SAGE Open Medicine
Volume 12: 1–8

DOI: 10.1177/20503121241239538
journals.sagepub.com/home/smo
Sage

An updated review on Guillain-Barré syndrome: Challenges in infection prevention and control in low- and middle-income countries

Sakif Ahamed Khan, Proma Rani Das, Zabun Nahar and Syed Masudur Rahman Dewan

Abstract

Guillain-Barré syndrome is a rare condition that can be potentially life-threatening. Guillain-Barré syndrome does not have a definitive etiological agent. It is a syndrome that can arise from multiple factors, including various infectious diseases and immunizations. The severity of Guillain-Barré syndrome is exacerbated by these variables, especially in low-income and middle-income countries where healthcare systems are already constrained and struggle to meet the demands of other diseases. The primary aim of our article is to comprehensively examine the life-threatening nature and intensity of Guillain-Barré syndrome by assessing its etiology, progression, and prevalence in low- and middle-income nations while also considering global trends. Furthermore, we proposed the implementation of standard and efficacious treatment and diagnostic resources that are readily accessible and successful in affluent nations and should also be readily accessible in impoverished nations without any unnecessary delay. Our study also emphasized the epidemiological data with molecular epidemiological analysis and the utilization of artificial technology in low- and middle-income nations. The goal was to decrease the incidence of Guillain-Barré syndrome cases and facilitate early detection.

Keywords

Guillain-Barré syndrome, infectious disease, low- and middle-income countries, disease management

Date received: 16 December 2023; accepted: 28 February 2024

Background

Guillain-Barré syndrome (GBS) is an inflammatory polyneuropathy distinguished by a gradual onset of symmetrical, flaccid hypoxia in the ascending extremities, accompanied by motor symptoms with or without sensory manifestations.[1] It is a severe inflammatory peripheral radiculopathy and neuropathy with a reported global incidence of about 100,000 new cases per year. It is characterized by progressive limb weakness, sensory deficits, cranial nerve involvement, tendon areflexia, and cerebrospinal fluid (CSF) albuminocytological dissociation.[2] There are different forms of GBS as the most prevalent type in the United States and Europe (85%–90%) is acute inflammatory demyelinating polyneuropathy; 5% of cases in the United States; and 25% in Japan involve Miller Fisher syndrome; although China, Japan, and Mexico often see cases of acute motor axonal neuropathy and acute sensorimotor axonal neuropathy, the United States only sees between 5% and 10% of these cases; acute pandysautonomia is one of the known rare GBS variations.[3] Currently, GBS is not adequately documented in low- and middle-income countries (LMICs). However, the existing scant evidence indicates that GBS exhibits a more severe clinical progression in LMICs and that patients in these countries experience worsening conditions compared to those in high-income countries (HICs). This article focuses on the severity and clinical aspects of GBS in LMICs, as well as the challenges

Department of Pharmacy, School of Medicine, University of Asia Pacific, Dhaka, Bangladesh

Corresponding author:
Syed Masudur Rahman Dewan, Department of Pharmacy, School of Medicine, University of Asia Pacific, 74/A Green Road, Dhaka 1205, Bangladesh.
Email: gobeshok.d@gmail.com

faced in its management, approaches to treatment, and preventive strategies aimed at reducing potential risk factors.

Etiologies and development of GBS: Conventional and updates

GBS is a syndrome that can arise from a range of circumstances, including numerous infectious diseases and vaccinations, among others. The exact cause and progression of GBS have not yet been conclusively established. This complicates the process of determining the exact mechanisms or pathways of development. Nevertheless, certain likely reasons and mechanisms have been identified and will be further elucidated in this discussion.

The pathogen most frequently responsible for the initial infection that leads to GBS is *Campylobacter jejuni* (*C. jejuni*).[4] *C. jejuni* is most commonly spread to people through their diet, specifically by the ingestion of raw or undercooked poultry meat and fish, unpasteurized milk, or water contaminated with the bacteria.[5] The prevalence of *C. jejuni* infection is significantly higher in individuals diagnosed with GBS from Curaçao, China, and Bangladesh, with rates ranging from around 60% to 70%, compared to patients from all other countries, where the prevalence ranges from 30% to 32%.[6,7] The elevated incidence of *C. jejuni* infection in various geographical areas can potentially be attributed to factors such as the quality of their sanitation systems, environmental conditions, and host-related variables, which may encompass dietary practices.[8] A 45-year-old man who acquired GBS with irreparable neurological impairment 2 weeks after contracting *C. jejuni*-associated gastroenteritis was the first to demonstrate the correlation between *C. jejuni* infection and the onset of GBS in 1982.[9]

As previously stated, GBS is an immune-mediated, post-infectious disorder; it is not known whether a specific disease-causing agent is responsible for GBS; therefore, the condition is referred to as a syndrome rather than a disease. Immune systems that are cellular and humoral are likely involved in the development of GBS. The majority of patients claim to have had an infectious disease in the weeks before developing GBS. Numerous infectious agents have been identified, and it is believed that many of these agents cause the body to produce antibodies against particular gangliosides and glycolipids found in the peripheral nerve system myelin, including GM1 and GD1b.[10] Molecular mimicry is the most widely accepted theory for how autoimmune diseases arise. Antibodies are generated in response to *C. jejuni* infection, triggering the complement system and ultimately resulting in phagocytosis of the bacteria. However, in very uncommon events, antibodies generated against particular *C. jejuni* antigens will also bind to gangliosides of nervous tissue, leading to complement activation and destruction by phagocytes. Damage to the peripheral nervous system causes demyelination and axonal damage.[11]

Antibodies targeting ganglioside GM1 have been identified in 20%–71% of patients with *C. jejuni*-associated GBS, a significantly higher occurrence compared to other GBS cases. The probable cause of this discovery is the occurrence of molecular mimicry between ganglioside GM1 and the lipopolysaccharide of *C. jejuni*.[12] Another instance is the Miller-Fischer syndrome variant's target, the GQ1b ganglioside. It is believed that the presence of particular antigens in *C. jejuni's* capsule that are related to nerves is what gives the organism its pathogenicity. Antibodies produced by immune reactions against the capsular elements interact with myelin to trigger demyelination. The peripheral nervous system appears to be immunologically damaged by ganglioside GM1, which appears to cross-react with *C. jejuni* lipopolysaccharide antigens.[13] The final outcome of autoimmune attacks is muscle paralysis along with possible sensory or autonomic abnormalities when the immune system incorrectly targets the peripheral nervous system. Autoimmune attacks can also produce myelin inflammation and conduction blockage.[14]

The Vaccine Adverse Event Reporting System defines vaccine-associated GBS as the emergence of GBS symptoms within 6 weeks of vaccination.[15] According to certain studies, vaccinations such as the meningococcal vaccine, poliovirus vaccine, flu vaccine, and rabies vaccine are capable of triggering GBS.[16] A girl aged 15 years reported lower limb paralysis 24 days after getting an anti-rabies vaccination made from neural tissue from sheep brains. This case was observed in a tertiary care hospital in Pakistan.[17] A study revealed that the monovalent inactivated influenza A (H1N1) 2009 vaccine had been associated with a slight spike in the incidence of GBS. According to this finding, there were 1.6 more instances of GBS per million Americans who received vaccinations.[18] A 13-year-old female patient who had received the first dose of the recombinant protein subunit COVID-19 vaccination (Corbevax, BECOV2D) 4 days prior reported to the emergency room of a tertiary care academic center in north India with bilateral upper limb and lower limb paralysis; the patient had GBS, according to the diagnostic results and clinical symptoms.[19] The precise mechanism of GBS after receiving the COVID-19 vaccine has not been determined yet. There may be a molecular mimicry mechanism functioning in COVID-19 vaccine-associated GBS[20] which needs to be investigated with importance.

GBS prevalence

To fully comprehend the severity of the GBS syndrome, it is essential to meticulously analyze and evaluate the prevalence data regarding GBS throughout various countries globally. This can provide us with crucial insights into the potentially serious consequences of the condition. GBS exhibits variations in its epidemiology, clinical features, management, and outcome across LMICs and HICs. Currently, there is a scarcity of data regarding GBS in

LMICs, and the actual occurrence of GBS in many LMICs is still uncertain due to insufficient healthcare infrastructure.[21]

The global incidence of GBS in 2019 amounted to 150,095 cases, leading to a total of 44,407 years lived with disability.[22] The median incidence rate of GBS in western populations residing in high-income countries has been estimated to be 1.10 per 100,000 person-years in the United States,[23] 1.6 in Canada,[24] 1.33 in the United Kingdom,[25] and 1.59 in Denmark.[26]

Due to an unprecedented rise in GBS patients in several parts of the nation, the National Center for Epidemiology, Prevention, and Disease Control (CDC) of Peru issued an epidemiological notice in June 2023. Between epidemiological weeks 1 and 28 (January–July 2023), a total of 231 suspected instances of GBS had been recorded in Peru, an upper-middle-income country in Latin America.[27] Notably, the period spanning epidemiological weeks 23 (June 2023) to 28 (July 2023) accounted for 56% of the reported cases, amounting to 130 cases. In the year 2019, Peru experienced an unexpected epidemic of GBS, which subsequently disseminated over several regions of the country, leading to the confirmation of approximately 700 recorded cases.[27]

The disease is most commonly found in Bangladesh, a lower-middle-income country, with a rate of 2.5 cases per 100,000 person-years in adults and 3.25 cases per 100,000 person-years in pediatric patients. It is also prevalent in Latin America, with a rate of 2.12 cases per 100,000 person-years, and in North America and Europe, with rates ranging from 0.81 to 1.91 cases per 100,000 person-years. In East Asia, the rate is between 0.44 and 0.67 cases per 100,000 person-years. The prevalence of the condition seems to rise by 20% for every 10-year increment in age. In contrast to other autoimmune disorders, males are more frequently impacted. Despite the existing immunotherapies, the death rate remains approximately 5%, and up to 20% of individuals are unable to walk unassisted 1 year after the disease begins.[28]

Globally, most studies report a 2%–10% death rate with GBS, while regional variations are noteworthy. As an example, recorded mortality rates are 2%–7% in North America and Europe, 13% in Hong Kong, 14%–17% in Bangladesh, and 16% in Egypt.[21] A Taiwanese cohort found 5469 cases of GBS in Asian nations, with a crude incidence of 1.71 per 100,000 person-years and an in-patient mortality rate of 1.61% (88/5,469).[29] In Korea, the incidence rate grew from 1.28 to 1.82 per 100,000 people between 2010 and 2016.[30] The analysis of prevalence data allows for an examination of the severity of GBS in an upper-middle-income country such as China. In addition, the significance of prevalence data is emphasized to ascertain the level of severity in low-income countries. GBS prevalence in China: From 2016 to 2019, 5548 hospital records from 38,861 GBS patients were evaluated: 9163 in 2016, 9485 in 2017, 11,519 in 2018, and 8694 in 2019.[31] During the years 2006 and 2007, a lower-middle income country, Bangladesh, documented a total of 1619 and 1844 instances of acute flaccid paralysis in children under the age of 15. Out of these cases, 608 (37%) and 855 (46%) were confirmed as GBS based on the established criteria.[32] The majority of studies investigating the prevalence of GBS have been conducted in populations residing in HICs, with only a limited number of studies including populations from LMICs. The incidence of GBS has been reported to range from 0.16 to 3.0 cases per 100,000 individuals per year[33]; this significant variability in incidence rates may be attributed, at least in part, to differences in geographical locations. For example, an upper middle-income country, Brazil, reported an incidence rate of approximately 0.40 cases per 100,000 individuals per year, whereas Europe and North America reported rates ranging from 0.84 to 1.91 cases per 100,000 individuals per year. Iran, Curaçao, and Bangladesh had higher incidence rates, ranging from 2.1 to 3.0 cases per 100,000 individuals per year.[7,23,32] However, due to a lack of facilities for diagnosis, treatment, prevention, and public awareness in LMICs, the precise epidemiological data may not be recorded.

Australia, a high-income country, and the other, Burkina Faso, a low-income country; an Australian cohort study was conducted, reviewing 46 people with GBS (54% male), with an average age of 55 years. 61% of cases were found to have a previous infection. Twenty-eight percent of individuals experienced previous immunogenic events or disorders. The most prevalent category, accounting for 78% of cases, was acute inflammatory demyelinating polyradiculoneuropathy. Forty-three percent of the cases had CSF albuminocytologic separation. The most common finding in electrodiagnostic testing was demyelination, observed in 64% of cases. Ninety-eight percent of the subjects underwent immunotherapy, with the majority receiving intravenous immunoglobulin (IVIg) treatment (93%). Twenty-two percent of individuals underwent additional treatment as a result of changes in their treatment or a lack of improvement. Thirteen percent of patients necessitated intensive care unit (ICU) stay, while 46% required rehabilitation. No fatalities or requirements for mechanical ventilation were reported. At the 6-month mark, 71% of the follow-up sample still experienced some level of disability, although it was often not severe.[34]

A cross-sectional study was done to evaluate the epidemiological, clinical, and treatment profile of GBS patients hospitalized in the neurology department in Burkina Faso over the past 18 years. The incidence of GBS in their specific environment was extremely low, with only 1.9 cases occurring each year. The scarcity of neurologists and neurophysiologists in the nation could account for this predicament.[35] Approximately 14.2% of patients had experienced an infectious condition before being admitted to the neurology department. Based on the literature, about two-thirds of cases of GBS are preceded by an acute infection occurring within a period of 3–4 weeks.[36] The limited incidence of infections can be attributed to the prolonged duration patients have to wait before being admitted to the hospital.[35] The

diagnosis of GBS relies on the patient's medical history and assessments of their neurological function, electrophysiology, and CSF.[36] However, in settings with limited resources, the diagnosis is typically done based on clinical observations with some additional laboratory tests. 88.5% of instances had a lumbar puncture, but only a small percentage of patients (17.1%) had access to an electromyogram. The restricted availability of electromyogram machines, with only two in the entire country, along with the high cost of 30,000 fcfa per test, and the severity of the patients, can account for the poor rate of completion of this procedure. Albuminocytological dissociation was observed in 42.8% of cases, which is similar to the rate reported by Basse et al.[35] in Senegal (45.4%).

Challenges in GBS prevention and management

Due to the varied clinical presentation and the vast range in prognosis among individuals, GBS can be difficult to identify and manage. Managing GBS can be particularly difficult when epidemics caused by infectious diseases occur. The severity of the condition among the human population is further exacerbated by the fact that there are numerous causes or contributors to GBS and that numerous studies are still being conducted in this area. Therefore, to prevent the consequences of this disease, thorough diagnosis, treatment management, and a variety of preventative measures should be implemented in all countries, especially in LMICs.

The process of diagnosing medical conditions is often more difficult in LMICs. This is due to limited access to resources such as facilities for CSF testing and nerve conduction examinations, which are essential for accurate diagnosis. As a result, patients in LMICs may be referred to many healthcare providers, leading to delays in receiving a proper diagnosis. In a prospective global cohort study, it was observed that the median duration between the initiation of weakness and the commencement of the study was found to be 5 days in the Netherlands, whereas it was 10 days in Bangladesh.[37] Inadequate or delayed diagnosis, restricted availability of appropriate diagnostic and healthcare resources, insufficient reporting of GBS, and lack of awareness in poor and middle-income countries may lead to the untimely demise of patients with severe infections prior to their arrival at the hospital.

Patients with GBS who are at risk of immediate respiratory insufficiency, acute autonomic dysfunction with cardiovascular instability, severe swallowing dysfunction, reduced cough reflex, or quickly progressing weakness are advised to be admitted to the ICU. Nevertheless, in LMICs, the availability of ICU beds is restricted, and the provision of ICU services in private hospitals is prohibitively expensive, with costs ranging from approximately US$300 to US$1,200 per day, making it unaffordable for the majority of patients.[21] A study conducted in Bangladesh revealed that the lack of ICU support, when necessary, was the most significant risk factor associated with mortality in patients diagnosed with GBS.[38] Given the inadequacy of the healthcare system in LMICs, it is essential that all hospitals, including those serving underprivileged communities, regardless of their location, have fair and ample access to essential healthcare resources. These resources include critical care equipment such as ICU beds, ventilators, and life support systems, as well as necessary treatment and diagnostic facilities, and an adequate number of healthcare personnel. It is essential to strengthen the healthcare system's ability to handle serious life-threatening situations.

Enhanced comprehension of GBS in LMICs is necessary due to the susceptibility of populations in LMICs to GBS outbreaks, resulting from poor hygiene practices and heightened exposure to infections. In addition, the limited availability of diagnostic and healthcare resources in LMICs contributes to the delayed diagnosis of individuals with severe manifestations of GBS. Furthermore, the absence of comprehensive clinical guidelines at the national level, along with the unavailability of affordable and efficacious therapies, exacerbates the adverse outcomes and increased mortality rates observed in LMICs compared to HICs. In the context of health system issues in LMICs, there is a pressing need to develop novel, focused, and affordable treatment options.[21]

The diagnosis and care of patients with GBS might be hindered by resource constraints in LMICs, such as the scarcity of electrodiagnostic machines, hospital and ICU beds, and rehabilitation clinics.[39] A recent study conducted in China revealed a decreased occurrence of antecedent *C. jejuni* infection in cases of GBS. The study compared data from 2013 to 2017, where the incidence was found to be 27%, with data from 1991 to 1992, where the incidence was reported to be 66%. This decline can be attributed to the advancements in health care that have taken place over the past 5 decades. Enhancing healthcare infrastructure in low- and middle-income countries can effectively reduce the prevalence of severe GBS syndrome.[21] The New Zealand government implemented a nationwide effort to mitigate the presence of *Campylobacter* spp. in chicken, aiming to decrease infection levels. In a span of 2 years, the nation successfully recorded a 52% decrease in campylobacteriosis cases and a concurrent 13% drop in hospital admissions related to GBS.[40] It is imperative to examine and implement infection control measures in LMICs.

GBS cases have been linked to vector-borne viruses, such as chikungunya and dengue, which are carried by the same *Aedes* family of mosquitoes as the Zika virus. These infectious diseases are more prevalent in sub-Saharan Africa and portions of Asia, which are primarily composed of LMICs. Consequently, these areas are especially vulnerable to new occurrences of GBS.[21] To mitigate the life-threatening condition and reduce the severity of GBS, it is imperative to implement measures to manage these infectious diseases transmitted by vectors.

As stated before, there is concern over the transmission of GBS through food. Therefore, it is crucial to take the necessary measures to increase the public's awareness about the severe consequences of the condition. GBS has the potential to be fatal because it can induce respiratory difficulties and almost complete paralysis. People with GBS should receive treatment and monitoring as soon as feasible; some may require emergency medical attention, intensive care, and life support. It is important to keep an eye out for consequences in all GBS patients, which might include irregular heartbeat, infections, blood clots, and high or low blood pressure. Supportive care, which includes monitoring of the heartbeat, blood pressure, and respiration, should be publicly accessible. To facilitate research on GBS in LMICs, it is imperative to establish a sustainable clinical trial infrastructure. This infrastructure should encompass physical healthcare facilities and a well-trained group of health professionals. In addition, it is crucial to develop high-quality diagnostic laboratories and implement training programs for healthcare professionals involved in the care of GBS patients and clinical research.

Within 1–2 days of the commencement of the illness, the severity of GBS can occasionally progress to a fulminating condition with quadriplegia and the need for ventilator support. Such sorts of GBS cases exhibit slower healing, significant residual disability, and axonal deterioration, and are unable to walk without assistance for 6 months or a year following the onset of sickness.[41] Given that GBS lacks a definitive preventive measure and can arise from various causes and conditions, it is imperative that low- and middle-income nations have widespread access to comprehensive treatment and diagnostic facilities. The recommended therapies are outlined below.

The primary sources of biomarkers for GBS include serum, CSF, and peripheral nerve tissue. There are several ways to diagnose *C. jejuni* infection in GBS, including the isolation of *C. jejuni* from stool culture, enzyme-linked immunosorbent assay as a serological test, real-time polymerase chain reaction, and lymphocyte transformation test.[41] These tests along with biomarkers ought to be widely accessible in all nations, particularly in low- and middle-income nations with inadequate healthcare infrastructure because preventing illness severity requires first identifying the disease.

When a patient is unable to walk by themselves, therapies including IVIg and plasma exchange are recommended because corticosteroids are typically useless in GBS. Plasmapheresis, which has been the gold standard treatment for GBS for the past 2 decades, successfully eliminates specific inflammatory molecules (cytokines, complement, antibodies, etc.) from the blood.[41] Since plasma exchange is cheaper than intravenous immunoglobulin, it may be the preferred method of treating GBS in LMICs.[42]

GBS does not have a specific treatment, however giving intravenous immunoglobulin or performing therapeutic plasma exchange early on can speed up motor recovery, reduce the time spent on ventilator support, and are regarded as the standard of care for GBS. Nevertheless, these medicines are frequently inaccessible to numerous patients in LMICs due to their exorbitant cost, limited accessibility, and the need for specialist skills in prescribing them.[43] For example, most patients in Bangladesh cannot afford intravenous immunoglobulin or plasma exchange due to the country's poor per capita income and lack of coverage by the national health insurance system. Consequently, even though the majority of GBS patients in Bangladesh suffer from severe symptoms, only 10%–12% of patients in Bangladesh receive one of these medications.[44] Less expensive options, such as modified therapeutic plasma exchange, exchange blood transfusion, rituximab, and pulse steroid therapy, have been used in various regions and have demonstrated effectiveness, although the data are limited.[43] Therefore, it is imperative to thoroughly examine and expeditiously make accessible these alternative, economically viable treatments in all LMICs.

In recent years, there has been growing attention regarding the possible use of monoclonal antibodies as a therapy option for GBS, in line with their established efficacy in other autoimmune neurological disorders.[45] The therapy of GBS in HICs has recently shifted its attention toward complement inhibitors. The investigation of Eculizumab, a humanized monoclonal recombinant antibody targeting complement factor 5, is presently underway in the United Kingdom and Japan.[46] ANX005, a humanized antibody targeting complement factor 3, has demonstrated safety and tolerability in patients with GBS. Currently, effectiveness trials for this therapeutic drug are underway in Europe, the United States, and Asia.[6] The antibody-cleaving enzyme known as Imlifidase and the cord-blood-derived T-regulatory cell product called CK0801 are presently undergoing investigation in human studies for their potential application in the treatment of GBS.[45] The considerable expense associated with biologic therapies is expected to impose significant limitations on their utilization among patients with GBS in LMICs. However, it is possible that these medications could become accessible for certain applications within LMICs at more affordable price points in the future. Although GBS has no known cure, treatments can help alleviate its symptoms and abbreviate its duration. Therefore, these treatment facilities must be accessible at all times to save lives and mitigate the severity of GBS disease.

To mitigate the occurrence of any disease, it is essential to ascertain the total number of affected cases and identify the underlying reasons before implementing preventive measures. Epidemiology is the scientific discipline that investigates the occurrence and determinants of diseases in different populations. Epidemiological data are necessary for the implementation and extension of disease prevention programs, as well as the care of persons with early-onset sickness. The widespread implementation of GBS "molecular-epidemiological" data gathering demands priorities in LMICs as it is crucial to ensure a sound healthcare

system throughout a nation in case of both communicable and non-communicable diseases.[47,48]

Currently, the diagnosis of GBS in lower-income countries poses significant challenges. However, considering the potential avenues discussed in this comprehensive article, it is recommended that additional research be conducted at the molecular level, in conjunction with epidemiological analysis.[48] This approach would enhance our comprehension of the molecular epidemiology of GBS, thereby facilitating the development of effective public health policies for disease prevention and treatment.[49]

Recent research has shown that machine learning enhances the effectiveness of image processing by identifying early signs of diseases that cannot be detected using conventional methods. Indeed, artificial intelligence (AI) approaches often play a crucial role in facilitating the detection and treatment of cancer. This remains significant even in underdeveloped countries when the provision of optimal healthcare is hindered by limited resources, the high cost of medical services, and various other challenges. A recent study revealed the capability of utilizing deep learning and rudimentary imaging to develop an inexpensive point-of-care solution for lymphoma diagnosis.[50] Personalized medicine, also known as precision medicine, is an emerging healthcare approach that tailors the treatment and prevention of diseases to the specific situations of individuals. This includes taking into consideration their genetic information, psychosocial features, surroundings, and lifestyles. The abundance of data generated from this information necessitates the utilization of AI technology for analysis and integration.[51] Given the significant influence of AI technology on various diseases, including in undeveloped countries, it is imperative that sufficient attention and extensive research be directed toward the application of AI technology in the case of GBS. AI can analyze an individual's genetic data to discern personalized treatment alternatives. One of the most notable areas where AI exhibits great potential is in the realm of disease diagnosis and treatment. The utilization of a cluster algorithm for feature selection from datasets has demonstrated a notable level of accuracy in characterizing GBS through the application of AI.[52] This finding suggests the potential for computer-assisted GBS diagnosis. Recent advancements in technology have contributed to enhanced diagnostic accuracy for axonal GBS. These include the use of metabolite analysis, peripheral nerve ultrasonography, and feature selection by AI.[53] Additional research should be conducted to facilitate the development of advanced technology for addressing various forms of GBS. This would greatly contribute to the improvement of diagnostic and therapy management in LMICs.

The main limitation of our study was that we were unable to graphically represent the disease prevalence of GBS due to a lack of country-specific comprehensive data, hindering a detailed analysis of the disease situation in high-, middle-, and low-income countries. A meta-analysis on the topic of GBS disease may have provided an in-depth analysis of its global prevalence, distribution across different income countries, options for treatment, diagnosis techniques, and risk factors. The results of a meta-analysis may provide a more precise estimation of the treatment impact, disease risk factors, or other consequences compared to the individual studies included in the analysis. Therefore, additional meta-analytical research should be carried out to delve deeper into the GBS disease situation. Cohort studies allow for the examination of several outcomes associated with a single exposure, serving as a valuable method to explore the connection between exposure and outcomes, despite being costly and requiring a significant amount of time to carry out. To acquire the desired GBS scenario, doing a cohort study on the disease in high-, middle-, and low-income nations is necessary.

Conclusion

Due to the absence of an effective cure or preventive mechanism for GBS, the infection poses a heightened level of concern and fatality, particularly in low-income nations. To further progress in this particular discipline, it will be imperative for researchers to engage in a joint endeavor aimed at comprehensively analyzing the intricate processes involved in molecular mimicry and the resulting immune-mediated nerve injury. To mitigate the life-threatening condition and instances of GBS in LMICs, it is imperative to swiftly establish contemporary and efficient treatment facilities, as well as cost-effective treatment options and adequate diagnostic capabilities in all such countries. Furthermore, immediate measures must be implemented to strengthen the healthcare infrastructure in these nations.

Acknowledgements

The authors would like to thank the Department of Pharmacy, University of Asia Pacific for their support in completing the project.

Author contributions

SAK and PRD conceptualized and wrote the draft. ZN reviewed the manuscript. SMRD conceptualized, revised the manuscript, and supervised the project. All authors approved the final draft.

Data availability

Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

Ethics approval

Not applicable.

Informed consent

Not applicable.

ORCID iDs

Sakif Ahamed Khan https://orcid.org/0009-0008-9397-4299
Syed Masudur Rahman Dewan https://orcid.org/0000-0003-1443-7150

References

1. Shastri A, Al Aiyan A, Kishore U, et al. Immune-mediated neuropathies: pathophysiology and management. *Int J Mol Sci* 2023; 24: 7288.
2. Zheng X, Fang Y, Song Y, et al. Is there a causal nexus between COVID-19 infection, COVID-19 vaccination, and Guillain-Barré syndrome? *Eur J Med Res* 2023; 28: 98.
3. Mukerji S, Aloka F, Farooq MU, et al. Cardiovascular complications of the Guillain-Barré syndrome. *Am J Cardiol* 2009; 104: 1452–1455.
4. Willison HJ, Jacobs BC and Van Doorn PA. Guillain-Barré syndrome. *Lancet* 2016; 388: 717–727.
5. Lopes GV, Ramires T, Kleinubing NR, et al. Virulence factors of foodborne pathogen *Campylobacter jejuni. Microb Pathog* 2021; 161: 105265.
6. Islam Z, Jacobs BC, Van Belkum A, et al. Axonal variant of Guillain-Barre syndrome associated with Campylobacter infection in Bangladesh. *Neurology* 2010; 74: 581–587.
7. Van Koningsveld R, Rico R, Gerstenbluth I, et al. Gastroenteritis-associated Guillain-Barre syndrome on the Caribbean Island Curacao. *Neurology* 2001; 56: 1467–1472.
8. Dourado ME, Félix RH, Da Silva WKA, et al. Clinical characteristics of Guillain-Barré syndrome in a tropical country: a Brazilian experience: Guillain-Barré syndrome in the tropics. *Acta Neurol Scand* 2012; 125: 47–53.
9. Rhodes KM and Tattersfield AE. Guillain-Barre syndrome associated with Campylobacter infection. *BMJ* 1982; 285: 173–174.
10. Rees JH, Gregson NA and Hughes RA. Anti-ganglioside GM_1 antibodies in Guillain-Barré syndrome and their relationship to Campylobacter jejuni infection. *Ann Neurol* 1995; 38: 809–816.
11. Shahar E. Current therapeutic options in severe Guillain-Barré syndrome. *Clin Neuropharmacol* 2006; 29: 45–51.
12. Hadden RDM and Gregson NA. Guillain-Barre syndrome and Campylobacter jejuni infection. *J Appl Microbiol* 2001; 90: 145S–154S.
13. Jacobs BC, Van Doorn PA, Tio-Gillen AP, et al. Campylobacter jejuni infections and anti-GM1 antibodies in Guillain-Barré syndrome. *Ann Neurol* 1996; 40: 181–187.
14. Pithadia AB and Kakadia N. Guillain-Barré syndrome (GBS). *Pharmacol Rep* 2010; 62: 220–232.
15. Chen R, Rastogi S, Mullen J, et al. The vaccine adverse event reporting system (VAERS). *Vaccine* 1994; 12: 542–550.
16. Haber P, Sejvar J, Mikaeloff Y, et al. Vaccines and Guillain-Barré syndrome. *Drug Saf* 2009; 32: 309–323.
17. Wajih Ullah M, Qaseem A and Amray A. Post vaccination Guillain-Barre syndrome: a case report. *Cureus* 2018; 10(4): e2511.
18. Salmon DA, Proschan M, Forshee R, et al. Association between Guillain-Barré syndrome and influenza A (H1N1) 2009 monovalent inactivated vaccines in the USA: a meta-analysis. *Lancet* 2013; 381: 1461–1468.
19. Rohilla R, Kakkar AK, Divyashree K, et al. Recombinant protein subunit COVID-19 vaccine-induced Guillain-Barré syndrome in an adolescent: a case report. *Br J Clin Pharmacol* 2023; 89: 556–560.
20. Lucchese G and Flöel A. SARS-CoV-2 and Guillain-Barré syndrome: molecular mimicry with human heat shock proteins as potential pathogenic mechanism. *Cell Stress Chaperones* 2020; 25: 731–735.
21. Papri N, Islam Z, Leonhard SE, et al. Guillain–Barré syndrome in low-income and middle-income countries: challenges and prospects. *Nat Rev Neurol* 2021; 17: 285–296.
22. Bragazzi NL, Kolahi A-A, Nejadghaderi SA, et al. Global, regional, and national burden of Guillain–Barré syndrome and its underlying causes from 1990 to 2019. *J Neuroinflammation* 2021; 18: 264.
23. Sejvar JJ, Baughman AL, Wise M, et al. Population incidence of Guillain-Barré syndrome: a systematic review and meta-analysis. *Neuroepidemiology* 2011; 36: 123–133.
24. Hauck LJ, White C, Feasby TE, et al. Incidence of Guillain-Barre syndrome in Alberta, Canada: an administrative data study. *J Neurol Neurosurg Psychiatry* 2008; 79: 318–320.
25. Hughes RA, Charlton J, Latinovic R, et al. No association between immunization and Guillain-Barré syndrome in the United Kingdom, 1992 to 2000. *Arch Intern Med* 2006; 166: 1301.
26. Al-Hakem H, Sindrup SH, Andersen H, et al. Guillain–Barré syndrome in Denmark: a population-based study on epidemiology, diagnosis and clinical severity. *J Neurol* 2019; 266: 440–449.
27. Guillain-Barré Syndrome – Peru. World Health Organization (WHO), https://www.who.int/emergencies/disease-outbreak-news/item/2023-DON477 (2023, accessed 2 November 2023).
28. Madden J, Spadaro A, Koyfman A, et al. High risk and low prevalence diseases: Guillain-Barré syndrome. *Am J Emerg Med* 2024; 75: 90–97.
29. Liou L-S, Chung C-H, Wu Y-T, et al. Epidemiology and prognostic factors of inpatient mortality of Guillain-Barré syndrome: a nationwide population study over 14 years in Asian country. *J Neurol Sci* 2016; 369: 159–164.
30. Kim A-Y, Lee H, Lee Y-M, et al. Epidemiological features and economic burden of Guillain-Barré syndrome in South Korea: a nationwide population-based study. *J Clin Neurol* 2021; 17: 257.
31. Zheng P, Tian D-C, Xiu Y, et al. Incidence of Guillain-Barré syndrome (GBS) in China: a national population-based study. *Lancet Reg Health West Pac* 2022; 18: 100302.
32. Islam Z. High incidence of Guillain-Barre syndrome in children, Bangladesh. *Emerg Infect Dis* 2011; 17: 1317–1318.
33. McGrogan A, Madle GC, Seaman HE, et al. The epidemiology of Guillain-Barré syndrome worldwide. *Neuroepidemiology* 2009; 32: 150–163.
34. Barnes SL and Herkes GK. Guillain–Barré syndrome: clinical features, treatment choices and outcomes in an Australian cohort. *Intern Med J* 2020; 50: 1500–1504.

35. Basse AM, Boubacar S, Sow AD, et al. Epidemiology of acute polyradiculoneuritis at Fann department of neurology Dakar, Senegal. *Clin Neurol Neurosci* 2017; 1: 76–79.
36. Dabilgou AA, Kaboré R, Dravé A, et al. Guillain-Barré syndrome (GBS) in Sub-Saharan Africa: experience from a tertiary level hospital in Burkina Faso. *PAMJ Clin Med* 2022; 8: 15.
37. Islam MB, Islam Z, Farzana KS, et al. Guillain-Barré syndrome in Bangladesh: validation of Brighton criteria. *J Peripher Nerv Syst* 2016; 21: 345–351.
38. Ishaque T, Islam MB, Ara G, et al. High mortality from Guillain-Barré syndrome in Bangladesh. *J Peripher Nerv Syst* 2017; 22: 121–126.
39. Leonhard SE, Conde RM, De Assis Aquino Gondim F, et al. Diagnosis and treatment of Guillain-Barré syndrome during the Zika virus epidemic in Brazil: a national survey study. *J Peripher Nerv Syst* 2019; 24: 340–347.
40. Baker MG, Kvalsvig A, Zhang J, et al. Declining Guillain-Barré syndrome after campylobacteriosis control, New Zealand, 1988–2010. *Emerg Infect Dis* 2012; 18: 226–233.
41. Nyati KK and Nyati R. Role of Campylobacter jejuni infection in the pathogenesis of Guillain-Barré syndrome: an update. *BioMed Res Int* 2013; 2013: 1–13.
42. Willison HJ, Jacobs BC and Van Doorn PA. Guillain-Barré syndrome: surveillance and cost of treatment strategies—Authors' reply. *Lancet* 2017; 389: 253–254.
43. Nepal G, Shrestha GS, Shing YK, et al. Low-cost alternatives for the management of Guillain-Barré syndrome in low- and middle-income countries. *World Med Health Policy* 2021; 13: 749–757.
44. Islam B, Islam Z, Rahman S, et al. Small volume plasma exchange for Guillain-Barré syndrome in resource-limited settings: a phase II safety and feasibility study. *BMJ Open* 2018; 8: e022862.
45. Rajabally YA. Immunoglobulin and monoclonal antibody therapies in Guillain-Barré syndrome. *Neurotherapeutics* 2022; 19: 885–896.
46. Verboon C, Van Doorn PA and Jacobs BC. Treatment dilemmas in Guillain-Barré syndrome. *J Neurol Neurosurg Psychiatry* 2017; 88: 346–352.
47. Proma AY, Das PR, Akter S, et al. The urgent need for a policy on epidemiological data on cardiovascular diseases in Bangladesh. *Health Sci Rep* 2023; 6: e1410.
48. Dewan SMR. The risk of SARS-CoV-2 infection through sexual contact should be investigated: a timely call. *Immun Inflamm Dis* 2023; 11: e971.
49. Honardoost M, Rajabpour A and Vakil L. Molecular epidemiology; New but impressive. *Med J Islam Repub Iran* 2018; 32: 312–316.
50. Agrebi S and Larbi A. Use of artificial intelligence in infectious diseases. *Arti Intel Prec Health* 2020: 415–438.
51. Mesko B. The role of artificial intelligence in precision medicine. *Expert Rev Precis Med Drug Dev* 2017; 2: 239–241.
52. Hernández-Torruco J, Canul-Reich J, Frausto-Solís J, et al. Feature selection for better identification of subtypes of Guillain-Barré syndrome. *Comput Math Methods Med* 2014; 2014: 1–9.
53. Shang P, Zhu M, Wang Y, et al. Axonal variants of Guillain–Barré syndrome: an update. *J Neurol* 2021; 268: 2402–2419.