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CASE REPORT

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Paraparetic Guillain-Barre syndrome: An uncommon diagnosis of acute flaccid paralysis of the lower limbs

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Abstract

Apart from the usual differentials of transverse myelitis and cord compression, paraparetic GBS should be considered when sudden, flaccid paralysis of the lower limbs occurs, as prompt diagnosis and management can minimize sequel and unnecessary procedures. We do report a case wherein we managed a similar situation without the use of an immunomodulatory therapy.

KEYWORDS

flaccid paralysis, neuropathy, paraparetic GBS

1 | INTRODUCTION

Paraparetic Guillain-Barre syndrome is an uncommon variant of GBS. 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 was suggestive of paraparetic GBS, which were further confirmed by CSF and electrophysiologic findings.

Globally, GBS is the most common cause of acute flaccid paralysis in the current era of near elimination of poliomyelitis.[1] GBS is typically characterized by acute ascending bilateral symmetric weakness of varying degrees in the limbs, but it can present in a variety of ways, ranging from symptoms limited to specific parts of the body known as the "GBS variant" or "topographic variant" to quadriparesis with autonomic and respiratory involvement.[2,3] The diagnosis is reached after careful consideration of the clinical history, cerebrospinal fluid examination, and electrodiagnostic tests. In certain situations, neuroimaging is required to rule out differentials. We herein present a case of paraparetic GBS in a teenage boy who was managed conservatively and recovered fully in one and a half months.

2 | CASE PRESENTATION

A 19-year-male student presented to our center with chief complaints of progressive weakness of the lower limbs for 2 days. After waking up in the morning, the patient noticed heaviness of his legs and had difficulty in lifting them. When he tried to wear slippers, there was loosening and difficulty holding them in feet. In the evening, his condition increased to the point that he needed the assistance of his parents to get up from the chair. There was no

facial weakness, dysarthria, dysphagia, or diplopia. There was further worsening in weakness of lower limbs the next day, prompting the support of parents while walking. However, there was no upper limb weakness, sensory symptoms, or bowel and bladder involvement. Ten days before the onset of limb weakness, he experienced rhinorrhoea and a slight fever for about three days. He had no previous history of similar weakness, back discomfort, weight loss, shortness of breath, recent trauma, surgery, bug bite, or medication history.

On arrival at the emergency department, the patient's Glasgow Coma Scale score 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 the bulk of the limb muscles; tone was reduced in bilateral ankle and knees, normal in the rest of the joints. Power was 5/5 across all the joints in upper limbs whereas lower limb examination showed bilateral hip flexion 4/5, hip extension 3/5, knee flexion 4/5, knee extension 4/5, ankle dorsiflexion 2/5, and plantar flexion 3/5. Reflex was 2+ in bilateral biceps, triceps, and supinators. Left knee and ankle reflex were 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 percent, lymphocyte 30 percent, monocytes 8 percent, eosinophil 4 percent, and basophils 1 percent), hemoglobin 16 gm/dl, erythrocyte sedimentation rate 25 mm/h, random blood 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 percent lymphocytes, glucose 86 mg/dl, and protein 68 mg/dl. Chest X-ray and electrocardiogram findings were normal. Laboratory results including human immunodeficiency virus (HIV), 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 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 (Table 1) and preserved sensory amplitudes in both upper and lower limbs (Table 2).

We opted not to initiate immunomodulatory medication considering the degree of his disability and instead managed him conservatively with physiotherapy sessions. After 7 days, he was discharged with moderate improvement in motor power: bilateral hip flexion 4+/5, hip extension 4/5, ankle dorsiflexion 3/5, and rest were comparable to admission. On follow-up at 1.5 months, the power was 5/5 in the lower limbs with no residual deficits.

3 | DISCUSSION

The clinical spectrum of GBS encompasses Classical GBS, Miller Fisher variant, the Pharyngeal-Cervical-Brachial 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] Uncommon variants are usually diagnosed late and likely to land in the emergency or Intensive Care Units as full-blown GBS with respiratory failure and autonomic dysfunction.[1] The paraparetic variant of GBS is an uncommon variant with weakness 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 is no specific

TABLE 1 Nerve conduction study: Motor

	Latency (ms)		Amplitude (mv)			
Nerve	D	P	D	P	NCV (m/s)	F-Min (ms)
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

Abbreviations: 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

Abbreviations: Rt., right; Lt., left; NCV, nerve conduction velocity.

statistics on how frequent the paraparetic variety is, although several retrospective and prospective studies have indicated that it accounts for 1–8 percent of overall GBS cases.[1,6-8] Based on the existing data, it is evident that paraparetic GBS is not an usual variant, and we hope that our case contributes to the understanding of the diagnosis and management of this unusual variant.

With the patient's aforementioned presentation to us, typical differentials that came to mind were transverse myelitis and cauda equine neuritis. They were ruled out based on the 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, and intramedullary primary spinal cord tumor were excluded on the basis of absence of corresponding findings on spinal MRI. Infection (Cytomegalovirus, HIV), inflammation (eg, sarcoidosis), and endocrine (diabetes mellitus, hypothyroidism) conditions were ruled out based on laboratory and CSF findings.

In our case, a paraparetic variant of GBS is confirmed by clinical symptoms, characteristic disease progression and improvement, CSF findings, electrodiagnostic testing and ruling out focal lesions by neuroimaging. There was also a history of an antecedent upper respiratory tract viral infection, which has been documented in around 25 percent of overall paraparetic GBS cases.[7] Our patient had an intact sensory system, deep tendon reflexes, and NCT of the upper limbs throughout the course of illness. However, Berg et al reported sensory abnormalities or loss/absence of reflexes in the upper limbs or abnormal NCT in the upper limbs in 98 percent of 40 paraparetic GBS. One limitation could be that we were unable to do serial NCT in our patient.[5] In a study by Hiew et al, around 75 percent of paraparetic individuals exhibited an axonal pattern in NCT, similar to our case.[7] In a study by Berg et al, over 88 percent of total paraparetic GBS patients required therapy with intravenous immunoglobulin or plasmaphereis. Our patient, on the contrary, was not initiated on any immunomodulatory agents. In their study, patients with paraparesis had a better prognosis than those with quadriparesis after a 6-month follow-up.[5] Our patient likewise recovered fully without any impairments after 1.5 months of symptoms without the use of any immunomodulatory drugs.

4 | CONCLUSION

Paraparetic GBS is an uncommon manifestation that should be considered in the differential diagnosis of a patient with acute flaccid paralysis. Before a diagnosis of paraparetic GBS is established, common causes must be ruled out. Early detection and monitoring are crucial since some individuals may 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, and manuscript revision. RO involved in writing the manuscript concept, collection of case information, and manuscript revision. NN, NA, and SP participated in preparing a literature review and interpretation of clinical findings. BPG, RK, RR, NG, and AS involved in patient care team and collection of case information. All authors approved the final version.

ETHICS APPROVAL
I attest that my article submitted to Clinical Case Reports Journal. (1) This report has not been published in its entirety or in part anywhere else; (2) The manuscript is not currently being considered for publication in another journal; (3) I was personally and actively involved in the substantive effort that resulted in the revised manuscript, and they will be jointly and individually accountable for its content.

DATA AVAILABILITY STATEMENT
Data available on request from the authors

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REFERENCES
1. McGrogan A, Madle GC, Seaman HE, de Vries CS. The epidemiology of Guillain-Barré syndrome worldwide. A systematic literature review. *Neuroepidemiology*. 2009;32(2):150-163. doi:10.1159/000184748

2. Aljaafari D, Ishaque N. Paraparetic Variant of Guillain-Barré Syndrome in First 24 Hours of Postpartum Period: A case report. *Sultan Qaboos University Med J*. 2020;20(2):227-230. doi:10.18295/squmj.2020.20.02.015
3. Dimachkie MM, Barohn RJ. Guillain-Barré syndrome and variants. *Neurol Clin*. 2013;31(2):491-510. doi:10.1016/j.ncl.2013.01.005
4. Progress M, Yuki N, Hartung H. Guillain–Barré Syndrome; 2012.
5. van den Berg B, Fokke C, Drenthen J, van Doorn PA, Jacobs BC. Paraparetic Guillain-Barré syndrome. *Neurology*. 2014;82(22):1984-1989. doi:10.1212/WNL.0000000000000481
6. Ropper AH. Unusual clinical variants and signs in Guillain-Barré syndrome. *Arch Neurol*. 1986;43(11):1150-1152. doi:10.1001/archneur.1986.00520110044012
7. Hiew FL, Ramlan R, Viswanathan S, Puvanarajah S. Guillain-Barré Syndrome, variants & forms fruste: Reclassification with new criteria. *Clin. Neurol. Neurosurg*. 2017;158:114-118. doi:10.1016/j.clineuro.2017.05.006
8. Pokalkar D, Narisetty V, Polusani R, Kamera S, Poosarla SSL. A study on topographic variants of Guillain-Barré Syndrome in a tertiary care hospital in South India. *IAIM*. 2015;2:32-38.

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