

Posttraumatic Guillain-Barré Syndrome Immediately Following a Traffic Accident

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Guillain-Barré syndrome (GBS) is an inflammatory demyelinating polyneuropathy characterized by areflexic paralysis. Most cases of GBS are preceded by an infection, however, posttraumatic GBS has also recently been reported. We report a case of posttraumatic GBS immediately following a traffic accident. We think this case is of clinical significance for practitioners because of the rare cause of a sudden flaccid paralysis following trauma.

Key Words: Posttraumatic GBS, Guillain-Barré syndrome, Trauma

INTRODUCTION

Guillain-Barré syndrome (GBS) is an inflammatory demyelinating polyneuropathy characterized by areflexic paralysis that is not commonly encountered in the neurosurgical field^{5,19}. Most cases of GBS are preceded by an infection, such as an upper respiratory infection or enteritis. However, posttraumatic GBS has also recently been reported^{3,4,9,16}.

Here, we report a case of abrupt paralysis of the lower extremities immediately following a traffic accident. The patient was confirmed as having GBS with an electromyogram/nerve conduction study (EMG/NCS) and cerebrospinal fluid (CSF) assessment, and was successfully treated with administration of intravenous immunoglobulin (IVIG).

To our knowledge, this is a unique case of posttraumatic GBS in which the symptom onset was immediately after the trauma. We think that this case is of clinical significance for practitioners because of the rare cause of a sudden flaccid paralysis following trauma.

CASE REPORT

A 36-year-old previously healthy man presented with abrupt flaccid paralysis of the lower limbs immediately following a traffic accident. Examination showed profound weakness of both legs (grade 1) combined with severe lancinating paresthesia at the posterior surface of the right leg. His left leg weakness spontaneously improved to grade IV within a few hours after injury. Deep tendon reflexes were absent. Bladder dysfunction was noted and catheterization was required. Pathologic reflexes, such as Babinski sign and ankle clonus, were absent.

To treat apparent spinal cord injury or cauda equina syndrome, the patient was administered a high-dose steroid. However, a few days later, the patient complained of mild weakness and tremor in both upper extremities without improvement of right leg weakness.

Whole spine magnetic resonance imaging (MRI) demonstrated mild central intervertebral disc herniation at L5/S1 that was of little clinical significance (Fig. 1). Otherwise, there was no significant pathology, including compressive lesion, cord contusion, or hemorrhage. Brain MRI also showed no significant abnormality. EMG/NCS was conducted 7 days after the accident, which did not demonstrate any abnormal findings. However, 3 weeks after the accident, a follow-up EMG/NCS was conducted. In the motor NCS, terminal latency was increased and conduction velocity was decreased in both upper and lower extremities. Sensory NCS showed decreased conduction velocity in both upper and lower extremities. There was increased F-wave latency in all 4 extremities. The assessment showed signs of demyelinating sensorimotor polyneuropathy. Furthermore, CSF assessment showed a mild elevation of protein levels, consistent with inflammatory demyelinating polyneuropathy and leading to a final diagnosis of GBS. The patient was transferred to the department of neurology, where he was treated with administration of IVIG.

Three months after the accident, symptoms showed gradual improvement and the patient could walk stably with a cane. Bladder dysfunction had also improved. At the final follow-up, 5 months after the accident, the patient's condition was stable. Further EMG/NCS still showed demyelinating sensorimotor polyneuropathy without significant changes since previous assessments.

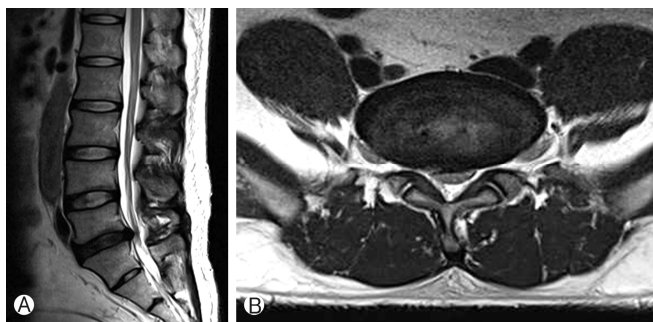


Fig. 1. Lumbar spine magnetic resonance imaging (MRI) demonstrated herniated intervertebral disc at L5/S1 level in sagittal and axial images, respectively (A and B). Muscle weakness of the lower extremities of the patient could not be explained by MRI findings.

DISCUSSION

In the present case, we report a case of posttraumatic GBS. We believe the case is of particular importance for clinicians for several reasons. First, to the best of our knowledge, this is the first reported case of GBS that developed immediately following a traffic accident. Second, such a case of sudden paralysis of the lower extremities after traffic accident without significant disc herniation or compressive lesion upon cauda equina may be difficult to diagnose, leading to confusion and embarrassment for both doctors and patients. Finally, such cases might result in disputes for insurance compensation, since the relationship between GBS and trauma has not been well established.

GBS is an inflammatory demyelinating polyradiculopathy, characterized by acute areflexic paralysis and demyelinating changes in the peripheral nervous system. The majority of cases are preceded by upper respiratory infection or diarrhea, with the most frequently identified infectious agent being *Campylobacter jejuni*, followed by cytomegalovirus^{6,8,10}. The main feature of GBS is progressive bilateral and relatively symmetric weakness of the limbs, with the weakness progressing over a period of 12 hours to 28 days before a plateau is reached¹⁵. Electrophysiologic study can confirm the presence and classification of GBS²⁰. The typical finding from a lumbar puncture is albuminocytologic dissociation²⁰.

In contrast to well-established infectious etiology, the concept of posttraumatic GBS preceded by no risk factors other than trauma has been recently introduced. There have already been several reports of posttraumatic GBS, following physical damage from traffic accidents, falling, or surgery^{1,7,11-14,17,18}. However, in all previous cases, the time from trauma to symptom onset was more than several days. The main difference of the present case from those previously reported is the immediate onset of flaccid weakness following trauma, which could lead to suspicions of mechanical compressive lesions rather than an inflammatory process.

The main pathological pathway for GBS is a triggered immune-mediated response resulting in multifocal mononuclear cell

infiltration throughout the peripheral nervous system²). In the case of posttraumatic GBS, it is postulated that the underlying mechanisms are based on a trauma-related disruption of the cellular and humoral immune system¹¹. In the present case, however, it is difficult to understand how such immune-mediated reactions happened so promptly following trauma, and the possible mechanism is yet to be explained.

Because of the rarity of posttraumatic GBS and the lack of systematic research, the role of immunological treatment has not yet been established. However, there exist some studies reporting the value of an empiric course of IVIG or plasma exchange^{7,14,17}. In the present case, the patient showed considerable recovery with IVIG without relapse for 6 months. Similar to the treatment for other cases of GBS, immunotherapy with IVIG or plasma exchange may be viable options for the management of posttraumatic GBS.

Posttraumatic GBS is a rapidly progressive disorder, resulting in severe neurological complications. Clinicians should take into account the possibility of posttraumatic GBS in cases of unexplained muscle weakness after trauma, because appropriate corresponding measures including general medical care and immunotherapy could relieve and improve the condition.

CONCLUSION

Posttraumatic GBS can occur immediately after a trauma, such as a traffic accident. This rare condition can be successfully treated with immunotherapy, and should be considered in unexplained paralytic patients without significant radiologic abnormalities following trauma.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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