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Review Article

Guillain-Barre Syndrome and Its Association with Viral Infections

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ABSTRACT

Guillain Barré syndrome is one of the best examples of a post infectious immune disease and offers insights into the mechanism of tissue damage in other more common autoimmune diseases. Controlled epidemiological studies have linked it to infection with Campylobacter jejunal in addition to other viruses including cytomegalovirus and Epstein Barr virus. The syndrome includes several pathological subtypes, of which the most common is a multifocal demyelinating disorder of the peripheral nerves in close association with macrophages. Evidence from histological examination of peripheral nerve biopsy and post-mortem samples suggests that both cell mediated and humoral mechanisms are involved in the pathogenesis. Immunological studies suggest that at least one third of patients have antibodies nerve gangliosides, which in some cases also react with constituents of the liposaccharide of C jejunal. In the Miller Fisher variant of the disease, these antiganglioside antibodies have been shown to produce neuromuscular block, and may in part explain the clinical signs of that disorder. Treatment with both intravenous immunoglobulin and plasma exchange reduces the time taken for recovery to occur, although mortality remains around 8%, with about 20% of patients remaining disabled.

INTRODUCTION

Guillain-Barré Syndrome (GBS) is a major cause of sudden paralysis. It often shows signs like fibbers in the peripheral nervous system (PNS) [1,2]. The incidence of GBS varies between 0.81 and 1.89 cases per 100,000 people each year, with a median of 1.11 cases. Recent data suggests that GBS is becoming more common [3].

GBS usually happens after an infection when the immune system attacks the nerves. About two-thirds of patients have stomach or breathing symptoms before GBS starts. A bacteria called Campylobacter jejunal is the most commonly known trigger, causing about 1 in every 1000 GBS cases [4]. Genetics may also play a role in GBS, but specific genetic risk factors are not yet clearly identified [5]. Overall, GBS seems to be

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influenced by both genetic and environmental factors.

Recent studies show that some viruses, such as COVID-19 (caused by SARS-CoV-2) and Varicella-zoster virus (VZV), might also be linked to GBS. However, the research results are mixed. Some meta-analyses found a connection between COVID-19 and GBS [6,7], but other studies did not confirm this [8]. Research also shows that COVID-19 patients have a higher risk of neurological and psychiatric problems compared to patients with influenza or other respiratory illnesses. People with pre-existing neurological conditions also have a higher risk of dying from COVID-19 than healthy people [9].

VZV causes chickenpox in the first infection, then stays inactive in nerve cells and can reactivate later as herpes zoster (shingles) [10]. Although there are many reports of GBS following VZV reactivation [11], it is believed that GBS rarely happens after shingles [12]. Because both GBS and shingles are common, it is hard to tell whether the relationship is real or just a coincidence.

Overall, the link between viral infections and GBS is still unclear. Observational studies are often affected by confounding factors and reverse causality, making it hard to prove a direct cause-and-effect relationship. Therefore, a strong, well-designed study is urgently needed to clarify if certain viral infections truly cause GBS.

Genome-wide association studies (GWAS) help find genetic factors linked to diseases. Mendelian randomization (MR), which uses GWAS data, is a powerful method to study causality [13]. MR minimizes confounding by using genetic information fixed at birth, avoiding some of the problems seen in observational studies [14].

In this study, we used two-sample MR to investigate whether genetic susceptibility to infections by different viruses—including HIV, SARS-CoV-2 (COVID-19), varicella-zoster virus (VZV), herpes simplex virus (HSV), Epstein-Barr virus (EBV), hepatitis B virus (HBV), and influenza virus—is linked to the risk of developing GBS.

Methods:

Exposure GWAS dataset in this research, the following exposure variables and dataset had been meticulously curated to delineate us.

Case studies:

exposure was explored using the fnn-b_AB1_HIV dataset from the Finden database [15], which included 357 cases, 218,435 controls, and a comprehensive set of 16,380,466 SNPs.COVID-19 was examined through the ebi-a-GCST011081 dataset provided by the COVID-19 Host Genetics Initiative [16], comprising 9986 cases, 1,877,672 controls, and 8,107,040 SNPs, sourced from Gavriella (VZV) was investigated with the fnn-b-AB1_VARICELLA dataset from the latest Finsen, encompassing 710 cases, 211,856 controls, and 16,380,433 Spheres' zoster (VZV) was studied using the ebi-a-GCST90018941 dataset from GWAS, which contained 522 cases, 351,740 controls, and 19,078,292 Sep.'s was analysed with the fnn-b_AB1_HER-PES_SIMPLEX dataset from the Finnigan database, consisting of 1,595 cases, 211,856 controls, and 16,380,457 Sops've was assessed using the fnn-b-AB1_EBV data-set from Finnegan (release 9), which included 1,238 cases, 213,666 controls, and 16,380,461 Snaps' was investigated with the ebi-a-GCST90018804 dataset from GWAS, containing 145 cases, 351,740 controls, and 19,079,722 SNPs [17].Outcome GWAS dataset data for GBS as an outcome were sourced from the Finnish database,

which includes 213 cases and 215,718 controls, with a total of 16,380,463 SNPs, all from individuals of European descent. Instrument identification Instrumental variables need to satisfy the following three assumptions: SNPs must be strongly associated with the exposure factor (objective criterion: p<5×10^-8), independent of confounding factors, and not directly associated with the outcome [18]. Given that only a small proportion of the SNPs for the exposure factor under study meet the condition of strong association with the outcome, we had adjusted the p-value to p<5×10^-6. Meanwhile, to avoid instruments with linkage disequilibrium and to exclude non-random associations between certain genes and specific traits, we set the parameters to 2=0.001 and kb=10,000.

We further refined our analysis by computing the variance and employing F-statistics to evaluate the robustness of the genetic instrument utilized in our study. Instruments with an F-statistic greater than 10 were defined as strong instruments [19]. Finally, we used the of the National Institutes of Health to exclude confounders-related SNPs and determine the final instrumental variables. Two-sample MR analysis In the R computing environment, we performed MR analysis employing the Townscaper package, which coordinated and integrated the exposure and outcome datasets. The analysis employed five methods of two-sample MR analysis: MR-Egger regression [20], weighted median estimator [21], Inverse Variance Weighted (IVW), weighted mode [22], and Simple mode. Previous studies had shown that the IVW method was not affected by horizontal pleiotropy, which in turn minimizes the impact of confounding factors and providing unbiased estimates [23]. As a result, we primarily relied on the IVW method to determine positive outcomes, while utilizing other methods for supplementary validation. Ten evaluated the effect size using the β value, odds ratio (OR), and 95% confidence interval (CI). second step was the outlier test. We applied MR-PRESSO to detect any outliers that may indicate pleiotropic bias in the reported results [24]. If outliers are present, they needed to be manually removed and then MR analysis should be conducted again. third step was sensitivity analysis, which aimed to test whether the results of MR analysis were reliable, mainly including heterogeneity test (Cochran's Q test) ($p < 0.05$ indicates heterogeneity), pleiotropy test (Egger Intercept test) ($p < 0.05$, indicating that there is pleiotropy in the data), single SNP test and retention one method analysis. Finally, we conducted the MR-Steiger directional test to determine whether there is a reverse causal relationship between the exposure and the outcome. To above steps about the two sample MR are detailed in flow- chart below (Fig. 1).

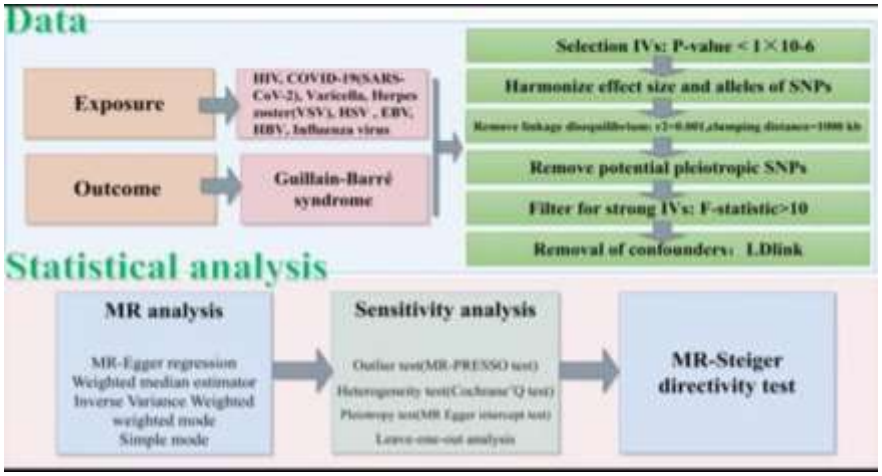
RESULT:

Our study identified a significant causal link between HIV infection and the occurrence of GBS, indicating that HIV increases the risk of GBS (IVW: p=0.010, OR [95% CI] 1.240 [1.052–1.463]). However, no causal relationship was found between GBS and COVID-19 (IVW: p=0.275, OR[95% CI] 0.831[0.596–1.159]), varicella (IVW: p=0.543, OR [95% CI] 0.919 [0.701–1.206]), herpes zoster (IVW: p=0.563, OR [95% CI] 0.941 [0.766–1.156]), herpes simplex (IVW: p=0.280, OR [95% CI] 1.244 [0.837–1.851]), Epstein-Barr virus (EBV) (IVW: p=0.218, OR [95% CI] 0.883 [0.724–1.076]), hepatitis B virus (HBV) (IVW: p=0.179, OR [95% CI] 1.072 [0.969–1.187]), or influenza virus (IVW: p=0.917, OR [95% CI] 0.971 [0.553–1.703]). The results of MR Egger, Weighted Median, Simple Mode, and Weighted Mode analyses are consistent with the directionality of IVW, all indicating that there is no causal relationship with GBS (Figs. 2, 3). For

HIV, the MR-PRESSO analysis did not identify any potential SNP outliers. At this point, MR Egger's p (Q-statistic) =0.190, and IVW's p (Q-statistic) =0.253, indicating a lack of heterogeneity. Furthermore, the MR-Egger analysis showed no evidence of horizontal pleiotropy (intercept=− 0.045, p=0.715). The leave-one-out analysis reinforced the robustness of the results, as all SNP p-values were found to be greater than 0, indicating that the causal relationship between HIV and GBS is deemed reliable. For COVID-19, varicella, herpes zoster, HSV, EBV, HBV, and influenza virus, no abnormal SNPs were identified, and there was no indication of heterogeneity or horizontal pleiotropy (Table 1). All of these have confirmed the absence of a causal relationship. Meanwhile, the results of the MR-Steger directional test indicate that there is no reverse causal relationship between all viral infections and GBS. (Table 2)

Discussion in Simple Words:

Guillain-Barré syndrome (GBS) is usually seen as an autoimmune disease that randomly affects the nerves, often after an infection. However, a few cases in families have been found, suggesting that genes might also play a role along with infections in causing GBS. [25,26]



In our study, we found a strong causal link between HIV and GBS, but we did not find such a link with other viruses. To our knowledge, this is the first study using a method called Mendelian Randomization (MR) to prove that a virus (HIV) can directly cause GBS. This method is better than older studies because it reduces errors like confusing cause and effect or other hidden factors.

In GBS, the immune system mistakenly attacks the body's own nerves because it can't tell the difference between "self" and "foreign" anymore.[27] HIV quickly spreads to the central nervous system (CNS) after infection. HIV might cause nerve damage through immune cells like macrophages or T lymphocytes that attack the nerves [28]. Also, HIV may trigger the body to make harmful autoantibodies through a process called molecular mimicry [29,30] Additionally, HIV infection can cause neutrophils (a type of immune cell) to release traps (NETs) and activate immune sensors called TLR7 and TLR9[31,32].

Previous research has already suggested that HIV can drive GBS, and how it shows up depends on the person's own risk factors, how strong their immune system reacts to HIV treatments, and the type of HIV [33]. In our study, the MR analysis showed a clear link between HIV and GBS with one main method (IVW with P<0.05), and all five

methods we used pointed in the same direction. This suggests that genes making a person more likely to get HIV also make them more likely to develop GBS. Even though we still don't fully understand the genetic causes of GBS, our findings show that genes related to HIV infection need to be studied more [34].

For patients suspected of having GBS, doctors should test for HIV early because HIV treatment can help slow down nerve damage [35,37].

During the COVID-19 pandemic, many reports suggested that COVID-19 might cause GBS. However, big studies looking backward (retrospective) and forward (prospective) did not find a strong causal link between COVID-19 and GBS. In our study, MR analysis also showed that COVID-19 infection does **not** increase the risk of GBS (P=0.275; OR 0.831 [0.596–1.159]), supporting the idea that COVID-19 does not cause GBS [38].

Even though some reports linked COVID-19 to GBS through the production of harmful antibodies or confusion of the immune system (molecular mimicry), the overall evidence does not support a real cause-effect relationship.

Similarly, some viruses like Varicella-Zoster Virus (VZV), Herpes Simplex Virus (HSV), Epstein-Barr Virus (EBV), Hepatitis B Virus (HBV).

The Eponym and 100 Years of Clinical Advances:

George Guillain (pronounced "ghee-lain") was a student of Pierre Marie, who had an important position at the famous Salpêtrière hospital in France [39]. Guillain was one of Marie's best students and later took over his role in 1925.

In 1916, during World War I, George Guillain, Jean-Alexandre Barré, and André Stroh treated two soldiers at an army hospital during the Battle of the Somme in northern France. These soldiers had sudden and severe weakness in their arms and legs. The doctors noticed two important things:

- The soldiers' spinal fluid had high protein levels without signs of infection, which had never been seen before in cases of sudden paralysis.
- Both soldiers completely recovered.

At first, the doctors thought it might be poisoning, syphilis affecting the nerves, or trench fever (a disease common among soldiers).

Guillain and Barré (but not Stroh) quickly started calling the illness "our syndrome." Later, people also included Landry's name because, years earlier, Landry had described a similar but often deadly illness where weakness started in the limbs and moved upward, even affecting breathing and facial muscles [40]. Patients usually had fever and severe pain before becoming weak.

Back then, Landry's treatments included unusual methods like breathing chloroform, taking opium, using electrical stimulation, and even eating pork chops and drinking warm Bordeaux wine!

It's very possible that this disease had been seen even earlier. Historical records show similar cases described by François Comely in 1828, James War drop in 1834, Louis-Stanislaw Domenic in 1864, Robert Graves in 1884, and even William Osler in 1892 (though Osler's case might have been a different illness) [41].

Interestingly, just one month after Guillain, Barré, and Stroh published their findings, another report came out by Pierre Marie and Charles Chatelaine.

They described three soldiers with the same symptoms. In a small note, they admitted they only found out about Guillain and Barre's work after writing their own report.

When Guillain, Barré, and Stroh presented their findings to the Society of Neurology, some people wondered: could the disease have been named after Marie and Chatelaine instead? After all, they also reported similar cases.

It's a bit strange that Guillain and Barre's 1916 paper — which wasn't even considered very special at the time — ended up creating such a famous medical name. Stroh's name was left out, and no one knows exactly why, though it may be because he had a different specialty and wasn't as involved when people started debating the signs of the illness.

Over the last 100 years, there have been many important discoveries about Guillain-Barré Syndrome (GBS). Doctors started using another name too: acute inflammatory demyelinating polyradiculoneuropathy [42] (AIDP), which describes how the nerves are damaged.

GBS happens all around the world, and many important studies have helped us understand it better:

- In 1949, Haymaker and Kernohan studied 50 people who died from GBS and found that the disease damages nerves through an immune attack.
- In 1964, Wiederholt, Mulder, and Lambert showed that GBS mostly damages the nerve coverings (myelin) and that steroids didn't help much.
- In 1976, Kurland and others found a link between a swine flu vaccine and an outbreak of GBS.

For many years, GBS was thought to only involve nerve covering damage. But in 1986, in Canada, Feasibly [44] and his team discovered a form that attacks the nerve fibbers themselves (axonal type). Then in 1990, doctors from Johns Hopkins University, University of Pennsylvania, and Hebei Medical University in China found that a bacteria called Campylobacter jejunal could trigger the axonal type of GBS [43].

Also in 1990, Japanese researcher Yuki and his team found special antibodies that attack nerve parts, showing a process called "molecular mimicry" — when the immune system mistakes nerves for bacteria and attacks them.

Big treatment breakthroughs also happened:

- In 1985, a North American study showed that plasma exchange (removing bad antibodies from blood) helped GBS patients recover faster.
- In 1992[46], a Dutch study showed that intravenous immunoglobulin (IVIG) — giving patients helpful antibodies through a drip — also worked really well.
- In 1997[47], a major study showed that combining plasma exchange and IVIG didn't work better than using just one of them. This led the American Academy of Neurology to recommend using either plasma exchange or IVIG as treatment [48].

PATHOGENESIS:

Multiple antecedents and potentially triggering events have been reported [49]. The association with infections is established in not only C jejunal but also cytomegalovirus, Epstein-Barr virus, influenza A, Mycoplasma pneumoniae, Haemophilus influenzae, hepatitis (A, B, and E). and Zika virus. [50,51] The risk of GBS from influenza vaccine varies from 3 cases per million

to as low as zero. [52,53] surgery may predispose patients to GBS (more likely in patients with prior malignant or autoimmune disorders) but is exceedingly rare in our experience.[54] Guillain-Barré syndrome is often a post infectious, immune-mediated nerve injury. Three phenotypes are likely purely demyelinating, purely axonal, and demyelinating with axonal involvement. Immunopathogenesis differs in each of these conceptual models, and outcome (possibly a response to treatment) is also different. The current working hypothesis of GBS immunopathogenesis is depicted in Figure 2. Although both elements of the immune response (T cells and B cells) play a role, current understanding holds that GBS is antibody mediated. Not all antiganglioside antibodies are neurotoxic, but antibodies binding to GM1 or GD1a gangliosides (at nodes of Ranvier) activate myelin-destroying complement.[55] The predominance of motor axonal involvement has led to the designation acute motor axonal neuropathy. Campylobacter jejunal infection is the main known instigator of this mechanism, and molecular mimicry between C jejunal lip oligosaccharide and GM1 and GD1a has been found. [56,57] For patients presenting with the ataxic sensory variant, the most commonly identified ganglioside antigen.

In one type of Guillain-Barré Syndrome (GBS), the body attacks a molecule called GQ1b [58]. Another type, called acute motor-sensory axonal neuropathy, is grouped under a broader name, axonal GBS.

For the regular demyelinating form (where the nerve covering is damaged), doctors still don't know exactly what molecule the immune system attacks.

Even though there are different types, the treatment is usually the same. However, the axonal type tends to be worse, with slower recovery and more long-term disability.

Clinical Presentations

Guillain-Barré Syndrome (GBS) usually has a clear pattern of symptoms that worsen over 1 to 2 weeks. People often first feel severe back pain and tingling in their hands and feet, with a feeling like a tight band around the body. These sensations spread upward. Although tingling is common, the ability to feel things stays mostly normal or is only slightly reduced. Muscle weakness usually starts in muscles closer to the canter of the body, making it hard to climb stairs or get up from a chair. Weakness becomes noticeable 1 or 2 days after tingling begins, and reflexes like the knee-jerk reflex are often reduced or absent [60,61].

The legs are more affected than the arms, creating the appearance of paralysis moving upward. In about half of patients, face and throat muscles are also affected, sometimes as the first sign. Breathing can become shallow, with short, broken sentences when talking. There are special forms of GBS, like Miller Fisher syndrome (affecting the eyes and balance), and other rare types that may first show different symptoms but later become more typical [63]. One of the most serious problems in GBS is weakness of the breathing muscles, which can cause respiratory failure. This is sometimes missed because early signs can be subtle, and some patients may suddenly stop breathing even if they seemed stable. Normally, breathing is mainly controlled by the diaphragm muscle, supported by chest and neck muscles. In GBS, weak breathing muscles cause shallow breaths, poor oxygen exchange, faster breathing, and eventually a buildup of carbon dioxide in the blood. In severe cases, a "rocking horse" movement of the chest and abdomen called paradoxical breathing may be seen [64]

Abnormal mechanics of breathing:

Normal breathing mostly happens because the diaphragm [65,66], a large muscle under the lungs, contracts. The diaphragm does about two-thirds of the work needed to breathe in, with help from other muscles like the external intercostals, scalene, and sternocleidomastoids. Breathing out usually happens naturally as the chest relaxes, but the abdominal muscles help when a strong breath out or a cough is needed. Air moves into the lungs by overcoming a "respiratory load," which includes the resistance of the airways, chest wall, lungs, and pressure at the end of breathing out. When breathing muscles are weak, they can't fully overcome this load, leading to reduced airflow and parts of the lungs collapsing. This results in shallow breathing and poor oxygen exchange [67].

Dysautonomia Patterns

Another manifestation of a developing neurocritical illness is dysautonomia, recognized by extreme blood pressure fluctuation and exaggerated drug responses, cardiac arrhythmias, hypersecretions, gastrointestinal tract dysfunction, and bladder dysfunction. [68,69] Baroreflex abnormalities altered function due to vagal nerve demyelination may cause this blood pressure fluctuation [70].

FUTURE DIRECTION AND IMMEDIATE:

Although GBS was initially described 100 years ago, its recent occurrence in association with the Zika virus emphasizes the importance of ongoing study of this disorder.[71] In those affected, the most common presentation was that of acute motor axonal neuropathy, with 19% having novel autoantibodies to the glycolipid GA1. The virus is spread by mosquito-borne infection and sexual transmission and will most likely spread to the Americas. Epidemiological research to identify and control outbreaks continues to be important. Additionally, many areas with a higher infection risk also have poor sanitation and limited access to modern normotensive management. Lastly, we need to translate increased understanding of pathogenesis to meaningful treatment or prevention. Immunotherapeutic trials of blocking the complement cascade (i.e., eculizumab [72,73], monoclonal antibodies binding to complement factor 5) are under way.

CONCLUSION:

Guillain-Barré syndrome is a well-recognized, acute, disabling neurologic illness. Management is supportive with immunomodulating therapy but may involve prolonged intensive care and long-term neurorehabilitation. Without a full understanding of its pathophysiology, it will be difficult to find a treatment that dramatically hastens the slow recovery trajectory. Guillain-Barré syndrome can profoundly affect a patient's life and family because it may take years to resolve completely. Patients with GBS struggle and strive while undergoing normotensive care and long-term rehabilitation, but the outcome is good for many

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