ventilatory support was needed and number of Plasmapheresis were not mentioned [7]. 2 cases were reported following vaccination. In 1987,

Newton.N et al [8], reported a case of Guillain-Barré syndrome after vaccination with purified tetanus toxoid. Similar case was reported by Bakshi R et al [9] after combined tetanus-diphtheria toxoid vaccination.

CONCLUSION

In our case , the clinical, biochemical and nerve conduction studies were all suggestive of GBS. There were no antecedent factors other than snake bite , administration of ASV or TT. Also taking into the reference of 4 cases reported in literature, we conclude it as GBS secondary to above factors.

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