Journal of Teachers Association
https://tajrmc.com/taj
Vol. 38, No. 2, 2025

pISSN 1019-8555
eISSN 2408-8854

RESEARCH ARTICLE | OPEN ACCESS **DOI:** https://doi.org/10.70818/taj.v38i02.0286

Electrophysiological Subtypes of Guillain-Barre Syndrome in the Bangladeshi Population: A Cross-Sectional Study

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Citation:
Haque ABMM, Zaman R, Uddin S, Parveen E, Sarker MAM, Rahman M, Yousuf A, Nasrin S, Habibah M; Electrophysiological Subtypes of Guillain-Barre Syndrome in the Bangladeshi Population: A Cross-Sectional Study. Journal of Teachers Association. 2025;38(2):12-19

Article History:
Received: 03.02.2025
Accepted: 18.04.2025
Published: 01.06.2025

ABSTRACT: ***Background:*** Guillain–Barré syndrome (GBS) exhibits significant regional differences in demographics, symptoms, electrophysiological subtypes, diagnostic features, and prognosis. Acute Inflammatory Demyelinating Polyradiculoneuropathy (AIDP) predominates in Western populations, the axonal variants—Acute Motor Axonal Neuropathy (AMAN) and Acute Motor and Sensory Axonal Neuropathy (AMSAN)—are more common in Asia. However, data on GBS subtypes in Bangladesh remain limited. This study aims to determine the distribution of GBS subtypes and their associated clinical and electrophysiological features. ***Methods:*** A cross-sectional study was conducted at Rajshahi Medical College Hospital from January 2016 to December 2017. A total of 45 clinically diagnosed GBS patients underwent nerve conduction studies (NCS) to classify subtypes. Clinical parameters were analyzed, including age, preceding infections, and motor function. Statistical associations between GBS subtypes and clinical findings were assessed using chi-square and t-tests. ***Results:*** Among the 45 patients, 55.6% were diagnosed with AMAN, 37.8% with AIDP, and 6.6% with AMSAN. AMAN was significantly more prevalent in younger patients, whereas AIDP was more common in older individuals. Preceding diarrheal illness was associated with AMAN and AMSAN ($p = 0.026$), while respiratory infections were linked to AIDP ($p = 0.030$). Axonal subtypes demonstrated lower CMAP amplitudes, while AIDP showed prolonged distal motor latency and reduced conduction velocity, indicating demyelination. ***Conclusions:*** AMAN is the predominant GBS subtype in Bangladesh, aligning with regional trends. Electrophysiological findings are crucial for early diagnosis and management of GBS. Further research is needed to investigate genetic and environmental factors influencing GBS subtypes in this population.

Keywords: Guillain-Barré Syndrome, AIDP, AMAN, AMSAN, Electrophysiology.

Article at a glance:

Study Purpose*: The study aims to determine the distribution of GBS subtypes and explore associated clinical and electrophysiological features.*

Key findings: *55.6% had AMAN, 37.8% had AIDP, and 6.6% had AMSAN. Preceding infections were associated with different subtypes.*

Newer findings: *AMAN is predominant in Bangladesh, aligning with regional trends, and highlighting the importance of early diagnosis through NCS.*

Abbreviations: *GBS - Guillain-Barré Syndrome, AIDP - Acute Inflammatory Demyelinating Polyradiculoneuropathy, AMAN - Acute Motor Axonal Neuropathy, AMSAN - Acute Motor and Sensory Axonal Neuropathy, MFS - Miller Fisher Syndrome, NCS - Nerve Conduction Study, CMAP - Compound Muscle Action Potential, SNAP - Sensory Nerve Action Potential, NCV - Nerve Conduction Velocity, DML - Distal Motor Latency, AMR - Antimicrobial Resistance.*

INTRODUCTION

Guillain-Barré syndrome (GBS) is an inflammatory polyneuropathy characterized by a progressive onset of symmetrical, flaccid hypoxia in the ascending limbs, followed by motor symptoms with or without sensory abnormalities.[1] It is a significant inflammatory peripheral radiculopathy and neuropathy marked by advancing limb

weakness, sensory impairments, cranial nerve involvement, tendon areflexia, and cerebrospinal fluid (CSF) albumin cytological dissociation.[2]

Approximately 100,000 people are affected by GBS globally each year. A population-based study showed the annual incidence of GBS ranges from 0.4 to 1.7 per 100,000 individuals, with a higher prevalence in males and older adults.[3,4] The incidence of GBS rises from 1.7/100,000 to 3.3/100,000 beyond the age of 50, in contrast to younger patients.[5] Approximately two-thirds of GBS infections are linked with antecedent infection.[6] Numerous microorganisms have been linked to Guillain-Barré syndrome, including *Campylobacter jejuni*, Zika virus, and severe acute respiratory syndrome coronavirus-2. In Guillain-Barré syndrome associated with *C. jejuni*, substantial evidence indicates an autoantibody-mediated immunological mechanism initiated by molecular mimicry between the structural elements of peripheral nerves and the pathogen.[7]

GBS presents various clinical manifestations, including unique variants such as Miller-Fisher syndrome, characterized by ophthalmoplegia, ataxia, and areflexia. Other variants involve different nerve fiber types, leading to diverse symptoms.[8] In severe cases, cranial nerve involvement can lead to facial weakness, and autonomic dysfunction may result in blood pressure fluctuations and cardiac arrhythmias.[9] The diagnosis of Guillain-Barré Syndrome (GBS) relies on patient history, neurological assessments, electrophysiological evaluations, and cerebrospinal fluid (CSF) analysis.[10–12]

The prevalent variations of Guillain-Barré Syndrome (GBS) are Acute Inflammatory Demyelinating Polyradiculoneuropathy (AIDP), a motor sensory demyelinating condition, and Acute Motor Axonal Neuropathy (AMAN) and Acute Motor and Sensory Axonal Neuropathy (AMSAN), all classified as axonal disorders. Additional rare variants of Guillain-Barré Syndrome (GBS) encompass: Miller Fisher Syndrome (MFS), paraparetic Guillain-Barré Syndrome (GBS), pharyngeal-cervical-brachial weakness, bilateral facial palsy with paresthesia (BFP), Bickerstaff brainstem encephalitis (BBE), which may overlap with MFS, polyneuritis cranialis, and acute autonomic neuropathy, which, akin to acute pure sensory neuropathy, has an indeterminate correlation with other GBS variants. [13,14] AIDP was previously a frequently occurring variant, but its occurrence has recently diminished. Acute motor axonal neuropathy (AMAN) is on the rise in South Asian nations. Furthermore, the uncommon variant of GBS, namely acute sensory axonal neuropathy (ASAN), along with the mixed form of acute polyradiculo-neuropathy, has seen an increasing patient population in recent years.[15] The GBS electrodiagnostic criteria, known as the 'Hadden criteria,' differentiate between primary demyelinating (acute inflammatory demyelinating polyradiculoneuropathy) and primary axonal subtypes (acute motor axonal neuropathy or acute motor sensory axonal neuropathy), along with inexcitable and equivocal forms.[16–18] Additionally, a revised set of electrodiagnostic criteria, known as the 'Rajabally criteria,' has recently been introduced, facilitating enhanced early electrodiagnosis using a single nerve conduction study (NCS).[19]

The extent and severity of GBS vary geographically. AIDP is the predominant type of GBS in Western countries. However, in Europe and North America, around 90–95% of individuals with Guillain-Barré syndrome (GBS) exhibit AIDP, whereas the remainder present with AMAN or AMSAN.[8,20,21] In contrast, Asia and Central and South America report higher incidences of axonal variants like AMAN, comprising 30–65% of cases.[22–24] These differences may be attributed to variations in genetic susceptibility, environmental factors, and the prevalence of specific antecedent infections.

Global studies have documented variations in the prevalence of these subtypes across different regions. In Bangladesh, existing research indicates a predominance of the axonal variant; however, comprehensive data detailing the distribution of electrophysiological subtypes within the Bangladeshi population remains scarce. Understanding the specific subtype distribution is crucial, as it influences clinical management, prognostication, and healthcare resource allocation. This study aims to determine the distribution of electrophysiological subtypes of Guillain-Barré Syndrome (GBS) in the Bangladeshi population. Specifically, it seeks to classify GBS patients using standardized nerve conduction study criteria, analyze the demographic and clinical characteristics associated with each subtype, and compare these findings with regional and global data to identify patterns unique to Bangladesh.

METHODS

It was a cross-sectional study incorporating both descriptive and analytical methods. The study was conducted between January 2016 and December 2017 in the word of Neurology at Rajshahi Medical College Hospital, Rajshahi, Bangladesh. The sample size for this study was determined using the formula: $n = \frac{z^2pq}{d^2}$, where n represents the desired sample size, Z is the standard normal deviate corresponding to a 95% confidence interval (1.96), P denotes the estimated prevalence (2.4% based on a previous study by Kutepov et. al; [25], q=1−P and d is the margin of error (5 %). Using these parameters, the calculated sample size was n=35.97. Although the calculated sample size was close to 36, we collected data from 45 participants. This sample size was sufficient to ensure statistical power and validity for the study's objectives. By using the purposive sampling technique, we collected all the data from the study population. The study included patients with a clinical diagnosis of Guillain-Barre Syndrome (GBS) referred to the Neurology Ward of Rajshahi Medical College Hospital for Nerve Conduction Studies (NCS), confirmed electrophysiologically as GBS. Patients aged over 18 years and both sexes were included. Patients with diabetes mellitus, chronic kidney or liver disease, a history of alcoholism or exposure to toxins (e.g., lead) causing neuropathy, a history of trauma affecting nerves and muscles, and pre-existing conditions such as motor neuron disease, myopathy, multifocal motor neuropathy, or genetic disorders affecting nerves and muscles were excluded. Data was collected from patients with clinically suspected Guillain-Barre Syndrome (GBS) admitted to the Neurology Ward of Rajshahi Medical College Hospital between January 2016 and December 2017. Participants were selected based on predefined inclusion and exclusion criteria. A structured questionnaire and written informed consent (available in both Bangla and English) were administered to each participant. Electrophysiological studies (Nerve Conduction Studies, NCS) were performed at least seven days after the onset of symptoms to confirm the diagnosis of GBS. Motor NCS were conducted in both upper and lower extremities, with measurements including compound muscle action potential (CMAP) amplitude, distal motor latency, nerve conduction velocity, F wave latency, and sensory nerve action potential (SNAP) amplitude. Abnormalities were confirmed by comparing results with the laboratory's normal reference values, adjusted for patient age. The procedure was conducted in the electrophysiology laboratory using an NIHON KOHDEN electrophysiology machine (Model MEB 9400K). Data was analyzed using SPSS version 16. Descriptive statistics were used to summarize baseline characteristics of the participants, expressed as mean ± standard deviation (SD) for continuous variables and as frequencies and percentages for categorical variables. Differences in proportions and means were assessed using the Chi-square test and Student t-test, respectively. For comparisons of electrophysiological findings between subtypes of Guillain-Barre Syndrome (GBS), unpaired t-tests were performed to determine statistical significance. The association between clinical features, CSF findings, and subtypes of GBS was evaluated using a chi-square test. A p-value < 0.05 was considered as significant.

RESULTS

A total of 45 patients were eligible for analysis after applying inclusion and exclusion criteria. Participants' mean age was 37.25 ± 15.40 years with a range between 18 and 80. The maximum (47%) respondents belonged to the young adult (18-40 years, 47%), followed by the middle-aged (41-64 years, 44%), and elderly group (≥65 years, 9%). Males and females were nearly equal with females being 23 persons in this study. Based on electrophysiological criteria, 25 patients (55.5%) were classified as AMAN, 17 (37.8%) as AIDP, and 3 (6.7%) patients as AMSAN. None of them were found to have been affected by MFS (Miller-Fisher Syndrome) (Table 1).

Table 1: Demographic And Clinical Characteristics of Guillain-Barré Syndrome (GBS) Patients

Variable	Frequency	Percentage
Gender		
Male	22	48.9%
Female	23	51.1%
Age (Mean ± SD)	37.25 ± 15.40	
Young Adults (18-40 years)	21	46.7%

Middle-aged (41-64 years)	20	44.4%
Elderly (≥65 years)	4	9.89%
Types of GBS		
AMAN	25	55.6%
AIDP	17	37.8%
AMSAN	3	6.6%
Preceding Infection		
Diarrhea	24	53.3%
RTI	4	8.9%
No Infection	17	37.8%

Table 2 showed that males were predominant in AMSAN (66.67%) and AMAN (52.0%), whereas females were slightly more prevalent in AIDP (58.83%). However, there was no significant association between gender and GBS subtype (p=0.65). This study found a significant association between age and GBS subtypes (p = 0.001). AMAN was most prevalent among young adults (18-40 years, 80%), while AIDP was primarily observed in middle-aged patients (41-64 years, 76. 5%). In contrast, elderly patients (≥65 years) were exclusively affected by AIDP (23.5%). A history of antecedent infections was reported and 62.22% of the patients were associated with GBS. A highly significant association was found between preceding infections and GBS subtypes (p = 0.03). Among the 53.33% of patients with a history of diarrhea, the majority had AMAN (64%) and AMSAN (66.7%). Respiratory tract infections (RTI) were observed more frequently in AMSAN (33.3%), whereas AIDP had the highest proportion of patients (58.82%) with no preceding infection history.

Table 2: Association of Demographic Characteristics and Preceding Infections Among GBS Subtypes

Variable	AMAN	AIDP	AMSAN	P value
Gender				
Male	13 (52.0%)	7 (41.17%)	2 (66.67%)	0.650
Female	12 (48.0%)	10 (58.83%)	1 (33.33%)	
Age				
Young Adults (18-40 years)	20 (80.0%)	0 (0.0%)	1 (33.3%)	
Middle-aged (41-64 years)	5 (20.0%)	13 (76.5%)	2 (66.7)	0.001
Elderly (≥65 years)	0 (0.0%)	4 (23.5%)	0 (0.0%)	
Preceding Infection (62.22%)				
Diarrhea				
Yes	6(35.30)	16 (64.0)	2 (66.7)	0.026
No	9 (36.0)	11 (64.70)	1 (33.3)	
Respiratory Tract Infection				
Yes	1 (5.88)	2 (8.0)	1 (33.3)	0.030
No	10 (58.82)	7 (28.0)	0 (0.0)	

Table 3 revealed that the CSF white blood cell (WBC) count was significantly higher in AIDP (6.12 ± 2.7 cells/mm³) compared to axonal subtypes (AMAN/AMSAN) (4.07 ± 0.9 cells/mm³, p < 0.01). In contrast, CSF protein levels were slightly higher in axonal subtypes (171.32 ± 127.6 mg/dl) compared to AIDP (144.35 ± 73.75 mg/dl); however, this difference was not statistically significant (p = 0.09). Electrophysiological assessments revealed that the CMAP (Compound Muscle Action Potential) amplitudes were significantly lower in axonal subtypes (AMAN/AMSAN) across all tested nerves (p < 0.001). In contrast, AIDP showed relatively preserved CMAP amplitudes, consistent with a demyelinating pathology. Furthermore, Nerve conduction velocity (NCV) was significantly slower in AIDP than in axonal subtypes (p < 0.001), particularly in the median, ulnar, tibial, and peroneal nerves. Similarly, Distal motor latency (DML) was significantly prolonged in AIDP (p < 0.001), confirming the presence of conduction delay, a key feature of demyelination. AIDP patients had

markedly prolonged F-latencies compared to axonal subtypes ($p < 0.001$), particularly in the right median and left tibial nerves. F-wave studies further reinforced the electrophysiological differences between the subtypes. AIDP patients exhibited markedly prolonged F-latencies compared to axonal subtypes ($p < 0.001$), particularly in the right median and left tibial nerves. In sensory NCS, latency was significantly prolonged in AIDP compared to axonal subtypes ($p < 0.001$), with marked differences in the median, ulnar, and sural nerves. Moreover, Sensory nerve amplitudes were significantly lower in AIDP than in axonal variants ($p < 0.001$).

Table 3: Comparison of Cerebrospinal Fluid (CSF) Findings and Electrophysiological Parameters Between Demyelinating and Axonal Subtypes of GBS

Category	Parameter	Demyelinating (AIDP) Mean ± SD / n (%)	Axonal (AMAN/AMSAN) Mean ± SD / n (%)	P Value
CSF Findings	WBC (cells/mm³)	6.12 ± 2.7	4.07 ± 0.9	<0.001
	Protein (mg/dl)	144.35 ± 73.75	171.32 ± 127.6	0.09
Motor Nerve Conduction				
CMAP Amplitude (mV)	Upper Limb (Rt Median)	2.86 ± 0.31	0.96 ± 0.31	<0.001
	Upper Limb (Rt Ulnar)	2.91 ± 0.31	0.95 ± 0.55	<0.001
	Lower Limb (Lt Tibial)	3.73 ± 1.77	0.79 ± 0.75	<0.001
	Lower Limb (Lt Peroneal)	2.49 ± 0.62	0.90 ± 0.74	<0.001
Conduction Velocity (m/s)	Upper Limb (Rt Median)	47.04 ± 1.48	51.65 ± 0.84	<0.001
	Upper Limb (Rt Ulnar)	46.75 ± 1.87	51.57 ± 0.56	<0.001
	Lower Limb (Lt Tibial)	46.67 ± 2.76	50.99 ± 0.51	<0.001
	Lower Limb (Lt Peroneal)	48.23 ± 1.04	52.49 ± 1.23	<0.001
Distal Motor Latency (ms)	Upper Limb (Rt Median)	6.25 ± 1.58	3.87 ± 0.61	<0.001
	Upper Limb (Rt Ulnar)	5.92 ± 0.80	2.68 ± 0.78	<0.001
	Lower Limb (Lt Tibial)	6.92 ± 1.68	4.05 ± 0.61	<0.001
	Lower Limb (Lt Peroneal)	6.32 ± 1.11	3.32 ± 0.25	<0.001
F-Latency and F-Wave Studies				
F-Latency (ms)	Median (Right)	37.91 ± 3.30	30.57 ± 1.43	<0.001
	Tibial (Left)	56.11 ± 4.88	45.25 ± 2.11	<0.001
F-Wave Occurrence	Median (Right)	31.36 ± 3.27	87.14 ± 5.34	<0.001
Sensory NCS				
Latency (ms)	Right Median	3.27 ± 0.42	2.22 ± 0.88	<0.001
	Right Ulnar	3.59 ± 0.03	2.44 ± 0.93	<0.001
	Left Sural	3.68 ± 0.14	2.63 ± 1.07	<0.001
Amplitude (mV)	Right Median	8.07 ± 4.90	22.29 ± 8.64	<0.001
	Right Ulnar	8.45 ± 3.04	20.99 ± 7.52	<0.001
	Left Sural	8.73 ± 5.45	20.21 ± 7.37	<0.001
Sensory NCS				
Latency (ms)	Right Median	3.27± 0.42	2.22± 0.88	<0.001
	Right Ulnar	3.59±0.03	2.44± 0.93	<0.001
	Left Sural	3.68 ±0.14	2.63 ±1.07	<0.001

This study also explored the differences in key electrophysiological abnormalities, including conduction block and prolonged F-wave latency, among AIDP, AMAN, and AMSAN subtypes. AIDP exhibited a significantly higher prevalence of conduction block (88.24%) compared to AMAN (24.0%) and AMSAN (33.3%) ($p < 0.001$), consistent with its demyelinating pathology. Additionally, prolonged F-wave latency (>120% of the upper limit of normal) was most frequently observed in AIDP

(41.18%), while it was rare in AMAN (4.0%) and absent in AMSAN (p = 0.006) (Table 4).

Table 4: Comparison of Conduction Block and Prolonged F-Wave Latency Among GBS Subtypes

Complication	GBS subtypes				P value
	Parameter	AIDP n (%)	AMAN n (%)	AMSAN n (%)	
Conduction Block	Present	15 (88.24)	6(24.0)	1 (33.3)	<0.001
	Absent	2 (11.76)	19 (76.0)	3 (66.7)	
PROLONG F WAVE	>120% of the upper limit of normal	7 (41.18)	1(4.0)	0 (0.0)	0.006
	<120% of the upper limit of normal	10 (58.82)	24 (96.0)	3(100.0)	

DISCUSSION

This study was conducted at a tertiary care hospital and investigated the electrophysiological subtypes of Guillain-Barré Syndrome (GBS) in the Bangladeshi population and their demographic, clinical, and electrophysiological characteristics. Our study found that AMAN was the most prevalent GBS subtype (55.6%), followed by AIDP (37.8%), and AMSAN (6.6%). These results align with previous studies in China, Japan, and other South Asian countries, which report a higher incidence of axonal subtypes compared to Western populations, where AIDP predominants.[22, 26, 27] The predominance of AMAN in Bangladesh could be attributed to environmental, genetic, and infectious factors, particularly *Campylobacter jejuni* infections, which have been associated with this subtype. Age was significantly associated with the distribution of GBS subtypes. AMAN was most common among young adults (18-40 years), whereas AIDP was more frequently observed in middle-aged (41-64 years) and elderly (≥65 years) patients. However, we didn't find any specific information to align this outcome. Some prior studies have suggested a male predominance in GBS overall, but our data did not indicate significantly a gender disparity in subtype distribution.[28, 29] This study found a significant association between preceding infections and GBS subtypes. Diarrheal illness, likely caused by *Campylobacter jejuni*, was the most common preceding infection, observed in the majority of the patients, with the highest prevalence in AMAN and AMSAN. This supports the well-established link between *Campylobacter jejuni* infections and the development of axonal GBS subtypes.[30] In contrast, respiratory tract infections (RTI) were more frequently associated with AMSAN and AIDP of the patients with no preceding infections, which is consistent with previous reports that suggest RTI is a common antecedent in demyelinating GBS.[31] CSF analysis revealed a significantly higher white blood cell (WBC) count in AIDP compared to axonal subtypes (AMAN/AMSAN). This finding is consistent with previous studies showing that albuminocytological dissociation (elevated protein without significant pleocytosis) is a hallmark of GBS, but the extent of CSF WBC elevation is not as pronounced in AIDP. However, this distinction may align with findings from previous studies, which have reported that while albuminocytological dissociation is common across GBS variants, the extent of CSF protein elevation and WBC count can differ based on the specific pathophysiological mechanisms involved.[32] Electrophysiological parameters demonstrated marked differences between axonal and demyelinating GBS subtypes. Compound Muscle Action Potential (CMAP) amplitudes were significantly lower in axonal subtypes (AMAN/AMSAN) across all tested nerves ($p<0.001$), confirming the characteristic axonal degeneration observed in these variants. In contrast, AIDP exhibited preserved CMAP amplitudes but significantly reduced nerve conduction velocity (NCV), prolonged distal motor latency (DML), and delayed F-wave latencies, findings indicative of demyelination. These electrophysiological differences are critical for the accurate classification of GBS subtypes, as they have direct implications for prognosis and therapeutic approaches.[33–35] Additionally, we found conduction block and prolonged F-wave latency were the hallmark features of AIDP, distinguishing it from axonal variants. In AIDP, immune-mediated demyelination may disrupt saltatory conduction, leading to conduction blocks and delayed F-wave latencies. Conversely, AMAN and AMSAN primarily

involve axonal degeneration, where the myelin sheath remains intact, resulting in less frequent conduction blocks and normal F-wave latencies. However, reversible conduction failure can occur in AMAN's early stages, potentially causing transient conduction blocks. In Addition, Sumaira et. al; also found that prolonged F-wave latencies were observed exclusively in AIDP cases, while conduction blocks were rare in AMAN patients.[36] This underscores the role of demyelination in AIDP and axonal involvement in AMAN.

Limitations of the Study

Despite providing valuable insights into the electrophysiological subtypes of Guillain-Barré Syndrome (GBS) in the Bangladeshi population, this study has several limitations. First, the sample size was relatively small, which may limit the generalizability of the findings. A larger cohort could provide a more comprehensive understanding of subtype distribution. Second, the study was conducted in a single tertiary care hospital, making the findings not fully representative of the broader Bangladeshi population. Finally, potential confounding factors such as genetic predisposition and environmental influences were not extensively analyzed, which could impact the interpretation of results.

CONCLUSION

This study provides crucial insights into the electrophysiological subtypes of Guillain-Barré Syndrome (GBS) in the Bangladeshi population, highlighting the predominance of the axonal variant, particularly Acute Motor Axonal Neuropathy (AMAN). The findings align with regional trends and emphasize the role of preceding infections, especially diarrhea, in subtype variation. Significant differences in electrophysiological parameters between axonal and demyelinating subtypes were observed, reinforcing their distinct pathophysiological mechanisms. Understanding these variations is essential for improving diagnostic accuracy and treatment strategies. Further large-scale, multi-center studies are needed to confirm these findings and explore genetic and environmental influences on GBS subtypes.

Funding

The study was conducted without any financial support or specific grants from public, commercial, or non-profit funding agencies.

Ethical Approval

This study was approved by the Institutional Review Board (IRB) of Bangabandhu Sheikh Mujib Medical University (BSMMU).

Conflict of Interest: There is no conflict of interest.

Consent for Publication

All authors have reviewed and approved the final manuscript for publication. They confirm that the study findings can be publicly shared without restrictions.

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