

ORIGINAL ARTICLE

Clinical profile and predictors of outcomes of guillain-barré syndrome variants among patients admitted in tertiary care hospital.

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ABSTRACT... Objective: To evaluate the clinical profile, outcomes, and independent predictors of poor prognosis in patients diagnosed with Guillain-Barré Syndrome. **Study Design:** Cross-sectional Observational study. **Setting:** Department of Neurology, Nishtar Hospital, Multan. **Period:** November 2024 to April 2025. **Methods:** A total of 134 adult patients diagnosed with GBS were enrolled using non-probability consecutive sampling. Data were collected prospectively through structured clinical assessment, nerve conduction studies, and cerebrospinal fluid analysis. Outcome was measured using the Hughes Disability Score. Statistical analysis was performed in SPSS v26.0, applying chi-square test and binary logistic regression with $p < 0.05$ considered significant. **Results:** Among 134 Guillain-Barré Syndrome patients, 72.4% were aged 18–45 years and 63.4% were male. Gastrointestinal infection (40.3%) was the most common antecedent. Acute Inflammatory Demyelinating Polyradiculoneuropathy (43.3%) was the predominant variant; albuminocytologic dissociation was present in 79.1%. Symmetric ascending weakness (91.0%), quadriparesis (67.9%), and facial palsy (29.9%) were frequent. Mechanical ventilation (21.6%) and ICU admission (30.6%) were required. Good outcome occurred in 72.4% of cases. Poor outcome (27.6%) was significantly associated with older age ($p < 0.001$), subacute progression ($p = 0.039$), absent ACD ($p < 0.001$), neck weakness, ventilation, and ICU admission ($p < 0.05$). **Conclusion:** Favorable outcomes in GBS were associated with early presentation and AIDP subtype, while poor prognosis correlated with old age, delayed admission, absence of albuminocytologic dissociation, and ICU care.

Key words: Albuminocytologic Dissociation, Clinical Profile, Guillain-Barré Syndrome, Intravenous Immunoglobulin, Outcome Predictors.

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INTRODUCTION

Guillain-Barré Syndrome (GBS) represents a complex, immune-mediated acute polyradiculoneuropathy characterized by its remarkable clinical heterogeneity and potentially devastating neurological consequences.¹ Globally, the syndrome manifests with an annual incidence ranging from 0.4 to 3 per 100,000 population, with notable geographic variations and increasing prevalence in certain regions.² The disease demonstrates a distinct epidemiological profile, with risk incrementally rising by approximately 20% per decade of life and a notable male preponderance, presenting a male to female ratio of 1.5–2.3:1.³ Risk factors encompass a broad spectrum of demographic and physiological variables, with age, prior infections, and individual immunological susceptibility playing crucial roles in syndrome manifestation.⁴

The clinical spectrum of GBS is varied and influenced by geographic, environmental, and genetic factors.⁵ In developed countries, Acute Inflammatory Demyelinating Polyradiculoneuropathy (AIDP) is the predominant subtype, whereas Acute Motor Axonal Neuropathy (AMAN) and Acute Motor and Sensory Axonal Neuropathy (AMSAN) are more common in Asian regions including India, Pakistan, Bangladesh, and China, with reported axonal variant incidences ranging from 28% to 67%.^{6,7} The axonal variants, particularly AMAN and AMSAN, are frequently linked with more rapid progression, severe disease, and poorer prognosis compared to demyelinating forms.⁸

Typically, GBS is prefaced by an antecedent illness, most commonly gastrointestinal or respiratory infections, notably *Campylobacter jejuni*.

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These infections are hypothesized to trigger immune-mediated injury through molecular mimicry, resulting in demyelination or axonal degeneration.^{9,10} Clinically, patients may present with ascending weakness, paresthesia, cranial nerve palsies, bulbar dysfunction, respiratory distress, and autonomic instability.¹¹ The disease course is variable, ranging from full recovery to prolonged disability or death. Early cranial nerve involvement, rapid disease progression (within 7 days), respiratory failure, and axonal electrophysiological patterns are poor predictors of outcomes.^{12,13}

Early diagnosis and classification using nerve conduction studies (NCS) and cerebrospinal fluid (CSF) analysis are pivotal. NCS not only confirms the diagnosis but also classifies the disease into electrophysiological subtypes and aids in prognostication. AIDP typically shows demyelinating features, while axonal variants exhibit low or absent CMAP amplitudes. The presence of albuminocytologic dissociation in CSF is seen in over 80% of cases by the second week of illness.¹³ Despite the availability of specific immunotherapies such as IVIg and plasmapheresis, which are most effective when initiated early, outcomes in GBS remain variable. Approximately 20–30% of patients need mechanical ventilation, and 3–14% die during the acute phase, especially in cases with rapid disease progression, bulbar involvement, or autonomic dysfunction.¹⁴

In South Asia, particularly in Pakistan, data on the clinical spectrum, electrophysiological variants, and outcome predictors of GBS are limited. Existing regional studies report a high prevalence of axonal variants and young male predominance, but prognostic indicators remain inconsistently described. Given these gaps, this study aims to describe the clinical and demographic profile of adult GBS patients, determine the frequency of electrophysiological variants using NCS, evaluate outcomes including recovery, ICU admission, need for mechanical ventilation, and in-hospital mortality, and identify predictors of poor outcomes.

METHODS

A cross-sectional observational study was executed at Nishtar Hospital, Multan, in the Neurology

Department from November 2024 to April 2025. Ethical approval for the study was obtained from the Institutional Ethical Review Board of Nishtar Medical University, Multan (Approval Reference No. 18937/NMU), and informed consent was taken from all patients before enrollment. A non-probability consecutive sampling technique was utilized for participant enrollment. A sample size of 134 patients was calculated using WHO sample size calculator with a confidence interval of 95%, expected proportion of AMAN as 14.6% and absolute precision of 6%.¹⁵

Inclusion and Exclusion Criteria

Patients aged 18 to 75 years, diagnosed with Guillain-Barré Syndrome as per Brighton Collaboration criteria and confirmed by NCS and CSF analysis (CSF protein >45 mg/dL with leukocyte count <50/mm³), were included after informed consent. Only those admitted within 2 weeks of symptom onset were enrolled. Patients with alternative causes of acute flaccid paralysis (e.g., poliomyelitis, botulism, myasthenia gravis, vasculitic or toxic neuropathies), central nervous system disorders, or incomplete diagnostic evaluation were excluded. Additionally, patients who were pregnant or lactating, HIV reactive, known to have selective IgA deficiency, or receiving immunosuppressive therapy including recent corticosteroid use were not included.

Data Collection Procedure

A proforma was used to gather the baseline demographic and clinical data, including age, sex, residence and antecedent illnesses. Duration from symptom onset to admission and disease progression classified as acute (≤ 7 days) or subacute (8–14 days) were noted. A comprehensive neurological exam was performed. Motor strength was graded using the MRC scale (0–5). Symmetric ascending weakness was defined by bilateral limb involvement with ≤ 1 grade inter-limb difference. Paraparesis involved lower limbs only, while quadriparesis included all four limbs. Neck flexor weakness was identified as inability to lift the head against gravity (MRC ≤ 3). Muscle pain in proximal regions was considered significant if VAS score was ≥ 4 . Sensory findings including paresthesia were confirmed through light touch, pinprick, and vibration testing with a 128 Hz tuning fork.

Cranial nerve involvement was assessed clinically. Facial palsy was graded \geq II on the House-Brackmann scale. Ophthalmoplegia was based on restricted eye movements. Dysphagia was scored \geq 3 using the DOSS, and dysarthria was noted with a Frenchay score \geq 2. Bulbar weakness was recorded based on clinical signs and bedside swallow assessment. Autonomic features included bladder dysfunction—defined as urinary retention (post-void residual \geq 100 mL) or incontinence—and blood pressure fluctuations, defined as systolic changes \geq 30 mmHg or diastolic \geq 15 mmHg over 24 hours, monitored every 4 hours. NCS was performed and interpreted by neurophysiologists to classify patients into specific GBS variants, including AIDP, AMAN, AMSAN and MFS variant. Outcomes were assessed using the Hughes Disability Score (0–6) at admission and discharge. Good outcome was defined as HDS \leq 2, confirmed at discharge. ICU admission was based on clinical need—breath count \leq 20, autonomic instability, bulbar dysfunction, or respiratory compromise. In-hospital death was also. Data were collected prospectively by trained investigators under supervision, ensuring accuracy and adherence to predefined clinical and diagnostic criteria.

Statistical Analysis

Data were analyzed using SPSS version 26.0. Quantitative variables (age, duration from onset to admission, and Hughes Disability Scores) were presented as mean \pm standard deviation and compared using the independent samples t-test. Categorical variables (gender, residence, clinical features, treatment, and outcomes) were expressed as frequencies and percentages and compared using the Chi-square test. Variables with $p < 0.05$ in Chi-square analysis were included in a binary logistic regression model using backward stepwise likelihood ratio method to identify independent predictors of poor outcome. Adjusted odds ratios (ORs) with 95% confidence intervals (CIs) were reported. A p -value < 0.05 was considered statistically significant.

RESULTS

The mean age of patients diagnosed with GBS was 39.87 ± 13.04 years. The average duration from symptom onset to hospital admission was 6.61

± 2.95 days. Among 134 patients with GBS, the majority were aged 18–45 years (72.4%), male (63.4%), and residing in urban areas (61.2%). The most frequent antecedent illness was gastrointestinal infection (40.3%). Acute disease progression (\leq 7 days) was observed in 72.4% of patients, and albuminocytologic dissociation was present in 79.1%. AIDP was the predominant variant (43.3%), followed by AMAN (29.9%) and AMSAN (13.4%). IVIG was the most administered therapy (50.0%), and 21.6% required mechanical ventilation. Good outcome was achieved in 72.4% of patients, whereas 7.5% succumbed to illness. Detailed distribution of baseline and clinical characteristics is provided in Table-I.

TABLE-I

Baseline demographic and clinical characteristics of patients diagnosed with guillain-barré syndrome (n = 134)

Variable	n (%)
Age Group	
18 to 45 years	97 (72.4)
46 to 75 years	37 (27.6)
Gender	
Male	85 (63.4)
Female	49 (36.6)
Residence	
Urban	82 (61.2)
Rural	52 (38.8)
Antecedent Illness	
Gastrointestinal Infection	54 (40.3)
Respiratory Infection	31 (23.1)
Fever	25 (18.7)
Surgery	9 (6.7)
None	15 (11.2)
Disease Progression	
Acute (\leq 7 days)	97 (72.4)
Subacute ($>$ 7 days)	37 (27.6)
Albuminocytologic Dissociation	106 (79.1)
GBS Variants	
AIDP	58 (43.3)
AMAN	40 (29.9)
AMSAN	18 (13.4)
MFS	3 (2.2)
Not Classified	15 (11.2)
Motor Features	

Symmetric Ascending Weakness	122 (91.0)
Paraparesis	16 (11.9)
Quadriparesis	91 (67.9)
Neck Flexor Weakness	60 (44.8)
Muscle Pain	45 (33.6)
Sensory Feature	
Paresthesia	45 (33.6)
Cranial Nerve Involvement	
Facial Palsy	40 (29.9)
Ophthalmoplegia	6 (4.5)
Dysarthria	21 (15.7)
Dysphagia	16 (11.9)
Bulbar Weakness	18 (13.4)
Autonomic Features	
Bladder Involvement	12 (9.0)
BP Fluctuations	15 (11.2)
Treatment Received	
IVIg Only	67 (50.0)
Plasmapheresis Only	19 (14.2)
Both (IVIg + Plasmapheresis)	9 (6.7)
Supportive Care Only	39 (29.1)
Need for Mechanical Ventilation	
ICU Admission	41 (30.6)
Outcome	
Good Outcome	97 (72.4)
Poor Outcome	37 (27.6)
Mortality	
	10 (7.5)

AIDP – Acute Inflammatory Demyelinating Polyneuropathy; AMAN – Acute Motor Axonal Neuropathy; AMSAN – Acute Motor-Sensory Axonal Neuropathy; MFS – Miller Fisher Syndrome; IVIG – Intravenous Immunoglobulin; BP – Blood Pressure.

The mean age was lower in patients with good outcomes (37.16 ± 10.70 years) compared to those with poor outcomes (46.97 ± 15.85 years). Similarly, the average duration from symptom onset to admission was shorter in good outcome group (5.62 ± 2.15 days) than in the poor outcome group (9.22 ± 3.20 days). The mean Hughes Disability Score at admission was 3.63 ± 0.49 in the good outcome group versus 4.76 ± 0.44 in the poor outcome group, while at discharge it was 1.00 ± 0.61 and 3.76 ± 0.44 , respectively ($p < 0.001$).

Patients aged 46–75 years had significantly higher

poor outcomes compared to younger patients ($p < 0.001$). Subacute disease progression (>7 days) was more frequent among those with poor outcomes (40.5% vs. 22.7%, $p = 0.039$). Absence of albuminocytologic dissociation was significantly associated with poor outcomes (43.2% vs. 12.4%, $p < 0.001$). Among GBS variants, AMAN and AMSAN were more common in poor outcome groups, whereas AIDP was predominant in those with good recovery ($p = 0.009$). Neck flexor weakness ($p = 0.012$), paresthesia ($p = 0.023$), facial palsy ($p = 0.001$), bulbar weakness ($p = 0.022$), and autonomic features including bladder involvement ($p < 0.001$) and blood pressure fluctuations ($p = 0.003$) were significantly linked with poor outcomes. Supportive care alone was linked to a higher proportion of poor outcomes, while patients receiving both IVIG and plasmapheresis showed only good outcomes ($p = 0.006$). Need for mechanical ventilation ($p = 0.001$), ICU admission ($p < 0.001$), and in-hospital mortality ($p < 0.001$) were also significantly linked with adverse outcomes (Table-II).

TABLE-II

Association of baseline demographic and clinical characteristics with patient outcomes in guillain-barré syndrome (GBS) (n = 134)

Characteristics	Good Outcome n = 97	Poor Outcome n = 37	P-Value	
Age Group				
18 to 45 years	79 (81.4%)	18 (48.6%)	< 0.001	
46 to 75 years	18 (18.6%)	19 (51.4%)		
Gender				
Male	64 (66.0%)	21 (56.8%)	0.322	
Female	33 (34.0%)	16 (43.2%)		
Residence				
Urban	60 (61.9%)	22 (59.5%)	0.799	
Rural	37 (38.1%)	15 (40.5%)		
Antecedent Illness				
Gastrointestinal Infection	45 (46.4%)	9 (24.3%)	0.019	
Respiratory Infection	21 (21.6%)	10 (27.0%)		
Fever	19 (19.6%)	6 (16.2%)		
Surgery	3 (3.1%)	6 (16.2%)		
None	9 (9.3%)	6 (16.2%)		
Disease Progression				
				0.039

Acute (≤ 7 days)	75 (77.3%)	22 (59.5%)	
Subacute (> 7 days)	22 (22.7%)	15 (40.5%)	
Albuminocytologic Dissociation	85 (87.6%)	21 (56.8%)	< 0.001
GBS Variants			0.009
AIDP	49 (50.5%)	9 (24.3%)	
AMAN	24 (24.7%)	16 (43.2%)	
AMSAN	9 (9.3%)	9 (24.3%)	
MFS	3 (3.1%)	0 (0.0%)	
Not Classified	12 (12.4%)	3 (8.1%)	
Motor Features			
Symmetric Ascending Weakness	88 (90.7%)	34 (91.9%)	0.832
Paraparesis	12 (12.4%)	4 (10.8%)	0.803
Quadriparesis	69 (71.1%)	22 (59.5%)	0.196
Neck Flexor Weakness	37 (38.1%)	23 (62.2%)	0.012
Muscle Pain	37 (38.1%)	8 (21.6%)	0.070
Sensory Feature			
Paresthesia	27 (27.8%)	18 (48.6%)	0.023
Cranial Nerve Involvement			
Facial Palsy	21 (21.6%)	19 (51.4%)	0.001
Ophthalmoplegia	6 (6.2%)	0 (0.0%)	0.122
Dysarthria	12 (12.4%)	9 (24.3%)	0.089
Dysphagia	9 (9.3%)	7 (18.9%)	0.124
Bulbar Weakness	9 (9.3%)	9 (24.3%)	0.022
Autonomic Features			
Bladder Involvement	3 (3.1%)	9 (24.3%)	< 0.001
BP Fluctuations	6 (6.2%)	9 (24.3%)	0.003
Treatment Received			0.006
IVIg Only	54 (55.7%)	13 (35.1%)	
Plasmapheresis Only	13 (13.4%)	6 (16.2%)	
Both (IVIg + Plasmapheresis)	9 (9.3%)	0 (0.0%)	
Supportive Care Only	21 (21.6%)	18 (48.6%)	
Need for Mechanical Ventilation	14 (14.4%)	15 (40.5%)	0.001
ICU Admission	19 (19.6%)	22 (59.5%)	< 0.001
Mortality	0 (0.0%)	10 (27.0%)	< 0.001

Pearson Chi-Square test was applied to assess the association

between clinical and demographic characteristics and outcomes in patients with GBS. A p-value < 0.05 was considered statistically significant.

Binary logistic regression analysis identified several independent predictors of poor outcome in patients with GBS. Age between 46 and 75 years was significantly linked with higher odds of poor recovery (AOR = 5.053; 95% CI: 1.621–15.750; $p = 0.005$). Subacute disease progression exceeding seven days also increased the likelihood of poor outcome (AOR = 6.201; 95% CI: 1.925–19.979; $p = 0.002$). Absence of albuminocytologic dissociation was a strong negative prognostic indicator (AOR = 8.527; 95% CI: 2.502–29.061; $p = 0.001$). Conversely, the presence of neck flexor weakness (AOR = 0.175; 95% CI: 0.053–0.574; $p = 0.004$), the need for mechanical ventilation (AOR = 0.208; 95% CI: 0.062–0.701; $p = 0.011$), and ICU admission (AOR = 0.099; 95% CI: 0.031–0.321; $p < 0.001$) were significantly correlated with unfavorable outcomes (Table-III).

TABLE-III

Binary Logistic Regression Analysis for Predictors of Poor Outcome in Patients with Guillain-Barré Syndrome (n = 134)

Predictors	P-Value	Adjusted Odds Ratio	95% CI
Age Group (46–75 years)	0.005	5.053	1.621 – 15.750
Disease Progression (>7 days)	0.002	6.201	1.925 – 19.979
Albuminocytologic Dissociation (No)	0.001	8.527	2.502 – 29.061
Neck Flexor Weakness (Yes)	0.004	0.175	0.053 – 0.574
Mechanical Ventilation (Yes)	0.011	0.208	0.062 – 0.701
ICU Admission (Yes)	<0.001	0.099	0.031 – 0.321

Binary logistic regression using the Enter method was applied to identify independent predictors of poor outcome among patients with GBS. The variables retained in the final model were age group, disease progression duration, absence of albuminocytologic dissociation, presence of neck flexor weakness, need for mechanical ventilation, and ICU admission. A p-value < 0.05 was considered statistically significant.

DISCUSSION

This study analyzed the clinical profile, management strategies, and outcomes of 134 patients diagnosed with GBS at a tertiary care hospital in Pakistan. The average age of patients was 39.87 ± 13.04 years, with the majority (72.4%) aged between 18 and 45 years. These findings are aligned with those of Tewedaj et al., who reported 76.7% of their patients in the 14 to 34 year range, and Siddiqui et al., where 61% were under 40 years.^{13,16} In contrast, Rajabally and Uncini reported a wider age distribution (18–84 years), with increased mortality in older adults. Importantly, advancing age was observed as an independent predictor of poor outcome in the present study (AOR = 5.053; 95% CI: 1.621–15.750; $p = 0.005$), consistent with Kalita et al. and their finding of age ≥ 50 years as a significant prognostic factor (OR = 1.96; $p = 0.03$).^{7,17}

A history of gastrointestinal illness was the most common antecedent factor in the present study (40.3%), followed by respiratory infections (23.1%) and fever (18.7%). These findings are aligned with Siddiqui et al., who reported gastrointestinal symptoms in 21% and respiratory illness in 14.5%. Kalita et al. similarly noted diarrhea in 19.4% and sore throat or URTI in 20.7% of their cases.^{7,13} Acute progression (≤ 7 days) was observed in 72.4% of the study population. Siddiqui et al. reported similar findings, with 71% presenting within 7 days. Kalita et al. recorded a median symptom onset of 8 days, while Tewedaj et al. documented a mean of 8.77 ± 7.25 days.^{7,13,16} In the present study, subacute progression (> 7 days) was significantly correlated with poor outcome ($p = 0.039$), and regression analysis confirmed this association (AOR = 6.201; $p = 0.002$). A similar association was highlighted by Rafique et al. (2023) (OR = 3.2; $p = 0.01$).¹⁸

AIDP was the predominant GBS variant in this study (43.3%), followed by AMAN (29.9%) and AMSAN (13.4%). These results are comparable to those reported by Siddiqui et al. (AIDP: 53%, AMAN: 29%, AMSAN: 11%) and Rafique et al. (AIDP: 41.3%, AMAN: 26.1%, AMSAN: 30.4%).^{13,18} In the current study, AMAN and AMSAN were significantly more prevalent in poor outcome groups ($p = 0.009$). Albuminocytologic dissociation (ACD) was detected in 79.1% of patients. This rate closely matches

findings by Ruiz-Sandoval et al. (81%) and Tewedaj et al. (82.9%). Siddiqui et al. reported ACD in 64% of patients who underwent lumbar puncture.^{13,16,19} In this study, absence of ACD was an independent predictor of poor outcome (AOR = 8.527; $p = 0.001$), highlighting its prognostic relevance.

Symmetric ascending weakness was observed in 91.0% of patients, comparable to findings by Siddiqui et al. (93%) and Sharma et al. (90%).^{13,20} Quadriparesis and paraparesis were observed in 67.9% and 11.9% of cases, respectively. Notably, neck flexor weakness was significantly associated with poor outcome ($p = 0.012$), and also emerged as a negative prognostic marker in regression analysis (AOR = 0.175; $p = 0.004$), in line with Verma et al. and Khedr et al.^{20,21} Sensory involvement (paresthesia) was documented in 33.6% of patients, higher than the 3% reported by Siddiqui et al. but similar to Sharma et al. (31.7%) and Kalita et al. (52.7%). Among cranial nerve findings, facial palsy (29.9%) and bulbar weakness (13.4%) were significantly correlated with poor outcome ($p = 0.001$ and $p = 0.022$, respectively), consistent with Kalita et al. and Rafique et al., who all observed strong associations with disease severity and ventilation requirements.^{7,20}

Treatment modalities showed 50% of patients receiving IVIG, 14.2% plasmapheresis, 6.7% both, and 29.1% managed with supportive care alone. Patients treated with IVIG or plasmapheresis had better outcomes, while supportive care alone was associated with poor prognosis ($p = 0.006$). Bhatia et al. similarly reported the protective role of both treatments in outcome improvement. However, studies by Kalita et al. and Tewedaj et al. reported no significant outcome differences by treatment type, possibly due to delayed therapy.^{7,16} Mechanical ventilation was needed in 21.6% of patients and was independently associated with poor outcome (AOR = 0.208; $p = 0.011$), consistent with findings from Verma et al., Sharma et al. and Tewedaj et al. ICU admission was also significantly associated with poor prognosis (AOR = 0.099; $p < 0.001$), aligning with Ahmed et al. and Singh et al.^{14,19}

This study offers comprehensive analysis of clinical predictors and outcomes in Guillain-Barré

Syndrome, integrating both bivariate and multivariate statistical models with a relatively robust sample size. However, limitations include its single-center design and short-term outcome assessment. Moreover, potential confounding variables such as comorbidities were not explored. Future multicenter studies with extended follow-up, inclusion of neurophysiological and biomarker profiling, and assessment of long-term disability and quality of life are recommended to validate and expand upon the current findings for improved risk stratification and patient care.

CONCLUSION

This study identified key clinical and demographic predictors influencing outcomes in patients with Guillain-Barré Syndrome. Younger age, early presentation, presence of albuminocytologic dissociation, and absence of neck flexor weakness were significantly associated with favorable outcomes. In contrast, age above 45 years, subacute progression, absence of albuminocytologic dissociation, need for mechanical ventilation, and ICU admission independently predicted poor outcomes. These findings underscore the importance of early diagnosis, timely intervention, and prognostic stratification to improve clinical outcomes in GBS patients.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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AUTHORSHIP AND CONTRIBUTION DECLARATION

1	Anahita Khan: Conception and design of study, methodology, data collection, and final approval of the manuscript.
2	Sohaib Hassan: Study design methodology, data collection, and final approval of the manuscript.
3	Muhammad Ali Qureshi: Data analysis, methodology, interpretation and final approval of the manuscript.
4	Muhammad Irfan Jamil: Data analysis, results formulation, discussion writing and final approval.
5	Adeel Ahmed: Study design methodology, data collection, and final approval of the manuscript.
6	Maria Jabeen: Data analysis, methodology, interpretation and final approval of the manuscript.