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Gullain Barre Syndrome: Acute Motor Axonal Neuropathy

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TABLES

The laboratory report on cerebrospinal fluid (CSF) analysis following a lumbar puncture provides critical insights into cell count**,** cell type, glucose levels, and protein concentration as follows in table 1

Table 1: CSF Report Analysis

Sl.no	Parameter	Result	Normal range
1.	Cell count	04	0–5 cells/µL
2.	Cell type	100% lymphocytes	Predominantly lymphocytes (no Polymorphonuclear leukocytes)
3.	Sugar CSF	62	50–80 mg/dL (or >60% of serum glucose)
4.	Proteins CSF	27.9	15–45 mg/dL

Plasmapheresis is a therapeutic procedure used to remove harmful substances from the blood, particularly in autoimmune and neurological disorders procedure done for the patient on following date mentioned in the table 2.

Table 2: Plasmapheresis cycle

Date	Number of plasmapheresis cycle
06/10/2022	1st cycle
08/10/2022	2nd cycle
10/10/2022	3rd cycle

12/10/2022	4th cycle
14/10/2022	5th cycle

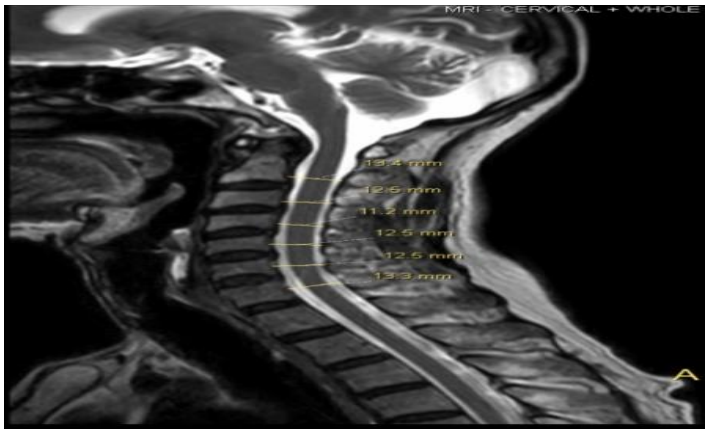

Figure.1: Magnetic Resonance Image(MRI) scan of the cervical spine

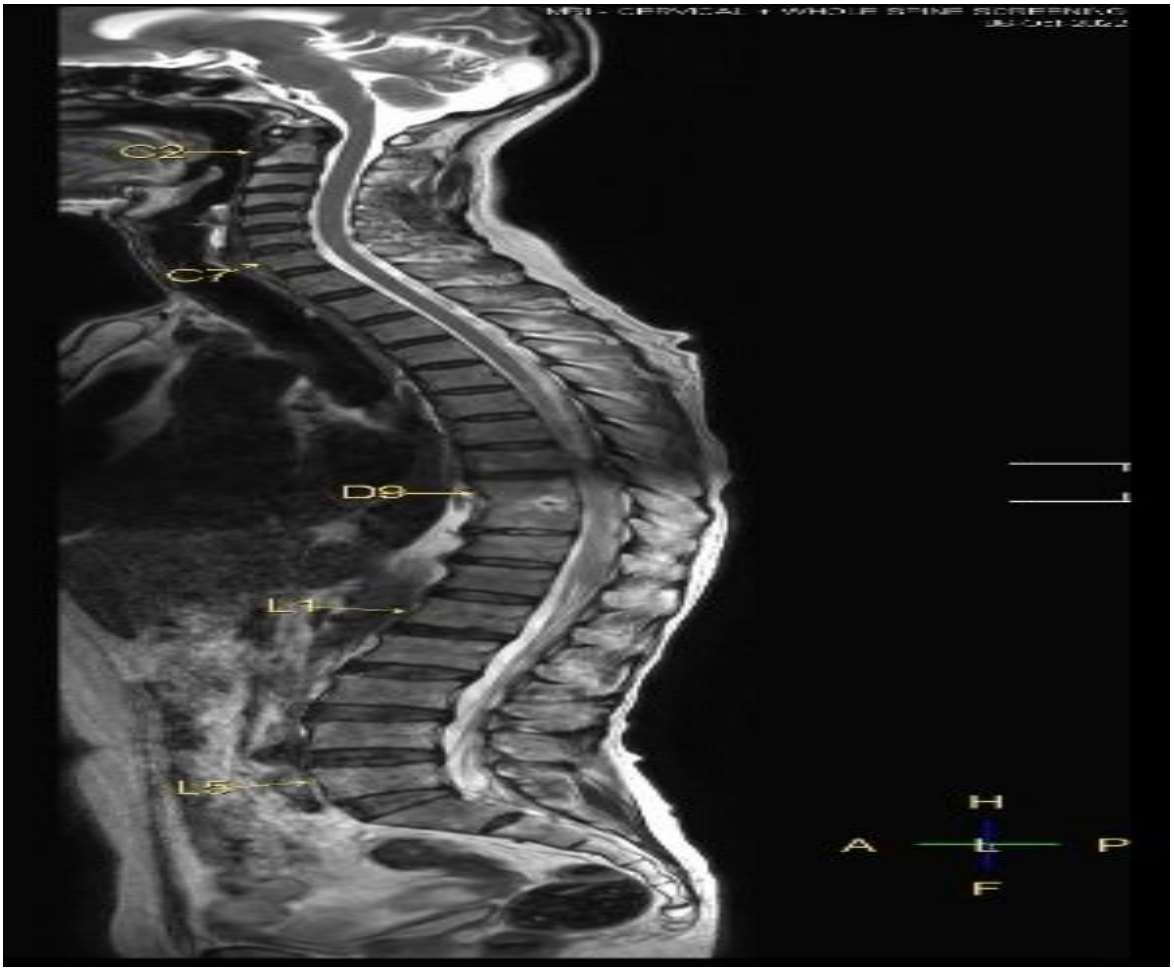

Figure.2: Magnetic Resonance Image(MRI) scan of the whole spine

ABSTRACT

BACKGROUND

Guillain-Barré Syndrome (GBS) is an acute immune-mediated polyneuropathy that results in ascending paralysis, autonomic dysfunction, and neuromuscular impairment.

OBJECTIVES

This case highlights the presentation, diagnosis, and therapeutic management of Acute Motor Axonal Neuropathy (AMAN), a severe GBS variant.

MATERIAL AND METHODS:

A 73-year-old male patient case report, Cerebrospinal fluid (CSF) analysis showed mild pleocytosis with albuminocytologic dissociation**,** and MRI findings revealed an incidental posterior fossa arachnoid cyst.

RESULT

A 73-year-old male presented with gait ataxia, progressive limb weakness, and numbness**.** Cerebrospinal fluid (CSF) analysis showed mild pleocytosis with albuminocytologic dissociation**,** and MRI findings revealed an incidental posterior fossa arachnoid cyst. The patient underwent plasmapheresis (five cycles) along with neuro-rehabilitative therapy to remove pathogenic circulating autoantibodies and enhance recovery. Following plasmapheresis and physiotherapy, the patient demonstrated substantial neurological improvement**,** with restored motor function, preserved bulbar reflexes, and safe oral intake**.**

CONCLUSION

The critical role of early immunomodulatory intervention**,** particularly plasmapheresis and rehabilitation**,** in managing GBS variants like AMAN**.** Multidisciplinary care, close outpatient monitoring, and structured rehabilitative support are essential in optimizing functional outcomes and minimizing recurrence risks**.**

KEY WORDS: *Gullian barre syndrome, Axoglial junction, Plasmapheresis, Neuro rehabilitation*.

1. INTRODUCTION

Guillain-Barré syndrome (GBS) is a rare autoimmune condition where the body's immune system erroneously targets peripheral nerves, resulting in muscle weakness, numbness, and, in severe instances, paralysis.[1] 150,095 people worldwide were estimated to have Guillain-Barré syndrome (GBS) in 2019. Around the world, the condition was responsible for 44,407 years lived with disability (YLDs). GBS's age-standardized prevalence increased 6.4% from 1990 to 2019.[1] Geographically, the incidence of GBS varies; China had one of the lowest age-standardized prevalence rates (0.8 per 100,000), while Japan had the highest rate (6.4 per 100,000). According to a population-based study conducted in urban China between 2013 and 2017, the yearly incidence of GBS ranged from 0.41 to 0.58 per 100,000 person-years.[2] The yearly incidence of GBS in India is between 0.3 and 1.3 cases per 100,000 people under the age of 18.[3] There are various subtypes of Guillain-Barré syndrome (GBS), and each one has a unique impact on the peripheral nervous system: AIDP stands for acute inflammatory demyelinating polyneuropathy. Immune-mediated demyelination, which causes muscle weakness, sensory abnormalities, and autonomic dysfunction, is the most prevalent type in Western nations.[5] AMAN, or acute motor axonal neuropathy, An axonal variation that mainly affects motor nerves without sensory involvement is more common in Asia and Latin America. It is frequently linked to an infection with Campylobacter jejuni. Acute Motor and Sensory Axonal Neuropathy (AMSAN) A severe variant similar to AMAN but involving both motor and sensory nerves, leading to prolonged recovery.[4] Miller Fisher Syndrome (MFS) A rare subtype characterized by ophthalmoplegia (eye muscle paralysis), ataxia (lack of coordination), and areflexia (loss of reflexes)**.** It is strongly linked to anti-GQ1b antibodies.[4] Infections including Campylobacter jejuni, Cytomegalovirus (CMV), Epstein-Barr virus (EBV), Zika virus, influenza, or COVID-19 frequently cause the illness because of molecular mimicry that causes the immune system to unintentionally target nerve components. Rarely, several vaccines have been linked to GBS, but the risk is far smaller than the chance of getting the illness after exposure. Furthermore, in certain situations, significant surgical procedures have been linked to inflammation of the immune system. Despite not being inherited, genetic predisposition may enhance susceptibility to GBS. Recovery outcomes can be enhanced by early risk factor identification and timely management with therapies like intravenous immunoglobulin (IVIG) or plasmapheresis.[5]

2. CASE PRESENTATION

A 73-year-old male was admitted in neuro ward at a tertiary care hospital on October 6, 2022, with complaints of abrupt onset of giddiness followed by difficulties walking and unbalance when walking around 5 p.m. that same day. He had been experiencing numbness in both of his upper and lower limbs over three days. There is no history of blurred vision, loss of consciousness, slurred speech, seizure, fever, chest pain, or tongue bite.

The patient's neurological and physical examination indicates preserved consciousness and orientation, with the ability to follow commands and actively move all four limbs. Muscle strength assessment shows mild weakness, with a power rating of 4+/5 in the left upper and lower limbs**,** 4+/5 in the right upper limb**,** and 4/5 in the left limb, suggesting mild to moderate weakness. The presence of gait ataxia signifies difficulty in coordinating movements while walking, potentially indicative of neurological impairment**.** Pupillary response is normal, with both pupils equally reactive to light, suggesting

intact cranial nerve function. Additionally, all peripheral pulses are palpable, indicating adequate vascular circulation. These findings provide insight into the patient's neurological status**,** potentially aiding in the diagnosis of neuromuscular disorders like Guillain-Barré syndrome (GBS).

As shown in table 1. The CSF analysis results indicate a mildly elevated cell count (4 cells/µL) with 100% lymphocytes, suggesting a non-bacterial inflammatory or autoimmune process. The CSF glucose level (62 mg/dL**)** falls within the normal range (50–80 mg/dL), ruling out bacterial meningiti**s**, which typically presents with low glucose levels. The CSF protein concentration (27.9 mg/dL) is also within the normal range (15–45 mg/dL), though Guillain-Barré syndrome (GBS) often shows elevated protein levels without an increase in cell count.These findings are consistent with viral or autoimmune neurological conditions, potentially aligning with early-stage GBS, where CSF protein may rise later in the disease course.

The MRI scan Figure 1. of the cervical spine conducted on 06/10/2022 and revealed a posterior fossa arachnoid cyst, which is a fluid-filled sac located in the region near the brainstem and cerebellum. However, no other significant abnormalities were detected in the brain parenchyma, and there were no signs of vascular compromise in the intracranial arteries or Dural venous sinuses. This suggests that the arachnoid cyst is likely an incidental finding unless associated with clinical symptoms such as headache, dizziness, or balance issues.

The MRI scan Figure 2. of the whole spine done on 06/10/2022 and revealed indicates mild degenerative changes in the cervical and lumbar spine, consistent with spondylitis. Additionally, there is a congenital fusion of the D9-D10 vertebrae (block vertebra), accompanied by moderate narrowing of the neural foramina at these levels. The scan also shows mild posterior disc bulges at L3-L4, L4-L5, and L5-S1, which do not significantly obstruct the spinal canal but contribute to mild to moderate foramina narrowing at L3-L4 and L4-L5. These findings suggest structural changes that may lead to localized discomfort or nerve compression.

3. DISCUSSION

The patient presents with sudden onset gait instability, limb weakness, and numbness, indicating a rapidly evolving neuromuscular condition. Neurological examination reveals mild to moderate motor weakness**,** with muscle power ranging from 4+/5 to 4/5 across different limbs. Notably, cranial nerve function remains intact, and the patient retains full consciousness and orientation**,** strongly suggesting a peripheral neurological disorder rather than a central nervous system pathology**.**[6 7]

The cerebrospinal fluid (CSF) analysis reveals a mild pleocytosis with a cell count of 4 cells/µL, falling within the normal range yet slightly elevated, suggesting an immune-mediated response rather than an infectious aetiology. The presence of 100% lymphocytes without polymorph nuclear leukocytes strongly supports an autoimmune or post-infectious neurological process, effectively ruling out bacterial meningitis. The CSF protein concentration (27.9 mg/dL) remains within physiological limits, a finding not uncommon in early-stage Guillain-Barré Syndrome (GBS), where albuminocytologic dissociation an elevated protein level without pleocytosis—may manifest later. Additionally, glucose levels (62 mg/dL) are well-preserved, eliminating concerns for infectious meningeal involvement. Collectively, these findings align with Acute Motor Axonal Neuropathy (AMAN), a variant of GBS, where axonal degeneration predominates without significant demyelination, leading to motor dysfunction while largely sparing sensory modalities.[7]

The MRI of the cervical spine revealed a posterior fossa arachnoid cyst, likely an incidental finding unless clinically significant symptoms—such as headache, dizziness, or coordination difficulties manifest. The whole-spine MRI indicated mild degenerative spondylitic changes**,** congenital D9-D10 vertebral fusion, and neural foraminal narrowing. While these structural abnormalities may contribute to localized discomfort, they are insufficient to account for the progressive neuromuscular impairment observed in this patient.

Correlation with Guillain-Barré Syndrome (GBS)

GBS is characterized by autoimmune-mediated peripheral nerve demyelination or axonal injury, typically post-infectious, leading to ascending paralysis, sensory deficits, and autonomic dysfunction. This patient’s progressive limb weakness and numbness, combined with CSF findings, strongly suggest early-stage GBS, potentially evolving into Acute Motor Axonal Neuropathy (AMAN).[1] The presence of ataxia, absent cranial nerve involvement, indicates a rare GBS variant requiring electrophysiological confirmation.

Early recognition and intervention are critical for GBS**,** given its potential for respiratory failure and autonomic instability**.** Intravenous immunoglobulin (IVIG) or plasmapheresis remains the cornerstone of treatment, while physical rehabilitation is crucial for long-term functional recovery.[6] Continuous monitoring for bulbar involvement, autonomic dysfunction, and respiratory compromise is advised.

Clinical Progress and Therapeutic Intervention

The patient was started on steroid therapy Inj. Solumedrol, dose 1g through IV in 500ml normal saline slow infusion was give once a day for 5 days and 5 cycles of plasmapheresis was administered. 1 cycle every alternative day along with other neuro-supportive treatments that is physiotherapy. Following the initiation of physiotherapy, the patient

demonstrated a favourable clinical response, exhibiting improved sensorimotor function. He is now conscious, fully oriented, and obeys verbal commands appropriately. His cough reflex is intact, signifying preserved bulbar function, and he has regained the ability to safely consume oral nutrition, eliminating concerns of aspiration.

The patient and caregivers have been counselled on the pathophysiology of Guillain-Barré Syndrome (GBS), the expected recovery trajectory, and the importance of scheduled follow-ups for monitoring potential residual neurological deficits. He has been discharged with advice to continue outpatient follow-up in the Neurology department, ensuring long-term clinical oversight and rehabilitation.

Plasmapheresis in Guillain-Barré Syndrome

Plasmapheresis is an extracorporeal therapy that selectively removes circulating pathogenic antibodies and immune complexes implicated in the autoimmune-mediated attack on peripheral nerves. This technique involves separation of blood components, yielding a filtered plasma product, effectively reducing immune-mediated neurotoxicity, and facilitating neural recovery.[8]

The patient underwent five cycles of plasmapheresis, performed on alternate days, with continuous hemodynamic monitoring as shown in the table 2. Throughout the procedure, he remained clinically stable, without significant cardiovascular or autonomic fluctuations. Following completion of the therapeutic regimen, he was discharged on 15/10/2022, with neurological improvement and functional stability.[9]

4. LIMITATION

Single-Case Focus: The study is based on a single case report, limiting generalizability to broader populations affected by Guillain-Barré Syndrome (GBS), particularly the Acute Motor Axonal Neuropathy (AMAN) variant.

Short-Term Follow-Up: The study primarily evaluates immediate clinical progress post-plasmapheresis but lacks long-term follow-up data to assess sustained neurological recovery and potential relapse risks.

5. CONCLUSION

Guillain-Barré Syndrome (GBS), particularly the Acute Motor Axonal Neuropathy (AMAN) variant, presents as a rapidly progressive immune-mediated polyneuropathy resulting in motor weakness, absent reflexes, and autonomic dysfunction. Early diagnosis, guided by clinical evaluation, cerebrospinal fluid (CSF) analysis, nerve conduction studies, and MRI findings, is crucial for appropriate intervention.

This case highlights the significance of plasmapheresis as a primary therapeutic strategy, effectively reducing the burden of circulating pathogenic antibodies and mitigating neuromuscular impairment. The patient's clinical improvement following steroid therapy and plasmapheresis underscores the efficacy of early immunomodulatory treatment. The successful neurological recovery, evidenced by restoration of bulbar function, motor strength, and safe oral intake, emphasizes the importance of rehabilitation and continuous follow-up to optimize long-term functional outcomes.

Given the immune-mediated nature of GBS, structured outpatient neurology follow-up remains imperative to monitor for residual deficits**,** autonomic instability, and recurrence risk. This case reinforces the necessity of early therapeutic intervention**,** multidisciplinary management, and patient education, which collectively enhance prognosis and functional independence.

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REFERENCE

[1] Bragazzi NL, Kolahi AA, Nejadghaderi SA, Lochner P, Brigo F, Naldi A, et al. Global, regional, and national burden of Guillain–Barré syndrome and its underlying causes from 1990 to 2019. Journal of Neuroinflammation [Internet]. 2021 Nov 11;18(1).Available from: https://jneuroinflammation.biomedcentral.com/articles/10.1186/s12974-021-02319-4

[2] Xu L, Zhao C, Bao Y, Liu Y, Liang Y, Wei J, et al. Variation in worldwide incidence of Guillain-Barré syndrome: a population-based study in urban China and existing global evidence. Frontiers in Immunology. 2024 Sep 10;15.

[3] Kumar R, Co-Authors S, Suthar R. Indian Academy of Pediatrics (IAP) Under the Auspices of the IAP Action Plan 2022 Upendra Kinjawadekar Guillain-Barré Syndrome [Internet]. Available from: https://iapindia.org/pdf/Ch-053-stg-guillain-barre-syndrome.pdf

[4] What are the types of Guillain-Barre syndrome? [Internet]. www.medicalnewstoday.com. 2024. Available from: https://www.medicalnewstoday.com/articles/types-of-guillain-barre-syndrome

[5] Mayo Clinic. Guillain-Barre syndrome - Symptoms and causes [Internet]. Mayo Clinic. 2024. Available from: https://www.mayoclinic.org/diseases-conditions/guillain-barre-syndrome/symptoms-causes/syc-20362793

[6] Willison HJ, Jacobs BC, van Doorn PA. Guillain-Barré syndrome. Lancet (London, England) [Internet]. 2016 Aug 13;388(10045):717–27. Available from: https://www.ncbi.nlm.nih.gov/pubmed/26948435?dopt=Abstract

[7] Sejvar JJ, Baughman AL, Wise M, Morgan OW. Population Incidence of Guillain-Barré Syndrome: A Systematic Review and Meta-Analysis. Neuroepidemiology [Internet]. 2011;36(2):123–33. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5703046/

[8] Palma D, Dm P, An C, Dl C, Plasmapheresis T, Barré G. Neurological Case Reports Plasmapheresis and Guillain-Barré Syndrome: Desperate Therapy for Desperate Patient? A Case Report OPEN ACCESS Citation. 2022 [cited 2025 May 8];5. Available from: https://www.remedypublications.com/open-access/plasmapheresis-and-guillainndashbarreacute-syndrome-desperate-therapy-for-8573.pdf

[9] Osterman PO, Fagius J, Safwenberg J, Danersund A, Wallin BG, Nordesjo LO . Treatment of the Guillain-Barre Syndrome by Plasmapheresis. Archives of Neurology. 1982 Mar 1;39(3):148–54.

[10] Zito A, Alfonsi E, Franciotta D, Todisco M, Gastaldi M, Cotta Ramusino M, et al. COVID-19 and Guillain–Barré Syndrome: A Case Report and Review of Literature. Frontiers in Neurology. 2020 Aug 21;11.

[11] Nilse Querino Santos, Cláudia A, Antonio Alberto Lopes, Costa G, Aroldo Bacellar. Guillain-Barré syndrome in the course of dengue: case report. 2004 Mar 1;62(1):144–6.

[12] Hartung HP, Toyka KV, Pollard JD, Harvey GK. Immunopathogenesis and treatment of the guillain-barr?syndrome?part I. Muscle & Nerve. 1995 Feb;18(2):137–53.

[13] Meena A, Murthy JMK, Khadilkar S. Treatment guidelines for Guillain-Barré Syndrome. Annals of Indian Academy of Neurology [Internet]. 2011;14(5):73.
Availablefrom:https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3152164/