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Overview of Guillain–Barré Syndrome (GBS)

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ABSTRACT

Guillain–Barré syndrome (GBS) is a disorder of the immune system that impacts the peripheral nervous system (PNS). It is marked by severe inflammation that progresses rapidly and is mainly manifested through muscle weakness. The current literature review aims to provide a general overview of GBS, that includes its etiology, pathophysiology, and management, to gain insight into the relevant research that has already been executed.Anti-GM1 along with anti-GQ1B are two of the aforementioned antibodies that have been revealed to be especially crucial in targeting and damaging peripheral nerves or else neuromuscular junctions (NMJ). Furthermore, it has been discovered that patients' anti-GD1a antibodies attach to both the paranodal myelin of the affected nerves and the nodes of Ranvier of the fringe nerves in addition to the NMJ. According to reports, this infection has been implicated in a variety of neuropathies that develop as a consequence of immune-mediated post-infection consequences. Furthermore, because it has been investigated in animal models, atomic mimicry has been revealed to be fundamentally linked to the onset of

the disease in another investigation. It has previously been observed that the gastrointestinal infection causing bacterium *Campylobacter jejuni* performs a role in the development of GBS in humans. In any case, researchers have revealed that plasma exchange (PE)along with intravenous immunoglobulins (IVIG) stay the vital and useful components in the illnessmanagement. Subsequent trials have evaluated her methods, which are less effective and may result in significant negative events and difficulties.

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INTRODUCTION

An immune system disorder is Guillain–Barré syndrome (GBS) which results in polyradiculoneuropathy. It is distinguished by intense inflammation which quickly impacts the PNS (peripheral nervous system), primarily showing clinical signs through muscle weakness along with associated sensory symptoms. [1] The symptoms can develop either suddenly or gradually over several days to weeks. Severity of the infection can vary significantly, ranging from mild instances to very severe cases, which could lead to serious health issues for example respiratory failure, autonomic dysfunction, as well as tetraplegia. [2].

As per estimates, the mortality rate from GBS in patients from developed nations is around 2 to 3percent. However, previous research shows that administering IVIG(intravenous immunoglobulins) and conducting PEmay greatly improve the disease prognosis as well as promote recovery. While many cases of the condition generally have a favorable outcome, it has been computed that about 15percent of GBS patients experience poor consequences in spite of sufficient treatment through established methods mainly because of serious complications, especially weakness in the respiratory muscles, which might require mechanical ventilation. Since GBS is currently the primary cause of both acute and subacute forms of flaccid paralysis, subsequent findings from Western nations have further demonstrated that the national efforts aim to eradicate poliomyelitis. Seven different global studies suggest that earlier research has indicated the occurrence of GBS varies from 0.16 to 0.4 instances for every 100,000 people across all age groups each year [2,3]

The clinical consequences that patients typically experience are the main basis for diagnosing GBS. This involves the disease's monophasic nature, diminished or absence of deep reflexes, and a rise in lower limb muscular weakness. Furthermore, the cerebrospinal fluid (CSF) examination along with nerve conduction testing are crucial diagnostic procedures that may be utilized to validate the diagnosis. [4,5] In addition, the malady incorporates several variations which vary from one another within the clinical presentation pattern connected to seriousness as well as forecast of this condition. In addition, past research has shown that these variations moreover vary from one another within the research facility discoveries and infection's pathophysiology. To understand the present concepts that are essential to this show writing review, this current review aims to discuss the etiology, pathophysiology, along with management of GBS in general. [2,3]

The clinical consequences that patients typically experience are the main basis for determining the GBS. This comprises enhancement of the muscle weakness in the lower limbs, the reduction or absence of deep reflexes, as well as monophasic pattern of infection. Additionally, CSF examination and nerve conductivity tests constitute crucial demonstration procedures that can help to validate the diagnosis. [4,5]Furthermore, the illness has a variety of variances that vary from one another in the pattern of clinical presentations pertaining to the severity and prognosis of the disorder. Furthermore, previous publications have demonstrated that these variations also vary from one another in terms of the pathophysiology of the illness and the discoveries made by the research center. In order to clarify current concepts that are significant to this literature review, show writing review aims to discuss the etiology, pathophysiology, and management of GBS.

ETIOLOGY

Particular components are already stated to connect with improvement of GBS along with its variations. As per reports, the infection has been linked to different kinds of neuropathies that arise as immune-mediated, post-infection consequences. Previous studies using animal models have suggested that molecular mimicry might contribute to the advancement of infections. *C.jejuni*, a bacterium which is responsible for gastrointestinal infections, are found to elevate the GBS risk in humans. This connection likely stems from the resemblance between the antigen structures of gangliosides in the peripheral nerve cells along with lipooligosaccharide found in bacterial outer membrane, causing an

antibody-driven response targeting these nerve cells. As with the infection, the neuropathic effects of GBS will probably manifest as an immune-mediated response.

In expansion to the *C.jejuni*, additional diseases causing gastrointestinal as well as respiratory diseases have moreover been related to advancement of the GBS. Over 2/3 of the affected patients have been found to have a history of infection throughout the first 1-6weeks of triggering GBS-related symptoms.[5]For example, Dirlikov et al. have already described the amount of GBS cases have been examined and documented by tracking the Zika virus's contamination throughout a past epidemic.[7]

Additionally, past instances have detailed that a few other etiologies except the viral contaminations as a few medicines along with surgical controls may also incline to GBS development. Within early inoculation campaigns programme against flu A/H1N1 by the flu vaccines in the year 1976, it had be enevident that numerous instances of GBS and neuropathies were created in connection to vaccination organizations, in spite of the fact that it was detailed that within the taking after a long time, the numbers of GBS cases taking after immunization has essentially diminished to as it were 1 case per 1million vaccines managed. Research conducted in past has shown that individuals who have experienced the flu are seven times more prone to develop GBS in contrast with those who have not individuals who have been vaccinated against the disease.[9,8]

Clinical Features and Variants

Common Symptoms

The foremost common clinical introduction of GBS is bilateral upper and lower limb weakness.GBS ordinarily starts suddenly with distal, generally symmetrical paraesthesias, rapidly followed by progressive limb weakness Pain could be a conspicuous indication for numerous patients [20].

Diagnostic Criteria

The progressive weakening of both legs, along with the presence of areflexia, serves as a critical factor for diagnosis. Steady auxiliary testing includes CSF analysis and electrodiagnostic testing. The Brighton criteria are applied to assess GBS, featuring a range that spans from high to low certainty regarding symptoms. [21].

	Patterns of limb weakness	Sensory involvement	Cranial nerve involvement	CNS involvement	Serial neural conduction	IgG against ganglioside type	Proportion of patients with Guillain-Barré syndrome
Guillain-Barré syndrome spectrum							
Classic							
Demyelinating	Upper and lower limbs	Yes	Yes	No	AIDP	Unknown	69–90%
Axonal	Upper and lower limbs	Yes in AMSAN, no in AMAN	Yes	No	AMSAN, RCF	GM1, GD1a	<22%
Pure motor	Upper and lower limbs	No	Yes	No	AMAN, RCF	GM1, GD1a	5–70%
Pure sensory	None	Yes	No	No	Abnormal SNAPs	GD1b	<1%
Paraparetic	Lower limbs	Yes	No	No	Axonal	GM1, GD1b	5–10%
Facial diplegia and paraesthesia	None	Yes (distal)	Facial	No	AIDP	Unknown	<5%
Pharyngeal, cervical, brachial	Proximal upper limbs	Supportive	Bulbar	No	Equivocal	GT1a, GQ1b	<5%
Acute bulbar palsy	None	Supportive	Bulbar	No	Equivocal	GT1a	<1%
Guillain-Barré syndrome with hyperreflexia	Upper and lower limbs	Yes	Yes	No	Axonal	GM1	<1%
Miller Fisher syndrome spectrum							
Classic	None	Ataxia	Ocular motor nerves	No	Abnormal SNAPs	GQ1b, GT1a	4–25%
Acute ophthalmoplegia	None	Supportive	Ocular motor nerves	No	Normal	GQ1b	<1%
Acute ataxic neuropathy	None	Ataxia	No	No	Axonal	GM1	<5%
Acute ptosis	None	Supportive	Ptosis only	No	Normal	GQ1b	<1%
Acute mydriasis	None	Supportive	Dilated pupils	No	Normal	Unknown	<1%
Acute vestibular syndrome	None	Supportive	Nystagmus	Nystagmus	Normal	GQ1b	<1%
Bickerstaff brainstem encephalitis							
Classic	None	Supportive	Ocular motor nerves	Yes	Axonal	GQ1b, GT1a	<5%
Acute ataxic hypersomnolence	None	Ataxia	No	Yes	Normal	GQ1b	<1%

AIDP=acute inflammatory demyelinating polyneuropathy. AMAN=acute motor axonal neuropathy. AMSAN=acute motor and sensory neuropathy. RCF=reversible conduction failure. SNAP=sensory nerve action potential.

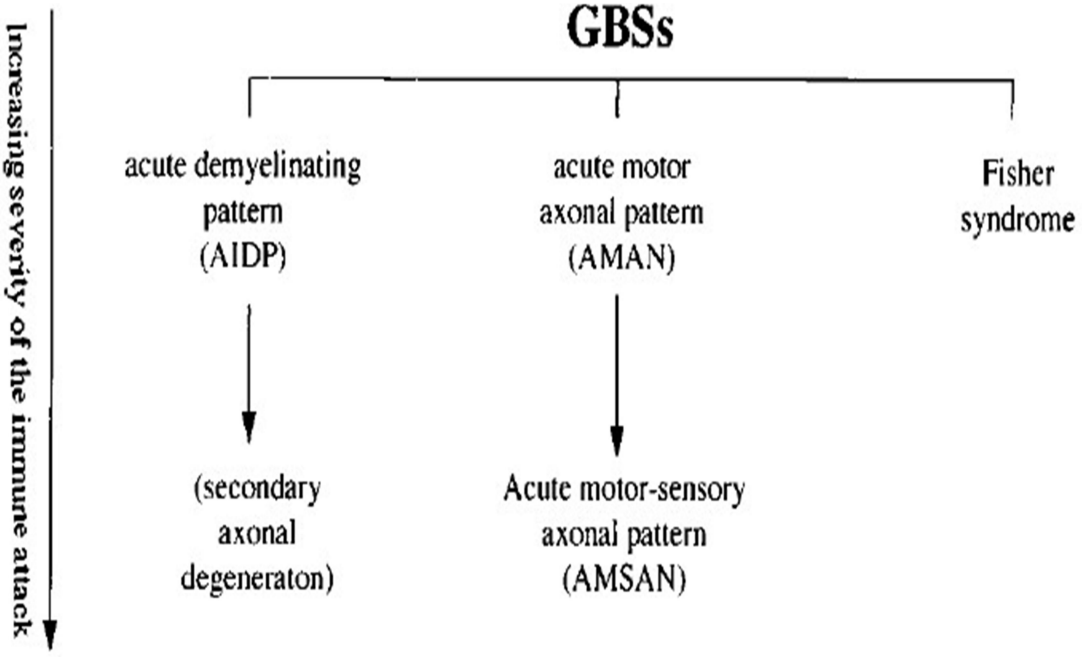
Table 3: **Clinical classification of Guillain-Barré syndrome**

GBS Variants

GBS can be classified into various subtypes according to its clinical symptoms, underlying mechanisms, and neurophysiological traits. The acronym GBS is associated with (AIDP). The spectrum of conditions included in this classification has expanded to encompass axonal variants and more specific types, for example,pharyngeal-cervical-brachial syndrome, Miller Fisher syndrome, bifacial weakness with limb paresthesia (BFP), and paraparetic conditions.[20,22,23]

Regional Differences in GBS Subtypes

The variations of GBS vary across different regions globally.AIDP accounts for 90percent of cases in North America and Europe, therefore being the most predominant subtype. AMAN, on the contrary hand, is the most widespread subtype in China and Japan. In India, the situation is moderate, with nearly equal shares of AIDP, AMAN, though AMAN tends to be more prevalent among younger patients [20].



Pathophysiology

This region is extensively analyzed in relation to this document. As per previous investigations, 70% of individuals with GBS had a history of infections, suggesting that illnesses may have a role in the development of GBS. In order to better understand the possible component and atomic resemblance that the infecting organisms might have with the tissues and cells of the infected individuals that caused GBS, especially the axonal kind, efforts were coordinated. This has previously been demonstrated by Yuki et al., who revealed that the lipooligosaccharide that is widely present in the structure of *C. jejuni* is comparable to the atomic structures of the gangliosides that exist inside the fringe nerves. Research facility testing in rabbits exposed to a lipooligosaccharide that replicates human gangliosides has demonstrated this notion in previous investigations. The research on fringe nerves showed that the rabbits in the study developed symptoms akin to GBS (specifically acute motor axonal neuropathy), which led to flaccid tetraplegia.[6,7]

Antibodies that target gangliosides at the same time affect various elements of the PNS. The GM1 and GQ1B antibodies are recognized for their ability to damage the NMJ or the peripheral nerves. Observed that in patients, existence of anti-GD1a antibodies is attached to NMJ, with nodes of Ranvier in peripheral nerves as well asparanodalmyelin of affected nerves. Considering the various elements

affected by distinct antibodies and the stimulation of antigens, it is recommended that these variances are main reasons for varying progression of disease.

Aside from that, a previous study conducted by Susuki et al revealed, following an infection with a pathogenic organism exhibiting similar antigenic properties, complement activation had been viewed in infected patients, implying a significant role for it in the pathophysiology and improvement of the illness.As already said, It has been discovered that some antibodies may be more likely to exhibit particular GBS variants and symptoms. For example, anti-GM1 antibodies have been found to improve the variety of axonal motor neuropathy, but anti-GQ1B antibodies are additionally connected to Miller-Fisher disorder. [11,12]Additionally, it has been established that anti-GT1A antibodies are connected to an improved GBS variant of pharyngeal-cervicalbrachial syndrome.[13]

Effectiveness of these antibodies, encompassing both their specificity as well as effectiveness in detecting, identifying various strains, is limited and should only be regarded as supplementary for verification of theevaluation and conclusion. Therefore, more research may be necessary to support the explanation of the possible key roles that these antibodies may play in the pathophysiology and differentiation of GBS and its variants, which are evident in the utilization of considerably better methods for presenting and treating the condition. Despite the ongoing monitoring of acute inflammatory demyelinating polyneuropathy, which is most frequent form of GBS in the US, the underlying mechanisms of this condition remain poorly understood and warrant further investigation are required.

DIAGNOSIS

Diagnosis can performed by analyzing CSF and conducting electrodiagnostic tests. A thorough physical assessment and a detailed medical history are crucial for achieving an accurate diagnosis. It is crucial to perform by analyzing CSF and conducting in high level of clinical awareness for GBS, particularly in acute medical environments, as cases may be overlooked if fundamental clinical skills are ignored. [20]

1.Cerebrospinal Fluid (CSF) Analysis

CSF analysis indicates elevated protein levels and keeping cell count within the normal range. However, a mild pleocytosis (5-50 cells/l) is detected in some cases. An enhanced concentration of the protein in

CSF is significantly influenced by when a lumbar puncture has been executed following initial appearance of weakness.

2. Electrodiagnostic Testing

Electrodiagnostic testing plays crucial role in diagnosis of GBS. This testing reveals signs of demyelination, variations in signal timing, notably reduced conduction speeds, and extended F-wave latencies. Observations of demyelination, conduction block and irregular slowdowns in conduction speeds, are particularly informative so these results are indicative of acquired demyelinating neuropathies. [22]

1.Brighton Criteria

The Brighton criteria are a collection of diagnostic guidelines utilized to categorize patients with GBS according to the degree of diagnostic certainty. These criteria take into account clinical characteristics, CSF results, and electrophysiological examinations to classify patients into various diagnostic categories.

TREATMENT

Immunotherapy, that includes PE and IVIg, is a key component of GBS treatment These treatments have been shown to reduce mortality and disease-related issues by addressing secondary complications developed during the hospital course. Despite management with immunotherapy, some patients still require mechanical ventilation [20].

Plasma Exchange (PE)

Plasma exchange (PE) is an effective immunotherapy for GBS, particularly when initiated early in the disease course. PE remove plasma containing harmful antibodies and replaces it with replacement fluids. PE shows improvement in recovery of walking ability, percentage requiring artificial ventilation, duration of complete strength recovery after 1year.

Intravenous Immunoglobulin (IVIg)

IVIg is an efficient treatment option for GBS. Over the duration 5days, a dosage of 0.4g/kg of body weight is administered [1]. RCTs have indicated that IVIg is as efficient as PE in improving disability grade at four weeks, mortality, and disability

	Distinguishing clinical features	CSF findings	Neural conduction findings	Other supportive tests
Brain				
Encephalitis	Drowsiness, seizures	Pleocytosis	Normal	Brain MRI for hyperintense lesions, EEG for slowing epileptiform discharges
Brainstem stroke	Hyperacute sudden onset, cranial and limb weakness	Normal	Normal	Brain MRI and magnetic resonance angiography for corresponding infarct and vascular occlusion
Spinal cord				
Transverse myelitis	Sensory level, brisk reflexes	Normal	Normal	Abnormal spine MRI for hyperintense lesions
Malignant infiltration	Cauda equina syndrome	Malignant cells	Normal	Abnormal spine MRI for enhancing lesions, investigations for primary lesions
Anterior horn cell				
Infection with Poliovirus, enterovirus 71, or enterovirus D68	Fever, flaccid paralysis	Pleocytosis	Motor neuronopathy	Presence of virus
Plexus				
Neuralgic amyotrophy	Asymmetry, pain, and findings limited to affected nerves	Normal	Abnormal in affected nerves	Brachial plexus MRI for nerve enhancement
Nerve roots				
Cytomegalovirus and HIV radiculitis	Subacute presentation	Pleocytosis	Delayed or absent F waves and H waves	HIV and cytomegalovirus serology
Chronic inflammatory demyelinating polyneuropathy	Subacute presentation and relapsing–remitting pattern	Albumin-cytological dissociation	Demyelinating neuropathy	Nerve ultrasound for enlarged nerve roots
Peripheral nerves				
Chronic inflammatory demyelinating polyneuropathy	Subacute presentation and relapsing–remitting pattern	Albumin-cytological dissociation	Demyelinating neuropathy	Nerve ultrasound for enlarged nerve roots, and proximal and distal nerves
Porphyria	Family history, concomitant psychiatric and abdominal pain	Normal	Axonal neuropathy	Increased urinary porphobilinogen
Lyme disease or other tick-borne diseases	History of exposure, characteristic rash (erythema migrans in Lyme disease)	Normal	Axonal neuropathy	Antibodies against *Borrelia burgdorferi* (Lyme disease) or the related tick species
Thiamine deficiency	Predisposing factors (eg, hyperemesis gravidarum, alcohol misuse, nutritional deficiency, and other neurological features such as Wernicke encephalopathy)	Normal	Axonal neuropathy	Reduced blood thiamine and erythrocyte transketolase activity
Diphtheria	Laryngeal infection	Increased total protein	Demyelinating neuropathy	Isolation of *Corynebacterium diphtheriae* on cultures
Critical illness polyneuropathy	Prolonged illness or ventilation	Normal	Axonal neuropathy	Overlap with myopathy
Metabolic or electrolyte imbalance	Predisposing factors	Normal	Normal	Low serum concentrations of abnormal electrolyte
Neuromuscular junction				
Myasthenia gravis	Fatigable weakness, relapsing–remitting pattern	Normal	Repetitive nerve stimulation for a decremental response	Acetylcholine receptor antibodies
Botulism	Rapid progression, pupillary abnormalities, dysautonomia, and descending paralysis	Normal	Rapid repetitive nerve stimulation for an incremental increase	Botulism toxin
Lambert-Eaton syndrome	Proximal weakness, depressed tendon reflexes, and autonomic changes	Normal	Repetitive nerve stimulation for post-tetanic facilitation	Antibodies against voltage-gated calcium channels
Muscle				
Inflammatory myositis	Proximal weakness, normal reflexes and sensation	Normal	Normal sensory potentials	Increased serum creatine kinase, myopathic electromyography
Critical illness myopathy	Prolonged illness or ventilation	Normal	Normal sensory potentials	Overlap with neuropathy
Hypokalaemic periodic paralysis	Transient weakness, family history, triggering factors (eg, fasting, exercise, and carbohydrate-rich meals)	Normal	Abnormal exercise test	Low serum potassium concentrations, genetic mutation
Miscellaneous				
Functional disorder	Inconsistent, variable presentation	Normal	Normal	Psychological evaluation

Table 2: **Differential diagnosis of Guillain-Barré syndrome by anatomical site and illness**

Comparative Effectiveness of PE and IVIg

The plasmapheresis (PE) along with IVIg are theefficient treatments for GBS; however, there is no agreement on which treatment is more effective. The effectiveness of IVIg versus PE in individuals with

severe GBS symptoms has been reviewed and analyzed in a systematic manner. In areas where resources are scarce, it may be appropriate to administer smaller doses of PE.

Supportive Care

Supportive care performs a crucial role in managing patients with GBS, particularly for those experiencing serious symptoms. Observation of fluctuations in blood pressure along with heart rate, while also being aware of possible complications such as ileus. Patients who show symptoms but are able to walk on their own may be managed conservatively in specialized care settings.

Mechanical Ventilation

Mechanical ventilation is required for patients with respiratory failure. Indicators that respiratory failure is approaching include rapid breathing (tachypnea), increased heart rate (tachycardia), sweating on the forehead, uncoordinated movements of chest and abdomen, a vital capacity below 20mL/kg, and maximal inspiratory pressures lower than 30mm H2O. Simple bedside tests, for example, a single breath count, can be good predictors of the requirement for mechanical ventilation [20]

Management

The effectiveness of previous modalities in the treatment of GBS has been demonstrated by previous randomized controlled trials. The two most effective and currently authorized treatments for GBS patients are plasma exchange and IVIG. Prior research has demonstrated the efficacy of plasma exchange in improving the removal of humoral mediators, complement proteins, as well as antibodies that are generally thought to essentially contribute to the development and pathogenesis of GBS. It has been recommended that five separate sessions be utilized for the volume exchange of plasma. Additional research is necessary to properly understand the precise mechanism of plasma exchange. Even though it has been stated that the mechanism underlying IVIG's physiology and activity in playing this role is still up for debate and has not been proven, it has been observed that IVIG may have potent immune-modulating properties. A total dose of two grams per kilogram should be taken into consideration, and IVIG should be administered over a period of 5days. Prior research comparing safety along with effectiveness of various treatment approaches indicates that they are equally beneficial in managing the condition. [14]

According to previous studies, both approaches must be successful in administering the virus. Any one of them should be treated for four weeks in a row without giving any one of them an edge over

the others. In addition, any of these modalities must be utilized for treatment within the first 2weeks for a more robust and effective management strategy. Corticosteroids are utilized for many years to address immune-mediated responses, yet recent studies indicate that the effectiveness of using corticosteroids, whether administered intravenously or orally in combination, may vary andmust belower than that of utilizing fake treatment or when in contrast with the viability of utilizing IVIG and plasma trade if alone or else when utilized in the combination. Utilizing these treatment options has been observed to considerably reduce the recovery duration from GBS.Previous research has indicated that individuals receiving these treatments could achieve independent walking within 32 days after their illness began. [14,15,16] Reports suggest that prognosis forGBS patients is usuallyfavorable, with estimates suggesting that approximately 80percent or more have a strong likelihood of recovery as well asdecline complications. Nevertheless, remaining percentage of individuals typically experience complications and severe issues. Furthermore, administering plasma exchange along with intravenous immunoglobulin (IVIG) at intervals, or using steroids following IVIG treatment, did not lead to significant positive outcomes. A randomized controlled trial carried out in 2021 confirmed this conclusion, indicating that giving a second dose of IVIG did not correspond with any notable enhancements inpatients sufferingfrom severe GBS and could potentially lead to a less favorable prognosis due to various side effects.An observational study by Verboon et al. previously reported similar findings, revealing that more IVIG doses do not significantly improve the prognosis for patients suffering from severe GBS. Although prior studies have examined the effectiveness of several alternative treatment methods, none have been shown to provide considerable benefits in contrast with plasma exchange along with IVIG. [17,18,19]

Management of Dysautonomia

For people suffering from GBS, acute dysautonomia is a major cause of death. Postural tachycardia is a typical manifestation of cardiac as well as hemodynamic abnormalities. Management involves closely observing the condition and, in certain instances, the placement of a pacemaker for bradycardia orele sinus arrest.

Pain Management

Pain is a significant symptom experienced by patients suffering from GBS and necessitates careful management. Opioid analogs must be administered carefully because they can reduce bowel movement

activity.Additional drugs, that includecarbamazepine, gabapentin, NSAIDs, acetaminophen, tricyclic antidepressants, may be utilized.

Nutritional Support

Nutritional assistance is crucial, particularly for patients who need mechanical ventilation.Gastric tube or else nasogastric feeding must be instituted gradually. A high-energy (40 to 45 nonprotein kcal), high-protein (2-2.5g/kg) diet has beensuggested to assist with weaning from mechanical ventilation [20].

PROGNOSIS AND OUTCOMES

Factors Influencing Prognosis

The long-lasting effects of GBS can influence patients' professional and personal lives for as long as 3 to 6 years following the illness. About 20-30% of children experience ongoing disabilities. A considerable portion of patients, roughly one-third, experience significant fatigue as a persistent effect.

Mortality and Disability

GBS mortality rates in Spain have seen improvement from 1999 to 2013.Age of death has risen, while mortality rate has declined. However, mortality rates can vary by region, with higher rates observed in areas with limited resources.

Predictors of Poor Outcome

ImprovedsNfL(serum neurofilament light) chain concentration specifies a poor consequence in GBS. Higher sNfL levels correlate with longer hospitalization times and decline likelihood of discharge with favorable walking ability. Clinical characteristics such as weakness in bulbar muscles and a quick onset of maximum limb weakness can indicate the likelihood of extended mechanical ventilation.

Rehabilitation

Rehabilitation plays a critical role in the recovery of GBS patients.Functional independence can be enhanced by physical therapy procedures that emphasize functional mobility, cardiovascular endurance, balance, gait training, as well as lower extremity strengthening. A biopsychosocial perspective that takes into account biological, psychological, and social elements can also facilitate recovery.

GBS and Vaccination

Association with Influenza Vaccination.

There is substantial evidence indicating a connection among influenza vaccination and GBS. Occurrence of GBS among those who received the swine flu vaccine was approximately 1 to 2 cases/million.

Association with COVID-19 Vaccination

GBS has been reported as a complication of COVID-19 vaccines. Some instances of GBS have been statedsubsequentVaxzevria COVID-19 vaccine. However, COVID-19 vaccine has many more advantages than risks.

Causality and Further Research

Numerous instances have expressed a temporal correlation between the onset of GBS and the Ad26.COV2.S vaccination. The cross-reactivity of antibodies triggered by peripheral nerve glycoproteins and adenovirus vaccination components is proposed as a viable underlying pathogenic mechanism. More research is needed to establish the connection between vaccines and GBS [21,23]

CONCLUSION

GBS is a multifaceted and varied condition that is exhibited with diverse clinical manifestations and outcomes.Timely diagnosis and immediate treatment with immunotherapy and supportive measures are crucial for enhancing patient results.Further investigation is needed to enhance our comprehension of the development of GBS, identify markers for disease progression, and create new therapeutic strategies.

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