

Case Report**Hyperreflexia — an Uncommon Presentation of Guillain-Barré Syndrome**N. S. Neki¹, Gagandeep Singh Shergill², Amanpreet Kaur³

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doi: <http://dx.doi.org/10.3329/jemc.v7i2.32658>**Abstract**

Guillain-Barré syndrome (GBS) is an acquired acute autoimmune polyradiculoneuropathy. Progressive motor weakness and areflexia are essential for diagnosis. But in some cases hyperreflexia can be seen. Diagnosis of GBS was made based on history and clinical findings and was supported by cerebrospinal fluid (CSF) studies and nerve conduction study (NCS). We hereby report a case of a 42-year-old male presenting with acute onset flaccid quadriparesis. There was frank hyperreflexia in all four limbs. Although reflex preservation and hyperreflexia can be noted in axonal variant of GBS in Chinese, Japanese, and European populations, it is uncommon in India.

Key words: Guillain-Barré syndrome; Hyperreflexia; Axonal variant

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Introduction

Guillain-Barré syndrome (GBS) is an acquired acute inflammatory demyelinating polyradiculoneuropathy (AIDP), which manifests as areflexic, flaccid paralysis with variable sensory disturbances, and increased cerebrospinal fluid (CSF) protein without pleocytosis i.e. albumino-cytological dissociation. Even though hyporeflexia or areflexia is necessary for diagnosis of GBS, hyperreflexia does not exclude a GBS variant. The subtypes of GBS are acute inflammatory demyelinating polyneuropathy (AIDP), acute motor axonal neuropathy (AMAN), acute motor sensory axonal neuropathy (AMSAN), Miller Fischer syndrome (MFS).^{1,2}

Case report

A 42-year-old male presented with complaints of acute onset flaccid weakness in all four limbs for 10 days. There was no history suggestive of sensory

abnormality. Bladder and bowel habits were normal. Past history revealed an episode of diarrhea 7–8 days back which resolved spontaneously. He was almost bed-ridden, unable to move his limbs and unable to lift his head from the bed or move from side to side following the event. Detailed neurological examination revealed a normal higher mental function without any cranial nerve involvement or neck rigidity. Limbs were flaccid, and power of the proximal muscles of the lower limb was 3/5 (Medical Research Council grading) and distal muscles was 2/5. In the distal upper limb muscles, power was 3/5. There was mild truncal weakness. Deep tendon reflexes were brisk throughout the course of illness and plantars were flexor bilaterally. Examination of sensory system and cerebellar system were unremarkable. All superficial reflexes were normal. Cardiovascular, respiratory and gastrointestinal examination did not reveal any

1. Professor, Department of Medicine, Government Medical College/Guru Nanak Dev Hospital, Amritsar, India-143001

2. Junior Resident, Department of Medicine, Government Medical College/Guru Nanak Dev Hospital, Amritsar, India-143001

3. Consultant Gynaecologist, Civil Hospital, Fatehgarhsahib, Punjab, India-140406

Correspondence N. S. Neki, Email: drneki123@gmail.com

abnormality. MRI of cervical spine was normal with no significant cord compression effects. Routine blood counts and biochemistry with electrolytes were normal. CSF examination revealed albuminocytological dissociation with cell count of $5/\text{mm}^3$, protein of 106 mg/dL and glucose 71 mg/dL with serum glucose 106 mg/dL. Nerve conduction study of all four limbs revealed decreased nerve conduction velocities in upper and lower limbs along with conduction block in bilateral tibial nerves signifying an acquired demyelinating type of neuropathy. Diminished amplitude was also noted in few motor nerves signifying an axonal involvement. Thus a diagnosis of acquired predominantly demyelinating (with axonal) type of motor polyradiculoneuropathy was made.

Discussion

GBS is broadly classified on a pathologic basis into demyelinating and axonal forms. Axonal GBS has been subclassified into acute motor axonal neuropathy (AMAN) and acute motor and sensory axonal neuropathy (AMSAN). Although hyporeflexia or areflexia is a cardinal feature of GBS, preserved reflexes or hyperreflexia is not a finding inconsistent with GBS.³ Hyperreflexia seen in GBS is usually associated with antecedent *Campylobacter jejuni* infection with most patients having history of abdominal pain and diarrhea. Almost all of them have IgG anti-GM1 ganglioside antibodies although anti-Campylobacter jejuni antibodies are frequently negative.¹ These cases are usually clinically mild and bulbar/respiratory involvement is uncommon. The variants most commonly reported to be associated with retained or brisk reflexes are AMAN.⁴ Moreover, 48% of Chinese and 33% of Japanese patients with AMAN showed hyperreflexia in the recovery phase. Our patient also had prior history of diarrhea and no bulbar/autonomic involvement. Although preservation of reflexes may simply be due to sparing of the sensory afferent pathway, the occurrence of brisk reflexes suggests a central mechanism. It has been suggested that the most common sites of nerve involvement in patients with GBS are not randomly distributed throughout the nerve. The distal motor nerves, the proximal segments, and the sites prone to

compression are most vulnerable for involvement.⁵ Early in the disease, electrophysiologic abnormalities are often mild or nonspecific. Motor conduction blocks have been documented in only 2–15% of patients with GBS within 3 weeks from disease onset, and conduction block in intermediate nerve segments in the first days of the disease is uncommon.⁶ The presence of normal sensory nerve function rather than motor is required for tendon jerks. As tendon jerks are dependent on synchronized volley of impulses, a purely axonal lesion would preserve tendon jerks better than a demyelinating lesion.

Conclusion

GBS should be considered in patients with acute pure motor quadriplegia with normal or brisk reflexes even though as shown in the criteria for AIDP, hyporeflexia or areflexia is a diagnostic requirement. MRI cervical spine should be done to rule out any compressive myelopathy at cervical region. This case report is to increase the awareness of treating physicians and neurologists regarding normo/hyperreflexic variant of GBS.

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