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Case Report

Acute Demyelinating Polyradiculoneuropathy in Guillain-Barré Syndrome: A Case Report Highlighting Diagnostic Challenges and Management Strategies

Prathap Arumugam, Angelin Grace Thomas, Ann Jency Arul Durai Singam, Dhivyaprasath Palaniyappan*

Department of Pharmacy Practice, Swamy Vivekanandha College of Pharmacy, Elayampalayam, Tiruchengode, Namakkal, Tamil Nadu, INDIA.

ABSTRACT

A 73-year-old female with fever, muscle weakness, and back pain was diagnosed with Acute Inflammatory Demyelinating Polyradiculoneuropathy (AIDP), a subtype of Guillain-Barré Syndrome (GBS). Laboratory findings showed elevated white blood cells, neutrophilia, and albuminocytological dissociation in cerebrospinal fluid. Nerve conduction studies confirmed demyelination, and imaging revealed degenerative spinal changes. She received Immunomodulatory therapy (IVIG) and supportive care, with careful management of comorbidities like hypertension and COPD. Early diagnosis and multidisciplinary care led to a positive outcome, highlighting the importance of timely intervention and monitoring to prevent complications and long-term disability.

Keywords: Electrodiagnostic studies, Cerebrospinal fluid analysis, Immunotherapy, Rehabilitation, Nerve conduction studies.

Correspondence:
Dr. Dhivyaprasath Palaniyappan
Assistant Professor, Department of Pharmacy Practice, Swamy Vivekanandha College of Pharmacy, Elayampalayam, Tiruchengode-637205, Namakkal, Tamil Nadu, INDIA.
Email: drdpsvcp@gmail.com

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INTRODUCTION

Neurological disorder Guillain-Barré Syndrome (GBS) results in abrupt muscle weakness that might either become crippling or, in extreme circumstances, lead to breathing difficulties that may be fatal. It still poses a challenging medical mystery to answer (Malek and Salameh, 2019). Anti-ganglioside antibodies are produced as a result of molecular mimicry brought on by an earlier infection, and these antibodies target proteins found in the axonal membrane. Aggression like this can damage cranial nerves and sensory fibres by causing fast-progressing ascending flaccid paresis. Electrophysiological studies and the clinical profile can help GBS be more broadly categorised as acute inflammatory demyelinating polyradiculoneuropathy, Acute Motor Axonal Neuropathy (AMAN), acute motor sensory axonal neuropathy, and Acute Motor-Sensory Axonal Neuropathy (AMSAN) (Amoretti *et al*., 2002). Usually caused by infections, GBS is particularly viral and bacterial Pathogens; Zika virus, Epstein-Barr virus, *Campylobacter jejuni*, Cytomegalovirus, CMV (Uncini and Yuki, 2012). Autonomic dysfunction; cranial nerve, Deep and some of its many aspects include involvement, ascending weakness, and sensory abnormalities varied clinical symptoms (Berg *et al*., 2014). The clinical path of GBS is marked by a fast advance to peak weeks of improvement, then a plateau phase and months-to-year recovery period. The rehabilitation process, though, varies greatly; whereas some people could get a total or, while some may suffer long-term effects or partial relief from their symptom's continuous impairment (Uncini *et al*., 2013). GBS must be identified quickly when individuals have rapidly worsening paralysis. Every GBS patient requires close observation, and early initiation of targeted therapy and supportive care might be beneficial (Esposito and Longo, 2017). This case report highlights important difficulties and factors in diagnosing and treating Guillain-Barré Syndrome by summarising the clinical presentation, diagnostic methodology, care, and outcome of a patient with the condition.

CASE DESCRIPTION

A 73-year-old female presents with complaints of backache, difficulty standing and walking for 3 days, and fever for 10 days. She had a past medical history of hypertension and COPD for 7 years and was on medication. Tab. Olmesartan 20 mg + Chlorthalidone 12.5 mg 1-0-0, Salmeterol 50 mcg + Fluticasone propionate 250 mcg (Powder for Inhalation) 1-0-0, T. Ferrous fumarate + Folic acid + Zinc sulphate 0-0-1, T. Cetirizine 10 mg 0-0-1/2. On assessing the nerve conduction study, it showed demyelinating changes suggestive of demyelinating polyradiculoneuropathy. On peripheral smear study showed normocytic normochromic anaemia with neutrophil preponderance. Gram stain (CSF) shows occasional WBCs and no bacteria. CSF analysis shows an

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increase in protein CSF (Table 1). On assessing the India INK preparation, nonencapsulated yeast was seen. On assessing the complete blood count, there is an increase in WBC, neutrophil, and absolute neutrophil count than normal, and haemoglobin, PCV, lymphocyte, total RBC, and absolute lymphocyte count were decreased than normal (Table 2). On assessing the sodium level, it was decreased from day 4 (Figure 1). The Gram stain result of the tracheal secretion suggests a relatively clean sample with minimal inflammatory response. The presence of moderate epithelial cells indicates some cellular material from the respiratory tract, while the 0-2 pus cells suggest minimal active infection. The occasional Gram-negative bacilli detected might represent normal respiratory tract colonisation or a potential early stage of bacterial presence. The neurological examination demonstrated symmetric weakness in the lower limbs, with proximal muscle strength rated at 3/5 and distal strength at 2/5 on the MRC scale. The upper limbs exhibited mild weakness, graded at 4/5. Deep tendon reflexes were absent in all four limbs. Sensory testing revealed diminished vibration and proprioception in the feet, while pinprick and temperature sensations remained intact. Cranial nerve assessment showed bilateral facial weakness, with preserved extraocular movements and no signs of dysphagia. The patient was unable to walk without assistance due to combined motor weakness and proprioceptive deficits. Autonomic evaluation indicated intermittent tachycardia and fluctuating blood pressure, suggestive of autonomic involvement. These clinical features were consistent with Guillain-Barré Syndrome (GBS), despite initial Cerebrospinal Fluid (CSF) analysis and Nerve Conduction Studies (NCS) being inconclusive. MRI Cervical spine with screening of the whole spine report shows diffuse osteopenia with diffuse degenerative spondylosis, diffuse disc bulge at the C4-C5-C6 level with significant thecal sac, cord, and nerve root compression, and posterolateral disc bulge at the C5-C6-C7 level on the left side causing significant neural foraminal narrowing and nerve root compression (Figure 2). The patient was prescribed medications: Inj. Piperacillin + Tazobactam 4.5 mg TDS, Inj. Levetiracetam 500 mg BD, Inj. Pantoprazole 40 mg 1-0-1, T. Fluconazole 150 mg 0-0-1, T. Ivabradine 5 mg 1-0-1, T. Neurobion forte 0-0-1, Inj. Paracetamol 1 g SOS, Neb. Glycopyrronium bromide 1-0-1, Neb. Acetyl Cysteine 1-0-1, Syp. Potassium chloride 10 mL TDS, T. Ferrous fumarate + Folic acid 1-0-0. ET Culture shows the presence of Pseudomonas aeruginosa. Sensitivity to Piperacillin + Tazobactam, Gentamicin, and Meropenem.

DISCUSSION

Guillain-Barré Syndrome (GBS) is a rare, acute, immune-mediated polyneuropathy characterised by rapid-onset weakness, ascending paralysis, and areflexia. It typically occurs after an antecedent infection, such as respiratory or gastrointestinal illness caused by pathogens like *Campylobacter jejuni*, cytomegalovirus, Epstein-Barr virus, or influenza. GBS is thought to result from molecular mimicry, where an immune response to the

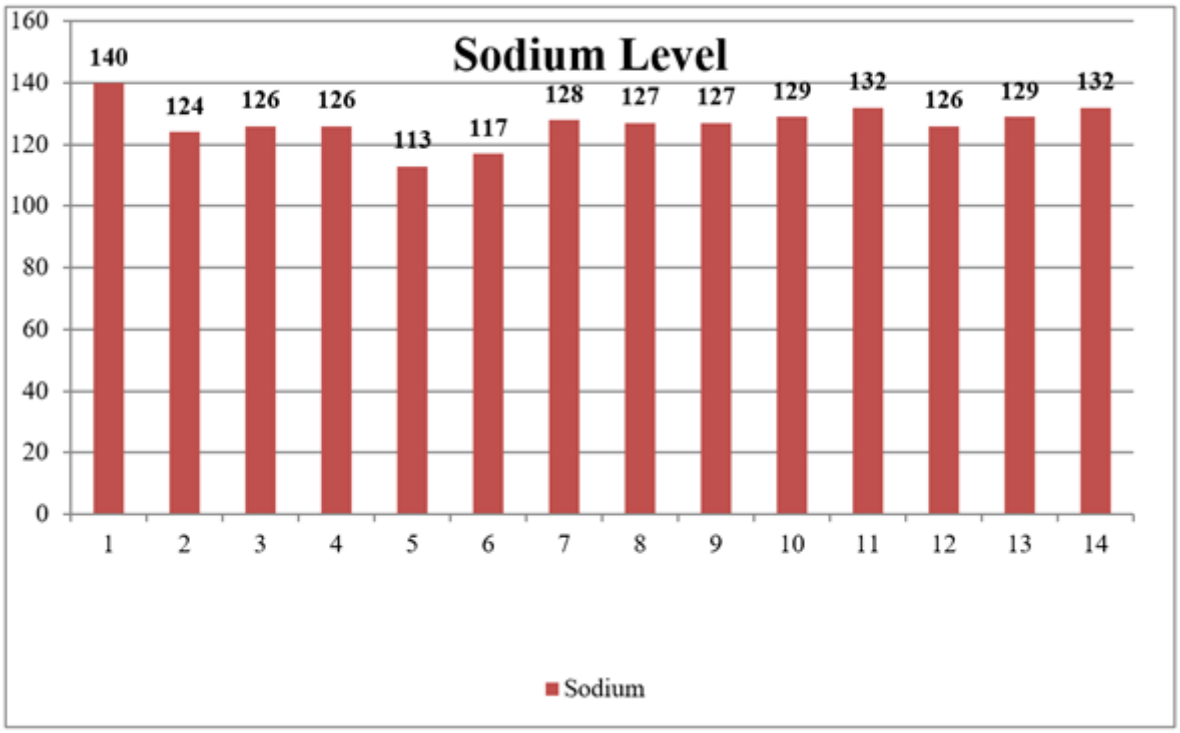

Figure 1: Serum Sodium level. This bar chart shows the sodium levels (in mmol/L) for individual patient, numbered from 1 to 14 based on their latest measurements. Each bar gives a clear picture of one patient's sodium level, with the values displayed right at the top of each bar for easy reference.

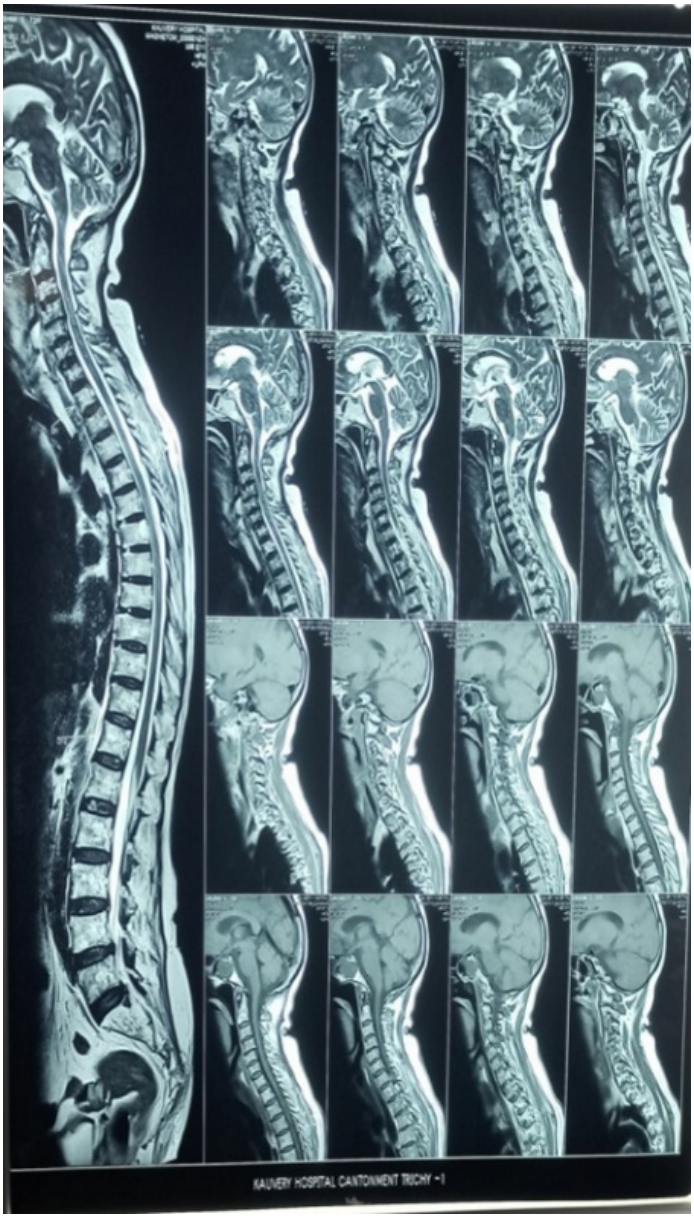

Figure 2: MRI Cervical Spine. We're looking at sagittal and axial MRI images of the cervical and upper thoracic spine, captured through various sequences like T1, T2, and STIR. These images help us assess the spinal cord and vertebral column, checking for structural integrity and looking out for issues like demyelination, inflammation, compression, or other pathologies. The images are taken from different levels and angles to ensure a thorough evaluation.

infection cross-reacts with peripheral nerve antigens, leading to demyelination or axonal degeneration.

In this case, the patient presented with progressive weakness, difficulty walking, and elevated CSF protein with normal glucose levels (albuminocytological dissociation), classic findings for GBS. Nerve conduction studies confirmed demyelinating changes, suggestive of Acute Inflammatory Demyelinating Polyradiculoneuropathy (AIDP), the most common variant of GBS. The elevated WBC count and neutrophilia may reflect an underlying infection, which could have triggered the immune response. Hyponatraemia observed in the patient might result from the Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH), commonly associated with GBS. Although the patient ultimately fulfilled both the clinical and electrophysiological criteria for Guillain-Barré Syndrome (GBS), the initially normal Cerebrospinal Fluid (CSF) protein levels and non-specific early Nerve Conduction Study (NCS) findings created significant diagnostic uncertainty, which delayed the initiation of definitive treatment.

Management usually consists of immunomodulatory treatments like supportive care, Intravenous Immunoglobulin (IVIG) or plasmapheresis; tight checking for respiratory disease of the autonomic system, and secondary infections. Treatment in this patient should concentrate on treating the immune-mediated neuropathy while controlling comorbid diseases like those from COPD, anaemia, and electrolytes. Early physiotherapy and rehabilitation are essential for GBS, given that the forecast of GBS differs greatly, and some patients recover completely, while others have residual neurological impairments. After completing a 5-day course of Intravenous Immunoglobulin (IVIG), the patient was managed in a high-dependency unit with comprehensive supportive care, including Deep Vein Thrombosis (DVT) prophylaxis, nutritional support, and physiotherapy. Neurological improvement became evident by day 10, marked by a gradual recovery of motor strength. By the end of the second week, upper limb strength had improved to 4/5 and lower limb strength to 3/5. The patient was able to sit independently by day 18 and managed a few assisted steps by day 21. Although reflexes remained reduced, they began to return during the third week. At a 6-week follow-up, the patient had regained independent walking ability, with only mild residual distal weakness and no cranial nerve involvement. By the 3-month follow-up, the patient had returned to baseline functional status. This instance emphasises the importance of prompt diagnosis and multidisciplinary management in achieving better results in GBS.

The patient's history of hypertension and Chronic Obstructive Pulmonary Disease (COPD) played a critical role in guiding both treatment decisions and ongoing clinical management. Due to the heightened cardiovascular risk and underlying pulmonary impairment, Intravenous Immunoglobulin (IVIG) was preferred over plasma exchange, given its more favourable safety profile in elderly individuals with respiratory compromise. Careful monitoring of fluid balance was essential to prevent worsening hypertension or cardiac strain. The presence of COPD raised concerns about potential respiratory deterioration, prompting close observation of pulmonary function and early engagement of respiratory therapy. Although mechanical ventilation was not required, the patient experienced a slower recovery trajectory, necessitating extended inpatient rehabilitation. These comorbidities underscore the need for personalised treatment approaches and vigilant supportive care in managing Guillain-Barré Syndrome (GBS) in older adults.

Table 1: CSF Analysis.

Sl. No.	Parameters	Patient value	Normal value
1.	Glucose CSF	67 mg/dL	50-80 mg/dL
2.	Protein CSF	56 mg/dL	15-45 mg/dL

Table 2: Complete blood count.

Sl. No.	Parameters	D1	D6	D26	D28	D31	D34	D38	D46	D49	D51	D54	D57	D58	Normal Range
1.	RBC	2.55	2.80	2.84	2.85	3.05	2.53	2.68	3.19	3.76	3.79	3.82	4.08	4.22	4.5-5.5 M/ cu.mm
2.	HB	10.6	10.2	10	9.2	9.9	8	8.2	9.5	11.1	11.2	11.4	12.3	12.4	12-15 g/dl
3.	WBC	9200	12700	17300	13400	21000	11500	12500	7950	15950	15670	14410	22980	16370	4000-10000 cells/cu.mm
4.	Neutrophil	78.1	87.1	79.7	82	56	78	80.5	63.8	77	59.9	67.2	77.2	70.3	40-80%
5.	Lymphocytes	15.5	6.5	14.8	13	36	15	14.3	25.9	15.6	31.4	25.6	16.2	19.4	25-40%
6.	Eosinophil	1.3	0.1	0.6	2	3	1	0.3	3.9	3.2	1	0.2	0.4	0.2	1-6%
7.	Total RBC	3.39	3.28	3.16	-	-	-	-	-	-	-	-	-	-	4.5-6 ML/10^9
8.	Absolute Neutrophil Count	7200	11100	13800	-	-	-	-	-	-	-	-	-	-	1400-6500 cells/ cu.mm
9.	Absolute Lymphocyte Count	1400	800	2600	-	-	-	-	-	-	-	-	-	-	1200-3400 cells/ cu.mm
10.	Basophil	0	0	0	0.4	0.6	0.3	0.3	0.3	0.2	0.3	-	-	-	1-2%
11.	Platelet Count	5.08	2.91	5.79	2.4	2.25	4.26	5.97	5.75	5.43	4.64	-	-	-	140000-450000/ cu.mm

Paybast S, *et al.* present a rare case of familial Guillain-Barré Syndrome (GBS) associated with confirmed COVID-19 infection, emphasising the role of SARS-CoV-2 as a potential trigger for immune-mediated neurological disorders. Their report highlights the need for heightened clinical vigilance during viral outbreaks, especially when symptoms appear in familial clusters.

In contrast, our case underscores the importance of early recognition and comprehensive, individualised management of GBS in elderly patients with significant comorbidities. While the clinical course followed a typical pattern of Acute Inflammatory Demyelinating Polyneuropathy (AIDP) and responded appropriately to IVIG therapy, the coexisting conditions-particularly COPD and SIADH-introduced additional complexity, necessitating tailored monitoring and supportive care. Compared to similar case reports, this patient's progress was consistent with expected outcomes, though recovery was more gradual. These observations point to the need for further research and case comparisons to better understand how comorbidities may influence the prognosis and recovery trajectory in GBS.

CONCLUSION

This instance underlines the difficulty in properly treating and diagnosing Guillain-Barré Syndrome (GBS), an uncommon but possibly fatal syndrome. Early identification of symptoms, such along with diagnostic methods such nerve conduction, progressive weakness and flexia, is essential for prompt intervention. The presentation of the patient with Elevated CSF protein and demyelinating alterations highlights the need for the detection of GBS subtypes helps to direct treatment modalities such as Intravenous Immunoglobulin (IVIG) or plasma exchange. Managing comorbidities, treating issues like autonomic dysfunction and hyponatraemia, and improving prognosis depend on multidisciplinary attention. With appropriate training, most GBS patients have remarkable recovery except for therapy and rehabilitation. Long-term follow-up is necessary to monitor for residual deficits or recurrence. This particular situation highlights the requirement for awareness and a comprehensive strategy to guarantee the best results in GBS management.

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ABBREVIATIONS

GBS: Guillain-Barré Syndrome; **AIDP:** Acute Inflammatory Demyelinating Polyradiculoneuropathy; **CSF:** Cerebrospinal fluid; **AMAN:** Acute Motor Axonal Neuropathy; **CMV:**

Cytomegalovirus; **SIADH:** Syndrome of Inappropriate Antidiuretic Hormone Secretion.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

AUTHOR'S CONTRIBUTION

Dr. Dhivya Prasath Palaniyappan helped with the study's idea and design, while Angelin Grace Thomas and Ann Jency Arul Durai Singam helped with data collection, analysis, and text drafting.

SUMMARY

A 73-year-old woman with COPD and hypertension arrived with fever, back pain, and increasing weakness. Acute Inflammatory Demyelinating Polyradiculoneuropathy (AIDP), a subtype of Guillain-Barré Syndrome (GBS), was validated by clinical examination, nerve conduction investigations, and CSF analysis demonstrating albuminocytological separation. Lab results showed neutrophilia and hyponatraemia, while MRI showed degenerative spinal abnormalities. In addition to comorbidity management, she was treated with intravenous immunoglobulin, electrolyte correction, antibiotic treatment, and supportive care. A successful recovery resulted from early diagnosis and interdisciplinary management. In order to improve outcomes for senior GBS patients, this example emphasises the difficulties in diagnosing the condition, the need for prompt treatment, and the necessity of thorough monitoring and rehabilitation.

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