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Original Article

Impact of Diabetes Mellitus on the Clinical Course and Early Outcomes in Guillain–Barré Syndrome: Single Center Experience

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Abstract

Background: This research was designed to evaluate whether comorbid diabetes mellitus (DM) influences the clinical presentation, severity of illness, and short-term functional recovery in individuals suffering from Guillain–Barré syndrome (GBS).

Methods: We performed a retrospective cohort study of 77 adult patients with GBS managed at a tertiary care neurological center from January 2020 through December 2025. Participants were categorized into two distinct groups: those with pre-existing type 2 diabetes mellitus (T2DM) (GBS+DM) and those without (GBS–DM). The comparison focused on demographic data, electrophysiological variants, Medical Research Council (MRC) sum scores, Modified Erasmus GBS Outcome Scores (mEGOS), and discharge functional capacity measured by the Hughes disability scale.

Results: In the total sample, 14 patients (18.2%) had diabetes mellitus. The GBS+DM group tended to be older; however, this difference was not statistically significant ($p = 0.142$). The mean mEGOS score was slightly higher in the diabetic cohort (3.8 ± 2.4) than in the non-diabetic group (3.4 ± 2.7), but the difference was not statistically significant ($p = 0.364$). No significant differences were observed regarding electrophysiological subtypes or early functional recovery between the cohorts. Nevertheless, a consistent numerical trend toward greater disability and less favorable prognostic markers was noted in the DM group.

Conclusion: Although not statistically significant, our data suggest that the presence of DM might be associated with a more severe clinical course and slower early recovery in GBS patients. Further validation through large-scale, prospective, multicenter trials is essential to definitively characterize the impact of DM on GBS outcomes.

Keywords: Guillain-Barre Syndrome; Diabetes Mellitus; Prognosis; Muscle Strength; Treatment Outcome; Autoimmune Diseases

INTRODUCTION

Guillain–Barré syndrome (GBS) is an acute-onset, immune-mediated inflammatory polyradiculoneuropathy affecting the peripheral nerves and nerve roots. With an annual incidence of approximately 1–2 per 100,000, GBS represents the most common cause of acute paralytic neuropathy in adults (1,2). Although the clinical course is typically monophasic, disease severity and recovery trajectories may vary substantially among patients. Despite effective immunomodulatory treatments such as intravenous immunoglobulin (IVIg) or plasmapheresis, a considerable proportion of patients develop persistent neurological sequelae. While overall mortality is reported to be below 5%, approximately 15–20% of patients are unable to walk independently six months after disease onset (3,4). These findings underscore the clinical importance of early prognostic assessment in GBS.

Several prognostic scoring systems have been developed to predict early disease course and long-term functional outcomes in GBS. Among these, the modified Erasmus GBS Outcome Score (mEGOS) is a widely validated and commonly used tool in clinical practice. The mEGOS incorporates patient age, history of preceding diarrhea, and the severity of muscle weakness as reflected by the Medical Research Council (MRC) sum score to estimate the risk of inability to walk independently at six months after disease onset (5–8). Higher mEGOS scores have consistently been associated with poorer functional outcomes.

Diabetes mellitus (DM) is a chronic metabolic disorder with increasing global prevalence and multisystem involvement. The peripheral nervous system is among the most commonly affected systems, and diabetic neuropathy may lead to progressive axonal degeneration and myelin damage over time (9). In recent years, accumulating evidence suggests that the presence of DM may adversely affect the clinical course and prognosis of GBS. In a retrospective study including 257 patients with GBS, Perić et al. reported that 17% of patients had concomitant DM and demonstrated that DM was significantly associated with poor short-term outcomes even after adjustment for age (10). Similarly, in a prospective study by Bae et al., patients with DM exhibited significantly worse functional outcomes at three months, and DM was identified as an independent risk factor for failure to regain independent ambulation. Notably, sudden cardiac arrest and deaths related to autonomic instability were observed exclusively in patients with DM in that study (11).

Proposed mechanisms underlying the unfavorable impact of DM on GBS include the presence of subclinical or overt axonal damage related to chronic hyperglycemia, which may exacerbate acute immune-mediated nerve injury. In addition, hyperglycemia-induced inflammatory activation, microvascular perfusion impairment, and metabolic stress may further aggravate peripheral nerve damage (12). Nevertheless, studies directly comparing the clinical characteristics and early outcomes of patients with GBS with and without DM across different populations remain limited.

In this study, we aimed to evaluate the impact of concomitant diabetes mellitus on clinical characteristics, disease severity, and early functional outcomes in patients with GBS treated at our center. By comparing patients with and without DM in terms of demographic features, baseline muscle strength (MRC sum score), mEGOS, need for mechanical ventilation, and functional status at discharge, we sought to elucidate the potential prognostic role of DM in GBS. Evidence regarding the impact of diabetes mellitus on the early clinical course of Guillain–Barré syndrome remains limited, particularly in real-world clinical cohorts.

METHODS

Protocol and Search Strategy

This retrospective cohort study, conducted at a single tertiary center, involved adult patients admitted to our Neurology Department with a diagnosis of GBS between January 2020 and December 2025. The Brighton criteria, which integrate clinical findings with electrophysiological and laboratory evidence, were used to confirm GBS diagnoses. Ethical approval for the protocol was granted by the local ethics committee on November 20, 2025 (Decision No: 848), and the study adhered to the principles of the Declaration of Helsinki.

The study population was categorized into two distinct cohorts based on glycemic status: the “GBS with DM” group, comprising patients with pre-existing type 2 diabetes mellitus, and the “GBS without DM” group, consisting of those without a history of diabetes mellitus. Enrollment followed a consecutive pattern, including all patients irrespective of clinical subtype, disease severity, or therapeutic interventions received.

Data on patient demographics (age and sex), potential triggers occurring within the six weeks preceding GBS (such as vaccinations, infections, or diarrheal episodes), clinical examination findings, and laboratory parameters were systematically retrieved from electronic medical records. Based on the results of nerve conduction studies, GBS cases were classified into electrophysiological subtypes: acute inflammatory demyelinating polyradiculoneuropathy (AIDP) or axonal variants, including acute motor/motor–sensory axonal neuropathy (AMAN/AMSAN) (13,14).

Assessment of motor strength was performed at admission and during hospitalization using the Medical Research Council (MRC) scale. The MRC sum score, ranging from 0 to 60, was calculated by assessing three muscle groups in each limb. Higher scores indicate greater muscle strength. For descriptive purposes, MRC scores were categorized into five groups reflecting the severity of motor weakness: (1) normal strength (60), (2) mild weakness (48–59), (3) moderate weakness (36–47), (4) severe weakness (24–35), and (5) extreme weakness (<24). To estimate the probability of regaining independent walking at six months, the modified Erasmus GBS Outcome Score (mEGOS) was calculated for each patient, incorporating admission MRC score, age, and history of preceding diarrhea.

Functional recovery was assessed using the GBS disability scale at discharge and, where available, at three-month follow-up. Secondary clinical endpoints included the need for mechanical ventilation, duration of intensive care unit stay, and total length of hospital stay.

Nerve Conduction Studies

Electrophysiological evaluations were performed using a Cadwell Sierra Summit system (Cadwell Laboratories Inc., USA) in the neurophysiology laboratory. All procedures followed standardized clinical protocols, maintaining skin temperature above 32°C for the upper extremities and 30°C for the lower extremities. Motor and sensory nerve conduction velocities, distal

latencies, and compound muscle action potential (CMAP) amplitudes were assessed in the median, ulnar, peroneal, and tibial nerves. F-wave responses were recorded to evaluate proximal nerve segments. Sensory nerve action potentials (SNAPs) were analyzed for the median, ulnar, and sural nerves. Based on the criteria established by Rajabally et al., patients were classified into the electrophysiological subtypes of AIDP, AMAN, or AMSAN (13).

STATISTICAL ANALYSIS

Statistical analyses and data processing were performed using IBM SPSS Statistics software (Version 25.0; IBM Corp., Armonk, NY, USA). Depending on data distribution, continuous variables are presented as mean ± standard deviation or median (interquartile range). Categorical variables are summarized as frequencies and percentages. To compare mEGOS and early functional outcomes between the cohorts, the independent samples t-test was used for normally distributed variables, whereas the Mann–Whitney U test was used for non-normally distributed variables. Categorical variables were compared using the Pearson chi-square test or Fisher exact test. Statistical significance was defined as a p-value < 0.05.

RESULTS

Study Characteristics

A total of 77 patients with GBS were included in the study. The mean age was 51.9 ± 18.4 years; 46 patients (59.7%) were male and 31 (40.3%) were female. Fourteen patients (18.2%) had concomitant diabetes mellitus, while 63 patients (81.8%) did not. In the DM group, 10 patients (71.4%) were male, compared with 36 patients (57.1%) in the non-DM group. No statistically significant differences were observed between the groups regarding age or sex distribution (both p > 0.05).

Table 1. Demographic and Clinical Characteristics of Patients With Guillain–Barré Syndrome With and Without Diabetes Mellitus

Characteristics	DM (+), n=63	DM (+), n=14	p value
Age, mean	50.7 ± 19.2	58.7 ± 11.7	0.142
Medical history			
Hypertension, n	3	0	0.029
CAD, n	5	4	0.052
Hyperlipidemia, n	18	6	0.297
MRC total score	2.6 ± 1.3	2.6 ± 1.1	0.779
Electrophysiological classification (Rajabally criteria)			
AIDP	50	13	
AMAN	8	1	
AMSAN	1	0	
Unclassified	4	0	

GBS, Guillain–Barré syndrome; DM, diabetes mellitus; CAD, coronary artery disease; MRC, Medical Research Council; AIDP, acute inflammatory demyelinating polyradiculoneuropathy; AMAN, acute motor axonal neuropathy; AMSAN, acute motor–sensory axonal neuropathy.

Electrophysiological evaluation revealed demyelinating features in 13 patients (92.9%) and axonal involvement in 1 patient (7.1%) in the DM group. In the non-DM group, 50 patients (79.4%) exhibited demyelinating features and 13 patients (20.6%) showed axonal involvement. There was no statistically significant difference in electrophysiological subtype distribution between the groups (p = 0.444) (**Table 1**).

Disease Severity and Clinical Features

The mean admission mEGOS score was 3.38 ± 2.67 in the non-DM group and 3.78 ± 2.35 in the DM group, with no statistically significant difference between the groups (p = 0.364) (**Table 2**).

The severity of motor weakness was evaluated using the Medical Research Council (MRC) sum score. Mild weakness (corresponding to higher MRC scores) was observed in 3 patients (21.4%) in the DM group and in 13 patients (20.6%) in the non-DM group, with no statistically significant difference between groups (p > 0.05).

Early Functional Outcomes

Early functional outcome, as assessed by the GBS disability scale at discharge, was 15.04 ± 17.16 in the non-DM group and 15.57 ± 16.56 in the DM group. No statistically significant difference was detected between the groups (p = 0.443).

Overall, no statistically significant differences were observed between patients with GBS with and without DM in terms of demographic characteristics, electrophysiological subtypes, baseline disease severity, or early functional parameters. However, numerically higher admission mEGOS and GBS disability scale scores in the DM group suggest that further studies are warranted to clarify the potential impact of diabetes mellitus on GBS prognosis.

DISCUSSION

In the present study, no statistically significant differences were observed between GBS patients with and without concomitant DM regarding demographic features, electrophysiological subtypes, baseline clinical

Table 2. Modified Erasmus Guillain–Barré Syndrome Outcome Score (mEGOS) and Early Prognostic Parameters

Parameter	DM (+), n=63	DM (+), n=14	p value
mEGOS score	3.4 ± 2.7	3.8 ± 2.4	0.364
Probability of improvement at 6 months (%)	15.1 ± 17.2	15.6 ± 16.6	0.443

GBS, Guillain–Barré syndrome; mEGOS, modified Erasmus Guillain–Barré Syndrome Outcome Score; DM, diabetes mellitus.

severity, or early functional outcomes. Nevertheless, the observation of numerically higher admission mEGOS and GBS disability scales in the DM group suggests that the potential influence of diabetes mellitus on the clinical course of GBS merits further investigation in larger, prospective cohorts.

Bae et al. reported significantly worse three-month functional outcomes in GBS patients with DM and identified DM as an independent risk factor for failure to regain independent ambulation (11). Similarly, Perić et al. demonstrated that DM was significantly associated with poor short-term outcomes even after adjustment for age (10). In contrast, although mEGOS and disability scores were numerically higher in our DM group, these differences did not reach statistical significance.

Several pathophysiological mechanisms have been proposed to explain the adverse impact of DM on GBS outcomes. Chronic hyperglycemia may lead to subclinical or overt axonal damage in peripheral nerves, thereby amplifying the effects of acute immune-mediated nerve injury. In our study, we focused on patients with a pre-existing diagnosis of type 2 DM. However, emerging evidence suggests that not only overt diabetes but also impaired fasting glucose or stress-induced glycemic fluctuations may correlate with GBS severity. While we did not perform oral glucose tolerance tests due to the retrospective design and the potential for acute illness-related stress to confound results, the interplay between early-stage glucose metabolism and neuroinflammation remains a critical area for investigation. Indeed, Bae et al. reported more pronounced distal nerve conduction abnormalities in GBS patients with DM, suggesting the additive effect of diabetic axonal damage (11). Furthermore, hyperglycemia may exacerbate peripheral nerve injury through increased inflammatory cytokine production, impaired microvascular perfusion, and enhanced oxidative stress (12,15-17).

Previous studies have also suggested an increased risk of autonomic instability, cardiac arrhythmias, and sudden cardiac arrest in GBS patients with DM (11). The absence of mortality in our cohort may be attributable to the limited sample size and the relatively small number of elderly or hyperacute cases.

From a clinical perspective, these findings have important implications. In patients with GBS and concomitant DM, clinicians should be aware of the potential for a more severe disease course and maintain a lower threshold for intensive care monitoring due to the risk of respiratory failure and autonomic dysfunction. Early planning of rehabilitation and appropriate counseling of patients and caregivers regarding potentially slower functional recovery are also essential. Prognostic tools such as the mEGOS may provide valuable guidance in clinical decision-making, particularly in patients with DM.

Although this study contributes to the limited body of literature examining the interaction between metabolic disorders and immune-mediated neuropathies, several important limitations should be considered when interpreting the findings. First, the relatively small sample size, particularly within the diabetic subgroup, may have reduced the statistical power required to detect subtle differences between the groups. Another limitation is the lack of documentation of diabetes-specific clinical parameters, including HbA1c levels, duration of diabetes, treatment adherence, and the severity of pre-existing diabetic complications. In addition, the absence of data regarding pre-admission glycemic control prevented us from determining whether well-controlled versus poorly controlled diabetes mellitus has differential effects on Guillain–Barré syndrome outcomes. Future multicenter prospective studies with larger cohorts, incorporating detailed metabolic parameters such as HbA1c levels, duration of diabetes, and metabolic control status, as well as systematic screening for glucose intolerance in non-diabetic GBS patients, may provide a more comprehensive understanding of the relationship between diabetes mellitus and Guillain–Barré syndrome.

CONCLUSION

In conclusion, diabetes mellitus in patients with Guillain–Barré syndrome should be considered a potential risk factor influencing early functional outcomes. Larger, multicenter, prospective studies are required to clarify the underlying mechanisms and to develop targeted strategies aimed at improving prognosis in patients with GBS and concomitant diabetes mellitus.

DECLARATIONS

Ethics Committee Approval: Ethical approval for the protocol was granted by the Adana City Training and Research Hospital Scientific Research Ethics Committee on November 20, 2025 (Decision No: 848), and the study strictly adhered to the principles of the Declaration of Helsinki.

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