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A CASE STUDY ON GUILLAIN-BARRE SYNDROME

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ABSTRACT

Guillain-Barre Syndrome (GBS) is a rare autoimmune neurological disorder where the immune system attacks the peripheral nerves, causing weakness, numbness and paralysis. GBS is more common in older adult (peak incidence occurs between 50 and 70 years of age). This is a case study of AIDP type of GBS admitted to hospital with chief complaints of bilateral hands and feet tingling sensation, weakness and pain since one day back with backache. Based on these symptoms, they underwent NCS which revealed sensory motor demyelinating polyradiculo neuropathy (GBS-AIDP). For this, treatment was initiated with IVIg (2g/kg-20g/day, each bottle over 5 hours) with physiotherapy including pain management. This case highlights the importance of diagnosing GBS early and managing it with a team approach, especially in older patients with other health issues. Ongoing research is crucial to augment comprehension, expedite diagnosis, and refine management strategies for Guillain-Barre Syndrome across diverse patient populations.

KEYWORDS: Guillain-Barre Syndrome (GBS), Acute inflammatory demyelinating polyneuropathy (AIDP), Nerve conduction study (NCS), Intravenous Immunoglobulin (IVIg).

INTRODUCTION

Guillain-barre syndrome (GBS) is a rare neurological disorder in which a person's immune system mistakenly attacks part of their peripheral nervous system- the network of nerve that

carries signals from the brain and spinal cord to the rest of the body, leading to symptoms such weakness, numbness and paralysis.

GBS can onset abruptly and may escalate in severity over hours, days or weeks leading to a point where certain muscles become completely unusable. While some instances of GBS are relativelymild, characterized only by transient weakness, others can result in nearly catastrophic paralysis, rendering the individual incapable of independent breathing. In such situations, the conditions pose a significant threat to life, as it may disrupt breathing, blood pressure, or heart rate. There is no known cure for Guillain Barre Syndrome but several treatment options can ease symptoms and help recovery. Fortunately, the majority of individuals typically recover from even the most critical forms of GBS. However, post recovery, some individuals may still experience residual weakness.[1]

Types

1. Acute inflammatory demyelinating polyneuropathy (AIDP): It damages the nerves 'myelin sheaths' causing weakness that starts in the lower body and progresses up.
2. Acute motor axonal neuropathy (AMAN): It impacts the only movement-linked nerves and is frequently associated with infections such as Campylobacter jejuni.
3. Acute motor-sensory axonal neuropathy (AMSAN): Widespread paralysis and sensory abnormalities are caused by this variant with a prolonged recovery time.
4. Miller-Fisher syndrome (MFS): It is mainly characterized by ophthalmoplegia, ataxia and areflexia which frequently occurs without noticeable limb weakness.[2]

EPIDEMIOLOGY

It is impossible to overlook the impact that GBS has on medical facilities around the world. The estimated incidence of GBS ranges from 0.4 to 0.2 per 100,000 populations based on the population-based studies from North America and Europe despite the fact that it is classified as an uncommon disorder that affects certain minor populations. According to the estimation, GBS is diagnosed in male patients more often than in female patients (1.5:1).

Because of variations of exposure to infectious organisms, estimates of annual incidence (per 100,000) are highest in Chile (2.12) and Bangladesh (3.25) and lowest in Japan (0.44), China (0.67), Tanzania (0.83) and Finland (0.84). Seasonal changes are explained and GBS surges have been documented after viral outbreaks, particularly those involving Zika virus and

Campylobacter jejuni. GBS is more common in older adult (peak incidence occurs between 50 and 70 years of age).[3]

CAUSES

Previous reports have indicated a correlation between certain conditions and the development of GBS and its variations. According to reports, the illness is classified as a unique type of neuropathy that manifests as immune-mediated, post-infection sequelae. According to earlier research using animal models, molecular mimicry shows a substantial correlation with the progression of the disease. It has been previously documented that approximately 1 in 1000 patients having gastrointestinal infection-causing bacteria **Campylobacter jejuni** predisposes to develop GBS.[4]

In addition to Campylobacter jejuni, 0.6to2.2 cases of **Cytomegalovirus** (viral CMV) per 1000 persons developed GBS especially AIDP (upto 70%), AMSAN, AMAN (7%) and MFS (6%). Dirlikov et al. previously reported that numerous cases of GBS were detected and reported after **Zika virus** infection during relevant prior pandemic.

According to previous reports shown that, aside from viral infections, factors like certain medications and surgeries can also triggers GBS. Although there was increase in GBS cases during the 1976 **H1N1 vaccination** campaign, rates eventually decreased to roughly one case per million immunizations. Interestingly, research indicates that the risk of contracting GBS is seven times greater following a real influenza illness than following vaccination.[5]

CLINICAL PRESENTATION

The hallmark feature of GBS is ascending weakness, which usually starts in the lower extremities and moves proximally to cause symmetrical flaccid paralysis. Functional mobility is made more difficult by concurrent sensory abnormalities, such as paresthesia or numbness, which frequently accompany motor dysfunction.[6]

In addition to motor and sensory defects of GBS may include autonomic dysfunction, which can show up as respiratory compromise, gastrointestinal dysmotility and cardiovascular instability. In order to prevent hemodynamic instability and cardiac arrhythmias, cardiovascular symptoms such as tachycardia, bradycardia and blood pressure swings may call for the close observation and supportive measures.[7] The clinical trajectory of GBS is

marked by rapid progression to maximal deficits within weeks, followed by plateau phase and subsequent recovery over months to years.[8]

PATHOPHYSIOLOGY

The pathophysiology of GBS involves a series of immune mediated events triggered by an antecedent infection or immune stimulants leading to a nerve damage and clinical symptoms. It is explained in the following manner:

Antecedent infection/ Immune trigger (Camylobactorjejuni, CMV, Zika virus, Vaccination)

⬇

Molecular Mimicry (Pathogen antigens resemble peripheral nerve gangliosides)

⬇

Autoantibody formation (Anti-ganglioside antibodies, complement activation)

⬇

Immune- mediated nerve damage

- AIDP: Segmental demyelination (Macrophage and T-cell mediated)
- AMAN/AMSAN: Direct axonal degeneration via antibody/ complement attack

⬇

Disrupted nerve conduction (Slowed conduction, conduction block)

⬇

Clinical manifestations

Ascending flaccid paralysis, areflexia, sensory symptoms and autonomic dysfunction

DIAGNOSIS

GBS diagnosis involves a combination of clinical evaluation, nerve function tests, and cerebrospinal fluid analysis and assess symptoms like muscle weakness, sensory changes (tingling, numbness) and reflexes and may order nerve conduction studies (NCS) and electromyography (EMG) to evaluate nerve and muscle electrical activity.[9]

TREATMENT

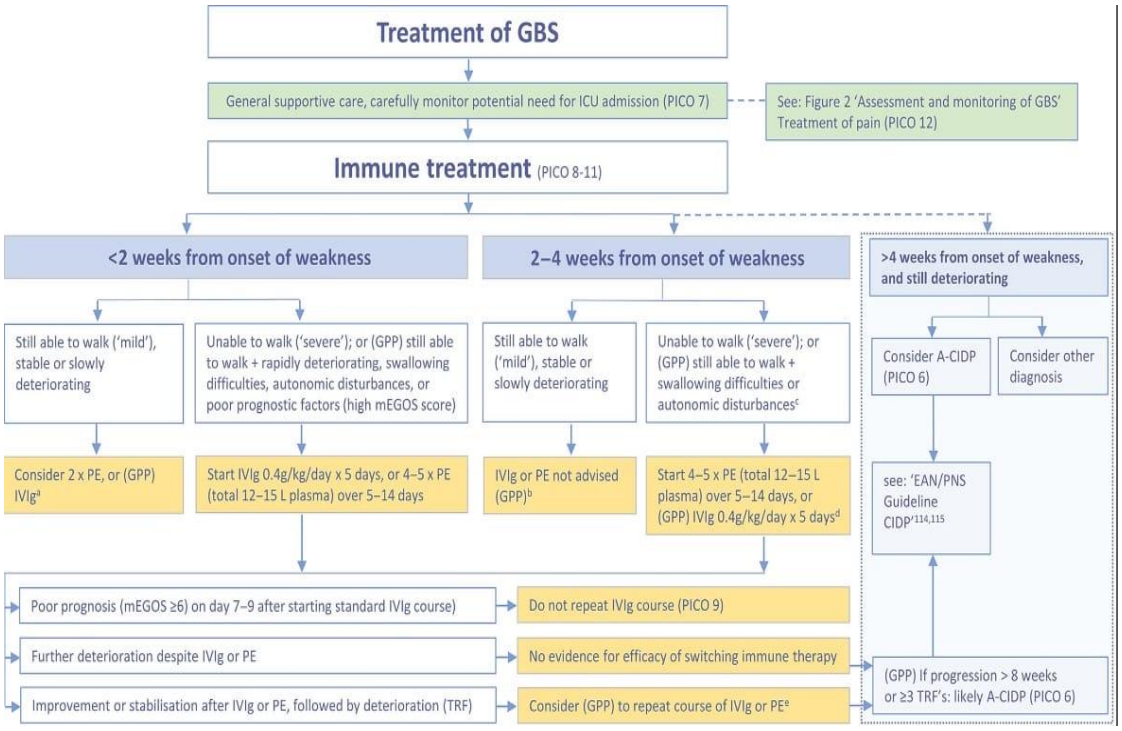

Fig. 1: Treatment of GBS.

Pharmacological treatment

- **Interavenous Immunoglobulin (IVIg) and Plasma exchange(PE)(PICO 8 and 9):** These are the primary treatments for patients who cannot walk on their own. It is best to start within 2 weeks and PE within 4 weeks of the onset. Quick action is important. PE includes 4 to 5 exchanges, totaling 12 to 15 liters of plasma, over 1 to 2 weeks. The IVIg dose is o.4 g/kg/day for 5 days. There is a weak recommendation for use in mildly affected, but quickly worsening patients (GBS-DS grade2). It is strongly advsed against combining PE with IVIg.
- **Corticosteriods (PICO 10)**: Oral Corticosteriods are strongly discouraged due to their lack of effectiveness and potential harm, sach as a delaying recovery. IV methylprednisone is a weakly advised against, whether used alone or with IVIg.
- **Other Immunotherapies (PICO 11):** Treatments besides IVIg, PE, or corticosteroids, such as eculizumab, alemtuzumab, mycophenolate mofetil, interferons, cyclophosphamide, and immuno adsorption, are strongly discouraged due to insufficient evidence, possible harm, or no proven benefit.
- **Pain management (PICO 12):** The task force weekly recommend gabapentinoids (Gabapentin, pregabalin), tricyclic antidepressants,or carbamazepine for managing

neuropathic pain in GBS. High dose corticosteroids for pain are weakly discouraged. Opiods can be used cautiously for severe pain. It is important to assess and differentiate between neuropathic and nociceptive pain to determine the right treatment.

- **Fatigue Treatment:** There is no effective pharmacological treatment for fatigue. Amantadine is weakly discouraged for reducing fatigue.

Non pharmacological treatment

- **Rehabilitation:** Early and ongoing physiotherapy, occupational therapy, and speech / swallowing therapy are cric for functional recovery and minimizing complications.
- **Monitoring and supportive care:** regular assessments of respiratory function using clinical scores (like mEGRIS), spirometry and monitoring autonomic signs are essential. Decisions about intensive care admission should be based on risk evaluations.
- **Psychological and emotional support:** Adress the emotional needs of both patients and caregivers throughout the recovery.[10]

CASE PRESENTATION

A 67 year old male patient was admitted to Basaveshwara Medical College & Hospital (BMCH), Chitradurga (Karnataka) with the chief complaints of bilateral hands and feet tingling sensation, weakness and pain since one day back with backache. Patient was apparently normal till one day ago then he developed pain and tingling sensation of bilateral feet progressive to hands, insidious in onset gradually progressive, non aggrevating or relieving factors were noted. Patient also had complaints of backache since one day, insidious onset, gradually progressive, dragging type. For above complaints patient was admitted to neurology department in BMCH. No history of sensory loss, incoordination, bowel and bladder incontinence, difficulty in gripping sandals and fever.

Past History

- Known case of Type2 DM on medications-Tab. Dapagliflozin/Metformin (10/1000) 0-0-1/2 and Tab. Voglibose GM 3 0-0-1/2
- Known case of IHD on medication- Tab. Ecospirin AV 75/20 (0-0-1)

Family History: Nothing significant

On examination

Patient was conscious and oriented

- Bp-140/100mmHg.

- PR-86bpm
- SPO2-97% @RA
- GRBS-235mg/dl
- Plantar- right: absent, left: flexor
- Reflexes-mute
- Tone-normal
- Power-upper limb-3/5, lower limb-3/5
- Pupils bilaterally equally reactive to light (BERL)
- Hand grip-normal
- Sensory examination-normal
- Cerebellar signs- absent

Laboratory investigations

- Neutrophils: 80.7 % (40-75%)
- Lymphocytes: 12.6% (20-45%)
- Eosinophils: 0.3% (1-6%)
- PCV: 25.8% (39-54%)
- CRP: 67.3mg/L (0-10mg/L)
- Sodium: 127mmol/L (135-155mmol/L)
- Chloride: 89mml/L (98-107mml/L)
- D Dimer: 960ng/ml (90-500ng/ml)
- Blood urea: 66mg/dl (10-45mg/dl)

Additional tests

- X-ray Chest: Right lower lobe consolidation
- 2D Echo: Normal
- NCS: Suggestive of sensory motor demyelinatingpolyradiculo neuropathy (GBS-AIDP)

TREATMENT

Table 1 presents the details of medications provided for the treatment. Patient was initiated with IVIg (2g/kg-20g/day, each bottle over 5 hours) as the main therapy to stop immune mediated nerve damage and promote recovery in GBS with aggressive physiotherapy. Pregabalin + Methylcobalaminewas used for neuropathic pain and nerve healing. Meanwhile he developed shortness of breath for which X Ray was done suggestive of LRTI and was

treated with antibiotics. Pulmonary embolism treated with LMWH and continued his current medications for his known cases. Table 2 presents the details of discharge medications.

Table 1: Treatment regimen.

Sl.No	MEDICATION	DOSE	ROUTE	FREQUENCY
1	IVF Normal saline	50ml/hr	IV	4 days
2	IV Immunoglobulin	2g/kg- 20g/day each bottle over 4 hrs	IV	5 days
3	Inj. Ceftriaxone	1g	IV	BID for 5days
4	Tab. Azithromycin	500mg	PO	OD for 4 days
5	Inj. Pantoprazole	40mg	IV	OD for 5 days
6	Inj. LMWH (Low molecular weight heparin)	0.5mg	SC	BID for 5 days
7	Tab.Montelukast+ levocetirizine	10mg+ 5mg	PO	OD for 5 days
8	Tab. Voglibose+ Glimepiride+ Metformin	0.3 mg+ 2mg+ 500mg	PO	½ tab HS for 6 days
9	Tab. Dapagliflozin+ Metformin	10mg + 1000mg	PO	½ tab HS for 6 days
10	Tab. Aspirin+ Clopidogrel+ Atorvastatin	75mg+ 75mg+ 20mg	PO	OD for 6 days
11	Tab. Pregabalin+ Methylcobalamin	75mg+ 750mcg	PO	OD for 6 days
12	Syp. Liquid Parrafin+Milk of Magnesia+ Sodium Picosulfate	4 tsp (20ml)	PO	OD for 6 days
13	Aggressive Physiotherapy	-	-	-

**BID: Twice a day, OD: Once a day, HS: At bed time.*

Table 2: Discharge medication.

Sl.No	MEDICATION	DOSE	ROUTE	FREQUENCY
1.	Tab. Apixaban	2.5mg	PO	BID (To continue)
2.	Tab Voglibose+Glimepiride+ Metformin	0.3mg+1mg+500mg	PO	½ tab HS (To continue)
3.	Tab Dapagliflozin+ Metformin	10mg+ 1000mg	PO	½ tab HS (To continue
4.	Tab. Atorvastatin+ Aspirin	20mg+75mg	PO	OD (To continue)
5.	Tab. Melatonin	5mg	PO	OD (To continue)
6.	Tab. Amlodipine	5mg	PO	OD (To continue)
7.	Tab. Pregabalin+ Methylcobalamine	75mg+750mcg	PO	OD for 10 days
8.	Syp. Liquid Parrafin+Milk of Magnesia+ Sodium Picosulfate	4 tsp (20ml)	PO	SOS

**BID: Twice a day, OD: Once a day, HS: At bed time*

DISCUSSION

GBS is an acute immune related polyradiculo neuropathy that leads to fast developing, loss of reflexes and varying sensory issues. The current case involves a 67 years old man with a diabetes and a heart disease who suddenly experience tingling and weakness in his limbs. Nerve conduction studies later confirmed this as an acute inflammatory demyelinating polyneuropathy (AIDP). The patient was effectively treated with IVIg and supportive care. It often occurs after an infection triggers an immune response that attacks peripheral nerves through molecular mimicry. The most common infectious agents linked to GBS include Campylobacter jejuni, Cytomegalovirus, EBV and Zika virus.[9,11,12] The immune response produces antibodies against gangliosides such as GM1, GD1a and GQ1b. The result is either demyelination or damage to the axons, depending on the type of GBS.[13] In this patient. Demyelinating form (AIDP) aligns with the most common subtype seen in western countries.[14]

The diagnosis of GBS mainly relies on clinical assessment, along with electrophysiological studies and cerebrospinal fluid (CSF) analysis. Nerve conduction studies showing prolonged distal latencies, slope conduction velocities and conduction block confirmed demyelination.[7] In this case, the nerve conduction findings matched those of sensory motor demyelination polyneuropathy, further supporting the diagnosis of AIDP. Managing GBS focuses on stopping immune damage and supporting essential functions. The both IVIg and plasmapheresis, are the first line treatments with similar effectiveness and should ideally start within 2 weeks of symptom onset.[15,16] In this case, the use of IVIg let to a significant clinical improvement, which is consistent with the evidence from randomized control trials showing a quicker recovery and a reduced need for a mechanical ventilation with IVIg therapy.[17] Pain and fatigue are common but often over looked problems in GBS. Neuropathic pain affects upto 70% of patients can be effectively treated with pregabalin, gabapentin or tricyclic antidepressants.[18] Our patient had neuropathic pain, which was managed with pregabalin and methylcobalamin supplementation. Supportive measures such as monitoring respiratory functions, preventing blood clots and aggressive physiotherapy are crucial in preventing complications like deep vein thrombosis and respiratory failure.[19] The outlook for GBS is generally positive. About 80% of patients regain their ability to walk independently within six months.[20] However, factors like older age, severe initial weakness, and the need for respiratory support can lead to worse outcomes.[21] Early diagnosis and prompt IVIg treatment in our patient likely aided his steady recovery and improved function.

This case highlights the importance of diagnosing GBS early and managing it with a team approach, especially in older patients with other health issues. Timely IVIg therapy, along with careful monitoring and rehabilitation support, can greatly enhance clinical outcomes and lower the chances of long-term disability.

CONCLUSION

Guillain-Barre Syndrome is a rare and potentially fatal neuropathy that necessitates prompt identification and collaborative care to optimize outcomes. This case emphasizes the necessity of considering Guillain-Barre Syndrome in patients exhibiting sudden and progressive limb weakness, especially among older adults with preexisting health conditions such as diabetes and cardiovascular disease. Early diagnosis and prompt initiation of intravenous immunoglobulin therapy, coupled with continuous monitoring, efficient analgesia, and rigorous physiotherapy, are essential for mitigating complications and facilitating recovery. A cooperative approach helps a lot of patients become independent again and lowers the chance of long-term disability. Continuous follow-up and rehabilitation are essential for managing persistent symptoms and promoting long-term health, highlighting the significance of care from diagnosis to recovery. Ongoing research is crucial to augment comprehension, expedite diagnosis, and refine management strategies for Guillain-Barré Syndrome across diverse patient populations.

REFERENCES

1. https://www.ninds.nih.gov/health-information/disorders/guillain-barre-syndrome
2. Dimachkie MM, BarohnRJ: Guillain-Barre Syndrome and variants. NeurolClin, May 2013; 31(2): 491-510.
3. Bellanti R, Rinaldi S. Guillain-Barre Syndrome: A comprehensive review. Eur J Neurol, 2014; 31: 16365.
4. Rahman RS, Bauthman MS, Alanazi AM, Alsillah NN, Alanazi ZM, Almuhaysin MI, et al. Guillain–Barré syndrome: pathophysiology, etiology, causes, and treatment. Int J Community Med Public Health, 2021; 8: 3624-8.
5. JastiAK, Selmi C, et al. Guillain-Barré syndrome: causes, immunopathogenic mechanisms and treatment. AERM, 2016; 12,11: 1175-89.
6. Kuitwaard K, Bos-Eyssen ME, Blomkwist-Markens PH, van Doorn PA Recurrences, vaccinations and long-term symptoms in GBS and CIDP. J PeripherNerv Syst., 2009; 14: 310–5.

7. Hadden RD, Cornblath DR, Hughes RA, et al. Electrophysiologicalclassification of Guillain-Barré syndrome: clinical associations and outcome. Plasma Exchange/SandoglobulinGuillain-Barré Syndrome TrialGroup. Ann Neurol, 1998; 44: 780–8.
8. Elendu C, Osamuyi EI, Afolayan IA, Opara NC, Chinedu-Anunaso NA, Okoro CB, Nwankwo AU, Ezidiegwu DO, Anunaso CA, OgbuCC, Aghahowa SO, Atuchukwu CS, Akpa EU, Peterson JC. Clinical presentationandsymptomatology of Guillain-Barré syndrome: A literature review. Medicine, 2024; 103: 30(e38890).
9. Willison HJ, Jacobs BC, van Doorn PA. Guillain-Barré syndrome. Lancet, 2016; 388(10045): 717-27.
10. Van Doorn PA, Van den Bergh PYK, Hadden RDM, et al. European Academy of Neurology/Peripheral Nerve Society Guideline on diagnosis and treatment of Guillain–Barré syndrome. J Peripher Nerv Syst., 2023; 28(4): 535-563.
11. Sejvar JJ, Baughman AL, Wise M, Morgan OW. Population incidence of Guillain-Barré syndrome: a systematic review and meta-analysis. Neuroepidemiology, 2011; 36(2): 123–33.
12. Dirlikov E, Major CG, Medina NA, et al. Guillain–Barré Syndrome and Zika Virus Infection in Puerto Rico. N Engl J Med., 2016; 375(16): 1501–3.
13. Willison HJ, Yuki N. Peripheral neuropathies and anti-glycolipid antibodies. Brain, 2002; 125(12): 2591–625.
14. McGrogan A, Madle GC, Seaman HE, de Vries CS. The epidemiology of Guillain–Barré syndrome worldwide: a systematic literature review. Neuroepidemiology, 2009; 32(2): 150–63.
15. Hughes RA, Swan AV, van Doorn PA. Intravenous immunoglobulin for Guillain–Barré syndrome. Cochrane Database Syst Rev., 2014; 2014(9): CD002063.
16. Plasma Exchange/Sandoglobulin Guillain-Barré Syndrome Trial Group. Randomised trial of plasma exchange, intravenous immunoglobulin, and combined treatments in Guillain–Barré syndrome. Lancet, 1997; 349(9047): 225–30.
17. Kuwabara S, Mori M, Misawa S, et al. Intravenous immunoglobulin therapy for Guillain–Barré syndrome: efficacy and mechanisms. Clin Exp Neuroimmunol, 2018; 9(2): 99–106.
18. Ruts L, Drenthen J, Jongen JL, et al. Pain in Guillain–Barré syndrome: a long-term follow-up study. Neurology, 2010; 75(16): 1439–47.
19. Leonhard SE, Mandarakas MR, Gondim FAA, et al. Diagnosis and management of Guillain–Barré syndrome in ten steps. Nat Rev Neurol, 2019; 15(11): 671–83.

20. Kuwabara S, Yuki N. Axonal Guillain–Barré syndrome: concepts and controversies. Lancet Neurol, 2013; 12(12): 1180–8.
21. Walgaard C, Lingsma HF, Ruts L, van Doorn PA, Steyerberg EW, Jacobs BC. Prediction of respiratory insufficiency in Guillain–Barré syndrome. Ann Neurol, 2010; 67(6): 7.