



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

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Abstract

Keywords

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

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.

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 imper-sistent 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 GBS⁶⁻⁹; 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 | Quadripareis 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 quadripareis, facial diplegia. Respiratory failure | 3 d | NCS: increased latency, decreased amplitude, no F-wave | NA | NA |
| Lin et al ¹⁶ 2006 | 22 | Progressive asymmetric quadripareis | 7 d | NCS: no F-wave | Protein 0.5 mg/dL | NA |
| Rivas et al ¹⁷ 2008 | 55 | Progressive quadripareis | 1 wk | NCS: inexcitability of all nerves with active denervation | Elevated protein levels | Absent |
| Tan et al ¹⁸ 2010 | 44 | Flaccid areflexic quadripareis, 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 | Quadripareis, swallowing difficulty, bilateral facial palsy | 14 d | EMG: demyelinating neuropathy | ACD | Absent |
| Carr et al ⁵ 2015 | 58 | Progressive quadripareis, 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 | Quadripareis, 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 | Quadripareis | 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 antemyelin 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 quadriplegia. 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 quadriplegia following TBI.

Conflict of Interest

None declared.

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