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Association of dysautonomia and different risk factors in patients with Guillain-Barré syndrome in a tertiary hospital in the Philippines

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Abstract

Background Guillain-Barré syndrome (GBS) presents with progressive ascending weakness, but it can also present with dysautonomia such as tachycardia, blood pressure fluctuations, diaphoresis, ileus, and urinary retention. GBS patients with dysautonomia was observed to have longer hospital stays and higher mortality rates than those without dysautonomia. We aimed to determine the risk factors for dysautonomia and its manifestations among patients with GBS and compared their features to those without dysautonomia.

Methods We conducted a 10 year-retrospective review of GBS patients admitted at the Philippine General Hospital. The patient demographics, comorbidities, GBS disability status scale (GBS-DS), GBS variants, parameters of dysautonomia, treatment, and outcome were recorded and analyzed. Simple and multiple logistic regression analysis were conducted to determine the factors associated with dysautonomia and the relationships were expressed using odds ratio.

Results 71 patients were included, and 49% developed dysautonomia. Hypertension and tachycardia were the most prominent manifestations. There was an increase in the odds of developing dysautonomia in a one-year increase in age (OR: 1.11, $p=0.001$) and a point increase in GBS-DS (OR:1.65, $p=0.037$) during admission. Pre-morbid hypertension (OR:0.13, $p=0.028$) and alcoholism (OR: 0.17, $p=0.037$) are shown to decrease the odds of developing dysautonomia. Although GBS patients with dysautonomia had longer hospital stay (12.33 days), it only predicts 5.5% of the variability.

Discussions The prevalence of cardiovascular manifestations was postulated from cardiosympathetic hyperactivity between arterial baroreceptors, cardiac parasympathetic fibers, and preganglionic sympathetic vasomotor fibers. The protective mechanism of premorbid hypertension could be attributed to the prior intake of antihypertensive medications, which mitigate cardiosympathetic fluctuations, while the protective effect of alcoholism needs to be further studied.

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Conclusion Patients who are older and with a high GBS-DS on admission, prompt close monitoring for the development of dysautonomia. The protective effects of premorbid hypertension and alcoholism needs further evaluation. The odds of developing pneumonia and being on a mechanical ventilator, while not statistically significant, could contribute to longer hospital stay of patients with dysautonomia. A larger prospective study is warranted to confirm these results.

Keywords Guillain-Barré Syndrome, Dysautonomia, Autoimmune inflammatory demyelinating polyneuropathy, IVIG, Plasmapheresis

Introduction

Guillain-Barré Syndrome (GBS) is the most common cause of acute or subacute generalized paralysis with an estimated global incidence of 0.4–1.7 per 100,000 per year affecting children and adults of all ages and sexes [1]. The major typical clinical manifestation is a progressive, ascending weakness accompanied by variable sensory loss over several days to weeks, which in severe cases, may lead to respiratory failure. GBS can be further subclassified based on the major immunopathology (demyelinating or axonal) as well as clinical presentation, namely, acute inflammatory demyelinating polyneuropathy (AIDP), acute motor axonal neuropathy (AMAN), acute motor-sensory axonal neuropathy (AMSAN), Miller Fisher syndrome (MFS), Bickerstaff brainstemencephalitis, pan dysautonomia, pure sensory and pure motor neuropathies [1].

Dysautonomia, characterized by various aberrancies of normal autonomic function, may occur in approximately 40–66% of patients diagnosed with GBS [2, 3]. These disturbances are typified by sinus tachycardia, bradycardia, facial flushing, blood pressure fluctuations, loss of sweating or diaphoresis, ileus, and urinary retention, among many others [2, 3]. Sympathetic predominance over afferent arterial baroreceptors, efferent cardiac parasympathetic fibers, and preganglionic sympathetic fibers with vasomotor and sudomotor functions has been implicated in its pathogenesis [3]. Another proposed theory posits a catecholamine surge from an afferent peripheral nerve conduction block [2, 3]. While generally transient, published literature on exact duration is sparse. Moreover, several lines of evidence show that subclinical indices of autonomic dysfunction remain detectable on laboratory testing even after the resolution of GBS [3, 4]. Several observational studies especially done by Chakraborty and colleagues demonstrated that GBS patients with dysautonomia, compared to those without, fared worse in terms of length of hospital stay, discharge outcomes, and mortality rates (6–13% vs. 2%) [2, 3]. To date, predictors of dysautonomia occurrence and GBS outcomes represent a void in the diagnostic and therapeutic management of GBS. In this study, we aimed to identify clinical and paraclinical factors associated with the development of dysautonomia and determine the impact of dysautonomia on clinical outcomes in a cohort of patients with GBS.

Methods

Study design and setting

This study utilized a cross-sectional study design with retrospective data gathering from June to July 2022 of all adult GBS patients with and without dysautonomia who were admitted at the University of the Philippines – Philippine General Hospital (UP-PGH) from January 2013 to June 2022.

Study population

Cases were identified using a review of patient medical records cross-referenced with the Database for Research and Information for the Neurosciences and results of nerve conduction studies (NCS) of the UP-PGH neurophysiology unit. In this 10-year retrospective review, all adult patients diagnosed with GBS based on the Brighton criteria at levels 1 and 2 of diagnostic certainty were included. From this cohort, cases with dysautonomia were defined as individuals who presented with at least one of the following features upon or during admission:

- **Cardiac –** Arrhythmia (sudden tachycardia and bradycardia), sustained sinus tachycardia (> 120 beats per minute or > 100 beats per minute on beta-blocker lasting for 2 h), sustained bradycardia (< 50 beats per minute for over 2 h) [2, 3].
- **Vascular** – Hypertension, defined as systolic blood pressure > 180 or > 160 mmHg while off or on antihypertensive medications, respectively: maximum systolic blood pressure, minimum diastolic blood pressure, hypotension with systolic blood pressure < 90 mmHg, and daily systolic blood pressure variation of > 85mmHg [2–4].
- **Gastrointestinal dysfunction** – Ileus, diagnosed radiographically and/or clinically requiring pharmacological treatment or mechanical decompression; presence of fecal incontinence [2–4].
- **Genitourinary dysfunction** – Urinary incontinence [2–4].
- **Sudomotor dysfunction** – Hyperhidrosis, fever > 37.8 degrees Celsius, and hypothermia < 36 degrees Celsius [2–4].

Using the criteria outlined above, patients were classified based on the presence or absence dysautonomia. We did

not include findings from objective tests for evaluating dysautonomia such as Ewing tests, isometric hand grip tests, and urodynamic tests. Patients who subsequently received a diagnosis of CIDP or non-immune-mediated inflammatory polyneuropathies after additional work-up were excluded.

Data collection

Data were obtained from the review of the medical records and supplemented by official results of the nerve conduction tests and cerebrospinal fluid (CSF) analysis, where applicable. Relevant baseline and clinical data collected included age, sex, and comorbidities (diabetes mellitus, hypertension, heart disease, alcoholism, pulmonary disease, malignancy, dyslipidemia, thyroid disease, chronic kidney disease). In terms of GBS-related data, information related to day of illness and corresponding Guillain – Barré Disability Disability Score (GBS-DS) on admission and discharge, antecedent events (infection, vaccination, surgery, trauma), GBS variant (classified as AIDP, AMAN, AMSAN, or MFS depending on the electrophysiologic findings and clinical history), and CSF protein and leukocyte levels were obtained. The criteria for diagnosing AIDP were as follows: At least two of the following: Motor conduction velocity of < 70% of lower limit of normal (LLN), distal motor latency of > 130% of upper limit of normal (ULN), distal compound muscle action potential (CMAP) amplitude duration > 120% ULN, proximal-to-distal CMAP duration ratio > 130%, F-wave latency > 120% ULN; or one of the above plus either or the following: Absent F-waves in two nerves with distal CMAP > 20% LLN, abnormal ulnar SNAP amplitude and normal sural SNAP amplitude. The criteria for diagnosing AMAN were as follows: None of the AIDP features in any nerve and at least one of the following in each of two nerves: distal CMAP < 80% LLN, proximal-to-distal CMAP amplitude ratio < 0.7 (excluding tibial nerve), isolated F-wave absence (< 20% persistence).

Data analysis

Data analysis was carried out using Microsoft Excel and STATA BE 17 (Texas, USA). Alpha was set at < 0.05 to determine statistical significance. Where appropriate, descriptive statistics with frequencies and proportions, or medians and interquartile ranges (IQR) was used to summarize categorical and continuous variables. Between-group comparisons of proportions were done using Chi-square or Fisher exact test whichever was applicable. Continuous variables were compared using independent t-test or rank sum test depending on normality. Simple and multiple logistic regressions analysis were conducted to the determine factors associated with dysautonomia; relationships were expressed using odds ratios (OR) with 95% confidence interval (CI).

Results

Demographic information and baseline characteristics

A total of 162 patient records with a consideration of GBS were identified, of which, only 81 satisfied the inclusion criteria but 10 had missing records, hence only 71 GBS patients were included in the study (Fig. 1). Thirty-five patients had dysautonomia (49.3%). Baseline demographic and clinical characteristics of GBS patients with and without dysautonomia are summarized (Table 1). The median age of GBS patients with dysautonomia were higher at 42.1 years old compared to those without at 31.6 years old. There were no differences in the proportion of gender, comorbidities, antecedent events, GBS variants, CSF studies found between groups.

Clinical features of dysautonomia

On average, manifestation of dysautonomia occurred within the 12th day of hospitalization. The most common manifestation of dysautonomia among the patients were cardiovascular signs such as hypertension (80%), sustained tachycardia (38.6%), hypotension (31%), and arrhythmia (22.9%). Disturbances in the genitourinary and gastrointestinal were the second most common presenting as fecal incontinence (17.1%), ileus (14.3%), and urinary incontinence (8.6%). (Fig. 2).

Association of dysautonomia with possible risk factors

Simple logistic regression analysis revealed that age is a significant factor in the development of dysautonomia, such that each year increase in age was associated with a 5% increase in the odds of developing dysautonomia (OR: 1.05, $p = 0.005$). (Table 2). Multiple logistic regression analysis was done to further identify the risks factors associated with dysautonomia and to control for possible confounders. Those with p-values less than 0.4 from simple logistic regression analysis were included in the independent factors. Age (OR: 1.11, $p = 0.001$), hypertension (OR:0.13, $p = 0.028$), alcoholism (OR: 0.17, $p = 0.037$), and GBS-DS (OR:1.65, $p = 0.037$) during admission were statistically significant risk factors for dysautonomia. A 1-year increase in age and a point increase in GBS-DS during admission, increased the odds of dysautonomia by 10% and 65% correspondingly. Premorbid hypertension (diagnosed with hypertension before admission) and alcoholism decreased the odds by 88% and 83% respectively.

Respiratory failure, mortality, treatments, and outcome

In patients with dysautonomia, 54% (19/35) received intravenous immunoglobulin (IVIG) while 28% (10/35) had plasma exchange (PLEX). Only one mortality was identified among the GBS patients in our cohort. However, association between dysautonomia and different outcome parameters such as the use of a mechanical

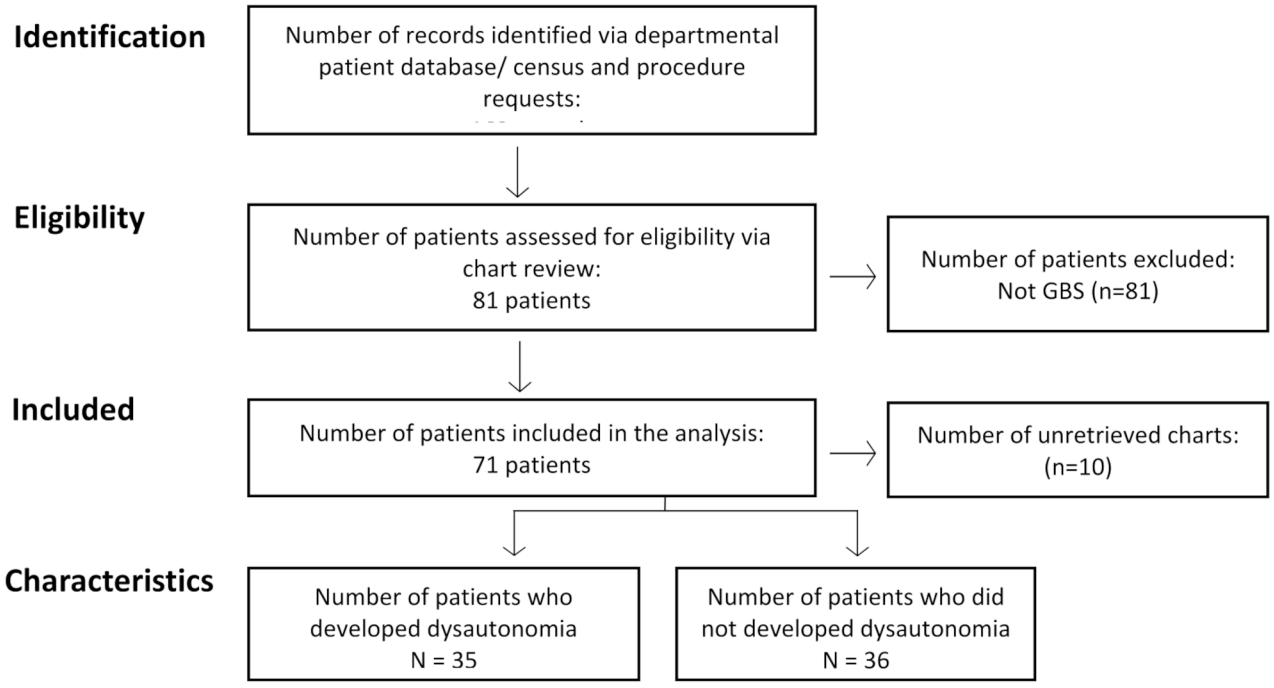

Fig. 1 Flow diagram of patient inclusion

ventilator, use of IVIG, PLEX, the occurrence of pneumonia, and duration of hospitalization were determined. 17% (6/35) of GBS patients with dysautonomia were intubated comparing to only 8% (3/36) to those without. Using simple logistic regression analysis, those with dysautonomia had 2.28 times and 2.6 times odds of being on a ventilator ($Z = 1.09$, $p = 0.274$) and having pneumonia during admission ($Z = 1.68$, $p = 0.093$) than those without; however, the differences were not statistically significant. GBS patients with dysautonomia have 1.67 times odds of being treated using IVIG ($Z = 1.07$, $p = 0.286$) and 1.2 times odds of being treated using PLEX ($Z = 0.34$, $p = 0.734$), but the associations again were not significant. Those with dysautonomia stayed longer in the hospital by 12.23 days. Although the association was significant, the presence of dysautonomia only predicts 5.5% of the variability in the number of hospital days ($R^2 = 5.5\%$, $p = 0.049$). Majority of our patients were followed-up on our out-patient clinic within 2–4 weeks, 28.6% of GBS patients with dysautonomia had a GBS DS score of 2 within one month compared to 33.3% of GBS patients without dysautonomia.

Discussion

In this retrospective cohort study, we showed that approximately half of clinically diagnosed GBS patients developed one or more features of dysautonomia, with increasing age and GBS-DS being positively associated with its development. This is in contrast with the findings of Chakraborty that found that age was not associated with the development of dysautonomia. This difference may be attributed to relatively similar mean age of GBS patients in their study between those with dysautonomia (55 +/- 16) and those without (54 +/- 16), furthermore, out cohort are relatively younger than their study [3]. The significant contribution of age in the manifestation of dysautonomia could be attributed to age-associated dysfunction in the autonomic nervous system. A study done by Keir et al. demonstrated an increase in muscle sympathetic nerve activity (MSNA) were seen with increasing age. MSNA is directly involved in short- and long-term regulation of cardiac output and total peripheral resistance [5]. Advancing age was also correlated with greater heart rate variability (HRV) due to a decrease in the following markers: standard deviation of R-R intervals, high frequency cyclic fluctuations of HRV, low frequency cyclic fluctuations of HRV, and very low frequency cyclic fluctuations of HRV [6]. With the proposed sympathetic overdrive in dysautonomia, this imbalance could be further worsened by diminished parasympathetic activity in aging as evidenced by age-related loss in cardiac-projecting vagal preganglionic neurons, reduction of acetylcholine synthesis, blunted changes in acetylcholine transporter, and reduced baroreflex sensitivity [6]. Our study also showed a male predominance with respect to GBS diagnosis and development of dysautonomia, reflecting the available data on global incidence [3, 7, 8].

Table 1 Patient demographics and clinical features

Characteristic	Dysautonomia (*n* = 35)	No Dysautonomia (*n* = 36)	*P* value
Age (y)	42.1 (13)	31.6 (15.3)	0.0014
Male	57.1	58.3	0.54
Comorbidities			
Diabetes Mellitus	5.7	8.3	0.67
Premorbid Hypertension [21]	11.4	19.4	0.82
Heart Disease	0	2.8	0.84
Alcoholism	14.3	25.0	0.87
Pulmonary Disease	2.9	2.8	0.49
Malignancy	5.7	5.6	0.48
Dyslipidemia	5.7	5.6	0.48
GBS-DS on admission	3.82	3.6	-
Antecedent Event			
Respiratory Infection	42.9	25.0	0.06
Gastrointestinal Infection	8.6	5.6	0.31
Vaccination	0	2.8	0.84
Trauma	0	0	-
Surgery	8.6	5.6	0.31
GBS Variant			
AIDP	40.0	52.8	0.86
AMSAN	28.6	19.4	0.18
AMAN	14.3	16.7	0.6
MFS	14.3	11.1	0.34
BBE	0	0	-
Cerebrospinal Fluid Studies			
Protein	298.7 (504.2)	95.3 (110.1)	0.3
Leukocytes	2.3	1.2	-

AIDP – Acute Inflammatory Demyelinating Polyneuropathy, AMAN – Acute Motor Axonal Neuropathy, AMSAN - Acute Motor and Sensory Axonal Neuropathy, BBE – Bickerstaff Brainstem Encephalitis, GBS – Guillain-Barré Syndrome, GBS-DS – Guillain-Barré Syndrome - Disability Score, MFS – Miller Fisher Syndrome

In terms of a possible antecedent infection, our study showed that a prior respiratory infection is the most common trigger for GBS which is reflective of the global cohort, especially in low to middle income countries [9]. The low number of preceding gastroenteritis infection in our study can be explained by the predominant variant of GBS in our cohort which is AIDP compared to AMAN variant which is more associated with *C. jejuni* infection [10]. Although countries in the Asian region have a high proportions of patients with AMAN or AMSAN, AIDP overall is still the most frequent variant [9].

Our study showed that the most common manifestation of dysautonomia involves the cardiovascular system which are the same with previous studies [1, 3, 11, 12]. This in turn contributes to a significant morbidity and mortality in GBS patients [13]. Although the mechanism is not yet fully understood, in terms of the proportion of GBS subtypes to the manifestation of dysautonomia, AIDP is characterized by cardio sympathetic hyperactivity while AMAN has reduced sudomotor function and only in severe cases [2, 14]. The pathological mechanism of GBS is still unknown, although molecular mimicry is the leading pathogenic concept in post-infectious GBS [15]. Available evidence shows that the underlying pathophysiology in dysautonomia predominantly involves an imbalance of sympathetic predominance between the afferent arterial baroreceptors, efferent cardiac parasympathetic fibers, and preganglionic sympathetic fibers with vasomotor and sudomotor functions [3]. Another proposed theory implicates a catecholamine surge from an afferent peripheral nerve conduction block [3]. A cardiovascular collapse may result from the drastic fluctuations of blood pressure from the alteration of feedback control or the inappropriate ectopic discharges from the demyelination of preganglionic sympathetic axons or axonal degeneration in postganglionic axons [2]. These hypotheses can be supported by findings of a study that analyzed autonomic nerves among GBS autopsy cases that revealed the presence of demyelination and mononuclear cell infiltration on the vagus nerve and sympathetic ganglia [13]. One limitation of our findings is that we are unable to differentiate between tachycardia as a manifestation of dysautonomia or as reactive sign from infection. Further analysis with more rigorous parameters can be done in future studies to differentiate between these two. As previously shown by Chakraborty and reflected in our study, the subtype of GBS do not predict the development of dysautonomia. This is supported by a

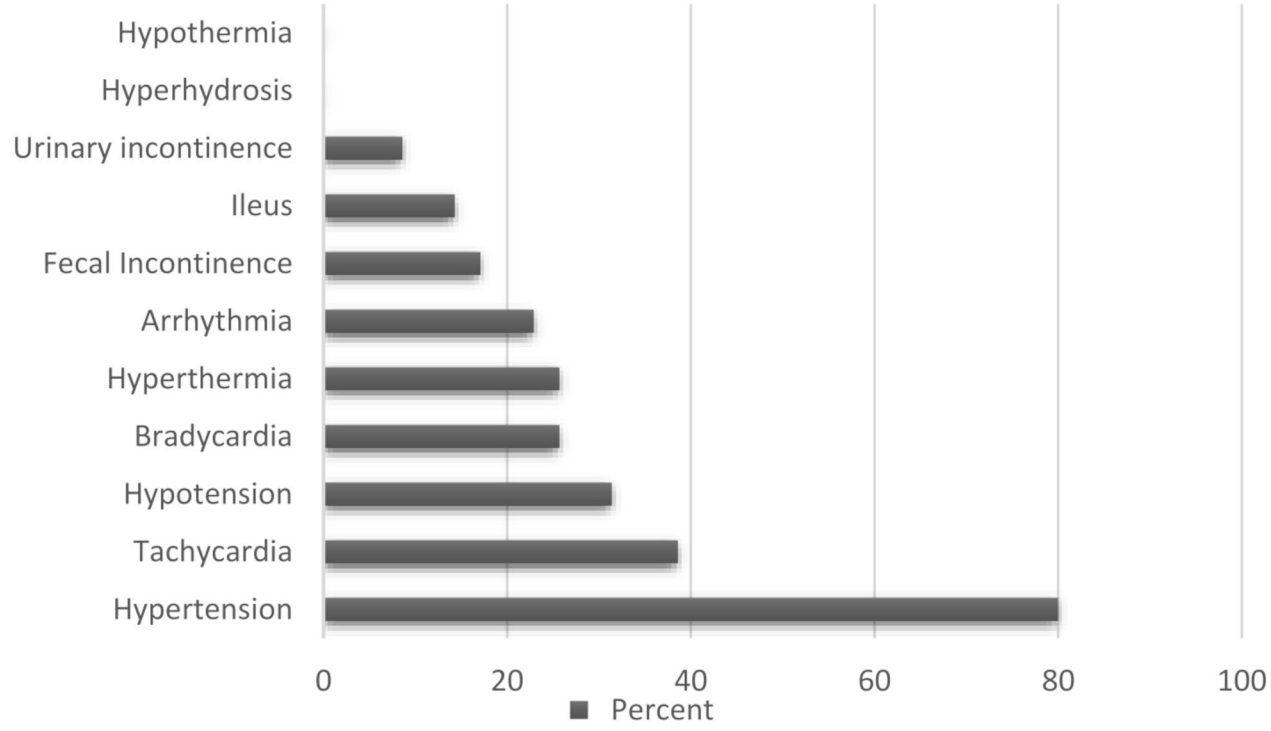

Fig. 2 Features of Dysautonomia ($n = 35$)

Table 2 Association of clinical features, risk factors, variants, and dysautonomia

Variable	Simple logistic regression analysis OR	*p*-value	Multiple logistic regression analysis OR	*p*-value
Age (y)	1.05	0.005	1.11	0.001
Male	0.95	0.92		
Comorbidities				
Diabetes Mellitus	0.67	0.67		
Hypertension	0.53	0.36	0.13	0.028*
Heart Disease	-	-		
Alcoholism	0.5	0.26	0.17	0.037*
Pulmonary Disease	1.03	0.98		
Malignancy	1.03	0.98		
Dyslipidemia	1.03	0.98		
GBS-DS				
Admission	1.22	0.24		
Antecedent Event				
Respiratory Infection	2.25	0.115	1.73	0.44
Gastrointestinal Infection	1.6	0.6	-	-
Vaccination	-	-	-	-
Trauma	-	-	-	-
Surgery	1.6	0.6	-	-
GBS Variant				
AIDP	0.6	0.28	0.08	0.065
AMSAN	1.7	0.37	0.18	0.28
AMAN	0.8	0.78	0.20	0.28
MFS	-	-	-	-
BBE	-	-	-	-

AIDP – Acute Inflammatory Demyelinating Polyneuropathy, AMAN – Acute Motor Axonal Neuropathy, AMSAN - Acute Motor and Sensory Axonal Neuropathy, BBE – Bickerstaff Brainstem Encephalitis, GBS DS – Guillain-Barré Syndrome Disability Score, MFS – Miller Fisher Syndrome, OR – Odds ratio

literature review done by Kaida that did not show any significant association between autonomic neuropathy and the presence of anti-glycolipid antibodies [13].

The study done by Chakraborty reported ileus as the most common manifestation of autonomic dysfunction in up to 42% of patients, in contrast, our study identified only 14.3% of patients with ileus [3]. The difference in the population can be attributed to varying sample sizes and the different cutoff values and duration used in classifications. Furthermore, a bias in practice can also be cited especially in our institution such that bedbound patients are routinely given with laxatives and are placed on urinary catheters which in turn results in under recognition and underreporting.

Interestingly, hypertension and alcoholism were noted to possibly cause decreased incidence of dysautonomia. This is despite the results showing hypertension as the most common feature of dysautonomia. Alcohol-induced autonomic dysfunction and alcohol-related large fiber peripheral neuropathy is a known consequence of chronic and excessive alcohol consumption [16]. A systematic review done by Julian and colleagues revealed that autonomic dysfunction among alcoholics tend to be asymptomatic and correlated with the total lifetime dose of ethanol [16]. Their study however has a heterogenous population hence appropriate conclusion requires further investigation. The most important risk factor for alcohol-related autonomic dysfunction is total lifetime dose of ethanol, which we failed to obtain from our review of records to further explore this correlation [16, 17]. Through review of evidence, studies showing the protective effect of alcohol consumption with dysautonomia are still lacking and given the small sample size, appropriate conclusions cannot be drawn. The protective effect of pre-morbid hypertension on the other hand can be explained intake of antihypertensive medications of these patients which could offset the development of dysautonomia. During admission, most of the patient's medications were continued if no contraindications were present in which most of them include antihypertensives. However, given the contradictory nature of these findings, further studies must be undertaken to further explore this correlation.

IVIG and PLEX are the gold-standard treatment for GBS with equal efficacy, however it is not known whether one has an advantage over the other in terms of reducing the severity of dysautonomia [2, 18]. This is supported by a meta-analysis done by Zaki and his colleagues which showed that IVIG and PLEX has similar curative effects, length of hospitalization, duration of mechanical ventilation, and risk of relapse [19]. Their study however did not compare the effects of IVIG versus PLEX in addressing dysautonomia in these patients. Both our study and the ones by Chakraborty further corroborates the general consensus that both treatment modalities are comparable in addressing dysautonomia such that both neither improves nor complicates the course of the disease [3]. Given these findings, clinicians tend to favor IVIG administration due to relative better availability of the drug and lesser contraindication than PLEX, another reason that could be explained was the significantly lower rate of discontinuation of IVIG than the PLEX group [19]. Aside from being more invasive, PLEX was also postulated to induce autonomic dysfunction with hypo-/hypertension due to an imbalance of fluids and electrolytes, however, prospective studies have not been done to confirm these [20].

Several observational studies have demonstrated that GBS patients with dysautonomia had poorer outcomes in terms of length of hospital stay, discharge outcomes, and mortality rates (6–13% vs. 2%) than those without dysautonomia [2, 3, 20]. In our study, GBS patients with dysautonomia had 2.26 times odds of being on a ventilator and 2.6 times odds of developing pneumonia compared to patients without dysautonomia. This highlights the need for closer monitoring for respiratory failure in these patients. Those with dysautonomia have a longer hospital stay of 12.23 days which could contribute to significant morbidity such as increased risk of nosocomial infection.

This study has several limitations. The retrospective nature of data collection increases its risk for selection and information bias. The secondary nature of data from different physicians who obtained them through the years may result to under or overestimation of the outcomes in different variables. For example, the prevalence of hypothermia and hyperhidrosis due to lesser clinical severity, and lower urinary incontinence due to bladder catheterization before retention becomes evident might have been underestimated. Given the relatively small sample size and a single-center setting of our cohort, our findings cannot be generalized to all patients with dysautonomia especially with the difference in their demographic characteristics such as race, age distribution, GBS subtype, and treatments received. One may argue that the prevalence of dysautonomia may have been overestimated since only severe cases go to the hospital, however, GBS patients whether mild or severe tend to be admitted as reflected by the number of MFS patients included in our cohort.

Conclusion

Our study showed that almost half of GBS patients developed dysautonomia, with most experiencing cardiovascular manifestations during their admission. Older patients and those with a higher GBS-DS on admission are more likely to develop dysautonomia during their hospitalization. The protective effects of premorbid hypertension and alcoholism needs further evaluation

due to theoretically conflicting mechanisms. Although not statistically significant, the higher odds of these patients to develop pneumonia and need for ventilatory support eventually leads to longer hospital stay and significant morbidities. Early identification of these patients prompts closer monitoring and timely intervention to manage, or event prevent dysautonomia and other complications of GBS. Furthermore, we would like highlight that the management of GBS patients is not only geared towards its sensorimotor manifestations but also on dysautonomia. A larger prospective study is warranted to confirm these results.

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Author contributions
NGR and GTP conceptualized the study protocol. GTP and LCD performed the review of records and data collection. MBP performed the statistical analyses of the data. All of the authors contributed to the generation of the protocol and finalization of the manuscript.

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Data availability
The datasets generated and/or analyzed during the current study are not publicly available due to patient privacy and confidentiality of records but are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
The protocol for this research project has been approved by the Research Ethics Board of the University of the Philippines Manila (Code: UPMREB 2021-0711-01) and it conforms to the provisions of the Declaration of Helsinki. No human participant was involved in this study. The consent to participate was waived by the research ethics board in accordance with the National Ethical Guidelines for Health and Health-Related Research.

Consent for publication
N/A.

Competing interests
The authors declare no competing interests.

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