

Clinical Profile and Treatment Outcome of Guillain-Barre Syndrome: A Three Year Tertiary Care Experience from Kerala

Krishnan Balagopal¹, Ria Elsa George², Riya Ann Koshy³

^{1,2,3} Department of Neurology, MOSC Medical College, Kolenchery, Kochi, India

Date of Submission: 9th September 2025

Date of Acceptance: 20th October 2025

Date of Publication: 15th December 2025

Abstract

Introduction: Guillain Barre Syndrome or GBS is an acute immune mediated polyradiculoneuropathy which continues to be a serious problem worldwide, causing respiratory insufficiency requiring mechanical ventilation in up to 30 percent of patients. This study aims to look at the clinico epidemiological profile and treatment outcome in GBS patients admitted in a tertiary level hospital in India.

Materials and Methods: We conducted a retrospective study of adult patients with Guillain barre Syndrome admitted at MOSC Hospital Kolenchery, Kerala, a tertiary care centre in south India, from January 2021 to January 2024. The case records of the study population were retrieved from medical records department and the clinical profile and outcomes were studied.

Results: A total of 28 patients were recruited on the study of which 17 (61%) were male and 11 female. The mean age of the study population was found to be 50 years. A preceding history of infection was found in 68% of patients and included respiratory infections and gastroenteritis. Limb weakness was the most common symptom noted followed by sensory symptoms including pain and paraesthesiae in the extremities. Intravenous immunoglobulin was the most common treatment modality given in 65% of patients. The most common clinical variant was Acute Motor Axonal Neuropathy (AMAN) seen in 54 % of patients followed by the demyelinating and bulbar onset forms. Ventilatory support was needed in 18% of patients and no cases of mortality were reported in the study population. Good treatment outcomes as calculated by the Hughes disability grading was achieved in 71 % of patients. Significant association was found between male sex and good outcomes.

Conclusions: Guillain Barre syndrome affects patients of all ages with a male predominance and a preceding infection seen in the majority of patients. Axonal form of GBS is the most common variety seen and a good outcome is noted in the majority of patients.

Keywords: Guillain Barre Syndrome, Clinical profile, Ventilatory Failure, Acute Motor Axonal Neuropathy

Introduction

Guillain-Barre syndrome (GBS) is the leading cause of acute neuromuscular weakness in the countries of the developed world. Guillain-Barré syndrome (GBS) also known as Acute Inflammatory Demyelinating Polyneuropathy (AIDP) is characterized by progressive symmetric muscle weakness with reduced or absent deep tendon reflexes. GBS occurs world over with an incidence of 1 to 2 cases per 1,00,000 population per

year¹. The mortality rate of GBS ranges between 5-20 percent². In spite of early diagnosis and treatment, GBS continues to be a severe disease. One-quarter of patients will require mechanical ventilation for respiratory failure or airway protection and 3-11% will die of GBS related complications³. It presents as progressive, flaccid, symmetrical muscle weakness with reduced or absent reflexes. It is having a variable presentation from mild disease to involvement of all four limbs with respiratory muscles paralysis, cranial nerves and even autonomic nervous system affection. The progressive ascending weakness reaches a maximum between 7 - 28 days. Diagnosis of GBS is mostly clinical. CSF examination shows albumin cytochemical dissociation at the end of first week. NCV studies if available are of use. Most nerve conduction studies in the classical form show evidence of peripheral nerve demyelination including prolonged distal latencies and reduced conduction velocities along with presence of conduction blocks⁴. A certain subset of patients with axonal GBS show reduced motor amplitudes early in the course of the illness⁵.

Other variants of GBS include the pure sensory variant, ataxic variant and the pharyngo cervico brachial variant⁶. Prior infections such as loose stools and fever with cough is a well-established antecedent event in the development of GBS by 2-3 weeks⁷. Approximately two-thirds of all GBS cases are preceded by an infections such as upper respiratory infection

Access this article online

Website: <https://www.nepjol.info/index.php/NJN>

DOI: <https://doi.org/10.3126/njn.v22i24.75201>



HOW TO CITE

Balagopal, K., George, R. E., & Koshy, R. A. Clinical Profile and Treatment Outcome of Guillain-Barre Syndrome: A Three Year Tertiary Care Experience from Kerala. NJNS. 2025;22(4)47-52

Address for correspondence:

Krishnan Balagopal
Consultant Neurologist, Department of Neurology MOSC Medical College,
Kolenchery, Kochi, India
Email: krishnan.balagopal@gmail.com

Copyright © 2023 Nepalese Society of Neurosurgeons (NESON)

ISSN: 1813-1948 (Print), 1813-1956 (Online)



This work is licensed under a Creative Commons Attribution-Non Commercial 4.0 International License.

, viral fevers or diarrhea⁸. Antecedent infection causes immune response against the host neural antigens through the mechanism of molecular mimicry⁹. Seasonal clustering of cases may be seen due to infection patterns in the affected areas¹⁰.

Therapeutic plasma exchange (TPE) and intravenous immunoglobulin (IVIg) are effective immunotherapies for adult and paediatric patients with GBS if given during the first few weeks of the disease. Also, appropriate supportive care is essential to minimize the risk of mortality and clinical risk and eventually improving the outcomes. Supportive care including ventilatory management is of great importance in reducing the morbidity and mortality of this condition¹¹. Physiotherapy and occupational therapy is considered as an integral part of the supportive management in reducing the incidence of complications such as respiratory failure, deep vein thrombosis (DVT), pain management and ambulation delay¹².

There is limited data in literature regarding clinical profile and outcome of the patients with GBS especially from developing countries. Identifying patients with poor prognostic factors can help in the appropriate distribution of resources in health care systems. To fulfil this unmet need in the management of patients with GBS in developing countries with limited resources, we have conducted a retrospective study among patients with GBS admitted to the Department of Neurology at MOSC Medical College, Kolenchery, Kerala to determine the clinico epidemiological profile, mortality and outcome of patients with GBS.

Materials and Methods

The aim of the study was to look at the clinical profile, mortality and treatment outcomes of patients admitted with Guillain Barre Syndrome in a tertiary level hospital

Objectives

1. To study the clinical profile of patients with GBS
2. To study the mortality of patients with GBS
3. To study the treatment outcomes of patients with GBS

Study Design

Retrospective chart analysis of patients with GBS admitted in the Neurology department over an 36 month period from Jan 2021 to Jan 2024

The study period taken was three years from the start of study. The setting for the study was patients admitted in the Department of Neurology MOSC Medical College, Kolenchery during the study period.

Study procedure

•Patients who were admitted in the Neurology Department, MOSC medical college, Kolenchery with Guillain Barre Syndrome over a period of 3 years were recruited according to inclusion and exclusion criteria.

•A semi structured proforma meeting the objective of the study was prepared for the collection of data.

Inclusion Criteria

1. All patients with Guillain Barre Syndrome diagnosed by Asbury's criteria admitted to the hospital during the study period

Exclusion Criteria

1. Early and prominent bladder and bowel dysfunction
2. Marked and persistent asymmetry of symptoms and signs

3. Presence of persistent sharp sensory level
4. Features of other diseases like myasthenia gravis, botulism, porphyria and diphtheria

Data Collection

All data from patients was recorded in a proforma. This included data like:

- Preceding history of infection
 - Duration of symptoms
 - Clinical features
 - Neurological examination
 - Laboratory values
 - Nerve conduction studies with attention to distal latencies, amplitudes and conduction velocities
 - CSF analysis if carried out looking for albumino cytological dissociation
 - Treatment given including IvIG and plasmapheresis
 - Overall mortality rate
 - Outcomes after treatment assessed at time of discharge
- Patients disability at discharge was evaluated using Hughes functional Grading scale. Muscle power was expressed using MRC grading.

Statistical Analysis

Data from the proforma were filled into MS Excel 2010 and analyzed by SPSS 25 version. For descriptive analysis frequency, percentage, mean and standard deviation were used & presented in tabular form. The significance of association of certain factors like the treatment adopted and poor prognosticators with the outcome variables like death, ventilator need, tracheostomy and bedridden state was measured by stepwise logistic regression analysis. Statistical significance was considered when the p-value was < 0.05.

Institutional Review Board (IRB) approval taken on 03/04/2024
-approval number
MOSC/IEC/136/2024

Outcome Measure

Functional outcome of the patients was assessed by Hughes motor scale at the time of discharge¹³. Hughes motor scale ranges from 0 to 6 where 0 is asymptomatic, 1 is having mild signs or symptoms but able to run, 2 is able to walk unaided for 5 meters, 3 is able to walk 5 meters with support, 4 is bedridden or chair bound, 5 is requiring ventilator assistance & 6 death of patient.

Results

Demography:

A total of 28 patients were recruited in the study of which 17 (61%) were male and 11 (39%) were female (Table 1)

Table 1: Descriptive Statistics of Study Participants (N = 28)

Variables (N = 28)	N (%)	
Demographic Information		
Age (Mean ± SD)	50.43 ± 18.281	
Gender	Male	17 (60.7%)
	Female	11 (39.3%)
Hospitalization Details		
Time from onset to arrival in hospital	< 1 week	20 (71.4%)
	≥ 1 weeks	8 (28.6%)
Length of Hospital Stay (week)	≤ 1 week	6 (21.4%)
	1 – 2 weeks	14 (50%)
	>2 weeks	8 (28.6%)
ICU Stay (week)	No ICU stay	9 (32.1%)
	≤ 1 week	10 (35.7%)
	1 – 2 weeks	7 (25%)
	>2 weeks	2 (7.1%)
Clinical Information		
Presenting Complaints	Weakness of Limb	25 (89%)
	Pain and Numbness	17 (60%)
	Bulbar Symptoms	5 (17.9%)
	Difficulty in Swallowing	4 (14.3%)
	Others	17 (60.7%)
Previous Infections		
Previous Infections	Yes	19(68%)
	Fever	10 (50%)
	Diarrhea	4 (14.3%)
	COVID 19	2 (7.1%)
	Respiratory tract infection	2 (7.1%)
	Others	3 (10.7%)
Subtypes of GBS	Others(bulbar/miller fisher/sensory)	7(14.3%)
	Axonal type	15 (64.3%)
	Demyelinating	6 (21.4%)
Treatment Information		
Treatment	IVIg/plasmapheresis	20 (71.4%)
	Inj. Methylprednisolone	9 (32.1%)
Mechanical ventilation		5 (17.9%)
Outcomes		
HUGHES Grade at Discharge	1	9 (32.1%)
	2	11 (39.3%)
	3	7 (25%)
	4	1 (3.6%)
Outcome	Good	20 (71.4%)
	Poor	8 (28.6%)
Mortality		NIL

The mean age of the study population was found to be 50.43 years. The ages ranged from a maximum of 82 years to a minimum of 16 years. The maximum number of cases -15- were in the age group of 30 to 60 years (54%). There were 8 cases more than 60 years (29%) and 5 cases less than 30 years (17%). Seasonal clustering of cases was looked for in the study. The maximum number of cases seen were seen in the months of July, August and September. Out of the total patients, 14 of them (50%) presented in the above three months. These are the monsoon season months and the increased incidence of viral and other infections could be a possible reason for this clustering seen.

Previous Infection:

A history of previous infection was obtained in about 19 out of the 28 patients (68%). Fever without any focus was the most common infection noted. This was seen in about 10 out of the 19 patients. Acute gastroenteritis was seen in 4 patients. Upper respiratory tract infection seen in 2 patients. Preceding COVID 19 infection was seen in 2 of the study population. Acute viral Hepatitis A was seen in one patient. The mean duration of antecedent infections to onset of neurological symptoms was noted to be about 10 days with a range from 2 days to 30 days.

Co morbidities:

Hypertension was the commonest co morbid condition seen in 11 patients (39%). Diabetes Mellitus was seen in 10 patients (36%). Hypothyroidism was seen in 2 patients. Smoking was seen only in one patient while there was no history of alcohol consumption.

Duration:

The mean duration of symptoms from disease onset to admission in hospital was 5 days ranging from a maximum of 28 days to a minimum of 1 day. The mean duration of hospital stay in our study was found to be 16 days ranging from a maximum of 75 days to a minimum of 5 days. ICU admission and stay was required in 19 patients (68%). The mean duration of ICU stay was found to be 12 days ranging from a maximum of 60 days to a minimum of 4 days. Mechanical ventilation was required in 5 patients (18%). Tracheostomy was done in 2 out of the 5 patients.

Clinical Presentation:

Weakness of all the 4 limbs was the most common presenting symptom seen in 25 out of 28 patients (89%) (Figure 1)

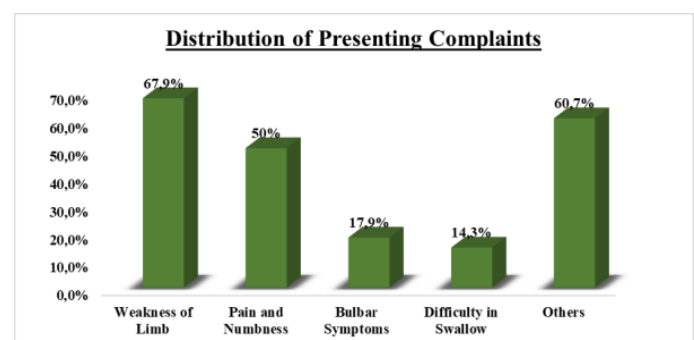


Figure 1: Graphical representation of presenting complaints distribution

The weakness started in lower limbs in most of the cases and ascended upwards to involve the upper limbs and neck. Paraesthesiae and pain of the extremities was the second commonest symptom seen in 17 patients(60%) Bulbar palsy as the presenting symptom was seen in 4 patients. Other symptoms seen included double vision and gait unsteadiness. Examination findings included symmetrical quadriparesis seen in 25 patients(89%) Absent deep tendon reflexes were seen in all of the above Bifacial palsy was seen only in 5 patients(18%) Ptosis and restriction of extraocular movements was seen in 2 patients Respiratory failure requiring ventilation was seen in 5 patients Autonomic involvement was seen only in 2 patients.

Type Of GBS:

Based on the clinical picture and the nerve conduction studies obtained, the patients were classified into the following variants. Classical GBS or the demyelinating form was seen in only 6 patients(21%) Axonal form of GBS including both AMAN and AMSAN were seen in 15 patients(64%) Other forms include 3 bulbar onset GBS and 3 forms presenting as Miller Fisher syndrome (MFS) of which 2 tested positive for anti GQ1B antibody One form was a pure sensory variant of GBS 2 of the patients had a prior history of GBS- one 12 years back and another 5 years back. Ganglioside antibody testing was done in both, which was negative.

CSF Study:

CSF analysis was done in only 10 patients(35%) Albumino cytological dissociation defined as cells less than 5 and protein level greater than 45 was seen in 8 of the 10 patients. All CSF cultures and serology were normal

Treatment:

18 of the patients received Intravenous Immunoglobulin injections(IVIG) at the dose of 2 grams per kg of body weight 2 patients received therapeutic plasmapheresis 8 patients who were not affordable for IVIG or plasmapheresis received pulse dose of methylprednisolone

Outcomes:

All the patients survived and there was no case of mortality in our study population Good outcome was defined as Hughes Grade 1 or 2 at discharge or the ability to walk a short distance without support while Grade 3 and above was calculated as a poor outcome 20 patients (71%) were able to achieve a good outcome in our study. Out of these, 9 patients were able to walk long distances without support(Hughes grade 1) while 11 patients were able to walk more than 10 metres without support(Grade 2) (Figure 2)

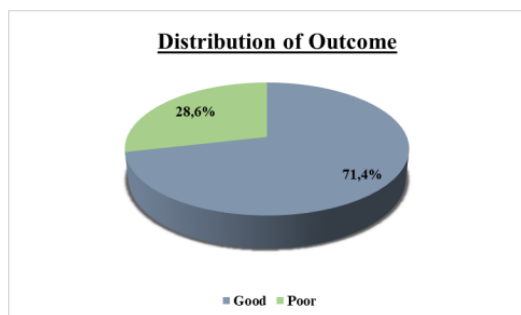


Figure 2 : Graphical representation of Outcome

Out of the 8 patients with poor outcomes, 7 of them required support while walking(Hughes Grade 3) and one person was wheelchair bound(Grade 4) Out of the poor outcomes, 3 had axonal variant, 2 had the demyelinating variant, 2 had the bulbar onset variant and 1 had Miller Fisher phenotype None of the patients died in the study

Correlations:

Table 2 presents the logistic regression analysis of factors associated with outcomes. The study included 28 participants, with 20 having a good outcome and 8 a poor outcome.

Age was not significantly associated with outcomes (OR: 0.983, 95% CI: 0.939-1.028, p=0.454). (Table 2)

Table 2: Logistic Regression Analysis of Factors Associated with Outcomes

Variables	Outcome		OR (95% CI)	p-value
	Good (N=20)	Poor (N=8)		
Age			0.983 (0.939 – 1.028)	0.454
Gender	Male	15 (75%)	0.111 (0.017 – 0.738)	0.023*
	Female	5 (25%)		
Time from onset to arrival in hospital	< 1 week	14 (70%)	0.778 (0.121 – 5.018)	0.792
	≥ 1 weeks	6 (30%)		
Length of Hospital Stay (week)	< 1 week	5 (25%)	Ref	
	1 – 2 weeks	11 (55%)	1.364 (0.112 – 16.577)	0.808
	> 2 weeks	4 (20%)	5 (0.388 – 64.387)	0.217

*p-value < 0.05 shows statistical significance

Gender showed a significant association, with males having a better outcome (OR: 0.111, 95% CI: 0.017-0.738, p=0.023).

Time from onset to hospital arrival within a week versus a week or more showed no significant impact on outcomes (OR: 0.778, 95% CI: 0.121-5.018, p=0.792).

Length of hospital stay was not significantly associated with outcomes, though patients staying more than two weeks showed a higher, but not statistically significant, odds of poor outcome (OR: 5, 95% CI: 0.388-64.387, p=0.217).

There was no association noted between subtype of GBS and final outcome in the analysis

Discussion

Guillain Barre Syndrome is one of the most common causes of acute flaccid paralysis worldwide. We conducted this study to look at the clinical profile and outcome of GBS in our local population in comparison to other studies. The incidence of GBS was commoner in males than in females in our study. This was in keeping with Sundar et al¹³, Sharma et al¹⁴ and Dhadke et al¹⁵ where male predominance was found. An Italian study by Ropper et al¹⁶ also showed similar findings. The mean age of the study population was found to be high at 50 years which could be due to most of the patients being adults in the study. This was higher than previous studies done from India¹⁷, Nepal¹⁸ and Bangladesh¹⁹ This was similar to other studies done from Australia²⁰ with a mean age of 52 years and from Iran²¹ with a mean age of 48 years.

The maximum number of patients were in the age group of 30 to 60 years as in various studies done including Stany et al²² and Dhadke et al. A prior history of infection was noted in most cases. The most common were fever without any source, followed by gastroenteritis and respiratory tract infections.

A similar study by Thota et al²³ showed fever to be the most common preceding event followed by respiratory and gastrointestinal infections. Similar findings were noted in studies by Webb et al²⁴ and Koga et al²⁵.

We had 2 cases of preceding COVID 19 infection. There are various case reports of COVID related GBS including one by Pimentel et al²⁶ which found that the demyelinating form of GBS was seen more commonly following COVID infection and a case series by Aladawi et al²⁷.

The mean duration of interval from infection to onset of symptoms was found to be 10 days in our study. This was similar to various studies done in India including Dave et al which showed infections to be occurring within the last two weeks of onset of symptoms. Seasonal clustering of cases was seen in our study in the monsoon months of July to September. A similar Indian study by Prasad et al showed maximum cases in the rainy season(41%) with a second peak in summer. The most common clinical findings seen were weakness of both lower and upper limbs followed by sensory symptoms and bulbar and facial involvement. This was similar to a study done by Sundar et al²⁸ of 50 patients who found that the most common presentation was motor weakness followed by tingling and numbness and respiratory involvement. In their study, the most common cranial nerve to be involved was the facial nerve. In another study by Kaur et al²⁹, facial nerve involvement was very common, seen in almost 41% of patients. The incidence of respiratory failure and mechanical ventilation was 18 % in our study. This was similar to studies done by Bhagat et al (16.1%) and a Chinese study done by Zhang et al³⁰(13.9%) A large case series from India done by Khadiolkar et al³¹ found that one third of hospitalized GBS patients require mechanical ventilation due to respiratory muscle or oropharyngeal weakness. Bulbar palsy and reduction in vital capacity appears to be independently associated with the same. Most patients in our study received IViG as treatment modality. This was similar to other studies done by Sundar et al and Habib et al The most common variant of GBS seen in our study was the axonal forms seen in about 54 % of the patients. This was similar to other Indian studies done by Kanjalkar et al and Kumbhar et al where the incidence of AMAN and AMSAN was found to be more than 60 % of all patients studied. CSF albumino cytological dissociation was observed in about 36 % which was lower than other studies where it was seen in more than 50 percent of cases. Most of these were in cases done in the second week of illness. Our study shows a lower incidence because CSF was done early in most cases. The overall mortality rate was zero which was better than most other Indian studies.

This could be because of early diagnosis and institution of treatment in most of our cases leading to lesser mortality. Positive outcome was seen in more than 70 % of cases in our study which was also seen in various other studies including ones by Neto et al³² and Rees et al³³ where more than 80 percent of patients improved. A significant co relation was found between male gender and overall good outcomes. There was no significant association between age, duration of illness, length of hospital stay and final outcomes.

LIMITATIONS OF STUDY:

The main limitation of the study was its small sample size due which further studies are needed to look at its conclusions Long term follow up of the patients was not looked at to assess the outcomes.

Conclusion

Guillain Barre syndrome is a very important cause of acute flaccid paralysis in developing countries. It affects people of all ages and a history of preceding infection is seen in most of the cases. Early diagnosis by clinical examination and nerve conduction studies is of great importance. Effective treatment early in the disease course leads to good outcomes in the majority of the patients.

References

1. Mateen FJ, Cornblath DR, Jafari H, Shinohara RT, Khandit D, Ahuja B, Bahl S, Sutter RW. Guillain-Barré Syndrome in India: population-based validation of the Brighton criteria. *Vaccine*. 2011 Dec 6;29(52):9697-701. doi:10.1016/j.vaccine.2011.09.123. Epub 2011 Oct 11. PMID: 22001121; PMCID: PMC3638251
2. McGrogan A, Madle GC, Seaman HE, de Vries CS. The epidemiology of Guillain-Barré syndrome worldwide. A systematic literature review. *Neuroepidemiology*. 2009;32(2):150-63. doi: 10.1159/000184748. Epub 2008 Dec 17. PMID: 19088488.
3. van den Berg B, Walgaard C, Drenthen J, Fokke C, Jacobs BC, van Doorn PA. Guillain-Barré syndrome: pathogenesis, diagnosis, treatment and prognosis. *Nat Rev Neurol*. 2014 Aug;10(8):469-82. doi: 10.1038/nrneurol.2014.121. Epub 2014 Jul 15. PMID: 25023340
4. Verma R, Chaudhari TS, Raut TP, Garg RK. Clinico-electrophysiological profile and predictors of functional outcome in Guillain-Barre syndrome (GBS). *J Neurol Sci*. 2013 Dec 15;335(1-2):105-11. doi: 10.1016/j.jns.2013.09.002. Epub 2013 Sep 10. PMID: 24064258.
5. Verma R, Chaudhari TS, Raut TP, Garg RK. Clinico-electrophysiological profile and predictors of functional outcome in Guillain-Barre syndrome (GBS). *J Neurol Sci*. 2013 Dec 15;335(1-2):105-11. doi: 10.1016/j.jns.2013.09.002. Epub 2013 Sep 10. PMID: 24064258
6. Seneviratne U. Guillain-Barré syndrome. *Postgrad Med J*. 2000 Dec;76(902):774-82. doi: 10.1136/pmj.76.902.774. PMID: 11085768; PMCID: PMC1741839
7. Färkkilä M, Kinnunen E, Haapanen E, Iivanainen M. Guillain-Barré syndrome: quantitative measurement of plasma exchange therapy. *Neurology*. 1987 May;37(5):837-40. doi: 10.1212/wnl.37.5.837. PMID: 3553986
8. Rees J. Guillain-Barré syndrome. Clinical manifestations and directions for treatment. *Drugs*. 1995 Jun;49(6):912-20. doi: 10.2165/00003495-199549060-00005. PMID: 7641605
9. Hu W, Dehmel T, Pirhonen J, Hartung HP, Kieseier BC. Interleukin 23 in acute inflammatory demyelination of the peripheral nerve. *Arch Neurol*. 2006 Jun;63(6):858-64. doi: 10.1001/archneur.63.6.858. PMID: 16769867
10. Kalita J, Misra UK, Das M. Neurophysiological criteria in the diagnosis of different clinical types of Guillain-Barre syndrome. *J Neurol Neurosurg Psychiatry*. 2008 Mar;79(3):289-93. doi: 10.1136/jnnp.2007.118000. Epub 2007 Jul 5. PMID: 17615164
11. Raphaël JC, Chevret S, Hughes RA, Annane D. Plas-

- ma exchange for Guillain-Barré syndrome. *Cochrane Database Syst Rev.* 2012 Jul 11;(7):CD001798. doi: 10.1002/14651858.CD001798.pub2. Update in: *Cochrane Database Syst Rev.* 2017 Feb 27;2:CD001798. doi: 10.1002/14651858.CD001798.pub3. PMID: 22786475
12. Hughes RA, Wijidicks EF, Benson E, Cornblath DR, Hahn AF, Meythaler JM, Sladky JT, Barohn RJ, Stevens JC; Multidisciplinary Consensus Group. Supportive care for patients with Guillain-Barré syndrome. *Arch Neurol.* 2005 Aug;62(8):1194-8. doi: 10.1001/archneur.62.8.1194. PMID: 16087757
 13. Sundar K, Vasanthan K, Vengadkrishnan K, Satyamurthy P, Sudhakar MK. Clinical Profile of Guillain-Barre Syndrome in a Tertiary Care Center. *Int J Sci Stud* 2016;4(9):27-3. doi: <https://dx.doi.org/10.18535/jmscr/v5i5.192>
 14. Sharma G, Sood S, Sharma S. Seasonal, Age & Gender Variation of Guillain Barre Syndrome in a Tertiary Referral Center in India. *Neuroscience & Medicine* 2013; 4:23-28. doi: <http://dx.doi.org/10.4236/nm.2013.41004>
 15. Dhadke SV, Dhadke VN, Bangar SS, Korade MB. Clinical profile of Guillain Barre syndrome. *J Assoc Physicians India.* 2013 Mar;61(3):168-72. PMID: 24475678.
 16. Clinical E, Neurology EP. Guillain-Barré syndrome variants in Emilia-Romagna, Italy, 1992-3: incidence, clinical features, and prognosis. Emilia-Romagna Study Group on Clinical and Epidemiological Problems in Neurology. *J Neurol Neurosurg Psychiatry.* 1998 Aug;65(2):218-24. doi: 10.1136/jnnp.65.2.218. PMID: 9703176; PMCID: PMC2170214
 17. Paul BS, Bhatia R, Prasad K, Padma MV, Tripathi M, Singh MB. Clinical predictors of mechanical ventilation in Guillain-Barré syndrome. *Neurol India.* 2012 Mar-Apr;60(2):150-3. doi: 10.4103/0028-3886.96383. PMID: 22626694
 18. Bhagat SK, Sidhant S, Bhatta M, Ghimire A, Shah B. Clinical Profile, Functional Outcome, and Mortality of Guillain-Barre Syndrome: A Five-Year Tertiary Care Experience from Nepal. *Neurol Res Int.* 2019 Jun 2;2019:3867946. doi: 10.1155/2019/3867946. PMID: 31275647; PMCID: PMC6582782
 19. Kabir, A. H., Rahman, M., Ali, B., Kabiruzzaman, .-, & Ahmad, M. (2015). Pattern of presentation of Guillain-Barre syndrome in three tertiary level Hospital in Bangladesh. *Journal of Armed Forces Medical College, Bangladesh,* 10(1), 50–52. doi: <https://doi.org/10.3329/jafmc.v10i1.22923>
 20. Blum S, Reddel S, Spies J, McCombe P. Clinical features of patients with Guillain-Barré syndrome at seven hospitals on the East Coast of Australia. *J Peripher Nerv Syst.* 2013 Dec;18(4):316-20. doi: 10.1111/jns5.12045. PMID: 24172315
 21. Yadegari S, Kazemi N, Nafissi S. Clinical and electrophysiological features of Guillain-Barré syndrome in Iran. *J Clin Neurosci.* 2014 Sep;21(9):1554-7. doi: 10.1016/j.jocn.2013.11.041. Epub 2014 Apr 29. PMID: 24786718
 22. Anto Ignat Stany M, Susan Dsouza, Peter George. Clinical Profile and Outcomes Of Guillain-Barré Syndrome At A Tertiary Care Centre In Southern India. *International Journal of Scientific Research.* 2018;7(10); 1-3. doi: <https://www.doi.org/10.36106/ijsr>
 23. Thota B, Mukkara M, Samantaray A, Mohan A, Vengamma B. A study of clinical presentation and outcome of patients with Guillain-Barré syndrome: A prospective observational study at a tertiary care teaching hospital. *J Clin Sci Res* 2019;8:182-7. doi: https://doi.org/10.4103/JCSR.JCSR_93_19
 24. Webb AJ, Brain SA, Wood R, Rinaldi S, Turner MR. Seasonal variation in Guillain-Barré syndrome: a systematic review, meta-analysis and Oxfordshire cohort study. *J Neurol Neurosurg Psychiatry.* 2015 Nov;86(11):1196-201. doi: 10.1136/jnnp-2014-309056. Epub 2014 Dec 24. PMID: 25540247
 25. Koga M, Yuki N, Hirata K. Antecedent symptoms in Guillain-Barré syndrome: an important indicator for clinical and serological subgroups. *Acta Neurol Scand.* 2001 May;103(5):278-87. doi: 10.1034/j.1600-0404.2001.103005278.x. PMID: 11328202.
 26. Pimentel V, Luchsinger VW, Carvalho GL, Alcará AM, Esper NB, Marinowic D, Zanirati G, da Costa JC. Guillain-Barré syndrome associated with COVID-19: A systematic review. *Brain Behav Immun Health.* 2023 Mar;28:100578. doi: 10.1016/j.bbih.2022.100578. Epub 2023 Jan 17. PMID: 36686624; PMCID: PMC9842533
 27. Aladawi M, Elfil M, Abu-Esheh B, Abu Jazar D, Armouti A, Bayoumi A, Piccione E. Guillain Barre Syndrome as a Complication of COVID-19: A Systematic Review. *Can J Neurol Sci.* 2022 Jan;49(1):38-48. doi: 10.1017/cjn.2021.102. Epub 2021 May 5. PMID: 33949300; PMCID: PMC8267336
 28. Sundar K, Vasanthan K, Vengadkrishnan K, Satyamurthy P, Sudhakar MK. Clinical Profile of Guillain-Barre Syndrome in a Tertiary Care Center. *Int J Sci Stud* 2016;4(9):27-30. doi: <https://dx.doi.org/10.18535/jmscr/v5i5.192>
 29. Kaur U, Chopra JS, Prabhakar S, Radhakrishnan K, Rana S. Guillain-Barré syndrome. A clinical electrophysiological and biochemical study. *Acta Neurol Scand.* 1986 Apr;73(4):394-402. doi: 10.1111/j.1600-0404.1986.tb03295.x. PMID: 3727915
 30. Zhang B, Wu X, Shen D, Li T, Li C, Mao M, Zhang HL, Liu K. The clinical characteristics and short-term prognosis in elderly patients with Guillain-Barré syndrome. *Medicine (Baltimore).* 2017 Jan;96(1):e5848. doi: 10.1097/MD.0000000000005848. PMID: 28072747; PMCID: PMC5228707
 31. Meena AK, Khadilkar SV, Murthy JM. Treatment guidelines for Guillain-Barré Syndrome. *Ann Indian Acad Neurol.* 2011 Jul;14(Suppl 1):S73-81. doi: 10.4103/0972-2327.83087. PMID: 21847334; PMCID: PMC3152164
 32. Netto AB, Taly AB, Kulkarni GB, Uma Maheshwara Rao GS, Rao S. Prognosis of patients with Guillain-Barré syndrome requiring mechanical ventilation. *Neurol India.* 2011 Sep-Oct;59(5):707-11. doi: 10.4103/0028-3886.86545. PMID: 22019655
 33. Rees JH, Thompson RD, Smeeton NC, Hughes RA. Epidemiological study of Guillain-Barré syndrome in south east England. *J Neurol Neurosurg Psychiatry.* 1998 Jan;64(1):74-7. doi: 10.1136/jnnp.64.1.74. PMID: 9436731; PMCID: PMC2169900