GUILLAIN-BARRE SYNDROME AN UNUSUAL COMPLICATION AFTER SNAKE BITE, IN A CHILD.
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Abstract: 11 years old girl had a history of snake bite (krait) at her home, within few hours taken to government hospital and had received antivenin venom on the day of the bite. She had clinical, biochemical, and electrophysiological features of Guillain-Barre syndrome (GBS), with motor and sensory neuropathy. Though some cases of GBS following snake bite have been reported in adults, such presentation is rare in children. This case of GBS following snake bite in a child is reported for its rarity.

Keyword: GUILLAIN-BARRE SYNDROME, SNAKE BITE, NEUROPATHY, ANTI-SNAKEVENOM.

INTRODUCTION:
Guillain-Barré syndrome (GBS) is an acute, frequently severe, and fulminant polyradiculoneuropathy that is autoimmune in nature (1). Guillain-Barré syndrome is preceded by various antecedent events. GBS can occur after viral or bacterial infections, in autoimmune diseases, after administration of certain drugs and vaccines (vaccines against rabies, influenza, and oral poliomyelitis) (2), following surgery or organ transplantation, or following snake bite.(3). In this article, we present one such case of GBS following snake bite in a child and discuss the possible mechanism of immuno pathogenesis of GBS after snake bite.

THE GBS CHILD
Case Summary:
11 year old girl child had a history of snake bite (krait) at her home in right middle finger on 17/2/12 at 12:15 am. She was taken for some native treatment and later within few hours taken to nearby government hospital. Child was received in an irritable state with bite mark in right middle finger with cellulitis. Her 20 min whole blood clotting time was normal. Child initially had bilateral ptosis with slurring of speech, was irritable with increasing respiratory distress. Child was intubated and ventilated. Anti-snake venom (20 vials) was completed with intermittent cessation, due to anaphylactic reactions;

followed by injection atropine and injection neostigmine. Slowly child slipped into a state of complete neuroparalysis within 6 hours of admission. On day 3 child regained consciousness in the form of response to oral commands. On 20/2/12 she was with weak spontaneous respiratory efforts. Other blood & urine investigations were normal. On day 6 the child continued to be in mechanical ventilation and referred here to our hospital for further management. At our hospital intensive care unit, child was put on ventilator with minimal settings. On examination airway was intubated with poor spontaneous efforts, heart rate of 110/min, normal pulse volume, Blood-pressure of 100/70 mm hg. On examining central nervous system child was conscious, obeying commands, cranial nerves were normal, bilateral pupils were equal and reacting to light. There was no ptosis, able to close both eyes tightly. On examining motor system , bulk was normal , tone decreased in all 4 limbs , power 2/5 in all 4 limbs , DTR absent , bilateral plantar no response , no meningeal signs.

ELICITATION OF PLANTAR RESPONSE
Child was extubated at the end of second week. After 3 weeks proximal power in both upper and lower limbs improved to 3/5; however distal weakness persisted and bilateral plantar no response. By fifth week in addition child had hyperesthesia in the soles and superficial sensations were normal. Joint position sense, vibration was lost in toes. Child was able to walk with support, bilateral foot drop was present By sixth week (on discharge) paresthesia improved. In upper limb, proximal power was 4/5 and distal 3/5.In lower limb, proximal power was 4/5 and distal power was 2/5, with plantar no response. Superficial sensations were present. Joint position sense, vibration were lost in toes.

TONE AND POWER IMPROVED
ON DISCHARGE

The problems in this child were Snake bite, bilateral ptosis, respiratory muscle weakness, anaphylactic reactions to anti-snake venom, flaccid quadriparesis (distal>proximal), areflexia, plantar– no response, with sensory involvement (deep sensations). RNS showed no decremental response. NCS showed axonal motor sensory neuropathy, CSF protein levels were increased, serum CPK was normal, CT brain & MRI brain and cervical spine (entire spine) was normal. So, this child was diagnosed as Acute motor sensory axonal neuropathy—GBS variant, as the child had persistent flaccid weakness (L>U), (distal>proximal), areflexia, absent plantar, NCS—axonal motor sensory neuropathy, CSF– elevated proteins. Asbury Diagnostic Criteria for Typical Guillain-Barré Syndrome
Features required for diagnosis
1. Progressive weakness in both arms and legs
2. Areflexia
Features strongly supporting diagnosis
- a. Progression of symptoms over days, up to four weeks
- b. Relative symmetry of symptoms
- c. Mild sensory symptoms or signs
- d. Cranial nerve involvement, especially bilateral weakness of facial muscles
- e. Recovery beginning two to four weeks after progression ceases
- f. Autonomic dysfunction
- g. Absence of fever at onset
- h. High concentration of protein in cerebrospinal fluid, with fewer than 10 cells per cubic millimeter
- i. Typical electrodiagnostic features

Features excluding diagnosis
- a. Diagnosis of botulism, myasthenia, poliomyelitis, or toxic neuropathy
- b. Abnormal porphyrin metabolism
- c. Recent diphtheria
- d. Purely sensory syndrome, without weakness Based on this criteria and clinical picture the child was diagnosed as GBS. This case of GBS following snake bite in a child is reported for its rarity.

DISCUSSION:
The features of GBS in our patient could be attributed to the snake bite or the administration of anti-snake venom. One case of GBS following snake bite has been reported in the literature. (3). One case each has been reported after tetanus toxoid administration (4) and after administration of combined diphtheria and tetanus toxoid. (5). The neuromuscular complications of snake bite include neuromuscular junction abnormality, which can be caused by elapid and some sea snake venom, and myotoxicity, which can be caused by elapid as well as viper venom. Chuang et al. (3) described a patient who developed axonal GBS following the bite of a Formosan krait. The patient presented with symmetric paresis and sensory signs in the upper and lower limbs, autonomic dysfunction, facial nerve involvement, and mild elevation of cerebrospinal fluid (CSF) protein at about 4 weeks after the bite, but had good functional recovery. Electrodiagnostic studies revealed profound sensory and motor polyneuropathy. Repeat electrophysiologic examination confirmed nerve regeneration. One case of axonal sensory motor neuropathy following snake bite has been reported from India (6) with severe bilateral weakness of cranial nerves but with autonomic dysfunction. CSF study, even at 2 weeks after the illness, was normal and favored critical illness neuropathy rather than GBS. (7).

Newton and Janati (4) reported a case of GBS that developed after the injection of pure tetanus toxoid. They demonstrated a hyper-sensitivity lymphoblastic transformation occurring in response to purified tetanus toxoid antigen; also, typing for disease-associated antigens was homozogous for HLA-B8. The National Immunization Program, Centers for Disease Control and Prevention, USA, (8) had conducted a study based on previous active surveillance epidemiological studies of GBS and vaccination history and reported a background rate of 0.3 cases of GBS per million person-weeks. Although there is no direct evidence to prove the correlation between snake bite and GBS, the child had acquired no antecedent viral flulike illness or infection, nor had she required surgery, and the snake envenomization was the only preceding condition known. Bungarotoxin and/or antivenom therapy, however, may have been contributory in the pathogenesis of GBS generation in this child. Noterman and associates (8) reported some patients suspected of having botulism who were later diagnosed as having GBS. They postulated that the anti-GTlb antibody accounts for a central element of GBS development. Curiously, botulism causes a clinical syndrome very similar to the Miller-Fisher syndrome-GBS variant, and it was explained that the neurotoxin and anti-GQlb antibody bind to the same GQlb (or GQlb crossreactive) receptor observed in some patients so far reported. (9). Since bungarotoxin and botulium are biologically similar, a host immune response to the neurotoxin in venom, followed by autoimmunity and/or antivenom-venom immuno-complex binding to gangliosides, indicates the plausible hypothesis. However, there is also another possibility that heterogeneous antivenom serum itself is as likely to trigger GBS as the American swine influenza vaccine (10) was.
REFERENCES:
1. Harrison’s principles of internal medicine. 18th edition