

Clinical and Evaluative Profile of Guillain-Barré Syndrome in Burkina Faso: Retrospective Study of 49 Patients Collected in 16 Years (2003-2018)

Djingri Labodi Lompo^{1,*}, Raphael M P Kaboré¹, Adja Mariam Ouédraogo², Kadari Cissé², Adama Ramdé¹, Christian Napon³

¹Tingandogo University Hospital, Health Sciences Training and Research Unit, Joseph Ki-Zerbo University of Ouagadougou, Ouagadougou, Burkina Faso

²Health Sciences Research Institute, Department of Medical Biology and Public Health of Ouagadougou, Ouagadougou, Burkina Faso

³Bogodogo University Hospital, Health Sciences Training and Research Unit, Joseph Ki-Zerbo University of Ouagadougou, Ouagadougou, Burkina Faso

Email address:

labodilompo@yahoo.fr (Djingri Labodi Lompo), raphkabore@yahoo.fr (Raphael M P kaboré),

adjamariam@yahoo.fr (Adja Mariam Ouédraogo), cisskad4@yahoo.fr (Kadari Cissé), adamsramde69@gmail.com (Adama Ramdé),

naponc@yahoo.fr (Christian Napon)

*Corresponding author

To cite this article:

Djingri Labodi Lompo, Raphael M P kaboré, Adja Mariam Ouédraogo, Kadari Cissé, Adama Ramdé, Christian Napon. Clinical and Evaluative Profile of Guillain-Barré Syndrome in Burkina Faso: Retrospective Study of 49 Patients Collected in 16 Years (2003-2018). *Clinical Neurology and Neuroscience*. Vol. 7, No. 3, 2023, pp. 56-64. doi: 10.11648/j.cnn.20230703.13

Received: September 24, 2023; **Accepted:** October 17, 2023; **Published:** November 9, 2023

Abstract: *Introduction:* The aim of our study was to describe the epidemiological, paraclinical and clinical profile of Guillain-Barré syndrome (GBS) in the University Hospitals of Ouagadougou, Burkina Faso. *Patients and methods:* This was a retrospective, multicenter, descriptive study of the records of patients hospitalized for GBS in 3 Ouagadougou University Hospitals for a period of 16 years, from March 2003 to May 2018. Included in the study were the records of patients aged ≥ 16 years who were hospitalized for GBS during the study period, according to the modified Brighton diagnostic criteria. Patients who were HIV-positive or had cytorachy > 10 elements/mm³ were not included. Socio-demographic, climatic, clinical variables, Electroneuromyography (ENMG) data, cerebro spinal fluid (CSF) examination and evolution were studied. The intra-hospital clinical course of patients was assessed according to the Guillain-Barré Disability Scale (GBDS). *Results:* A total of 49 cases of GBS were hospitalized. The average age was 36 years, with a predominance of females (51%) and during the cold dry season (40.8%). In the state phase, all patients had a motor deficit of all 4 limbs and 15 patients (30.6%) had dysautonomia. The mean durations of the extension and plateau phases were 10 days and 19 days respectively. At the ENMG, the axonal form (71.4%) predominated. Pulmonary infection (36.7%), exacerbation of hypertension (32.6%) and electrolyte disorders (23%) were the most frequently encountered intra-hospital complications. Intra-hospital mortality was 18.4%. Among the survivors, 30% were confined to a wheelchair with or without respiratory assistance. After univariate analysis, intra-hospital infectious complications ($p=0.04$), exposure to mechanical ventilation ($p=0.05$) and severe clinical presentations ($p=0.005$) were the variables significantly influencing intra-hospital mortality. *Conclusion:* GBS affects more often young patients, occurs more frequently in the cold dry season. It is characterized by a late hospitalization of the patients, a predominant axonal damage. Early admission of patients, early use of quality intensive care units, availability of Polyvalent Intravenous immunoglobulin and plasma exchange, will significantly improve the prognosis of GBS in Burkina Faso and Sub-Saharan Africa (SSA).

Keywords: Guillain-Barré Syndrome, Dysautonomia, Cold Dry Season, Mortality, Burkina Faso

1. Introduction

Guillain-Barré syndrome (GBS) is an acute polyneuropathy with a variable degree of weakness that reaches. The disease is mostly preceded by an infection, following a bacterial or viral infection, mainly by *Campylobacter jejuni*, Cytomegalovirus, Epstein-Barr Virus or *Mycoplasma pneumoniae*. It is manifested by rapidly progressive, bilateral, relatively symmetrical paralysis of all four limbs, with or without damage to the respiratory muscles or muscles innervated by the cranial nerves, hypo or areflexia of the tendons, frequent albuminocytological dissociation of the cerebrospinal fluid (CSF) [1]. The natural history of GBS includes an extension phase lasting less than 4 weeks, a plateau phase of variable duration, from a few days to several weeks or months, and a recovery phase, also of variable duration. GBS remains a serious disease due to the respiratory, dysautonomic and thromboembolic complications that can occur [2].

The average incidence of the pathology is estimated between 1 and 2 cases/100,000 inhabitants/year. GBS is present in all regions of the world, with a discrete male predominance and affects all ages with 2 peaks in frequency, one at 15-34 years and the other at 50-74 years [3]. A seasonal distribution with winter and spring predominance of cases has been frequently reported [4-6].

The spectrum of GBS is quite heterogeneous and includes acute inflammatory demyelinating polyradiculoneuropathy (AIDP), predominant in Europe and North America [3, 7], acute motor axonal neuropathy (AMAN) and acute sensory-motor axonal neuropathy (ASMAN) predominant in Asia, South and Central America [8] and Miller-Fisher syndrome (MFS) and other overlapping syndromes [1].

In high-income countries, therapeutic advances combining multivalent immunoglobulin (IVIg), plasma exchange (PE) and resuscitation measures have greatly improved the prognosis of GBS [3]. However, GBS is still a serious disease: about 25% of patients temporarily require artificial ventilation, about 20% of patients are still unable to walk after 6 months and 3 to 10% of patients die [1]. In low-income countries, due

to the insufficiency or unavailability of IVIg, PE and adequate intensive care or resuscitation units, the vital and functional prognosis is often still poor, with mortality rates sometimes reaching 20% in India [9] and up to 25.9% in Ethiopia [10]. In Africa, few recent studies have been devoted to GBS [10, 11] and seem to indicate a similar epidemiological, clinical and paraclinical profile with the rest of the world, except for a younger age of patients and a poorer vital prognosis.

In a context of a low income, Sahelian climate, marked by low availability and poor accessibility of therapeutics of proven efficacy on the disease, insufficient and/or low quality intensive care and resuscitation units, the present study was initiated in order to contribute to a better knowledge of GBS, and then to help improve the prognosis of this disease, in Sub-Saharan Africa (SSA).

The objectives of this work were to describe the clinical and evolutionary profile of GBS in Burkina Faso through a retrospective, multicentre, hospital-based study based on the files of patients hospitalized in the university hospitals of Ouagadougou, Burkina Faso, and to identify the factors influencing intra-hospital mortality.

2. Patients and Methods

This was a cross-sectional study retrospective, multicenter, descriptive and analytical of the records of patients hospitalized for GBS in the 4 University Hospitals of Ouagadougou, Yalgado Ouédraogo, Tingandogo, Bogodogo and Pediatric Charles de Gaulle, in Burkina Faso, over a 16-year period from March 2003 to May 2018. Included in the study were the records of patients, regardless of age or sex, who were hospitalized in the so-called CHU during the study period for GBS according to the modified Brighton criteria [12]. These criteria are based on clinical manifestations and the results of neurophysiological examinations and lumbar puncture. Patients were placed into two categories including Level 1 (highest degree of diagnostic certainty) and Level 2 (intermediate degree of diagnostic certainty). The modified Brighton criteria used for the diagnosis of the patients in our study are summarized in Table 1 below.

Table 1. Modified Brighton Criteria Used for the Diagnostic Criteria of the Patients in our Study.

Level 1 diagnostic certainty	Level 2 diagnostic certainty
Bilateral and flaccid weakness of limbs Bilateral weakness and limb flabbiness; AND Decreased or absent deep tendon reflex in weak limbs; AND Monophasic course and time between onset-nadir 12 h to 28 days, followed by a clinical plateau; AND Absence of alternative diagnosis for weakness AND Albuminocytologic dissociation (i.e. increase in CSF protein* above normal laboratory values, with CSF white blood cell count <10/μl); AND GBS-compatible electrophysiological analysis results	Bilateral and flaccid weakness of limbs AND Decreased or absent deep tendon reflex in weak limbs; AND Monophasic course and time between onset-nadir 12 h to 28 days, followed by a clinical plateau; AND Absence of alternative diagnosis for weakness AND Total number of white blood cells <10/μl in the CSF (with or without an increase in CSF protein above normal laboratory values); OR GBS-compatible electrophysiological test results if CSF is not collected or results are not available.

The intra-hospital clinical course of patients was assessed according to the Guillain-Barré Disability Scale (GBDS), summarized in Table 2 below.

Table 2. *Guillain-Barre's syndrome disability scale, adapted from Hughes et al. (1978).*

0	A healthy state
1	Minor symptoms and capable of running
2	Able to walk 5 m or more without assistance but unable to run
3	Able to walk 5 m across an open space with help of a person, a walker, or canes
4	Bedridden or chair bound, cannot walk even with an aid.
5	Patient placed on assisted ventilation
6	Dead

Not included in the study were incomplete or unusable records, records of patients who were unable to perform lumbar puncture (LP) or electroencephalogram (ENMG), records of patients who developed fever at the onset of neurological symptoms, records of patients who were HIV-positive, or who had cytorachy > 10 elements/mm³. In addition, the following disease entities were formally excluded on the basis of clinical and sometimes biological criteria: porphyria, botulism, myasthenia gravis, acute anterior poliomyelitis, toxic neuropathies, diphtheria, pure sensory peripheral neurogenic syndromes without motor impairment, periodic dyskaemic paralysis. Data were collected from clinical records of patients, emergency department hospitalization records, neurology, multi-purpose resuscitation, available computer databases. The data were collected on a standardised collection form for all patients, which allowed the study of the following parameters: sociodemographic, climatic, clinical, paraclinical, therapeutic and evolutionary. The following variables were studied: socio-demographic and climatic variables (distribution according to age, sex, university hospital, month, year, season), clinical variables (admission time, mode of installation, severity and distribution of motor deficit, chronology of the different phases of the disease, dysautonomic symptoms), paraclinical variables (cytorachy, proteinachy, albumino-cytological dissociation (ACD),

ENMG data), therapeutic and evolutionary variables (specific immunotherapy, treatment of complications, length of hospitalisation, intra-hospital intercurrent complications, intra-hospital death, becoming functional at the end of hospitalisation).

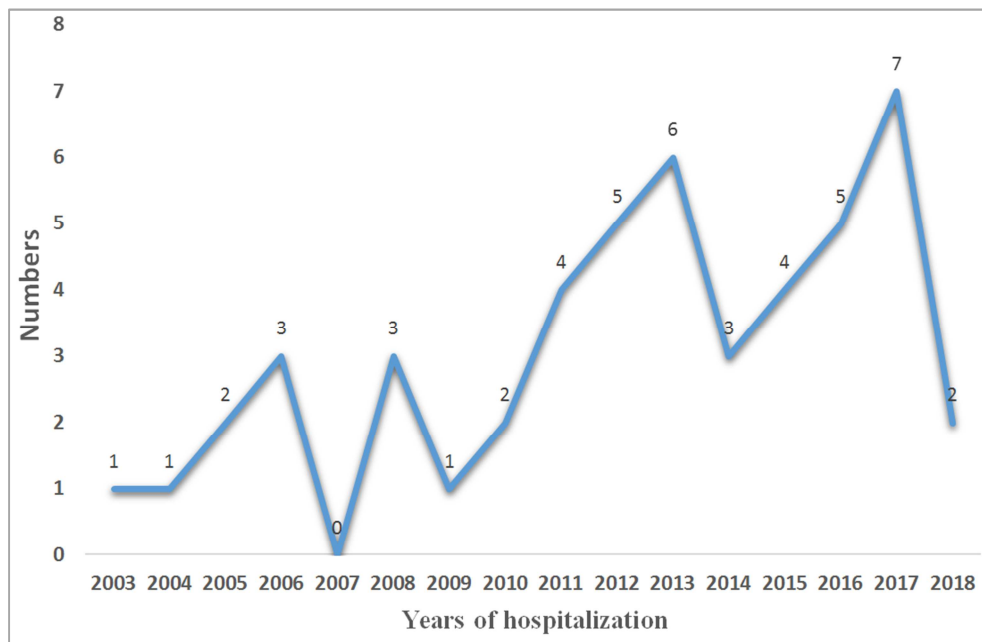
The data were processed by the Epi-info software in its version 7.2.1.0.

In order to identify the factors influencing the intra-hospital mortality of GBS, a bivariate analysis between independent (sociodemographic, climatic, clinical, paraclinical, therapeutic and evolutionary) and dependent (deceased/survivors) variables was performed, using chi-square or Fischer exact tests. The values from $p \leq 0.05$ were considered statistically significant.

The study was carried out after authorization by the Burkina Faso Bioethics Committee and the authorization of the administrations of the various university hospitals. Patient anonymity was respected.

3. Results

A total of 49 cases of GBS were hospitalized in Ouagadougou's university hospitals over a period of 16 years, from March 2003 to May 2018, corresponding to an annual hospitalization frequency of 3.1 cases per year. Figure 1 shows the annual distribution of hospitalized GBS cases.

**Figure 1.** *Distribution of Patients by Year of Hospitalization (n=49).*

The mean age of the patients was 36 \pm 16.4 years (range 15 - 79 years); the age range 15 - 45 years with 38 patients (77.5%) was the most representative. There were 25 females (51%) and 24 males (49%), giving a sex ratio of 0.96. The average age of the patients was 36 years \pm 16.4 years (extremes 15 - 79 years); the age range 15 - 45 years with 38

patients (77.5%) was the most representative. The cumulative monthly frequency of hospitalized GBS cases showed three peaks, in January, May and November with frequencies of 12.2%, 18.4% and 16.3% respectively. Figure 2 shows the cumulative monthly frequency of GBS hospitalized cases over 16 years.

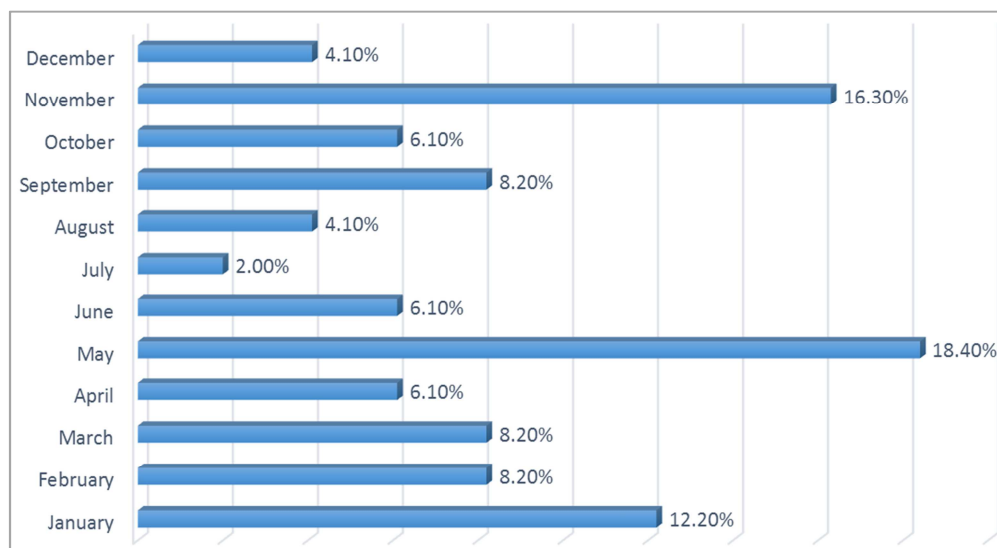


Figure 2. Distribution of patients according to cumulative monthly GBS frequencies over 16 years (n=49).

The seasonal distribution of hospitalized GBS cases according to the 3 seasons of the year, the cold dry season or harmattan (November-February), the hot dry season (March-May), and the rainy season (June-October), showed that GBS occurs throughout the year with a higher frequency of cases during the harmattan (40.8%). Figure 3 shows the seasonal distribution of GBS.

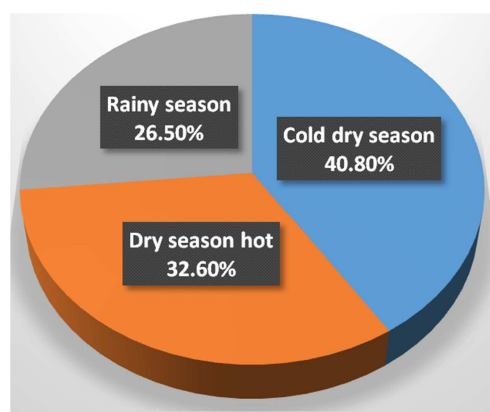


Figure 3. Distribution of GBS patients by season of the year.

The average length of stay in hospital was 16.2 days \pm 7.6 days (range 4 - 32 days). The majority of patients (36.7%) were hospitalized in neurology between the 8th and 14th day and only 12.2% of patients were hospitalized during the first week, following the onset of neurological signs. Table 3 below presents the distribution of patients according to the length of hospital stay.

Table 3. Distribution of patients by length of stay in hospital.

Consultation period (days)	Number	Percentage (%)
≤ 7	6	12.2
[8-14]	18	36.7
[15-21]	10	20.4
≥ 22	15	30.6
Total	49	100.0

Of the 49 patients in our series, 24 (49%) had a history of a recent infectious episode in the two to three weeks prior to the onset of neurological signs. The main infectious events were gastroenteritis with 10 cases (41.7%) and pneumopathy with 6 cases (25%).

Figure 4 below shows the distribution of patients according to infectious events preceding GBS.

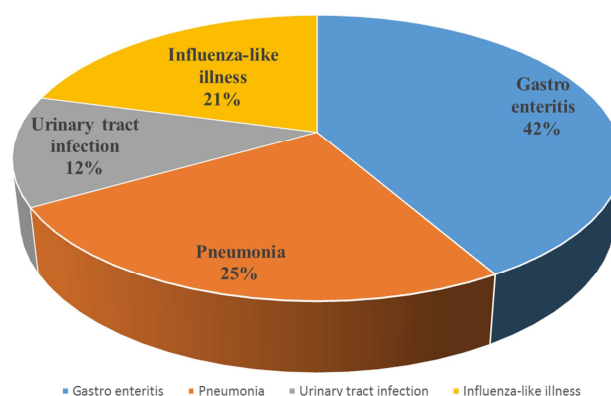


Figure 4. Distribution of patients according to precesive infectious events (n=24).

Pregnancy or postpartum were found as circumstances of discovery in 9 cases (18.4%) and 3 cases (6.1%) respectively.

On admission, all 49 patients (100%) presented with motor deficit of all 4 limbs; tetraparesis, with 22 cases (44.9%), lower limb plegia associated with upper limb paresis with 20 cases (40.8%) and tetraplegia with 7 cases (14.3%) were the main motor deficit profiles of the limbs represented. Cranial nerve damage was noted in 21 patients (42.8%), i.e. facial damage with 14 cases (28.5%), bulbar damage with 12 cases (24.5%) and oculomotor damage with 4 cases (8.2%), isolated or associated.

Osteotendinous reflexes were attenuated or abolished in all our patients, an abolition of osteotendinous reflexes in 46 patients (91.8%) and attenuation in 3 patients (8.2%).

Respiratory distress was present on admission in 9 patients (18.4%).

Dysautonomic disorders were observed in 15 patients (30.6%), mainly represented by a flare-up of hypertension in 8 patients (32.6%), stubborn constipation in 3 patients (18.4%) and cardiac disorders in 6 patients (16.3%). Table 4 below shows the distribution of patients according to dysautonomic disorders.

Table 4. Distribution of Patients by Dysautonomic Disorders (n=15).

Type of dysautonomy	Numbers (n)	Frequency (%)
Pushes of Arterial blood pressure	7	46.7
Cardiac arrhythmia	4	26.6
Hypotension	1	6.7
Persistent constipation	3	20

Type of dysautonomy	Numbers (n)	Frequency (%)
Urine retention	2	13.3
Hypersudation	1	6.7

The mean durations of the extension and plateau phases were 10 days \pm 5.7 (extremes 1 and 21 days) and 19 days \pm 5.9 (extremes 11 and 44 days), respectively.

Lumbar puncture (LP) was performed in all our patients (100%), mostly after the second week (81.6% of cases). Cerebrospinal fluid (CSF) was clear in 46 patients (93.9%). Cytorachy averaged 6 white blood cells (WBCs)/ml (extremes 0 and 42 elements/ml); it was normal (≤ 10 WBCs/ml) in 45 patients (91.8%); it was elevated (between 11 and 42 WBCs/ml) in 4 patients (8.2%). Proteinachy averaged 1.9g/l \pm 1.6g/l (extremes 0.3 and 6.3g/l); albuminocytological dissociation (ACD) was observed in 46 patients (93.9%). Electroneuromyography was performed in only 7 patients (14.3%), demonstrating axonal and demyelinating forms in 5 patients (71.4%) and 2 patients (28.6%), respectively.

Specific treatment with polyvalent intravenous immunoglobulins was administered in only 3 patients (6.1%). No patients received plasmapheresis.

Pulmonary infection with 18 cases (36.7%), an exacerbation of hypertension with 16 cases (32.6%) and electrolyte disorders with 11 cases (23%) were the most frequently encountered intra-hospital complications. Table 5 below summarizes the different intra-hospital complications observed.

Table 5. Distribution of patients according to intra-hospital complications (n=49).

Complications	Numbers (n)	Frequency (%)
Cardiovascular complications		
HTA	16	32.6
Hypotension	6	12.2
Tachycardia	4	8.2
Bradycardia	3	6.1
Cardiac arrhythmia	2	4.1
Infectious complications	33	67.3
Lung infections	18	36.7
Urinary tract infections	6	12.2
Other infections	2	4.1
Respiratory distress	13	26.5
Electrolytic disorders	11	23.0
Undernutrition	5	10.2
Escarres	1	2.1

Of all hospitalized patients, 15 cases (30.6%) were transferred to multi-purpose intensive care unit. The main reasons for transfer to intensive care were respiratory distress in 13 cases (26.5%) and severe dysautonomia in 2 cases (4.1%). Among the patients admitted to resuscitation, 5 patients (33.3%) benefited from mechanical ventilation, 3 patients (20%) from tracheotomy + mechanical ventilation; the 7 other patients (42.7%) only benefited from monitoring.

The average length of hospitalisation was 24 days \pm 19.5 (extremes 5 and 105 days). At the end of hospitalisation, 9 patients died, i.e. an intra-hospital mortality rate of 18.4%. The immediate causes of death were fatal respiratory distress in 3 cases, respiratory infection and/or septic shock in 4 cases,

cardiac arrest of dysautonomic origin in 1 case, and death of undetermined cause in 1 case. According to the GBDS (Guillain-Barré Disability Scale), of all hospitalized patients, 11 patients (22.4%) were able to walk unaided (scores 1 and 2), 17 patients (34.7%) were able to walk with assistance (score 3), 11 patients (22.4%) were confined to bed or chair (score 4) and only one patient (2%) was on respiratory assistance (score 5). Considering only the 40 surviving patients, 27.5% had a GBDS score of 1 & 2; 42.5% had a GBDS score of 3; 27.5% had a GBDS score of 4; and 2.5% had a GBDS score of 5. Figure 5 below summarises the clinical outcome of patients at the end of hospitalisation according to GBDS scores (in grades).

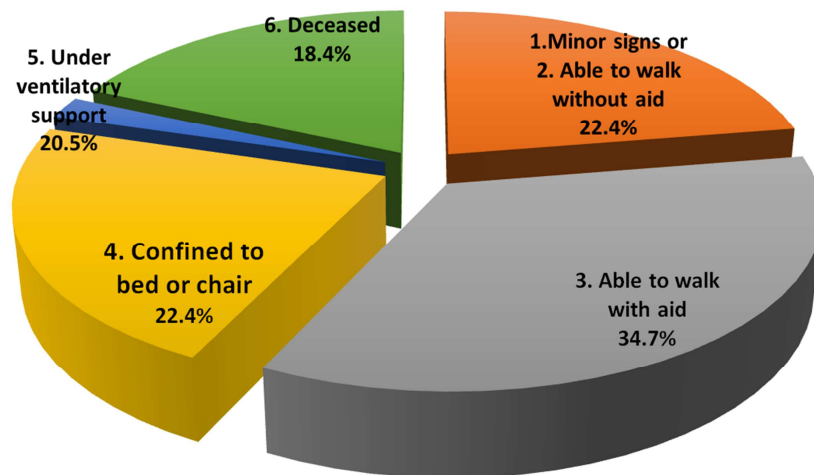


Figure 5. Clinical outcome of patients at the end of hospitalization according to Hughes score (in grade) (n=49).

After univariate analysis, intra-hospital infectious complications ($p=0.04$), exposure to mechanical ventilation ($p=0.05$) and serious clinical presentations on admission ($p=0.005$) were the variables significantly influencing

intra-hospital mortality. The results of the univariate analysis of the variables influencing in-hospital mortality of GBS inpatients are presented in Table 6 below.

Table 6. Results of univariate analysis of variables influencing in-hospital mortality of GBS inpatients.

Factors	Dead	Alive	P-value
Rapidly progressive installation	7 (77.8%)	21 (52.5%)	$p=0.31$
Axial injury at EMG	3 (100.0%)	2 (50.0%)	$p=0.22$
Mechanical ventilation	3 (33.3%)	2 (5.0%)	$p=0.05$
Length of hospital stay ≤ 21 days	6 (66.7%)	20 (50.0%)	$p=0.59$
Age ≤ 25 years old	4 (44.4%)	11 (27.5%)	$p=0.40$
Duration of the extension phase ≤ 14 days	9 (100.0%)	17 (42.5%)	$p=0.005$
Intra-hospital infectious complications	8 (88.9%)	18 (45.0%)	$p=0.04$
Intra-hospital cardiac complications	7 (77.8%)	18 (45.0%)	$p=0.15$

4. Discussion

We recorded a total of 49 hospitalized GBS cases over a 16-year period, with an average annual frequency of 3.1 cases per year, whereas most epidemiological studies find an average annual incidence of 1.3 to 1.5 cases/100,000 population/year [3]. This indicates a probable underestimation of GBS cases in our context. Indeed, the hospitalized nature of our series did not allow us to take into account non-hospitalized cases and cases treated in private clinics in particular.

Our study, with 77.5% of patients under 45 years old and an average age of 36 years, confirms the young age of GBS patients in Africa, already reported in previous studies [10, 11, 13, 14]. In contrast to the African series, patients in the European series, with an average age ranging from 54 to 77 years, are significantly older [2, 15, 16].

A slight peak in frequency observed in older adolescents and young adults corresponding to the age groups from 15 years to under 40 years, and the elderly. An increased risk of cytomegalovirus (CMV) and *Campylobacter jejuni* infections in young people, and an increased susceptibility of the elderly to autoimmune diseases such as GBS due to decreased

immunosuppressive mechanisms, may explain these observations [4, 17].

While male predominance seems to be the rule [9, 14], we paradoxically found a slight female predominance in our series. This can be explained by the 24.5% of GBS cases that occurred in the context of pregnancy and/or childbirth, which are also recognized as triggering factors for the disease [18, 19].

We have noted a significant seasonal variation in GBS, marked by an upsurge of the disease during the harmattan from November to February and the hot dry season from March to May. Almost similar findings have been made in Sweden [20], France [4], Brazil [5] and China [6]. In these series, a higher incidence of GBS cases was observed mainly in winter and spring. The seasonality of GBS revealed by our study could be explained by the increase in respiratory infections due to cold, dry harmattan winds and gastroenteritis during the dry season due to the shortage of drinking water and the ecosystem. These gastroenteritis and respiratory tract infections were the frequent predecessors of GBS in our series. Indeed, in our series, 49% presented an infectious event in the 2 to 3 weeks prior to the installation of GBS. Previous infectious events, similar to ours, have been reported in India [9] and Ethiopia [10] respectively. However, higher

frequencies of history of infectious events have been observed in other series [6, 15, 21], ranging from 65.8% to 80% of cases. We also observed that among the infectious episodes preceding GBS, gastroenteritis (41.7%) was the main infectious event, followed by pneumopathy (25%). Our results are quite similar to those reported in Bangladesh [8, 22] where diarrhoea was found to be the main infectious event in 50% of the cases. The preponderance of gastroenteritis among the precessive infectious episodes in our series could be explained by inadequate hygiene and sanitation in our context.

Electrophysiological studies conducted in Asia, mainly China [23] and Japan [24], and in South and Central America [3, 8, 25] have shown that axonal forms of GBS constituted 30-47% of cases, while in Europe and North America, the demyelinating form (Acute Inflammatory Demyelinating Polyneuropathy), with 69-90% of cases, was predominant [3, 26]. In sub-Saharan Africa (SSA), in a recent Ethiopian study, the demyelinating form was predominant with 55.3% of cases [10], while in our series, where ENMG was performed in only 14.3%, the axonal form was largely predominant with 71.4% of cases. The predominance of the axonal form was also observed in Bangladesh [8], where it accounted for 86% of cases. In the African context, due to the scarcity of studies on GBS in general and electrophysiological examinations in particular, data on the distribution of the different electroclinical forms of GBS are still insufficient.

Progressively, acute respiratory distress (ARD) and dysautonomic disorders are the most severe forms of the disease, as are bulbar damage and decubitus complications [1, 3, 25]. ARD complicates the progression of GBS in 10 to 30% of cases [1, 28] the 26.5% ARD rate reported in our series is part of this framework. However, higher rates of 32.5% have been reported in India [9].

The use of artificial ventilation for ARD is reported on average in 25% of cases in hospital series in developed countries [3, 15, 25], whereas it is reported at lower rates, of the order of 10 to 20%, in series from low-income countries [8, 14], such as ours. This low rate of use of mechanical ventilation, in contrast to the fairly frequent use of ARD in low-income country series such as ours, could be explained by the low accessibility and/or availability of beds for medical resuscitation or neurological intensive care, particularly for GBS patients, at the onset of signs predictive of ARD or life-threatening dysautonomic complications. Factors predictive of ARD during GBS include rapidly progressing clinical forms, bulbar or upper extremity paralysis, and dysautonomia [1, 27]. For example, early transfer to an intensive care or resuscitation unit for prophylactic intubation is indicated at the onset of signs predictive of mechanical ventilation in developed countries [2, 3], whereas in low-income countries, transfer to an ICU or resuscitation is most often delayed at the onset of ARD or irreversible dysautonomia [28].

In the literature, dysautonomic disorders are found in 70% of cases and mainly include urine retention, paralytic ileus, sinus tachycardia, labile arterial hypertension, cardiac arrhythmia, orthostatic hypotension [1, 3]. In the series of

low-income countries, particularly in SSA [10.], India [29] and Brazil [5], lower rates of the order of 30 to 34% are reported, as in our series. Even lower rates of dysautonomy, however, of 8-25% have been reported among children in Bangladesh [8], Aman [30] and Taiwan [31]. The low incidence of dysautonomy reported in these different studies from low income countries may be related to the lack of bedside cardio-pressure and respiratory monitoring of patients most often hospitalized in general neurology wards.

The GBS early mortality rate of 18.4% reported in our series and those of 15.3% and 25.9% reported in SSA, Tanzania [11] and Ethiopia [10], respectively, remain very high compared to mortality rates reported in high-income countries and even some low-income countries. Thus, in Bangladesh, 6-month mortality rates of 12% [21] and 14% [32] respectively were recorded. In India, highly variable 3-month mortality rates of 3.5% [33], 8.2% [29], and up to 20% [9] have been reported. In Brazil [5], a mortality rate of 10% has been reported. In high-income countries, the lethality of GBS patients is lower [34]: in the USA, an in-hospital mortality rate of 2.5% has been found [34]; an average 6-month mortality rate of 6% (0-13%) has been recorded in Asia, i.e. 2%, 3% and 13%, respectively in Singapore, Japan and Hong Kong [16], and 6- and 12-month mortality rates of 2.8% and 3.9% respectively have been recorded in the Netherlands [35]. This favourable development of GBS in these high-income countries is explained by the greater availability of adequate and well-equipped intensive care units, early access of patients at the onset of signs predictive of complications, including ARD, early detection of severe dysautonomic complications by cardiorespiratory monitoring, and the availability of specific therapies of proven efficacy such as Polyvalent intravenous immunoglobulins and plasma exchange [35]. Conversely, in low-income countries such as ours, the still high GBS-related mortality seems to be associated with the non-availability and inaccessibility of neurological intensive care or medical resuscitation units, and the lack of intensive surveillance in general neurology inpatient units [16]. The high intra-hospital mortality rate in our context could also be explained by the non-availability of specific therapies of proven efficacy such as Polyvalent intravenous immunoglobulins or Plasma Exchanges, the high frequency of infectious complications, especially respiratory infections. Indeed, in our context, only 6.1% of patients were able to benefit from polyvalent intravenous immunoglobulins, sometimes at a late stage, due to the expensive nature and under-availability of this drug in our country. Plasma exchanges are not yet available in our country.

The occurrence of infectious complications during hospitalisation ($p=0.04$), exposure to mechanical ventilation ($p=0.05$) and a serious clinical presentation on admission ($p=0.005$) were the variables significantly associated with intra-hospital mortality.

The use of mechanical ventilation had already been

identified as a risk factor for death in GBS patients in several series worldwide [28, 30, 36]. More recently, other series [16, 21, 35] have confirmed that mortality increases significantly with mechanical ventilation. Infectious complications related to ventilation procedures and the severity of ARD may explain the increased mortality. The Asian series of Wong AH *et al.*, like us, also identified respiratory infections as a factor in the death of GBS patients [16].

Similarly, we identified severe clinical presentations on admission (tetraplegia + ARD) as a factor associated with death in our patients. This factor has also been reported in other series [34, 35].

However, other factors associated with frequently reported GBS deaths such as advanced age, a long phase of disease extension (> 7 days) [16, 34, 35, 37, 38], dysautonomy [34, 37] were not identified in our study.

Concerning the functional prognosis of GBS, globally 35% of patients recovered completely, 35% had minimal residual motor signs and 30% had moderate to severe residual paresis. [37]. Six months after the onset of the disease, about 80% of the patients are independent for walking at 6 months [8]. In our study, at the end of hospitalisation, although 72.5% of patients were still dependent (able to walk with assistance, bedridden or intubated), their functional prognosis was nevertheless likely to improve within 6 months to a year.

5. Limitations of Our Study

Due to the retrospective nature of our study, potential biases could be introduced, including missing or biased data.

Due to the hospital-based nature of our study, potential biases in the selection of serious cases and/or cases treated in tertiary hospitals of the public health system could be introduced. It is therefore possible that less serious cases hospitalised in private clinics or followed up on an outpatient basis could have been excluded from the scope of our study.

Our study also suffered from the small number of patients who were able to complete the ENMG and the fact that data from post-hospital follow-up were not available.

6. Conclusion

GBS is a relatively common condition in our context, affecting more often young patients with a predominance of females, occurring more frequently during the cold or hot dry season. It is most often preceded by an infectious episode of acute gastroenteritis. It is characterized by late admission to the tetraparesis phase, fairly frequent dysautonomy, complications such as respiratory distress and pulmonary infection, respectively in $\frac{1}{4}$ and $\frac{1}{3}$ of cases, and axonal damage in about $\frac{3}{4}$ of cases. The low or late use of mechanical ventilation, the unavailability of Polyvalent intravenous immunoglobulins and Plasma exchanges, are responsible for a rather heavy early mortality and a poor early functional prognosis. Early admission of patients, early use of quality intensive care units, availability of Polyvalent intravenous

immunoglobulins and Plasma exchanges, will significantly improve the prognosis of GBS in Burkina Faso and SSA.

Conflicts of Interest

All the authors do not have any possible conflicts of interest.

Disclosures

The manuscript has been read and approved by all the authors.

Acknowledgments

The authors thank the patients who subjected to the study.

References

- [1] van Doorn P A. Diagnosis, treatment and prognosis of Guillain-Barré syndrome (GBS). *Presse Med.* 2013; 42: e193–e201.
- [2] Garric JR, Conard M, Louis G, Bernard D *et al.* Le Syndrome de Guillain Barré en Réanimation: *Réanimation* (2014) 24: S92-S95.
- [3] Hughes RA, Cornblath DR. Guillain-Barré syndrome. *Lancet* 2005; 366: 1653-66.
- [4] Delannoy A, Rudant J, Chaignot C, *et al.* Incidence du syndrome de Guillain Barré en France: une analyse épidémiologique à partir des données du PMSI (2008-2013). *Revue épidémiologique et Santé Publique*; vol 65 N°S1 mars 2017 PS7. (5).
- [5] Pinol-Ripoll G, Larrodé P P, Garcés-Redondo M, & Iñiguez C M. (2008, March). Characteristics of Guillain-Barré syndrome in the healthy area III of Aragon Country. In *Anales de medicina interna* 2008; (Vol. 25, No. 3, pp. 108-112).
- [6] Xu X, Shen D Li T, Zhang B, Mao M *et al.* (2016). Distinct Clinical Characteristics of Pediatrics Guillain Barre Syndrome: A comparative Study between children and adults in Northeast China. *PLoS One* 2016; 11 (3): e0151611.
- [7] Yuki N, Hartung HP. Guillain-Barré syndrome. *N Engl J Med* 2012; 366: 2294.
- [8] Habib R, Saifuddin M, Islam R, Rahman A *et al.* Clinical Profile of Guillain Barré Syndrome-Observations from a tertiary Care Hospital of Bangladesh. *Birdem Med J* 2017; 7 (1): 38-42.
- [9] Dhadke SV, Dhadke VN, Bangar SS, Korade MB. Clinical profile of Guillain Barré syndrome. *J Assoc Physician India* 2013; 61 (3): 168-72.
- [10] Melaku Z, Zenebe G, & Bekele A. Guillain-Barré syndrome in Ethiopian patients. *Ethiopian medical journal* 2005; 43 (1), 21-26.
- [11] Howlett W P, Vedeler CA, Nyland H, & Aarli J A. Guillain-Barré syndrome in northern Tanzania: a comparison of epidemiological and clinical findings with western Norway. *Acta neurologica scandinavica* 1996, 93 (1), 44-49.

- [12] Sejvar JJ, Kohl KS, Gidudu J, Amato A, Bakshi N, Baxter R, Burwen DR, Cornblath DR, Cleerhout J, Edwards KM, Heininger U. Guillain-Barré syndrome and Fisher syndrome: case definitions and guidelines for collection, analysis, and presentation of immunization safety data. *Vaccine*. 2011 Jan 10; 29 (3): 599–612.
- [13] Bahemuka M. Guillain-Barre syndrome in Kenya: a clinical review of 54 patients; *J Neurol* (1988) 235: 418-421.
- [14] Kassimi E H, Abdelfettah Y, Elbouchikhi M, Khadir A, Naitkhchat A, Lmidmani F, & Elfatimi A. Handicap et devenir fonctionnel après un syndrome de Guillain-Barré (expérience du service de Casablanca): à propos de 19 cas. *Annals of Physical and Rehabilitation Medicine* 2013; (56), e17.
- [15] Forsberg A, Press R, de Pedro-Cuesta J, Widéon Holmqvist L. Use of health-care, patient satisfaction and burden of care in Guillain-Barré syndrome *Journal of Rehabilitation Medicine* 2006; 38: 230-236.
- [16] Wong AH, Umapathi T, Shahrizaila N, Chan YC, Fong MK et al. The mortality of comparing mortality of Guillain Barré syndrome across different regions. *J Neurol Sci*. 2014 Sep 15; 344 (1-2): 60-2.
- [17] van KR, van Doorn PA, Schmitz PI, Ang CW, van der Meche FG. Mild forms of Guillain-Barre syndrome in an epidemiologic survey in The Netherlands. *Neurology* 2000 Feb 8; 54 (3): 620-5.
- [18] Chan LY, Tsui MH, Leung TN. Guillain-Barre syndrome in pregnancy. *Acta Obstet Gynecol Scand* 2004 Apr; 83 (4): 319-25.
- [19] Vaduva C, de Seze J, Volatron AC, Stojkovic T, Piechno S, Husson J, et al. Syndrome de Guillain-Barré sévère et grossesse: deux cas d'amélioration rapide en post partum. *Rev Neurol (Paris)* 2006 Mar; 162 (3): 358-62.
- [20] De Pedro Cuesta J, Abaira V, Jiang GX, et al. Guillain Barre syndrome in south-west Stockholm, 1973-1991. 3. Clinicoepidemiological subgroups. *Acta Neurol Scand* 1996; 93: 175-83.
- [21] Ishaque T, Islam M B, Ara G, Endtz H P, Mohammad Q D, Jacobs B C, & Islam Z. High mortality from Guillain-Barré syndrome in Bangladesh. *Journal of the Peripheral Nervous System* 2017, 22 (2), 121-126.
- [22] Islam B, Islam Z, Rahman S, et al. Small volume plasma exchange for Guillain Barré Syndrome in resource-limited setting: a phase II safety and feasibility study. *BMJ Open* 2018; 8: e022862. Doi: 10.1136/bmjopen-2018-022862.
- [23] Ho TW, Mishu B, Li CY, et al. Guillain-Barré syndrome in northern China: relationship to *Campylobacter jejuni* and anti-glycolipid antibodies. *Brain* 1995; 118 (Pt 3): 597-605.
- [24] Ogawara K, Kuwabara S, Mori M, et al. Axonal Guillain-Barré syndrome: relation to anti-ganglioside antibodies and *Campylobacter jejuni* infection in Japan. *Ann Neurol* 2000; 48 (4): 624-31.
- [25] Kuwabara S. Guillain-Barré syndrome: epidemiology, pathophysiology and management. *Drugs* 2004; 64 (6): 597-610.
- [26] Yuki N. Acute motor axonal neuropathy and multifocal motor neuropathy: more in common than not. *Muscle Nerve* 2013; 48: 693-5.
- [27] Durand MC, Porcher R, Orlikowski D, Clair CD, Annane D, JeanLouis Gaillard JL et al. Clinical and electrophysiological predictors of respiratory failure in Guillain-Barre Syndrome: a prospective study. *The Lancet Neurology* 2006; 5: 1021-1028.
- [28] Dhar R, Stitt L, Hahn AF. The morbidity and outcome of patients with Guillain-Barré syndrome admitted to the intensive care unit. *J Neurol Sci*. 2008; 264: 121-8.
- [29] Verma R, Chaudhari T S, Raut T P, & Garg R K. Clinico-electrophysiological profile and predictors of functional outcome in Guillain-Barre syndrome (GBS). *Journal of the neurological sciences* 2013, 335 (1-2), 105-111.
- [30] Roshan K, Al-Futaisi A, Chacko A, Fazalullah M, Al Nabhani S, Al-Awaidy S,... & Al-Mahrooqi S. Clinical Characteristics of Childhood Guillain Barré Syndrome. *Oman Medical journal* July 2008; volume 13, Issue 3.
- [31] Hu M-H, Chen C-M, Lin-Lin K et al. Risk Factors of Respiratory Failure in Children with Guillain-Barré Syndrome: *Pediatrics and neonathology* 2012; 53, 295-299.
- [32] Islam Z, Jacobs B C, van Belkum A, Mohammad Q D, Islam M B, Herbrink P,... & Endtz H P. Axonal variant of Guillain-Barre syndrome associated with *Campylobacter* infection in Bangladesh. *Neurology* 2010, 74 (7), 581-587.
- [33] Kalita J, Misra U K, Goyal G, & Das M. (2014). Guillain-Barré syndrome: subtypes and predictors of outcome from India. *Journal of the Peripheral Nervous System* 2014; 19 (1), 36-43.
- [34] Ashekhlee A, Hussain Z, Sultan B, & Katirji B. Guillain-Barré syndrome: incidence and mortality rates in US hospitals. *Neurology* 2008; 70 (18), 1608-1613.
- [35] Van den Berg B, Bunschoten C, van Doorn PA, Jacobs BC. Mortality in Guillain-Barré syndrome. *Neurology*. 2013; 80: 1650-4.
- [36] Orlikowski D, Sharshar T, Porcher R, Annane D, Raphael JC, Clair B. Prognosis and risk factors of early onset pneumonia in ventilated patients with Guillain-Barré syndrome. *Intensive Care Med*. 2006; 32: 1962-9.
- [37] Netto AB, Taly AB, Kulkarni GB, Uma Maheshwara Rao G S, Rao S. Prognosis of patients with Guillain-Barré syndrome requiring mechanical ventilation. *Neurol India [serial online]* 2011 [cited 2016 Apr 7]; 59: 707-11.
- [38] Domínguez-Moreno R, Tolosa-Tort P, Patiño-Tamez A, Quintero-Bauman A, Collado-Frías D K, Miranda-Rodríguez M G,... & Estañol-Vidal B. Mortalidad asociada al diagnóstico de síndrome de Guillain-Barré en adultos ingresados en instituciones del sistema sanitario mexicano. *Rev Neurol* 2014; 58 (1), 4-10.