Traumatic Brain Injury and Guillain-Barré Syndrome: Tale of an Illicit Affair—Case Report and Brief Review of Literature

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Abstract

Guillain-Barré syndrome (GBS) is a common entity in neurology clinics. A variety of etiologies have been implicated in the presentation of GBS. Although rarely reported, traumatic brain injury (TBI) has also been reported to cause GBS. In this article, we report a similar case of GBS that occurred following TBI and the patient presented with acute flaccid paraparesis with intact strength in upper limbs. Paraparesis progressed to quadriparesis simulating a case of spinal injury, without any correlating imaging findings. Nerve conduction study findings, cerebrospinal fluid studies, and clinical examination led to the diagnosis of post-TBI GBS. A review of similar cases reported in literature is also attached. High index of suspicion should be maintained for GBS in all cases of imaging-negative post-TBI limb weakness which may simulate acute spinal injury.

Keywords
- Guillain-Barré syndrome
- head injury
- traumatic brain injury
- spinal cord injury
- paraplegia

Introduction

Guillain-Barré syndrome (GBS) is an immune-mediated acute inflammatory polyradiculoneuropathy characterized by flaccid paralysis and acute demyelinating changes in the peripheral nervous system.[1,2] Diagnosis is made by clinical features, cerebrospinal fluid (CSF) study, and electrophysiological findings.[3,4] A variety of causes may lead to GBS including but not limited to respiratory or gastrointestinal infection, vaccine-induced, drug-induced, postsurgery, and autoimmune disorders.[5] One rare reported cause is traumatic brain injury (TBI) and although a few hypotheses have been proposed, the exact pathophysiology of TBI causing GBS remains ambiguous and debatable. Here, we report a rare case of progressive quadriparesis starting with sudden-onset flaccid paralysis of both lower limbs immediately following a road traffic accident (RTA). The patient was clinically confirmed as having GBS, with supportive evidence from nerve conduction studies (NCSs) and CSF studies. We have also compiled highlights of other cases of post-TBI GBS reported in literature.

Case Report

A 29-year-old previously healthy man presented with sudden-onset flaccid paraparesis of the both lower limbs immediately following an RTA (bike driver, not wearing helmet). Examination revealed profound weakness of both lower limbs (►**Table 1**). Power of upper limbs was intact. Deep tendon reflexes were absent in both lower limbs. Plantar

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Table 1 Power (by Medical Research Council scale) of different muscle groups of all four limbs

	At admission		At discharge		At 1 month after discharge		At 3 months after discharge	
	Right	Left	Right	Left	Right	Left	Right	Left
Upper extremity Shoulder (adductors, abductors, flexors, extensors, lateral rotators, medial rotators)	5/5	5/5	5/5	5/5	5/5	5/5	5/5	5/5
Elbow (flexors, extenders)	5/5	5/5	5/5	5/5	5/5	5/5	5/5	5/5
Wrist (flexors, extensors)	5/5	5/5	4+/5	4+/5	5/5	5/5	5/5	5/5
Finger (adductors, abductors)	5/5	5/5	4/5	4/5	5/5	5/5	5/5	5/5
Lower extremity Hip (flexors, extensors, adductors, abductors)	1/5	3/5	0/5	2/5	4+/5	5/5	4+/5	5/5
Knee (flexors, extensors)	1/5	2/5	0/5	2/5	4+/5	5/5	4+/5	5/5
Plantar (flexion, dorsiflexion)	0/5	0/5	0/5	0/5	4/5	4/5	4+/5	4+/5
Small muscles of foot	0/5	0/5	0/5	0/5	3/5	3/5	4/5	4/5

reflexes were equivocal and abdominal reflex intact. Foley's catheter was applied for incontinent bladder. There was no backache. Rest of the neurological examination was unremarkable.

The patient was started on high-dose steroids to treat apparent spinal cord injury. Magnetic resonance imaging (MRI) of the spine revealed normal study. Computed tomography scan of brain and skull revealed a small hemorrhagic contusion in the left frontotemporal region, which did not warrant surgery. The patient was treated on the lines of spinal cord injury, with no improvement. The MRI study of the spine was unremarkable and did not correlate with the clinical findings. This diagnostic dilemma led to delay; weakness further progressed, and the patient developed quadriparesis over the next 3 weeks.

NCS of all four limbs was conducted 24 days after RTA. Motor nerve conduction study showed normal distal latencies, normal compound muscle action potential (CMAP) amplitudes, and normal conduction velocities of bilateral ulnar nerve. Distal latencies were significantly prolonged for median nerve; conduction velocities of the median nerve and amplitudes were however reduced. CMAPs were bilaterally absent for peroneal nerves. Bilateral tibial distal latency was prolonged and conduction velocities of tibial nerve amplitudes were reduced. Sensory nerve conduction study of the bilateral ulnar, median, and sural sensory nerve action potential was present with normal latencies and normal amplitudes. "F" wave of bilateral upper limbs were impersistent for upper limbs bilaterally and absent in lower limbs. "H" reflex was absent bilaterally. These findings suggested primary demyelination secondary to axonal radiculopathy involving the lower limbs more than the upper limbs. Serum ganglioside antibody tests were inconclusive. CSF studies showed mildly elevated protein levels, without rise in cell count, consistent with albuminocytological dissociation in inflammatory demyelinating polyradiculoneuropathy. Acute inflammatory demyelinating polyneuropathy, a variant of GBS, was considered based on NCS, clinical examination, and CSF studies. Patient denied treatment with intravenous immunoglobulins. At 1-month follow-up patient had improved and could stand with support. At 3 months the patient could walk with support.

Discussion

GBS is an immune-mediated acute inflammatory polyradiculoneuropathy characterized by flaccid paralysis and acute demyelinating changes in the peripheral nervous system. Majority of cases are preceded by upper respiratory infection or diarrhea, with the most frequently identified infectious agent being *Campylobacter jejuni*. Surgery, immunization, autoimmune inflammatory disorders, and drug adverse effect are other causes of GBS[6–9]; however, GBS following TBI has only sparsely been described in literature (►**Table 2**).

Following TBI, the mechanism of GBS is thought to be trauma-related disruption of the cellular immunity. Levels of B cells and immunoglobulins forming humoral immunity increase.[10] Allegedly, trauma often leads to transient immunosuppression thus altering the immune tolerance of the body, leading to clinical or subclinical exogenous infection, which could elicit cross-reactive antibodies. Following TBI and violation of blood–brain barrier, molecules such as myelin basic protein may cross into the central nervous system and be perceived as antigenic material, thus activating the immune-mediated polyradiculoneuropathy in GBS.[5,8,10] These findings have also been supported by

Table 2 Compilation of posttraumatic brain injury Guillain-Barre syndrome cases reported in the world literature

Study	Age (y)	Clinical findings	Onset	Findings	CSF study	Antiganglioside antibodies
Duncan and Kennedy[8] 1987	61	Quadriparesis with dysphagia, respiratory distress, left ptosis, LMN facial palsy	9 d	NCS: demyelinating motor peripheral neuropathy	NA	NA
De Freitas et al[9] 1997	29	Flaccid areflexic quadriparesis, facial diplegia. Respiratory failure	3 d	NCS: increased latency, decreased amplitude, no F-wave	NA	NA
Lin et al[16] 2006	22	Progressive asymmetric quadriparesis	7 d	NCS: no F-wave	Protein 0.5 mg/dL	NA
Rivas et al[17] 2008	55	Progressive quadriparesis	1 wk	NCS: inexcitability of all nerves with active denervation	Elevated protein levels	Absent
Tan et al[18] 2010	44	Flaccid areflexic quadriparesis, numbness, paresthesia	1 wk	NCS: absent sensory/motor responses, no blink reflexes	Protein 18.2 mg/dL, ACD	Not done
Battaglia et al[19] 2013	73	Quadriparesis, swallowing difficulty, bilateral facial palsy	14 d	EMG: demyelinating neuropathy	ACD	Absent
Carr et al[5] 2015	58	Progressive quadriparesis, bilateral facial palsy	24 d	EMG: prolonged F-wave latencies, evidence of conduction block in both upper extremities and lower extremities	ACD	GD1a, GD1b
Unal et al[20] 2016	63	Quadriparesis, facial paralysis, speech impairment, swallowing difficulty	17 d	EMG: sensory motor demyelinating polyneuropathy	Elevated protein level	NA
Jia et al[21] 2017	41	Sudden quadriplegia with areflexia, respiratory failure	2 wk	NCS: CMAP of right median nerve and sural nerve not elicited, CMAP of the common peroneal nerve on both sides were significantly decreased, F-waves not evoked in either upper limbs or lower limbs	Protein 18 mg/dL	Absent
Li et al[10] 2017	48	Quadriplegia with cranial nerves II, III, IV, VI involvement, respiratory muscle involvement	10 d	NCS: reduced CMAP and reduced F-wave persistence	Protein 64 mg/dL ACD	GM1
Harsh et al (current study)	29	Quadriparesis	Immediate	NCS: primary demyelination secondary axonal polyradiculopathy	Protein 50.6 mg/dL	Absent

Abbreviations: ACD, albuminocytologic dissociation; CMAP, compound muscle action potential; CSF, cerebrospinal fluid; EMG, electromyography; LMN, lower motor neuron; NA, not available; NCS, nerve conduction study.

some recent studies demonstrating that the levels of antibodies, such as antimyelin antibodies or antiganglioside antibodies, are increased following TBI.[10] There is targeting of ganglioside antibodies of axolemma of the ventral and dorsal roots, leading to axonal damage, thus resulting in reduced neuronal transmission.[11] These deficits are identified as prolonged distal latencies and reduced amplitudes in NCS studies as was seen in our case. In CSF study, the classical finding in GBS is albumin-cytological dissociation, there is increased protein level and elevated CSF/serum albumin ratio during the second week.[12]

Owing to limited reports on post-TBI GBS, the role of immunological treatment has not yet been established. An empiric course of intravenous immunoglobulins or plasma exchange might be valuable as it has shown to improve progression of the symptoms.[13,14] Few cases have shown partial clinical improvement, while others did not, when treated with high doses of intravenous methylprednisolone.[1,15] Further research on immunological treatment of posttraumatic GBS is thus required.

Conclusion

Unlike in other reports when GBS followed days after trauma, sudden-onset paraparesis immediately following injury in this patient towed our attention toward compressive myelopathy; however, absence of backache and a negative MRI scan suggested otherwise. A "spinal concussion" may also lead to limb weakness, however, patients with spinal concussion improve over time, unlike our patient who deteriorated over the next 2 weeks and paraparesis progressed to quadriparesis. This is more likely a presentation of GBS.

In clinical practice, if we come across a case with sudden onset of paralysis of bilateral lower limbs following trauma, which cannot be explained by routine imaging and laboratory findings, GBS may be considered in the differential diagnosis. Early neurological examination, NCS, CSF studies, and tests for antiganglioside antibodies may further help in supporting the diagnosis of GBS in the case of quadriparesis following TBI.

Conflict of Interest
None declared.

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