**Paraparetic Guillian-Barre Syndrome: An uncommon diagnosis of acute flaccid paralysis of the lower limbs**

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**Key Clinical message**:

Upon encountering acute, flaccid paralysis of lower limbs, besides regular differentials of transverse myelitis, cord compression; paraparetic GBS should be considered since it’s timely diagnosis and management helps prevent complications and prevent unnecessary interventions. In addition we report a case where we managed similar case without use of immunomodulatory agent.

**Abstract**:

Paraparetic Guillian-Barre Syndrome (GBS) is an uncommon variant of GBS presenting as bilateral weakness of the lower limbs with loss of reflexes without involvement of the upper limbs or cranial nerves. We describe a case of a teenage boy, who developed progressive, symmetric weakness of the lower limbs following an upper respiratory tract infection. Post-infectious, monophasic, acute, symmetric course of weakness were suggestive of paraparetic GBS which were further confirmed by cerebrospinal fluid (CSF) and electrophysiological findings.

**Keywords**

Paraparetic GBS, neuropathy, flaccid paralysis

**Introduction**

Globally, GBS is the most common cause of acute flaccid paralysis in the current era of near elimination of poliomyelitis. [1]Typically, GBS has an acute ascending bilateral symmetric weakness of varying degrees in limbs, but it can have a heterogeneous presentation ranging from symptoms restricted to certain parts of the body called “GBS variant” or “ topographic variant” to quadriparesis with autonomic and respiratory involvement. [2][3] Diagnosis is made after proper correlation of clinical history, cerebrospinal fluid analysis, electrodiagnostic test and neuroimaging is needed in some cases to rule out differentials. Herein, we present a case of paraparetic GBS in a teenage boy who was managed conservatively and recovered fully in one and a half months.

**Case presentation**

A 19-year-male student presented to our centre with chief complains of  progressive weakness of the lower limbs for 2 days. While waking up in the morning, the patient found his legs were feeling heavy and had difficulty in lifting his legs. While going to the washroom, he was unable to grip his slippers properly. In the evening, his weakness further worsened so that he needed the support of his parents for getting up from chair. There was no facial weakness, dysarthria, dysphagia, or diplopia. There was further worsening in weakness of lower limbs the next day, prompting support of parents during walking. However, there was no upper limb weakness, sensory symptoms, or bowel and bladder involvement. Ten days before the onset of limb weakness, he had rhinorrhoea and mild fever that lasted for about 3 days. He didn’t have any history of similar weakness in the past, back pain, weight loss, shortness of breath, recent trauma, surgery, insect bite, or drug history.

On arrival at the emergency department, the patient’s Glasgow Coma Scale was 15/15. Patient’s body weight was 65 kg and height 181cm, blood pressure was 110/80 mm Hg, pulse 88/min, temperature 98-degree Fahrenheit, and respiratory rate 16/min. There was no cyanosis, clubbing, jaundice, or pallor. There was no significant difference in muscle bulk of the limbs, tone reduced in bilateral ankle and knees, normal in rest joints. Power was 5/5 across all the joints in upper limbs while lower limb examination showed bilateral hip flexion 4/5, hip extension 3/5, knee flexion 4/5, knee extension 4/5 and ankle dorsi flexion 2/5 and plantar flexion 3/5. Reflex was 2+ in bilateral biceps, triceps and supinators. Left knee and ankle reflex was 1+, right knee and bilateral ankle reflexes were absent. Plantar response was down going on both sides.

On laboratory investigations, total leukocyte count was 6400 (Neutrophils 57 %, Lymphocyte 30%, monocytes 8%, Eosinophil 4%, Basophils 1%), Haemoglobin 16 gm/dl, Erythrocyte Sedimentation Rate 25 mm/hr, sugar 98 (mg/dl), urea 19 mg/dl, creatinine 0.6 mg/dl, Sodium 137 (mEq/l) and potassium 3.9 mEq/l. Lumbar puncture was done on the 7th day and CSF findings revealed a total white blood cell count <5 with 100% lymphocytes, glucose 86 mg/dl, protein 68 mg/dl. Chest X-ray and electrocardiogram findings were normal. Laboratory results including human immunodeficiency virus, syphilis, hepatitis B and C, serum angiotensin converting enzyme and thyroid stimulating hormone level were unremarkable. Magnetic Resonance Imaging (MRI) of the dorsal and lumbosacral spine showed no significant abnormalities that corroborated to clinical presentation. Nerve Conduction Test (NCT) performed on the 8th day of onset of symptoms showed a pattern of motor axonal neuropathy in the lower limbs; normal in the upper limbs; and preserved sensory amplitudes in both upper and lower limbs.

Weighing the severity of his weakness, we decided not to start immunomodulatory treatment and managed him conservatively with appropriate sessions of physiotherapy. He was discharged after 7 days of admission with mild improvement in motor power: bilateral hip flexion 4+/5, hip extension 4/5, ankle dorsi flexion 3/5 and rest were similar as admission. On follow-up at 1.5 months, he had fully recovered power in the lower limbs with no residual deficits.

**Discussion**

Clinical spectrum of GBS encompasses Classical GBS, Miller Fisher variant, the Pharyngeal-Cervical-Brachial (PCB) variant, paraparetic variant, pure motor variant GBS, GBS with acute pharyngeal weakness, bifacial weakness with paraesthesia, acute ptosis, acute mydriasis, acute ataxic neuropathy, acute ataxic hypersomnolence and Bickerstaff brainstem encephalitis. [1][4][5]Besides classical GBS, uncommon variants are usually late diagnosed and likely to land in the emergency or intensive care units with full-blown GBS with respiratory failure and autonomic dysfunction [1]*.* The paraparetic variant of GBS is an uncommon variant with weakness being confined to the legs. *Ropper et. al* first described this variant in 1986 as paraparesis with normal power, sensation and reflexes in the arms.[6] There’s no exact data revealing how common the paraparetic variant is, but different retrospective and prospective studies have revealed them to constitute 1-8% of total GBS [1][6][7][8] Looking at the available data, it is obvious that paraparetic GBS is not a common variant and we hope our case adds insights into the diagnosis and management of this uncommon variant.

# With the aforementioned presentation of the patient to us, common differentials borne in our mind were transverse myelitis and cauda equine neuritis. They were ruled out on the basis of absence of a discrete spinal cord lesion on spinal MRI, absence of well-defined sensory levels, or bowel and bladder involvement. Spinal cord compression secondary to spondylodiscitis, leptomeningeal malignancy, lymphoma, intramedullary primary spinal cord tumor were excluded due to the absence of corresponding findings on spinal MRI. Infection (Cytomegalovirus, HIV), inflammation (eg, Sarcoidosis), endocrine (diabetes mellitus, hypothyroidism) conditions were ruled out on the basis of laboratory and CSF findings.

In our case, a paraparetic variant of GBS, is confirmed by clinical symptoms, characteristics of disease progression and improvement, CSF findings, electrodiagnostic tests and ruling out focal lesions by neuroimaging. There was also a history of an antecedent upper respiratory tract viral infection which has been reported in about 25% of total paraparetic GBS.[7]Throughout the course of the illness, our patient had an intact sensory system and deep tendon reflexes and NCT of the upper limbs. However, *Berg et. al* has reported sensory abnormalities or loss / absence of reflexes in the upper limbs or abnormal NCS in the upper limbs in 98% of 40 paraparetic GBS. One limitation could be that we were unable to do serial NCTs in our patient.[5] In a study of *Hiew et. al*, about 75% of paraparetic patients had an axonal pattern in NCT, similar to our patient.  [7] About 88% of total paraparetic GBS required treatment with intravenous immunoglobulin or plasmaphereis in the study by *Berg et. al*, however, our patient was not started on any immunomodulatory agent. In their study, patients with paraparesis were found to have a better outcome than patients with quadriparesis in a follow-up of 6 months.[5] In consensus, our patient also had a full recovery without any deficits after 1.5 months of onset of symptoms without the use of any immunomodulatory agents.

**Conclusion**

Paraparetic GBS is an uncommon presentation and an alternative diagnosis should always be kept in mind until ruling out the differentials. Early diagnosis and monitoring are important, as some patients might develop respiratory compromise or severe dysautonomia.

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**Conflicts of interest**:

The authors declare they have no conflicts of interest.

**Authors Contributions:**

PL: involved in writing the manuscript, collection of case information, manuscript revision, RO: involved in writing the manuscript concept, collection of case information, manuscript revision. NN, NA, SP: participated in preparing a literature review and interpretation of clinical findings. BPG, RK, RR, NG, AS: involved in patient care team and collection of case information. All authors approved the final version.

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**Table 1: Nerve conduction study: Motor**

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| Nerve | Latency  (ms) | | Amplitude  (mv) | | NCV  (m/s) | F-Min |
|  | D | P | D | P |  |  |
| Rt. CPN | 5.37 | 12.19 | 1.58 | 1.14 | 46.92 | 51.27 |
| Lt. CPN | 4.31 | 11.19 | 1.81 | 0.98 | 46.51 | 50.00 |
| Rt. PTN | 5.69 | 17.31 | 4.48 | 2.51 | 48.86 | 46.87 |
| Lt. PTN | 5.56 | 16.12 | 1.98 | 1.17 | 44.51 | 51.37 |
| Rt. Median | 2.75 | 7.31 | 13.37 | 12.08 | 59.21 | 27.56 |
| Rt. Ulnar | 3.06 | 7.62 | 9.12 | 8.15 | 57.02 | 26.75 |
| Lt. Median | 3.81 | 8.50 | 8.49 | 7.86 | 55.44 | 29.31 |
| Lt. Ulnar | 3.25 | 8.87 | 10.01 | 8.12 | 54.11 | 29.81 |

D: Distal; P: Proximal; Rt.: Right; Lt.: Left; NCV: Nerve conduction velocity

**Table 2: Nerve conduction study: Sensory**

|  |  |  |  |
| --- | --- | --- | --- |
| Nerve | Latency(ms) | Amplitude(µv) | NCV(m/s) |
| Rt. Sural | 3.85 | 20.37 | 38.96 |
| Lt. Sural | 3.45 | 17.26 | 43.48 |
| Rt. Median | 2.78 | 38.09 | 61.15 |
| Rt. Ulnar | 2.48 | 26.64 | 60.48 |
| Lt. Median | 2.85 | 41.87 | 59.65 |
| Lt. Ulnar | 2.62 | 60.02 | 57.25 |

Rt.: Right; Lt.: Left; NCV: Nerve conduction velocity