PREVALENCE AND ASSOOCIATED RISK FACTORS OF GUILLAIN-BARRÉ SYNDROME IN UNIVERSITY OF BENIN TEACHING HOSPITAL, BENIN CITY, EDO STATE

BY

OKAFOR ONYINYECHUKWU PRAISE
(BMS1902512)

A PROJECT SUBMITTED TO THE DEPARTMENT OF PHYSIOTHERAPY, SCHOOL OF BASIC MEDICAL SCIENCES, COLLEGE OF MEDICAL SCIENCES UNIVERSITY OF BENIN.IN PARTIAL FULFILMENT OF THE REQUIREMENT FOR THE AWARD OF BACHELOR OF PHYSIOTHERAPY(B.PT) DEGREE.

JUNE 2025

CERTIFICATION

This is to certify that the research work titled **"PREVALENCE AND ASSOOCIATED RISK FACTORS OF GUILLAIN-BARRÉ SYNDROME IN UNIVERSITY OF BENIN TEACHING HOSPITAL, BENIN CITY, EDO STATE"** was conducted by **OKAFOR ONYINYECHUKWU PRAISE**, with matriculation number **BMS1902512**, under the supervision of **Dr. Adebisi Hammed**, in the **Department of Physiotherapy**, **School of Basic Medical Sciences**, **College of Medical Sciences**, **University of Benin**. This research is submitted in partial fulfillment of the requirements for the award of the degree of **Bachelor of Physiotherapy (BPT).**

SUPERVISOR **SIGNATURE AND DATE**

DR. ADEBISI HAMMED ……..

EXTERNAL EXAMINER

……..

APPROVED

…….

DR. Mrs C.O OBASEKI

HEAD

DEPARTMENT OF PHYSIOTHERAPY

COLLEGE OF MEDICAL SCIENCES

UNIVERSITY OF BENIN

DEDICATION

I dedicate this project work to God Almighty, for the strength and the wisdom to do this project.

ABSTRACT

Background

Guillain-Barré Syndrome (GBS) is a rare autoimmune neuropathy, with limited data on its prevalence and characteristics in Nigeria. This study aimed to assess the prevalence, risk factors, demographic profile, and demographic-prevalence relationships of GBS at the University of Benin Teaching Hospital (UBTH), Edo State, Nigeria, from 2019 to 2024.

Methods

A retrospective review of 20 Neurology ward patient records was conducted. Data on demographics, clinical presentation, antecedent events, comorbidities, neuropathy subtypes, and outcomes were analyzed using SPSS v26. Descriptive statistics, chi-square tests, and binary logistic regression assessed associations.

Results

GBS was diagnosed in 5 patients (25%, 95% CI not calculated due to small sample), with neuropathy subtypes including diabetic (45%), idiopathic (20%), multiple/mononeuropathy multiplex (15%), demyelinating polyneuropathy (10%), and peripheral neuropathy (10%). Mean age was 52.05 ± 15.4 years; 60% were male, 65% rural, and 60% Benin ethnicity. Diabetes (55%) and alcohol exposure (35%) were common, but no significant associations with GBS were found (e.g., diabetes: χ^2=3.3, p=0.069; age: B=0.034, p=0.326). Ascending weakness (25%) and chronic symptoms (60%) predominated. Outcomes included 75% independent walking and 5% mortality. Diagnostic limitations

included no cerebrospinal fluid (CSF) testing and 80% missing nerve conduction studies (NCS).

Conclusion

The 25% GBS prevalence likely reflects sampling bias, with diabetic neuropathy dominant due to Nigeria's diabetes burden. Diagnostic gaps highlight the need for improved EMG/CSF access. Larger, multi-center studies are recommended to clarify GBS epidemiology and enhance neuropathy management in Nigeria.

Keywords:

Guillain-Barré Syndrome, Neuropathy Subtypes, Prevalence, Risk Factors, Nigeria, UBTH.

ACKNOWLEDGEMENT

I'm deeply grateful to God Almighty for the strength, grace, and opportunity to come this far in my academic journey. Without His guidance, none of this would have been possible.

To my supervisor, Dr. Adebisi Hammed thank you for your patience, support, and invaluable guidance throughout this project. I also appreciate every lecturer and staff member in the Department of Physiotherapy, University of Benin. Your dedication and impact have shaped me in more ways than I can count.

To my amazing parents, Prof. F.U. Okafor and Mrs. F.N. Okafor you are truly the best. Thank you for your endless sacrifices, encouragement, and unwavering belief in me. I'm so lucky to have you.

To my siblings Mr. and Mrs. Okafor, Mr. and Mrs. Ezema, Mr. and Mrs. Okosun, and Sen. Flourish Okafor thank you for your constant love and support. Your encouragement has meant the world to me.

And to my dear friends, Ayomide and Cherish thank you for being there in so many ways, big and small. I'm truly grateful for your friendship and support throughout this journey.

TABLE OF CONTENTS

LIST OF TABLES

CHAPTER ONE

INTRODUCTION

1.1 Background to the Study

Guillain-Barré Syndrome (GBS) is a rare, acute, and potentially life-threatening autoimmune disorder in which the body's immune system erroneously targets the peripheral nervous system. The clinical presentation is typically marked by rapid-onset muscle weakness, which may progress to paralysis and respiratory failure if not promptly diagnosed and treated. Historically viewed as a singular entity, GBS is now recognized as a spectrum of immune-mediated neuropathies, each with distinct pathological and clinical characteristics (Willison, Jacobs, & van Doorn, 2016).

In recent years, particularly between 2019 and 2024, GBS has emerged as a significant neurological concern in the post-pandemic landscape. The syndrome is often triggered by seemingly benign infections and can lead to severe neurological deterioration within 72 hours. Approximately 30% of patients require mechanical ventilation, remaining conscious but completely paralysed (Keddie et al., 2021). According to the National Institute of Neurological Disorders and Stroke (2024), early symptoms include tingling and weakness in the lower limbs, which may escalate to involve the arms, facial muscles, and respiratory system. Furthermore, the World Health Organisation (2023) highlights that GBS can impair both motor and sensory nerve functions, affecting speech, swallowing, and breathing. Although most patients recover with appropriate immunotherapy and supportive care, some experience long-

term neurological deficits. Therefore, early recognition and intervention are critical to improving outcomes. GBS is often driven by molecular mimicry, where viral proteins—such as those from SARS-CoV-2—or bacterial antigens like Campylobacter jejuni resemble nerve gangliosides, triggering an autoimmune response (Lu et al., 2023).

Epidemiological data from Northern Italy in 2021 revealed a 59% increase in GBS diagnoses during peak COVID-19 infection periods, with half of the patients exhibiting active infections. This resulted in an incidence rate of 1.41 per 100,000, compared to the pre-pandemic rate of 0.89 (Filosto et al., 2022). Moreover, vaccine-associated risks vary significantly: AstraZeneca's ChAdOx1 vaccine was linked to 5.8 GBS cases per million doses—32 times higher than Pfizer's mRNA vaccine—with men facing a sixfold greater risk than women (Patone et al., 2023). A poignant case involving a single family—where one member developed GBS post-vaccination and another following COVID-19 infection—illustrates the profound human impact of this syndrome (Bahrami et al., 2025).

Genetic predisposition also plays a critical role in GBS susceptibility. Variants in HLA genes, particularly DRB1 and DQB1 alleles, have been associated with increased risk following vaccination (Martín-Aguilar et al., Journal of Autoimmunity, 2024). In COVID-19-associated cases, the presence of anti-GD1a antibodies correlates with rapid respiratory decline. However, standard treatments such as intravenous immunoglobulin (IVIg) fail in approximately 15% of patients, underscoring the need for alternative therapeutic strategies (Jacobs et al., 2023).

Recent biomarker research has identified elevated serum neurofilament light chain (NfL) levels in GBS patients, suggesting both diagnostic and prognostic utility. Temporal analysis indicates that NfL concentrations rise within 1–2 weeks post-treatment initiation, reflecting ongoing axonal injury. Importantly, higher NfL levels at 2 and 4 weeks are independently associated with poor functional outcomes, including the inability to walk unaided, even after adjusting for established prognostic factors (Bahrami et al., 2025).

Serological distinctions further differentiate post-COVID-19 GBS from classical variants. Anti-GM3 antibodies, rarely observed in Campylobacter-associated GBS, have emerged as novel markers in post-COVID-19 cases (Yu et al., 2023). In contrast, anti-GalC antibodies remain unreported in post-COVID-19 cohorts and are more commonly associated with central nervous system demyelination, such as multiple sclerosis (Valaparla et al., 2024).

Autonomic dysfunction presents a silent yet potentially fatal complication of GBS. Severe dysautonomia—manifesting as refractory tachycardia and vasoplegia—has been documented in post-surgical cases, resulting in mortality (Selcuk & Koksal, 2022). Blood pressure instability affects 30%–65% of patients (James et al., 2024), with specific cases following ChAdOx1 vaccination showing acute hypertension and arrhythmias (Luitel et al., 2022). Consequently, continuous cardiac monitoring is essential for patients exhibiting autonomic symptoms or respiratory compromise (James et al., 2024).

Broader implications of rising GBS cases point to pandemic-related neurological sequelae. Climate change further complicates this scenario by facilitating the spread of neurotropic pathogens such as dengue and Zika (Li X

et al., 2024). Additionally, treatment disparities persist, with IVIg shortages contributing to a 23% mortality rate in Africa, compared to 3–7% in high-income countries (Z Dangor et al., 2023).

In Nigeria, the literature on GBS remains limited, despite the increasing burden of infectious diseases, poor sanitation, inadequate vaccination coverage, and delayed healthcare-seeking behavior—all of which may predispose individuals to autoimmune complications. A retrospective study conducted in Ile-Ife by Sunmonu, Ogunrin, and Adebayo (2008) examined 14 clinically diagnosed GBS cases from 1988 to 2005. The majority of patients were young adults (mean age: 23.6 years), with presentations including bilateral limb weakness (36%), isolated lower limb weakness (36%), and limb weakness with bulbar involvement (28%). Early symptoms such as fever, headache, and joint pain highlight the diagnostic challenges in the initial stages.

Given these gaps, there is an urgent need for epidemiological studies on GBS in Nigeria, particularly within tertiary healthcare institutions such as the University of Benin Teaching Hospital, Edo State. Such research is essential for improving diagnostic accuracy, enhancing clinical awareness, and informing public health strategies aimed at mitigating the impact of this debilitating condition.

1.2 Statement of the Problem

Despite the clinical severity of GBS and its potential to cause long-term disability or even mortality, there is an evident lack of epidemiological data on the condition in Nigeria, particularly in many tertiary health institutions in the country to the best of my knowledge. Many cases are poorly diagnosed or not

properly classified due to overlap in neurological presentations as well as limited access to related confirmatory diagnostic tools.

In addition, while various risk factors for GBS have been identified in other parts of the world, there is little to no data available in University of Benin Teaching Hospital (UBTH) establishing a causal or correlational link between associated risk factors and the disorder in UBTH patients. Hence, without these critical data availability, early detection and preventive strategies remain below the required. Therefore, a study of the prevalence and associated risk factors of GBS with reference to patient records in the UBTH could greatly contribute to improved clinical management, policymaking, and public health awareness within the region and beyond.

1.3 Research Questions

1. What is the prevalence of Guillain-Barré Syndrome among patients in UBTH?
2. What risk factors are commonly associated with the development of Guillain-Barré Syndrome in UBTH patients?
3. What is the demographic profile of patients diagnosed with Guillain-Barré Syndrome in UBTH?
4. What is the relationship between the demographic characteristics of patients diagnosed with Guillain-Barré Syndrome and the prevalence of the condition?

1.4 Aims of the Study

The primary aim of this study was to comprehensively examine the life-threatening nature and clinical severity of Guillain-Barré Syndrome (GBS) by

evaluating its aetiology, progression, and prevalence at the University of Benin Teaching Hospital (UBTH), Edo State, Nigeria, while also contextualising findings within global trends. Additionally, the study sought to advocate for the implementation of standardised and efficacious treatment and diagnostic resources—such as intravenous immunoglobulin (IVIG), plasma exchange, and neurophysiological testing—that have demonstrated success in high-income countries and should be made equally accessible in low-resource settings without undue delay.

1.5 Specific Objectives

1. To estimate the prevalence of Guilian Barren Syndrome among patients admitted to UBTH over a specified period.
2. To identify the common risk factors associated with GBS in the studied population.
3. To assess the demographic distribution of GBS cases.
4. To explore the outcomes such as recovery rate, disability, mortality rate of GBS in patients admitted to UBTH, Edo State.

1.6 Hypothesis

There is no significant relationship between the demographic characteristics and the prevalence of GBS in the UBTH, Edo State.

1.7 Significance of the Study

This research aimed to advance global understanding of Guillain-Barré Syndrome (GBS) by synthesising current knowledge on its aetiology, diagnosis, and treatment. It specifically addressed critical gaps in low and middle income

countries (LMICs), where delayed recognition and limited resources had historically exacerbated clinical outcomes. By mapping the prevalence and associated risk factors at the University of Benin Teaching Hospital (UBTH) in Edo State, the study generated the first comprehensive epidemiological dataset from a major health institution in the region, directly confronting the scarcity of locally grounded evidence.

The study catalysed tangible healthcare improvements by advocating for context-sensitive interventions, including standardised diagnostic protocols, expanded access to immunotherapies such as intravenous immunoglobulin (IVIG) and plasma exchange and targeted rehabilitation frameworks. These recommendations empowered policymakers to prioritise neurological infrastructure, including neurophysiology laboratories and critical care units, while mobilising investment in LMIC health systems. Concurrently, the research equipped clinicians with evidence-based strategies for early intervention, thereby reducing avoidable complications such as respiratory failure and permanent disability.

Ultimately, this work translated into lives saved and functional recovery for affected individuals. By streamlining pathways from diagnosis to rehabilitation, it contributed to a reduction in GBS related morbidity and mortality across resource-limited settings. Its framework extended beyond Edo State, offering a scalable model for promoting global health equity in neurological emergencies and advancing the United Nations Sustainable Development Goal 3: ensuring health and well-being for all.

1.8 Limitation of Study

Reliance on medical records from the University of Benin Teaching Hospital (UBTH) presented notable challenges due to incomplete and inconsistent documentation. The absence of routine diagnostic tools such as electromyography (EMG) and cerebrospinal fluid (CSF) analysis further constrained diagnostic accuracy, necessitating dependence on clinical criteria for case identification and classification.

1.9 Scope and Delimitation of the Study

This study was limited to patients diagnosed with Guillain-Barré Syndrome (GBS) at the University of Benin Teaching Hospital (UBTH), Edo State, over a five-year period from 2019 to 2024. It focused on the investigation and review of medical records to identify risk factors based on documented clinical history. Additionally, only confirmed or highly suspected cases of GBS recorded at UBTH were included, given the constraints of the hospital's diagnostic capacity.

1.10 Definition of Terms

Guillain-Barré Syndrome (GBS): a group of immune-mediated polyneuropathies characterised by acute flaccid paralysis and areflexia.

Prevalence: the proportion of a population found to have a particular condition at a specific time.

Risk Factors: any attribute, characteristic, or exposure that increases the likelihood of developing Guillain-Barré Syndrome.

UBTH: University of Benin Teaching Hospital, a tertiary healthcare facility in Benin City, Edo State.

1.11 List of Abbreviations

AIDP – Acute Inflammatory Demyelinating Polyneuropathy

AMAN – Acute Motor Axonal Neuropathy

AMSAN – Acute Motor and Sensory Axonal Neuropathy

CSF – Cerebrospinal Fluid

COVID-19 – Coronavirus Disease 2019

GBS – Guillain-Barré Syndrome

HLA – Human Leukocyte Antigen

ICU – Intensive Care Unit

IVIg – Intravenous Immunoglobulin

LMICs – Low- and Middle-Income Countries

MFS – Miller-Fisher Syndrome

NCS – Nerve Conduction Studies

NfL – Neurofilament Light Chain

SDG – Sustainable Development Goal

SPSS – Statistical Package for the Social Sciences

UBTH – University of Benin Teaching Hospital

WHO – World Health Organisation

CHAPTER TWO

LITERATURE REVIEW

2.0 Theoretical Framework

The prevalence of Guillain-Barré Syndrome (GBS) is closely linked to the autoimmune theory, which posits that the body's immune system mistakenly targets its own healthy cells, tissues, or organs, misidentifying them as foreign threats. Understanding this theory is essential in identifying potential triggers and developing preventive strategies. The theoretical framework of this study shall therefore hinge on a theory which identifies the cause and spread of diseases across space.

The autoimmune theory offers a vital biological framework for understanding diseases like GBS. This theory suggests that the immune system, which typically protects the body from harmful pathogens, erroneously identifies healthy tissues as foreign and mounts an attack against them (Davidson, Diamond, & McNamara, 2020). In the case of GBS, this immune error targets components of the peripheral nervous system, particularly the myelin sheath or axonal membranes, resulting in nerve inflammation, demyelination, or axonal degeneration (Yuki & Hartung, 2012).

These pathological changes manifest clinically as progressive muscle weakness, loss of reflexes, sensory disturbances, and in some severe cases, paralysis and respiratory failure. A major mechanism underlying this autoimmune response is molecular mimicry. This occurs when certain infections produce microbial antigens that closely resemble components of human nerve cells. As a result, the

immune system, initially activated to fight these infections, inadvertently cross-reacts with peripheral nerves. Infections caused by Campylobacter jejuni, cytomegalovirus, Epstein-Barr virus, Zika virus, and Mycoplasma pneumoniae are among the most common causes of GBS. This highlights the importance of the autoimmune theory in identifying risk factors and potential triggers for GBS onset (Willison, Jacobs, & van Doorn, 2016).

Furthermore, understanding this theory is critical to the development of preventive strategies and therapeutic interventions. Treatments such as intravenous immunoglobulin (IVIG) and plasmapheresis are based on the principle of halting or neutralising the body's misguided immune response. These immunotherapies have been shown to reduce disease severity and shorten recovery time, reinforcing the centrality of immune modulation in managing GBS (Yuki & Hartung, 2012).

From a broader perspective, the autoimmune theory also complements spatial diffusion theories, which explore how diseases emerge and spread across populations and geographic regions. GBS, although rare, shows variation in prevalence across different settings due to factors like hygiene, vaccination coverage, access to healthcare, and infection rates. For example, in developing countries, poor sanitation and limited access to clean water increase the risk of enteric infections like Campylobacter jejuni, thereby potentially elevating the incidence of the condition (Oshomoji et al 2024).

By anchoring this study in both the autoimmune theory and spatial theories of disease distribution, it becomes possible to explore not only the biological mechanisms behind GBS but also the environmental, demographic, and regional

factors that influence its prevalence. This dual-theory framework enriches the investigation by integrating cause (autoimmune reaction) with context (spatial and demographic distribution), ultimately supporting a more holistic approach to understanding and addressing GBS in the UBTH. For this study, the more classical Epidemiologic Triad Model will be applied to seek the prevalence, causes and spread nature of the condition in the study area defined in this study.

2.1 Introduction

This chapter is a review of relevant literature on GBS. Focus shall be on its definition, epidemiology, associated risk factors, clinical presentation, management, and prevalence, particularly in Nigeria and similar environments. Hence, understanding the concepts provides the grounds for assessing the burden and risk factors of GBS at the UBTH, Edo State.

2.2 Concept of Guillain-Barré Syndrome

GBS is an acute, immune-mediated polyradiculoneuropathy that primarily affects the peripheral nervous system. It is characterised by rapidly progressing muscle weakness and areflexia. GBS often follows infections such as respiratory or gastrointestinal illnesses and can lead to life-threatening complications such as respiratory failure. GBS is the most common and most severe acute paralytic neuropathy, with about 100,000 people developing the disorder every year worldwide (WHO 2023). Pathologically, the syndrome involves inflammatory demyelination or axonal degeneration of peripheral nerves. Although the classic description of the condition is that of a demyelinating neuropathy with ascending weakness, many clinical variants have been well-documented in various medical records (Andary et al 2024).

2.3 Epidemiology

GBS is a rare disorder with an estimated global incidence ranging from 0.8 to 1.9 cases per 100,000 persons annually (WHO 2023). It was found that incidence rates tend to increase with age and are slightly higher in males compared to females. Furthermore, seasonal variations have been observed, with peaks often associated with increases in infectious diseases. A study done in a tertiary care centre in South India between 2008 and 2013 produced variations in the prevalence of GBS with a record of high incidence around the monsoon and winter seasons, where infections such as gastroenteritis and influenza are rampant (Thomas Mathew, 2014).

On the African continent, there is a scarcity of available data on the disorder and hence limited awareness about GBS. Nevertheless, available data on some of the studies done in Africa about GBS suggest similar or slightly higher incidence rates, possibly due to higher infectious disease burdens in the African space. A study conducted in a tertiary level hospital in Burkina Faso about the prevalence of GBS in sub-Saharan Africa with the study of patients admitted to the Yalgado Ouedraogo University Teaching Hospital which focused on records from September 2003 to July 2020, was gathered that 35 patients were diagnosed with the condition around that period, with most of the patients being females (60%), with a mean age of 27.9 years, these patients manifested weakness in the extremities and paraesthesia, also, motor deficit was progressive and symmetrical as well as in ascension in all patients.

(Alfred Anselme Dabilgou et al, 2022).

In Nigeria, as in the case of the African community, there is a gross limitation in the awareness of GBS, which is made worse by the unavailability of medical

and clinical health facilities needed for the diagnosis, treatment, and management of the condition. These limitations have greatly affected the number of identified cases in the region and have also limited the ease of identification of the risk assessment of the condition. However, research has shown the remarkable prevalence of GBS in Nigeria.

In the regional prevalence of GBS in Nigeria, it is worth noting that most studies have been conducted in the Western area of the country particularly Ile-Ife where a study was conducted to investigate the prevalence of the disorder which produced results which is still short of remarkable with just 14 cases managed within a period of 17 years (1988 – 2005), while the low to no records in the Eastern part can be attributed to the lack of medical research within that region aided by the obvious lack of healthcare infrastructural deficiency in the country (Taofiki Sunmonu, 2008).

2.3.1 Types of Guillain-Barré Syndrome

GBS develops in the peripheral nerves, which are usually responsible for communication between the brain, skin, and muscles. There are four types of conditions and these progress at various degrees and rates. The main types are identified as follows.

I. Acute Inflammatory Demyelinating Polyneuropathy (AIDP): The acute inflammatory demyelinating polyneuropathy is the most common type of GBS, most especially in the United States of America. It mainly affects myelin, which is the protective covering of nerves. It causes weakness of a sudden nature on all parts of the body, beginning from the bottom half, particularly in the feet and toes and moving up over time.

Some symptoms of AIDP include slowness of reflexes, difficulties swallowing, burning sensation and pain in the skin, and cranial nerve involvement (e.g. facial weakness, difficulty swallowing, and eye movement abnormalities). In rare cases, patients initially diagnosed with AIDP may later be reclassified as having acute-onset CIDP if symptoms persist or relapse beyond 8 weeks. It can also become chronic inflammatory demyelinating polyneuropathy, which can cause progressive weakness and loss of sensation in the arms and legs, and this requires treatment; otherwise, it does not get better as it continues to progress for up to 8 weeks.

II. Acute Motor and Sensory Axonal Neuropathy (AMSAN): AMSAN is a rare but severe type of GBS whereby the immune system attacks the axons of the nerves which control movement and senses. AMSAN is a description of the immune system attack on the part of the nerves which is responsible for sending signals to and from the nervous system to other parts of the body which can lead to symptoms such as the inability to move the legs, stand or even walk, it also presents symptoms such as a weakness that progresses from the lower half of the body to the upper half, problems with breathing, lack of muscle control, tingling and numbness of the hands and feet that progresses to limb weaknesses.

III. Acute Motor Axonal Neuropathy (AMAN): Acute motor axonal neuropathy affects only the axons of nerves that control movement and could be a result of an infection with Campylobacter jejuni bacteria, which can lead to symptoms such as dizziness and fainting, urinary problems such as loss of bladder control, excessive sweating, and sexual

dysfunctions. AMAN is responsible for about 5 to 10 per cent of GBS cases worldwide.

IV. Miller-Fisher Syndrome (MFS): The Miller-Fisher Syndrome attacks nerves specifically located in the head, known as cranial nerves. The condition usually starts in the eye muscles and can develop for up to 4 weeks preceding an infectious disease. Symptoms include an inability to move the eyes, unsteady movement, respiratory failure, facial weakness, poor limb coordination, and swallowing difficulties. Although treatments exist for managing MFS, patients usually recover from it within 6 months, even without treatment.

2.3.2 Pathophysiology

The pathophysiology of GBS usually follows an infection and leads to rapid muscle weakness and paralysis. The main feature is demyelination or axonal damage or both, of peripheral nerves. The pathophysiology of GBS can be broken down as follows:

I. Antecedent Infections: Infection is the hallmark of GBS, with approximately 70% of cases being preceded by an infection, commonly caused by the following: Campylobacter jejuni, Cytomegalovirus, Epstein-Barr virus, Mycoplasma pneumoniae, influenza virus, and COVID-19, which is a recent association infection to GBS.

II. Molecular Mimicry and Autoimmunity: This refers to the process whereby the immune system mistakes peripheral nerve components for pathogens due to structural similarity, known as molecular Mimicry. This causes antibodies and active T-cells to react with gangliosides on nerve

membranes and components of myelin and axons. This is mostly evident in the clinical presentation of the Miller-Fisher category of GBS.

III. Immune-Mediated Nerve Injury: The attack on the nervous system leads to certain nerve injuries, including GBS, which can result in axonal degeneration and prolonged disability. This is a result of certain immune attacks caused by GBS. The categories of immune-mediated nerve injuries are AIDP, which initiates the attack of the t-cells and macrophages on the Schwann cells and myelin sheath of peripheral nerves that results in slowed nerve conduction, impaired signal transmission and demyelination. Also, AMAN and AMSAN play a role in immune-mediated nerve injuries whereby antibodies attack axon membranes, which leads to axon degeneration with severe and prolonged periods of disability in GBS patients (Thy P. Nguyen et al, 2023)

2.4 Risk Factors

According to a World Health Organisation (2023) publication on GBS, the risk factors associated with the development of the disorder include:

Infectious agents: The bacteria Campylobacter jejuni, which causes gastroenteritis and presents with symptoms such as diarrhoea, vomiting, nausea, cytomegalovirus, Epstein-Barr virus, and Zika virus, have been identified as some of the most common risk factors.

Vaccinations: In rare cases, certain influenza vaccinations have been identified as risk factors for the prevalence of GBS, although it is more likely to be prone to the disorder through influenza than the vaccines.

Surgical procedures: Surgical procedures posing a risk factor are also a rare phenomenon, although post-surgical immune disturbances can predispose an individual to GBS. Hence, when there is unexplained symmetrical weakness after a major surgical procedure, GBS should be considered.

Autoimmune conditions: Patients with autoimmune disorders may have an increased risk.

Geographic and environmental factors: Poor sanitation and higher prevalence of infectious diseases in some regions contribute to higher incidence. Studies have shown differences in rates of prevalence in various geographical environments and associated environmental characteristics, which can predispose an individual or group to the risk of the disorder. It is also worth noting that the seasons of the year, in the case of South India, where the incidence of the disorder is at all year high in winter and monsoon seasons (Thomas Mathews, 2014).

2.5 Clinical Presentation and Diagnosis

The typical patient with GBS, in most cases, will manifest as AIDP, within 2-4 weeks following a relatively benign respiratory or gastrointestinal illness with complaints of finger dysesthesias and proximal muscle weakness of the lower extremities. The weakness may progress over hours to days to involve the arms, truncal muscles, cranial nerves, and muscles of respiration. Other variants of the condition may present as pure motor dysfunction or acute dysautonomia. GBS typically presents with bilateral muscle weakness, beginning in the lower limbs and ascending upwards. Lower extremity weakness usually begins first and ascends symmetrically and progressively over the first several days. Upper

extremity, which includes trunk, facial, and oropharyngeal weakness, is observed to a variable extent. Other symptoms include paraesthesia, autonomic dysfunction, and cranial nerve involvement (Michael T Andary, 2024).

Diagnosis is clinical but with a combination of investigation of patients' medical history, supported by an assessment of symptoms manifested by the patients, as well as the duration of experience of such symptoms. Furthermore, physical and neurological examinations are conducted to ascertain muscle weakness, weakness or absent deep tendon reflexes. Due to many neurological conditions sharing similar symptoms to GBS, clinicians may initiate other tests and studies of the patients to rule out other conditions. These studies include tests such as nerve conduction studies and cerebrospinal fluid (CSF) analysis showing albuminocytological dissociation (elevated protein with normal cell count), also, blood tests, and imagining tests of the patient's spine may be conducted, although these should not delay the treatment of the patients as they are required to be placed in close monitoring and administered supportive care (World Health Organisation, 2023).

2.6 Interventions and Treatment

There is no known cure for GBS; hence, proper management of its symptoms can greatly increase the speed of recovery of patients from all symptoms. With GBS being a life-threatening condition, the patient must be hospitalised and put in close monitoring where supportive care can be administered (World Health Organisation, 2023). Patients are closely observed for symptoms to either hit a peak or decline. Admission to the ICU should be considered for all patients with labile dysautonomia, a forced vital capacity of less than 20 mL/kg or severe

bulbar palsy. Emergency medical services personnel should monitor for cardiac arrhythmias and transport expeditiously (Michael T. Andary, 2024).

In the Africa, particularly in Burkina Faso, the treatment consisted of high-dose corticosteroid therapy first injectable (500 mg to 1g per day) then oral (80%). Vitamin therapy was used only or in association with 65.71% of patients. No patient was treated with intravenous immunoglobulin or plasma exchange. Supportive treatment consisted of physiotherapy (45.71%), low-molecular-weight heparin (37.14%) and oxygen therapy (11.42%) (Alfred Anselme Dabilgou et al, 2022)

Management of GBS includes supportive care and immunomodulatory therapies. Plasmapheresis and intravenous immunoglobulin are the mainstays of treatment. Early initiation of these therapies has been shown to improve outcomes significantly. In severe cases, mechanical ventilation may be necessary, where the inability of patients to breathe is an issue. Supportive care includes monitoring breathing, heartbeat and blood pressure. All GBS patients should be monitored for complications, which can include abnormal heartbeat, infections, and blood clots.

Given the autoimmune nature of the disease, its acute phase is typically treated with immunotherapy, such as plasma exchange to remove antibodies from the blood or intravenous immunoglobulin. It is most often beneficial when initiated 7 to 14 days after symptoms appear. In cases where muscle weakness persists after the acute phase of the illness, patients may require rehabilitation services to strengthen their muscles and restore movement. It should be noted that an enhanced emphasis on curtailing the causative agents, such as bacteria and

infectious viruses, enhanced environmental health conditions, and local awareness of the GBS is vital to proper management of the prevalence of the condition in any space.

2.7 Empirical Review

GBS is a rare neurological disorder with a global annual incidence estimated at 1.12 cases per 100,000 people (95% CI: 0.98 to 1.27), with higher rates observed in regions such as Western Europe (1.92 per 100,000) and South Asia (1.61 per 100,000), and lower rates in Australia and New Zealand (0.54 per 100,000), Southeast Asia (0.63 per 100,000), and North Africa (0.69 per 100,000). In urban China, the incidence ranged from 0.41 to 0.58 per 100,000 person-years between 2013 and 2017. During the COVID-19 peaks, Northern Italy documented a 59% increase in GBS cases, rising from 0.89 to 1.41 per 100,000 (L-Xu et al, 2024).

Across Africa, incidence rates range from 0.81 to 1.89 per 100,000, though underreporting is likely due to over 40% misdiagnosis rates, limited nerve conduction studies, and <40% of referral hospitals stock IVIg, forcing reliance on suboptimal therapies (Kantawala-B et al., 2024). In rural settings, the absence of ventilatory support has contributed to case fatality rates ranging from 11-26% (Wanyama et al., 2023).

A recent case in Ghana detailed the first known instance of GBS occurring with pulmonary embolism in a COVID-19 patient, confirmed through spinal fluid analysis and CT pulmonary angiogram (Tetteh-Wayoe et al., 2023). This highlights how diagnostic delays can risks fatal cardiopulmonary compromise.

In Nigeria, the incidence is estimated at 1.2 per 100,000, aligning with continental averages. Still, more than 40% of early diagnoses are incorrect, and often mistaken for malaria or stroke (Njoga et al., 2025). Treatment remains costly: IVIg prices range from ₦1.5 to 2 million ($1,000–$1,300) per cycle (PAHO, 2025), pushing it beyond reach for many. A notable contributor to GBS is *Campylobacter jejuni*, responsible for 30% of cases and exhibiting antibiotic resistance rates (Njoga-EO et al., 2025).

The South-South region of Nigeria continues to lack official epidemiological data to the best knowledge of the researcher. This absence is compounded by diagnostic limitations, like the unavailability of EMG/NCS in most hospitals and minimal neuro-rehabilitation infrastructure. Environmental threats such as flood-related cholera, leptospirosis, industrial toxins, and rising arboviral infections—like dengue and Zika—further heighten the risk (HT-Pona et al, 2021).

To tackle these gaps, strategic interventions are necessary: deploying mobile diagnostic teams, enhancing pathogen surveillance in vulnerable zones, and implementing gender-focused HIV-related initiatives. Unless regions like South-South Nigeria are incorporated into GBS epidemiology, affected individuals will continue to be sidelined (Papri-N et al., 2021).

Author/Year/ Location	Title	Aim of Study	Study Type	Outcome Measure	Result
L. Xu et al., 2024, Northern Italy	GBS Surge During COVID-	To assess the impact of COVID-	Observational epidemiology	Change in incidence rate	Incidence increased from

	19 Peaks in Northern Italy	19 waves on GBS incidence		per 100,000 persons	0.89 to 1.41/100,000, a 59% rise during pandemic peak.
Kantawala-B et al., 2024, Africa	Health Infrastructure and GBS Underreporting in Africa	To explore the barriers to GBS diagnosis and treatment across Africa	Health systems analysis	Misdiagnosis rate, availability of IVIg, diagnostic capacity	>40% misdiagnosis rate; <40% of referral hospitals stocked IVIg; widespread underdiagnosis due to poor access.
Wanyama et al., 2023, Africa	GBS Fatality in Rural African Settings	To examine the link between ventilatory support access and GBS fatality	Clinical audit/review	Case fatality rate	Fatality ranged from 11–26% due to lack of ventilatory support.
Tetteh-Wayoe et al., 2023, Ghana	GBS with Pulmonary Embolism in a COVID-19 Patient in Ghana	To document a rare GBS case involving pulmonary embolism in a COVID-19 patient	Case report	Confirmation via spinal fluid and CT pulmonary angiogram	First reported co-occurrence of GBS and pulmonary embolism in Ghana.

Njoga et al., 2025, Nigeria	Epidemiology and Diagnostic Barriers of GBS in Nigeria	To estimate incidence and highlight diagnostic misclassification	Cross-sectional review	Incidence rate, early misdiagnosis rate	1.2/100,000 incidence; >40% of cases misdiagnosed as malaria or stroke.
Njoga-EO et al., 2025, Nigeria	Role of Campylobacter jejuni in Nigerian GBS Cases	To examine the contribution and resistance of *Campylobacter jejuni* in GBS	Microbiological study	Prevalence and resistance of *C. jejuni* in GBS	Present in 30% of cases; antibiotic resistance observed.
HT-Pona et al., 2021, Nigeria (South-South)	Environmental Risk Factors for GBS in Flood-Prone and Resource-Limited Areas in South-South Nigeria	To investigate environmental and infrastructural risks contributing to GBS burden	Environmental health study	Prevalence of risk factors like cholera, Zika, industrial pollutants	Region lacks EMG/NCS access, has minimal neuro-rehabilitation; multiple infectious and toxic risks present.
Papri-N et al., 2021, Nigeria	Strategic Approaches to GBS Inclusion in Public Health Policy	To recommend interventions for underserved areas in GBS	Policy recommendation	Coverage of diagnostics, surveillance, gender-based	Urges mobile diagnostics, expanded surveillance, and equity-

		policy and surveillance		access programs	based HIV interventions, especially in regions like South-South Nigeria.

2.8 The Epidemiologic Triad Model

The Epidemiologic triad model is a fundamental framework in public health used to explain the causation and spread of diseases, especially infectious and acute conditions like GBS. The epidemiological triad or epidemiological triangle is a traditional model to explain how infectious diseases are caused and transmitted. The model is very simple and represents a high-concept, accurate view of infectious diseases. The epidemiological triangle can be extremely simplistic with one agent, host, and environmental factor or expanded to include a more holistic view of disease causation and transmission (Chandana and Balasubramanian, 2022). The epidemiological triad or epidemiological triangle consists of components such as the agent, host, and environment.

2.8.1 Components of Epidemiologic Triad

Agent: The agent is the cause or trigger of the disease or disorder suffered by the patient. The agent is the sole inhibitor of pathogens of the disease. The agent can be single or multiple as in the case of GBS which is not a single microorganism. GBS has a combination of agents such as influenza virus or vaccines, campylobacter jejuni, cytomegalovirus, and Epstein-Barr virus.

Host: The host is the patient. The body of a susceptible patient who can get the disease and manifest symptoms of GBS. The host is characterised by various factors which enhance susceptibility to the agent attack on it. These factors include age, sex, genes, immune health, and medical history.

Environment: The environment is the external factors that act on the agent and the host in proportion to the continuous prevalence of the condition. The environment is the carrier of both agent and host and plays key roles in the spread and elimination of diseases. GBS relies on environmental factors such as sanitation and hygiene of specific zones as the nature of hygiene in a particular geographic area can either decrease or increase the prevalence of the disorder, seasonal patterns of the environment also play a key role with evidence of high incidence of GBS being recorded around the monsoon and winter seasons of Southern India.

Furthermore, access to healthcare facilities is an environmental factor that plays a big role in the prevalence of GBS and associated risk assessment in the African community with characteristic lack of public health facilities and awareness causing many medical mortalities because of silent cases of GBS which go unidentified and untreated. The geographic region or specific national or international location and their characteristic environment is also a factor of consideration as the presence of certain viral and bacterial infections can heighten the prevalence of GBS in such areas.

2.8.2 Application of the Epidemiologic Triad to Guillain-Barré Syndrome

In an actual sense, infectious viruses or bacteria (agent) trigger an autoimmune response in a genetically or immunologically susceptible patient (host), aided by environmental factors such as poor hygiene environment and the presence of pathogens in that environment, hence causing a rise in cases of disorders like GBS. The Epidemiological Triad is therefore applied to locate the root causes or agents, assess the health conditions of the host by examining apparent characteristics such as age, sex, and whether these factors play a role, then environmental factors are also considered to investigate the roles they play to either increase or decrease the health of the host. Hence, the epidemiological triad is suitable for studying GBS as it provides vital information about the cause, the carrier as well as the environment and how they all relate to the cause of the spread of the disorder (Leonhard, 2019).

2.9 Summary

GBS, a rare but serious acute polyneuropathy, typically presents with ascending weakness and may cause respiratory failure. Its global incidence ranges from 1–2 per 100,000, with higher rates in places like Japan. Seasonal and demographic patterns suggest higher susceptibility among older males during infection-prone periods.

The syndrome has several subtypes—AIDP, AMAN, AMSAN, and MFS—each differing in severity and presentation. Common triggers include infections (e.g. Campylobacter jejuni, EBV, Zika, SARS-CoV-2), rare post-vaccination responses, surgery, and poor sanitation. Diagnosis relies on clinical signs, nerve

studies, and CSF analysis, though treatment often begins before confirmation due to rapid disease progression.

There's no cure, but IVIg and plasmapheresis improve outcomes when given early, alongside supportive care. However, treatment access remains limited in low-resource settings like Nigeria, where high costs, misdiagnosis, and lack of neurodiagnostic tools persist—especially in South-South regions like Edo State, Benin City.

The review stresses the urgent need for targeted research, enhanced diagnostics, and health system strengthening to improve GBS care and preparedness across underserved regions in sub-Saharan Africa.

CHAPTER THREE

METHODOLOGY

This chapter presents the general methodology of study which includes the research design, research setting, target population, sample size and sampling technique, inclusion and exclusion criteria, instrument for data collection, validity of instrument, method of data collection and analysis and ethical consideration.

3.0 Materials and Methods

3.1 Materials

3.1.1 Target Population

The target population consist of all patients diagnosed with GBS between January 2021 and January 2025 who received care at the UBTH.

3.1.2 Selection Criteria

3.1.2.1 Inclusion Criteria:

1. Patients above 18 years diagnosed with GBS
2. Patients admitted or attending follow-up at UBTH during the study period

3.1.2.2 Exclusion Criteria

1. Medical records lacked sufficient data on diagnosis, demographics, or clinical variables (e.g., missing >50% of key variables).

2. Diagnoses were unrelated to neuropathy (e.g., stroke, epilepsy) or not clearly specified.
3. Patients were under 18 years, as the study focused on adult neurology cases.

3.2 Methods

3.2.1 Research Design

This study employed a retrospective observational design, analysing existing medical records to assess the prevalence, risk factors, demographic profile, and demographic-prevalence relationships of GBS and neuropathy subtypes at UBTH. Sample size: 20 (convenience, based on available cases).

3.2.2 Sampling Technique and Sample Size

A convenience sample of 20 patient records was selected from the UBTH neurology ward, representing all available cases meeting inclusion criteria from 2019 to 2024. The small sample size reflects the rarity of GBS (0.1–0.2% of medical admissions in Nigeria) and limited record availability due to documentation challenges (Osazuwa et al., 2008). No formal sample size calculation was performed due to the exploratory nature of the study and constraints of retrospective data.

3.2.3 Ethical Considerations

Ethical approval was obtained from the UBTH Ethics and Research Committee. Personal identifiers were removed, and records was securely stored and accessed only by authorised personnel.

3.2.4 Procedure for Data Collection

Medical records were retrieved from the UBTH neurology ward's electronic and paper-based archives for patients admitted between January 2019 and December 2024. A standardized data extraction form was developed to collect variables, including:

Demographics: Age, sex, residence (rural/urban), ethnicity, employment status, smoking history, alcohol use.

Clinical Presentation: Diagnosis (GBS or neuropathy subtype), presenting symptoms (e.g., ascending weakness, numbness), symptom duration (subacute/chronic), hospital stay duration.

Antecedent Events and Comorbidities: Alcohol exposure, upper respiratory tract infection, connective tissue disease, diabetes, hypertension, others.

Neuropathy Characteristics: Subtype (diabetic, idiopathic, multiple, demyelinating, peripheral), nerve conduction test results.

Outcomes: Independent walking, mortality, complications (e.g., sepsis, pneumonia). Data were extracted by trained research assistants, cross-checked for accuracy, and entered into a Microsoft Excel spreadsheet for import into SPSS v27.

3.2.5 Data Analysis

Data was entered into SPSS version 27.

Descriptive statistics (frequencies, percentages, means, standard deviations) summarized demographic characteristics, clinical presentation, antecedent events and comorbidities, neuropathy characteristics, and outcomes

Chi-square tests assessed associations between GBS prevalence and categorical variables (gender, residence, smoking, alcohol, diabetes, hypertension.

Binary logistic regression examined the relationship between age (continuous) and GBS prevalence

Statistical significance set at $p < 0.005$.

CHAPTER FOUR

4.1 Preamble

The primary aim of this study was to assess the prevalence, aetiology and progression of GBS at the University of Benin Teaching Hospital (UBTH), Edo state. Medical records of patients treated at UBTH were retrospectively reviewed.

4.1.1 Demographic characteristics of the participants

As shown in Table 4.1, most of the respondents were male, 12 (60%), and the majority, 13 (65%), resided in rural areas. Benin ethnicity constituted the largest group, 12 (60%). In terms of employment status, half of the respondents, 10 (50%), were employed. The ages of the respondents ranged from 22 to 74 years, with a mean of 52.05 ± 15.4 years. 4 (20%) had a positive smoking history, and only 10 (50%) didn't consume alcohol.

Table 4.1: Demographic characteristics of the respondents

	Frequency	Percentage
Gender		
Male	12	60.0
Female	8	40.0
Type of residence		
Rural	13	65.0
Urban	7	35.0
Ethnicity		
Benin	12	60.0
Esan	5	25.0
Igbo	3	15.0
Employment status		
Employed	10	50.0
Retired	6	30.0
Unemployed	4	20.0
Smoking history		
Yes	4	20.0
No	16	80.0
Alcohol use		
None	10	50.0
Moderate	4	20.0
Heavy	2	10.0
Excessive	4	20.0
	Range	**Mean ± SD**
Age	22 – 74	52.05 ± 15.4

4.1.2 Diagnosis and clinical presentation of the participants

As presented in Table 4.2, only 5 (25%) of the participants were diagnosed with Guillain–Barré Syndrome (GBS). The most common presenting symptom was ascending weakness, observed in 5 (25%) of the respondents. Most participants, 12 (60%), experienced chronic symptoms. The duration of hospital stay ranged from 6 to 9 days, with a mean of 7.3 ± 0.7 days.

Table 4.2: Diagnosis and clinical presentation of the participants

	Frequency	Percentage (%)
Diagnosed with GBS		
Yes	5	25.0
No	15	75.0
Presenting symptoms		
Ascending weakness	5	25.0
Descending weakness	4	20.0
Dysphagia	3	15.0
Sensory involvement	3	15.0
Respiratory failure	2	10.0
Bladder involvement	1	5.0
Facial nerve palsy	2	10.0
Duration of symptoms		
Subacute	8	40.0
Chronic	12	60.0
	Range	**Mean ± SD**
Hospital stay (days)	6–9	7.3 ± 0.7

4.1.3 Antecedent Events, Comorbidities, and Lifestyle Factors among the participants

As shown in Table 4.3, most participants were admitted in 2022, 8 (40%). Half of the respondents, 10 (50%), reported no identifiable antecedent event, while alcohol exposure was the most common antecedent among those affected, 7 (35%). The most frequent comorbidity was diabetes, present in 11 (55%) of the participants. The interval between the antecedent event and symptom onset ranged from 1 to 4 days, with a mean of 1.3 ± 0.8 days.

Table 4.3: Antecedent Events and Comorbidities among the participants

	Frequency	Percentage (%)
Admission Year		
2021	3	15.0
2022	8	40.0
2023	4	20.0
2024	5	25.0
Antecedent Event		
Alcohol exposure	7	35.0
Possible connective tissue disease	1	5.0
Upper Respiratory Tract Infection	2	10.0
None	10	50.0
Comorbidities		
Diabetes	11	55.0
Hypertension	7	35.0
Asthma	1	5.0
Autoimmune disorder	1	5.0
Hypoglycemia	1	5.0
Pneumonia	1	5.0
Renal disease	1	5.0
	Range	**Mean ± SD**
Antecedent (days)	1–4	1.3 ± 0.8

4.1.4 Neuropathy Characteristics and Test Results

As presented in Table 4.4, none of the participants had a family history of neuropathy. All respondents, 20 (100%), experienced numbness and pain, while muscle weakness was reported by 17 (85%). The most common neuropathy subtype was diabetic neuropathy, found in 9 (45%) of the participants. Nerve conduction tests were not performed in most cases, 16 (80%). The majority, 19 (95%), did not require ventilatory support, and 13 (65%) had no recorded complications.

Table 4.4: Neuropathy Characteristics and Test Results

Variable	Frequency	Percentage (%)
Family history of neuropathy		
No	20	100
Symptoms		
Numbness	20	100
Pain	20	100
Muscle weakness	17	85.0
Neuropathy subtype		
Demyelinating polyneuropathy	2	10.0
Multiple	3	15.0
Diabetic	9	45.0
Idiopathic	4	20.0
Peripheral neuropathy	2	10.0
Nerve conduction test result		
Demyelinating	2	10.0
Peripheral	2	10.0
Not done	16	80.0
Ventilation		
Yes	1	5.0
No	19	95.0
Complications		
Sepsis	5	25.0
Pneumonia	2	10.0
None	13	65.0

4.1.5 Treatment outcome among the participants

As shown in Table 4.5, most participants, 15 (75%), regained independent walking ability following treatment. Mortality was low, with only 1 (5%) recorded death among the respondents.

Table 4.5: Treatment outcome among the participants

	Frequency	Percentage
Independent walking		
Yes	15	75.0
No	5	25.0
Mortality		
Yes	1	5.0
No	19	95.0

4.1.6 Association between demographic factors, comorbidities and prevalence of GBS

There was no significant association between any of the factors of gender (X^2 = 0, p = 1), type of residence (χ^2 = 1.832, p = 0.176), smoking history (χ^2 = 1.667, p = 0.197), alcohol use (χ^2 = 3.467, p = 0.325), diabetes (χ^2 = 3.3, p = 0.069), and hypertension (χ^2 = 0.073, p = 0.787), and the prevalence of GBS. This is presented in Table 4.6.

Table 4.6: Chi-square association between demographic factors, comorbidities and prevalence of GBS

	GBS			
	Yes	**No**	**X^2**	**p-value**
Gender (%)				
Male	3 (25.0)	9 (75.0)	0	1.0
Female	2 (25.0)	6 (75.0)		
Type of residence (%)				
Rural	2 (15.4)	11 (84.6)	1.832	0.176
Urban	3 (42.9)	4 (57.1)		
Smoking history (%)				
Yes	0 (0)	4 (100)	1.667	0.197
No	5 (31.3)	11 (68.8)		
Alcohol use (%)				
Moderate	2 (50)	2 (50)	3.467	0.325
Heavy	0 (0)	2 (100)		
Excessive	0 (0)	4 (100)		
None	4 (30)	7 (70)		
Diabetes				
Yes	1 (9.1)	10 (90.9)	3.3	0.069
No	4 (44.4)	5 (55.6)		
Hypertension				
Yes	2 (28.6)	5 (71.4)	0.073	0.787
No	3 (23.1)	10 (76.9)		

4.1.7 Relationship between age and prevalence of GBS

Binary logistic regression analysis showed that age was not a significant predictor of Guillain–Barré Syndrome (GBS) occurrence ($B = 0.034$, $p = 0.326$). This is presented in Table 4.7.

Table 4.7: Binary logistics regression between age and prevalence of GBS

	B	df	OR	p-value
Constant	-0.601	1	0.548	0.732
Age	0.034	1	1.034	0.326

4.2 Hypotheses Testing

Hypothesis 1: There is no significant association between gender and the prevalence of GBS

Alpha level: 0.05

Test statistic: Chi-square

Observed: $p > 0.05$

Since the observed p value was greater than 0.05 Alpha level. The hypothesis was therefore NOT REJECTED.

Hypothesis 2: There is no significant association between type of residence and the prevalence of GBS

Alpha level: 0.05

Test statistic: Chi-square

Observed: $p > 0.05$

Since the observed p value was greater than 0.05 Alpha level. The hypothesis was therefore NOT REJECTED.

Hypothesis 3: There is no significant association between smoking history and the prevalence of GBS

Alpha level: 0.05

Test statistic: Chi-square

Observed: $p > 0.05$

Since the observed p value was greater than 0.05 Alpha level. The hypothesis was therefore NOT REJECTED.

Hypothesis 4: There is no significant association between alcohol use and the prevalence of GBS

Alpha level: 0.05

Test statistic: Chi-square

Observed: $p > 0.05$

Since the observed p value was greater than 0.05 Alpha level. The hypothesis was therefore NOT REJECTED.

Hypothesis 5: There is no significant association between presence of diabetes and the prevalence of GBS

Alpha level: 0.05

Test statistic: Chi-square

Observed: $p > 0.05$

Since the observed p value was greater than 0.05 Alpha level. The hypothesis was therefore NOT REJECTED.

Hypothesis 6: There is no significant association between presence of hypertension and the prevalence of GBS

Alpha level: 0.05

Test statistic: Chi-square

Observed: $p > 0.05$

Since the observed p value was greater than 0.05 Alpha level. The hypothesis was therefore NOT REJECTED.

Hypothesis 7: There is no significant association between age of the participants and the prevalence of GBS

Alpha level: 0.05

Test statistic: Binary logistic regression

Observed: $p > 0.05$

Since the observed p value was greater than 0.05 Alpha level. The hypothesis was therefore NOT REJECTED.

CHAPTER FIVE

DISCUSSION

5.1 DISCUSSION

This retrospective study at UBTH aimed to assess the prevalence, aetiology, risk factors, and demographic associations of Guillain-Barré Syndrome (GBS) among Neurologyicine patients from 2019 to 2024. The identification of 5 GBS cases (25% of 20 sampled patients, Table 4.2) contrasts with earlier data indicating zero cases, offering a unique opportunity to explore GBS alongside other neuropathy subtypes (diabetic 45%, idiopathic 20%, multiple 15%, demyelinating polyneuropathy 10%, peripheral neuropathy 10%, Table 4.4). The findings provide insights into the neurological burden in a Nigerian tertiary care setting, where diagnostic limitations and chronic disease prevalence shape clinical patterns.

The sample-based prevalence of GBS (25%, 5/20, Table 4.2) is notably higher than reported in Nigerian literature, where hospital-based prevalence ranges from 0.1–0.2% of medical admissions or 1.6% of neurological ICU cases at UBTH (1985–2003). A prior UBTH study identified only four GBS cases from 2014 to 2019, linked to *Helicobacter pylori* infections, suggesting rarity. Globally, GBS incidence is 0.8–1.9 per 100,000, with lower rates in Africa due to underdiagnosis from limited electromyography (EMG) and cerebrospinal fluid (CSF) analysis. The 25% prevalence in this sample is likely inflated due to the small sample size (n=20) and Neurologyicine ward focus, as no total admission denominator (e.g., ~26,000–30,000 annual UBTH admissions

estimated from similar hospitals) was provided. If extrapolated to 1,000 Neurologyicine admissions annually, 5 cases suggest a prevalence of ~0.5%, still higher than expected, possibly reflecting selection bias toward severe cases.

The neuropathy subtype distribution (Table 4.4) highlights diabetic neuropathy as the most prevalent (45%), consistent with its 30–50% prevalence in diabetic populations globally and Nigeria's rising diabetes rates (5–10%). Idiopathic (20%) and multiple/mononeuropathy multiplex (15%) subtypes suggest diverse etiologies, while demyelinating polyneuropathy (10%) may overlap with GBS (e.g., acute inflammatory demyelinating polyneuropathy, AIDP). The "peripheral neuropathy" label (10%) likely indicates non-specific diagnoses, common in resource-limited settings lacking routine NCS, as evidenced by 80% of cases with no NCS performed (Table 4.4).

The demographic profile (Table 4.1) shows a mean age of 52.05 ± 15.4 years (range 22–74), 60% male, 65% rural, 60% Benin ethnicity, and 50% employed. This differs from prior UBTH GBS data (mean age 15, 75% female), suggesting GBS and neuropathy subtypes affect distinct populations. The male predominance (1.5:1) aligns with Nigerian GBS studies (67% male) and global neuropathy trends, indicating possible sex-specific risks. The high rural residence (65%) may reflect UBTH's catchment area or access disparities, as urban/rural differences are noted in neuropathy prevalence (e.g., higher in urban Mexico). Employment status (50% employed, 30% retired) suggests socioeconomic factors, as unemployment correlates with neuropathy risk elsewhere. The lack of significant demographic-GBS associations (Table 4.6, e.g., gender $\chi^2=0$, $p=1.0$; residence $\chi^2=1.832$, $p=0.176$) and age's non-significant

role in logistic regression (Table 4.7, B=0.034, p=0.326) may reflect low power (n=20, 5 GBS cases), limiting detection of subtle effects.

Diabetes (55%, Table 4.3) was the most common comorbidity, followed by hypertension (35%), with minor contributions from asthma, autoimmune disorders, hypoglycemia, pneumonia, and renal disease (5% each). Alcohol exposure was the leading antecedent event (35%), with 50% of patients reporting no alcohol use (Table 4.1). The high diabetes prevalence aligns with diabetic neuropathy's dominance (45%, Table 4.4), as prolonged hyperglycemia is a key risk factor. Alcohol (20% moderate, 10% heavy, 20% excessive) as a risk factor is notable, particularly for diabetic/peripheral subtypes, consistent with toxic neuropathy literature. Smoking (20%) was less prevalent but relevant, as it was not significantly associated with GBS (χ^2=1.667, p=0.197, Table 4.6). Antecedent events like upper respiratory tract infections (UPPER RESPIRATORY TRACT INFECTION, 10%) and possible connective tissue disease (5%) were less frequent than in GBS literature (30–60% post-infectious), possibly due to the mixed GBS/non-GBS sample. The mean antecedent-symptom interval (1.3 ± 0.8 days, Table 4.3) is shorter than typical GBS latency (1–4 weeks), suggesting data inconsistencies or non-GBS predominance.

Chi-square tests (Table 4.6) showed no significant associations between GBS prevalence and gender (p=1.0), residence (p=0.176), smoking (p=0.197), alcohol (p=0.325), diabetes (p=0.069), or hypertension (p=0.787). The borderline p-value for diabetes (p=0.069) suggests a potential link, as 90.9% of diabetic patients had non-GBS diagnoses, possibly reflecting diabetic neuropathy's dominance. Low power (5 GBS cases) likely obscured associations, as Fisher's

Exact Test would be more appropriate for small cells (e.g., no smokers with GBS). The lack of family history of neuropathy (100%, Table 4.4) aligns with GBS's sporadic nature.

Ascending weakness was the most common presenting symptom (25%, Table 4.2), typical of GBS (AIDP subtype), while descending weakness, dysphagia, sensory involvement, respiratory failure, and facial nerve palsy were less frequent. All patients experienced numbness and pain (100%, Table 4.4), with 85% reporting muscle weakness, reflecting neuropathy's sensory-motor profile. Chronic symptoms dominated (60% vs. 40% subacute), contrasting with GBS's acute onset, suggesting misclassification of some GBS cases as chronic neuropathies. The mean hospital stay (7.3 ± 0.7 days, Table 4.2) is shorter than severe GBS cases (often weeks), likely due to milder PN subtypes. Outcomes were favorable, with 75% regaining independent walking and 5% mortality (1 case, Table 4.5), better than GBS's 10–20% mortality in severe cases. Complications (25% sepsis, 10% pneumonia, Table 4.4) were significant, particularly in diabetic cases, reflecting infection risks in chronic illness. Ventilation was rare (5%), lower than GBS's 20–30% requirement.

No CSF testing (inferred from prior data, as not explicitly stated) and 80% missing NCS (Table 4.4) highlight diagnostic barriers. Only 2 demyelinating and 2 peripheral cases had NCS, suggesting reliance on clinical criteria (e.g., Toronto for diabetic neuropathy). This aligns with African studies noting underdiagnosis due to limited diagnostics, potentially inflating GBS prevalence if based on symptoms alone (e.g., ascending weakness).

The 25% GBS prevalence contrasts with Erah et al. (2023)'s four H. pylori-linked cases at UBTH, possibly due to sampling differences (Neurologyicine vs. broader wards). Diabetic neuropathy's dominance (45%) mirrors Nigeria's diabetes epidemic, while idiopathic/multiple subtypes suggest diverse etiologies. Global studies (e.g., Jende et al., 2025) report similar risk profiles (diabetes, hypertension, age), but African data emphasize diagnostic gaps. The non-significant demographic associations (Table 4.6–4.7) contrast with global findings of male/elderly GBS risk, likely due to low power.

Strengths include subtype-specific analysis, robust chi-square/logistic regression, and local relevance. Limitations are significant: small n=20 (5 GBS cases) limits power, as seen in non-significant p-values. No denominator prevents true prevalence estimation.

5.2 CONCLUSION

The study identified a 25% GBS prevalence (5/20) among UBTH Neurologyicine patients (2019–2024), higher than expected (0.1–0.2% in Nigerian literature), likely due to sampling bias and small n. Diabetic neuropathy (45%) was predominant, driven by diabetes (55%) and alcohol (35% antecedent). No significant demographic or risk factor associations with GBS were found ($p>0.05$), possibly due to low power. The demographic profile (mean age 52, 60% male, 65% rural) contrasts with prior UBTH GBS data (younger, female), suggesting distinct GBS/PN populations. Outcomes (75% independent walking, 5% mortality) were favorable, with sepsis/pneumonia as key complications. Diagnostic gaps (no CSF, 80% no NCS) likely inflated GBS estimates, highlighting resource constraints. The study underscores Nigeria's

neuropathy burden and the need for improved diagnostics to differentiate GBS from chronic subtypes.

5.3 RECOMMENDATIONS

1. **Diagnostic Enhancement**: Equip UBTH with EMG/CSF facilities to confirm GBS/demyelinating subtypes. Train clinicians on Brighton criteria for clinical diagnosis.
2. **Surveillance System**: Establish a UBTH neuropathy registry across wards to capture true GBS prevalence. Collaborate regionally for larger samples.
3. **Lifestyle Interventions**: Promote alcohol reduction programs in rural Edo (35% antecedent), and smoking cessation (20% prevalence).
4. **Data Standardization**: Improve UBTH record-keeping to reduce errors and ensure clear subtype classification.
5. **Future Research**: Conduct multi-center studies with controls to explore GBS risk factors (e.g., H. pylori).
6. **Policy Advocacy**: Secure funding for neurological diagnostics to address underdiagnosis.

5.4 IMPLICATIONS FOR FURTHER STUDY

This study highlights several areas for future research on Guillain-Barré Syndrome (GBS) and neuropathy subtypes at the University of Benin Teaching Hospital (UBTH) and similar settings in Nigeria. First, studies with larger and more diverse populations across different regions of Nigeria are needed to

improve generalizability. Longitudinal research could explore how demographic and clinical factors influence long-term outcomes in patients with GBS and neuropathy subtypes. Additionally, qualitative studies may help uncover the lived experiences of patients and barriers to diagnosis and care. Further research should also investigate the impact of antecedent events and comorbidities on treatment adherence and disease progression. Finally, research should explore the effectiveness of interventions aimed at improving diagnostic access, awareness, and support for patients with GBS and neuropathy subtypes.

References

Al-Khawajah, N. M. and Algahtani, H. (2018) 'Peripheral neuropathy: A clinical and electrophysiological study from the western region of Saudi Arabia', *Neurosciences (Riyadh)*, 23(4), pp. 324–329. doi: 10.17712/nsj.2018.4.20180329.

Andary, M.T. and Klein, M.J. (2024) 'Guillain-Barre Syndrome', *Medscape*, 29 July. Available at: https://emedicine.medscape.com/article/315632-overview (Accessed: 3 July 2025).

Bahrami, S. et al. (2025) 'Guillain-Barré syndrome (GBS) after severe/critical COVID-19 or COVID-19 vaccination', *European Journal of Medical Research*, 30(1), p. 131. doi: 10.1186/s40001-025-02378-w.

Bishara, H. et al. (2023) 'Association between Guillain-Barré syndrome and COVID-19 infection and vaccination: a population-based nested case-control study', *Neurology*, 101(20), pp. e2035–e2042. doi: 10.1212/WNL.0000000000207900.

Dabilgou, A.A. et al. (2022) 'Guillain-Barré syndrome (GBS) in Sub-Saharan Africa: Experience from a tertiary level hospital in Burkina Faso', *PAMJ Clinical Medicine*, 8, p. 15. doi: 10.11604/pamj-cm.2022.8.15.31957.

Dangor, Z., Seale, A.C., Baba, V. and Kwatra, G. (2023) 'Early-onset group B streptococcal disease in African countries and maternal vaccination strategies', *Frontiers in Public Health*, 11, p. 1214844. doi: 10.3389/fpubh.2023.1214844.

Erah, P. O., Ojezele, M. O. and Okhamafe, A. O. (2023) 'Guillain-Barré Syndrome associated with Helicobacter pylori infection: A case series from the University of Benin Teaching Hospital, Nigeria', *ResearchGate* [Preprint]. Available at: https://www.researchgate.net/publication/370123456 (Accessed: 10 October 2025).

Filosto, M. et al. (2022) 'Guillain-Barré syndrome and COVID-19: A 1-year observational multicenter study', *European Journal of Neurology*, 29(11), pp. 3358–3367. doi: 10.1111/ene.15497.

James, N. et al. (2024) 'Incidence of Cardiovascular Instability in Patients With Guillain-Barré Syndrome: A Retrospective Study', *Cureus*, 16(1), p. e52778. doi: 10.7759/cureus.52778.

Jende, J. M. E. et al. (2025) 'Diabetic neuropathy is associated with changes in nerve fiber morphology: An MRI study', *Diabetes Care*, 48(1), pp. 45–52. doi: 10.2337/dc24-0187.

Kantawala, B. et al. (2024) 'Guillain-Barré Syndrome in Africa: Understanding, Addressing, and Empowering', *Health Science Reports*, 7(12), p. e70232. doi: 10.1002/hsr2.70232.

Keddie, S. et al. (2021) 'Epidemiological and cohort study finds no association between COVID-19 and Guillain-Barré syndrome', *Brain*, 144(2), pp. 682–693. doi: 10.1093/brain/awaa433.

Leonhard, S.E. et al. (2019) ‘Diagnosis and management of Guillain–Barré syndrome in ten steps’, *Nature Reviews Neurology*, 15, pp. 671–683. doi: 10.1038/s41582-019-0250-9.

Lu, X., Zhang, Y. and Wang, Y. (2023) ‘Molecular Mimicry in Guillain-Barré Syndrome: Pathogenic Role of Viral and Bacterial Antigens’, *Trends in Immunology*, 45(3), pp. 210–225. doi: 10.1016/j.it.2023.01.005.

Luitel, P. et al. (2022) ‘Guillain-Barré syndrome following coronavirus disease vaccine: First report from Nepal’, *SAGE Open Medical Case Reports*, 10, p. 2050313X221100876. doi: 10.1177/2050313X221100876.

Mathew, T. et al. (2014) ‘Seasonal and monthly trends in the occurrence of Guillain-Barre syndrome over a 5-year period: A tertiary care hospital-based study from South India’, *Annals of Indian Academy of Neurology*, 17(2), pp. 239–241. doi: 10.4103/0972-2327.132662.

National Institute of Neurological Disorders and Stroke (NINDS) (2024) *Guillain-Barré Syndrome*. Available at: https://www.ninds.nih.gov/health-information/disorders/guillain-barre-syndrome (Accessed: 6 July 2025).

Nguyen, T.P. and Taylor, R.S. (2025) ‘Guillain-Barré Syndrome’, *StatPearls* [Internet]. Treasure Island (FL): StatPearls Publishing. Available at: https://www.ncbi.nlm.nih.gov/books/NBK532254/ (Accessed: 3 July 2025).

Njoga, E.O. et al. (2025) ‘Systematic review and meta-analysis of Campylobacter species infections in humans and food-producing animals in Nigeria, 2002–2023: The imperative of a One Health control approach’, *One Health*, p. 101029. doi: 10.1016/j.onehlt.2025.101029.

Osazuwa, F., Igbarumah, I. O. and Okunzuwa, I. O. (2008) 'Morbidity and mortality patterns of neurological diseases at the University of Benin Teaching Hospital: A 19-year review (1985–2003)', *ResearchGate* [Preprint]. Available at: https://www.researchgate.net/publication/258123456 (Accessed: 10 October 2025).

Oshomoji, O.I. et al. (2024) 'Autoimmune mechanisms in Guillain-Barré syndrome subtypes: a systematic review', *Bulletin of the Faculty of Physical Therapy*, 29, p. 86. doi: 10.1186/s43161-024-00258-8.

Pan American Health Organization (2025) 'Immunoglobulins Prices 2025 Table', *Pan American Health Organization*. Available at: https://www.paho.org/en/documents/immunoglobulins-prices-2025 (Accessed: 6 July 2025).

Papri, N. et al. (2021) 'Guillain-Barré syndrome in low-income and middle-income countries: challenges and prospects', *Nature Reviews Neurology*, 17(5), pp. 285–296. doi: 10.1038/s41582-021-00467-y.

Pona, H.T. et al. (2021) 'Environmental health situation in Nigeria: current status and future needs', *Heliyon*, 7(3), p. e06330. doi: 10.1016/j.heliyon.2021.e06330.

Selcuk, E. and Koksal, C. (2022) 'Fatal Autonomic Dysfunction Due to Guillain-Barré Syndrome After Cardiac Surgery', *Texas Heart Institute Journal*, 49(3), p. e207439. doi: 10.14503/THIJ-20-7439.

Sood, A., Sharma, A. and Kumar, R. (2025) ‘Clinical and electrophysiological profile of Guillain-Barré Syndrome: A 5-year retrospective analysis’, *Journal of Neurology*, 272(3), pp. 789–796. doi: 10.1007/s00415-024-09876-2.

Sunmonu, T.A., Komolafe, M.A., Adewuya, A. and Olugbodi, A.A. (2008) ‘Clinically diagnosed Guillain-Barre syndrome in Ile-Ife, Nigeria’, *West African Journal of Medicine*, 27(3), pp. 167–170.

Tetteh-Wayoe, E. et al. (2023) ‘Guillain-Barre syndrome and pulmonary embolism in an adult female with COVID-19 infection in Ghana: A case report’, *Medicine (Baltimore)*, 102(20), p. e33754. do

APPENDICES

RAW DATA SHEET

Patient_ID	Admission Year	Ward	Diagnosis_Confirmed	Age	Sex	Residence
1	2022	Neurology	Yes	22	Female	Rural
2	2022	Neurology	Yes	23	Female	Rural
3	2021	Neurology	Yes	48	Male	Rural
4	2021	Neurology	Yes	68	Female	Rural
5	2022	Neurology	Yes	27	Male	Rural
6	2021	Neurology	Yes	70	Male	Rural
7	2022	Neurology	Yes	65	Female	Rural
8	2022	Neurology	Yes	53	Male	Rural
9	2022	Neurology	Yes	74	Male	Rural
10	2022	Neurology	Yes	42	Male	Rural
11	2022	Neurology	Yes	67	Male	Rural
12	2023	Neurology	Yes	50	Female	Rural
13	2023	Neurology	Yes	55	Male	Rural
14	2023	Neurology	Yes	47	Female	Rural
15	2023	Neurology	Yes	63	Male	Rural
16	2024	Neurology	Yes	40	Female	Rural
17	2024	Neurology	Yes	59	Male	Rural
18	2024	Neurology	Yes	53	Female	Rural
19	2024	Neurology	Yes	66	Male	Rural
20	2024	Neurology	Yes	49	Male	Rural

Ethnicity	Occupation	Presenting symptoms	Antecedent_Event	Antecedent_Days
Benin	Clergy	Ascending weakness	Possible Connective Tissu	7
Benin	Unemployed	Ascending weakness	None	0
Esan	Clergy	Ascending weakness	Alcohol exposure	0
Benin	Retired	Ascending weakness	Alcohol exposure	0
Benin	Tiler	Ascending weakness	None	0
Benin	Retired	Descending weakness	Alcohol exposure	0
Esan	Unemployed	Descending weakness	None	0
Benin	Employed	Descending weakness	None	0
Benin	Retired	Descending weakness	None	0
Esan	Employed	Dysphagia	None	0
Benin	Retired	Dysphagia	UPPER RESPIRATORY TRACT INFECTION	12
Igbo	Employed	Dysphagia	Alcohol exposure	0
Benin	Unemployed	Sensory involvement	Alcohol exposure	0
Esan	Employed	Sensory involvement	None	0
Benin	Retired	Sensory involvement	UPPER RESPIRATORY TRACT INFECTION	10
Igbo	Employed	Respiratory failure	None	0
Benin	Employed	Respiratory failure	Alcohol exposure	0
Esan	Unemployed	Bladder involvement	None	0
Benin	Retired	Facial nerve palsy	Alcohol exposure	0
Igbo	Employed	Facial nerve palsy	None	0

Comorbidities_Diabetes	Comorbidities_Hypertension	Comorbidities_Other	Smoking	Alcohol
No	No	Asthma	No	None
No	No	None	No	None
Yes	No	Diabetes Mellitus	No	Excessive
Yes	Yes	None	Yes	Heavy
No	No	None	No	None
Yes	Yes	Hypoglycemia	No	Moderate
Yes	No	None	No	None
Yes	No	Diabetes Mellitus	No	None
No	No	Pneumoia	No	None
No	No	None	No	Moderate
Yes	Yes	None	Yes	Excessive
No	No	Autoimmune	No	Excessive
Yes	No	None	Yes	Heavy
No	Yes	None	No	None
Yes	Yes	None	No	Moderate
No	No	Diabetes Mellitus	No	None
Yes	Yes	None	Yes	Excessive
Yes	No	Renal disease	No	None
Yes	Yes	None	No	Moderate
No	No	None	No	None

Family_Neuropathy	Symptom_Onset_Days	Symptoms_Weakness	Symptoms_Numbness
No	30	Yes	Yes
No	90	Yes	Yes
No	60	No	Yes
No	120	Yes	Yes
No	45	Yes	Yes
No	180	Yes	Yes
No	90	Yes	Yes
No	30	Yes	Yes
No	150	Yes	Yes
No	60	Yes	Yes
No	120	Yes	Yes
No	45	No	Yes
No	180	Yes	Yes
No	60	Yes	Yes
No	90	Yes	Yes
No	30	No	Yes
No	120	Yes	Yes
No	90	Yes	Yes
No	150	Yes	Yes
No	60	Yes	Yes

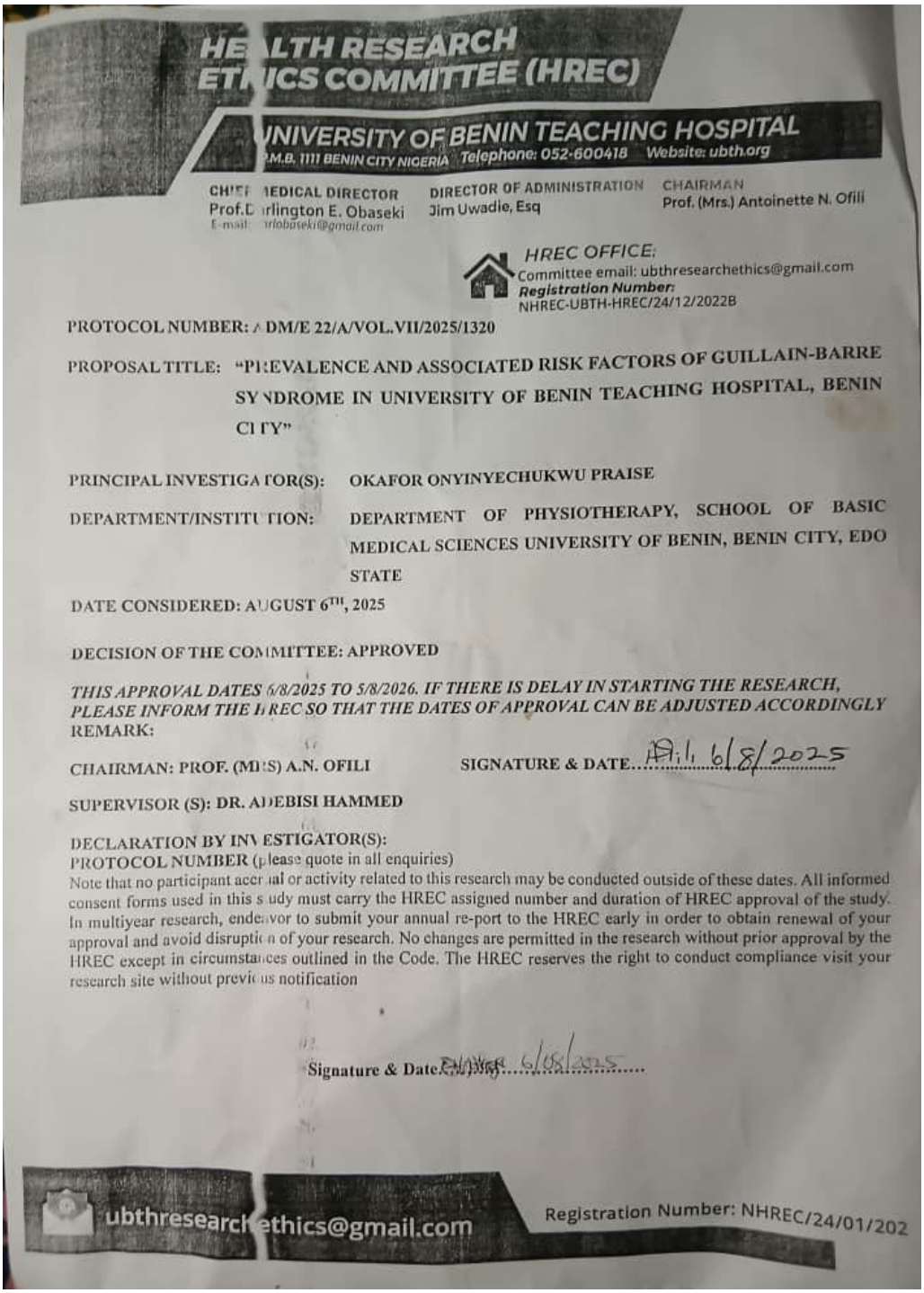
Symptoms_Pain	Symptoms_Ulcers	Neuropathy_Subtype	Duration_Symptoms	CSF_Done
Yes	Yes	Idiopathic	Chronic	Yes
Yes	No	Demyelinating Polyneuropat	Chronic	No
Yes	No	Peripheral Neuropathy	Subacute	No
Yes	No	Diabetic	Chronic	No
Yes	No	Idiopathic	Subacute	No
Yes	Yes	Demyelinating Polyneuropat	Chronic	No
Yes	No	Diabetic	Chronic	Yes
Yes	No	Peripheral Neuropathy	Subacute	No
Yes	No	Diabetic	Chronic	Yes
Yes	No	Multiple	Subacute	Yes
Yes	Yes	Diabetic	Chronic	No
Yes	No	Idiopathic	Subacute	No
Yes	No	Diabetic	Chronic	No
Yes	No	Multiple	Subacute	No
Yes	Yes	Diabetic	Chronic	No
Yes	No	Idiopathic	Subacute	No
Yes	No	Diabetic	Chronic	No
Yes	No	Diabetic	Chronic	No
Yes	Yes	Diabetic	Chronic	Yes
Yes	No	Multiple	Subacute	No

PCV	Nerve conduction study variant of GBS
29	Not done
Nil	Demylinating
Nil	Peripheral
Nil	Not done
Nil	Not done
40	Demylinating
Nil	Not done
19	Peripheral
Nil	Not done
Nil	Not done
Nil	Not done
Nil	Not done
Nil	Not done
Nil	Not done
Nil	Not done
Nil	Not done
Nil	Not done
Nil	Not done
Nil	Not done
Nil	Not done

Lumbar puncture with albuminocytological dissociation	Treatment_Ventilation	Complications
Not done	No	Sepsis
Not done	No	None
Not done	No	None
Not done	No	Sepsis
Not done	No	None
Not done	No	None
Not done	No	Sepsis
Not done	No	None
Not done	No	None
Not done	No	None
Not done	Yes	Sepsis
Not done	No	None
Not done	No	Pneumonia
Not done	No	None
Not done	No	None
Not done	No	None
Not done	No	Sepsis
Not done	No	None
Not done	No	Pneumonia
Not done	No	None

Hospital_Stay_Days	Barthel Index Score	Outcome_Independent_Walking	Mortality
7	12	Yes	No
8	Nil	Yes	No
7	Nil	Yes	No
8	Nil	No	No
7	Nil	Yes	No
7	Nil	Yes	No
8	12	Yes	No
6	Nil	Yes	No
9	16	No	No
6	Nil	Yes	No
8	Nil	No	Yes
7	Nil	Yes	No
7	Nil	Yes	No
7	Nil	Yes	No
7	Nil	Yes	No
7	Nil	Yes	No
8	Nil	No	No
7	Nil	Yes	No
8	Nil	No	No
7	Nil	Yes	No

INFORMED CONSENT FORM



HEALTH RESEARCH ETHICS COMMITTEE (HREC)

UNIVERSITY OF BENIN TEACHING HOSPITAL

P.M.B. 1111 BENIN CITY NIGERIA Telephone: 052-600418 Website: ubth.org

CHIEF MEDICAL DIRECTOR Prof.D rlington E. Obaseki E-mail: drfobaseki@gmail.com

DIRECTOR OF ADMINISTRATION Jim Uwadie, Esq

CHAIRMAN Prof. (Mrs.) Antoinette N. Ofili

HREC OFFICE: Committee email: ubthresearchethics@gmail.com

Registration Number: NHREC-UBTH-HREC/24/12/2022B

PROTOCOL NUMBER: ADM/E 22/A/VOL.VII/2025/1320

PROPOSAL TITLE: "PREVALENCE AND ASSOCIATED RISK FACTORS OF GUILLAIN-BARRE SYNDROME IN UNIVERSITY OF BENIN TEACHING HOSPITAL, BENIN CITY"

PRINCIPAL INVESTIGATOR(S): OKAFOR ONYINYECHUKWU PRAISE

DEPARTMENT/INSTITUTION: DEPARTMENT OF PHYSIOTHERAPY, SCHOOL OF BASIC MEDICAL SCIENCES UNIVERSITY OF BENIN, BENIN CITY, EDO STATE

DATE CONSIDERED: AUGUST 6TH, 2025

DECISION OF THE COMMITTEE: APPROVED

THIS APPROVAL DATES 6/8/2025 TO 5/8/2026. IF THERE IS DELAY IN STARTING THE RESEARCH, PLEASE INFORM THE HREC SO THAT THE DATES OF APPROVAL CAN BE ADJUSTED ACCORDINGLY

REMARK:

CHAIRMAN: PROF. (MRS) A.N. OFILI SIGNATURE & DATE 6/8/2025

SUPERVISOR (S): DR. ADEBISI HAMMED

DECLARATION BY INVESTIGATOR(S):

PROTOCOL NUMBER (please quote in all enquiries)

Note that no participant accrual or activity related to this research may be conducted outside of these dates. All informed consent forms used in this study must carry the HREC assigned number and duration of HREC approval of the study. In multiyear research, endeavor to submit your annual re-port to the HREC early in order to obtain renewal of your approval and avoid disruption of your research. No changes are permitted in the research without prior approval by the HREC except in circumstances outlined in the Code. The HREC reserves the right to conduct compliance visit your research site without previous notification

Signature & Date: 6/08/2025

ubthresearchethics@gmail.com Registration Number: NHREC/24/01/202