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## *CASE REPORT*

# GBS with Bilateral plantar extensor – A case report

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**Abstract:**

*GBS is an immune mediated polyradiculoneuropathy classically characterized by acute symmetrical ascending lower motor type weakness and areflexia. But sometimes, in axonal variants of GBS, reflexes are preserved or exaggerated. We report a case of GBS with bilateral extensor plantar response during the course of the disease. A 36-year-old male presented with acute quadriplegia with asymmetrical muscle weakness and extensor plantar response. Sensory, bowel and bladder function was intact. He was treated with intravenous methylprednisolone daily for 5 days without improvement. NCS revealed AIDP and AMAN variants of GBS. So, in any patient presenting with acute quadriplegia with extensor plantar response, GBS should be considered as differential diagnosis.*

***Abbreviation:*** *NCS ( nerve conduction study), AIDP(acute inflammatory demyelinating polyradiculoneuropathy), AMAN ((acute motor and axonal polyradiculoneuropathy), GBS (Guillain-Barré syndrome), CSF( cerebrospinal fluid ), AMSAN(acute motor sensory axonal neuropathy).*

**Introduction:**

Guillain-Barré syndrome is an acute, immune mediated, frequently severe and fulminant polyradiculoneuropathy[1]. It is clinically characterized by acute, progressive, symmetrical ascending muscle weakness and areflexia with or without sensory, autonomic or brainstem involvements. Cranial nerve involvement occurs in 45% to 75% of cases in different series. Facial paresis, usually bilateral, is present in 50% of affected individuals[2]. Although, the diagnosis of GBS is based on clinical criteria, the presence of suggestive findings in the nerve conduction studies (NCS) or albuminocytological dissociation in the cerebrospinal fluid (CSF) analysis help to confirm the diagnosis[3]. We reported a case of GBS with asymmetrical weakness and extensor plantar response during the course of the disease.

**Case presentation:**

A 36-year-old male was admitted with weakness of all 4 limbs for 5 days. It was sudden onset and gradually progressive. Weakness started in left upper limb, then right upper limb and subsequently involved both lower limbs 1 day later. Initially, he performed his daily activities with assistance, later 2 days prior his admission, he become bedridden. There was no history of fever, respiratory tract infection, diarrhea, vaccination prior to his illness within 1 month. On examination, bulk and tone of muscles were normal. The weakness of all 4 limbs were asymmetrical and muscle power were of 2/5 in both upper limbs and 3/5 in both lower limbs with more marked on proximal than distal part. All deep tendon reflexes were present with bilateral plantar extensors. All modalities of sensation were intact except $C_5$ and $C_6$ was absent. There were no involvement of cranial nerves, respiratory system and autonomic systems. He was treated with intravenous methylprednisolone daily for 5 days without improvement. The investigations showed normal findings of total and differential leukocyte counts and serum electrolytes. Vasculitis screening was negative. MRI of brain (Fig. 1), MRI of cervical spine with screening of whole spine was normal. CSF examination showed

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albuminocytological dissociation. CSF protein was 100 mg/dl and cell count only 2 (100% lymphocyte). All causes of infectious radiculopathies were ruled out by analysis of serological test for infectious agent. Nerve conduction study(Table I) showed demyelinating and axonal polyradiculopathy. Later on the patient was treated with 5 courses of plasmapharesis every alternate day. The patient was gradually improved after plasmapheresis and subsequently he was discharged from hospital.

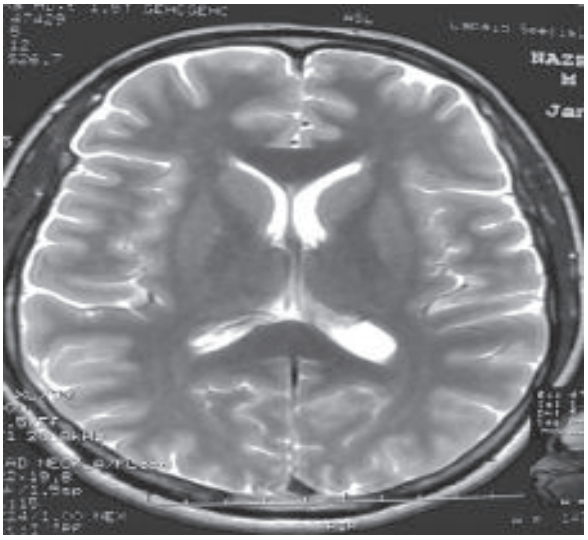


**Fig.-1:** *MRI of Brain revealed Normal.*

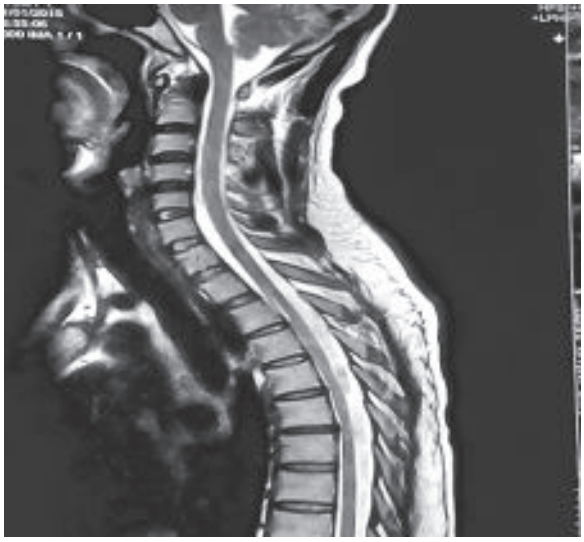
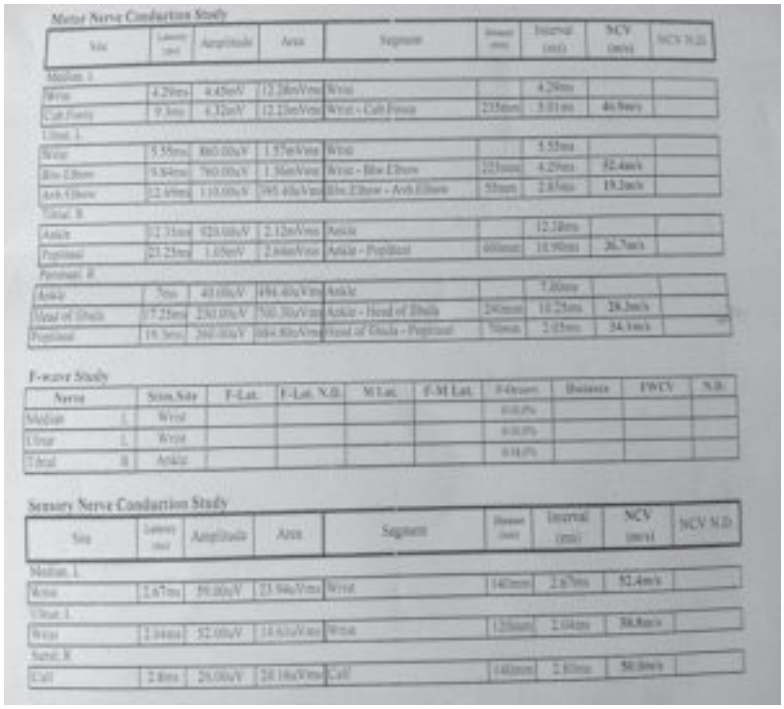


**Fig.-2:** *MRI of cervical spine and dorsal spine with screening of other spine revealed normal.*

**Table-I**

*Nerve conduction study (NCS) revealed demyelinating and axonal polyradiculoneuropathy*



Motor Nerve Conduction Study

| Site | Latency (ms) | Amplitude | Area | Segment | Distance (mm) | Interval (ms) | NCV (m/s) | NCV N.D |
|---|---|---|---|---|---|---|---|---|
| Median, L | | | | | | | | |
| Wrist | 4.29ms | 4.45mV | 12.28mVms | Wrist | | 4.29ms | | |
| Cub Fossa | 9.3ms | 4.32mV | 12.23mVms | Wrist - Cub Fossa | 235mm | 5.01ms | 46.9m/s | |
| Ulnar, L | | | | | | | | |
| Wrist | 5.55ms | 860.00uV | 1.97mVms | Wrist | | 5.55ms | | |
| Bw Elbow | 9.84ms | 760.00uV | 1.56mVms | Wrist - Bw Elbow | 223mm | 4.29ms | 52.4m/s | |
| Arb Elbow | 12.69ms | 110.00uV | 295.40uVms | Bw Elbow - Arb Elbow | 55mm | 2.85ms | 19.2m/s | |
| Tibial, R | | | | | | | | |
| Ankle | 12.33ms | 920.00uV | 2.12mVms | Ankle | | 12.38ms | | |
| Popliteal | 23.25ms | 1.05mV | 2.64mVms | Ankle - Popliteal | 400mm | 10.90ms | 36.7m/s | |
| Peroneal, R | | | | | | | | |
| Ankle | 7ms | 40.00uV | 484.40uVms | Ankle | | 7.00ms | | |
| Head of Fibula | 17.25ms | 230.00uV | 760.30uVms | Ankle - Head of Fibula | 240mm | 10.25ms | 28.3m/s | |
| Popliteal | 19.5ms | 360.00uV | 884.80uVms | Head of Fibula - Popliteal | 55mm | 2.05ms | 34.1m/s | |

F-wave Study

| Nerve | Stim.Site | F-Lat. | F-Lat. N.D | M-Lat. | F-M Lat. | F-Amp | Distance | FWCV | N.D. |
|---|---|---|---|---|---|---|---|---|---|
| Median L | Wrist | | | | | 0.0uV | | | |
| Ulnar L | Wrist | | | | | 0.0uV | | | |
| Tibial R | Ankle | | | | | 0.0uV | | | |

Sensory Nerve Conduction Study

| Site | Latency (ms) | Amplitude | Area | Segment | Distance (mm) | Interval (ms) | NCV (m/s) | NCV N.D |
|---|---|---|---|---|---|---|---|---|
| Median, L | | | | | | | | |
| Wrist | 2.67ms | 59.00uV | 23.94uVms | Wrist | 140mm | 2.67ms | 52.4m/s | |
| Ulnar, L | | | | | | | | |
| Wrist | 2.04ms | 52.00uV | 18.61uVms | Wrist | 120mm | 2.04ms | 58.8m/s | |
| Sural, R | | | | | | | | |
| Calf | 2.8ms | 26.00uV | 20.18uVms | Calf | 140mm | 2.83ms | 50.0m/s | |

**Discussion:**

This is a case of GBS with atypical clinical presentation, characterized by acute quadriplegia with asymmetrical weakness and bilateral extensor plantar responses. Our patient had no history of antecedent infection. We diagnosed him as GBS on the basis of acute progressive quadriplegia, albuminocytological dissociation on CSF and NCS revealed demyelinating and axonal polyradiculo-neuropathy. Despite an atypical pattern of clinical signs and symptoms, the plasmapharesis was started which leaded to the functional recovery of our patient.

GBS is an immune mediated acute progressive inflammatory polyradiculoneuropathy that characterized by symmetrical muscle weakness and areflexia. Several types of GBS are recognized, acute inflammatory demyelinating poly-radiculoneuropathy (AIDP) is common variant. Additionally, there are two axonal variants, are well recognized that are acute motor axonal neuropathy (AMAN) and acute motor sensory axonal neuropathy (AMSAN)[2]. Axonal variants are commonly associated with preserved or brisk reflexes. Hyperreflexia seen in GBS has a common association with antecedent C jejuni infection and positive anti-$GM_1$ ganglioside antibody. Although, all patients have IgG anti-GM1 ganglioside antibody and anti-c jejuni antibodies are frequently negative[4]. Antibody testing is not widely available in our country which makes the diagnosis.

**Conclusion:**

GBS patient may present with signs and symptoms associated with CNS involvement. So, Neurologist as well as internist should have a high degree of suspicion towards the diagnosis of GBS, if a patient present with acute motor paraparesis or quadriparesis with extensor plantar response. In that case, NCS and CSF analysis can confirm the clinical findings. Early diagnosis and treatment of GBS may prevent mortality and morbidity.

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