Comprehensive Approach to Guillain-Barre Syndrome: A Case Report

ABSTRACT:

Aim: Guillain-Barré Syndrome (GBS) is an autoimmune disorder of the peripheral nervous system that often presents with progressive limb weakness and sensory disturbances. Globally, the incidence rate of Guillain-Barré Syndrome (GBS) is approximately 0.001% to 0.002% of the population annually, or 1-2 cases per 100,000 people.

Presentation of case:A 61-year-old male presented with severe lower back pain, progressing to limb weakness and tingling. Diagnosed with Guillain-Barré Syndrome (GBS) with motor axonal neuropathy and albumin-cytological dissociation via nerve conduction studies, CSF analysis, and imaging, he was treated with plasmapheresis, IVIG, antibiotics for a suspected UTI, neuroprotective medications, and physiotherapy. The patient showed significant motor improvement and stable vitals post-treatment.

Discussion and Conclusion: We discuss the treatment included plasmapheresis, IVIG, antibiotics, physiotherapy, and neuroprotective medications. The patient showed significant improvement, with enhanced limb strength and stable vitals, and is now focusing on rehabilitation and long-term care.

KEYWORDS:*Guillain Barre Syndrome, Acute motor axonal neuropathy, Acute inflammatory demyelinating polyradiculoneuropathy, Acute motor sensory axonal neuropathy (AMSAN), Intravenous IVIG.*

INTRODUCTION:

Guillain-Barré Syndrome (GBS) is an inflammatory condition of the peripheral nervous system and the leading cause of acute flaccid paralysis. It can occur at any age but is more common in men, with its prevalence increasing with age. While GBS manifests in various clinical forms, it typically begins with weakness and sensory disturbances in the legs that progress to the arms and cranial muscles. Diagnosis relies on the patient's medical history, neurological examination, and findings from electrophysiological and cerebrospinal fluid (CSF) studies. Electrophysiological tests not only confirm peripheral nerve damage but also distinguish between subtypes of GBS: acute motor axonal neuropathy (AMAN), acute inflammatory demyelinating polyradiculoneuropathy (AIDP), and acute motor sensory axonal neuropathy (AMSAN).[1] Globally, the incidence rate of Guillain-Barré Syndrome (GBS) is approximately 0.001% to 0.002% of the population annually, or 1-2 cases per 100,000 people. This translates to about 1 to 2 individuals affected for every 100,000 people each year.[1,9]

The exact cause of Guillain-Barré Syndrome (GBS) is unclear, but it is believed to result from an abnormal immune response to infections that damage peripheral nerves. In some patients, antibodies against gangliosides are detected, leading to complement activation, macrophage infiltration, and edema in the affected nerves. Infections before GBS are thought to trigger immune activation, causing demyelination and axonal injury in susceptible individuals. The typical dosage is 0.4 g/kg daily for five days, with similar effectiveness if given over two days. Studies have shown that higher increases in blood IgG levels after IVIg

treatment are linked to better outcomes, such as independent walking after six months. If IgG increases are low, a higher dose or additional IVIg cycles may be recommended.[2]

Patients with Guillain-Barré Syndrome require careful monitoring and comprehensive supportive care. Approximately 25% of patients may require mechanical ventilation, and many experiences autonomic disturbances, necessitating admission to high-dependency or intensive care units. Symptoms typically peak within four weeks, followed by a recovery phase that can extend for months or even years, during which the immune response diminishes, and the peripheral nerves undergo natural repair.[8]
The standard treatments for Guillain-Barré Syndrome (GBS) include intravenous immunoglobulin (IVIG) and plasma exchange (PLEX). IVIG is thought to work through immune modulation, although the exact mechanism is not fully understood. It is usually administered at a dose of 2 grams per kilogram over a period of five days. Plasma exchange is believed to function by removing harmful antibodies, humoral mediators, and complement proteins involved in the development of GBS, though its exact mechanism also remains unclear. Corticosteroids are not recommended, as intravenous methylprednisolone (IVMP) does not improve neurological outcomes, and combining intravenous methylprednisolone with IVIG provides no added benefit. Oral corticosteroids may even hinder recovery or lead to side effects.

The focus of treatment should be on early diagnosis and intervention to prevent severe axonal damage and minimize long-term disability. Meanwhile, ongoing research into new therapies and neuropathy fluid biomarkers may lead to advancements in clinical management soon.[3,4]

CASE REPORT:

A 61-year-old male patient presented to the hospital with severe lower back pain, followed by weakness and a tingling sensation in both lower limbs for 20 days. He had been evaluated at another facility, where an MRI of the spine revealed L3-L4, L4-L5, and L5-S1 PIVD with nerve root compression at L5 and cervical lordosis. Subsequently, the patient experienced weakness in both upper limbs accompanied by a tingling sensation. There was no history of fever, loose stools, or double vision. The patient was then admitted for further evaluation and management. On neurological examination, he was found to be conscious, oriented, and without any abnormalities. Nerve conduction velocity which shows the study showed diffuse motor axonal with secondary demyelinating neuropathy LL>UL with preserved sensory in all limbs and ultrasound abdomen and pelvis shows Internal echoes in urinary bladder suggested URE correlation to rule out cystitis.

Fig 1: X RAY LUMBOSACRAL SPINE A.P/ LAT	Degenerative changes are noted in the form of anterior and lateral marginal osteophytes, suggesting mild lumbar spine spondylosis.
Fig 2: MRI LUMBOSACRAL SPINE	Mild lumbar spondylosis with mild disc bulges from L3-L4 TO L5-S1 LEVELS indenting anterior thecal sac with mild narrowing of neural foramina. cervical spondylosis causing moderate central canal stenosis at C5-C6 and C6-C7 levels with mind indentation on the cord and mild cord

	edema.
Fig 3: 3T MRI BRAIN WITH MRA AND MRV	Few chronic ischemic foci in bilateral periventricular and frontoparietal white matter regions. No evidence of acute infarction or intracranial arteries and dural venous sinuses.
Table 1: Diagnostic Findings and Imaging Results	

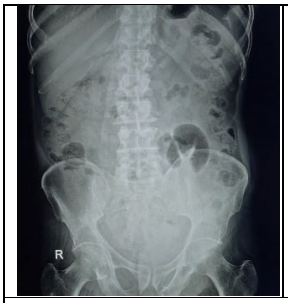

Fig 1: X RAY LUMBOSACRAL SPINE

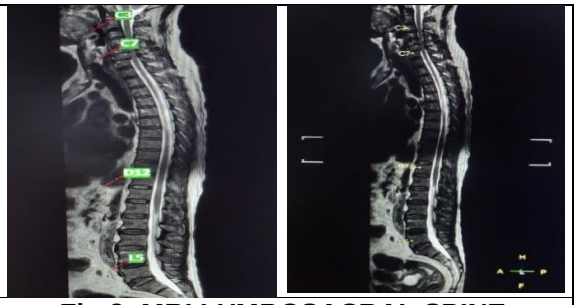

Fig 2: MRI LUMBOSACRAL SPINE

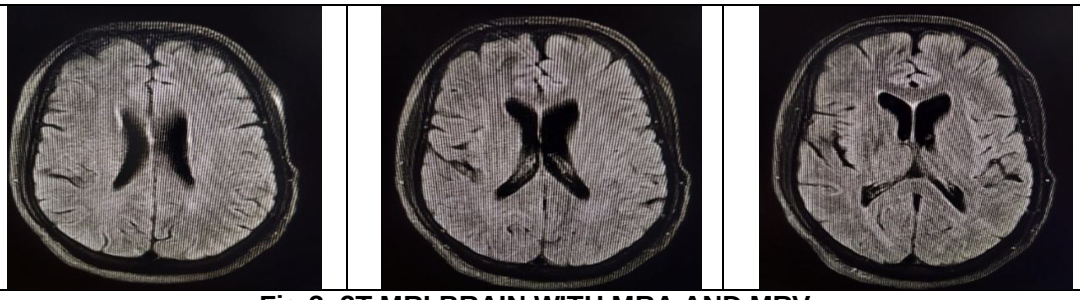

Fig 3: 3T MRI BRAIN WITH MRA AND MRV

PROCEDURE	
LUMBAR PUNCTURE	11-03-2024
5 CYCLES PLASMAPHERESIS:	
1ST CYCLE	07-03-2024
2ND CYCLE	08-03-2024
3RD CYCLE	09-03-2024
4TH CYCLE	11-03-2024
5TH CYCLE	12-03-2024
Table 2: Medical Procedure and timeline.	

The patient was admitted to the Neuro ICU for further evaluation and management. He was started on neuro-supportive medications. Routine investigations revealed an elevated total count of 18.0, a CRP level of 60.49, and low sodium of 127. The patient was initiated on IV antibiotics (ceftriaxone 2 g) and treatment for hyponatremia. Nerve conduction velocity (NCV) testing of all four limbs showed diffuse motor axonal neuropathy with secondary demyelination (LL>UL) and preserved sensory function in all limbs. Based on the patient's

clinical condition and NCV findings, Guillain-Barré Syndrome was suspected. After discussion with the patient's family, he underwent five cycles of plasmapheresis (Table 2).

Following the initial cycles of plasmapheresis, his results showed a cell count of 08, 100% lymphocytes, sugar level of 76, and protein of 106.39 (indicating albumin-cytological dissociation). Due to recurrent fever spikes, urine was sent for routine analysis and culture. The urine routine revealed pus cells of 989, RBC count of 24, and numerous bacteria, although the urine culture showed no growth. A urologist recommended oral nitrofurantoin 100 mg. Antibiotics were escalated from IV ceftriaxone to IV meropenem 1 g. The patient was also prescribed with multivitamins, including intravenousMethylcobalamin, intravenousThiamine 200 mg, and oralVitamin E 400 mg to enhance immunity.

Following the escalation of antibiotics, the patient did not experience any further fever spikes and was transferred to the ward in a stable condition. After completing five cycles of plasmapheresis, he was started on IVIG therapy, receiving 10 grams per cycle for two days. Physiotherapy was initiated. Due to bladder disturbances, a urology consultation was sought, and the advice provided was followed. The patient responded well to the treatment and did not develop any new symptoms. He was conscious, coherent, and obeying commands. His speech was normal, and he was able to move all four limbs, with upper limb power assessed at 4/5 and lower limb power at 2/5. He could eat orally and maintained stable vitals. The patient's relatives were counselled and educated about the nature of the illness, the necessity of long-term care, and the importance of continued physiotherapy and rehabilitation. The patient was hemodynamically stable and was planned for discharge.

DISCUSSION:

Guillain–Barré syndrome (GBS) is an autoimmune disorder affecting the peripheral nerves, marked by reduced or absent reflexes, neuromuscular paralysis, and elevated protein levels in the cerebrospinal fluid without a corresponding increase in white blood cells.[5]

The diagnostic methods involve nerve conduction velocity tests and cerebrospinal fluid examination. GBS is marked by progressive complications, which can range from muscular weakness to cardiovascular problems. Physiotherapy plays a crucial role in the assessment and treatment of Guillain-Barré Syndrome (GBS), focusing on comprehensive evaluations and tailored rehabilitation strategies to aid recovery and enhance quality of life. The discussion includes multidisciplinary rehabilitation, exercise training, pulmonary rehabilitation, electrical stimulation, robotic rehabilitation, virtual reality, and alternative therapies.[6]

The diagnosis of Guillain-Barré Syndrome (GBS) relies on the patient's clinical presentation and electrophysiological findings, which vary depending on the specific variant. The classic clinical manifestation includes bilateral, symmetrical, ascending weakness accompanied by hyporeflexia. Electrophysiological evidence of demyelination may include reduced motor nerve conduction velocity, prolonged distal motor latency, increased F-wave latency, conduction blocks, and temporal dispersion.[7]

This case describes a 61-year-old male presenting with progressive neurological symptoms, including lower back pain, limb weakness, and tingling, later diagnosed as Guillain-Barré Syndrome (GBS) based on nerve conduction studies showing diffuse motor axonal neuropathy with demyelination and cerebrospinal fluid analysis revealing albumin-cytological

dissociation. Imaging identified cervical and lumbar spondylosis with mild neural compromise but did not account for the acute presentation. Management included five cycles of plasmapheresis followed by IVIG therapy, alongside antibiotics for a suspected urinary tract infection causing fever. Supportive care, including physiotherapy and neuroprotective medications, was initiated. The patient responded well, with improved motor function, stable vitals, and no new symptoms, leading to discharge with long-term rehabilitation plans.

CONCLUSION:

This case highlights the importance of early diagnosis and a multidisciplinary approach in managing Guillain-Barré Syndrome (GBS), a serious autoimmune disorder. A 61-year-old male presented with progressive limb weakness and sensory symptoms. Diagnostic findings, including nerve conduction studies and cerebrospinal fluid analysis, confirmed GBS with motor axonal neuropathy and albumin-cytological dissociation. Imaging showed incidental cervical and lumbar spondylosis without acute significance. Treatment included five cycles of plasmapheresis followed by IVIG therapy, alongside management of a suspected urinary tract infection with antibiotics and supportive care. Physiotherapy was initiated to improve motor function, and neuroprotective medications addressed associated complications. The patient demonstrated significant improvement, with enhanced limb strength, stable vitals, and no further symptoms. Patient was further focused on rehabilitation and long-term care.

Disclaimer (Artificial intelligence)

Option 1:

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc.) and text-to-image generators have been used during the writing or editing of this manuscript.

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