# Case Report of Guillain–Barré Syndrome Presenting as Acute Paraparesis.

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## Abstract:

Guillain–Barre syndrome (GBS) is an acute immune-mediated inflammatory demyelinating polyradiculoneuropathy. The classic presentation characterises by symmetrical limb weakness and areflexia. Sometimes, it is difficult to diagnose GBS because of its variants. Here, we describe a rare presentation of GBS as acute paraparesis. A 65-year-old woman presented with progressive weakness both legs and numbness at fingers of both hands and feet for 9 days. On examination, sensory was involved in both upper and lower limbs, but weakness showed only in lower extremities. Nerve conduction study showed sensory-motor demyelinating polyneuropathy and hence GBS was diagnosed. The patient received intravenous immunoglobulin (IVIG), then she completely recovered within 3 weeks.

**Keywords:** Guillain–Barré Syndrome, Parapararetic variant, Acute paraparesis

## Introduction:

Guillain–Barre syndrome (GBS) is an acute immune-mediated inflammatory demyelinating polyradiculoneuropathy. The classic presentation is acute onset ascending sensorimotor polyneuropathy resulting generalized weakness and areflexia. However, the disease can present atypically or as a clinical variant.[1] We describe a case of GBS presenting as acute paraparesis which is a rare presentation. It is important for clinician to aware atypical presentation of GBS in order to provide pharmacological treatments such as plasmapheresis or intravenous immunoglobulin (IVIG) when a patient has significant disability.

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## Patient presentation:

A 65-year-old woman presented with progressive weakness both legs and numbness at fingers of both hands and feet for 9 days. There is no weakness on her upper limbs. She also compliant about muscle pain in both arms and legs 3 weeks earlier. She had a background of hyperlipidemia. None of her family members had history of muscle weakness or experienced similar symptoms.

On admission, her general appearance, vital and systemic examinations were normal. Neurological examination revealed normal function of the cranial nerves. Her muscle strength was 5/5 in the upper limbs (both the proximal and distal muscles), 3/5 of both iliopsoas, gluteus maximus, hamstring muscles, 4/5 of both adductors, extensor hallucis longus muscles, and 5/5 of both quadriceps, tibialis anterior, gastrocnemius and gluteus medius muscles. Her pinprick sensation and proprioception were intact in both upper and lower extremities, but her vibratory sensation decreased at both feet and toes. Deep tendon reflexes were 1+ in the upper extremities and areflexia in the lower extremities. The plantar responses were normal bilaterally. Cerebellar examination was normal.

She went to private hospital 2 days before this admission. MRI lumbosacral spine with screening whole sagittal spine was performed, and the results were normal (**Figure 1**). After that, she came to our outpatient department (OPD). GBS was in the differential diagnosis, therefore, we performed lumbar puncture. Cerebrospinal fluid (CSF) analysis showed that the protein level was high (158.8 mg / dL), while the white blood cell count was 4 with all mononuclear cells. Nerve conduction study (NCS) results, in sensory NCS showed low SNAP amplitude [2.7] of the right sural nerve. Motor NCS showed definite conduction block (by proximal to distal amplitude criteria) [Rt tibial proximal 3.0 to distal 8.6 mV] [Rt peroneal proximal 3.5 to distal 1.5 mV] and slightly slow CV [Rt tibial 35.4 m/s, Rt peroneal 36.4 m/s] of the right tibial and peroneal nerves and partial conduction block and slow CV [Rt median 42.9 m/s, Rt ulnar 38.8 m/s] of the right median and ulnar nerves. The F wave was absent in the right peroneal nerve and the rest of the testing were normal (**Figure 2**). According to the NCS results, there were electrophysiological evidence of sensory-motor demyelinating polyneuropathy. The patient received intravenous immunoglobulin (0.4 kg/day) for 5 days. 3 weeks follow-up after treatment, her leg weakness had recovered completely.

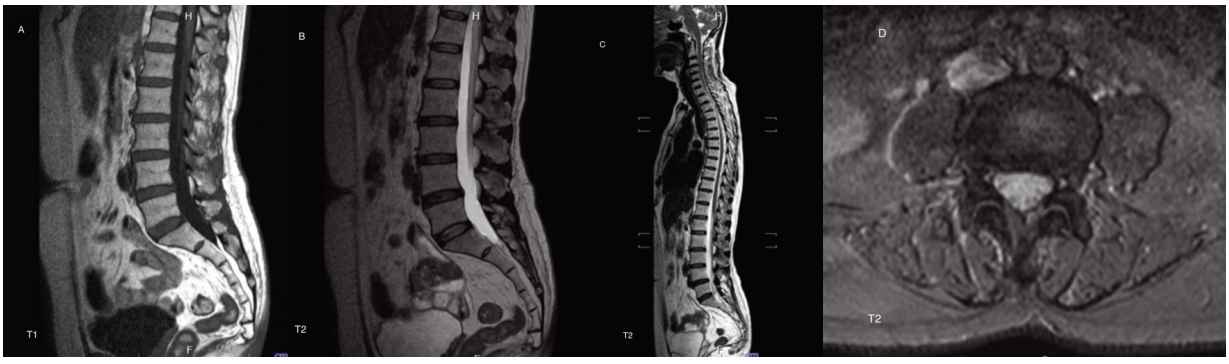


**Figure 1**: Magnetic resonance imaging of lumbosacral spine with screening whole sagittal spine revealed normal. (A) Sagittal T1 sequence of LS spine; (B) Sagittal T2 sequence of LS spine; (C) Sagittal T2 sequence of whole spine; (D) Axial T2 sequence of LS spine.

Sensory NCS

| Nerve/Sites | Onset Lat (ms) | Peak Lat (ms) | Peak Amp (uV) | Duration (ms) | Distance (cm) | Velocity (m/s) |
|---|---|---|---|---|---|---|
| R Median - Digit II (Antidromic) | | | | | | |
| Wrist | 2.6 | 3.2 | 18.3 | 1.4 | 13 | 49.9 |
| R Ulnar - Digit V (Antidromic) | | | | | | |
| Wrist | 2 | 2.7 | 23.2 | 1 .4 | 11 | 55.6 |
| R Sural - Ankle (A 7 cm, B 14 cm, C 21 cm) | | | | | | |
| Calf(A) | 3.4 | 4.2 | 2.7 | 2.1 | 7 | 20.7 |
| | | | | | 14 | |

F Wave

| Nerve | Min M Lat (ms) | Min F Lat (ms) | Min F-M (ms) |
|---|---|---|---|
| R Peroneal - EDB | 4.3 | - | |
| R Tibial -AH | 4 | 33.6 | 29.5 |
| R Median - APB | 2.9 | 36.3 | 33.4 |
| R Ulnar - ADM | 2.5 | 34.7 | 32.2 |

Motor NCS

| Nerve/Site | Onset Lat (ms) | PK Amp (mV) | Duration (ms) | Area (mVms) | Distance (cm) | Lat Diff (ms) | Velocity (m/s) |
|---|---|---|---|---|---|---|---|
| R Median - APB | | | | | | | |
| Wrist | 2.9 | 13.1 | 4.7 | 31.4 | 7 | | |
| Elbow | 7.8 | 7.5 | 5.9 | 21.3 | 21 | 4.9 | 42.9 |
| R Ulnar - ADM | | | | | | | |
| Wrist | 2.4 | 1 1.7 | 5.1 | 32 | 7 | | |
| B.Elbow | 7.9 | 8.4 | 6.1 | 26.6 | 21 | 5.4 | 38.8 |
| A. Elbow | 9.5 | 6.2 | 6 | 19.8 | 10 | 1.7 | 60 |
| R Peroneal - EDB | | | | | | | |
| Ankle | 3.5 | 3.5 | 6.1 | 11 .4 | 8 | | |
| Fib head | 12.3 | 1 .5 | 7.7 | 6.4 | 32 | 8.8 | 36.4 |
| Pop fossa | 15.7 | 1.2 | 6.1 | 4 | | 3.4 | |
| R Tibial -AH | | | | | | | |
| Ankle | 3.8 | 8.6 | 5.3 | 20.5 | 8 | | |
| Pop fossa | 14.5 | 3 | 5.5 | 8.1 | 38 | 10.7 | 35.4 |

**Figure 2**: Nerve conduction study (NCS) results, in sensory NCS showed normal dLat and SNAP amplitude of the right median and ulnar nerves, normal dat and low SNAP amplitude [2.7] of the right sural nerve. Motor NCS showed normal dLat, normal distal CMAP amplitude, definite conduction block (by proximal to distal amplitude criteria) [Rt tibial proximal 3.0 to distal 8.6 mV] [Rt peroneal proximal 3.5 to distal 1.5 mV] and slightly slow CV [Rt tibial 35.4 m/s, Rt peroneal 36.4 m/s] of the right tibial and peroneal nerves. Normal dLat, normal distal CMAP amplitude, partial conduction block and slow CV [Rt median 42.9 m/s, Rt ulnar 38.8 m/s] of the right median and ulnar nerves. The F wave was absent in the right peroneal nerve and The F wave latency of the tibial, median and ulnar nerves were normal.

## Discussion:

Guillain Barre syndrome (GBS) is an inflammatory disease of the peripheral nervous system caused by an aberrant immune response to infections.[1] Currently, GBS is the leading cause of acute paralytic disease after the virtual eradication of acute poliomyelitis.[1] Incidence of GBS was 0.48 to 0.93 in Thailand and 1 to 2 per 100,000 population worldwide.[1,2]

So called classic GBS is the most common manifestation, which is symmetrical distal paresthesia, accompanied or followed by weakness that starts in the legs and progresses to the arms. It accounts for in 30-85% of all GBS patients worldwide.[1] It may involve facial and oropharyngeal muscles, respiratory muscles, as well as autonomic nervous system.[3] Interestingly, Some GBS patients have a distinct clinical variant which can be mimicker of other neurological diseases. These variants include 1) pure motor variant, 2) pure sensory, 3) bilateral facial palsy with paraesthesias, 4) pharyngeal–cervical–brachial weakness, 5) paraparetic variant, 6) Miller Fisher syndrome and 7) Bickerstaff brainstem encephalitis.[1]

We demonstrate a patient with paraparetic variant of GBS and review difference between paraparetic variant and classic GBS. Paraparetic variant, described as an isolated weakness of both lower limbs, was reported as 5-10% of all GBS[1], which should be distinguished from other causes of paraparesis particularly spinal cord lesion.

Paraparetic variant of GBS has many features similar to classic GBS. CSF profile typically showed albuminocytologic dissociation. There were only minority about 16% of paraparetic variant had slight lymphocytic pleocytosis greater than 10 cells/mm.[4] Moreover, patients with paraparetic variant showed demyelinating features in 44%, axonopathy in 6% and equivocal 50%, without significant difference from quadriparetic group.[4] However, a study in India showed that 59% had axonal subtype, while 33% had demyelinating subtype.[5]

Interestingly, according to a cohort study of 40 paraparetic GBS patients, 50% had clinical sensory deficit arms and 89% had abnormal electrophysiologic study in the upper extremities. In addition, 28% of them had emerging arm weakness during follow up.[4] Because of many similar features and developing arm weakness in some paraparetic patient, paraparetic variant may be an intermediate diagnosis of classic GBS, eventually, leading to the diagnosis of classic GBS.

On the other hand, there were some features distinguish from classic GBS. Approximately 25% of paraparetic GBS patients had a history of preceding viral prodrome symptom[6], while in classic GBS reported up to three quarters.[6] Besides, cranial nerve involvement was also found significantly less in paraparetic variant in 32% compared to classic GBS in 54%.[4] Currently, there was no clear reason to explain these differences.

In our case, she had only symmetric weakness and parethesia both lower extremities with evidence of demyelination by conduction block in electrodiagnostic study. There was no abnormal symptom or abnormal value on electrodiagnostic study of her upper extremities. In addition, Her CSF profile showed albuminocytologic dissociation. Thereby her condition was compatible with paraparetic variant of GBS. She did not have a history if preceding viral prodome symptom and cranial nerve involvement.

Most patients of paraparetic variant, about 88%, required therapy with intravenous immunoglobulin or plasmaphereis. However, it is significantly less than quadriparetic GBS patients who required therapy in about 97%. Their paraparetic GBS patients had a better prognosis than those with quadriparesis after a 6-month follow-up.[4] However a case report show a patient with paraparetic variant GBS who fully recovered after 1.5 months of symptoms without the use of any immunomodulatory drugs.[7]

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