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Case Series

Clinical spectrum and treatment outcomes in variants of Guillain-Barré syndrome: a case series

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ABSTRACT

Guillain-Barré syndrome (GBS) is an autoimmune polyradiculoneuropathy that is acute, typically severe, and fulminant. GBS has an incidence of 0.81-1.89 (median 1.11) per 100,000 person-years, and men are slightly more susceptible to GBS than females. 70% of individuals acquire this acute flaccid paralysis condition within 1-4 weeks following a respiratory infection or diarrhoea (especially *Campylobacter jejuni*). There are several identified subtypes of GBS, with acute inflammatory demyelinating polyneuropathy (AIDP) being the most prevalent. Additionally, there are two "axonal" subtypes: acute motor axonal neuropathy (AMAN) and acute motor sensory axonal neuropathy (AMSAN), both of which are clinically severe. The clinical trial of ophthalmoplegia, ataxia, and areflexia characterizes a different subtype called Miller Fisher syndrome (MFS) linked to anti-GQ1b antibodies. The patient's medical history, along with neurological, electrophysiological, and cerebrospinal fluid tests, are used to diagnose GBS. Intravenous immunoglobulin (IVIG) and plasma exchange are effective treatments; however, newer approaches are required because 25% of patients eventually need mechanical ventilation, 20% are unable to walk, and 2-5% of patients may experience relapses.

Keywords: GBS, AIDP, AMAN, AMSAN, MFS, IVIG

INTRODUCTION

Guillain-Barré syndrome (GBS) is a heterogeneous disease defined by rapidly progressing symmetrical limb weakness with hyporeflexia or areflexia; sensory abnormalities and cranial nerve involvement occur in some patients. The pathophysiology of GBS involves molecular mimicry, antiganglioside antibodies, and probably complement activation. Based on the clinical picture, electrophysiological results, and CSF findings, GBS subtypes and variations are categorized. In Europe and North America, acute inflammatory polyneuropathy is most commonly demyelinating AIDP. Axonal variants, both motor AMAN or sensorimotor AMSAN, are more common in China and Japan. In Western countries, the MFS accounts for 6% of all GBS cases.[1] Currently, the most effective therapies for GBS are plasma exchange and IVIG.[2] However, many individuals continue to have significant disease progression, pain, and residual deficits despite these therapeutic alternatives.

CASE SERIES

Case 1

A 17-year-old male with no known past medical history presented with a 5-day history of progressive weakness of both upper and lower limbs. He reported difficulty in walking and episodes of knee buckling, requiring support for ambulation. Before the weakness, he experienced a 1-week illness characterized by high-grade fever, cough, and minimal mucoid sputum production. There was no

history of diarrhea, vomiting, or immunization. Cranial nerves were intact. Neurological examination revealed acute flaccid areflexic quadriparesis, bilateral symmetry, and a distal-to-proximal ascending pattern of weakness. Nerve conduction studies confirmed the diagnosis of the AMAN variant of GBS, demonstrating reduced compound muscle action potential (CMAP) amplitudes with preserved sensory nerve action potentials (SNAPs). The patient was admitted to the ICU and promptly initiated IVIG therapy to regulate the immune response. Additionally, we commenced a tailored physiotherapy program to promote muscle recovery and prevent complications. Due to his young age, the patient demonstrated an exceptional response to the treatment regimen. His journey towards recovery was marked by a steady and remarkable improvement in his muscle strength. Each week brought tangible progress as the once-weakened muscles regained their power and function. After a ten-day stay in the hospital, where he received round-the-clock care and intensive therapy, the patient was deemed fit for discharge.

Case 2

A 17-year-old male with a history of GBS at 30 months old presented with weakness in all four limbs. He had a history of difficulty in squatting and bilateral foot deformities since childhood. For two days before presentation, he experienced fever and loose stools (2-3 episodes per day), followed by progressive weakness in both upper and lower limbs, initially affecting proximal and later distal muscles. Flailness also developed in all limbs. On examination, bilateral facial weakness was observed. After 2 days, neck and trunk muscle weakness developed, as evidenced by the inability to turn around the bed and the difficulty in lifting the neck from the pillow. His clinical presentation showed a rapid and significant deterioration within 24 hours. He exhibited acute flaccid areflexic quadriparesis, which progressed to quadriplegia. Nerve conduction studies revealed severe motor axonal neuropathy with normal SNAPs and reduced CMAP amplitude. These clinical and electrophysiological features suggested an AMAN variant. The patient experienced respiratory failure, with a respiratory rate of 34/min, SpO_2 at 93%, and SBC less than 5 seconds. Consequently, the patient underwent intubation and was placed on mechanical ventilation. The patient was promptly admitted to the intensive care unit and initiated on IVIG and DVT prophylaxis, and underwent limb and chest physiotherapy. Following IVIG, the patient's clinical course showed gradual improvement, but impending respiratory failure necessitated intubation. While the patient had made promising progress in resolving the symptoms after a month of treatment, significant challenges remain. Although we successfully extubated him, he is currently unable to walk or move around independently due to limited motor improvement. This progress needs to be evaluated in the context of the specific severity and potential for recurrence associated with the AMAN variant of GBS the patient was diagnosed with. A deeper comprehension of the underlying factors impacting recovery is essential for determining the best course of action moving forward.

Case 3

A 31-year-old male with a history of occasional alcohol consumption and no prior medical conditions presented with a cascade of debilitating symptoms. It began with a 3-day history of loose stools (4 episodes/day), followed by generalized body pain for 3 days. Over the next 2 days, he developed progressive numbness and weakness in both upper and lower limbs. The weakness initially began in the distal parts of both lower limbs and then progressed to both upper limbs within 2-3 days. There was no history of recent vaccinations. Upon neurological examination, the patient had hypotonia, global areflexia, and quadriparesis in all four limbs. This weakness worsened over time, eventually leading to complete paralysis of all four limbs. In subsequent days, he developed bilateral lower motor neuron facial weakness and neck muscle weakness. Nerve conduction studies revealed conduction block in multiple limbs (left peroneal, left tibial, right peroneal, and right ulnar) and prolonged distal latency in the left tibial nerve with an absent CMAP in the right tibial nerve. Sensory conduction studies were normal in all four limbs. These features, along with clinical correlation, suggested AIDP. In the neurological ICU setup, the patient was started on IVIG. Despite receiving IVIG therapy, further complications ensued on day three. He had respiratory failure, aspiration pneumonia, and autonomic dysfunction symptoms such as sinus tachycardia, fluctuating blood pressure, and infrequent episodes of profuse sweating. Facing impending respiratory failure (single breath count 7, respiratory rate 33/min), the patient was intubated and placed on mechanical ventilation. The patient was approached with multimodal, supportive treatment. Exhibiting signs of recovery and following improvement from respiratory failure, the patient successfully underwent a weaning process and was subsequently extubated. He is undergoing physiotherapy for his limbs and chest. This multifaceted approach has yielded impressive results, as the patient can now maintain an upright position unaided and even ambulate with support under the guidance of the rehabilitation team.

Case 4

A 48-year-old female with a known case of diabetes mellitus presented with a seven-day history of pricking sensations in bilateral lower and upper limbs, progressively leading to an inability to stand and walk for the past 4 days. The patient exhibited acute ascending weakness of all four limbs, along with non-radiating back pain of insidious onset and gradual progression. A low-grade fever for 2 days was reported before the onset of weakness. He had no history of diarrhea, vomiting, or vaccination. Upon examination, the patient had

hypertension (180-110 mmHg). Neurological examination revealed acute flaccid global areflexic quadriparesis with sensory loss and neck weakness. A nerve conduction study showed severe demyelinating changes in the bilateral median and tibial nerves. The clinical features, along with NCS findings, led to the diagnosis of GBS with the AIDP variant. The patient received a comprehensive treatment approach, including IVIG for immune modulation, with symptomatic relief provided. During the second week of illness, the patient developed an altered sensorium with significant hyponatremia (serum sodium of 116 mEq/L) secondary to SIADH (Syndrome of inappropriate antidiuretic hormone ADH release), a recognized complication of GBS. Correction of euvolemic hyponatremia involved free water restriction and administering 3% saline, while insulin addressed hyperglycemia. Hypertension was managed with amlodipine and enalapril. Limb and chest physiotherapy were incorporated to enhance recovery. The patient exhibited gradual improvement with the instituted management plan. The resolution of quadriparesis, sensory deficits, and improvement in reflexes were observed during the two months of hospitalization. Following this positive trajectory, the patient was eventually discharged.

Case 5

A 46-year-old female with a known case of diabetes mellitus presented with weakness in all four limbs for 25 days. The patient reported a single episode of loose stools, a day before the onset of weakness. She had no prior history of receiving any vaccinations. Neurological examination showed acute flaccid areflexic quadriparesis. Nerve conduction studies revealed a severe demyelinating neuropathy, characterized by prolonged distal motor latencies and reduced nerve conduction velocities, suggestive of AIDP with secondary axonal involvement. The patient was admitted to the ICU. The necessity for an immunosuppressant was discussed since it was pure motor involvement without further progression. The progression of motor weakness halted, and the disease had entered a plateau phase. Following the protocol, immunotherapy wasn't necessary during the plateau stage. Therefore, we focused on supportive care and physiotherapy to help the patient recover. The patient's ten-day hospital stay presented a positive outcome, marked by a gradual, spontaneous recovery of symptoms. This encouraging progress suggests the patient's body effectively mounted an immune response against the underlying cause of GBS, leading to a natural improvement in her condition. Her ability to move and perform daily activities had progressively improved, potentially culminating in a regained independence in tasks like walking or eating. Following this, the patient was deemed suitable for discharge with appropriate monitoring and follow-up. This case underscores that not all cases of GBS necessitate immunotherapy as a mandatory treatment.

Case 6

A 60-year-old woman with a known case of hypertension and type 2 diabetes mellitus presented with a one-week history of progressive weakness in bilateral upper and lower limbs, both proximal and distal, causing buckling of the knees while walking. In subsequent days, she noticed unsteadiness of gait and swaying while walking. There was also a history of truncal and neck muscle weakness. She experienced numbness in both hands up to the wrist. She had no history of diarrhea, fever, or vaccination. The patient's vital signs were normal except for high blood pressure (170/100 mmHg). Neurological examination revealed global areflexia, hypotonia, quadriparesis, and bilateral ptosis. Sensory testing showed decreased touch, pain, and temperature sensation in both hands up to the wrist. The cerebellar function was impaired. A gait examination revealed stance ataxia. Cerebrospinal fluid analysis showed elevated protein (45 mg/dl) with acellular count and negative cultures. Nerve conduction studies revealed sensory-motor axonal polyradiculoneuropathy with sensory predominance. The MRI brain scan was unremarkable. IVIG therapy was initiated immediately upon diagnosis by admission in a neurological ICU setup. The patient received antihypertensive medications (amlodipine and enalapril), atorvastatin for hyperlipidemia, and insulin for hyperglycemia. Chest and limb physiotherapy were also provided. With multifaceted treatment, the patient's symptoms improved significantly. The weakness gradually subsided, and she regained full muscle strength. Sensory deficits were also resolved completely. After achieving complete recovery, the patient was discharged after thirteen days of hospital stay.

Case 7

A 62-year-old male, who is a chronic smoker and a known case of hypertension, coronary artery disease, and recently diagnosed heart failure with an ejection fraction of 40%, presented with a two-day history of progressive weakness in both upper and lower limbs. He initially experienced difficulty in walking, which progressed to requiring support. Subsequently, he developed weakness in his hands and numbness in both soles and palms, with decreased touch and pain sensation. On probing the history, he had two days of fever and loose stools, with no prior vaccinations. On examination, the patient had a blood pressure of 170/100 mmHg and profuse sweating suggestive of autonomic dysfunction. The patient had hypotonia in bilateral lower limbs, absent deep tendon reflexes, and reduced sensory pain and touch in the fingers and soles. The cerebellar function was bilaterally impaired. Nerve conduction studies demonstrated reduced conduction velocity, increased distal latency, and absent SNAPs in the bilateral median and ulnar nerves, indicating sensorimotor demyelinating poly-radiculomyelopathy with bilateral sural nerve sparing. In combination with clinical presentation and nerve conduction studies, the patient was diagnosed with

AMSAN. An echocardiogram revealed ischemic heart disease, left ventricular segment hypokinesia, an ejection fraction of 40%, a mild murmur, and a sclerotic aortic valve. D-dimer test results were elevated at 1608 ng/ml. The patient's cardiac comorbidities, including CAD and HFrEF, complicated the overall clinical picture. He was promptly admitted to the ICU. Therapy selection was challenging in this case due to the interplay of autonomic dysfunction and underlying heart failure, requiring careful consideration of potential alternatives to IVIG. Plasmapheresis, another potential treatment, was also ruled out after considering the potential risks and benefits in light of the autonomic dysfunction. Following informed consent and clearance from a cardiologist, the patient received IVIG therapy. Concurrent administration of heparin was initiated for prophylactic management of potential embolic complications. Along with symptomatic relief measures, the patient received a comprehensive cardiac medication consisting of aspirin, metoprolol, clopidogrel, atorvastatin, and aldactone. Additionally, enalapril was given to control hypertension. The patient's journey towards recovery was intricate, marked by underlying cardiac conditions that added a layer of complexity to his overall clinical presentation. Recognizing the potential challenges posed by these comorbidities, we meticulously reviewed expert opinions and relevant medical literature to formulate the most effective treatment plan. This tailored approach proved successful, and the patient's condition began to show positive signs of improvement. Following a month-long stay in the hospital, during which his symptoms demonstrably improved, the patient was discharged ambulant without any complications.

Case 8

A 43-year-old female with no known comorbidities presented with complaints of a three-day history of progressive weakness in all four limbs, preceded by paraesthesia in both lower limbs (feet). Before the onset of weakness, she had a three-day history of low-grade fever and a two-day history of loose stools. There was no history of vaccination. The cranial nerve examination was normal. Neurological examination revealed acute flaccid areflexic quadriparesis, consistent with GBS. Nerve conduction studies indicated motor axonal neuropathy (AMAN) of bilateral median and ulnar nerves, with normal SNAPs and reduced CMAP amplitude. The results of the potassium test and the ECG were normal. Requiring immediate critical care, the patient was admitted to the ICU, and IVIG therapy was initiated to modulate the immune response. Additionally, a supportive pharmacological regimen was administered and tailored physiotherapy started to enhance recovery. The patient exhibited significant improvement in motor function and paraesthesia following the treatment. The patient exhibited outstanding progress, attaining a full recovery devoid of any unforeseen complications. Following a twelve-day hospital stay and a meticulous assessment, she was discharged with a rehabilitation plan.

Case 9

A 58-year-old female with a known case of hypertension and type 2 diabetes mellitus presented with weakness in both lower limbs for one week, followed by weakness in both upper limbs for six days. She had a two-day history of high-grade fever. Concurrent symptoms included numbness, paraesthesia, and a sensation of limb heaviness. There was no history of previous immunization. Neurological examination revealed acute flaccid areflexic quadriparesis. Sensory testing showed decreased vibration and joint position sensation in both lower limbs. Nerve conduction studies confirmed severe sensorimotor axonal neuropathy of bilateral upper and lower limbs with reduced CMAPs and absent SNAPs. This solidified the diagnosis of GBS with the AMSAN variant. The patient was admitted to the ICU and immediate treatment was initiated with IVIG, along with supportive medications for symptomatic relief. Amlodipine was administered for hypertension control, and insulin was used to manage hyper-glycemia. Physiotherapy was incorporated into the treatment plan to aid in the patient's recovery. Despite the patient having hypertension and diabetes mellitus, it did not significantly impede her recovery from the illness. The patient exhibited remarkable resilience in her recovery journey. Following the prompt initiation of treatment, her progress was steady and gradual. Supportive measures, including physiotherapy, proved invaluable in her rehabilitation journey. After a successful three-week hospitalization, the patient was discharged with a tailored management plan.

Case 10

A previously healthy 23-year-old male patient presented with a four-day history of progressive weakness and numbness in both upper and lower limbs. The initial symptoms began with numbness in the proximal regions of his legs, which gradually spread to encompass the entire lower limbs. He had a history of buckling both knees. Subsequently, numbness and weakness progressed to involve the distal parts of his bilateral upper limbs. He had no history of fever, vomiting, loose stools, or vaccinations. Neurological examination revealed hypotonia in all four limbs, quadriparesis, and areflexia. Tests assessing cerebellum function showed impairment. Sensory examination revealed decreased sensitivity to touch, pain, and temperature in both the lower limbs and the upper limbs up to the elbows. A nerve conduction study confirmed a severe motor demyelinating polyneuropathy with conduction block and secondary axonal changes. Both clinical and electrophysiological investigations pointed towards a diagnosis of AIDP with co-occurring features of AMSAN. He was admitted to the ICU. Since the patient had no progression in both motor and sensory weakness, the disease process attained a plateau stage clinically; therefore, we don’t require immunotherapy as per protocol. We managed him conservatively. Despite the presence of overlapping

AIDP and AMSAN, the patient demonstrated a steady and sustained improvement in both motor and sensory functions throughout the ten-day treatment regimen. Follow-up assessments revealed positive progress, suggesting a favourable prognosis for the patient's recovery.

DISCUSSION

Clinical features

GBS is a monophasic disease that usually reaches its peak (nadir) within 4 weeks. Distal paraesthesia and limb pains precede rapidly ascending muscle weakness from the lower to upper limbs, more marked proximally than distally. GBS is characterized by hyporeflexia or areflexia, which may be absent early in the course of the disease. Cranial nerve involvement occurs in 45% to 75% of patients in different series. Facial and bulbar weakness are prevalent, and respiratory weakness that needs ventilatory support occurs in 20% of cases.[3] Approximately 70% of patients experience moderate to severe pain in the limbs, interscapular area, or back during the acute phase of the disease. Autonomic dysfunction, to varying degrees, has been documented. The majority of clinically significant autonomic dysfunction occurs during the first 2 to 4 weeks of the illness, during the peak time of paralysis.[4]

Table 1: Subtypes of GBS.

Subtype	Features	Electro diagnosis	Antibodies	Pathology
AIDP	Adults are more affected than children, with 90% of cases in Western world. Recovery is rapid, often accompanied by sensory-motor GBS, cranial nerve deficits, and frequent autonomic dysfunction	Demyelinating	Various	The initial attack on Schwann cell surface results in extensive myelin damage, macrophage activation, lymphocytic infiltration, and variable secondary axonal damage.
AMAN	The disease is prevalent in children and young adults in China and Mexico, with rapid recovery and rarely affecting cranial nerves.	Axonal	GM1a, GM1b GD1a GalNAc-GD1a	First attack occurs at Ranvier motor nodes, involving macrophage activation, few lymphocytes, frequent peri-axonal macrophages, and a highly variable extent of axonal damage.
AMSAN	Adults often experience slow and incomplete recovery, which resembles severe AMAN but affects sensory fibers, leading to sensory deficits.	Axonal	GM1, GD1a	Similar to AMAN, it affects sensory nerves and roots, causing severe axonal damage.
MFS	Children and adults are affected. Ophthalmoplegia, ataxia, and areflexia	Axonal or demyelinating	GQ1b (90%)	Few cases were examined; it resembles AIDP

Pathogenesis

Clinical evidence suggests that GBS is an immune-mediated organ-specific condition and is brought on by a synergistic interaction of humoral and cell-mediated immune responses to antigens in the peripheral nervous system. One to four weeks before the onset of neurological symptoms, approximately two-thirds of patients report a preceding event, most commonly a gastrointestinal infection, upper respiratory infection, immunization, or surgery. CMV, Epstein-Barr virus, varicella-zoster virus (VZV), hepatitis A and B, HIV, *Mycoplasma pneumoniae*, and *Haemophilus influenzae* are specific pathogenic agents related to GBS. *C. jejuni* is the most prevalent bacterial species that has been related to GBS, especially its axonal variants.[5] The Thr51 variation of the *C. jejuni* cstII gene is linked to the prevalence of GBS. GBS is more common in patients with lymphoma, HIV-seropositive individuals, and systemic lupus erythematosus (SLE). During the COVID-19 pandemic, SARS-CoV-2 infection in GBS has recently been identified.

Diagnosis

Asbury and Cornblath have reassessed the electrodiagnostic, CSF, and clinical criteria for GBS. Albumino-cytological dissociation, characterized by a high protein content in the CSF (<10 cells/μL) in conjunction with normal cell counts, is considered GBS. The clinical diagnosis of GBS may be supported by nerve conduction tests (NCS), which can distinguish between axonal and demyelinating subtypes. Electrodiagnostic studies in the early stages of the disease frequently reveal reduced muscle response strength, slower nerve signal transmission, and blockages in motor nerve pathways, either individually or together. Prolonged distal latencies, indicative of distal nerve block, and prolonged or absent

F-responses, suggestive of proximal nerve and root involvement are key findings for diagnosing focal demyelinating conditions. In most cases, the H-reflex is either absent or significantly delayed. Even if a limited electrodiagnostic evaluation shows no abnormalities early in the disease course, a more comprehensive investigation, including measurement of late responses, practically always demonstrates disordered conduction in the affected limb within a few days of symptom manifestation. Magnetic resonance imaging of cauda equina roots in acute GBS patients often shows gadolinium enhancement, potentially aiding in complex diagnostics. In response to the need for epidemiologic studies of immunization and estimating GBS risks, the Brighton collaboration created a new set of case criteria for GBS. About 10% of patients experience abnormal liver function, likely due to recent or ongoing viral hepatitis, typically caused by CMV or EBV infections. Hyponatremia is common, especially in ventilated patients.[6]

Table 2: Uncini criteria set employed for electrodiagnosis of GBS subtypes.[7]

AIDP	AMAN	AMSAN	Unexcitable	Equivocal
A minimum of one of the following is present in at least two nerves: **MCV <70% LLN** **DML>130% ULN** **dCMAP duration >120% ULN** **pCMAP/dCMAP duration ratio >130%** **F-response latency >120% ULN** **Alternatively, one of the aforementioned is in a single nerve, plus:** **Absent F waves in two nerves with dCMAP > 20% LLN** **Abnormal ulnar SNAP amplitude and normal sural SNAP amplitude**	Neither of the AIDP characteristics in any nerve (demyelinating features allowed in one nerve if dCMAP <20% LLN) Additionally, in each of the two nerves, at least one of the following: dCMAP<80% LLN pCMAP/dCMAP amplitude ratio <0.7 (excluding tibial nerve) Isolated F wave absence (or <20% persistence)	Same criteria as AMAN in motor nerves, plus SNAP amplitudes <50% LLN in at least two nerves	Distal CMAP absent in all nerves (or present in only one with a distal CMAP <10% LLN)	Abnormal findings not fulfilling specific criteria for other subtypes

Management

GBS is typically treated with immunotherapy and multidisciplinary, supportive medical care. Patients with a GBS disability scale score ≥3 and difficulty walking 10 meters alone typically begin immunotherapy.[8] Immunotherapy options include receiving IVIG at a daily dose of 0.4 g/kg body weight for 5 days or undergoing plasma exchange with 200-250 ml plasma/kg body weight across five sessions. For patients with GBS who have an ongoing infection, antimicrobial or antiviral treatment may be considered. Glucocorticoids are not beneficial in the treatment of GBS.[9] Patients with worsening GBS require critical care monitoring, focusing on vital capacity, heart rhythm, blood pressure, nutrition, deep-vein thrombosis prophylaxis, cardiovascular status, and early tracheotomy and chest physiotherapy.[10] Treatment-related fluctuation (TRF) affects 10% of GBS patients treated with IVIG or plasma exchange, recommending retreatment with IVIG (2 g/kg over 5 days) in those developing TRF.

CONCLUSION

GBS and its variants manifest a diverse clinical spectrum, ranging from AIDP, AMAN, AMSAN, and MFS to rarer forms. Treatment primarily focuses on supportive care and immunotherapy, with plasmapheresis and IVIG demonstrating efficacy in shortening the disease course. The vast majority of patients experience complete or near-complete recovery, with a favourable overall prognosis. However, long-term residual deficits can occur, highlighting the need for effective rehabilitation and supportive measures to improve quality of life. Further research is crucial to refining diagnostic tools, identifying prognostic factors, and developing personalized treatment strategies for optimal outcomes in GBS.

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