VOLUME - 14, ISSUE - 01, JANUARY - 2025 • PRINT ISSN No. 2277 - 8160 • DOI : 10.36106/gjra

Original Research Paper

Internal Medicine

ATYPICAL PRESENTATION OF GUILLAINE BARRE SYNDROME - EARLY BLADDER AND BOWEL INVOLVEMENT.

Dr. Mohit Relwani	3rd Year M.D. Medicine
Dr. Manisha Panchal	Associate Professor, M.D. Medicine
Dr. Manthan Modi	2nd Year M.D. Medicine

ABSTRACT Guillain-Barré Syndrome is an acute, autoimmune-mediated polyradiculoneuropathy characterized by progressive motor paralysis, sensory disturbances, and autonomic dysfunction. While bladder and bowel involvement are uncommon in classic GBS presentations, their early and pronounced presence can signify an atypical variant, posing diagnostic challenges. This report details the case of a 57-year-old male who presented with fever history of AGE 10-15 days back followed by progressive limb weakness, urinary and fecal incontinence, and lower back pain. Diagnostic evaluation, including cerebrospinal fluid analysis and nerve conduction studies, confirmed GBS. Imaging studies excluded structural lesions, and the patient demonstrated significant improvement following intravenous immunoglobulin (IVIG) therapy. This case underscores the importance of recognizing atypical manifestations to ensure timely diagnosis and treatment. Increased awareness of such presentations can aid in differentiating GBS from other neuromuscular and spinal pathologies, optimizing patient outcomes.

KEYWORDS : Guillain-Barré Syndrome, atypical presentation, bladder dysfunction, polyradiculoneuropathy, autonomic dysfunction

INTRODUCTION

Guillain-Barré Syndrome (GBS) is a rare but potentially life-threatening disorder characterized by acute immune-mediated damage to peripheral nerves. While classic GBS involves ascending motor paralysis, areflexia, and mild sensory symptoms, atypical features, such as prominent bladder and bowel dysfunction, can complicate diagnosis. GBS is often triggered by infections or, less commonly, vaccinations, with the immune response directed against nerve gangliosides via molecular mimicry (Hughes et al., 2021).

This report describes an atypical case of GBS in a 57-year-old male, focusing on early bladder and bowel involvement.

Case Report

A 57-year-old male auto driver with no prior medical history presented with complaint of loose stools 10-15 days back followed by fever and chills lasting two days, followed by nausea ,vomiting Over the subsequent days, he developed bilateral lower and upper limb tingling, progressive weakness in the lower limbs, and urinary and fecal incontinence. He also reported lower back pain. There was no history of slurred speech, altered sensorium, breathlessness, or other neurological symptoms.

Clinical Examination

On examination, the patient was vitally stable and fully oriented.

Hughes Disability Score : 4

Neurological Findings

- Reduced tone and power (2/5) in the lower limbs (Flaccid weakness)
- Absent deep tendon reflexes in the lower extremities
- Preserved cranial nerve function.
- Sensory examination was normal.
- Babinski's sign was absent.

Investigations:

Hb	14.5
TC	9600
PC	298000
CREAT	0.80
NA+	136
K+	4.70
UREA	21.40
T. BIL	0.80
ALT	40.0
ALB	4.30
CRP	4.5
INR	0.83
Hba1c	5.4
CRP	NEGATIVE
HIV	NON REACTIVE
HBSAG	NON REACTIVE
HCV	NON REACTIVE

USG APK : Kidneys : normal , Urinary bladder : overdistended (600 ml) Prostate : normal (LMN BLADDER)

Cerebrospinal Fluid (CSF): Analysis showed elevated protein (202.5 mg/dL) with normal cell count, consistent with albuminocytologic dissociation.

MRI Lumbar Spine: Mild enhancement of nerve roots and cauda equina at D12–L2, suggestive of inflammatory changes without compression.

Nerve Conduction Studies (NCS):

- Decreased conduction velocity
- Increased distal latency
- F wave latency prolonged
- Sural nerve sparing

Findings indicated a predominantly demyelinating pattern of pure motor polyradiculoneuropathy, consistent with GBS.

Differential Diagnosis

Inflammatory : transverse myelitis, acute CIDP, Vasculitis.
Metabolic neuropathies : Diabetes, Porphyria
Pure motor : botulism, NMJ disorders
Infectious : HIV
However, imaging and laboratory findings supported the diagnosis of GBS.
Brighton's Criteria : LEVEL 1

Treatment And Outcome

The patient received IVIG (2 g/kg over five days). Significant clinical improvement was observed, with increased lower limb strength (4+), resolution of pain, and improved respiratory effort. Urinary and fecal incontinence also resolved.

HUGHES DISABILITY SCORE improved from 4 (on admission) to 2 (able to walk 10m or more without support

DISCUSSION

Overview of GBS

GBS encompasses a spectrum of autoimmune neuropathies, with acute inflammatory demyelinating polyneuropathy (AIDP) being the most common variant.

Others acute motor axonal neuropathy (AMAN), acute motor-sensory axonal neuropathy (AMSAN), Miller Fisher Syndrome (MFS) (Hughes et al., 2021).

Also Pharyngocervicobrachial variant, multiple cranial nerve variant, pandysautonomic variant etc.

The pathogenesis involves immune-mediated damage to myelin or axons, often triggered by antecedent infections such as Campylobacter jejuni, cytomegalovirus, or Zika virus.

Atypical Features in GBS

Bladder and bowel dysfunction are rare in GBS, typically appearing in severe or atypical cases. These symptoms may result from:

Autonomic Nervous System Dysfunction: Parasympathetic control of the bladder via sacral spinal segments (S2–S4) is impaired, causing detrusor areflexia and overflow incontinence.

Somatic Nerve Involvement: Pudendal nerve dysfunction affects sphincter control.

Spinal Shock-Like Phenomenon: Diffuse nerve inflammation can lead to temporary loss of bladder control (Walgaard et al., 2011).

Pain, although common in GBS, often manifests as back or limb pain and rarely contributes to initial diagnostic confusion. Early recognition of atypical features is crucial to avoid misdiagnosis, particularly in conditions mimicking GBS, such as transverse myelitis or metabolic neuropathies.

Diagnostic Challenges

The presence of urinary and fecal incontinence necessitated exclusion of structural spinal cord lesions, metabolic disorders, and infections. Imaging and CSF analysis were pivotal in confirming GBS. Electrodiagnostic studies further supported the diagnosis, highlighting the role of multimodal investigation in atypical cases.

Management

IVIG and plasma exchange are the mainstays of treatment, with early initiation significantly improving outcomes. Corticosteroids, though less effective in classic GBS, may have a role in addressing associated inflammatory changes (Hughes et al., 2021).

Prognosis

Most patients with GBS achieve significant recovery within months, although residual deficits may persist in severe cases. Early recognition and intervention are critical in optimizing functional outcomes, particularly in atypical presentations.

CONCLUSION

This case illustrates the diagnostic complexity of atypical GBS presentations, such as prominent bladder and bowel involvement. While rare, these features highlight the diverse manifestations of GBS and the importance of a thorough clinical and diagnostic evaluation. Increased awareness of atypical forms can aid in timely differentiation from other neuromuscular and spinal conditions.

REFERENCES

1. Hughes, R. A., Swan, A. V., van Doorn, P. A. (2021). Intravenous immunoglobulin for Guillain-Barré syndrome. Cochrane Database of Systematic Reviews, 2021(1). https://doi.org/10.1002/14651858.Cd002063.pub4
2. Willison, H. J., Jacobs, B. C., & van Doorn, P. A. (2016). Guillain-Barré syndrome. The Lancet, 388(10045), 717–727. https://doi.org/10.1016/S0140-6736(16)00339-1
3. Walgaard, C., Lingsma, H. F., Ruts, L., van Doorn, P. A., & Jacobs, B. C. (2011). Early recognition of poor prognosis in Guillain-Barré syndrome. Neurology, 76(11), 968–975. https://doi.org/10.1212/WNL.0b013e3182104407