A Clinical Study of Prognostic Factors of Guillain Barré Syndrome in a Tertiary Care Hospital of North-East India

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ABSTRACT

Introduction: Guillain-Barré syndrome (GBS) is an acute, frequently severe, and fulminant polyradiculoneuropathy that is autoimmune in nature. Various factors play an important role in the mortality and morbidity of the disease. Aim of the study is to look for the prognostic factors.

Methods: We conducted a hospital based observational, descriptive study comprising of 52 patients of Guillain Barré Syndrome who had been diagnosed based on the criteria laid down by Asbury AK, Cornblath DR (1990), admitted in Gauhati Medical College and Hospital, Guwahati, Assam (India) and fulfilled the inclusion and exclusion criteria. Statistical analysis was performed using GraphPad InStat version 3.00 for Windows 7, GraphPad Software, San Diego California USA.

Results: In our study, 32 were male and 20 were female; 38.46% were in the third decade of life followed by the second decade (21.15%) and sixth decade (13.46%). On follow up at the end of 1 month, 30 out of 52 patients (57.7%) had good prognosis while the rest (42.3%) had a poor prognosis (GBS disability score of >2). Amongst the patients with poor prognosis, 40.91% of the patients were more than 50 years of age (p-value 0.02), 68.18% had a low MRC sum score (<30) on presentation to the hospital (p-value 0.0002), 54.55% had an antecedent history of diarrhoea (p-value 0.0023), 68.18% showed autonomic dysfunction (p-value 0.023) during hospital stay and 72.72% had cranial nerve involvement. Mortality was 7.69% due to ventilator associated pneumonia.

Conclusion: Early identification of several clinical factors are crucial to predict the prognosis of the disease and early initiation of treatment ensures a better outcome.

KEYWORDS: Autonomic dysfunction, Guillain-Barré syndrome (GBS), GBS disability score, Polyradiculoneuropathy, Poor prognosis.

INTRODUCTION

Guillain-Barré Syndrome is an acquired immunologically mediated inflammatory polyradiculoneuropathy. It initially presents with weakness with or without paresthetic sensory symptoms. The fairly symmetrical weakness of the lower limbs ascends proximally over hours to several days and may subsequently involve arm, facial, and oropharyngeal muscles, and in severe cases, respiratory muscles. Its severity varies from mild, in which patients are still capable of walking unassisted, to a nearly total quadriplegia1. This is the most common cause of acute or subacute generalized paralysis in practice2.

It has a yearly incidence rate between 1.1 and 1.8 per 100000 in western countries. The lifetime likelihood of any individual acquiring GBS is 1:1000. GBS is equally common in men and women and can occur at any age. In western countries, GBS is common in the 5th decade, but in India it occurs more commonly at a younger age3. There is a male preponderance among the hospitalized population4.

GBS causes rapidly progressive acute flaccid diffuse proximal and distal weakness of the four limbs with or without sensory loss; the maximal weakness is reached within 4 weeks. The usual pattern is an ascending paralysis. A rapid tempo of progression with facial, bulbar and/or respiratory muscle weakness is frequent during the first week of symptoms5. Autonomic nervous system involvement is common and may occur even in

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patients whose GBS is otherwise mild. Several subtypes of GBS are recognized. Guillain-Barré syndrome (GBS) is usually associated with a good prognosis. However, it can have a poor prognosis needing ventilatory support and causing major deficits at discharge. GBS remains fatal in about 4% of cases; only about 20% of patients are able to walk unaided after 4 weeks. After 1 year only 60% recover full motor strength and 14% are left with a severe disability.

Regarding clinical prognostic factors, most studies demonstrate higher age (>40 or >50 years) as poor outcome. Also preceding diarrhoea, severe weakness leading to a low MRC score on admission and high early GBS disability grade are all reliable predictors of a poor outcome. González-Suárez et al. (2013) demonstrated cranial nerve involvement and need for mechanical ventilation also leads to a poor prognosis.

MATERIALS AND METHODS

Patients

In this descriptive, observational study, conducted from June 2014 to May 2015, a total of 52 patients were included. These patients, all above the age of 12 years, had attended Medicine O.P.D./Ward or Neurology O.P.D./Ward in Gauhati Medical College and Hospital, Assam, India; and had been diagnosed based on the criteria laid down by Asbury AK, Cornblath DR (1990). Patients with acute neuromuscular weakness due to other causes (e.g., myasthenia gravis, botulism, poliomyelitis, toxic neuropathy, diphtheria, vasculitic neuropathy) and those with major illness like CAD, DM, Hypertension, HIV and tuberculous infection were excluded.

Ethical clearance was obtained from the ethical committee of Gauhati Medical College & Hospital.

Assessment

Data was collected by taking proper history from patients and attendants, thorough clinical examination and relevant investigations. Data was recorded in preformed proforma. Clinical data mainly comprised of age, sex, date of onset of disease, date of presentation to the hospital, duration of progression of symptoms, disability at the time of presentation, preceding and concurrent infections, sensory symptoms, bladder and bowel complaints, exposure to toxins, heavy metals and recent vaccination. In clinical examination due importance was given to pulse, blood pressure, respiratory rate, single breath count and neurological examination including cranial nerves especially III, IV, VI, VII, IX, X, tone and power of the muscles, reflexes (both deep and superficial) and sensory system examination. Power of the muscles was assessed by MRC sum score (Annexure 2), a summation of the MRC grades (range 0–5) given in full numbers of the following muscle pairs: upper arm abductors, elbow flexors, wrist extensors, hip flexors, knee extensors and foot dorsal flexors as given by Kleyweg RP et al (1991). The MRC sum score ranged from 0 (“total paralysis”) to 60 (“normal strength”). Laboratory data consisted of Complete Blood Count, ESR, RFT, LFT, serum electrolytes, RBS, Urine routine examination and Urine for Porphobilinogen, HIV ELISA, Chest X-Ray, ECG and Nerve Conduction Velocity (NCV) testing.

Patients were followed up after 1 month and were assessed by the GBS Disability Score (Annexure 1) advocated by Hughes RA et al (1978). Poor prognosis was defined as the inability to walk unaided 10 meters across an open space (GBS disability score of 3 or higher) as given by Walgaard et al (2011). GBS disability score of ≤2 was taken as good prognosis.

Statistics

All the statistical graphs were prepared using Microsoft Excel 2007 and Microsoft Word 2007. Statistical analysis was performed using GraphPad InStat version 3.00 for Windows 7, GraphPad Software, San Diego California USA. (www.graphpad.com).

RESULTS

Out of the 52 cases, 30 (57.69%) were males and 22 (42.31%) were females. Male to Female ratio was 1.36:1; the age ranged from 12 to 80 years and mean age was 34.02±16.78 years. Majority (38.46%) were in the age group of 21-30 years followed by 12-20 years (21.15%).

The highest incidence of GBS (38.46%) (20 cases) was seen in the summer months from May to July. There were 16 (30.76%) cases in the spring season from February to April, 10 (19.24%) in the winter season from November to January and 6 (11.54%) in the rainy season from August to October. Majority of the patients (38.46%) achieved clinical nadir, which is the maximum disease activity after the onset of the disease, during the first week of presentation. By the end of the 2nd week, 28.84% had achieved nadir. Mean number of days for nadir was 9.71 ± 6.5 days.

Thirty three (33) out of 52 patients (63.46%) of GBS had some antecedent event while 19 (36.54%) reported no such clinical event. Gastrointestinal infection was reported by 16 (30.77%) patients. Most of the patients (93.94%) developed GBS within 28 days from the antecedent event and 66.67% had a latent period of < 2 weeks. The mean latent period was 14.08 ± 8.21 days.

In nerve conduction studies, 38 (73.08%) patients were found to have AIDP. AMAN was diagnosed in 8 (15.38%) patients while AMSAN was diagnosed in 6 (11.54%) of the cases.

Cranial nerve involvement was seen in 28 out of 52 (53.84%) of the patients while 46.16% of patients had no cranial nerve involvement. Respiratory muscle weakness was seen in 14 (26.92%) patients and 73.08% of the patients did not show respiratory muscle weakness.
Five (9.62%) patients developed aspiration pneumonia and 4 (7.69%) patients had ventilator associated pneumonia during hospital stay. Respiratory muscle weakness was seen in 26.31%, 37.5% and 16.67% of patients with AIDP, AMAN and AMSAN respectively. Sensory Symptoms in the form of pain was seen in 21 patients (40.38%) and 37 patients (71.15%) had parasthesias. Objective sensory deficit was seen in 17 patients (32.69%). Autonomic dysfunctions were seen in 25 out of 52 patients (48.07%) while 27 (51.93%) patients had no evidence of autonomic dysfunctions. Out of the 52 patients studied 4 patients expired. Mortality was 7.69%. The cause of death in all the patients was ventilator associated pneumonia. Two out of the 4 patients who died, also had autonomic dysfunction. Mortality in AIDP patients was 7.9% and in AMAN patients it was 12.5%.

Thirty out of 52 patients had good prognosis at the end of 1 month (57.7%) while the rest had a poor prognosis (42.3%). Maximum numbers of patients with poor prognosis were of age group 51-60 years (27.27%).

Statistical analysis testing reveals a p-value of 0.02 which is significant. Majority of the patients with poor prognosis (68.18%) had a low MRC sum score (<30) on presentation to the hospital. The p-value is 0.002 on statistical analysis which is significant. There was history of diarrhoea in 54.55% of the patients with poor prognosis. Statistical analysis test reveals a p-value of 0.003 which is significant. There were autonomic dysfunctions in 68.18% of the patients with poor prognosis. Statistical analysis test shows a p value of 0.023 which is significant. Sixteen out of 22(72.72%) patients with poor prognosis had cranial nerve involvement while cranial nerve was not involved in 6 (27.28%) patients with poor prognosis. Statistical test shows p-value to be 0.49 which is significant. Amongst patients with poor prognosis electrophysiological studies showing axonal neuropathy was found in 40.91% of the patients and demyelinating neuropathy was detected in 59.09% of the patients with poor prognosis. Statistical analysis test shows a p value of 0.064 which is insignificant.

**DISCUSSION**

**Gender Distribution**

In our study, we found a male to female ratio of 1.36:1. An Indian study Dhadke SV et al. 2013\textsuperscript{13}, McGrogan A et al. 2009\textsuperscript{14}, González-Suárez et al. 2013\textsuperscript{8} have also reported small predominance of male gender.

**Age Distribution**

In this study the youngest age was 13 years and the oldest age was 80 years with a mean age of 34.02±16.78 years. Maximum number of cases were in the third decade of life (38.46%) followed by the second decade (21.15%) and sixth decade (13.46%). Dhadke SV et al.
Table 1: Prognosis based on age.

<table>
<thead>
<tr>
<th>Age (yrs)</th>
<th>Prognosis</th>
<th>p-value</th>
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<tbody>
<tr>
<td>12-20</td>
<td>Good 8 (26.68%)</td>
<td>Poor 3 (13.64%)</td>
</tr>
<tr>
<td>21-30</td>
<td>Good 15 (50%)</td>
<td>Poor 5 (22.72%)</td>
</tr>
<tr>
<td>31-40</td>
<td>Good 4 (13.33%)</td>
<td>Poor 2 (9.09%)</td>
</tr>
<tr>
<td>41-50</td>
<td>Good 1 (3.33%)</td>
<td>Poor 3 (13.64%)</td>
</tr>
<tr>
<td>51-60</td>
<td>Good 1 (3.33%)</td>
<td>Poor 6 (27.27%)</td>
</tr>
<tr>
<td>&gt;60</td>
<td>Good 1 (3.33%)</td>
<td>Poor 3 (13.64%)</td>
</tr>
<tr>
<td>Total</td>
<td>Good 30</td>
<td>Poor 22</td>
</tr>
</tbody>
</table>

Table 2: Prognosis based on weakness at presentation (MRC sum score)

<table>
<thead>
<tr>
<th>MRC sum score</th>
<th>Prognosis</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>51-60</td>
<td>Good 4 (13.33%)</td>
<td>Poor 2 (9.09%)</td>
</tr>
<tr>
<td>41-50</td>
<td>Good 11 (36.67%)</td>
<td>Poor 2 (9.09%)</td>
</tr>
<tr>
<td>31-40</td>
<td>Good 12 (40%)</td>
<td>Poor 3 (13.64%)</td>
</tr>
<tr>
<td>&lt;30</td>
<td>Good 3 (10%)</td>
<td>Poor 15 (68.18%)</td>
</tr>
</tbody>
</table>

**Seasonal Variation**

In this study maximum number of patients were seen in the summer season from May to July (38.46%) followed by the spring season from February to April (30.76%). Sharma G et al. (2013)\(^5\) in India, Zaheer M et al (2008)\(^16\) in Pakistan, Akbayram M et al (2011)\(^17\) in Turkey found maximum incidence of 41.53%, 64% and 40% respectively during summer season which correlates with our study.

**Clinical Nadir**

By the end of the second week of onset of disease, 68.3% of the patients achieved clinical nadir. All the patients (100%) had maximum weakness by 4 weeks. Maximum number of days from disease to nadir was 28 days and minimum was 1 day. According to Asbury AK et al, 1990\(^18\)~50% of patients reach clinical nadir by 2 weeks and more than 90% by 4 weeks. Sejvar JJ et al. 2011\(^19\) described that the weakness in GBS progresses over a period of 12 hours to 28 days before a plateau is reached.

**Antecedent Events**

Our study revealed 63.46% to have respiratory tract infection or gastrointestinal tract infection before the development of GBS. Two thirds of cases (66.67%) of GBS are preceded by upper respiratory tract infection or diarrhoea according to Yuki N et al. 2012\(^20\) which matches our study. Respiratory Tract Infection was present in 32.69% of our cases and gastrointestinal tract infection in 30.77%. González-Suárez et al. 2013\(^8\) have reported upper respiratory infection in 37.7% of cases and gastrointestinal infection in 27.4%.

**Latent Period between the antecedent event and onset of the disease**

The mean latent period, 14.08 ± 8.21 days, in our study is consistent with the study carried out by González-Suárez et al. 2013\(^8\). However, the study carried out by Dhadke SV et al. 2013\(^21\), majority i.e. 20 out of 22 (90.90%) patients developed neurologic illness within 4 weeks of antecedent event, while remaining 2 patients developed it between 1-3 months of preceding illness.

**Subtypes of GBS**

In our study the most common subtype of GBS was AIDP which was found in 73.08% of the patients. AMAN was diagnosed in 8 (15.38%) patients while AMSAN was diagnosed in 6 (11.54%) of the cases. We did not find any case of Miller Fisher Syndrome or any other variant of GBS. Kalita et al (2014)\(^22\) in Lucknow have reported AIDP in 73.4%, AMAN in 13.4% and AMSAN in 4.6% which is similar to our study. Gupta et al (2008)\(^23\) in Thrivananthapuram, Alexander et al (2001)\(^24\) in Vellore and Vengamma (2011)\(^25\) in Tirupati have reported the most common subtype of GBS as AIDP in 85.2%, 38.2% and 76.2% respectively followed by AMAN in 10.6%, 30.4% and 3.4% patients respectively.

**Cranial Nerve Involvement**

Cranial nerves were involved in 53.84% of the patients. According to Löffel et al. 1977\(^27\) and Winer et al. 1988\(^28\) cranial nerves are involved in 50% of the patients. González-Suárez et al. 2013\(^8\) have shown cranial nerve involvement in 35.5% of the patients. Most common nerve involvement was the VII cranial nerve bilaterally in our study (38.46%) followed by the IX and X cranial nerves (26.92%).

**Respiratory System Involvement**

This study showed respiratory muscle paralysis in 14 patients (26.92%). Ten (19.2%) patients needed ventilatory support. Four (7.69%) patients had aspiration pneumonia and 4 (7.69%) had ventilator associated pneumonia. Chio A et al. 2003\(^29\) and Ropper AH 1992\(^30\) have reported respiratory muscle paralysis in
20-30% of patients which is consistent with our study. González-Suárez et al. 2013³ have shown respiratory paralysis in 17% of cases. In Indian study by Dhadke SV et al. 2013¹ respiratory failure was observed in 32.5% patients.

**Autonomic Dysfunctions**

We found autonomic disturbances in 25 (48.07%) patients. Autonomic dysfunction is common in the Guillain–Barré syndrome, occurring in over 60 % cases according to Zochodne 1994⁴². Flachenecker P et al 1997³³ and Burns TM et al 2001³⁴ have all reported autonomic disturbances in over 50% of patients. Chatterjee A et al 2009³⁵ found autonomic signs in 35.3% of patients in their study. However, González-Suárez et al 2013³ have found autonomic abnormalities in 8.5% in their study which is less than that in our study.

**Mortality**

Four (7.69%) patients died in our study, all due to ventilator associated pneumonia. Winer JB et al. 1988³⁶ has reported mortality in the range of 3-13%. Hughes RAC et al. 2007³⁷, Rajabally YA et al. 2012³ have found mortality in GBS to be 5% and 4% respectively. In northern India, Kalita J et al (2014)³² have reported death in 3.4 % of the patients studied. Lawn and Wijdicks (1999)³⁸ did a comprehensive audit of in-hospital GBS-related deaths and found that deaths most commonly resulted from ventilator-associated pneumonia as found in our study.

**Prognosis of the patients**

On follow up at the end of 1 month in the study 30 out of 52 patients had good prognosis (57.7%) while the rest had a poor prognosis (42.3%). In the study done by Walgaard et al (2011)³⁹ 54% had poor outcome at 4 weeks, 29% at 3 months, and 15% at 6 months after hospital admission.

**Prognosis based on Age at one month**

In our study after 1 month 40.91% of the patients with poor prognosis were more than 50 years of age. Statistical analysis showed significant p-value of 0.02. The Plasma Exchange Sandoglobulin Trial⁴⁰ has shown a significant correlation between ages more than 50 years and death or inability to walk at 48 weeks after the disease. Visser et al (1999)⁴¹ using data of 147 patients who had participated in the Dutch GBS trial found by multivariate logistic regression analysis that age >50 was a predictor of a poor outcome. Walgaard et al (2011)⁴² has advocated age more than 60 years as a predictor of poor outcome of GBS.

**Prognosis based on weakness**

In this study, 15 (68.18%) out of 22 patients with poor prognosis had a low MRC sum score (<30) on presentation to the hospital. Statistical analysis showed p-value of 0.0002 which is statistically significant. Visser et al (1999)⁴³ have shown low MRC score (<40) to be a predictor of a poor outcome. Walgaard et al (2011)⁴² have reported low MRC score at admission to the hospital and at day 7 to be a predictor of poor outcome. In India, Verma R et al (2013)⁴⁵ have shown that MRC sum score <30 on hospital admission is a predictor of poor outcome at 6 months.

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**Prognosis based on history of diarrhoea**

Statistical analysis test revealed a p-value of 0.0023 between the two groups of patients with good and poor prognosis. Arami MA et al (2006)⁴⁴ and Walgaard et al (2011)⁴² have shown a significant correlation between history of diarrhoea and worse outcome at 6 months. Visser et al (1999)⁴³ have shown in their study previous gastrointestinal infection being associated with a poor outcome. History of diarrhoea leading to a poor outcome has also been shown by Hadden RDM et al (2001)⁴⁵ and van Koningsveld et al (2007)⁴⁶.

**Prognosis based on autonomic dysfunction**

15 out of 22 patients (68.18%) with poor prognosis showed autonomic dysfunction as against 10 out of 30 patients (33.33%) with good prognosis. Statistical analysis test showed the p-value to be 0.023 which is significant. In India Kalita J et al (2014)²² and Verma R et al (2013)⁴³ have reported autonomic dysfunction to be a marker of poor outcome. However, Singh NK et al (1987)³⁹ have found no association between autonomic dysfunction and clinical outcome of the patient.

**Prognosis based on cranial nerve involvement**

Out of 22 patients with poor prognosis 16 (72.72%) patients had cranial nerve involvement while cranial nerve was not involved in 6 (27.28%) patients. Statistical analysis showed significant p-value. Rajabally et al. 2012² have reported facial and/or bulbar weakness at admission was a strong predictor of mechanical ventilation and poor prognosis. Sundar U et al. 2005⁴⁸ reported that bulbar weakness is a predictor of poor outcome.

<table>
<thead>
<tr>
<th>Table 3: The prognostic factors evaluated.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Prognostic Factors</strong></td>
</tr>
<tr>
<td>1. Age</td>
</tr>
<tr>
<td>2. Antecedent history of Diarrhoea</td>
</tr>
<tr>
<td>3. Cranial Nerve involvement</td>
</tr>
<tr>
<td>4. Autonomic dysfunction</td>
</tr>
<tr>
<td>5. MRC sum score at presentation</td>
</tr>
<tr>
<td>6. Electrophysiological Study</td>
</tr>
</tbody>
</table>

**Prognosis based on electrophysiological studies**

Our study showed axonal features in 40.91% of the patients with poor prognosis by electrophysiological studies. Demyelinating neuropathy was detected in 59.09% of the patients with poor prognosis. Statistical analysis test showed p-value to be 0.064 which is insignificant. The Plasma Exchange Sandoglobulin Trial⁴⁰ has shown no difference of outcome between axonal and demyelinating variety of GBS. In India, Kalita J et al (2014)²² have reported better outcome in AIDP as compared with AMAN. Kuwabara et al
have reported that patients with axonal Guillain-Barré syndrome can show both rapid and slow recoveries.

CONCLUSION
This study concludes that there are several factors which play a key role in the prognosis of the disease. Most of the patients having poor prognosis were of advanced age, had severe disability at presentation, had a previous history of diarrhoea, had some autonomic dysfunctions or had cranial nerve involvement. It is, therefore, crucial that these factors are identified early with an aim to treat and manage them, and subsequently ensure better outcomes.

REFERENCES

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Conflict of Interest: None Declared.
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ANNEXURES

1. Guillain-Barré Syndrome Disability Score advocated by Hughes RA et al, 1978

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Healthy state</td>
</tr>
<tr>
<td>1</td>
<td>Minor symptoms and capable of running</td>
</tr>
<tr>
<td>2</td>
<td>Able to walk 10 m or more without assistance but unable to run</td>
</tr>
<tr>
<td>3</td>
<td>Able to walk 10 m across an open space with help</td>
</tr>
<tr>
<td>4</td>
<td>Bedridden or chair bound</td>
</tr>
<tr>
<td>5</td>
<td>Requiring assisted ventilation for at least part of the day</td>
</tr>
<tr>
<td>6</td>
<td>Dead</td>
</tr>
</tbody>
</table>

2. MRC sum score: Power of the muscles was assessed by MRC sum score, a summation of the MRC grades (range, 0–5) given in full numbers of the following muscle pairs: upper arm abductors, elbow flexors, wrist extensors, hip flexors, knee extensors, and foot dorsal flexors as given by Kleyweg RP et al, 1991. The MRC sum score ranged from 0 (“total paralysis”) to 50 (“normal strength”).

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No contraction</td>
</tr>
<tr>
<td>1</td>
<td>Flicker or trace of contraction</td>
</tr>
<tr>
<td>2</td>
<td>Active movement with gravity eliminated</td>
</tr>
<tr>
<td>3</td>
<td>Active movement against gravity</td>
</tr>
<tr>
<td>4</td>
<td>Active movement against gravity and resistance</td>
</tr>
<tr>
<td>5</td>
<td>Normal power</td>
</tr>
</tbody>
</table>