Case Report

A rare case of Guillain–Barre syndrome after snakebite in young male and review of literature

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Abstract

Guillain–Barre syndrome (GBS) is an autoimmune disorder affecting the peripheral nervous system, often triggered by infections. This important medical emergency can also have noninfectious causes, such as trauma, surgery, medication (including vaccinations), and other systemic disorders. Here, we present a rare case of GBS following a snakebite. A 19-year-old man presented to the emergency department with quadriparesis and areflexia after showing initial recovery from a snakebite. Nerve conduction studies revealed motor axonal polyneuropathy. He was treated with intravenous immunoglobulin, which improved his clinical condition. This case report highlights a rare cause of GBS and emphasizes that timely recognition and treatment can significantly reduce morbidity and mortality. Additionally, we reviewed past literature of this rare association.

Keywords: Emergency medicine, Guillain–Barre syndrome (GBS), snakebite

Introduction

Guillain–Barre syndrome (GBS) is an acute-onset acquired autoimmune disease affecting the peripheral nervous system (PNS). The annual incidence of GBS ranges from 0.5 to 2 cases per 100,000 individuals. It has variants like acute inflammatory demyelinating polyneuropathy, acute motor axonal neuropathy (AMAN), and Miller–Fisher syndrome.[1] Approximately, 70% of GBS cases follow infection, vaccination, or surgery within 2–4 weeks.[2] However, only a few cases of GBS following snakebite have been reported in the literature. Here, we report one such case in a young male patient.

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Case History

A 19-year-old male patient presented to the emergency department (ED) with a history of sudden onset quadriparesis since one day. Initially, he had limb weakness that progressed in 48 h, in the form of being unable to stand and unable to lift the upper limb.

He had a history of hospitalization at our institute 18 days prior for a snakebite while working in the field at 4:00 am. Initially, he visited a primary health center and received a tetanus toxoid injection (0.5 ml IM), and he was then referred to our ED. On arrival, his Glasgow coma score (GCS) was E3V3M3, oxygen saturation was 74% on 4 l of oxygen, respiratory rate (RR) was 12/min, bilateral pupils were dilated and reactive to light, heart rate (HR) was 80/min, blood pressure (BP) was 110/80 mmHg, and random blood sugar (RBS) was 116 mg/dl. Central nervous system examination revealed low tone in all limbs and absent deep tendon reflexes. His whole blood clotting time was less

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than 20 min every time when performed sequentially multiple times. He was clinically diagnosed as a case of neurotoxic snakebite. Along with antisnake venom, intravenous atropine and neostigmine were administered. He was intubated and started on the volume control/assist control mode of mechanical ventilation. On the second day, his GCS improved to E3VetM5. He was put on synchronized intermittent mandatory ventilation mode with an FIO_2 of 70%. He improved clinically and was extubated on day four of hospitalization. On day six, he was discharged with stable vitals and in an asymptomatic state with no residual weakness.

However, 12 days after discharge, he returned to the ED with quadriparesis. His vitals were BP 114/80 mmHg, HR 110/min, RR 16/min, and oxygen saturation 95% on room air, GCS 15/15, pupils normal and equal size and reactive to light bilaterally, and RBS 115 mg/dl. There was no history of diplopia, dysarthria, breathing difficulty, seizure episodes, altered mental status, or bowel and bladder involvement. There was no history of fever, headache, burning micturition, or loose stool, just prior to the episode of quadriparesis. He had no prior medical and drug use history. Furthermore, he had no history of addiction or substance abuse and no history of new insect bites. The general physical examination was unremarkable. During the neurological examination, decreased muscle power was observed in all limbs as 1/5 (which was previously 5/5 as he was working normally in daily life) and deep tendon reflexes were absent. Fundus examination revealed a normal disc. Cardiac, respiratory, and abdominal examinations were also within normal limits.

Blood gas analysis, complete blood count, liver and renal function tests, and electrolyte and procalcitonin levels were normal. Urine toxicology test was also unremarkable. Noncontrast computed tomography head [Figure 1] and MRI brain [Figure 2] revealed no abnormality, including hemorrhage or acute ischemia. Cerebrospinal fluid analysis was within normal limits. The nerve conduction study (NCS) indicated axonal polyneuropathy affecting motor fibers, suggesting GBS, particularly the AMAN-type variant [Tables 1a and 1b].

Subsequently, the patient was administered intravenous immunoglobulin (IVIG) 2 g/kg, for the next five days. The patient started improving with the ongoing IVIG therapy and in-hospital rehabilitation. He started moving the toes of the lower limb followed by walking with support over the next 20 days. Eventually, on day 22, he was discharged with an upper limb power of 4/5 and lower limb power of 3/5. At the two-month follow-up, he showed gradual improvement and resumed normal daily activities.

Table 1a: Amplitudes in the motor nerve conduction study (NCS) were severely reduced, and the velocity of impulse conduction was normal or mildly slowed in motor NCS. F wave latencies are nonrecordable

Motor nerve conduction study					
Site	**Latency (ms)**	**Duration (ms)**	**Amplitude (mV)**	**Area (mV ms)**	**NCV (m/s)**
Left median (wrist)	4.0	5.2	2.2	7.1	68.7
Left median (elbow)	7.3	5.5	1.3	4.5	68.7
Right median (wrist)	3.3	5.0	1.5	4.1	55.2
Right median (elbow)	7.5	5.5	1.5	4.9	55.2
Left ulnar (wrist)	2.2	5.3	1.2	0.8	55.6
Left ulnar (elbow)	6.7	7.7	1.2	4.1	55.6
Right ulnar (wrist)	2.5	4.5	1.0	1.2	66.1
Right ulnar (elbow)	6.3	5.9	0.9	2.1	66.1
Left peroneal (ankle)	5.7	7.9	0.3	1.2	20.5
Left peroneal (head of fibula)	22.8	5.3	0.2	0.4	20.5
Right peroneal (ankle)	6.0	8.7	0.8	2.7	23.7
Right peroneal (head of fibula)	19.9	7.9	0.2	0.7	23.7
Left tibial (ankle)	4.8	8.5	2.2	8.6	23.6
Left tibial (popliteal)	19.6	10.3	0.9	4.8	23.6
Right tibial (ankle)	6.1	8.4	3.5	16.0	23.6
Right tibial (popliteal)	21.0	8.3	1.1	5.1	23.6

Table 1b: Nerve conduction appeared normal in sensory fibers

Sensory nerve conduction study					
Site	**Latency 1 (ms)**	**Latency 2 (ms)**	**Amplitude (μV)**	**Area (μV ms)**	**NCV (m/s)**
Left median (wrist)	2.0	2.6	67.7	3.2	76.5
Right median (wrist)	2.4	2.8	59.2	0.6	62.0
Left ulnar (wrist)	2.0	2.6	25.7	2.5	63.7
Right ulnar (wrist)	2.4	2.8	27.8	0.5	54.6
Left sural	3.0	3.6	14.7	1.3	46.7
Right sural	2.9	3.6	16.5	0.9	47.9

Discussion

GBS is an immune-mediated polyradiculoneuropathy that affects the PNS, and patients usually present with bilaterally symmetrical ascending flaccid paralysis. While infectious illness, vaccination, and surgery are commonly reported preceding events, cases in the literature in which GBS has been associated with snakebite remain few.[3-8]

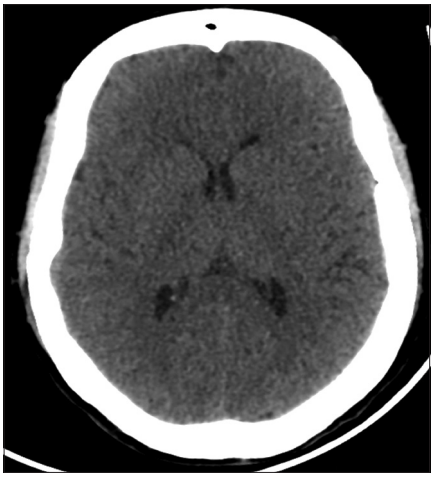

Figure 1: Non-contrast CT head with no abnormality

Cobra, krait, and sea snakes are common neurotoxic snakes in India.[8,9] Neuroparalysis resulting from neurotoxic snakes stems from the blockade of neuromuscular receptors, and the mechanism varies depending on the snake species involved. For example, krait bites affect pre- and postsynaptic neuromuscular receptors, whereas cobra bites primarily target the blockade of postsynaptic receptors. The bite of the Indian cobra (*Naja naja*) typically results in tender local swelling, blistering, and necrosis. Krait bites often lack skin manifestations. The common krait is primarily nocturnal, and its bite is usually painless.[10] Paralysis of proximal limb muscles occurs before distal ones. Occasionally, patients may present with complete quadriplegia and a locked-in syndrome.[8]

GBS pathogenesis is based on molecular mimicry, where immune responses to various pathogens cross-react with ganglioside components on peripheral nerves due to shared epitopes. Epitopes on the Schwann-cell surface membrane are associated with acute inflammatory demyelinating neuropathy, whereas epitomes on the axonal membrane are linked to acute axonal forms of GBS.[2]

To the best of our knowledge, all the data from previous reports are summarized in Table 2.

In this study, the patient fully recovered after the snakebite and was pursuing all his daily activities normally. However, after 18 days of

Table 2: Summary of previous literature

Author	Clinical features	Snake	Received 1) ASV dose 2) TT dose	Presentation time since 1) Bite 2) ASV	NCS	Treatment
Chuang *et al.* (Taiwan, 1996)[3]	Quadriparesis and difficulty in breathing and speech	Formosan krait (*Bungarus multicinctus*)	1) Received 2) Not received	1) 4 weeks 2) Beyond 25 days	Sensorimotor axonal-type polyneuropathy	Five sessions of plasmapheresis and methylprednisolone improved, the patient's clinical condition
Srivastava *et al.* (India, 2010)[4]	Quadriparesis, tingling, and numbness in all four extremities	Not known	1) Received 2) Received	1) Beyond 5 weeks 2) Beyond 5 weeks	Motor and sensory polyneuropathy—primarily demyelination with secondary axonal degeneration	Plasmapheresis was performed and the patient's condition improved at discharge
Neil *et al.* (France, 2011)[5]	Quadriparesis and paresthesia in all four extremities and gait ataxia	Viper (*Vipera aspis*)	1) Not received	1) Beyond 10–12 days	Sensorimotor polyneuropathy with conduction block	Treatment not known Study was conducted to establish the relationship between snake venom and GBS
Neto *et al.* (Brazil, 2014)[6]	Quadriparesis	Rattlesnake (*Crotalus* sp.)	1) Received 2) Received	1) Beyond 2 weeks 2) Beyond 2 weeks	Sensorimotor axonal polyradiculoneuropathy	IVIG was administered and the patient's clinical condition improved
Hameed *et al.* Pakistan, 2019)[7]	Quadriparesis and facial weakness	Sea snake (*Hydrophis platurus*)	1) Not received	1) Beyond 6 weeks	Acute motor axonal polyneuropathy	Plasmapheresis
Changadiya *et al.* (India, 2020) (2 cases)[8]	First—Quadriparesis and bulbar weakness Second—Bulbar weakness, quadriparesis, and autonomic dysfunction	First Common krait Second krait	First: 1) Received 2) Not known Second: 1) Received 2) Not known	First: 1) 15 days 2) 15 days Second: 1) 10 days 2) 10 days	First: Demyelinating motor polyneuropathy Second: Axonal motor polyneuropathy	First IVIG Second IVIG

ASV=Antisnake venom

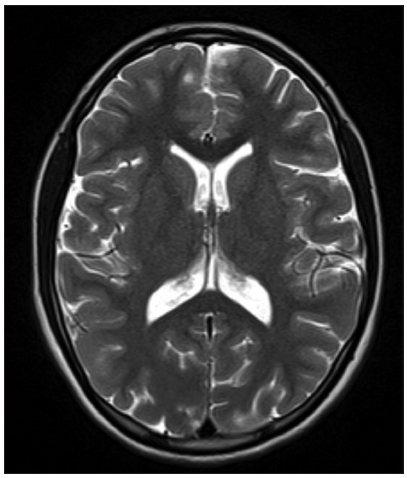

Figure 2: T2-weighted MRI brain with no abnormality

snakebite, he had unexplained limb weakness, which was eventually diagnosed as GBS (AMAN variant) by NCS. Therefore, the patient was started on IVIG therapy and showed eventual recovery.

Key messages

The possible association between GBS and snakebite should always be considered. Careful observation of limb weakness or other neurological symptoms for a few weeks after a snakebite is imperative. Early detection and intervention can be highly beneficial in such cases.

List of abbreviations

Abbreviation	Definition
GBS	Guillain–Barre syndrome
IVIG	Intravenous immunoglobulin
PNS	Peripheral nervous system
AIDP	Acute inflammatory demyelinating polyneuropathy
AMAN	Acute motor axonal neuropathy
MFS	Miller–Fisher syndrome
GCS	Glasgow Coma Scale
RR	Respiratory rate
HR	Heart rate
BP	Blood pressure
RBS	Random blood sugar
ASV	Antisnake venom
ED	Emergency department
NCCT	Noncontrast computed tomography
MRI	Magnetic resonance imaging
NCS	Nerve conduction study

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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