Eur Spine J
DOI 10.1007/s00586-016-4577-2

CASE REPORT

Guillain–Barré syndrome following elective spine surgery

Nikhil Sahai[1] · **Ki Soo Hwang**[1] · **Arash Emami**[1]

Received: 11 January 2016 / Revised: 20 April 2016 / Accepted: 21 April 2016

Abstract
Purpose There is a paucity of literature describing Guillain–Barré syndrome (GBS) in the elective orthopedic patient. We aim to report one such case following spine surgery.
Methods A morbidly obese 52-year-old male developed diminished reflexes as well as left upper and lower extremity weakness following surgical decompression and fusion at L4–5. The patient had persistent weakness and progressed to areflexia, at which point urgent lumbar puncture supported a diagnosis of GBS.
Results The patient was promptly started on intravenous immunoglobulin and made significant clinical improvement with near-complete resolution of symptoms by 3-month follow-up visit. By the sixth month, he was able to function and ambulate without a cane. GBS is a rare and potentially critical cause of diminished reflexes and weakness in the post-operative elective orthopedic patient. We propose that morbid obesity may have contributed to the patient's susceptibility of developing GBS following surgery.
Conclusion Neurologic symptoms of this autoimmune condition may also mimic the clinical picture of an elective spine patient, thus confounding diagnosis. If imaging cannot explain exam findings or new neurologic symptoms post-operatively, rare disease processes should be considered in the differential diagnosis.

✉ Ki Soo Hwang
Hwang_md@yahoo.com

[1] Department of Orthopaedic Surgery, St. Joseph's Regional Medical Center, University Spine Center, 504 Valley Road, Wayne, NJ 07470, USA

Keywords Guillain–Barré syndrome · Areflexia · Postoperative weakness · Diminished reflexes · Paralysis · Weakness

Introduction

Guillain–Barré syndrome (GBS) is an eponym that describes an acute, inflammatory demyelinating peripheral polyneuropathy with an incidence of 0.81–1.89 (median 1.11) per 100,000 person-years, and is more common in men than in women (ratio 3:2). Initial presenting symptoms of the condition include cranial nerve deficits or pain, numbness, paresthesia, and limb weakness that can progress to symmetric ascending flaccid paralysis and areflexia. Prompt diagnosis is critical as mild GBS can advance rapidly to involve the respiratory musculature and lead to death [1]. Though GBS is usually preceded by infection, it has been reported following both non-operative [2] and operative orthopedic management, including total hip arthroplasty, pelvic fracture fixation, and spine surgery [3–5]. Symptoms typically present 1 week postoperatively, peak within 4 weeks, then resolve over weeks to months [1].

Diagnosis of Guillain–Barré syndrome is primarily clinical with supporting evidence from cerebrospinal fluid (CSF) studies and neurophysiological testing. CSF studies often show albuminocytologic dissociation, or increased concentration of protein with a normal cell count. Increased number of mononuclear or polymorphonuclear cells in CSF (>50 cells per μl) should point away from a diagnosis of GBS. Nerve conduction studies will show either a slowing or complete blockage of normal conduction. Areflexia and diffuse ascending weakness are clinical hallmarks of the condition. Treatment begins with

Published online: 09 May 2016

intravenous immunoglobulin and steroids, with plasmapheresis reserved for severe cases [1]. We aim to describe the case of such a patient who was diagnosed with GBS following elective spinal surgery.

Case report

Consent was obtained after the patient was informed that data concerning his case would be submitted for publication. A 52-year-old, morbidly obese (BMI = 49) gentleman with a past medical history of hypertension initially presented complaining of progressive lower back pain with radiation to bilateral lower extremities. He was unable to ambulate without a cane due to excruciating pain. Examination showed neurogenic claudication and radiculopathy, and MRI demonstrated severe lumbar spinal stenosis at L4–5. Initially, lumbar epidural injections provided moderate relief of symptoms, but his pain persisted. He began to show neurological deficits, including ankle weakness and further deficits in ambulation.

After failure of conservative treatments, the patient underwent posterior spinal decompression and fusion using a minimally invasive transforaminal lumbar interbody fusion (MIS-TLIF) technique. He had almost immediate relief of radicular pain with residual left ankle weakness. The patient was discharged home within 24 h following surgery and was able to ambulate with minimal assistance. He demonstrated significant clinical improvement at 2 weeks postoperatively. However, within 72 h of that evaluation, the patient called in complaining of new onset left arm and left leg weakness. The patient was admitted urgently due to inability to ambulate with rapidly progressing left upper and lower extremity weakness.

On examination, the patient had 2/5 motor function to all major muscle groups of his left upper and lower extremity. The patient was alert and responded to all commands. He underwent MRI of the brain and whole spine. MRI of the brain failed to demonstrate obvious pathology. Per radiology report, there was multi-level disc displacement and facet osteoarthrosis from C2 to C5. This was most pronounced at C3–4 where severe central canal narrowing and cord impingement were seen with signal hyperintensity of the cord, likely related to myelopathy. Moderate-to-severe right and left neural foraminal narrowing was also noted. Lumbar spine MRI showed previously performed laminectomy at L4–5 with moderate broad-based disc bulge and moderate narrowing of the central canal, lateral recesses, and neural foramina from L1–S1. Neurology was consulted and intracranial pathology was ruled out. However, diminished upper and lower left extremity reflexes were noted.

The consulting neurologist recommended decompression of the cervical and lumbar spine for symptomatic progression of cervical and lumbar spinal stenosis. The patient then underwent anterior cervical discectomy and fusion (ACDF) for severe cervical spinal stenosis at C3–4 followed by revision posterior decompression from L1–S1 to improve symptoms thought to be due to spinal stenosis. However, within 48 h post-operatively, it was again clear that his neurological function had still not improved.

At this time, a second neurology consultation was obtained. The second neurologist noted persistent left arm and left leg weakness as well as moderate right leg weakness. The most significant clinical finding was the absence of deep tendon reflexes. The patient underwent lumbar puncture which showed high protein and normal cell count, thus substantiating the diagnosis of Guillian–Barré syndrome. The patient was immediately started on immunoglobulin therapy. The patient made significant clinical improvement with near-complete resolution of left arm weakness at 3-month follow-up visit. By the sixth month, he was able to function and ambulate without significant intervention.

Discussion

Guillain–Barré syndrome is usually a post-infectious disease, and most patients report symptoms of a respiratory or gastrointestinal tract infection before the onset of neurologic symptoms. GBS has been reported following infection with *Campylobacter jejuni, Mycoplasma pneumonia, Haemophilus influenza*, cytomegalovirus, Epstein–Barr virus, and other pathogens. The preceding infection is important as it launches the immune response that ultimately leads to the development of GBS and damage to peripheral nerves [1].

Four factors have been implicated in this process: antiganglioside antibodies, molecular mimicry and cross-reactivity, complement activation, and host factors. Gangliosides are glycolipid molecules that are distributed throughout the nervous system and play a role in maintenance of human peripheral nerve cell membranes. About half of patients with GBS have serum antibodies to a variety of these gangliosides. Antibodies generated against certain pathogen-borne antigens may mimic self and lead to the cross-reactive destruction of self gangliosides within peripheral nervous tissue. Thus, the severity and subtype of GBS that manifests may be dictated by the precedent infection and the specificity of such antibodies in susceptible individuals [1, 6].

While infrequent, the development of post-operative GBS has been documented following cervical, thoracic, lumbar, and sacral spine surgery [5, 7]. It remains unclear

if undergoing surgery actually increases risk of developing GBS. Staff et al. studied nerve biopsies of a cohort of 23 patients who developed nontraumatic post-operative neuropathy within 30 days following a wide variety of surgeries; twenty-one of these showed inflammatory change [8]. Steiner et al. reported GBS in four patients following epidural anesthesia. They postulated that direct trauma to a nerve root following epidural anesthesia may release antigens (possibly myelin) and ultimately initiate an auto-immunologic sequence that begins with sensitization to neural elements [9]. This process may result in demyelination and neuropathy in susceptible patients as described above.

It is unclear what made our patient susceptible to GBS, but it is reasonable to postulate that the patient's morbid obesity may have played a role since obesity alters normal immune response and increases risk of infection. Adipocytes secrete cytokines and adipokines that are linked to the inflammatory response (such as leptin, adiponectin, TNF-α, IFN-α), and this altered secretion may increase levels of these chemical transmitters thereby affecting the interaction between adipocytes and leukocytes. Circulating leptin level has been shown to reflect mass of adipose tissue, and serum adiponectin has been shown to predict mortality in critically ill patients. As a result, macrophage differentiation and the inflammatory response become dysregulated [10].

In this case, our patient presented with weakness and diminished reflexes in the left upper and lower extremities following decompression and fusion. After two subsequent operations, the patient had progressed to areflexia. This case illustrates that when physical exam findings in the postoperative patient cannot be explained by imaging studies, rare diagnoses may be warranted for consideration. Though this is an uncommon complication, it can be a life-threatening condition and should therefore be diagnosed and treated as quickly as possible.

Compliance with ethical standards

Conflict of interest None of the authors have any potential conflict of interest to disclose.

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