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Clinical characteristics, temporal patterns, and outcomes of Guillain–Barré syndrome

A 7-year cross-sectional study in a low-resource setting

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Abstract

Guillain–Barré syndrome (GBS) is a rare but serious neurological disorder that often leads to significant morbidity and mortality. This study aimed to investigate the clinical characteristics, epidemiological trends, and outcomes of GBS patients admitted to Jimma Medical Center (JMC). This was a mixed retrospective and prospective study conducted at JMC, a tertiary teaching hospital in southwest Ethiopia. Medical records of patients admitted with GBS from October 2017 to August 2021 were retrospectively reviewed, while patients admitted between September 2021 and October 2024 were prospectively recruited. Data were collected on socio-demographic information, clinical presentation, diagnostic tests, treatment, and discharge outcomes. Descriptive statistics, Kaplan–Meier survival analysis, and Geographic Information System mapping were used to analyze the data. A total of 93 patients with GBS were admitted during the 7 year study period. The median age was 30 years (IQR: 23–40), and most patients (54.8%) were within the 15 to 30 age group. Admissions peaked between November and January each year, with notable increase since the end of 2021. Among the 54 patients with complete medical records, the most common presenting symptoms were extremity weakness (92.6%) and difficulty walking (59.3%), while ascending paralysis was observed in 70.4% of cases. Bulbar symptoms occurred in 31.5% of patients. Preceding infections, particularly flu-like symptoms, were reported by 46.3% of them. Overall, 77.8% required ICU care, with 42.6% needing mechanical ventilation. Intravenous immunoglobulin therapy was administered to only 14.8% of patients. The in-hospital mortality rate was 25.9%, and complications such as quadriplegia (46.3%) and depression (35.2%) were common. Patients who required mechanical ventilation faced a significantly higher risk of mortality ($P < .05$), with survival rates dropping to 0% by day 165 in this group. This study highlights a significant burden of GBS at JMC, with notable seasonal trends, high ICU admission rates, and substantial mortality. Early diagnoses, improved access to immunotherapy, and enhanced supportive care, especially for those with respiratory failure, are critical to improving outcomes. The findings underscore the need for robust public health strategies to prevent, manage, and mitigate the impact of GBS, particularly in low-resource settings like Ethiopia.

Abbreviations: ACD = albuminocytologic dissociation, AEFI = adverse event following immunization, AIDP = acute inflammatory demyelinating polyneuropathy, ALP = alkaline phosphatase, ALT = alanine aminotransferase, AMAN = acute motor axonal neuropathy, ANA = antinuclear antibody, AST = aspartate aminotransferase, BUN = blood urea nitrogen, CSF = cerebrospinal fluid, EFDA = Ethiopian Food and Drug Administration, GBS = Guillain–Barré syndrome, GIS = Geographic Information System, HBsAg = hepatitis B surface antigen, HCV Ab = hepatitis C virus antibody, HFDS = hughes functional disability scale, HIV = human immunodeficiency virus, HMIS = health management information system, ICU = intensive care unit, IQR = interquartile range, IVIg = intravenous immunoglobulin, JMC = Jimma Medical Center, MFS = Miller Fisher syndrome, MRC = medical research council, MRN = medical registration number, NINDS = National Institute of Neurological Disorders and Stroke, RBS = random blood sugar, SARS-CoV-2 = severe acute respiratory syndrome coronavirus 2, SD = standard deviation, VDRL = Venereal Disease Research Laboratory, WHO = World Health Organization.

Keywords: Ethiopia, Guillain–Barré syndrome, intravenous immunoglobulin, mortality, quadriplegia, respiratory failure

TGW, EGA and EKG contributed equally to this article.

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1. Introduction

Guillain–Barré syndrome (GBS) is a rare but serious neurological disorder characterized by the immune system attacking the peripheral nervous system, leading to muscle weakness and, in severe cases, paralysis.[1] Globally, the incidence of GBS is estimated to range between 0.16 and 3 cases per 100,000 individuals annually.[2] The incidence of GBS varies geographically and is influenced by seasonal variations, with higher rates often observed during specific months that correlate with outbreaks of infections like *Campylobacter jejuni*, cytomegalovirus, influenza, Epstein–Barr virus, Zika virus,[3,4] and Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2).[5] While rare, certain vaccinations, such as those for influenza, measles-mumps-rubella, hepatitis B, and diphtheria, are considered potential risks for GBS.[6,7]

Recent studies have also suggested a possible association between viral vector-based SARS-CoV-2 vaccines and GBS.[5] Additionally, a research shows that GBS can occur during the early stages of human immunodeficiency virus (HIV), including seroconversion, likely due to immune dysregulation caused by the virus.[8] It often manifests in both acute HIV infection and in individuals with advanced HIV, suggesting a complex interplay of direct viral effects and immune-mediated responses.[9] Patients with HIV may experience more severe forms of GBS and longer recovery periods compared to non-HIV-infected individuals.[10]

The clinical presentation of GBS is heterogeneous, with Acute Inflammatory Demyelinating Polyneuropathy being the most common subtype, characterized by symmetrical ascending motor weakness and sensory disturbances. Other subtypes, including Acute Motor Axonal Neuropathy and Miller Fisher syndrome, present with distinct clinical features but share a common (autoimmune pathogenesis).[11]

Despite the serious nature of GBS, timely administration of treatments such as intravenous immunoglobulin (IVIg) and plasma exchange have been shown to significantly improve patient outcomes.[11] However, many patients continue to experience long-term complications, including residual weakness and pain.[12]

In Ethiopia, GBS represents a critical public health issue, particularly given the country's high prevalence of infectious diseases that can trigger this syndrome. The mortality rate ranges from 10% to 25.8%.[13–15] Understanding the clinical profiles, epidemiological trends, environmental influences, and treatment responses of patients with GBS is crucial for improving patient care and preparing healthcare systems to effectively respond to future outbreaks associated with the condition. This study aimed to investigate the temporal patterns of admission, clinical characteristics, and discharge outcomes of patients admitted with GBS at Jimma Medical Center (JMC), Ethiopia.

2. Methods

2.1. Study design and setting

The study was conducted at JMC, a tertiary teaching hospital located in southwest Ethiopia. It involved a retrospective review of medical records of patients admitted with GBS at JMC from October 2017 to August 2021. Patients admitted from September 2021 to October 2024 were prospectively recruited and followed during their hospital stays.

2.2. Eligibility criteria

Patients who were diagnosed with GBS according to the National Institute of Neurological Disorders and Stroke (NINDS) criteria,[16] and admitted to the medical wards of JMC during the specified study period were included. Patients with no medical records or those whose diagnosis was only documented without detailed records to NINDS diagnostic criteria were excluded.

2.3. Operational definitions

1. The NINDS criteria for the diagnosis of GBS are as follows[16]:
   - Rapid onset of bilateral weakness or paralysis
   - Areflexia (absence of reflexes)
   - Symptoms progressing over days to a maximum of 8 weeks.
   - Exclusion of alternative diagnoses that could explain the clinical presentation, such as myasthenia gravis, toxic neuropathy, or spinal cord disorders
2. Bulbar symptoms: refer to dysfunctions in the cranial nerves IX (glossopharyngeal), X (vagus), XI (accessory), and XII (hypoglossal), leading to difficulties in swallowing, speaking (dysarthria), and controlling the movements of the face and tongue.[17]
3. Albuminocytologic dissociation is characterized by elevated protein levels in cerebrospinal fluid (CSF) without an increase in white blood cell count.[18]
4. Quadriplegia is the paralysis of all 4 limbs (both arms and legs), resulting from damage to the cervical spinal cord or widespread peripheral nervous system damage.[19]
5. Medical Research Council (MRC) scale, was used to evaluate muscle strength upon admission and discharge across 6 key muscle groups (shoulder abduction, elbow flexion, wrist extension, hip flexion, knee extension, and ankle dorsiflexion) on a scale of 0 to 5, where 0 indicates no movement and 5 indicates normal strength.[20]
6. The Hughes Functional Disability Scale was employed to evaluate the functional status of patients at the time of discharge. This scale ranges from 0 to 6, with 0 indicating no disability and 6 indicating death. It assesses the degree of disability in daily activities, providing a comprehensive view of the patient's functional capabilities.[21,22]
7. Depression was defined based on clinical diagnoses made by attending psychiatrists during the hospital stay, relying on patient-reported symptoms and clinical evaluations. The symptoms identified were[23]:
   - Depressed mood most of the day
   - Loss of interest or pleasure in almost all activities
   - Significant weight loss or change in appetite
   - Insomnia or hypersomnia
   - Fatigue or loss of energy
   - Feelings of worthlessness or excessive guilt
   - Diminished ability to think or concentrate
   - Recurrent thoughts of death or suicidal ideation

2.4. Data collection procedures

To identify patients admitted with GBS, a comprehensive search was conducted using the Health Management Information System and inpatient admission logbooks at the hospital. Key information, including medical registration number (MRN), age, sex, address, date of admission, diagnosis, outcome, and discharge or death date, was extracted from these sources.

The MRN was used as a primary method for retrieving patient records from the hospital's main archive. When records could not be located using the MRN, patient names were used as an alternative search criterion. In certain cases, direct contact was made with patients via phone numbers listed in the registration book to assist in locating their records, and occasionally, medical charts were obtained directly from the patients.

The retrieved medical records were thoroughly reviewed by 2 physicians using data collection formats specifically developed for this study. These formats captured socio-demographic details, clinical data, risk factors, diagnostic tests, inpatient management, and discharge outcomes. In cases where medical records were missing or incomplete, attempts were made to contact patients via phone to gather the missing information. However, for the data remained incomplete, only the basic

Table 1

Socio-demographic characteristics of patients with GBS at JMC from 2017 to 2024 (N = 93).

Variables	Frequency (%)
Age group (yr)	
15–30	51 (54.8)
31–45	24 (25.8)
≥46	18 (19.4)
Sex	
Female	29 (31.2)
Male	64 (68.8)

GBS = Guillain–Barré syndrome, JMC = Jimma Medical Center.

information (age, sex, address, date of admission, and diagnosis) retrieved from Health Management Information System was used for analysis.

2.5. Data management and statistical analysis

The dataset was initially collected in Microsoft Excel and underwent comprehensive quality assessment before being analyzed in SPSS version 26 (IBM, New York). Geographic Information System mapping was used to characterize the spatial distribution of cases, and Moran I test was performed to assess spatial autocorrelation for the distribution of GBS cases. Formal sensitivity analyses were conducted to assess the significant association between data completeness and mortality outcomes. Potential selection bias was further quantified through extreme-scenario analyses to estimate mortality rates bounded between the scenarios where all missing cases survived and where all missing cases died, compared to the observed rate in documented cases. To address this limitation, 2 methodological safeguards were implemented: a strict separation of demographic analyses (full cohort, n = 93) from clinical outcome assessments (documented subcohort, n = 54), and transparent reporting of all exclusion criteria. A Mann-Kendall trend test conducted to assess seasonal variation in the admission data. Normality tests were performed using visual inspections of histograms, Q–Q plots, and the Kolmogorov–Smirnov and Shapiro–Wilk tests. Descriptive statistics were used to summarize the study variables, with categorical variables presented as frequencies and percentages, and continuous variables described using the mean and standard deviation (SD) or median and interquartile range (IQR). Log Rank (Mantel–Cox), Breslow (Generalized Wilcoxon), and Tarone-Ware tests were applied to assess the equality of survival distributions based on the need for mechanical ventilation. Additionally, Kaplan–Meier curves were used to evaluate patients' survival during their hospital stay. A *P*-value of <.05 was considered statistically significant for all analyses.

2.6. Ethical considerations

Ethical clearance was obtained from the Institutional Review Board of the Jimma University Institute of Health with a reference number JUIH/IRB/411/21. Informed consent was obtained from all patients who were followed prospectively. For patients with missing data, follow-up contact was made by phone. Prior to any data collection, patients were informed about the purpose of the follow-up and the nature of the information being requested. Participation was voluntary, and only those willing to provide the required information were included. Patient confidentiality was maintained throughout the research process, with access to patient information limited to the principal investigator, supervisor, and data collectors. All data were securely stored and used exclusively for the purposes of this study. This study was conducted in accordance with the guidelines of Good Clinical Practice and the Principles of the Declaration of Helsinki.

3. Result

Over the study period from October 10, 2017, to October 12, 2024, 93 patients were admitted to JMC with a GBS diagnosis. Of these, 39 patients (41.9%) had incomplete data, which were only included in the analysis of socio-demographic variables such as age, sex, geographic distribution, and the overall time trend, leaving only 54 patients for full analysis. The remaining 54 patients with complete records were used for a comprehensive analysis.

3.1. Socio-demographic characteristics

The median age of the 93 admitted patients was 30 years (IQR: 23–40), with an age range of 15 to 75 years. The majority, 51 (54.8%), were within the 15–30-year age group. The majority of patients were male, accounting for 64 (68.8%) of the total cases (Table 1).

3.2. Geographic distribution of the cases

The spatial distribution of GBS cases showed a clear concentration in certain areas, with approximately one-third (33.3%, n = 31) of the cases originating from Jimma Town. Other towns with higher case prevalence included Mizan, with 11 cases (11.8%), and Bonga, with 9 cases (9.7%). While the visual inspection of the distribution suggests clustering of cases in these regions (Fig. 1), statistical analysis using Moran I test did not reveal significant spatial autocorrelation for the distribution of GBS cases (Moran I = −0.073, *P* = .615).

3.3. Pattern of admission

Among the total 93 patients with GBS, the highest number of admissions consistently occurred between November and January across most years: November 2022 (6, 6.5%), December 2023 (7, 7.5%), and January 2022 (5, 5.4%) (Fig. 2). A Mann–Kendall trend test conducted to assess seasonal variation in the admission data revealed no significant trend over time (test statistic = 0.055, *P*-value = .634).

3.4. Clinical status at admission

Among the 54 patients with complete medical records, the median time from symptom onset to admission was 7 days (IQR: 4–11), with a range of 1 to 37 days. The median duration from symptom onset to the development of weakness was 4 (IQR: 2–7) days. Notably, 8 (14.8%) patients were admitted more than 14 days after symptom onset. The most common symptoms at presentation were extremity weakness (92.6%) and difficulty walking (59.3%), with the majority exhibiting ascending paralysis (70.4%). Cranial nerves IX and X were the most commonly affected, leading to bulbar symptoms in 31.5% of patients. Additionally, the majority of patients (51.9%) presented with severe muscle weakness upon admission, as assessed by the MRC scale for muscle strength (Table 2).

3.5. Antecedent events

Among the 54 admitted patients, possible antecedent events were identified in 37 (68.5%) cases of GBS. Nearly half of them, 25 (46.3%), reported recent flu-like symptoms, while 11 (20.4%) reported GI symptoms. Additionally, 1 patient,

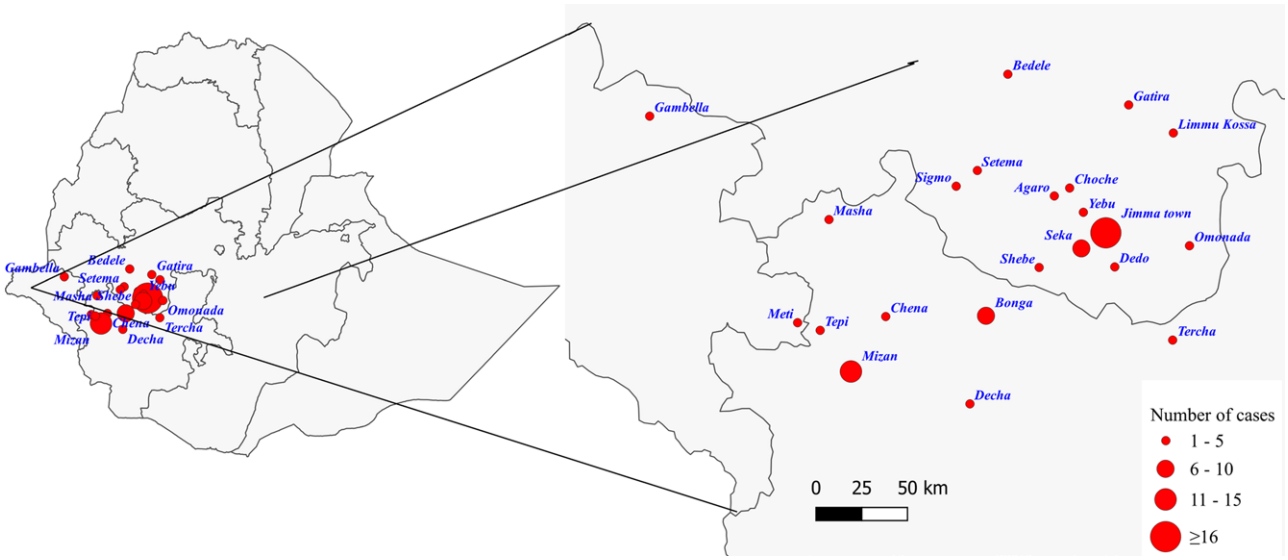


Figure 1. Geographic distribution of GBS by the number cases (N = 93). GBS = Guillain–Barré syndrome.

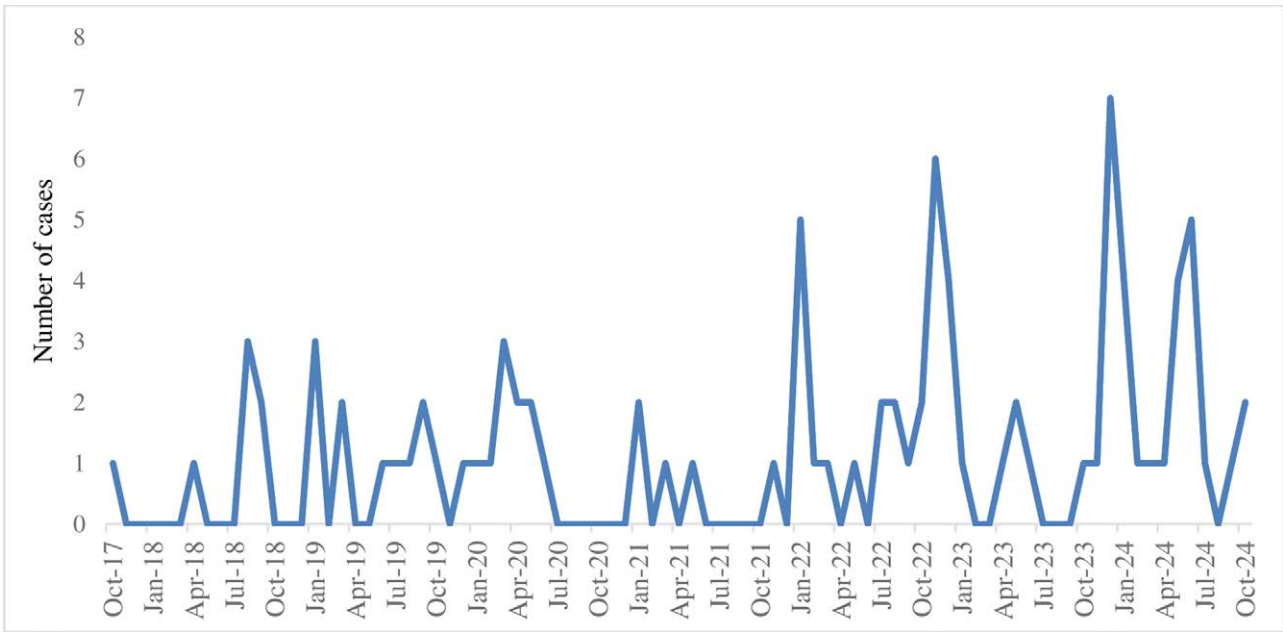


Figure 2. Pattern of admission among patients with GBS at JMC from 2017 to 2024 (N = 93). GBS = Guillain–Barré syndrome, JMC = Jimma Medical Center.

a 51-year-old male, had received a sheep brain derived Fermi type rabies vaccination 14 days prior to admission (Table 3).

3.6. Laboratory profile and neurodiagnostic testing

Among the 54 patients who underwent cerebrospinal fluid (CSF) analysis, 44 (81.5%) exhibited albuminocytologic dissociation. Nerve conduction studies were conducted on 2 patients only, both of whom were diagnosed with acute motor-sensory axonal neuropathy. Brain and whole spine computed tomography (CT) scans were performed for 42 patients, all of whom had normal findings. All 54 patients were tested using HIV rapid tests, as well as for HBsAg and HCV antibodies. The results showed that 2 patients (3.7%) were positive for HIV, 3 patients (5.6%) were positive for HBsAg, and 3 patients (5.6%) were positive for HCV antibodies. Additionally, antinuclear antibody (ANA) testing was performed for 25 patients, and only 1 (1.9%) tested positive (Table 4).

3.7. Management and course in the hospital

Of 54 patients, the majority, 42 (77.8%) were admitted to the ICU and managed with respiratory support. The mean duration of mechanical ventilation for the 23 patients (42.6%) who required ventilator support was 9.6 (SD: 11.3) days. Intravenous immunoglobulin therapy was administered to only 8 patients (14.8%) while none underwent plasmapheresis. Quadriplegia 25 (46.3%), depression 19 (35.2%), and bed sores 12 (22.2%) were the 3 most commonly documented complications. The median hospital stay was 11.5 (IQR: 5–22.3) days, with a range of 2 to 165 days (Table 5).

Table 2

Clinical characteristics of patients with GBS at JMC from 2017 to 2024 (n = 54).

Variables	Output
Days since symptom onset to admission, median (IQR)	7 (4–11)
Time to peak weakness (d), median (IQR)	4 (2–7)
Symptoms at presentation, n (%)	
Extremity weakness	50 (92.6)
Difficulty walking	32 (59.3)
Bulbar symptoms	17 (31.5)
Sensory symptoms	16 (29.6)
Others	13 (24.1)
Motor weakness pattern, n (%)	
Ascending and symmetric	34 (63)
Ascending and asymmetric	4 (7.4)
Descending and symmetric	7 (13)
Descending and asymmetric	7 (13)
Circular	2 (3.6)
Impaired swallow (gag-reflex), n (%)	19 (35.2)
Abnormal single breath count, n (%)	29 (53.7)
Diaphragmatic paralysis, n (%)	21 (38.9)
Muscle tone, n (%)	
Normal	3 (5.6)
Flaccid	51 (94.4)
Muscle strength, MRC scale (on admission), n (%)	
Severe	28 (51.9)
Moderate	18 (33.3)
Mild	8 (14.8)
Deep tendon reflex, n (%)	
Normal	4 (7.4)
Diminished	50 (92.6)
Plantar reflex, n (%)	
Normal	8 (14.8)
Abnormal	46 (85.2)

GBS = Guillain–Barré syndrome, IQR = interquartile range, JMC = Jimma Medical Center, MRC = Medical Research Council.

3.8. Discharge outcome and survival analysis

The in-hospital mortality rate was 25.9% (n = 14). Upon discharge, 31 (57.4%) of patients had severe muscle weakness based on the MRC scale, while 12 (22.2%) and 11 (20.4%) had mild and moderate weakness, respectively. Survival analysis showed that 52.2% of the 23 patients requiring mechanical ventilation did not survive. Patients with respiratory failure had significantly higher mortality risks than those without ($P < .05$). Survival analysis of mechanically ventilated patients (n = 23) revealed rapid early mortality, with survival declining to 60.6% by day 7. By day 165, the curve reached 0% survival due to the death of the last remaining hospitalized patient (from 1 at-risk patient at day 160), while 11 survivors (47.8%) had been discharged earlier and were censored. In contrast, non-ventilated patients (n = 24) maintained stable survival (91.7% at day 6), with only 2 deaths (Fig. S1, Supplemental digital Content, https://links.lww.com/MD/Q771).

3.9. Sensitivity analyses

Sensitivity analysis revealed a significant association between data completeness and mortality ($\chi^2 = 12.519$, df = 1, $P = .0004$), suggesting missingness may be outcome-related. Extreme-case scenarios estimated overall mortality rates ranging from 15.1% (if all missing cases survived) to 57.0% (if all died), compared to 25.9% observed among documented cases. To mitigate potential selection bias due to incomplete records, we analyzed clinical outcomes only among patients with complete data (n = 54), while demographic characteristics were reported for the full cohort (n = 93).

Table 3

Possible antecedent events of patients with GBS at JMC from 2017 to 2024, (n = 54).

Variables	Frequency (%)
Antecedent event	
No	17 (31.5)
Yes	37 (68.5)
Recent symptoms before weakness onset	
None	18 (3.3)
Flu symptoms	25 (46.3)
GI symptoms	11 (20.4)
Recent vaccination (immunization)	
Rabies vaccine (14 d back)	1 (1.9)

GBS = Guillain–Barré syndrome, GI = gastrointestinal, JMC = Jimma Medical Center.

4. Discussion

This study provides a comprehensive analysis of GBS cases admitted to a tertiary teaching hospital in Ethiopia, offering valuable insights into the clinical characteristics, potential antecedent events, and outcomes of GBS in a low-income setting. Our findings are consistent with global reports on the demographic profile, clinical presentation, and complications associated with GBS, while also highlighting some unique regional patterns that have important public health implications.

The majority of patients were young adults, with a median age of 30 years, and males were more frequently affected than females. This demographic trend aligns with previous studies which also reported a higher incidence of GBS in younger males.[24,25] The reasons for this may be related to genetic predispositions or behavioral factors that increase exposure to infections known to trigger GBS, such as *C jejuni* and other viral infections.[3,26]

Jimma Town accounted for the highest number of admissions, with cases from outside Jimma Town referred from larger towns such as Bonga and Mizan. This pattern may be attributed to factors such as population density, improved healthcare access, and environmental influences. The median time from symptom onset to admission in our study was 7 days (IQR: 4–11), consistent with previous studies, which reported ranges from 6 to 8.5 days.[2,13] The most common symptoms at presentation were extremity weakness and difficulty walking, with an ascending pattern, in line with the findings of previous studies.[27,28]

Albuminocytological dissociation (ACD), a hallmark diagnostic feature of GBS, was observed in 44 patients (81.5%) in our study. This finding is consistent with other studies that have reported a high prevalence of ACD in GBS patients.[2,13] The elevated prevalence of ACD in our cohort may be attributed to several factors, including delayed healthcare presentation and the timing of lumbar puncture procedures, which were likely conducted later in the disease progression. As GBS typically evolves over days to weeks, the likelihood of detecting ACD increases as the disease progresses.[29] Additionally, the median time from symptom onset to admission in our study was 7 days, which may have allowed sufficient time for the characteristic changes in the CSF to develop.

One of the key findings of this study was the identification of antecedent events in 68.5% of the GBS cases, with flu-like symptoms and GI symptoms reported by 46.3% and 20.4% of patients, respectively. This is in line with the established understanding that GBS often follows infections,[30] which can trigger an autoimmune response resulting in demyelination or axonal damage of the peripheral nervous system.[26] The association between flu-like symptoms and GBS suggests a possible link to seasonal influenza, which peaks in certain months that may correspond to the higher admission rates observed in our study. The seasonal pattern of peak admissions for GBS observed in

Table 4

Laboratory, nerve conduction and imaging profile of patients with GBS admitted at JMC from 2017 to 2024.

Variables	Output
CSF analysis, n (%)	
Albuminocytologic dissociation	44 (81.5)
Blood chemistry	
RBS, mean (SD)	134.8 (48.2)
Creatinine, median (IQR)	0.7 (0.5–1)
BUN, median (IQR)	7 (4.9–15)
ALT, median (IQR)	45 (25–81)
AST, mean (SD)	54.7 (83.3)
ALP, mean (SD)	152.7 (107.4)
Serum protein, mean (SD)	4.4 (0.5)
Serology, n (%)	
HBsAg, (n = 50) (Positive)	3 (5.6)
HCV Ab, (n = 50) (Positive)	3 (5.6)
VDRL, (n = 50) (Reactive)	1 (1.9)
HIV, (n = 50) (Reactive)	2 (3.7)
ANA (n = 27) (Positive)	1 (1.9)

ALP = alkaline phosphatase, ALT = alanine aminotransferase, ANA = antinuclear antibody, AST = aspartate aminotransferase, BUN = blood urea nitrogen, CSF = cerebrospinal fluid, HBsAg = hepatitis B surface antigen, HCVAb = hepatitis C virus antibody, HIV = human immunodeficiency virus, IQR = interquartile range, RBS = random blood sugar, SD = standard deviation, VDRL = venereal disease research laboratory.

Table 5

Management and hospital course of patients with GBS admitted to JMC from 2017 to 2024 (N = 54).

Variables	Frequency (%)
Admission place, n (%)	
ICU	42 (77.8)
Ward	12 (22.2)
Ventilator support	
Patients requiring ventilation, n (%)	23 (42.6)
Days on mechanical ventilator, mean (SD)	9.6 (11.3)
Patients requiring tracheostomy, n (%)	5 (9.3)
Days with tracheostomy, median (IQR)	24 (10.5–89)
Medications, n (%)	
Immunoglobulin	8 (14.8)
Other supportive medications	46 (85.2)
Complications during hospitalization, n (%)	
Quadriplegia	25 (46.3)
Depression	19 (35.2)
Bed sore	12 (22.2)
Other	20 (37)
Length of hospital stays (d), median (IQR)	11 (5–21)

ICU = intensive care unit, IQR = interquartile range, SD = standard deviation.

this study, particularly the significant increases in November, December, and January, suggests a potential link to specific environmental or epidemiological factors prevalent during these months. These peak periods coincide with typical flu seasons in Ethiopia,[31] where dry and summer months may lead to increased exposure to infectious agents, such as viral or gastrointestinal infections, that can trigger GBS. This observation aligns with findings from previous studies that have shown a correlation between influenza outbreaks and increased cases of GBS, possibly due to molecular mimicry where antibodies produced against the influenza virus cross-react with components of the peripheral nerves. The surge in GBS cases following infectious outbreaks appear to occur regardless of seasonal climate variations, underscoring the need for further investigation into these associations.[3,4]

Most importantly, the significant proportion of patients presenting with flu-like symptoms underscores the need for robust surveillance of influenza and other respiratory pathogens, particularly during peak seasons and in regions with limited healthcare resources. Effective vaccination campaigns are crucial in regions prone to influenza outbreaks, as they play a key role in preventing serious complications, including GBS. Although there have been concerns that influenza vaccination itself may slightly increase the risk of GBS,[32] evidence from high-income countries suggests that the benefits of vaccination outweigh this risk. Influenza vaccines have been shown to significantly reduce the incidence of influenza-related illnesses, which in turn lowers the likelihood of subsequent GBS onset.[33] Importantly, studies have found no significant association between influenza vaccination and GBS. For example, a study in Taiwan reported no increased risk among older adults,[34] and a report from Philadelphia, found no evidence linking GBS with influenza.[35] These findings further support the overall safety of influenza vaccines. However, low-income countries often face challenges such as limited healthcare infrastructure and vaccine availability, which may contribute to a higher incidence of postinfectious GBS.

For countries like Ethiopia, enhancing access to influenza vaccines could be beneficial not only for reducing influenza cases but also for potentially lowering the burden of GBS. Public health strategies to improve vaccine uptake may include increased awareness campaigns, subsidized or free vaccination programs, and integrating influenza vaccination into routine immunization schedules. By addressing these gaps, there is an opportunity to mitigate the dual burden of influenza and its associated complications, such as GBS, in resource-limited settings.

There has been an increase in cases since the end of 2021, which may be closely linked to the COVID-19 burden in Ethiopia. A study conducted at JMC revealed that severe COVID-19 cases were prevalent during these times, coinciding with the subsequent waves of the COVID-19 outbreak in Ethiopia, which were dominated by the Delta and Omicron variants.[36] This association suggests that the increase in GBS cases could be linked to the increased viral activity during these waves, as viral infections, including SARS-CoV-2, are known to trigger autoimmune responses that can lead to GBS.[37] The surge in COVID-19 cases may have contributed to this trend, reflecting post-viral complications, particularly in patients experiencing immune dysregulation associated with COVID-19. Research has documented an increased incidence of GBS following COVID-19 infection, further supporting this hypothesis.[3,5] Additionally, there may be a potential link between the rise in admissions and COVID-19 vaccination, as Ethiopia significantly scaled up vaccination efforts through campaigns at the end of 2021.[38]

The COVID-19 pandemic has expanded the understanding of GBS, with multiple case reports and cohort studies identifying its occurrence following SARS-CoV-2 infection.[39,40] While the precise mechanism remains unclear, it is thought that SARS-CoV-2 may induce autoimmune responses similar to those triggered by other viral pathogens. The pandemic has raised safety concerns around COVID-19 vaccines, especially viral vector-based vaccines, due to reports of GBS following vaccination.[41] In Ethiopia, the most widely administered vaccines were the Oxford-AstraZeneca vaccine (from March 22, 2020, to June 20, 2021)[42] and the Johnson & Johnson vaccine (from August 21, 2021, to June 20, 2022),[43] both of which are viral vector-based vaccines. Importantly, several large-scale studies have reported no significant increase in GBS risk following COVID-19 vaccination. For instance, a study involving over 58 million individuals found no association between mRNA-1273 vaccination and GBS,[44] and other analyses also found no increased risk following various COVID-19 vaccines.[45] These findings support the overall safety of COVID-19 vaccines. However, extensive studies and data from the World Health Organization (WHO) indicate that the risk of GBS following COVID-19 is significantly higher than the risk associated with vaccination, and the overall benefits of vaccination remain substantial.[46]

For low-income countries, these findings have critical implications. Healthcare systems already under strain may face further challenges if GBS emerges as a notable post-COVID complication. Strengthening diagnostic capabilities, improving vaccine access, and educating the public on vaccine safety are essential strategies to mitigate the risk of GBS associated with both COVID-19 infection and vaccination.

Our findings highlight the critical need for early diagnosis and management of GBS. The frequent occurrence of flu-like symptoms and gastrointestinal issues as antecedent events underscores the necessity for prompt evaluation of patients presenting with these prodromal signs, particularly during peak respiratory seasons. The observation that 42.6% of patients required ICU admission and mechanical ventilation underscores the severity of GBS and the urgency of early intervention to avert life-threatening complications. For healthcare providers in resource-limited settings, managing GBS poses significant challenges due to the demand for supportive care, including respiratory assistance and access to treatments like intravenous immunoglobulin (IVIg), which is often costly and scarce in low-income environments.[47]

These findings also advocate for enhanced surveillance systems to monitor GBS incidence, especially following viral outbreaks or mass vaccination campaigns. Public health authorities should consider integrating GBS surveillance with broader disease monitoring programs to facilitate early identification and management. Furthermore, raising awareness about the connection between specific infections, vaccinations, and GBS is essential. Effective communication strategies can help dispel misconceptions regarding vaccine safety, thereby promoting broader vaccine uptake, which may ultimately reduce the burden of GBS.

The clinical course of GBS observed in this study reveals high morbidity rates, with a substantial proportion of patients facing severe complications, including quadriplegia, depression, and prolonged ICU stays. The in-hospital mortality rate of 25.9% reflects the serious nature of this disease. In low-income countries, limited access to advanced diagnostic tools, specialized care, and expensive treatments such as IVIg or plasmapheresis may exacerbate poorer outcomes compared to high-income settings.[47] Strengthening healthcare infrastructure, improving access to essential therapies, and providing training for healthcare professionals on managing neurological emergencies are critical strategies for enhancing outcomes for GBS patients in these regions.

4.1. Strength and limitation

One of the key strengths of this study is its comprehensive review of medical records, allowing for a detailed analysis of patient demographics, clinical presentations, management strategies, and outcomes, particularly in resource-limited settings like Ethiopia.

However, there are notable limitations. The retrospective component may introduce biases due to significant incomplete medical records, which may not provide a full picture of the clinical characteristics and outcomes of GBS patients. Additionally, the findings may not fully represent the broader population of GBS patients in Ethiopia, as the data is limited to a single center. Moreover, limited access to advanced diagnostic tools, such as electrophysiological studies, critically hinders the ability to accurately differentiate between GBS subtypes, such as acute inflammatory demyelinating polyneuropathy and acute motor axonal neuropathy. This limitation impacts the ability to provide definitive subtype diagnoses, which are crucial for understanding disease patterns and guiding treatment. Furthermore, the inconsistent availability of immunotherapy and other advanced treatment options may have influenced patient outcomes, making it difficult to directly compare our results with those from higher-resource settings where such diagnostic and treatment options are routinely available. This limitation should be clearly acknowledged as a significant factor in the interpretation of our findings.

5. Conclusion

This study provides valuable insights into the epidemiology, clinical characteristics, and outcomes of GBS in Ethiopia, with broader implications for similar settings worldwide. The association with antecedent infections, seasonality, and the potential links to vaccinations underlines the need for robust public health strategies to mitigate the burden of this debilitating condition. The observed hospital mortality rate significantly exceeds the global average, highlighting the urgent need for improved healthcare access, timely diagnosis, and early intervention strategies. Future research should focus on understanding the pathogenesis of GBS in diverse populations, the long-term outcomes of patients, and the effectiveness of preventive measures, including vaccination programs in low-income countries. Overall, these findings emphasize the importance of raising awareness about GBS, improving diagnostic and treatment protocols, and enhancing healthcare infrastructure to better manage this serious neurological condition in Ethiopia and similar settings.

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References

[1] Piccione EA, Salame K, Katirji B. Guillain-Barré syndrome and related disorders. In: Katirji B, Kaminski HJ, Ruff RL, editors. Neuromuscular disorders in clinical practice. Springer; 2014. p. 573–603.
[2] Ancona P, Bailey M, Bellomo R. Characteristics, incidence and outcome of patients admitted to intensive care unit with Guillain-Barre syndrome in Australia and New Zealand. J Crit Care. 2018;45:58–64.
[3] Finsterer J. Triggers of Guillain–Barré syndrome: campylobacter jejuni predominates. Int J Mol Sci . 2022;23:14222.
[4] Israeli E, Agmon-Levin N, Blank M, Chapman J, Shoenfeld Y. Guillain–Barré syndrome—a classical autoimmune disease triggered by infection or vaccination. Clin Rev Allergy Immunol. 2012;42:121–30.
[5] Ha J, Park S, Kang H, et al. Real-world data on the incidence and risk of Guillain–Barré syndrome following SARS-CoV-2 vaccination: a prospective surveillance study. Sci Rep. 2023;13:3773.
[6] Salmon DA, Proschan M, Forshee R, et al; H1N1 GBS Meta-Analysis Working Group. Association between Guillain-Barré syndrome and influenza A (H1N1) 2009 monovalent inactivated vaccines in the USA: a meta-analysis. Lancet. 2013;381:1461–8.

[7] Chen Y, Zhang J, Chu X, Xu Y, Ma F. Vaccines and the risk of Guillain-Barré syndrome. Eur J Epidemiol. 2020;35:363–70.
[8] Wang Y, Yang J, Wen Y. The peculiarity of infection and immunity correlated with Guillain-Barre syndrome in the HIV-infected population. J Clin Med. 2023;12:907.
[9] Wagner JC, Bromberg MB. HIV infection presenting with motor axonal variant of Guillain-Barré syndrome. J Clin Neuromuscul Dis. 2007;9:303–5.
[10] Diale MI. Management, Morbidity and Mortality of Guillain Barré Syndrome Patients Admitted to ICU at Universitas Academic Hospital. University of the Free State; 2021.
[11] Van den Berg B, Walgaard C, Drenthen J, Fokke C, Jacobs BC, Van Doorn PA. Guillain–Barré syndrome: pathogenesis, diagnosis, treatment and prognosis. Nat Rev Neurol. 2014;10:469–82.
[12] van Doorn PA. Diagnosis, treatment and prognosis of Guillain-Barré syndrome (GBS). Presse Med. 2013;42:e193–201.
[13] Tewedaj ZD, Huluka DK, Kebede YT, et al. A retrospective analysis of the clinical profile and factors associated with mortality and poor hospital outcomes in adult Guillain–Barre syndrome patients. Sci Rep. 2024;14:15520.
[14] Teshome W, Dagne T, Getachew T. Assessing the predictors and outcomes of Guillain-Barré syndrome at St. Paul's and Zewditu hospitals in Addis Ababa, Ethiopia. Am J Nurs Heal Sci. 2022;3:73–83.
[15] Melaku Z, Zenebe G, Bekele A. Guillain-Barré syndrome in Ethiopian patients. Ethiop Med J. 2005;43:21–6.
[16] Guillain-Barré syndrome | National Institute of Neurological Disorders and Stroke. https://www.ninds.nih.gov/health-information/disorders/guillain-barre-syndrome. Accessed October 10, 2024.
[17] Asbury AK, Cornblath DR. Assessment of current diagnostic criteria for Guillain-Barré syndrome. Ann Neurol. 1990;27:S21–4.
[18] Feasby TE, Gilbert JJ, Brown WF, et al. An acute axonal form of guillain-barrée polyneuropathy. Brain. 1986;109 (Pt 6):1115–26.
[19] O'Connor JR, Leitner LA. Traumatic quadriplegia, a comprehensive review. J Rehabil. 1971;37:14–20.
[20] Kleyweg RP, Van der Meché FG, Schmitz PI. Interobserver agreement in the assessment of muscle strength and functional abilities in Guillain-Barré syndrome. Muscle & Nerve. 1991;14:1103-9.
[21] Hughes RAC, Newsom-Davis JM, Perkin GD, Pierce JM. Controlled trial of prednisolone in acute polyneuropathy. Lancet. 1978;312:750–3.
[22] Hughes AJ, Daniel SE, Kilford L, Lees AJ. Accuracy of clinical diagnosis of idiopathic Parkinson's disease: a clinico-pathological study of 100 cases. J Neurol Neurosurg Psychiatry. 1992;55:181–4.
[23] American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders: DSM-5, 5th ed. American Psychiatric Association; 2013. p. 160-8.
[24] Sharma G, Sood S, Sharma S. Seasonal, age & gender variation of Guillain Barre syndrome in a tertiary referral center in India. Neurosci Med. 2013;4:23–8.
[25] Verma R, Chaudhari TS, Raut TP, Garg RK. Clinico-electrophysiological profile and predictors of functional outcome in Guillain–Barre syndrome (GBS). J Neurol Sci. 2013;335:105–11.
[26] Mir F, Dar W, Yaqoob A, Wani M, Asmi R, Wani M. Seroprevalence of Campylobacter jejuni infection in common subtypes of Guillain–Barre syndrome in Kashmiri population. Egypt J Neurol Psychiatry Neurosurg. 2023;59:168.
[27] Amante JMG, Preysler MG. Clinical profile and outcomes of Guillain-Barre syndrome: a four-year retrospective study at a Tertiary Hospital in the Philippines. Int J Res Publ. 2021;73:119–28.
[28] Al Maawali SM, Al Shibani AY, Nadeem AS, Al-Salti AM. Guillain–Barre syndrome: demographics, clinical features, and outcome in a single tertiary care hospital, Oman. Neurosci J. 2020;25:369–74.
[29] Al-Hakem H, Doets AY, Stino AM, et al; IGOS Consortium. CSF Findings in relation to clinical characteristics, subtype, and disease course in patients with Guillain-Barré syndrome. Neurology. 2023;100:e2386–97.
[30] Sivadon-Tardy V, Orlikowski D, Porcher R, et al. Guillain-Barré syndrome and influenza virus infection. Clin Infect Dis. 2009;48:48–56.
[31] Tadesse M, Mengesha M, Tayachew A, et al. Burden and seasonality of medically attended influenza like illness (ILI) in Ethiopia, 2012 to 2017. BMC Infect Dis. 2020;20:1–13.
[32] Levison LS, Thomsen RW, Andersen H. Guillain–Barré syndrome following influenza vaccination: a 15-year nationwide population-based case–control study. Eur J Neurol. 2022;29:3389–94.
[33] Petráš M, Králová Lesná I, Dáňová J, Čelko AM. Is an increased risk of developing Guillain–Barré syndrome associated with seasonal influenza vaccination? A systematic review and meta-analysis. Vaccines. 2020;8:150.
[34] Yen CC, Wei KC, Wang WH, Huang YT, Chang YC. Risk of Guillain-Barré syndrome among older adults receiving influenza vaccine in Taiwan. JAMA Netw Open. 2022;5:e2232571.
[35] Feature Article — Guillain-Barré syndrome (GBS) & vaccines: the risks and recommendations | children's Hospital of Philadelphia. https://www.chop.edu/parents-pack/parents-pack-newsletter/newsletter-archive/feature-article-gbs-vaccines-risks-and-recommendations. Accessed September 6, 2025.
[36] Abera EG, Tukeni KN, Chala TK, Yilma D, Gudina EK. Clinical profiles and mortality predictors of hospitalized patients with COVID-19 in Ethiopia. BMC Infect Dis. 2024;24:908.
[37] Pimentel V, Luchsinger VW, Carvalho GL, et al. Guillain–Barré syndrome associated with COVID-19: a systematic review. Brain Behav Immun Health. 2023;28:100578.
[38] Galli M, Zardini A, Gamshie WN, et al. Priority age targets for COVID-19 vaccination in Ethiopia under limited vaccine supply. Sci Rep. 2023;13:5586.
[39] Filosto M, Cotti Piccinelli S, Gazzina S, et al. Guillain-Barré syndrome and COVID-19: a 1-year observational multicenter study. Eur J Neurol. 2022;29:3358–67.
[40] Webb S, Wallace VCJ, Martin-Lopez D, Yogarajah M. Guillain-Barré syndrome following COVID-19: a newly emerging post-infectious complication. BMJ Case Rep. 2020;13:e236182.
[41] Wise J. Covid-19: two rare vaccine side effects detected in large global study. BMJ. 2024;384:q488.
[42] EFDA. COVID-19 vaccine safety monitoring and surveillance findings in. 2021. http://www.efda.gov.et/wp-content/uploads/2023/06/COVISHIELD-vaccine-report.pdf. Accessed October 24, 2024.
[43] EFDA. Report on Janssen (Johnson & Johnson) COVID-19 Vaccine Active Surveillance in Ethiopia from August 21, 2021 to June 20, 2022. 2022:1–20. http://www.efda.gov.et/wp-content/uploads/2023/06/Janssen-AEFI-Active-surveillance.pdf. Accessed October 24, 2024
[44] Le Vu S, Bertrand M, Botton J, et al. Risk of Guillain-Barré syndrome following COVID-19 vaccines: a nationwide self-controlled case series study. Neurology. 2023;101:e2094–102.
[45] No significant increase in Guillain-Barré syndrome after COVID-19 vaccination, researchers find | Rutgers University. https://www.rutgers.edu/news/no-significant-increase-guillain-barre-syndrome-after-covid-19-vaccination-researchers-find?utm_source=chatgpt.com. Accessed September 6, 2025.
[46] Risk of Guillain-Barre syndrome 6 times higher after COVID infection, study suggests | CIDRAP. https://www.cidrap.umn.edu/covid-19/risk-guillain-barre-syndrome-6-times-higher-after-covid-infection-study-suggests. Accessed October 22, 2024.
[47] Papri N, Islam Z, Leonhard SE, Mohammad QD, Endtz HP, Jacobs BC. Guillain–Barré syndrome in low-income and middle-income countries: challenges and prospects. Nat Rev Neurol. 2021;17:285–96.