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An Up-to-Date Review on Guillain–Barre Syndrome

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Abstract

Guillain–Barré syndrome (GBS) is an infrequent acute paralytic polyneuropathy that impacts around one in every 1 lakh people. It can affect both children and adults. GBS is an autoimmune condition of the peripheral nervous system that is developed due to an immune-mediated response to antigens that react with the myelin sheath of peripheral nerves, causing demyelination and/or axonal injury. In serious conditions, GBS can cause respiratory failure and even death also. The treatment with intravenous immunoglobulin and/or plasmapheresis, with an aim to neutralize and dispose the circulating antibodies from the bloodstream, respectively, in addition to supportive measures to maintain motor function of nervous system is presently available.

Keywords: Acute paralytic polyneuropathy, Demyelination, Guillain–Barre Syndrome, Peripheral nerves

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Introduction

Guillain–Barré syndrome (GBS) is a condition, in which the immune system attacks the peripheral nervous system. This can also be defined as a monophasic, immune-mediated polyneuropathy with an acute onset that often occurs after an antecedent infection.[1] GBS is a common cause of acute flaccid paralysis characterized by symmetrical weakness of the limbs and hyporeflexia or areflexia that lasts for 4 weeks.[2] Sensory symptoms such as numbness or paresthesia usually begin distally and follow a symmetrical pattern.[3] Acute inflammatory demyelinating polyneuropathy and acute motor axonal neuropathy are the two most common subtypes of GBS. Miller–Fischer syndrome**,** characterized by ophthalmoplegia, ataxia, and areflexia, is a less common subtype of GBS.[4]

GBS is most commonly caused by an infectious disease that causes the immune system to produce antibodies that react with gangliosides on nerve membranes.[5] This autoimmune response causes nerve damage or blockade. *Campylobacter jejuni* is the most common pathogen that is considered to cause signs and symptoms of GBS.[6]

GBS is currently treated with intravenous immunoglobulin (IVIg) and plasma exchange, both of which have been shown to be effective. Despite these treatment options, many patients continue to experience severe disease, pain, and residual deficits.[7]

In the present review, the team of authors focused on the history, epidemiology, causes, pathogenesis, signs and symptoms, diagnosis, therapeutic strategy of GBS, and the life after GBS.

History

The clinical features of GBS were determined in 1859.[8] In some cases of peripheral neuropathy, lymphocytic inflammation of the nerve was described by Eichorst in 1877 and Leyden in 1880.[9] The characteristic cerebrospinal fluid findings of increased protein concentration and normal cell count in two French soldiers were described by Guillain, Barre, and Strohl in 1916.[10] Haymaker and Kernohan described the clinical and histopathological features of 50 fatal cases of GBS in 1949, including inflammatory changes of the peripheral nerve.[11]

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In the mid-1950s, Waksman and Adams used homologous or heterologous peripheral nerve tissue combined with Freund adjuvant to induce experimental allergic neuritis in animals.[12]

Plasma exchange was discovered to be an effective treatment in the 1980s, and intravenous immunoglobulin efficacy was demonstrated in the 1990s (IVIg).[13]

Epidemiology

An annual incidence rate of GBS has increased with age (0.06/100,000 in children and 27/100,000 in elderly people aged 80 and up), and males are slightly more prone than females to develop this disease.[14] Seasonal variations have been reported, presumably due to changes in infectious antecedents, but these findings are rarely statistically significant.[15] In the past 5 years, reports from a variety of geographic areas have been published, indicating that the disorder's local incidence rate may be higher in some areas, possibly due to higher rates of infectious organism exposure. There have been several outbreaks of GBS, the most recent of which was linked to *C. jejuni* infections. The disorder can affect multiple family members, but this is unusual and could be due to a coincidence, a common antecedent infectious history, or unknown heritable factors.[16]

Similarly, only a small percentage of infected people (roughly one percent) will mount the specific humoral immune response that causes GBS in *C. jejuni* outbreaks. Overall, the estimated lifetime risk of developing GBS for any individual is less than one in 1000, based on the incidence rate and life expectancy.[16]

Causes

The exact cause of GBS is not known. It can be triggered by a variety of infections, the most common of which is a respiratory infection that causes cold or influenza-like symptoms such as fever, runny nose, cough, and generalized aches and pains.[17] Reports are there as following infection with the Zika virus and COVID-19 virus. There is no link between the severity of symptoms and the development of GBS; a person is just as likely to develop the syndrome after a minor flu-like episode as they are after a severe one.[18]

The specific infectious organism is rarely identified, partly because the infection has usually subsided by the time GBS manifests, and partly because most doctors do not bother to look for it.[19]

Microorganisms that cause respiratory illness and can then trigger GBS include *Cytomegalovirus* and an unusual bacterium called *Mycoplasma*.[20]

Gastroenteritis is the second most common antecedent illness that causes diarrhea, nausea, and vomiting.[21] Again, the exact identity of the infecting organism is usually unknown, but infection with *C. jejuni*, a common food contaminant, is the most frequently identified cause when thoroughly investigated.[22]

The Epstein–Barr virus, which causes infectious mononucleosis, can also be a trigger. The virus that causes acquired immune deficiency syndrome that has been linked to GBS. It usually occurs in the early stages of human immunodeficiency virus infection, before the immune system has been severely compromised; it can happen as soon as a few weeks after the initial infection.[23]

Risk Factors

GBS can affect people of all ages. However, the risk increases with age. Males are more likely to suffer from it. Infection with campylobacter, a type of bacteria commonly found in undercooked poultry, influenza virus, cytomegalovirus, Epstein–Barr virus, Zika virus, hepatitis A, B, C, and E, HIV, the virus that causes AIDS, Mycoplasma pneumonia, surgery, trauma, Hodgkin's lymphoma, influenza vaccinations or childhood vaccinations, COVID-19 virus can all cause GBS.[16]

Complications

GBS wreaks havoc on the patients' nerves. People with GBS may experience the symptoms such as the weakness or paralysis can spread to the breathing muscles, which can be fatal. Up to 22% of people with GBS require temporary breathing assistance from a machine within the 1st week of being hospitalized for treatment. The majority of GBS patients recover completely or have only minor residual weakness, numbness, or tingling. GBS is known to cause blood pressure fluctuations and irregular heart rhythms (cardiac arrhythmias).[24] One-third of GBS patients experience severe nerve pain, which can be alleviated with medication. GBS can cause sluggish bowel function and urinary retention. People who are unable to move as a result of GBS are at risk of developing blood clots. Taking blood thinners and wearing support stockings may be advised until you are able to walk independently. Being immobile also increases the chances of developing bedsores (pressure sores). Frequent repositioning may aid in avoiding this issue.[2]

Pathogenesis

GBS is thought to be an immune-mediated demyelinating neuropathy with an acute onset in the majority of cases. Approximately two-thirds of cases are preceded by an acute influenza-like illness from which the patient has recovered by the time the neuropathy manifests. GBS is linked to infections with *C. jejuni*, *Cytomegalovirus*, *Epstein–Barr virus*, and *Mycoplasma pneumoniae*, as well as prior vaccination.[25]

There has been no evidence of an infectious agent in the affected nerves, so an immunologic reaction is the most likely cause. By immunizing experimental animals with a peripheral nerve myelin protein, a similar inflammatory disease of the peripheral nerves can be reproduced.[26]

Activated macrophages cause segmental demyelination, which triggers a T-cell-mediated immune response. When these T-cells are transferred to a naive animal, the lesions are similar. Furthermore, lymphocytes from people with GBS have been shown to cause demyelination in myelinated nerve fiber tissue cultures. Antibodies in the bloodstream that reacts with peripheral nerve components could also play a role.[27]

Signs and Symptoms

GBS can be triggered by a variety of infections, the most common of which is a respiratory infection that causes cold or influenza-like symptoms such as fever, runny nose, cough, and generalized aches and pains; pinprickling or needle sensations in distal areas such as the fingers, toes, and ankles; weakness in the lower extremities that spreads to the upper body; inability to walk or climb stairs, as well as difficulties with facial movements, speaking, chewing, and swallowing; dual vision and erroneous eye movement; severe pain that may be achy, shooting, or cramping in nature, and that is worse at night; difficulties with bladder or bowel function; tachycardia; low or high blood pressure; and difficulty in breathing.[17]

Some of them are discussed in detail:

Muscle Deficiency

Damage to the motor nerves that connect the brain to the muscles cause weakness, which is usually the most prominent clinical feature of GBS. Weakness usually starts in the legs and is symmetrical, affecting both sides of the body equally, though there may be some differences from side to side.[28]

A stroke, on the other hand, usually affects only one side of the body, resulting in weakness.[29] The distal muscles (the parts of the limbs farthest from the spinal cord) are the most affected in some people.[30] The muscles closest to the spinal cord (proximal muscles) are weaker in others.[31] GBS affects roughly half of the population, causing weakness in both the proximal and distal muscles. Their thighs, legs, ankles, and feet all weaken at the same time, causing their legs to become limp and flaccid.[32]

Abnormal Sensations

Although weakness is usually the most noticeable symptom of GBS, abnormal sensations are frequently the first symptoms, appearing

hours to days before weakness.[33] Sensory symptoms affect 50–70% of GBS patients, and they can take many different forms.[34] Paresthesias are a type of abnormal sensation. They are referred to as dysesthesias if they are particularly unpleasant.[35] Paresthesias include sensations such as pins and needles or tingling. Numbness or loss of sensation is also common complaints, but they are less common.[36] The toes, feet, or fingers are usually the parts of the limbs farthest from the spinal cord that experiences these sensations.[37]

Pain

GBS is a condition, in which the paralysis is so severe that it overshadows all other symptoms.[38] Pain is an unavoidable part of the disease, but it is not always given the attention it deserves.[39] Pain has been reported in more than 80% of people with GBS in some studies.[40]

Doctors frequently overlook and undertreat it, particularly in the early stages of awareness, but it can be detected through a neurologic examination. In rare cases, there may be a loss of sensation or an abnormal sensation with little or no weakness.[41]

Diagnosis

The clinical features of acutely evolving weakness and loss of reflexes following an antecedent illness, such as an upper respiratory tract infection or diarrhea, are used to diagnose GBS.[42]

Electrophysiologic studies and cerebrospinal fluid examinations, which are obtained through spinal tap, are the most commonly used diagnostic tests (lumbar puncture). The results of these tests are usually positive, confirming the clinical suspicion. If the diagnosis is still in doubt, additional tests may be required to rule out the possibility of another disease masquerading as GBS.[43]

GBS and its variants are diagnosed using electrodiagnostic testing of nerves and muscles. Furthermore, the tests can provide a prognosis estimate. The doctor can usually make a working diagnosis of GBS based on the patient's history and physical findings. A definitive diagnosis, on the other hand, is required for treatment planning, prognosis, and proper treatment. Given the risk of respiratory failure and the possibility of hastening recovery with appropriate immune-modulating therapies, it is critical to get a correct diagnosis as soon as possible so that treatment can begin in a timely and appropriate manner.[44]

Obviously, a person suffers from tick paralysis, arsenic poisoning, or another disorder that resembles GBS should not be treated as if they have the disease. A doctor, on the other hand, would not want to manage a person with GBS anywhere other than an intensive care unit.[45]

Nerve function testing can be performed in a variety of settings, including the emergency room and the intensive care unit, but it is most commonly done in a specialized laboratory. As soon as the test is finished, the results are available. The results of the electromyographic and nerve conduction studies can confirm that GBS is the correct diagnosis and rule out other causes of acutely developing weakness. Testing procedures available till date are safe and non-invasive. As a result, when a person is suspected of having GBS, electrical testing is the next step in the diagnostic process.[46]

Therapeutic Strategy

GBS is treated with a multi-disciplinary approach that includes both general medical treatment and immunological therapies. To avoid or manage complications, respiratory, cardiac, and hemodynamic function must be monitored, as well as prophylaxis for deep vein thrombosis, management of possible bladder and bowel dysfunction, early initiation of physiotherapy and rehabilitation, and psychosocial support. Furthermore, pain control with opioids or non-steroidal anti-inflammatory drugs is critical.[47]

Applicable treatments:

- Plasma exchange therapy (plasmapheresis): The liquid portion of the blood (plasma) is extracted and separated from the blood cells. The blood cells are then reintroduced into the body of the donor, which produces more plasma to compensate for what was lost. Plasmapheresis may work by removing antibodies from plasma that contributes to the immune system's attack on peripheral nerves.[48]
- Immunoglobulin therapy: A vein is used to administer immunoglobulin containing healthy antibodies from blood donors (intravenously). Immunoglobulin at high doses can block the damaging antibodies that may contribute to GBS.[49]

The GBS and Life After It

Patients who do not fully recover may experience pain, neuropathic pain, overuse pain, fatigue, emotional issues, and abnormal sensations. Pain is an underappreciated symptom of GBS that can last for years. It could be neuropathic pain or pain caused by overuse of muscles that have not fully recovered. During the paralytic stage of the illness, pain is also present.[50]

Pain is a vital defense mechanism that protects us from harming ourselves. When you put your hand in a flame, skin receptors detect the heat and quickly send the information to the brain, allowing the hand to be withdrawn before serious injury occurs. Nociceptive pain is the term for this type of physiologic pain. However, any neuropathic disease, including GBS, can damage the nerve fibers that transmit these messages in a normal situation, resulting in neuropathic pain.[51]

Persistent residual fatigue is one of the most common complaints of GBS patients following an acute paralytic illness, but it has received very little research attention; most early studies on the ultimate outcome of GBS do not even mention it.[52] Many of these studies are based on phone interviews or chart reviews conducted long after the patient has been discharged from the neurologist's care, and minor complaints may have been overlooked.[53]

Patients who are suffering from the aftereffects of the acute illness, such as weakness, pain, and fatigue, may also develop depression. Despite constant assurances that the disease almost never returns, anxiety about a possible recurrence is common. These concerns are shared by the patient and his or her entire family. Financial difficulties may have arisen as a result of the illness and may last for years, particularly if the patient has been unable to return to work.[54]

As they recover from GBS paralysis and become more active, some patients notice that continued activity, such as walking, causes abnormal sensations, such as tingling in the toes or fingers, and that continued activity causes fatigue and even collapse.[55] These unusual sensations serve as an early warning

sign of impending exhaustion. This type of warning should be used by the patient as a cue to sit down and rest. The tingling and fatigue usually go away with time.[56]

Conclusion

GBS is an immunocompromised pathological state, in which peripheral nerves are mainly affected. It happens rarely around the globe by a rate of about 0.1%. A mix infection of virus and bacteria in the upper respiratory tract are involved. GBS is an immunological issue therefore; immunoglobulin therapy, as well as medicines for symptomatic relief is advised for effective treatment.

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