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A review study on Guillain barre syndrome

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Abstract

Acute immune-mediated peripheral nerve system dysfunction known as Guillain-Barre Syndrome (GBS) is typified by rapidly worsening muscle weakness, which frequently begins in the lower limbs and can eventually result in paralysis. It is typically brought on by an infectious event, most often a bacterial or viral infection, with Campylobacter jejuni being the most often linked harmful organism. According to epidemiology, there are 1-2 instances of GBS for every 100,000 people each year, with a small male predominance and greater frequency in adults over 50. Nerve conduction investigations and cerebrospinal fluid analysis demonstrating albumin cytological separation complement the largely clinical diagnosis.

Keywords: Autoimmune Neurological Disorder; Peripheral Nerve Damage; Demyelination; Immune-mediated Nerve Injury; Plasma Exchange (PE); Intravenous Immunoglobulin (IVIg).

1. Introduction

Guillian–Barre syndrome is an autoimmune disease, and it is mainly caused by bending bacteria and jejunum virus, the serum of patients containing an autoantibody such as the IgM and IgG. A large number of immune complexes are deposited in the peripheral nerve, damaged nerve root, and ganglia and peripheral nerve, resulting in extensive demyelination of peripheral nerve tissue. Patients may appear to have myoparalysis breathing; clinical early treatment is the key to delaying the progress of the disease. PE can quickly eliminate autoantibodies, similar antibodies; complement components and immune complexes, all kinds of cytokines, and pathological substances such as endogenous or exogenous toxins in patients with GBS. The postulated mechanisms of action of IVIg include interference with costimulatory molecules involved in antigen presentation and modulation of autoantibodies, cyotokines, and adhesion molecule production as well as macrophage Fc receptors. This process also disrupts complement activation and membrane attack complex formation. Modulation of the expression and function of Fc receptors on macrophages; suppression of cytokines, chemokines, and adhesion molecules; and alteration of the activation, differentiation, and effector functions of T cells [1].

The morbidity of GBS depends on the severity of the disease and on the treatment given. Respiratory complications, including ventilator-associated pneumonia, adult respiratory distress syndrome (ARDS), sepsis, pulmonary embolism and unexplained cardiac arrest due to autonomic dysfunction are the primary causes of death in patients with GBS. In a retrospective study, which included 114 patients, 59% had respiratory complications, primarily pneumonia. In a French multicenter study, it was demonstrated that among the patients who could not be mobilized, 20-28% developed pneumonia. In the group of patients requiring respiratory therapy, 60-74% developed pneumonia. 80% of patients with GBS recover completely or have minor deficits in the form of foot drop, minor balance problems or dysesthesias. In 5-10%, the course is protracted with months of respiratory therapy and severe motor and sensory sequelae. Approx. 3%

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become permanent wheelchair users. 3-8% die as a result of sepsis, ARDS, pulmonary embolism or unexplained cardiac arrest [2].

Prognosis: Short of death, the worst-case scenario in GBS is tetraplegia within 24 hours, with incomplete recovery after 18 months or longer. The best-case scenario is mild difficulty walking, with recovery within weeks. The usual scenario, however, is peak weakness in 10-14 days, with recovery in weeks to months. Average time on a ventilator (without treatment) is 50 days. There are likely many mild cases of GBS that are never definitively diagnosed, and patients make full recovery without treatment. The spectrum of milder disease has not been well studied nor clarified. Approximately 80% patients with GBS walk independently at 6 months, and about 60% of patients attain full recovery of motor strength by 1 year. Recovery in approximately 5-10% of patients with GBS is prolonged, with several months of ventilator dependency and a very delayed, incomplete recovery. Mortality: A 2008 epidemiologic study reported a 2-12% mortality rate despite ICU management, although the rate may be less than 5% in tertiary care centers with a team of medical professionals who are familiar with GBS management. Causes of GBS-related death include acute respiratory distress syndrome (ARDS), sepsis, pneumonia, venous thromboembolic disease, and cardiac arrest. Most cases of mortality are due to severe autonomic instability or from the complications of prolonged intubation and paralysis. The leading cause of death in elderly patients with GBS is arrhythmia. GBS-associated mortality rates increase markedly with age. In the United States, the case-fatality ratio ranges from 0.7% in persons younger than 15 years to 8.6% in individuals older than 65 years. Survey data has shown that in patients aged 60 years or older, the risk of death is 6-fold that of persons aged 40-59 years and is 157-fold that of patients younger than 15 years. Although the death rate increases with age in males and females, after age 40 years males have a death rate that is 1.3 times greater than that of females. GBS-related deaths usually occur in ventilator-dependent patients, resulting from such complications as pneumonia, sepsis, acute respiratory distress syndrome, and, less frequently, autonomic dysfunction. Underlying pulmonary disease and the need for mechanical ventilation increase the risk of death, especially in elderly patients. Morbidity: A significant percentage of survivors of GBS have persistent motor sequelae. Estimates indicate that 15-20% of patients have moderate residual deficits from GBS and that 1-10% are left severely disabled. Although the exact prevalence is uncertain, up to 25,000-50,000 persons in the United States may have long-term functional deficits from GBS. The speed of recovery varies. Recovery often takes place within a few weeks or months; however, if axonal degeneration has occurred, recovery can be expected to progress slowly over many months, because regeneration may require 6-18 months. Length of hospital stay increases with advancing age, because of disease severity and associated medical complications. Patients may experience persistent weakness, areflexia, imbalance, or sensory loss. Approximately 7-15% of patients have permanent neurologic sequelae (although figures of as high as 40% have been estimated), including bilateral footdrop, intrinsic hand muscle wasting, sensory ataxia, and dysesthesia. Patients may also exhibit long-term differences in pain intensity, fatigability, and functional impairment compared with healthy controls. In extremely rare cases, patients may experience recurrent GBS. Numerous papers have addressed the issue of persistent fatigue after recovery from GBS. Studies have suggested that a large percentage of patients continue to have fatigue-related problems, subsequently limiting their function at home and at work, as well as during leisure activities. Treatment suggestions range from gentle exercise to improvement in sleep patterns to relief of pain or depression, if present. GBS can produce long-lasting changes in the psychosocial status of patients and their families. Changes in work and leisure activities can be observed in just over one third of these patients, and psychosocial functional health status can be impaired even years after the GBS event. Interestingly, psychosocial performance does not seem to correlate with the severity of residual problems with physical function. Poor conditioning and easy fatigability may be contributory factors. Rudolph et al determined that patients who have had GBS seem overall to have a reduced quality of life and physical functioning. Their findings were based on a study of 42 GBS patients who were examined after a median of 6 years post–disease onset using a variety of measures, including the visual analogue scale (VAS) for pain, the disability rating index (DRI), and the Medical Outcome Study 36-item short-form health status scale (SF-36). Prognostic factors: Preceding gastrointestinal infection or diarrheal illness, older age (57 years or older), poor upper extremity muscle strength, acute hospital stay of longer than 11 days, ICU requirement, need for mechanical ventilation, medical Research Council (MRC) score below 40 and discharge to rehabilitation [3].

Since there is currently no conclusive laboratory test for GBS, the diagnosis is still primarily made clinically. The patient's history, physical examination, and supporting tests, such as cerebrospinal fluid analysis and nerve conduction testing, are all used by clinicians. The CSF fluid's albumin cytological dissociation (an increased protein level with a normal white cell count), areflexia, and ascending paralysis are classic symptoms that are extremely are highly suggestive of GBS. Managing symptoms and lessening the degree of immune-mediated nerve damage are the goals of GBS treatment. The two cornerstones of immunotherapy are intravenous immunoglobulin (IVIg) and plasmapheresis (plasma exchange), both of which have been shown to be effective in shortening recovery times and enhancing results when given early in the course of the illness. Supportive care, such as physical therapy and breathing support, is also essential, especially in extreme situations.

2. Results and discussions

Fei-Fei Tan. *e*t, al, (2024), Atypical Guillain-Barre syndrome with positive anti-sulfatide, anti-GT1b, and anti-GT1a antibodies, GBS is an acute immune-mediated disease of the peripheral nervous system. It is reported that the pathogenesis of GBS is related to many antibodies, such as ganglioside antibodies (GM, GD, GQ, and GT) containing sialic acid residues and sulfatide antibodies containing sulfuric acid residues. These antibodies are closely related to the clinical manifestations and subtypes of GBS and are also important biomarkers for its diagnosis. The clinical manifestations, the number of cells and protein in cerebrospinal fluid, and demyelinating changes in neurophysiological examination, were consistent with typical GBS. It is different from typical GBS. First, the tendon reflexes were normal; second, the patient had obvious muscle pain, which is easily misdiagnosed as polymyositis; third, ECG examination showed sinus bradycardia. The heart rate returned to normal after treatment; lastly, the intravenous immunoglobulin treatment was satisfactory and resulted in a fine prognosis. In the classification and diagnostic criteria of 2014 [4].

Juhao Zeng. *et*, al, (2024), *Strongyloides stercoralis*-induced sepsis and acute respiratory distress syndrome in a patient with Guillain-Barre syndrome, Strongyloidiasis stercoralis can cause disease when larvae invade the human body through the skin or mucosa and can also infect a host when the host ingests its eggs. Strongyloidiasis lacks characteristic manifestations, and its clinical symptoms are related to the immune response of the host and the degree of infection. Immuno deficient patients with underlying disease or who are receiving long-term corticosteroid treatment are more prone to developing severe disease. The *Strongyloides stercoralis*-induced sepsis and acute respiratory distress syndrome (ARDS) in a patient with Guillain-Barre syndrome [5].

Hua Liu. *et*, al, (2023), Variant of Guillain-Barré syndrome with anti-sulfatide antibody positivity and spinal cord involvement, Anti-sulfatide antibodies have been observed in 5.2% of all GBS cases, mainly in those without a history of prior or occult infection, suggesting that these antibodies may be related to a specific variant or variants of GBS. In this case report, we present a patient with GBS positive for anti-sulfatide antibodies. The CSF showed cytoalbuminologic dissociation. Electromyography suggested multiple peripheral nerve damage. Immunoblotting of the CSF showed positivity for anti-sulfatide antibody. The patient also had symptoms of diaphoresis, positive bilateral pathological signs, normal bilateral deep sensation, and joint position sense. A cervical MRI showed a lamellar and slightly long T2 signal in the medulla at the level of the C2–C3 vertebrae, which we interpreted as evidence of spinal cord involvement. GBS differs from multiple sclerosis (MS) and neuromyelitis optic spectrum disorder (NMOSD). MS lesions mainly involve the intracranial region and spinal cord and are characterized by temporal and spatial diversity; GBS patients have no significant abnormalities in cranial MRI. NMOSD is characterized by transverse myelitis and acute optic neuritis, spinal cord lesions that are often larger than three spinal cord segments, and specific antibodies against aquaporin [6].

Mei Jin, *et*, al (2023). Analysis of the characteristics of sympathetic skin response in children with Guillain-Barre syndrome, Guillain-Barre syndrome (GBS) is the most common cause of acute flaccid paralysis in children. The diagnosis and subtype classification of GBS in children is relatively difficult, especially for young children, who are more dependent on auxiliary examinations. Neuroelectrophysiological testing is of great significance in the early diagnosis, subtype classification and prognosis assessment of GBS in children. Studies have shown that 2/3 of GBS patients have autonomic dysfunction (AD), which is mainly manifested by increased heart rate, increased blood pressure and abnormal skin sweating. AD is an independent risk factor for poor prognosis in GBS patients, and may even lead to serious adverse consequences such as death. Pay attention to the monitoring of AD in GBS patients, early detection and early intervention. The sympathetic skin response (SSR) technology records the sweating response of the skin through surface and then detects the function of sympathetic nerve fibres. It is an electrophysiological index that objectively reflects the function of autonomic nerve function. The changing characteristics of SSR in children with GBS, compared the value of SSR and traditional nerve conduction measurement in the early diagnosis of GBS, analysed the relationship between SSR and AD and disease severity, and then evaluated its predictive value for the prognosis of GBS [7].

Qiao-Lin Zhou. *et*, al, (2022), Guillain-Barré syndrome and hemophagocytic syndrome heralding the diagnosis of diffuse large B cell lymphoma, DLBCL is one of the most common types of lymphomas. Occasionally, HPS can be an initial manifestation of tumor factors and EBV infection. GBS is rarely diagnosed prior to lymphoma. The incidence of GBS in NHL is low. Almost all studies of lymphoma-related GBS are case reports. NHL combined with GBS is more common than Hodgkin's lymphoma alone. In the present case, GBS and HPS were simultaneously confirmed, heralding the diagnosis of lymphoma. According to the previous case reports, the lymphoma types in these cases combined with GBS include DLBCL, Burkitt lymphoma, splenic marginal zone lymphoma, and peripheral T-cell lymphoma. Only approximately 10 cases of DLBCL have been reported to date. GBS is an immune-mediated acute inflammatory peripheral neuropathy that manifests as damage to the multiple nerve roots and peripheral nerves. The main pathological feature is extensive inflammatory demyelination of the peripheral nerves. It is a motor neuropathy that progresses rapidly. The two most common types of GBS are acute inflammatory demyelinating polyneuropathy and

acute motor axonal neuropathy. Other types of GBS, including acute motor-sensory axonal neuropathy (AMSAN), Millen-Fisher syndrome, acute pan-autonomic neuropathy, and acute sensory neuropathy, are relatively rare [8].

Sun Ruidi. *et*, al, (2022), Study on the very early and early neuroelectrophysiological characteristics of Guillain-Barre syndrome in children, Neuroelectrophysiological diagnostic criteria have good adaptability and accuracy in GBS classification and diagnosis, but the rate of motor nerve conduction abnormalities fluctuates between 39.2% and 88.2% from 2 days to 3 weeks after onset. Foreign reports show that the most common neuroelectrophysiological abnormality in adult GBS patients within 7 days of onset is abnormal H reflex. Some studies also believe that involvement of type Is a sensory afferent fiber in the H reflex can explain the disappearance of GBS reflexes and precedes demyelination and/or axonal damage of motor and sensory nerves. Other studies believe that involvement of type Is a sensory afferent fibre in the H reflex is the cause of ataxia and reflex abnormalities in Miller-Fisher syndrome (MFS). Foreign case studies that children with MFS all had abnormal H reflexes. However, there are no reports in China on the neuroelectrophysiological characteristics of GBS in children in the early stage (within 14 days of onset), especially in the very early stage (within 7 days of onset). Therefore, this study retrospectively collected neuroelectrophysiological data of GBS in children, compared them in groups according to different examination times, and explored the abnormal rates of H reflex, sensory nerve conduction, and motor nerve conduction in GBS children, so as to provide a basis for the clinical diagnosis of GBS in children [9].

Liang Jufang. *et*, al, (2021), Clinical features and Brighton stratified diagnosis of Guillain-Barre syndrome in children, Is an autoimmune polyradiculoneuropathy and a common cause of acute flaccid paralysis in children. GBS is a self-limiting disease with a good prognosis in most cases. A small number of children may develop respiratory muscle weakness. Early and accurate diagnosis can help children receive timely treatment and reduce complications. Although the diagnostic criteria proposed by Asbury and Cornblath have been widely accepted and applied worldwide, the Brighton Collaboration, an international organization that monitors vaccine safety, has proposed a hierarchical diagnostic criterion for GBS based on case definitions. The Brighton criteria classify the certainty of GBS diagnosis (level 1 is the highest and level 4 is the lowest), which is more operational in the diagnosis of adult GBS at home and abroad. The clinical phenotype and neuroelectrophysiological characteristics of paediatric GBS are different from those of adult GBS. Children with GBS have a faster disease progression and more demyelinating GBS subtypes. Brighton stratified diagnosis has a high sensitivity in children with GBS in foreign countries, but there is currently no research on Brighton stratified diagnosis in children with GBS in China. Therefore, this study retrospectively analysed the clinical characteristics of children with GBS in Hubei Province and explored the significance of Brighton stratified diagnosis in children with GBS [10].

Li Cheng. *e*t, al, (2021), Analysis of risk factors for assisted ventilation in children with Guillain-Barre syndrome, Foreign literature shows that facial muscle and/or bulbar muscle weakness is associated with GBS assisted breathing, and impaired oropharyngeal reflexes affecting respiratory function are possible reasons prospective study of GBS in children, predictive models for assisted breathing, and meta-analyses all show that cranial nerve involvement or bulbar muscle weakness are risk factors for assisted breathing. The correlation between GBS electrophysiological subtype and assisted breathing type is the main subtype of GBS in Europe and North America, but the axonal type is the main subtype of GBS in Asia, and there are differences in the subtype composition in different regions. Reversible conduction block affects subtype classification, which may be a reason for the controversy over the relationship between different subtypes and assisted breathing. The data from this study showed that axonal subtype is not a risk factor for GBS requiring assisted breathing. The analysis may be related to the different axonal subtype compositions in the two studies. Whether electrophysiological subtype is a risk factor for GBS assisted breathing still needs to be verified by large-sample, multicentre studies [11].

A Sun Ruidi. *et,* al (2020), Relationship between motor nerve block and different subtypes of Guillain-Barre syndrome in children, IDP and AMAN are two different subtypes of GBS. In AIDP, different degrees of motor nerve demyelination can lead to slowed nerve conduction velocity and/or the occurrence of motor CB. Motor CB indicates that segmental demyelinated nerve impulses cannot be effectively transmitted, and limb weakness worsens. This also explains why the severity of the disease in AIDP children with motor CB is more severe than that in AIDP children without motor CB at the peak of the disease. Of course, motor CB can exist in nerve roots, plexuses or hidden parts. The clinical symptoms caused by motor CB in different parts are different. Therefore, not all motor CBs in all parts can represent the severity of AIDP. In AMAN, IgG antibodies bind to GM1 or GD1a on the axon membrane of motor fibers on the nodes of Ranvier, activate complement in situ, lead to the disappearance of node voltage-gated sodium channel clusters and damage to the axon-glial junction on the node side, and are accompanied by Wallerian degeneration in the later stage, manifested as a decrease in motor conduction CMAP. This is the development process of classic AMAN. The different neurophysiological characteristics of AIDP and AMAN can serve as an important auxiliary examination for determining

GBS subtypes. Although in most cases, the presence of motor CBs indicates motor demyelination, reversible motor CBs, as a special type of motor CB, are related to axonal subtypes [12].

Christine Verboon. *e*t, al, (2019), Current treatment practice of Guillain-Barr´e syndrome, Plasma exchange (PE) and IV immunoglobulin (IVIg) are the only proven effective treatments for Guillain-Barr´e syndrome (GBS), although there has been little formal exploration of optimal dosage and treatment duration for either. The implementation of these treatments in clinical practice is complicated by the variability in disease presentation and severity. Most therapeutic trials with PE or IVIg focused on adult patients who were unable to walk independently. At present, it is unclear whether these treatments are also effective in children, patients with mild GBS, or clinical variants including Miller Fisher syndrome (MFS). The treatment practice currently provided for GBS varies between patients, especially with respect to initial treatment of mild and variant forms, and retreatment of TRF and nonresponding patients. Such treatment could be beneficial in terms of clinical outcome and cost-effectiveness, but selective treatment trials are lacking and complicated because of the rarity and diversity of GBS. Whether such evidence can be generated by comparative treatment studies based on observational data needs to be determined. Further studies are required to develop evidence-based guidelines on the treatment of GBS [13].

Sonja E. Leonhard. *e*t, al (2019), Diagnosis and management of Guillain–Barre syndrome in ten steps, Guillain–Barré syndrome (GBS) is an inflammatory disease of the PNS and is the most common cause of acute flaccid paralysis, with an annual global incidence of approximately 1–2 per 100,000 person-years. GBS occurs more frequently in males than in females and the incidence increases with age, although all age groups can be affected. Patients with GBS typically present with weakness and sensory signs in the legs that progress to the arms and cranial muscles, although the clinical presentation of the disease is heterogeneous and several distinct clinical variants exist. Diagnosis of GBS is based on the patient history and neurological, electrophysiological and cerebrospinal fluid (CSF) examinations. Other diseases that have a similar clinical picture to GBS must be ruled out. Electrophysiological studies provide evidence of PNS dysfunction and can distinguish between the subtypes of GBS: acute inflammatory demyelinating polyradiculoneuropathy (AIDP), acute motor axonal neuropathy (AMAN) and acute motor sensory axonal neuropathy (AMSAN). To further improve the worldwide management of GBS, we aim to use this consensus report as a basis for the development of online information resources, training material and teaching courses. These resources will be directed towards healthcare workers, including clinical neurologists, as well as patients with GBS and their relatives [14].

Hang Liu. *e*t, al, (2019), Hepatitis E virus-associated Guillain–Barre syndrome: Revision of the literature, Guillain–Barre syndrome is a postinfectious and autoimmune-induced peripheroneural disorder, characterized by a rapidly progressive bilateral and symmetric weakness of limbs in its classic form (acute inflammatory demyelinative polyradiculoneuropathy, AIDP). Although AIDP was more common in reported cases, any other types of GBS may follow HEV infection. About two-thirds of patients have preceding infection within 3 weeks before onset of weakness. Some common infectious agents causing GBS are as follows: Campylobacter jejuni, cytomegalovirus (CMV), Epstein–Barr virus (EBV), Mycoplasma pneumoniae, Haemophilus influenzae, and hepatitis B virus. The purpose of this review is to clarify the pathogenesis of HEV associated GBS, the clinical presentations and diagnosis with a particular insight provided to the neurologists and hepatologists, and outline subsequent management and prevention. Although existing therapies are limited in providing a functional improvement, new programs of treatment should still be designed to employ in combination or sequential therapeutic strategies along with the scientific understanding of pathophysiological mechanisms of HEV-associated GBS. Hepatitis E virus infection was frequently associated with GBS or variants of GBS. Two possible pathogenesis mechanisms have been proposed and require further study to explore more details. Although PLEX or IVIG has been widely used to treat HEV-associated GBS, it is worth discussing whether antiviral monotherapy or combination of ribavirin and immunotherapy can be used as a novel treatment. If the pathogenesis was clarified sufficiently, the answer to the question would be straightforward because direct neural infection could respond well to antiviral therapy. Prevention and early diagnosis of HEV-associated GBS can be difficult and challenging because prodromal symptoms of infection are usually asymptomatic or mildly symptomatic [15].

Mario Emílio Dourado. (2018), Guillain-Barré syndrome (GBS), was introduced in 1927 by Drăgănescu and Claudian at the Congress of the Neurology Society of Paris, which was led by Barré. The reasons why Strohl was omitted from the eponym are not clear. The eponym is still used worldwide for its clarity in describing GBS, making its diagnosis easy and accessible to the non-neurological community. Interestingly, Guillain maintained that a microorganism would eventually be found as the cause of the syndrome, as described for poliomyelitis. We now know that approximately two-thirds of patients with GBS report a previous infectious illness, most commonly diarrhea or a respiratory disease, or vaccination, days to weeks preceding the onset of neurologic signs. The recent outbreak of Zika virus (ZIKV), in a naïve population in Latin America and, before that, in French Polynesia, further supports Guillain's original hypothesis of an infectious etiology for triggering the syndrome. Guillain-Barré syndrome is now considered an immune-mediated disorder; autoantibodies may form in response to a variety of antigenic stimuli, either of bacterial or viral origins. This

appears to be the mechanism induced by Campylobacter jejuni in the axonal subtype of GBS. The mechanism of the demyelinating variant associated with viral infections is still not understood. In the majority of cases the onset of neurological involvement occurred within days of the onset of symptoms and findings characteristic of ZIKV infection. The outbreak of ZIKV was also associated with microcephaly in newborns infected in utero. The ZIKV outbreak drew attention to the need for the medical community be aware of GBS. In the majority of patients, the treatment of GBS requires an intensive care unit, which is not available in many rural communities [16].

Shuang Liu, *e*t, al, (2018), Immunotherapy of Guillain-Barre syndrome, Guillain-Barre syndrome (GBS) is an acute onset, monophasic, immune-mediated peripheral nerve and nerve root disorder (termed polyradiculoneuropathy), which was first recognized as a distinct medical condition in 1916. GBS has become the most common cause of acute flaccid paralysis worldwide, following the near-eradication of poliomyelitis, and is a neurological emergency. The reported incidence of GBS in Europe and North America ranges from 1 to 2 cases per 100,000 adults, and 0.4 to 1.4 cases per 100,000 children per year. There also appears to be a linear increase in incidence with age and a slightly higher frequency in males compared to females. Despite significant advances in understanding the pathogenesis of immune-mediated disorders and the development of targeted molecular based therapies, current immunotherapies for GBS are non-specific and only partly efficacious. Improved collaboration is needed between neurologists, neuroscientists, immunologists, medical chemists and pharmacologists to deduce and design more effective immunotherapies for GBS. It is imperative to consider the role of the blood-nerve barrier in GBS pathogenesis and therapeutic development, and we advocate pathogenic leukocyte trafficking as a biologically relevant mechanistic target with translational potential for disease-specific immune modulatory therapy using function-neutralizing antagonists such as humanized monoclonal antibodies that do not require blood nerve barrier permeability and retention within peripheral nerve/ nerve root endoneurium [17].

Nazgol Motamed-Gorji. *e*t, al, (2017), Biological Drugs in Guillain-Barré Syndrome: An Update, Guillain-Barré Syndrome (GBS) is a term used to describe acute autoimmune peripheral neuropathy with specific characteristic of ascending symmetrical flaccid paralysis of limbs accompanied with hyporeflexia or areflexia. Many consider GBS as a post-infectious inflammatory disorder, since it is commonly preceded by a viral or bacterial infection. Due to near eradication of Poliomyelitis, GBS is now considered the most common universal cause of acute flaccid paralysis. It predominantly commences with a progressive bilateral weakness in muscles of lower limbs, which rapidly ascends and spreads to the muscles of upper body, upper limbs and face. This motor dysfunction is frequently associated with a loss or attenuation of deep tendon reflexes throughout body. In severe cases of GBS, patients require mechanical ventilation as a result of respiratory failure. Main GBS variants comprise acute inflammatory demyelinating polyneuropathy (AIDP), acute motor axonal neuropathy (AMAN), and Miller-Fisher syndrome (MFS). Treatment of GBS could present a challenge for patients and physicians. GBS is a disease with diverse outcomes and various severity in different cases. Although many of the patients reach full recovery with routine treatments (IVIg and PE), residual defects could be still detected in a high prevalence of the cases. In some cases, in spite of receiving immunotherapy with traditional treatments, patients remain unable to walk unaided 6 months after the onset of the disease. Advancing molecular understanding of GBS pathoimmunology accounts for extensive potential biological treatments. Evidence by far suggests various possible biological agents for modulating GBS pathogenesis. current review represents a summary of what is already investigated regarding immunotherapeutic biological approaches to GBS, and what progress is required to improve these approaches via future studies. It is to be hoped that by further investigating biological drugs, GBS patients will experience better clinical outcomes and earlier function retrieval [18].

Goodfellow. *e*t, al, (2016), Guillain-Barré syndrome: a century of progress, Guillain–Barré syndrome (GBS) is the commonest cause of acute flaccid paralysis and manifests as rapidly evolving weakness and sensory disturbance in arms, legs and in some patients, facial, bulbar and respiratory muscles. Many patients will make a good recovery over many months but in severe cases patients can require months of intensive care support and be left with permanent severe weakness, sensory disturbance and pain. Furthermore, around 5% die from complications including respiratory failure, pneumonia and arrhythmias, making it a medical emergency with a high morbidity and significant mortality. In the 100th anniversary year of GBS, a new challenge has also come to the fore in the association of Zika virus infection with GBS. An immediate and pressing question is how countries that are experiencing or anticipating a Zika virus outbreak can prepare for the expected increase in cases of GBS, which threaten to overwhelm hospital and intensive care services. The immediacy of the challenge also highlights to importance of building on our progress so far, and gaining an even greater understanding of GBS in the years to come [19].

Lucas Masiêro Araujo. *e*t, al, (2016), The Zika virus (ZIKV) was named after the Zika forest, located in Uganda. It was first isolated in April 1947 from specimens collected from a sentinel rhesus monkey. Since this pivotal event, the virus has progressively spread outbounds, being detected in the sub-Saharan region, and later on in India and Southeast Asia, although only small limited outbreaks and sparse cases were reported in the following decades. The infection

outbreak has been recognized as a major public health issue in Brazil since the beginning of 2015. The ZIKV belongs to the Flaviviridae family, the same family of Dengue, hepatitis C, and yellow fever virus. ZIKV is an arbovirosis transmitted by Aedes aegypti mosquitoes which are found widespread in Brazil. Currently, there is scant but worrisome evidence suggesting it might also be transmitted via sexual intercourse through blood transfusion, as well as by vertical transmission. It is imperative that our medical societies and agencies of public health urgently develop training programs for health personnel on clinical suspicion and treatment of GBS and other neurological complications. In addition, the creation of collaborative. networks among neurological centers and referral hospitals would be advisable. Finally, it is an obligation and an opportunity for Brazilian neurologists to have make precedence and thoroughly study the current outbreak, producing invaluable scientific knowledge that could aid in further understanding of the pathophysiology, clinical course and therapy response of GBS [20].

Ma Xiaoyun. *e*t, al (2016). [Clinical efficacy of different doses of gamma globulin combined with glucocorticoid in treatment of moderate/severe acute Guillain-Barre syndrome in children: a comparative analysis], Intravenous immunoglobulin is a passive immunotherapy and is also recognized as an effective treatment that can shorten the course of treatment and alleviate the disease. The mechanism of action of intravenous immunoglobulin in the treatment of GBS is as follows: it provides specific IgG antibodies and neutralizes pathogenic autoantigens, which can block the interaction between autoantibodies and autoantigens and reduce nerve damage; the genetic polymorphism of macrophage membrane Fc segment receptor (FcγR) is related to the occurrence and development of GBS. Immunoglobulin can bind to FeSR, causing it to lose its antigen presentation function, blocking the polyclonal activation of peripheral blood monocytes and macrophages and the production of autoantibodies such as IgG, and blocking immune inflammatory reactions; it can compete with autoantibodies to bind to target tissues, inhibit T cells and NK cells and reduce pathological immune responses; it acts as a receptor for activated complement components, preventing complement from binding to form membrane-soluble immune complexes and preventing complement-mediated immune damage. Immunoglobulin can play a dual role of immune replacement and immune regulation. Its use in the acute phase can reduce the immune system's attack and damage to its own tissues and alleviate the disease. Hormones are immunosuppressants [21].

Mehndiratta. *e*t, al, (2016), Electrophysiological features of Gullian Barre syndrome newer insights, Gullian Barre syndrome (GBS) is classically a clinical diagnosis but electrophysiology and nerve conduction studies (NCS) help in supporting the diagnosis, making a distinction between axonal and demyelinating variants as well as helping in prognostication. The characteristic findings supportive of acute inflammatory demyelinating polyradiculoneuropathy include prolonged distal motor latencies, reduced conduction velocities, conduction blocks at nonentrapment sites, temporal dispersion and prolonged F wave latencies. Further studies in this regard are required to determine appropriate prognostication markers and electrophysiological parameters for assessing the occurence of an early respiratory failure and for estimating the duration of mechanical ventilation in critically ill GBS patients [22].

Sreenivasa Rao Sudulagunta. *e*t, al, (2015), Guillain-Barré syndrome: clinical profile and management, Guillain-Barré syndrome (GBS) is a fulminant polyradiculoneuropathy that is acute, frequently severe and autoimmune in nature. GBS is the most common cause of acute or subacute generalized paralysis which at one time rivaled polio in frequency. GBS is also known as Landry-Guillain-Barré-Strohl syndrome and acute inflammatory demyelinating polyneuropathy (AIDP). Global annual incidence is reported to be 0.6–2.4 cases per 100,000 per year. Men are more commonly affected by approximately 1.5 times than women. Acute inflammatory demyelinating polyradiculoneuropathy (AIDP) is the most commonly occurring subtype in North America and Europe accounting for about 90% of all cases. However, in other parts of the world (Asia, Central and South America) axonal variants of GBS i.e. acute motor axonopathy (AMAN) and acute motor sensory axonopathy (AMSAN) are found to represent 30% to 47% of cases. This retrospective study has the limitations in accurate calculation of incidence. No gender difference is observed. Increased age is associated with worse prognosis and increased frequency of GBS occurrence. Seasonal occurrence predominantly in winter is noted. Peak flow test may be a predictor of assessing requirement of mechanical ventilation and prognosis. Conduction block is the major abnormality noted in electrophysiological studies and proximal nerve segment assessing with Erb's point stimulation has high predictive value. Proximal segment involvement is more common than distal segment involvement as per electrophysiology studies. No difference in complications and outcome is found in treatment regimens of IVIG and plasma exchange. Mortality rate is comparable. IVIG treatment is more expensive but is associated with less duration of hospital stay [23].

3. Conclusion

This study highlights the critical need for early diagnosis and prompt treatment in Guillain-Barré Syndrome (GBS), emphasizing the effectiveness of intravenous immunoglobulin (IVIg) and plasma exchange (PE) in improving patient outcomes. While most patients recover, some face prolonged disability or respiratory complications, making supportive

care essential. The study also underscores the psychosocial impact, including fatigue and quality of life issues, even after recovery. By improving understanding and management of GBS, this research will benefit society by fostering better treatment protocols and patient support systems, ultimately enhancing recovery rates and quality of life for individuals affected by GBS.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

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