



Case Report

Patient with Guillain-Barré Syndrome with Posterior Spinal Root Involvement: A Case Report

Nevin Kuloğlu Pazarıcı, Münevver Ece Güven, Dilek Necioğlu Örken

Department of Neurology, Şişli Hamidiye Etfal Training and Research Hospital, İstanbul, Turkey

Abstract

Guillain-Barré syndrome (GBS) is an acute inflammatory demyelinating polyradiculoneuropathy of the peripheral nervous system. Involvement of the dorsal root ganglia and the medulla spinalis in GBS is rare, especially in an axonal form. Herein, we report the case of a 53-year-old woman with classic GBS and involvement of the T8-L1 dorsal root segments. Dorsal root and spinal involvement should be kept in mind in all types of GBS when patients present with segmental or dermatomal sensory impairment.

Keywords: Demyelinating polyneuropathy; dorsal root involvement; Guillain-Barré syndrome.

Please cite this article as "Kuloğlu Pazarıcı N, Güven ME, Necioğlu Örken D. Patient with Guillain-Barré Syndrome with Posterior Spinal Root Involvement: A Case Report. Med Bull Sisli Etfal Hosp 2018;52(1):61-63".

Guillain-Barré syndrome (GBS) is a rare, rapid-onset, demyelinating polyneuropathy of the peripheral nervous system that is the result of an immune system disorder. It is characterized by symmetric loss of muscular strength and areflexia, typically starting in the lower extremities and ascending to the arms, face, and oropharyngeal muscles. The spectrum of GBS includes acute inflammatory demyelinating polyradiculoneuropathy (AIDP), the most common form, and acute motor axonal neuropathy (AMAN), acute motor-sensory axonal neuropathy (AMSAN) and Miller Fisher syndrome.^[1]

The incidence of GBS is 1 to 2/1000, and it may occur at any age. Generally, patients have experienced an upper respiratory tract or gastrointestinal tract infection, surgical intervention, vaccination, or childbirth 1 to 4 weeks before the onset of the disease. Sensory symptoms and numbness in the hands and feet may develop, followed by a loss

of strength. Facial paralysis and difficulty swallowing or breathing may accompany these symptoms. A progressive loss of muscular strength and the inability to elicit reflexive responses during a physical examination are sufficient to establish the diagnosis. The disease progresses rapidly, and maximum involvement is seen in half of patients at 2 weeks, and in 90% at 4 weeks. More than half of GBS patients cannot walk without assistance during the course of the disease. Nearly one-third of hospitalized patients need respiratory support and follow-up in an intensive care unit. Autonomic symptoms, such as cardiac arrhythmia, orthostatic hypotension, and hypertensive crisis, may be seen. Fifty percent of cases recover after a period of time without any sequelae. Mortality or severe sequelae are seen in 6% to 17% of cases.^[2]

Laboratory techniques that can aid in the establishment of the diagnosis include cerebrospinal fluid (CSF) analysis,

Address for correspondence: Nevin Kuloğlu Pazarıcı, MD, Department of Neurology, Sisli Hamidiye Etfal Training and Research Hospital, İstanbul, Turkey

Phone: +90 532 547 18 21 **E-mail:** nevinpazarcı@yahoo.com

Submitted Date: November 03, 2016 **Accepted Date:** February 14, 2017 **Available Online Date:** March 29, 2018

©Copyright 2018 by The Medical Bulletin of Sisli Etfal Hospital - Available online at www.sislietfalthip.org

This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc/4.0/>).



and an electromyography (EMG) examination. During the acute phase of GSB, the CSF characteristically includes albuminocytological dissociation, or a higher protein content and hypocellularity ($<10/mm^3$). Signs of demyelination, such as decreased nerve conduction velocity and conduction blocks may be observed on EMG.^[3]

Though GBS is a disease of the peripheral nervous system, only a few autopsy reports in the literature have indicated that inflammatory changes involved the anterior horn cells, posterior nerve roots, or the spinal cord. Clinical and electrophysiological findings support diagnosis of an axonal variant of GBS, such as AMAN, and AMSAN.^[4, 5]

Presently described is the case of a patient who was hospitalized with clinical manifestations of GBS, and following tests, was ultimately diagnosed with GBS. Sensory disturbances developed in dermatomes corresponding to the T8-L1 spinal segments.

Case Report

A 53-year-old female patient without any known disease presented at the intensive care unit with complaints of weakness in her arms and legs that had been increasing in severity for 6 days. Four days before the onset of her discomfort, she had started antibiotherapy for a dentine infection following a tooth extraction. Subsequently, the patient had difficulty moving her mouth and her speech was slurred, but this was initially attributed to the dental procedure and infection. Within 1 to 2 days, there was loss of strength and numbness in her fingertips and in her feet, and she had some difficulty walking. The patient was hospitalized and a neurological examination revealed bilateral eyelid weakness and an inability to raise her eyebrows, retract her oral commissures, or show her teeth. Muscular strength in the fingers of both hands in flexion and the toes in plantar and dorsiflexion was graded as 4/5. Deep tendon reflexes of both the upper and lower extremities could not be elicited. Bilateral plantar reflexes of the skin were absent. A sensory system examination revealed a glove-and-stockings-type sensory deficit, and a markedly decreased sense of position and vibration in the distal extremities. The results of routine tests of biochemical and hematological values; thyroid function; vitamin B12 and folic acid values; urinary porphobilinogen; Bence Jones protein; urine and serum immunofixation electrophoresis; immunoglobulins A, G, and M; tumor markers; and serology were within normal limits. CSF oligoclonal band analysis yielded negative results. Electrophysiological studies revealed acute sensory and motor involvement, preservation of the sural nerve, and signs consistent with demyelinating polyneuropathy.

Based on the clinical and electrophysiological findings, a

diagnosis of GBS was made, and intravenous immune globulin treatment was initiated at a daily dose of 0.4 g/kg for 5 days. The patient's symptoms continued to worsen, and on the fourth day of treatment, superficial and deep sensory deficits were detected on the T8-L1 segment dermatomes, and muscular weakness had further declined. Contrast-enhanced spinal magnetic resonance imaging findings were normal, and the treatment was maintained for a total of 5 days. One week after completion of the treatment, the sensory deficit detected on the T8-L1 segment dermatomes began to regress. A physiotherapy program was initiated, and the patient was discharged from the hospital. At 1 month after discharge, her sensory complaints were limited to the distal parts of her extremities and she could walk without support. A repeat electrophysiological examination revealed no significant lasting change.

Discussion

In GBS, in addition to multifocal segmental demyelination, symmetric motor and sensory symptoms of polyneuropathy may be seen. Following the decrease in the incidence of poliomyelitis in the population, GBS has become the most common cause of acute flaccid paralysis, with an incidence of 1 to 2/100.000. It generally occurs in individuals without any other concomitant disease; it is generally not associated with other systemic or autoimmune disease. Subgroups are defined by clinical and biochemical characteristics; however, in practice, the term GBS is used for the most frequently encountered form of the disease: the classic, inflammatory, demyelinating form. In Western countries, the demyelinating form is seen in more than 90% of cases, while the axonal form is seen in 60% of GBS cases in Asian countries. In studies performed in this country, the axonal form was more frequently seen in Eastern Anatolia, though not as often as in Asian countries.^[6]

The muscular weakness of GBS typically first involves the legs, and then progresses to the arms, oropharyngeal muscles, and in severe cases, the respiratory muscles. Occasionally, the disease may present with muscular weakness beginning in the arms or cranial muscles. Often, the muscle weakness is more extensive and involves proximal parts of the extremities, beyond what is seen in cases with polyneuropathy syndrome. Sensory complaints are generally not prominent, and typically consist of paresthesia of the distal ends of the extremities, or more rarely, pain in the back, waist, or lower extremities. On neurological examination, in addition to muscular weakness in the previously described areas, a widespread loss or decrease in tendon reflex is almost always, noted. Objective sensory findings are more often limited to mild superficial hypoesthesia of the distal ends of the extremities or decreased vibration and position

sensations.^[1]

The primary histopathological findings in the AIDP form of GBS consist of mononuclear inflammatory infiltration of the endoneurium and segmental demyelination of nerve fibers. Although these lesions may be seen on every part of the peripheral nerves, from the nerve roots to distal intramuscular nerve branches, mostly, the motor nerve roots and proximal plexus segments are involved. Rarely, these pathological changes extend beyond the posterior nerve roots and reach the medulla spinalis.^[1] This condition is called polyradiculomyelitis. Axonal damage without inflammatory infiltration is seen in the AMAN and AMSAN forms of the disease.^[4, 5]

Since the sensory deficits detected on the T8-L1 dermatomes did not reach the spinal cord in this case, we thought that the inflammatory changes responsible for the clinical manifestations were limited to the posterior nerve roots. Though this case had the clinical and electrophysiological characteristics of the typical form of GBS, it is unlike case reports in the literature. Perhaps the inflammation was prevented from reaching the medulla spinalis and limited the histopathological changes to some extent. Since spinal segmental sensory deficits are not expected manifestations in typical GBS, their presence may lead to confusion and suspicion in the diagnosis. This case may provide guidance in the diagnosis and treatment of similar cases.

This case was presented on a poster at the 32nd National Clinical Neurophysiology EEG-EMG Congress in Bodrum, Turkey,

April 27-May 1, 2016.

Disclosures

Informed consent: Written informed consent was obtained from the patient for the publication of the case report.

Peer-review: Externally peer-reviewed.

Conflict of Interest: None declared.

Authorship contributions: Concept – N.K.P.; Design – N.K.P.; Supervision – N.K.P.; Materials – M.E.G.; Data collection &/or processing – M.E.G.; Analysis and/or interpretation – N.K.P.; Literature search – N.K.P.; Writing – N.K.P.; Critical review – D.N.Ö.

References

1. Willison HJ, Jacobs BC, van Doorn PA. Guillain-Barré syndrome. *Lancet* 2016;388:717–27. [\[CrossRef\]](#)
2. Parman YG, Öge AE. İmmun kökenli polinöropatiler. *Klinik Gelişim Dergisi* 2009;22:31–5.
3. Öge AE. Periferik sinirlerin yaygın ve çok odaklı hastalıklarında tanı. *Klinik Gelişim Dergisi* 2010;23:91–8.
4. Rosenblum WI, Budzilovich G, Feigin I. Lesions of the spinal cord in polyradiculoneuropathy of unknown aetiology and a possible relationship with the Guillain-Barré syndrome. *J Neurol Neurosurg Psychiatry* 1966;29:69–76. [\[CrossRef\]](#)
5. Müller HD, Beckmann A, Schröder JM. Inflammatory infiltrates in the spinal cord of patients with Guillain-Barré syndrome. *Acta Neuropathol* 2003;106:509–17. [\[CrossRef\]](#)
6. Akıl E, Varol S, Taşkın A, Arıkanoğlu A, Tamam Y, Öztürk Ü. Guillain-Barre sendromunda klinik ve demografik özellikler. *Dicle Tıp Dergisi* 2014;41:707–11. [\[CrossRef\]](#)