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Guillain-Barré Syndrome Presenting as Pseudo-Cerebellar Syndrome: A Case Report from a Resource-Limited Setting in Sub-Saharan Africa

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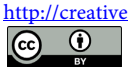

Abstract

Guillain-Barré Syndrome (GBS) is the most frequent cause of acute flaccid paralysis worldwide, with significant morbidity and mortality in the absence of timely treatment. It is an immune-mediated acute inflammatory polyradiculoneuropathy, usually triggered by infection. While the classical presentation is ascending symmetrical weakness with areflexia, atypical forms may mimic other neurological disorders and delay diagnosis. We report a rare pseudo-cerebellar presentation in a middle-aged man in a low-resource setting, highlighting diagnostic challenges and the importance of multidisciplinary supportive care. A 52-year-old teacher with no medical history presented with a 4-day history of progressive gait unsteadiness resembling cerebellar dysfunction. Neurological examination revealed global areflexia, wide-based stance, but preserved cognition and coordination tests. Brain and spine MRI were unremarkable. By day 7, cerebrospinal fluid (CSF) analysis showed albuminocytologic dissociation. On day 10, nerve conduction studies confirmed diffuse demyelination consistent with the acute inflammatory demyelinating polyradiculoneuropathy (AIDP) subtype of GBS. The patient subsequently developed ascending flaccid paraparesis with mild autonomic dysfunction, without respiratory compromise. Due to unavailability of intravenous immunoglobulin (IVIg) and plasmapheresis, treatment was limited to supportive care, physiotherapy, and preventive measures against complications. The patient showed gradual recovery over four weeks, regaining partial autonomy, and was referred for rehabilitation. This case underscores the diagnostic complexity of GBS variants that mimic central nervous system pathology. In resource-limited settings, reliance on clinical skills re-

mains crucial, especially where access to neurophysiology, CSF analysis, and disease-modifying therapies is limited. Despite the lack of specific treatment, supportive multidisciplinary management enabled significant recovery. Wider access to IVIg and plasmapheresis is urgently needed in low-income countries.

Subject Areas

Allergy & Clinical Immunology, Neurology

Keywords

Guillain-Barré Syndrome, Pseudo-Cerebellar Gait, Acute Polyradiculoneuropathy, Case Report, Low-Resource Settings

1. Introduction

Guillain-Barré Syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy that represents the most common cause of acute flaccid paralysis globally [1]. First described in 1916 by French neurologists Georges Guillain, Jean Barré, and André Strohl, it is characterized by rapidly progressive symmetrical weakness, areflexia, and variable sensory and autonomic dysfunction. Its annual incidence ranges from 0.81 to 1.89 per 100,000 population worldwide, with slightly higher rates in males and older adults [2] [3].

Pathophysiologically, GBS is thought to result from aberrant immune responses targeting peripheral nerve components, often triggered by preceding infections such as *Campylobacter jejuni*, cytomegalovirus, Epstein-Barr virus, or more recently, SARS-CoV-2 [4] [5]. The immune-mediated attack results in demyelination, axonal degeneration, or a combination of both, leading to impaired nerve conduction.

The classical clinical course involves progressive symmetrical weakness starting in the lower limbs and ascending proximally, accompanied by generalized areflexia. However, numerous variants have been described, including the Miller Fisher syndrome, pharyngeal-cervical-brachial weakness, paraparetic forms, and pure sensory variants [6]. These atypical presentations often complicate early diagnosis, particularly in settings with limited access to neuroimaging and electrophysiological studies.

Among the rarest variants is the pseudo-cerebellar presentation, in which patients initially manifest with gait ataxia mimicking cerebellar pathology. This form is challenging to recognize as central causes, including ischemic stroke, demyelinating diseases, and space-occupying lesions, are frequently suspected first. Misdiagnosis can lead to unnecessary investigations and delayed initiation of appropriate care [7].

The diagnostic process for GBS typically combines clinical assessment, cerebrospinal fluid (CSF) analysis showing albuminocytologic dissociation (elevated protein without pleocytosis), and nerve conduction studies demonstrating demyelination or axonal involvement [8]. However, in resource-constrained environ-

ments such as sub-Saharan Africa, these investigations may not be readily available, and diagnosis relies heavily on clinical acumen [9].

Treatment of GBS has been revolutionized by intravenous immunoglobulin (IVIg) and plasmapheresis, which significantly reduce disease progression and improve outcomes if administered early [10]. Unfortunately, in low-income settings, these therapies remain prohibitively expensive and inaccessible to most patients. Consequently, supportive management—including prevention of complications, physiotherapy, and multidisciplinary care—remains the cornerstone of treatment.

This report describes an unusual case of GBS with pseudo-cerebellar presentation in a 52-year-old male, managed at the Ignace Deen University Hospital in Guinea. We highlight the diagnostic challenges in differentiating GBS variants from central neurological pathologies, the difficulties of management in resource-limited settings, and the importance of supportive care. Additionally, we provide a brief review of recent literature (2020-2024) on atypical presentations and management strategies for GBS.

2. Case Presentation

2.1. Patient Information

A 52-year-old right-handed male teacher, with no significant medical history, presented to the neurology department of CHU Ignace Deen with a four-day history of progressive unsteadiness while walking. He denied fever, headache, nausea, vomiting, visual disturbance, dysarthria, or recent infections. There was no history of diabetes, hypertension, toxic exposure, alcohol misuse, or medication intake. Family history was unremarkable for neurological or autoimmune disease.

2.2. Clinical Findings at Admission (Day 1)

On neurological examination, the patient was alert, oriented, and cognitively intact. Gait assessment revealed marked unsteadiness with a broad-based stance, closely resembling cerebellar ataxia. However, coordination tests including finger-to-nose and heel-to-knee maneuvers were negative, arguing against true cerebellar dysfunction. Global areflexia was noted in both upper and lower extremities. No motor weakness, sensory loss, cranial nerve involvement, or sphincter disturbances were detected at this stage. Cardiovascular and respiratory examinations were normal.

The apparent discrepancy between the pseudo-cerebellar gait and preserved coordination tests initially raised suspicion for a central nervous system disorder such as cerebellar infarction, demyelinating lesion, or posterior fossa tumor.

2.3. Investigations

- **Magnetic Resonance Imaging (MRI):** Brain and spinal cord MRI performed on day 2 revealed no ischemic, hemorrhagic, demyelinating, or compressive lesions.
- **Routine Blood Tests:** Complete blood count, electrolytes, liver and renal func-

tion tests were all within normal limits.

- **Cerebrospinal Fluid (CSF) Analysis:** Lumbar puncture performed on day 7 revealed clear CSF with elevated protein (1.25 g/L) and absence of pleocytosis, consistent with albuminocytologic dissociation.
- **Nerve Conduction Studies (Day 10):** Electrophysiological examination demonstrated diffuse demyelination with slowed conduction velocities, prolonged distal latencies, and conduction block. These findings confirmed the diagnosis of the acute inflammatory demyelinating polyradiculoneuropathy (AIDP) subtype of GBS [3].

2.4. Clinical Evolution

The clinical course was characterized by progressive neurological decline:

- **Day 5:** Emerging distal weakness in the lower limbs, though mild at this stage.
- **Day 7 - 14:** Development of ascending flaccid paraparesis with global areflexia. Mild autonomic manifestations were noted, including constipation and orthostatic hypotension. Importantly, the patient did not develop respiratory distress, cranial nerve involvement, or bulbar weakness.
- **Day 14 - 28:** Gradual recovery phase, with proximal upper limb strength improving to Medical Research Council (MRC) grade 3/5 and distal strength 4/5 by the third week. By the fourth week, the patient achieved partial autonomy, including assisted standing and bed-to-chair transfers.

2.5. Therapeutic Interventions

Due to the unavailability of IVIg and plasmapheresis—both considered standard of care in GBS—the patient was managed with supportive measures only:

- **Hospital admission with close monitoring** for respiratory function and autonomic instability.
- **Preventive care:** pressure sore prevention, urinary tract infection prophylaxis, and deep vein thrombosis (DVT) prevention with compression stockings.
- **Daily physiotherapy** including passive and active mobilization, posture correction, and gait training.
- **Supportive treatment:** adequate hydration, analgesics for neuropathic pain, and laxatives for constipation.

2.6. Outcome and Follow-Up

At four weeks, the patient regained partial independence with marked functional improvement. He was subsequently referred to a rehabilitation center for long-term physiotherapy and occupational therapy. Written informed consent for publication of the case was obtained.

3. Discussion

3.1. Epidemiology and Global Burden

Guillain-Barré syndrome (GBS) remains the most frequent cause of acute flaccid

paralysis worldwide, surpassing poliomyelitis since the near-eradication of the latter [1]. The estimated global incidence is 1 - 2 cases per 100,000 inhabitants annually, with a slight male predominance and increasing risk with age [2]. In sub-Saharan Africa, epidemiological data are scarce, but available studies suggest a comparable or even higher incidence, likely underreported due to limited surveillance systems and diagnostic capacity [3].

In low-resource settings, the true burden of GBS is further amplified by diagnostic delays and the lack of access to disease-modifying therapies such as intravenous immunoglobulin (IVIg) or plasma exchange. Consequently, case fatality rates in these regions remain significantly higher (up to 15% - 20%) compared to high-income countries where mortality is usually below 5% [4] [5].

3.2. Clinical Spectrum and Atypical Presentations

GBS is traditionally characterized by acute ascending symmetrical weakness, areflexia, and variable sensory or autonomic dysfunction [6]. However, atypical variants are increasingly recognized, complicating early diagnosis. These include pure motor forms, Miller-Fisher syndrome (ophthalmoplegia, ataxia, areflexia), pharyngeal-cervical-brachial variants, and presentations mimicking central nervous system (CNS) disorders.

Our patient presented with an unusual pseudo-cerebellar gait without initial weakness or sensory changes. Such a presentation is rare but has been described in recent literature [7]. The ataxia in GBS is thought to arise not from cerebellar pathology but from impaired proprioceptive input secondary to large fiber involvement. This highlights the importance of distinguishing pseudo-cerebellar ataxia from true cerebellar dysfunction, especially when neuroimaging is unremarkable.

3.3. Diagnostic Challenges in Resource-Limited Settings

The diagnosis of GBS requires integration of clinical, biological, and electrophysiological findings. The classic triad includes:

1) Progressive weakness of more than one limb.

2) Generalized areflexia.

3) Albuminocytologic dissociation in the CSF.

In our case, all three criteria were eventually fulfilled, but only after more than one week of evolution. Access to MRI, lumbar puncture, and nerve conduction studies is often delayed in sub-Saharan Africa due to financial constraints and infrastructural limitations [8]. As in this report, such delays can mislead clinicians toward central etiologies, particularly when the presentation is atypical.

Recent guidelines stress the role of clinical acumen in low-resource contexts. Early recognition of areflexia, symmetrical progression, and lack of cerebellar signs (despite apparent ataxia) should alert clinicians to a peripheral process [9]. This case underlines the critical need for improved training and diagnostic pathways in African neurology centers.

3.4. Management and Therapeutic Limitations

Standard therapy for GBS includes IVIg (0.4 g/kg/day for 5 days) or plasma exchange (4 - 6 sessions over 2 weeks), both of which have been shown to reduce disease progression, shorten time to recovery, and decrease risk of respiratory failure [10] [11]. Unfortunately, neither treatment was available for this patient due to cost and resource constraints.

Instead, he was managed conservatively with multidisciplinary supportive care. Preventive nursing measures, intensive physiotherapy, and complication surveillance (DVT, respiratory failure, infections) were essential in facilitating recovery.

While IVIg and plasma exchange remain unattainable for many African patients, recent studies suggest potential adjuncts. Corticosteroids, once considered ineffective, may provide some benefit in specific subgroups when combined with rehabilitation [12]. Additionally, early mobilization and neurorehabilitation are increasingly recognized as crucial determinants of long-term functional outcome, even in the absence of immunotherapy [13].

3.5. Prognosis and Outcomes

Prognosis in GBS is influenced by several factors: age, rapidity of progression, requirement for mechanical ventilation, and access to immunotherapy [14]. In our patient, the absence of bulbar or respiratory involvement was a favorable prognostic marker. Despite lack of disease-modifying therapy, he regained partial autonomy within four weeks, underscoring the value of supportive care.

However, functional recovery in GBS is often prolonged, with up to 20% of patients experiencing persistent disability at one year [15]. Fatigue, neuropathic pain, and psychological sequelae are frequent long-term complications that require ongoing multidisciplinary management.

3.6. Lessons for Clinical Practice

This case highlights several important clinical lessons:

- **GBS can mimic central nervous system disorders** such as cerebellar disease, particularly through pseudo-ataxia.
- **Diagnostic vigilance** is crucial in resource-limited settings where access to confirmatory tests is delayed.
- **Supportive care and rehabilitation** can significantly improve outcomes even when IVIg or plasmapheresis are unavailable.
- **Health policy implications:** Wider access to affordable immunotherapy remains an urgent priority in low-income countries.

3.7. Recent Advances in GBS Research (2020-2024)

Several recent studies have advanced our understanding of GBS:

- **Immunopathogenesis:** Molecular mimicry between microbial antigens and peripheral nerve gangliosides remains central to GBS pathogenesis. New in-

sights from SARS-CoV-2-associated GBS suggest that viral infections may trigger autoimmune responses even without direct neurotropism [16].

- **COVID-19 and GBS:** A systematic review in 2021 reported over 300 cases of post-COVID GBS worldwide, reinforcing the link between emerging infections and GBS [17].
- **Biomarkers:** Neurofilament light chain (NfL) levels in CSF and serum are being investigated as prognostic biomarkers for axonal damage and long-term disability [18].
- **Novel therapies:** Clinical trials are ongoing to assess complement inhibitors (e.g., eculizumab, zilucoplan) as potential disease-modifying agents [19]. If proven effective, these may revolutionize GBS treatment, although affordability remains a challenge in low-resource regions.
- **Rehabilitation:** Recent data confirm that early intensive physiotherapy significantly improves mobility and quality of life in GBS survivors, even in the absence of IVIg [20].

4. Conclusions

Guillain-Barré syndrome (GBS) remains a major cause of acute flaccid paralysis worldwide, with substantial morbidity and mortality in regions where access to diagnosis and therapy is limited. This case illustrates the diagnostic complexity of atypical GBS presentations, particularly pseudo-cerebellar ataxia, which can mimic central nervous system disease and delay appropriate management. Despite the absence of immunomodulatory treatment, our patient demonstrated functional recovery with intensive supportive care and rehabilitation.

This report underscores the need for:

1) Increased awareness among clinicians of atypical GBS variants to facilitate earlier recognition and diagnosis.

2) Strengthening diagnostic capacity in resource-limited settings, including access to neurophysiology and CSF analysis.

3) Equitable access to IVIg and plasma exchange, which remain the standard of care but are largely unavailable in low-income countries due to cost and infrastructure barriers.

4) Integration of multidisciplinary supportive care, including physiotherapy, nursing, and rehabilitation, as an essential pillar of management when specific therapies are not accessible.

Recent advances (2020-2024) in biomarker research, immunotherapy development, and rehabilitation strategies offer hope for improved outcomes. However, their translation to low-resource contexts will depend on global health initiatives addressing disparities in neurological care.

Ultimately, this case highlights the resilience of patients and clinicians in navigating diagnostic and therapeutic challenges, while reinforcing the urgent need for systemic solutions to improve neurological care in sub-Saharan Africa and beyond.

5. Learning Points

- **GBS may mimic cerebellar syndromes**, particularly through pseudo-cerebellar ataxia, complicating diagnosis.
- **Clinical acumen remains central** in resource-limited settings where access to MRI, CSF analysis, and EMG is often delayed.
- **Supportive and rehabilitative care can significantly improve outcomes**, even in the absence of IVIg or plasmapheresis.
- **Multidisciplinary collaboration** (neurology, rehabilitation, nursing) is critical to optimize recovery.
- **Health policy and advocacy are needed** to ensure equitable access to essential neurological therapies.

Conflicts of Interest

The authors declare no conflicts of interest.

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