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Gullain-Baree syndrome: A comprehensive review about detailed basic information of the syndrome.

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ABSTRACT: Guillain-Barré Syndrome is the most common cause of acute neuromuscular paralysis worldwide, often triggered by infections such as Campylobacter jejuni, Epstein-Barr virus, or influenza. It is an autoimmune disorder where the body's immune system attacks the peripheral nerves, leading to sudden and progressive muscle weakness, sometimes resulting in respiratory failure. The syndrome includes several variants: Acute Inflammatory Demyelinating Polyneuropathy (AIDP), Acute Motor Axonal Neuropathy (AMAN), Acute Motor-Sensory Axonal Neuropathy (AMSAN), and Miller-Fisher Syndrome (MFS). Diagnosis is primarily clinical, supported by cerebrospinal fluid analysis and electrophysiological tests. Epidemiological data show GBS incidence varies by region and age, with higher rates in older adults and a male predominance. The condition's pathophysiology involves molecular mimicry, where antibodies generated by infections cross-react with nerve tissues, leading to demyelination or axonal damage. The review also discusses immunological aspects, historical pathology studies, vaccine associations (such as with influenza and COVID-19 vaccines), and includes a detailed case study of a 5-year-old boy with severe GBS. The document emphasizes the need for continued research into improved treatments and highlights recent advances in understanding GBS mechanisms and management.

KEYWORDS: Guillain-Barré Syndrome, autoimmune disorder, Acute Inflammatory Demyelinating Polyneuropathy (AIDP), Acute Motor Axonal Neuropathy (AMAN), Acute Motor-Sensory Axonal Neuropathy (AMSAN), axonal damage

I. INTRODUCTION

The most frequent cause of acute neuromuscular paralysis in the globe is Guillain-Barré syndrome (GBS). For a better prognosis, early treatment is crucial because it can be a serious and perhaps fatal condition. GBS is today one of the best characterized neuro inflammatory illnesses, offering important insights into the mechanisms of peripheral nerve inflammation more than a century after it was originally identified [1]. But there are still a lot of things about GBS that we don't fully understand, and its entire extent is still a mystery. GBS is known to be a monophasic, post infectious, immune-mediated polyradiculoneuropathy, and it is diagnosed mostly by clinical symptoms, with or without the assistance of electrophysiological and laboratory results. Due to respiratory failure, GBS patients eventually need mechanical breathing after developing a severe, widespread neuropathy. One patient out of every twenty passes away. Therapeutic advancements have not kept pace with growing knowledge of GBS, and intravenous immunoglobulin (IVIg) or plasma exchange (PLEX) continue to be the cornerstones of treatment. While the possible function of fluid biomarkers of neuropathy is still being investigated, outcome measures and interval neurological examinations are utilized to track disease activity and response to treatment. As you can see, new treatments are being investigated and could soon be used in clinical care. This review summarizes current knowledge and the best available evidence to help clinicians and scientists navigate the journey through Guillain-Barré syndrome in light of the new guidelines on diagnosis and treatment of GBS from the Peripheral Nerve Society (PNS) and the European Academy of Neurology (EAN) [2].

Guillain-Barré Syndrome (GBS) is a rare yet serious autoimmune condition that targets the peripheral nervous system, resulting in sudden neuromuscular paralysis. It is the leading cause of acute flaccid paralysis globally and can progress swiftly, necessitating immediate medical care. The disorder is often preceded by infections—such as Campylobacter jejuni, Epstein-Barr virus, cytomegalovirus, or influenza—that provoke an abnormal immune response. GBS encompasses a range of clinical variants, including Acute Inflammatory Demyelinating Polyneuropathy

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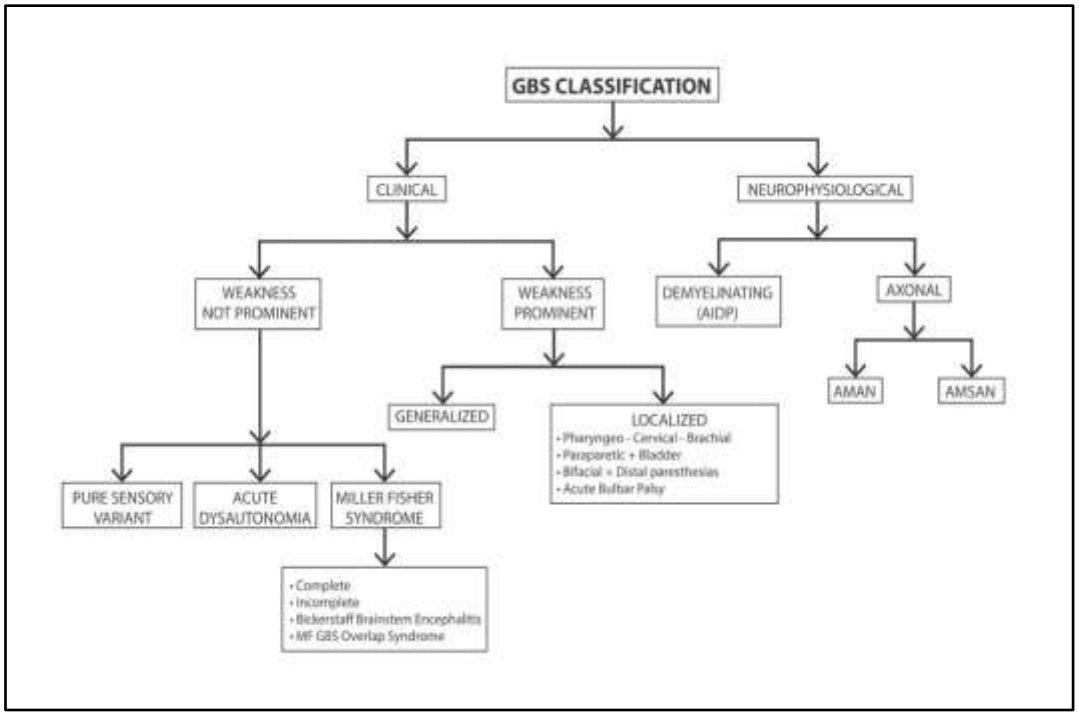
(AIDP), Acute Motor Axonal Neuropathy (AMAN), Acute Motor-Sensory Axonal Neuropathy (AMSAN), and Miller-Fisher Syndrome (MFS), each differing in severity and pathological features but united by a shared autoimmune mechanism. The annual incidence of GBS is estimated at 0.81 to 1.89 cases per 100,000 individuals, with a slightly higher occurrence in men and older populations[3]

Variants of GBS:

As mentioned, GBS is an umbrella term that describes a number of clinically and electrophysiological heterogeneous disorders that share the common feature of acute onset symmetric paralysis with or without sensory loss. GBS can be classified on the basis clinical features depending on topographic involvement or on the basis of electrophysiological features. Clinically, GBS has one important variant – the Miller Fisher syndrome (MFS) and both GBS and MFS have various forms frustes leading to a very heterogeneous clinical picture [4]. Electrophysiological GBS can have a demyelinating pattern – acute inflammatory, demyelinating polyradiculoneuropathy (AIDP) or an axonal pattern – acute motor axonal neuropathy (AMAN) and acute motor sensory axonalneuropathy(AMSAN).[3]



Although its exact origin is still uncertain, molecular mimicry—where microbial antigens resemble components of nerve cells—plays a central role in prompting the immune system to attack peripheral nerves. Prompt diagnosis and treatment are essential, with standard therapies including intravenous immunoglobulin (IVIG) and plasmaphoresis. While most patients achieve significant recovery, some may suffer lasting neurological impairments. This review delves into the pathophysiology, clinical features, diagnostic methods, treatment strategies, and current research on GBS, emphasizing recent progress and future prospects in its management.

II. EPIDEMIOLOGY

There are 100,000 new cases of GBS every year [5]. Estimates of yearly incidence (per 100,000 people) are lowest in Japan (0.44) [6], China (0.67) [7], Tanzania (0.83) [8],and Finland (0.84) [9], and high est in Chile (2.12) [10] and Bangladesh (3.25) [11], likely due to differ ences in exposure to infectious organisms. Seasonal variations are described [12], and spikes of GBS have been reported following in fectious outbreaks, most notably in relation to Campylobacter jejuni[13] and Zika virus (ZIKV) [14, 15]. Older people are more commonly affected (peak incidence of GBS is between 50 and 70 years of age) and the male: female ratio is 1.5:1.

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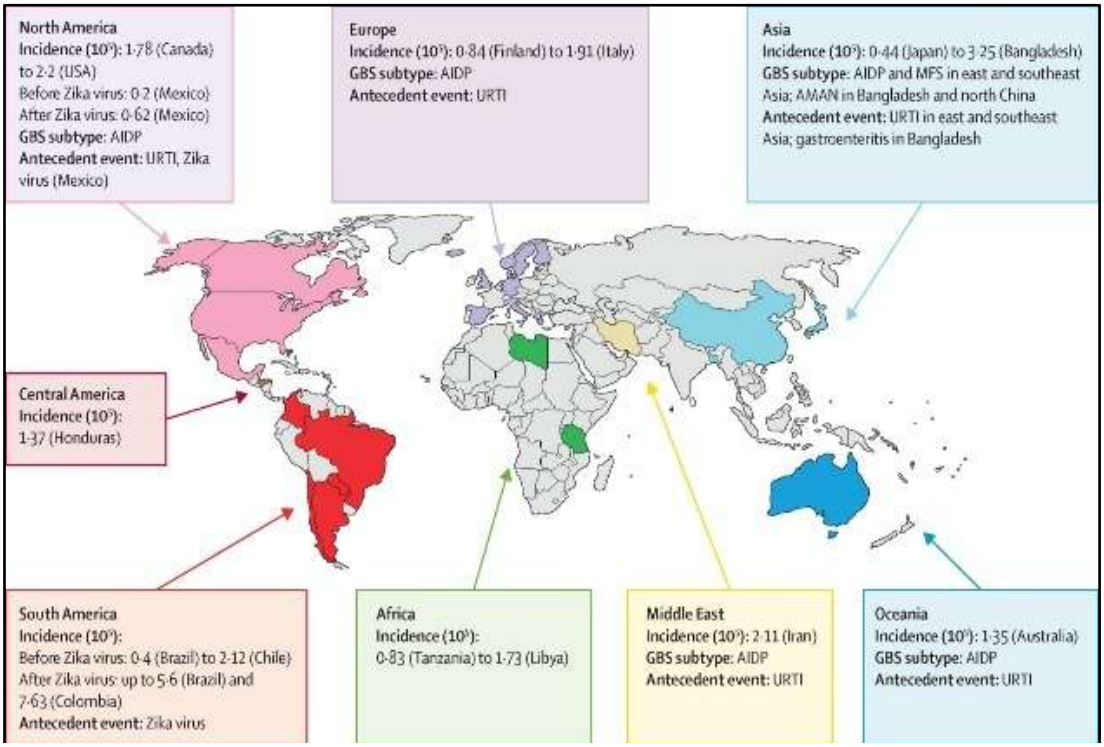

Guillain-Barré Syndrome (GBS) is a globally recognized neurological condition, with an annual incidence ranging from 0.81 to 1.89 cases per 100,000 individuals. While it can affect people of all ages, it is more commonly seen in men and older adults.[16][17].

Geographical Differences:

North America & Europe: Acute Inflammatory Demyelinating Polyneuropathy (AIDP) is the most frequently observed subtype

Asia & Latin America: There is a higher occurrence of Acute Motor Axonal Neuropathy (AMAN), often associated with Campylobacter jejuni infections.

Risk Factors and Triggers:

Infections:Around 60–70% of cases are preceded by viral or bacterial infections, particularly Campylobacter jejuni, Epstein-Barr virus, cytomegalovirus, and influenza.

Vaccination and Surgery:

In rare instances, GBS has been linked to certain vaccines (like flu and COVID-19) and surgical procedures.

Seasonal Trends: Some research indicates a rise in cases during winter and spring, coinciding with spikes in respiratory and gastrointestinal infection.

Mortality:

The death rate is between 3–7%, typically due to respiratory complications or autonomic dysfunction.

Recovery:

Most patients recover within 6 to 12 months, though approximately 20% may be left with lasting disabilities. [18]

The majority of GBS patients recover within 6 to 12 months; however, about 20% may experience long-term disabilities. Globally, around 100,000 new cases of Guillain-Barré Syndrome occur each year. The annual incidence rates per 100,000 population vary significantly across regions, with the lowest rates reported in Japan (0.44), China (0.67), Tanzania (0.83), and Finland (0.84), and the highest in Chile (2.12) and Bangladesh (3.25). These disparities are likely influenced by differences in exposure to infectious agents. Seasonal trends have been noted, with increases in cases often following infectious disease outbreaks, particularly those involving Campylobacter jejuni and the Zika virus. GBS is more common in older adults, with the highest incidence occurring between the ages of 50 and 70, and it affects males more frequently than females, with a male-to-female ratio of 1.5:1.[19]

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III. PATHOPHYSIOLOGY OF GBS:

The pathophysiology of GBS can be delineated into two pivotal stages: initiation by an immunological trigger and immune-mediated disruption of axons and/or myelin. Based on electrophysiology, GBS has been traditionally divided into two forms: acute inflammatory demyelinating polyradiculoneuropathy (AIDP) and acute motor axonal neuropathy (AMAN). The notion that this neurophysiological dichotomy reflects a true underlying pathological difference between primarily demyelinating versus axonal GBS is currently being challenged, and the new EAN/PNS guidelines no longer support the distinction between AIDP and AMAN.

Guillain-Barré Syndrome (GBS) is an autoimmune-mediated peripheral neuropathy where the body's immune system mistakenly attacks the myelin sheath or axons of peripheral nerves. This results in demyelination, inflammation, and nerve conduction abnormalities, leading to progressive muscle weakness and paralysis. Key Mechanisms of GBS Pathogenesis. [20] Triggering Event (Infection or Immune Stimulus) GBS is often preceded by infections (e.g., Campylobacter jejuni, Epstein-Barr virus, cytomegalovirus). The immune system produces antibodies that mistakenly recognize nerve components as foreign due to molecular mimicry.[21] Immune System Activation Antibodies target gangliosides (GM1, GD1a, GQ1b) on nerve membranes. Activation of T-cells and B-cells leads to inflammatory damage to nerves. Complement system activation worsens the damage, forming membrane attack complexes (MACs).[22] Types of Nerve Damage Demyelination (AIDP - Acute Inflammatory Demyelinating Polyneuropathy): Macrophages strip the myelin sheath, slowing nerve conduction. Axonal Degeneration (AMAN/AMSAN - Acute Motor/Sensory Axonal Neuropathy): Antibodies directly attack axons, leading to severe and prolonged paralysis.[23] Neurological Dysfunction Motor impairment: Weakness, paralysis. Sensory dysfunction: Numbness, tingling. Autonomic dysfunction: Irregular heartbeat, blood pressure instability.

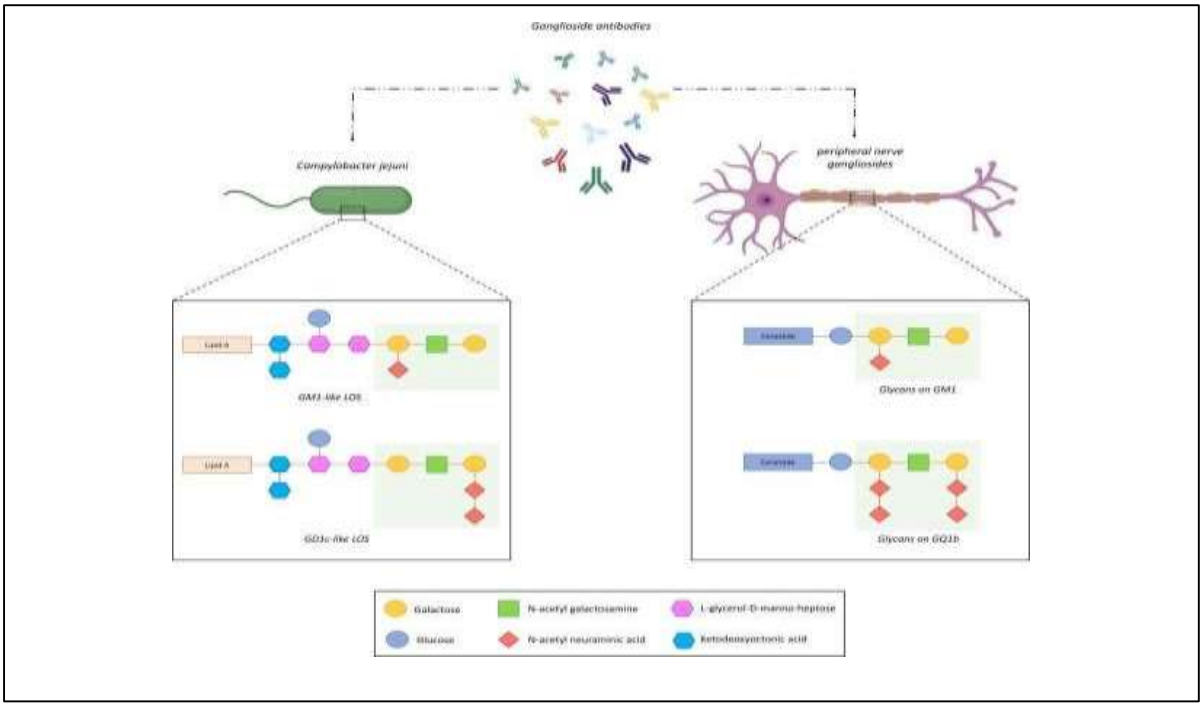

FIG. no.1: Lipo-oligosaccharide (LOS) on the outer membrane of Campylobacter jejune induces cross-reactive antibodies which, through molecular mimicry, bind to the structurally identical glycans (areas in green) present on peripheral nerve gangliosides (GM1 and GQ1b in the example above), resulting in damage to axons and Schwann cells. [Correction addedon 03 June 2024, after first online publication: Figure 1 has been corrected

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IV. CLINICAL FEATURES:

Criteria for the diagnosis of Guillain Barré syndrome were initially devised to investigate the possible association of this disease with swine flu vaccination in the 1970s.[24] These criteria have been redefined in the light of advances in the electrophysiology of Guillain Barré syndrome.[25] Required criteria for the diagnosis include progressive weakness of more than two limbs, areflexia, and progression for no more than four weeks. Other causes of an acute neuropathy such as lead poisoning, vasculitis, botulism, and porphyria require exclusion. Supportive criteria include relatively mild sensory signs, raised protein in the cerebrospinal fluid (CSF), with a relatively normal cell count, and neurophysiological evidence of conduction block. Weakness is frequently proximal and distal, unlike dying back axonopathies, and respiratory involvement occurs in about a quarter of cases. The CSF protein may be normal in the first week of the illness.[26] but may then rise to several g/dl. The CSF cell count usually remains below 500 cells/litre.

Guillain-Barré Syndrome (GBS) is characterized by acute, progressive, and symmetrical muscle weakness, often following an infection. Symptoms typically worsen over days to weeks, peaking within 4 weeks. The disease spectrum includes motor, sensory, and autonomic dysfunctions, with variations based on the subtype of GBS.

1. Motor Symptoms Progressive Weakness Starts in the lower limbs (ascending paralysis) and spreads to the upper limbs, trunk, and face. Can progress to quadriplegia in severe cases. Loss of deep tendon reflexes (areflexia) is a hallmark sign. In some cases, weakness descends (as seen in Miller-Fisher Syndrome). Facial and Bulbar Weakness Cranial nerve involvement occurs in 50% of cases, leading to: Facial weakness (bilateral in severe cases). Difficulty swallowing (dysphagia). Impaired speech (dysarthria). Respiratory Muscle Paralysis Occurs in 15-30% of cases, leading to respiratory failure. Patients may require mechanical ventilation if respiratory muscles weaken.[27]
2. Sensory Symptoms Paraesthesia (Tingling & Numbness) Begins in the feet and hands (glove-and-stocking pattern). May progress to involve the trunk and face. Pain Neuropathic pain is present in 50-80% of cases, described as burning, aching, or electric shock-like. Worse at night and exacerbated by movement.[28]
3. Autonomic Dysfunction Occurs in 70% of cases and can lead to life-threatening complications. Cardiovascular Symptoms Fluctuating blood pressure (hypertension or hypotension). Cardiac arrhythmias (bradycardia, tachycardia, or even sudden cardiac arrest). Gastrointestinal and Urinary Symptoms Bladder dysfunction (urinary retention or incontinence). Bowel dysfunction (constipation or ileus). Sweating Abnormalities Hyperhidrosis (excess sweating) or anhidrosis (lack of sweating) due to autonomic nerve involvement.[29]
4. Variants of GBS and Their Clinical Features Miller-Fisher Syndrome (MFS)Triad: Ophthalmoplegia (eye muscle paralysis), ataxia (loss of coordination), and areflexia. Acute Motor Axonal Neuropathy (AMAN)Purely motor variant with severe paralysis but no sensory involvement. Acute Motor-Sensory Axonal Neuropathy (AMSAN)More severe than AMAN, with both motor and sensory loss.[30]

V. HISTORY AND PHYSICAL:

Guillain-Barré syndrome (GBS) patients describe a fulminant course of symptoms that usually include ascending weakness and non-length dependent sensory symptoms. By definition, the nadir is usually reached within 4 weeks.

Symmetric involvement is a key feature of GBS. GBS is usually considered monophasic; therefore, a relapsing or remitting course at presentation would be considered a typical. Additionally, a prior GBS event (recurrent GBS) is also unusual, occurring in less than 10% of all patients. If the patient reports progression beyond 8 weeks, other diagnoses should be considered. GBS often presents (up to 70% of patients) within 1 to 6 weeks of antecedent illness. Other antecedent events that have been linked with GBS include vaccinations (specifically a 1976 strain of swine flu vaccine), surgery, trauma, or other infections.[31]

Classically, patients with GBS will have a pattern of proximal and distal weakness, which is flaccid and often profound if hospitalized. Significant neck flexion weakness may be present and can portend the need for intubation. Areflexia or hyporeflexia is usually present. (Rare cases without hypo/areflexia have been described, mostly in the AMAN variant of GBS). Besides the flaccid weakness and areflexia, patients experience non-

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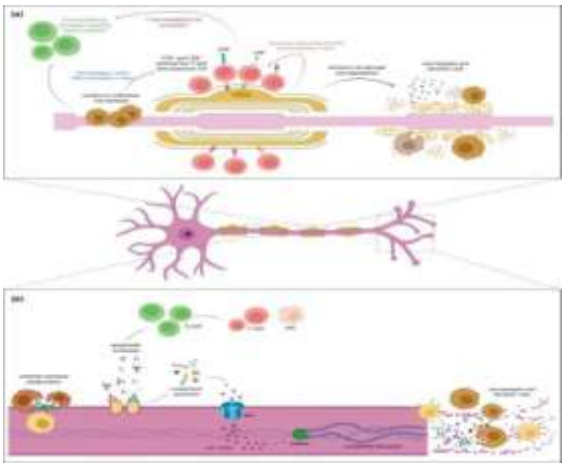
length-dependent sensory symptoms; therefore, unlike more common chronic neuropathies such as diabetic neuropathy, patients may report dysesthesias in the hands followed by the feet. Patients can develop facial diplegia due to the involvement of both facial cranial nerves. They can also develop dysphagia due to the involvement of the glossopharyngeal, vagus, and hypoglossal cranial nerves. Autonomic nerves can lead to significant morbidity; therefore, most physicians recommend monitoring in an intermediate or intensive care unit for cardiac arrhythmias or blood pressure lability.[31]

VI. PATHOLOGY:

The studies of Asbury and colleagues suggested that the earliest hall mark of Guillain Barré syndrome was the presence of perifas- cicular lymphocytic cuffs of small vessels in the endoneurium and perineurium. [32]Thisappears to be associated with demyelination, which is typically macrophage associated. [33] In this regard, the pathology has many similarities with the animal model, experimental allergic neuritis (EAN). [34]More recent pathological studies have shown that several pathological subtypes of Guillain Barré syndrome exist, although the demyelinating form of the disease is the most common, and probably represents at least 75% of cases. [35] Some cases of Guillain Barré syndrome are associated with a primarily axonal process, in which macrophages may be found in close proximity to the axon,with sparing of myelin.[36] This histological finding has been interpreted as indicating an immuno- logical attack on antigens of axonal origin, rather than a myelin antigen in demyelinating forms of the disease.



Still other cases of the disease appear to involve both sensory and motor axons and such cases are termed acute motor and sensory axonal neuropathy (AMSAN). This variant of the disease appears to be the most uncommon and perhaps accounts for only 5% of the clinical syndrome.

VII. IMMUNOLOGY

The earliest immunological studies of Guillain Barré syndrome were limited to crude complement fixation tests to nerve antigens. Such studies suggested minor abnormalities in only a small proportion of cases. [37] Nevertheless, the dramatic response of demyelinating cases of Guillain Barré syndrome to treatment with plasma exchange strengthened the view that a plasma derived factor must have a role in the aetiology of the syndrome. In the mid-1980s Koski et al described a C1 esterase technique that appeared to detect subtle complement fixation in most patients with Guillain Barré syndrome [38] and, furthermore, the concentrations fell during the convalescent stage of the disease. Unfortunately, this test proved di9cult to reproduce and few other laboratories could demonstrate such striking abnormalities. The discovery of antiganglioside antibodies in the serum of patients with Guillain Barré syndrome has sparked of an enormous proliferation of publications. The frequency of such antibodies varies from as low as 29% [39] up to nearly 70% [40], although the average figure is probably around 30%. Patients with Miller Fisher syndrome have detectable anti-GQ1b antibodies at a much higher frequency, prob- ably around 95% [41,42]. Gangliosides are widely distributed in the nervous system and may have a variety of functional roles.

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The structure of gangliosides involves several repeating subunits, which can be antigenic. Thus, antiganglioside antibodies have different specificities and these may overlap. Antibodies that recognise the NeuAC epitope will crossreact with several different gangliosides, making the importance of antiganglioside anti- bodies more difficult to interpret. The pattern of reactivity of a particular patient's serum with several different gangliosides helps to define the exact specificity of the antibody.

VIII. VACCINES ASSOCIATED WITH GBS:

Epidemiological evidence links some vaccines with a subsequent diagnosis of GBS. These include the vaccine forthe 'swine flu' (A/ New Jersey/76 influenza), the recombinant zoster vaccine (RZV), and the adenovirus-vector SARS-CoV-2 vaccines. The 'swine flu' vaccine campaign in the USA was associated with 4.9–5.9 GBS cases per mil- lion vaccinations [43]. Though initial concerns were raised about the H1N1 influenza A vaccination in 2009, subsequent surveillance reported only 1–1.6 cases per million doses.[44,45,46] Similarly, herpes zoster vaccination with RZV showed a marginally increased risk of GBS, with approximately 3 excess cases per million vaccination [47]. Adenovirus-vectored SARS-CoV-2 vaccines, such as ChAdOx1 and Janssen COVID-19 vaccines, have been linked to around 5.7 excess GBS cases per million first doses. [48,49] The 2023 EAN/PNS GBS guidelines conclude that the advantages of vaccination (reduction in morbidity and mortality related to infection and infection associated GBS) significantly outweigh any marginal elevation in the risk of post-vaccine GBS.[50] It remains uncertain whether repeat or future vaccination is safe following presumed vaccine-associated GBS, and certain individuals may be more susceptible to autoinflammation due to genetic predisposition. The risk of post-vaccine GBS should be acknowledged and balanced against the benefits of vaccination overall.

IX. CASE STUDY OF GBS

Patient information- A 5-year-old boy was taken to the Acharya Vinoba Bhave Rural Hospital with the chief complaints of Weakness in bilateral upper and lower limbs, trouble swallowing, inability to hold the neck, frothing from the mouth, fever spikes. Guillain-Barre Syndrome was discovered in him. He showed no improvement after treatment, and the patient status was unstable; he couldn't maintain saturation, and the patient was intubated on Ventilator support. The Patient's family is from a middle-class background. His family members were free of both communicable and non-communicable diseases. He and his family had good interpersonal relationships with relatives, neighbours, and other family members. The Ryle tube was inserted. He has a Fever (102°F) when he gets admitted. Blood test, cerebrospinal fluid examination, coagulation profile, liver function test, kidney function test, T3, T4, and TSH were done. Administration of immunoglobulin therapy, intravenous fluids, antipyretic, multivitamins, potassium, antibiotics, aminoglycosides, glucocorticoids, anti-allergic as per physician orders.

2. Physical Examination: On physical examination, the patient has experienced weakness in bilateral upper and lower limbs, Bulbar weakness is present, pain experienced in both legs, the gag reflex is absent, in the cardiovascular system, S1 and S2 sound is present, in the respiratory system, air entry is bilaterally equal (AEBE), pupils are reflected light, tone, and power of both upper and lower limbs are reduced, the plantar reflex is not examined, and then treatment was started as soon as possible.

3. Diagnostic Assessment: Blood test: Hb-11.3%, Total RBC count- 4.36millions/cu.mm, Total WBC Count- 5100/cu.mm, Total platelet Count 4.59lacs/cu.mm. In Cerebrospinal Fluid Examination, Glucose-CSF-72mg%, Protein CSF-105mg/dl, Lactic Dehydrogenasis-58I.U/L, PH-7.5. Prothrombin time -control -12.50secs, Prothrombin time-patients-20.60secs, INR-01.64, APTT-Control-30 secs, APTT- Patient- 36.30ecs in the Coagulation profile, Protein-9.9g/dl, Globulin-5.9gm/dl in liver function test. urea, creatinine, sodium, potassium, T3, T4 and TSH were all normal reading in the renal function test. [51]

X. TREATMENT

Treatment options for GBS are dependent on the severity of the disease. Currently, in mild cases there is typically no prescribed treatment. A mild case is defined as an individual who is still able to walk with or without assistance. In these cases, however, physical therapy may be beneficial. For more severe cases there are two treatment options, plasma exchange and intravenous immunoglobulin. Plasma exchange is a procedure in which the patient's blood is removed from their body and filtered before being returned to the body system. The goal of this treatment is to remove the

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harmful, soluble antibodies that are causing damage to the nerves from the blood. However, the timing of this treatment plays a key role in determining how effective it is. A study conducted in North America showed that plasma exchange had the greatest improvement when given within two weeks of the onset of symptoms. It was also found to be effective to a lesser degree at four weeks.Only individuals who could not walk without assistance were given the treatment in this study. Based on their findings it may be beneficial to repeat this experiment with individuals who have varying severity of the disease to determine if this timing applies to all cases of GBS.[52]

XI. CONCLUSION

Much has been learned about the mechanism of neuropathy in Guillain Barré syndrome but treatment remains disappointing after the major advances that occurred in the 1980s. Better and more specific treatments are clearly needed. It is hoped that recent advances in our understanding of pathogenesis may lead to better treatments in the next few years.

Since its initial description in 1916 by Georges Guillain, Jean Alexandre Barré, and André Strohl, there continues to be substantial developments in Guillain-Barré syndrome. IGOS has provided some clarity on geographical variations with more information likely to follow. The transient surge in patients with Guillain-Barré syndrome during the 2016 Zika virus outbreak and emerging reports of Guillain Barré syndrome in SARS-CoV-2 infection add to the growing list of antecedent infections. With the changing landscape in cancer therapy, reports of Guillain-Barré syndrome associated with immune checkpoint inhibitors have emerged. The medical community is continuously reminded of the need to remain vigilant of potential neurological complications with infective outbreaks and with the rise in novel immunotherapies. As new therapies enter clinical trials, and research is done into overcoming axonal degeneration and enhancing neural regeneration, the future outlook for Guillain-Barré syndrome is positive.

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