Review Article

Autoimmunity and Neuropathy: A Review of Guillain Barre Syndrome

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ABSTRACT

Guillain-Barre Syndrome (GBS) is an acute, immune-mediated neuropathy that often presents rapid-onset ascending paralysis and remains the leading global cause of acute flaccid paralysis. This article reviews the historical development of GBS, the role of antecedent infections such as *Camphylobacter jejuni* in disease pathogenesis the immunologic mechanisms including molecular mimicry and autoantibody-mediated nerve injury. Main subtypes, such as Acute Inflammatory Demyelinating Polyradiculoneuropathy (AIDP), Acute Motor Axonal Neuropathy (AMAN) and Miller Fisher Syndrome are described alongside their typical presentations. Epidemiological trends reveal a variable incidence worldwide, with most cases triggered by recent respiratory or gastrointestinal infection. Diagnosis relies on clinical criteria complimented by cerebrospinal fluid analysis, nerve conduction studies and detection of anti-ganglioside antibodies. Standard treatment includes supportive care, intravenous immunoglobulin, plasma exchange. Two illustrative case reports highlight common presentation, diagnostic challenges and outcomes in paediatric and familial forms. Advances in neuroimmunology, targeted therapies and rehabilitation are paving the way for improved prognosis and quality of life for GBS patients. This review synthesizes current knowledge and emerging approaches, emphasizing the importance of early recognition and multidisciplinary care.

Keywords: Guillain Barre Syndrome, Acute flaccid paralysis, Molecular mimicry, Immune mediated neuropathy.

INTRODUCTION

The most significant contribution of Jean Baptiste Octave Landry was the first description of ascending paralysis, which is now known as Guillain-Barré syndrome. A classic case of Landry's ascending paralysis was a 43-year old person with weakness, fever, pain and tingling in his toes and hands.[1] Presently acknowledged as the primary cause of acute ischemic paralysis worldwide, Guillain-Barré Syndrome (GBS) was first described by French neurologists Georges Guillain, Jean Alexandre Barré, and André Strohl in 1916.[2] Acute flaccid paralysis is most commonly caused by Guillain-Barré syndrome (GBS), an inflammatory disease of the PNS that affects 1-2 out of every 100,000 person-years worldwide each year.[3]

There are two subtypes of Guillain-Barré syndrome (GBS), a post-infectious immune-mediated neuropathy: axonal and demyelinating.[4] 50–70% of cases of GBS occur 1-2 weeks after a respiratory or gastrointestinal infection, or another immune stimulus that triggers an abnormal autoimmune response that targets peripheral nerves and their spinal roots. Studies of outcome in Guillain Barre Syndrome suggest that at the end of one year from onset of neuropathy 65% of patients achieve an almost complete cure so that they regain the ability to perform manual work and the 35% who do not.[5]

The first symptoms usually include limb pain, tingling, weakness, and numbness.[6] With different patterns of sensorimotor and motor impairments, respectively, Acute Inflammatory Demyelinating Polyradiculoneuropathy (AIDP) and Acute Motor Axonal Neuropathy (AMAN) are the most common forms. Additionally, rarer variants such as Miller Fisher Syndrome (MFS), characterized by ataxia, areflexia and ophthalmoplegia.[7] About two-thirds of GBS patients have an infection prior to neurological symptoms.[8] Although the precise etiology of GBS is still unknown, one theory is that an antecedent infection triggers the immune system and causes the production of cross-reactive antibodies against the nervous system, a process known as molecular mimicry.[9]

Two well-established, evidence-based immunomodulatory treatments for GBS are intravenous immunoglobulins (IVIg) and plasma exchange (PE).[10] However, a considerable percentage of GBS patients do not respond well to either therapy, highlighting the urgent need for new and more effective therapies. [11]

Both viruses and bacteria have been linked to the pathophysiology of GBS. Only *C. jejuni*, a major global cause of gastroenteritis, is certain to be the cause of GBS among the many microbial infections.[12] All GBS patients require close observation and can gain from early initiation of targeted treatment and supportive care.[13] More precise diagnosis of axonal GBS has been made possible by recent technological developments.[14] Because it can take years for Guillain-Barré syndrome to fully resolve, it can have a significant impact on a patient's life and family.[15]

This article aims to provide a clear overview of Guillain Barre Syndrome (GBS), from its pathogenesis to clinical outcomes and latest ways to diagnose and treat it. By looking at recent data and medical advances, it adds useful information that can help to improve early treatments and overall care for people with GBS.

History

The Somme River offensive was underway in France at the end of August 1916, during World War I, with the French and British engaged in the bloodiest battle in history against the Germans. Due to increasing weakness, two soldiers were admitted to the French Sixth Army's Neurological Service two weeks apart. They encountered three doctors there: André Strohl (29 years old), Jean Alexandre Barré (36 years old), and Georges Guillain (40 years old) which introduced the GBS. [16]

Octave Landry first described the clinical characteristics of GBS in 1859. But the Guillain-Barre Syndrome was originally described almost 100 years ago by french neurologists Georges Guillain, Jean-Alexender Barre and Andre Strohl. Guillain and Barre´ described the syndrome (an acute inflammatory paralytic neuropathy) in 1916. The description was provided after working closely with physician Andre Strohl (1887–1977), who had tested two French soldiers electrophysiologically and all these three authors' findings were published in a French medical journal that same year.[17] Guillain conducted a review of GBS in 1943 and documented eighteen clinical observations and Landry (1826–1865) had reported a case of a 43-year-old man who experienced acute ascending paralysis and passed away in a matter of days in 1859, which was half a century prior.[18]

GBS generates a variety of toxins, such as CAMP factor and beta-haemolysin/cytolysin, which lead to direct cytotoxic tissue damage. By mimicking human surface antigens and avoiding complement deposition, GBS evades the host immune response and interferes with host opsonization and phagocytosis.[19] Although the exact cause and pathophysiology of GBS are unknown, it is believed to be an immune-mediated process that arises from the production of inflammatory cells and autoimmune antibodies that react with epitopes on peripheral nerves and roots, causing demyelination, axonal damage, or both.[20] It is believed that a number of antigenic stimuli, including bacterial or viral infections, especially those caused by Campylobacter jejuni, trigger this immune response.[21]

Pathophisiology

Within a few days, Guillain-Barré syndrome (GBS), one of the most remarkable neurological conditions, can leave a person completely paralyzed and dependent on a ventilator. The rapid onset of limb weakness accompanied by loss of tendon reflexes and a normal cell count and elevated cerebrospinal fluid protein was identified by Guillain, Barré, and Strohl as a distinct clinical syndrome.[22] Molecular mimicry is the dominant theory, according to which microbial antigens that resemble parts of peripheral nerve tissues structurally can cause an autoimmune reaction. This autoimmune attack causes inflammation, which is followed by demyelination or axonal damage, which results in the characteristic motor and sensory impairments seen in GBS.[23] The axolemma and other peripheral nerve components contain high densities of gangliosides, which are the target of serum antibodies in a subset of GBS patients.[3] Cross-reactivity and the molecular mimicry phenomenon are linked to the development of GBS.[24]

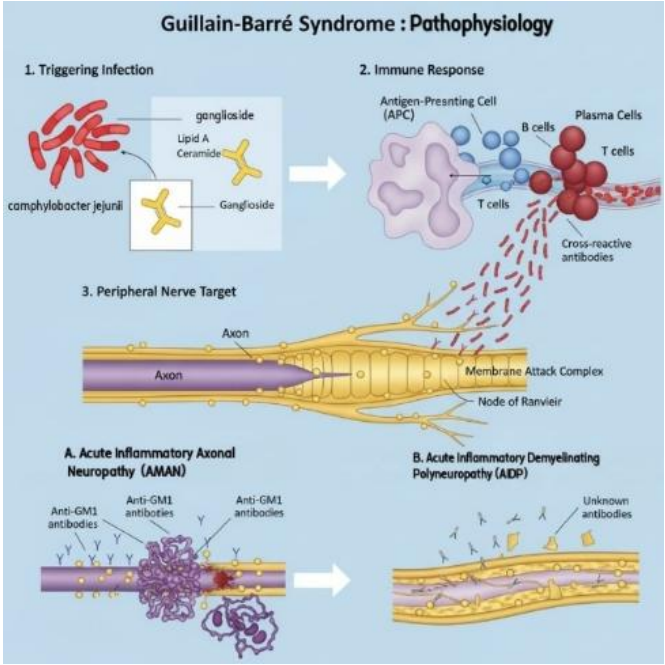

Figure 1: Pathophysiology of Guillain Barre Syndrome

Table 1: Major steps involve in pathophysiology of GBS

Microbial Trigger	Infectious agent with antigens that structurally resemble peripheral nerve components initiate the cascade
Molecular Mimicry	The immune system mistakenly identifies nerve tissues as foreign due to structural similarieties with microbial agents
Autoimmune Attack	Cross- reactivity leads to inflammatory response targeting peripheral nerve components
Nerve Damage	Demyelination or axonal damage results in characteristics motor and sensory impairements

Triggers

The gastrointestinal infection-causing pathogen *Campylobacter jejuni* has been shown to predispose humans to developing GBS. This is likely because the gangliosides involved in peripheral nerve cell structure and the lipooligosaccharide component of the bacterium's outer membranous layer have similar antigenicity, which in turn causes a similar antibody-mediated attack against these nerve cells.[25]

Most Important Pathogen such as

1) *Cytomegalovirus*
2) *Epstein- Barr virus*
3) *Influenza A*
4) *Mycoplasma*

5) *Pneumoniae*

6) *Haemophilus influenza*

7) *Hepatitis (A, B and C)*

8) *Zika virus*[15]

The type of antecedent infection, the development of pathogenic cross-reactive antibodies via molecular mimicry and the location of the target gangliosides affect the subtype and severity of the illness. Research is ongoing to further understand the pathogenesis of the disorder, find new biomarkers and develop more effective and specific treatments.[26] The incidence of dengue virus infections has increased over the past few years. West nile fever and dengue virus have been associated with neurological diseases, including GBS, but little has been published about this relationship.[27]

GBS is predominant pathogen of perinatal infection in Western countries and can cause serious harm to maternal and child health. In pregnant women, the manifestations of GBS commonly include asymptomatic bacteriuria, urinary tract infections, bacteremia, chorioamnionitis and placental abruption which responsible for adverse events such as premature birth and stillbirth.[28]

Table 2: Proposed infectious intecedents of GBS

Viruses	Bacteria
Adenovirus	*Camphylobacter jejuni*
Corona virus	*Haemophilus influenzae*
Cytomegalovirus	*Helicobacter pylori*
Epstein-Barr virus	*Mycoplasma pneumoniae*
Echo virus	*Salmonella typhosa*
Hepatitis A, B, C	*Shigella*
Herpes simplex	*Yersinica*
Herpes zoster	
Influenza A and B	
Mumps	
Parainfluenza	
Vaccinia	
West Nile virus	

TYPES OF GUILLAIN BARRE SYNDROME

1) **Acute Inflammatory Demyelinating Polyneuropathy (AIDP**)

One of the most frequent causes of acute flaccid paralysis in the world is acute inflammatory demyelinating polyneuropathy (AIDP).[29] When compared to AMAN, children with AIDP, the most prevalent subtype of GBS, recover better after three months.[30] AIDP typically manifests as progressive symmetric muscle weakness, typically in the lower extremities, accompanied by paresthesia of the hands and feet and either absent or reduced deep tendon reflexes.[9] Nevertheless, in certain AIDP patients, complement accumulates along the outer surface of Schwann cells at the peripheral nerves without any T-cells, indicating that autoantibodies to myelin antigens trigger complement and result in demyelination.[31]

2) **Acute Motor Axonal Neuropathy (AMAN**)

Through nerve conduction studies, the uncommon variant known as acute motor axonal neuropathy (AMAN) is confirmed.[32] Anti-ganglioside antibodies, like anti-GM1, which are known to trigger complement-mediated axonal damage, are linked to prior C. jejuni infections, which are strongly associated with AMAN.[33] Because of higher rates of C. jejuni infection, AMAN, the pure motor variant without sensory or autonomic nerve involvement, is more common in East Asia, Central America, and South America.[34] An acute motor axonal neuropathy (AMAN) diagnosis was validated by electromyography.[35]

3) **Acute Motor-Sensory Axonal Neuropathy (AMSAN)**

The rare but severe form of Guillain-Barré syndrome (GBS) known as acute motor-sensory axonal neuropathy (AMSAN) makes up less than 5%–10% of all GBS cases.[36] Griffin and colleagues verified that the acute motor-sensory axonal neuropathy (AMSAN) pattern of GBS was present.[37]

4) **Miller Fisher Syndrome (MFS)**

Guillain Barré syndrome (GBS), an acute post-infectious paralytic illness brought on by an inflammatory disruption of peripheral nerve integrity and function, has a clinical variant known as Miller Fisher syndrome (MFS).[38] In approximately 80% of cases, MFS manifests as the classic triad of symptoms: osteotendinous areflexia, ataxia, and ophthalmoparesis. [39]

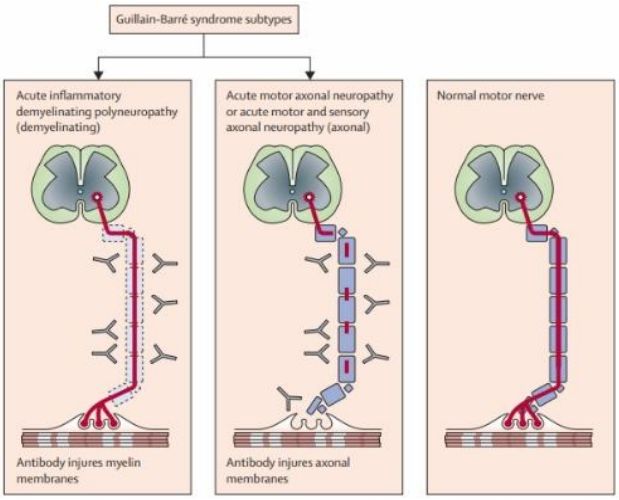

Figure 2: Major GBS subtypes GBS

Symptoms

- Most frequently reported symptoms such as

1) Fever

2) Cough

3) Sore throat

4) Upper respiratory symptoms[6]

- Cranial nerves are affected in over 50% of patients leading to
    1) Facial weakness
    2) Swallowing difficulties
    3) Eye muscle weakness
    4) Paralysis.[40]

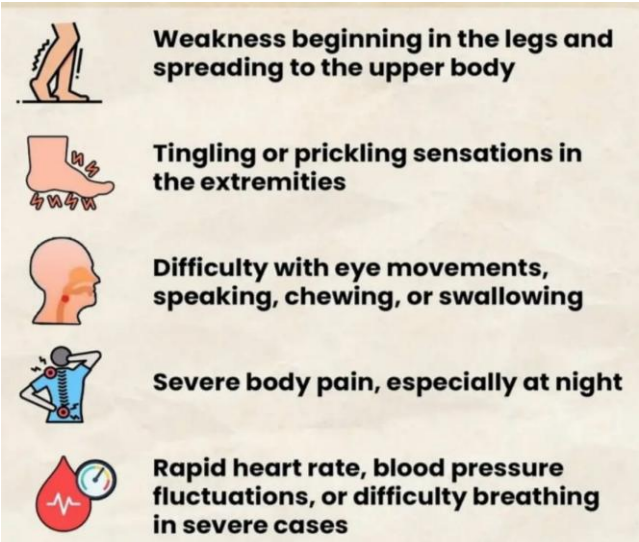

Figure 3: Symptoms of GBS

Patients with the AIDP subtype of GBS typically have weakness that starts in the legs and spreads to the arms as well as decreased or absent reflexes.[40] In most cases, a patient with GBS will have tingling dysesthesias in their extremities along with weakness. This weakness is prominent in the proximal muscle; legs are more often affected than arms.[41] Chronic fatigue is a common symptom in neurological diseases and can be defined based on its location in the body. Guillain-Barre Syndrome (GBS) is the most common cause of acute and severe generalized peripheral neuropathic weakness.[42] Treatment shortens the duration of GBS, decreases the amount of time needed to receive mechanical respiratory assistance, and lessens the overall severity, early diagnosis is crucial, even though the electrical abnormalities may not be widespread enough for a definitive diagnosis in the first two weeks.[43] Other common symptoms include back or limb pain and distal numbness. Some patients may also report altered gait or progressive limb weakness. [44]

DIAGNOSIS

It was based on diagnostic criteria of the National Institute of Neurological and Communicative Disorders and Stroke (NINCDS). Using specific criteria for demyelination and primary axonopathy, standard EMG examination with EMG/NC study apparatus.[45] When weakness is preceded by an infection within 1-3 weeks of onset, the diagnosis of GBS is frequently simple. However, in certain patients, the diagnosis can be more challenging, particularly if pain is present prior to the onset of weakness or if weakness initially only affects the legs.[46] In patients with suspected GBS, the diagnosis of GBS is more likely if there is a history of recent (within the previous 6 weeks) diarrhoea (sensitivity 13%–18%, specificity 89%–100%),49-53 Campylobacter infection,54 respiratory infection (sensitivity 21%–68%, specificity 59%–98%),49-53 fever (specificity up to 100%)49, 52 or influenza-like illness (specificity up to 100%).48, 53 However, around one third of GBS patients report none of these.[47] A number of internationally-accepted schemes and guidelines exist for the diagnosis and classification of GBS .[48] The clinical features of ascending weakness and sensory loss, along with hyporeflexia or areflexia, should raise suspicion of GBS.[49]

Cerebrospinal fluid (CSF) Analysis

Proteins found in cerebrospinal fluid (CSF) show promise in revealing crucial details about the pathomechanisms underlying GBS. A number of neurological disorders have been studied using the proteomic approach, which aims to identify and quantify the entire protein content (proteome) of the CSF in order to identify disease-related biomarkers.[50] Increased protein levels and a normal white blood cell count are common findings in the cerebrospinal fluid (CSF) of GBS patients. This condition is known as "cytoalbuminologic dissociation," and previous research examined the frequency of these abnormalities in GBS based on the length of the disease.[51]

Nerve Conduction Study (NCS)

Although Guillain-Barré syndrome is a clinically diagnosed disorder, nerve conduction studies (NCS) can separate axonal from demyelinating subtypes, support the diagnosis, and possibly even predict prognosis.[52] Acute inflammatory demyelinating polyneuropathy (AIDP) and acute motor axonal neuropathy (AMAN) are two additional conditions that are frequently distinguished from classical GBS using NCS. There have been several NCS criteria proposed for the diagnosis of GBS in the past. In one study, the sensitivity of older criteria for the diagnosis of AIDP ranged from 21% to 72%, while newer criteria had a higher proportion of axonal variants[53]

Anti-Ganglioside Antibodies

Some gangliosides linked to autoimmune diseases of the nervous system include GM1, GD1a, GalNAc-GD1a, GM1b, GD3, CD1b, GT1a, and GQ1b. When anti-ganglioside antibodies attach to the corresponding gangliosides, the complement system is triggered, which causes damage to the brain, including myelin loss and axonal degeneration[54]. Anti-ganglioside antibodies in serum were measured using ELISA. Tests were conducted for GT1a, GQ1b, GM1, GD1a, and GD1b.[55]

Electromyography (EMG)

Low amplitude or absent sensory nerve action potentials, an abnormal F wave, an absent H reflex, and other less common abnormalities are electrodiagnostic findings suggestive of GBS.[41]

Others

In typical presentations, magnetic resonance imaging (MRI) and ultrasound (USS) are not routine tests for the diagnosis of GBS; however, they may be taken into consideration when the diagnosis is unclear. For instance, a spinal cord MRI may be able to localize the disease to the nerve roots or help differentiate between a peripheral neuropathy and a myelopathy.[56] As far as we are aware, there are no sonographic studies that include the cervical nerves in GBS.[57]

TREATMENT AND MANAGEMENT

Disease Modifying Therapies

Hospitalization for GBS is necessary for supportive care, close monitoring of respiratory function, and the detection of dysautonomy symptoms. Both plasma exchange and intravenous immunoglobulin G therapy have demonstrated efficacy.[58]

1) **Intravenous Immunoglobulin (IVIG**)

IVIg may be a more practical and secure treatment.[59] Toxic antibodies, complement system proteins, and cytokines are thought to be eliminated by IVIG, which also inhibits antiganglioside binding, complement activation, and Fc-mediated immune cell activation. [60]The administration of immunoglobulins intravenously has a more intricate mechanism of action.[61] Blockage of Fc receptors, increased autoimmune immunoglobulin catabolism, and potential functions in supplying anti-idiotypic antibodies and promoting remyelination are likely among these.[62]

2) **Plasma Exchange**

Patients who received PE up to 30 days after the onset of the disease still experienced benefits, but PE is more effective when initiated within 7 days of the onset of the disease than when it is initiated later.[63] It has been shown to help patients recover more quickly and commence walking sooner.[64]

3) **Nutrition**

It is best to introduce gastric or nasogastric tube feeding gradually and early and to help with respiratory weaning and prevent muscle wasting, a high-protein diet (2–2.5 g/kg) and high energy (40–45 nonprotein kcal) have been suggested.[63]

4) **Pain Management**

Despite being common, Guillain-Barré syndrome (GBS) pain is frequently underdiagnosed and poorly treated. Numerous pharmacological treatment options have been explored in clinical trials for individuals with pain associated with GBS in recent years.[65]

5) **Supportive Care**

Vital sign monitoring, intravenous fluids, and oxygen therapy are examples of supportive care. Stabilizing the infant's condition and managing any potential complications require supportive care.[66]

6) **Rehabilitation**

On the basis of a precise function assessment, a rehabilitation plan is created. Patients must enlist the assistance of various members of the rehabilitation team based on the goals and objectives of the rehabilitation plan, which is created after the evaluation, data is compiled, and a list of issues is created. All pertinent disciplines were included in the treatment program, which consisted of up to three one-hour therapy sessions per week, contingent on team consensus and participant need.[67] Clinical psychology was used for counselling and support when needed, occupational therapy to improve daily function (domestic, community tasks), driving, and returning to work, and physiotherapy for strengthening, endurance, and gait training.[68]

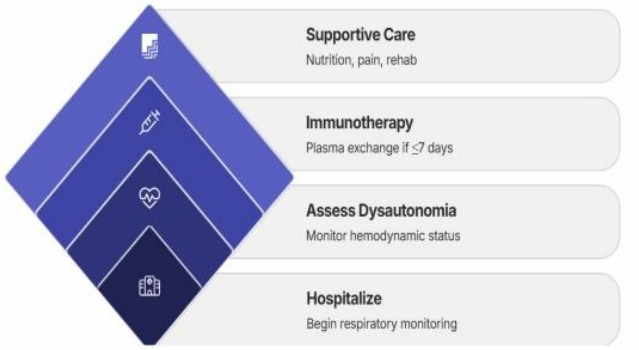

Figure 4: Treatment decision algorithms

CASE REPORT: 1

A case report of GBS in an eleven-month infant

A previously healthy 11- month old boy referred to a pediatric hospital because of weakness and difficulty feeding for 10 days. He developed fever and diarrhea 2 weeks before the onset of weakness. He was treated with trimethoprim and sulfamethoxazole for 7 days. After Two days the diarrhea started, presented weakness in the lower extremities and at 24h in both arms, and later inability to sit, feed and support the head. After examination, he was in the 50th percentile for weight, length and head circumference. The vital signs were in normal ranges according to his age. On detailed examination, he had flaccid paralysis with absence of inferior and superior deep tendon reflexes. There was no spontaneous movement in lower extremities, whereas there was only proximal muscle contraction in the upper extremities. Laboratory studies that included complete blood count, biochemical parameters and coagulation profile were normal. Examination of cerebrospinal fluid revealed an increase in the protein count (119mg/dL) and the cell count was in the normal range. Stool culture was negative. Electroneuromyography revealed normal motor and sensory nerve conduction velocity, greatly reduced amplitude of action potentials and an absence of F responses consistent with acute motor axonal neuropathy (AMAN) a variant of GBS.

Treatment

Patients received IVIG at 2g/kg single dose. Twenty- four hours later, evidence of proximal muscle contraction in upper and lower extremities, 72 h later horizontal movement of the lower extremities when gravity is eliminated. Two weeks later proximal movements against gravity are overcome in upper and lower extremities and he can hold his head . One month later he can grasp a toy but still has inability to stand alone.

CONCLUSION

GBS is rare disease and individual studies often lack a sufficient number of cases to make age specific incidence estimates. It has been seen that the pediatric patient has a better prognosis compared to the adult. Approximately 80% of patients who have suffered GBS are symptomatic within a period of 6 months and 20% persist with a moderate neurological sequela. It has been proven that the initiation of treatment in the early stages of the disease produces a faster recovery. [69]

CASE REPORT: 2

Simultaneous occurrence of Guillain-Barre Syndrome in three members of the same family: A case report

Case one

A 43-year-old women presented with a two week history of weakness involving both upper and lower limbs, preceded by diarrhea. Her pulse rate was 80 beats per minute, blood pressure was 120/70mmHg, respiratory rate was 16 breaths per minute. Neurological examination revealed a Medical Research Council (MRC) power grading of 2/5 in proximal while 1/5 in distal muscles of upper and lower limbs. On investigation, a complete blood picture showed haemoglobin of 10.5g/dL, mean corpuscular volume (MCV) of 76 fL, white blood cell (WBC) count of 10,300/cm² and platelets of 2,56,000/cm³. Stool culture were negative and electrocardiogram (ECG), thyroid function tests and metabolic profile revealed no abnormalities. Cerebrospinal fluid (CSF) examination showed normal cells and proteins. She was diagnosed as a case of GBS based on the nerve conduction study (NCS) which suggested acute motor axonal neuropathy (AMAN) variant of GBS. She received four sessions of plasmapheresis every 48 hours and responded gradually to treatment with a power of 4/5 in both upper and lower limbs at the time of discharge from the hospital.

Case two

A 40-year-old women, younger sister to the first patient, presented with weakness involving both upper and lower limbs for two weeks, She had no history of diarrhea and upper respiratory or viral infection. She started developing weakness in both lower limbs initially with a tingling sensation and eventually was unable to walk or stand unsupported with frequent falls. On physical examination, she had blood pressure of 140/90mmHg, pulse rate of 90 beats per minute, respiratory rate of 15 breaths per minute with a normal breathing pattern and a temperature of 98.6 °F. On neurological examination, MRC power grading of 3/5 in proximal while 1/5 in distal muscles of both upper and lower limbs was noted. On investigation, a complete blood picture showed haemoglobin of 12.3 g/Dl, MCV of 78 fL, WBC count of 9,260/cm² and platelets count of 2,15,000/cm³. She had no abnormalities on ECG, thyroid function tests were normal and stool culture were negative. CSF examination was unremarkable. She was also diagnosed as a case of GBS based on NCS which suggested the GBS variant AMAN.

Case 3

A 50-year-old man, husband to the younger sister and first cousin to both sisters, presented with a two-week history of weakness involving lower limbs. He also had a history of diarrhea preceding the weakness one month back. He had no weakness in upper and lower limbs. On physical examination, he had a blood pressure of 120/90 mmHg, pulse rate of 88 beats per minute, respiratory rate of 17 breaths per minute with a normal breathing pattern and a temperature of 98.6°F. MRC power grading of 1/5 was observed in all muscles groups of both lower limbs. On investigation, a complete blood picture showed haemoglobin og 13,5 g/dL, MCV of 78.2 fL , WBC count of 6,320/cm² and platelet count of 2,11,000/cm³. Thyroid function tests showed no abnormalities. ECG was normal and stool culture is negative. He was also diagnosed as a case of GBS based on NCS which suggested the GBS variant AMAN. He received four session of plamapheresis every 48 hours .

CONCLUSION

GBS is usually considered a sporadic autoimmune disease affecting nerves, usually preceded by an infection. However, a few familial cases have been reported suggesting genetic susceptibility. The presence of antecedent triggers in familial cases suggest a complex etiology of GBS. Further molecular and genetic studies need to be conducted in large population groups to determine the unexpected occurrence of familial cases in an otherwise known sporadic illness. [70]

RECENT ADVANCES AND FUTURE DIRECTION

Although there is no specific treatment for GBS, the standard of care for the condition is thought to be administering intravenous immunoglobulin or therapeutic plasma exchange at an early stage, which can hasten motor recovery and shorten the time spent on ventilator support. [71]. The zipper method is a new treatment approach that appears to improve outcomes for patients with severe Guillain-Barré syndrome who need intensive care by lowering mortality, accelerating weaning off of mechanical ventilation, and reducing hospital stays. For a variety of situations, this method is a promising immunomodulation approach.[72]

Novel Immunotherapies

It has been demonstrated in animal experiments that blocking the neonatal Fc receptor (FcRn) can lower the amount of IgG in the body and regulate the concentration of endogenous IgG. Mice deficient in FcRn exhibit markedly lower levels of anti-ganglioside antibodies. In mice, the use of FcRn inhibitors significantly lowers antibody levels, reducing nerve damage and clinical symptoms. This suggests that FcRn inhibitors may one day be used to treat GBS.[73] The development of targeted therapies and the identification of early biomarkers of disease subtype and progression depend on future immunological studies thoroughly characterizing autoreactive T and B cell immunity in well-defined patient cohorts stratified by GBS subtypes and disease stages. This will help to uncover their relative contributions to disease onset, progression, and clinical heterogeneity. Further study in this field is expected to enhance patient outcomes and deepen our understanding of GBS immunopathology[74]

Complement Inhibitors

Although complement activation has also been observed in CIDP patients, it is unclear whether this activation is a pathophysiological mechanism in CIDP that manifests independently of autoantibodies. Regardless, complement inhibition, a treatment approach that has proven effective in other autoantibody-mediated disorders, is also being researched as a possible future therapeutic approach in CIDP.[75]

Biomarkers

Reliable biomarkers for diagnosis, prognosis evaluation, and treatment response monitoring are scarce in immune-mediated neuropathies; however, IL8 in CSF has been confirmed as a diagnostic biomarker for GBS and CIDP and has the potential to be a prognostic biomarker for both short- and long-term outcomes in GBS.[76] Future studies should concentrate on determining more trustworthy biomarkers for GBS early detection and investigating the environmental and genetic variables that predispose people to the syndrome.[77] Finding biomarkers has been greatly impacted by the advent of single molecule array technology. Three biomarkers—neurodepartment light chain (NfL), peripherin, and total tau (T-tau)—have been demonstrated to have potential utility in GBS, and there is growing interest in serum and CSF biomarkers of peripheral nerve disease.[56]

Figure 5: The chart based on the current and failure direction

CONCLUSION

Guillain-Barre Syndrome (GBS) remains a critical neurological condition characterized by rapid onset, immune-mediated peripheral neuropathy, often triggered by preceding infections. Despite its relatively low incidence, the potential for life-threatening complications such as respiratory failure and long term disability. Advancements in clinical understanding, diagnostic modalities, immunotherapeutic strategies and plasma exchange have significantly improved patient outcomes. Ongoing research into the underlying immunopathology of GBS, including the role of molecular mimicry, anti-ganglioside antibodies and complement activation is paving the way for targeted therapies. Promising development such as FcRn inhibitors, complement blockers and stem cell based regenerative therapies represent future directions with the potential to revolutionize GBS management. Further efforts should focus on identifying reliable biomarkers for early diagnosis, subtype differentiation and prognosis assessment. Additionally interdisciplinary rehabilitation and long term supportive care remains essential for optimizing recovery and quality of life. Ultimately, a combination of early recognition, immunotherapy, supportive care and future precision medicine holds the key to improving survival and functional outcomes in GBS patients.

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