Spine

SPINE Volume 40, Number 6, pp E372-E374

CASE REPORT

Guillain-Barré Syndrome After Pelvic Fracture Fixation

A Rare Cause of Postoperative Paralysis

Tariq Hendawi, MD,* and Joseph M. Zavatsky, MD†

Study Design. Case report.

Objective. To describe the important epidemiology, clinical presentation, and pathophysiology of Guillain-Barré syndrome (GBS) after orthopedic surgery in a trauma patient.

Summary of Background Data. Little in the orthopedic literature describes trauma as an etiology of GBS. We report a case of post-traumatic GBS in a 52-year-old male who developed ascending weakness after experiencing pelvic trauma that required 2 separate orthopedic procedures for pelvic stabilization after a fall from a height of 12 ft. After the index operative procedure, the patient complained of left S1 numbness. Computed tomographic scan demonstrated the pelvic screw approximating the left S1 neuroforamen and correlated with the patient's immediate postoperative symptoms. A secondary procedure to reposition the screw alleviated the patient's left S1 numbness. Two weeks postoperatively, the patient developed profound ascending lower extremity weakness. This case highlights the importance of considering all etiologies, no matter how uncommon, in the differential diagnosis of lower extremity weakness.

Methods. Case report with literature search on GBS in orthopedic trauma patient.

Results. We propose that direct neural trauma from poorly positioned hardware resulting in clinical neurological symptoms may have been the inciting event that caused GBS in this trauma patient.

Conclusion. Post-traumatic GBS is a rare, potentially life-threatening cause of weakness. Once mechanical causes are ruled out with appropriate imaging, all etiologies in the differential diagnosis must be explored. If the image findings cannot explain the clinical examination, other biologic causes of weakness, including GBS, must be explored. The causes of GBS in the postoperative trauma patient include infection, trauma, surgery, or direct neural injury.

Key words: Guillain-Barré syndrome, pelvic trauma, postoperative paralysis, weakness, paralysis, ascending weakness, sacral fracture, SI joint dissociation.

Level of Evidence: 4

Spine 2015;40:E372–E374

Guillain-Barré syndrome (GBS) is an acute, autoimmune, inflammatory, demyelinating polyneuropathy affecting the peripheral nervous system. It causes symmetric ascending flaccid paralysis and areflexia.[1] The annual incidence is 1 to 2 cases per 100,000.[2] Presentation varies from mild peripheral symptoms to fulminant GBS involving facial, bulbar, or respiratory musculature, leading to death.[3] Guillain-Barré syndrome may be preceded by infection, immunization, surgery, or trauma.[4] Cross-reactivity of neural antigens with antibodies to infections such as *Campylobacter jejuni*, *Mycoplasma pneumoniae*, *Haemophilus influenzae*, cytomegalovirus, herpes simplex, and Epstein-Barr viruses are suspected.[5,6] In addition, trauma to nervous tissue can also expose myelin to the body's immune system, resulting in cross-reactivity and demyelination caused by deposition of circulating antigen-antibody complexes in the blood vessels of peripheral nerves.

CASE REPORT

A 52-year-old male smoker sustained multiple rib and lumbar transverse process fractures, along with a zone 1 sacral fracture with bilateral sacroiliac (SI) joint dissociation after a 12-ft fall. He underwent pelvic stabilization with an SI screw that traversed both SI joints. (Figure 1A–C) Postoperatively, the patient developed numbness in the left S1 dermatome, which corresponded with hardware approaching the S1 neuroforamen. Because of his clinical symptoms, the patient was taken back to the operating room for revision SI screw fixation (Figure 2), which resolved his S1 numbness.

Approximately 2 weeks after his initial injury, the patient presented to the emergency department with complaints of

From the *Department of Orthopaedic Surgery, Ochsner Clinic Foundation, Back and Spine Center, New Orleans, LA and †Spine, Scoliosis & Deformity Institute, Florida Orthopaedic Institute, Tampa, FL.

Acknowledgment date: April 19, 2014. Acceptance date: January 8, 2015.

The manuscript submitted does not contain information about medical device(s)/drug(s).

No funds were received in support of this work.

No relevant financial activities outside the submitted work.

Address correspondence and reprint requests to Joseph M. Zavatsky, MD, Florida Orthopaedic Institute, Spine, Scoliosis & Deformity Institute, 13020 Telecom Parkway North, Tampa, FL 33637; E-mail: josephzavatsky@yahoo.com

DOI: 10.1097/BRS.0000000000000779

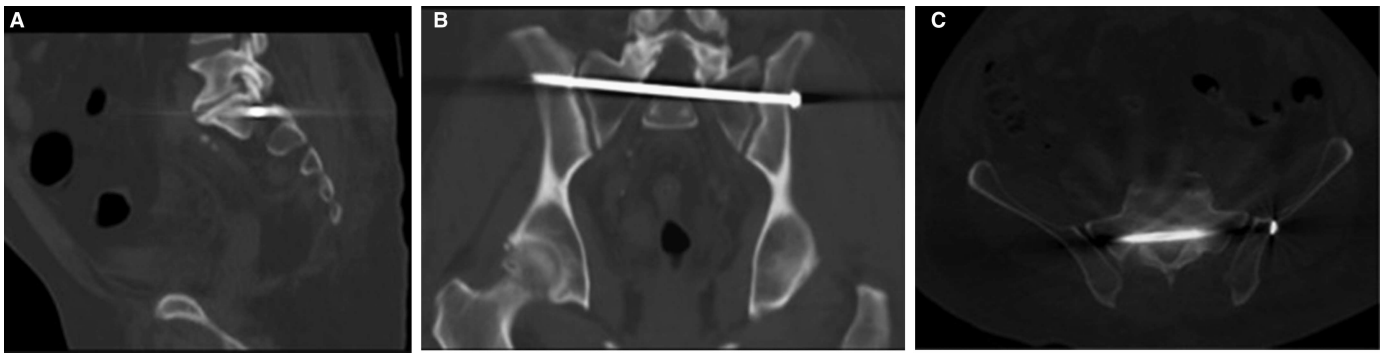

Figure 1. A–C, Computed tomographic cuts demonstrating sacroiliac screw fixation adjacent to the left S1 foramen and anterior thecal sac after initial procedure. The postoperative numbness in the left S1 distribution may represent the direct neural trauma or irritation that ultimately initiated the Guillain-Barré syndrome cascade.

profound bilateral lower extremity weakness and numbness that had progressed during a week's time. On examination, he had flaccid paralysis of his bilateral lower extremities (0/5), along with bilateral upper extremity weakness (3/5). He was areflexic in his lower extremities and hyporeflexic (+1) in the upper extremities. He denied any respiratory or swallowing difficulty. After emergency department and medicine consultations, the etiology of his new onset weakness could not be identified and orthopedics was consulted.

Further imaging showed no evidence of epidural collection, cerebrospinal fluid (CSF) leak, canal stenosis, or abnormal signal within the spinal thecal sac or nerves; computed tomographic scan showed well-aligned hardware. A spinal tap was ordered by the orthopedic resident and revealed a markedly elevated CSF protein consistent with GBS. Once the diagnosis was made, the patient was transferred to the intensive care unit and treated appropriately with intravenous immunoglobulin and steroids. Fortunately, his treatment was started before respiratory failure or intubation was required.

At last follow-up, the patient showed significant recovery in strength in both upper and lower extremities.

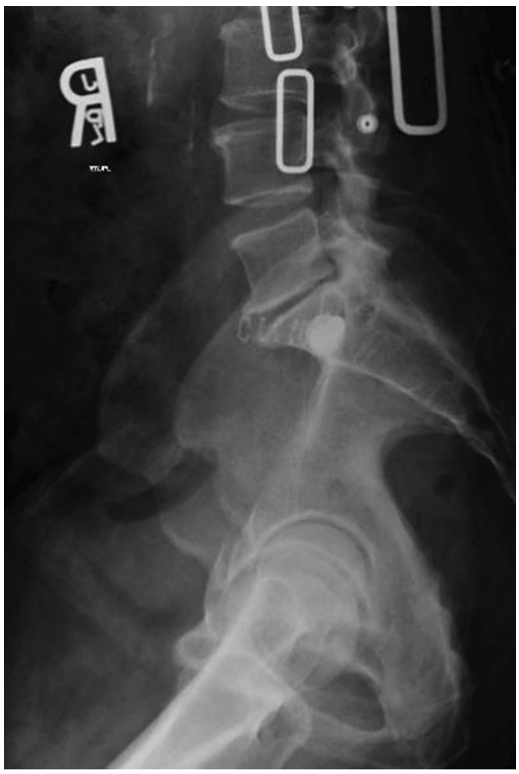

Figure 2. Revision sacroiliac screw placement demonstrating satisfactory placement. Clinically, this correlated with improvement in the left S1 radiculitis.

DISCUSSION

The pathophysiology of GBS in post-traumatic and postsurgical patients is not well understood, but the common pathway involves an autoimmune insult on the nervous system. Exposure to certain viruses, bacteria, vaccines, or even myelin itself may sensitize the body's immune system, which can trigger an autoimmune response on the nervous system.[1] Duncan *et al*[7] documented a case of GBS after head trauma. They hypothesized that the antibody complexes that arise in the central nervous system are only weakly immunopathogenic to the peripheral nervous system.[7] Nerve root irritation resulting in the patients' new onset S1 numbness may have resulted from the approximation of the SI screw relative to the left S1 neuroforamen. In selected patients, even minor neurological irritation possibly exposes the immune system to myelin, which sets off a cascade leading to immunogenicity. Steiner *et al*[8] reported a similar mechanism of direct nerve irritation causing GBS after epidural anesthesia, further supporting this theory.

Reports describing this phenomenon in postoperative patients are sparse in the orthopedic literature.[9] Guillain-Barré syndrome after orthopedic trauma has been reported in both operative[10–12] and nonoperative management.[9,13] The etiology in these cases is thought to be multifactorial, with T-cell dysfunction as the central theme. Common mechanical causes include malaligned hardware, bony fragments, hematoma, CSF, pyogenic infection, and fracture. Common biologic causes include acute neuropathies or plexopathies, transverse myelitis, multiple sclerosis, and electrolyte disturbances.

If the examination findings in a patient with postoperative weakness cannot be explained by the imaging, the differential diagnosis must be expanded to include more rare etiologies. If GBS is considered, a spinal tap with elevated CSF protein and normal WBC along with nerve conduction studies showing slowing or blockage of normal nerve conduction can confirm the diagnosis.

Prompt diagnosis of GBS is important because of its ability to progress rapidly to involve the respiratory musculature.[7,14] Once the diagnosis is made, patients can be treated with intravenous immunoglobulin, steroids, and, in severe cases, plasmapheresis.

➢ Key Points

- ❑ Guillain-Barré syndrome (GBS) is a rare cause of postoperative paralysis in the trauma patient.
- ❑ Causes of GBS in the trauma patient may be from direct neural trauma or infectious etiologies, among others.
- ❑ The diagnosis must be made promptly or progressive respiratory failure and death may ensue.

References

1. Hughes RA, Rees JH. Clinical and epidemiologic features of Guillain-Barré syndrome. *J Infect Dis* 1997;176(suppl 2):S92–8.
2. Arnason B, Soliven B. *Acute Inflammatory Demyelinating Polyneuropathy. Peripheral Neuropathy*. 3rd ed. Philadelphia, PA: WB Saunders; 1993:1437–97.
3. Rivas S, Douds GL, Ostdahl RH, et al. Fulminant Guillain-Barre syndrome after closed head injury: a potentially reversible cause of an ominous examination. *J Neurosurg* 2008;108:595–600.
4. Asbury AK, Cornblath DR. Assessment of current diagnostic for Guillain-Barré syndrome. *Ann Neurol* 1990;27:S21–4.
5. Walling A, Dickinson G. Guillain-Barré syndrome. *Am Fam Physician* 2013;87:191–7.
6. Jacobs BC. The spectrum of antecedent infections in Guillain-Barré syndrome. *Neurology* 1998;51:1110–5.
7. Duncan R, Kennedy PGE. Guillain- Barré syndrome following acute head trauma. *Postgrad Med J* 1987;63:479–80.
8. Steiner I, Argov Z, Cahan, et al. Guillain Barré syndrome after epidural anaesthesia: direct nerve root damage may trigger disease. *Neurology* 1985;35:1473–5.
9. Al-Hashel JY, John JK, Vembu P. Unusual Presentation of Guillain-Barre syndrome following traumatic bone injuries; report of two cases. *Med Princ Pract*. 2013;22:597–9.
10. Lee M, Campbell R, Born C. Guillain-Barré syndrome after failed pelvic fracture fixation. *J Trauma* 2009;67:E132–5.
11. Riebel GD, Heller JG, Hopkins LC. Guillain-Barré syndrome after an operation on the spine. *J Bone Joint Surg Am* 1995;77-A:1565–7.
12. Cheng J, Kahn DE. The acute motor-sensory axonal neuropathy variant of Guillain-Barré syndrome after thoracic spine surgery. *J Neurosurg Spine* 2011;15:605–9.
13. Freitas MRG, Nascimento OJM. Guillain-Barre syndrome after brachial plexus trauma. *Arq Neuropsiquiatr* 2006;64:1039–40.
14. Dong WS, Geun SS. Guillain-Barre syndrome following spinal fusion for thoracic vertebral fracture. *J Korean Neurosurg Soc* 2011;50:464–7.