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UNDERSTANDING THE GUILLAIN-BARRÉ SYNDROME SURGE IN MUMBAI IN CONTEXT OF THE RECENT PUNE GBS OUTBREAK - A CLINICAL AND ELECTROPHYSIOLOGICAL PROFILE

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ABSTRACT **Introduction:** During early 2025, a large cluster of Guillain–Barré Syndrome (GBS) cases was reported from Pune, India (225 cases, 11 deaths). Concurrently, an increased number of GBS admissions were observed at our tertiary care centre in Mumbai, prompting systematic evaluation. **Objective:** To describe the clinical profile, electrophysiological patterns, management, and short-term outcomes of GBS patients admitted between January and June 2025. **Methods:** This was a prospective, single-centre observational cohort study. Twenty consecutive patients diagnosed with GBS were included. Clinical features, antecedent events, electrophysiological subtypes, ICU requirement, ventilatory support, hospital stay, and outcomes were assessed using the Hughes Disability Scale at discharge and 28-day follow-up. Descriptive statistics were applied. **Results:** The mean age was 38.5 years, with male predominance (65%). Antecedent events were identified in 55%, most commonly gastrointestinal infections. Cranial nerve involvement occurred in 50%, and autonomic dysfunction in 35%. Electrophysiological studies (n=18) showed axonal variants in 10 patients and demyelinating variants in 5; three were normal. Six patients (30%) required invasive mechanical ventilation. The mean hospital stay was 19.4 days. In-hospital mortality was 25%. Among survivors, only 30% achieved complete recovery at 28 days. **Conclusions:** This study highlights a parallel surge of severe GBS cases in Mumbai during the Pune outbreak, marked by axonal predominance, high ICU utilisation, significant mortality, and early disability, underscoring the need for regional surveillance, early intensive care, and structured rehabilitation.

KEYWORDS : Guillain–Barré syndrome; Axonal neuropathy; Intensive Care; Mechanical Ventilation; Electrophysiology; India

INTRODUCTION

Guillain–Barré Syndrome (GBS) is an acute, immune-mediated polyradiculoneuropathy and a leading cause of acute flaccid paralysis worldwide. It typically presents with rapidly progressive limb weakness and may progress to bulbar or respiratory involvement requiring intensive care. Despite immunomodulatory therapy, morbidity remains significant. GBS has a global incidence of 1–2 cases per 100,000 population per year.[4] In January 2025, a large cluster of GBS cases was reported from Pune district, with 225 cases and 11 deaths.[3] During the same period, a surge of GBS cases was observed in our tertiary care centre in Mumbai, prompting the present study.

Subjects and Methods

This was a prospective, observational study conducted at a tertiary care centre in Mumbai, India, between January and June 2025. Patients of all ages who were admitted with a clinical diagnosis of Guillain–Barré Syndrome (GBS), based on the Brighton diagnostic criteria, were included.[4,7,8] Written informed consent was obtained from all participants or their legal guardians. Institutional ethics committee permission for this study was obtained. (IEC/258/25)

A total of 21 consecutive patients with suspected GBS were enrolled. One patient was subsequently excluded as she was diagnosed as Myasthenia Gravis. Data (20 patients) was collected using a structured clinical case form that included demographic details, comorbidities, antecedent infections or vaccinations within the preceding four weeks, and travel history. Clinical features at presentation, including motor weakness, cranial nerve involvement, sensory findings, autonomic dysfunction, and the need for ICU or ventilator support, were recorded. Neurological examination findings were documented using the Medical Research Council (MRC) muscle grading system and the Hughes Disability Scale[5,9].

Routine laboratory investigations including serum electrolytes cerebrospinal fluid (CSF) analysis, and infectious marker screening (HIV, hepatitis B surface antigen [HBsAg], hepatitis C virus [HCV], SARS-CoV-2, and diphtheria) were performed. Urine porphobilinogen testing was done to exclude porphyria when clinically suspected. Electrophysiological studies [nerve conduction study (NCS) and electromyography (EMG)] were conducted for 18 patients (two patients needed invasive ventilation soon after admission, hence could not be mobilised to the NCS room). Subtypes of GBS (demyelinating, axonal, or mixed) were determined based on established electrodiagnostic criteria.

All patients received standard care in accordance with institutional GBS management protocols. Treatment modalities included intravenous immunoglobulin (IVIG), supportive therapy, and early initiation of physiotherapy. ICU admission (19/20 patients) and mechanical ventilation were provided based on clinical indications. The duration of hospital stay, ICU stay, and ventilator support were recorded. Functional outcomes were assessed at discharge and telephonically on day 28 using the Hughes Disability Scale (HDS)[5]. Any complications, including those related to GBS or ICU stay, were also documented.

Descriptive statistics were used to summarize baseline characteristics and outcomes. Categorical variables were expressed as counts and percentages, while continuous variables were reported as mean ± standard deviation or median with interquartile range, as appropriate. Data analysis was performed using Microsoft Excel and cross-verified for consistency.

RESULTS

A total of twenty patients with confirmed diagnosis of Guillain-Barré Syndrome (GBS) were included in this observational study conducted at a tertiary care centre in Mumbai. Table 1 and 2 outlines the patient profile and clinical features respectively (n=20). The average age of the patients was 38.5 years, with the youngest being 12 and the oldest 75 years old. 11 out of 20 patients were in the working age population (19-59 years).

Analysing the geographical presentation, 13 were from Mumbai city and suburbs. Of the 6 patients outside Mumbai, 2 each were from Navi Mumbai, and Kalyan, where as one each was from Kalwa and Bhiwandi. These areas are within 100-150 square kilometre area of Mumbai. Only one patient was from Belgavi, Karnataka (440 km from Mumbai). Three patients had a travel history, 2 to Pune and one to Bhuj, Gujarat in last 2 weeks.

As outlined, 10 patients had a history of preceding infection, while one patient had received both Hepatitis B and Influenza Vaccine within last

4 weeks of illness. Seven patients were found to have co-morbid conditions, such as diabetes mellitus, hypertension, thyroid disorders, and chronic obstructive pulmonary disease. One patient was diagnosed with HIV during hospitalization, whereas two patients had a prior history of similar neurological illness.

Clinically, 15 out of 20 patients had a classical presentation of GBS with ascending lower motor neuron paraparesis. 4 out of 20 had a descending progression and one presented as a pharyngeal-cervical-brachial (PCB) variant. The patients had a varying degree of motor weakness while the average power scores for both upper and lower limbs were 2-3. Cranial nerve involvement was seen in ten patients. Among them, four had isolated bulbar dysfunction, one had isolated facial nerve involvement, four had both facial and bulbar nerve involvement, and one patient showed involvement of the facial, bulbar, and oculomotor nerves. Autonomic dysfunction was observed in seven patients. Out of these, two had tachycardia, two had blood pressure instability, one had excessive perspiration, one had a combination of tachycardia and blood pressure instability, and one had both perspiration and blood pressure fluctuations.

Cerebrospinal fluid (CSF) analysis was done in 19 patients; of which 9 showed albumin-cytological dissociation. One patient refused consent for lumbar puncture. Nerve conduction studies were performed in 18 out of 20 patients. These tests helped classify five patients as having demyelinating variants of GBS and ten as having axonal variants, including 6 Acute Motor Axonal Neuropathy (AMAN) and 4 Acute Motor Sensory Axonal Neuropathy (AMSAN), 3 had normal NCS. In addition, all patients were screened for relevant infectious markers, including COVID-19, HIV, hepatitis B and C, diphtheria, and porphyria, through throat swabs and urine analysis and one was diagnosed to be HIV positive.

The mean duration of hospital stay was 19.4 days, with the longest stay lasting up to 60 days. The average ICU stay was 10.05 days, with the maximum being 30 days. Only one patient with PCB variant of GBS did not need ICU stay and was managed in the general medical ward. Six patients required ventilator support, with a mean duration of 11.8 days of mechanical ventilation. All 20 patients received intravenous immunoglobulin (IVIG) within 48 hours of admission in standard weight adjusted dose (2gm/kg divided over 5 days). Appropriate supportive care including DVT prophylaxis and physiotherapy was initiated early in all patients and continued through their recovery.

The functional outcome at discharge was measured using the Hughes Disability Scale (HDS). About 30% percent of patients were still bedridden at the time of discharge. 25 percent (5 out of 20 patients) required assistance with walking or needed a wheelchair, while only 20 percent (4 out of 20 patients) showed minor symptoms or had recovered fully. A total of 5 patients succumbed to various complications including pneumonia with sepsis (3 out of 5) and labile blood pressure due to autonomic dysfunction (2 out of 5). At the 28-day follow-up, clinical improvement was noted in 9 out of 15 discharged patients. However, 6 out of 15 patients continued to suffer from residual limb weakness, which contributed to long-term morbidity.

DISCUSSION

The present observational study provides important insights into the clinical spectrum, severity, and short-term outcomes of Guillain–Barré Syndrome (GBS) cases admitted to a tertiary care centre in Mumbai during a period that coincided with the widely reported GBS outbreak in Pune in early 2025. Although Mumbai did not experience a formally declared outbreak, the temporal clustering of cases, their clinical severity, and the observed mortality suggest that the city may have been indirectly influenced by the same epidemiological drivers responsible for the Pune surge.

The mean age of 38.5 years with a male predominance in our cohort is consistent with prior Indian studies describing GBS as a disease predominantly affecting young and middle-aged adults.[1,10,11] Similar age distributions have been reported by Sharma et al. and Banerjee et al. during the recent Pune GBS outbreak, where approximately 90% of cases were under 60 years of age, and more than 55% were younger than 30 years.[2,3] This trend is further supported by the study by Salagre et al., which reported a mean age of 28.8 years, with the majority of patients belonging to the economically productive age group.[6] This demographic trend has significant socioeconomic implications, as emphasized by Sharma et al., who demonstrated the substantial indirect costs and productivity losses associated with prolonged hospitalization and disability in GBS.[3]

The geographical clustering of cases from Mumbai and its surrounding suburban regions, along with recent travel to Pune in a subset of patients, raises the possibility of shared environmental or infectious exposures. This observation supports the hypothesis that the Pune outbreak may have represented a regional phenomenon rather than a strictly localized event, with Mumbai experiencing a parallel, though less overt, increase in cases.

Antecedent events were identified in 11 of 20 patients (55%), with gastrointestinal illness being the most frequent trigger (7/11). Microbiological evidence from the recent Pune GBS outbreak, where stool analysis of 70 patients demonstrated Campylobacter jejuni in 39% and norovirus in 16%, reinforcing the role of enteric infections as key precipitants of GBS. C. jejuni is a well-established trigger for GBS, and similar outbreaks worldwide have been linked to contaminated water supplies, lending biological plausibility to the observed association.[2,3]

The predominance of axonal variants in our cohort further strengthens this association, as axonal GBS—particularly AMAN and AMSAN—is commonly linked to Campylobacter jejuni–related molecular mimicry and has been shown to be more prevalent in Asian populations and outbreak settings. Vaccination-associated GBS was observed in only one patient in the present cohort, supporting existing evidence that while vaccination is an uncommon trigger, it remains a recognised and clinically relevant precipitant of GBS and should be considered within an appropriate temporal context.[4,13,14]

Our cohort demonstrated a severe clinical phenotype, with quadriplegia in 95% (19/20) of patients, bulbar involvement in 45% (9/20), and respiratory muscle weakness in 30% (6/20). These findings are comparable to ICU-based series, including the study by Salagre et al., which reported quadriparesis, bulbar involvement, and respiratory weakness in 98%, 56.86%, and 47.05% of patients, respectively, all of which were strongly associated with ventilatory requirement and prolonged ICU stay.[6] Autonomic dysfunction was observed in 35% of patients in the present study and contributed to two deaths, whereas none was reported by Salagre et al.[6] Autonomic dysfunction could be a key marker of disease severity and predictor of mortality in this specific cluster of GBS.

The predominance of axonal variants in our cohort (10 of 18 patients undergoing electrophysiological testing) is particularly notable, as axonal GBS is associated with rapid progression, severe weakness, and delayed or incomplete recovery. Although exact electrophysiological data from the Pune outbreak are not fully reported, available clinical and epidemiological evidence suggests a similar pattern of severe, rapidly progressive disease, implying a shared pathogenic mechanism rather than coincidental temporal overlap.[2 3] Furthermore, the presence of atypical presentations, including descending weakness and the pharyngeal-cervical-brachial variant, as observed in our study, highlights the clinical heterogeneity observed during surge periods and reinforces the need for high diagnostic vigilance. 10% patients (2/20) had past history of GBS, this suggests that during "epidemic outbreaks" of GBS, patients with a past history of GBS are at a higher risk of relapse.

Despite early initiation of IVIG in all patients, 30% (6/20) of patients required invasive mechanical ventilation, with a mean ventilatory days of 11.8 and ICU stay of 10.05 days. The study by Salagre et al., reported a higher ventilator dependence (47%) with 26.3 mean ventilatory days and prolonged ICU stays (23.5 days) among severe GBS patients.[6]

The in-hospital mortality rate of 25% (5/20) in our study is higher than that reported in most non-outbreak Indian series (Salagre et al. 5.8%) and exceeds mortality figures from the Pune outbreak (4.8%).[2,6] This excess mortality likely reflects a combination of factors, including referral bias toward more severe cases, delayed presentation, autonomic instability (2/5 deaths), and infectious complications such as sepsis (3/5 deaths)—factors also highlighted as major contributors to mortality in ICU-based cohorts.[6]

Functional outcomes at discharge were generally poor, with only 4 of 20 (20%) patients achieving full recovery, compared to 9 of 51(17.6%) patients in the Salagre et al. cohort.[6] At 28-day follow-up in our study, 30% of patients had attained complete recovery, while a substantial proportion continued to experience residual limb weakness, necessitating ongoing physiotherapy and rehabilitation. This

highlights that survival alone underestimates the true burden of GBS, particularly among patients with axonal variants or those experiencing critical illness.[3,6,12]

The concurrence of severe GBS cases in Mumbai with the Pune outbreak highlights the need to view GBS outbreaks through a regional lens rather than a district-specific one. Urban centres such as Mumbai, with high population density and extensive intercity travel, may be particularly vulnerable to spillover effects during outbreak periods. Epidemiological surveillance from the Pune outbreak reported a hitherto unrecognised association of norovirus with gastrointestinal illness, identified in 16% of stool samples from 70 GBS patients, whereas norovirus screening was not available in our cohort.[3] Strengthening regional surveillance systems, improving early recognition of infectious triggers, and ensuring adequate ICU and rehabilitation capacity are essential to mitigate morbidity and mortality during future GBS surges.

Table 1 - Summary Table of Patient Characteristics (n = 20)

Section	Category / Description	Count (n) / Percentage (%)
(A) GENDER	Male	13 (65%)
	Female	7 (35%)
(B) AGE	Mean Age (±SD)	38.5 years
(C) Age Group*	Children (12-14 years)	2 (10%)
	Youth (15-24 years)	5 (25%)
	Adults (25-44 years)	6 (30%)
	Middle aged (45-59 years)	2 (10%)
	Senior (60 +)	5 (25%)
(D) Residence	Mumbai and suburbs	13 (65%)
	Outside Mumbai	6 (30%)
	Outside Maharashtra	1 (5%)
(E) Travel History (3/20)	Pune	2/3
	Others	1/3

*WHO Classification of Age

Table 2 - Clinical Characteristics and Outcome

(A) Precipitating Factor (2-4 weeks prior) (n=11/20) (55%)	Gastrointestinal	7
	URTI	2
	Undifferentiated Febrile illness	1
	Vaccination	1 (Hepatitis B and Influenza)
(B) Presentation	Ascending	15
	Descending	4
	OTHER • PCB	1
(C) Weakness distribution	Quadriparesis Paraparesis	19 1
(D) Days from onset to Nadir	Minimum Maximum Mean	1 10 5.8
(D) Complication	Bulbar weakness	9 (45%)
	Respiratory weakness	6 (30%)
	Autonomic symptoms	7(35%)
(E) Mechanical Ventilation (MV) n=6/20(30%)	Mean Ventilator Days	11.8
(F) Hospital Stay	Minimum Maximum mean	07 60 19.4
(G) ICU Stay (19/20)	Minimum Maximum Mean ICU stay	2 30 10.05
(H) Albumino-cytological dissociation in CSF	Present	9
	Absent	10
	Not Done	1
(I) Nerve conduction study	Demyelinating	5
	AMAN	6
	AMSAN	4
	NORMAL	3
	NOT DONE	2
(J) Outcome at Discharge	Full recovery (0–2 HDS*)	4 (20%)
	Partial recovery (3–4 HDS)	5 (25%)
	Severe (Bedridden)	6 (30%)
	Death	5 (25%)
(K) Outcome at 28 days after discharge	Full recovery (0-2 HDS)	6(30%)
	Partial recovery (3-4 HDS)	5(25%)
	Severe (Bedridden)	4(20%)

*HDS- Hughes Disability Score
*PCB – Pharyngeal-Cervical-Brachial

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