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Original Article

# Clinical Outcome of Intravenous Immunoglobulin in the treatment of Guillain Barre Syndrome in a Nepalese Tertiary Centre

*Rajeev Ojha[1], Ragesh Karn[1]*

*[1]Neurology Department, Tribhuvan University Teaching Hospital, Maharajgunj, Kathmandu, Nepal*

## ABSTRACT

**Introduction:** Intravenous Immunoglobulin is an approved therapy for Guillain Barre Syndrome. The objective of our study is to understand the management and outcome in Guillain Barre Syndrome patients treated with Immunoglobulin.

**Materials and Methods:** All consecutive patients were retrospectively evaluated in the study were of age ≥16 years and were being admitted in department of Neurology of Tribhuvan University Teaching Hospital, Kathmandu, Nepal from 2016 March to 2017 February.

**Results:** A total of 46 patients were included, mean age= 36.5±16.2 years, range = 16years to 80 years. Thirty-two patients (70%) were axonal variant, acute motor axonal neuropathy being more common (18 patients). Intravenous immunoglobulin was used in 23 patients (50%), 17 of them were axonal variant and 6 were demyelinating. Guillain Barre Syndrome patients with bilateral facial weakness (70% vs 30%; p<0.05) were likely to receive immunoglobulin therapy. Patients with immunoglobulin therapy were found to have higher ODSS at Nadir (9.3±1.8 vs 6.9±1.9; p <0.001) and discharge than patients without immunoglobulin treatment (6.2±1.7 vs 5.0±1.6; p=0.001). At Nadir, Patients with immunoglobulin therapy were found to have higher Guillain Barre Syndrome disability score (4.1±0.7 vs 3.2±0.9; p<0.095). In immunoglobulin group, Axonal variants were found to have higher ODSS score (9.6±1.9 vs 8.2±0.9, p=0.027) and Guillain Barre Syndrome disability score (4.2±0.7 vs 3.5±0.5; p=0.019) at nadir than demyelinating group.

**Conclusions:** Intravenous Immunoglobulin is easier to administer and is safe with less adverse effects. Although expensive, it is an effective treatment option in resource limited center. Axonal variants are clinically severe and likely to be need of Intravenous Immunoglobulin therapy.

**Keywords:** Axonal; Demyelinating; Guillain Barre Syndrome; Immunoglobulin; Neuropathy

**Correspondence:**
Dr Rajeev Ojha
Neurology Department, Tribhuvan University Teaching Hospital, Maharajgunj, Kathmandu, Nepal
ORCID ID: 0000-0001-7680-7036

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## INTRODUCTION

Guillain-Barré syndrome (GBS) is an acute neuromuscular paralysis characterized by rapidly progressive, symmetric weakness, ascending in pattern with areflexia. The incidence of GBS varies worldwide, ranges from 0.6-4 per 100,000 population.[1,2] Mortality usually occurs due to respiratory failure, aspiration pneumonia, autonomic dysfunction and pulmonary embolism.[3,4] Poor prognosis may be associated with older age, antecedent diarrhea, need for mechanical ventilation, rapid onset of weakness and severe muscle weakness during admission.[5] Despite treatment, mortality varies from 2.8% to 10% in various studies and 20% are still unable to walk after 6 months.[6,7]

Previously, steroids have been tried in the treatment of GBS, but was found to have no role.[8,9] Treatments like immunoglobulin and plasmapheresis have been found to be effective in acute period, helping in reducing the severity of disease and early recovery.[10] But the cost of these treatments are very high and are unaffordable to many people where health insurance is still in primitive phase.[11,12] To minimize the high cost, few studies have found low volume plasma exchange (PE) to be an effective treatment for autoimmune diseases like GBS.[12,13] Recent studies have found the efficacy of intravenous Immunoglobulin (IVIg) is comparable to plasma exchange.

Our study aims to understand the management and outcome in GBS patients in whom IVIg was used. There are very few studies published on IVIg use among GBS patients in South-Asia, and no such study has been reported from Nepal.

## MATERIALS AND METHODS

Patients were retrospectively evaluated who were admitted with diagnosis of GBS in Neurology department of Tribhuvan University Teaching Hospital, Kathmandu, Nepal from 2016 March to 2017 February. Inclusion criteria were patient of age more than 16 presenting with acute bilateral flaccid limb weakness along with hyporeflexia or areflexia or cranial nerve palsy or have history of antecedent viral infection; and nerve conduction study showing axonal or demyelinating neuropathy satisfying the pattern of any GBS variants. Excluded patients were the patients with peripheral neuropathy other than GBS. Historical, demographic, clinical and laboratory data were collected from all patients. Laboratory investigations included complete blood count, blood Urea, Creatinine, Sodium, Potassium and Sugar, Chest radiogram, electrocardiogram, nerve conduction studies, and cerebrospinal fluid analysis. Informed consents were obtained from patients. Ethics committee approval was given by institutional review board of Tribhuvan University Institute of Medicine, ref no: 21 (6-11-E).

Patients were evaluated for fulfillment of the diagnostic criteria of GBS from National institute of neurological disorders and stroke. Brighton's criteria[14] were also evaluated to look for the level of evidence met for diagnosis of GBS. Clinical severity was described by Overall disability sum score (ODSS) and GBS disability score. Medical Research Council (MRC) grading was used to measure the muscle strength of each joint. IVIg was given in patients with progressive weakness of limbs within 4 weeks of clinical history with significant disability or presented with bulbar or respiratory involvement. The dose of IVIg used in our patients was 0.4mg/kg for 5 days.

### STATISTICAL ANALYSIS

IBM SPSS statistics 20.0 was used for analyzing and storage of data. The statistical analysis included calculation of means, standard deviations, range, frequencies and percentages. Means of demographic profiles were compared by independent sample t test. Comparison of means of ODSS and GBS Disability score between GBS variants and IVIg and without IVIg group were also compared by independent sample t test. Chi-square tests were used for the descriptive statistics. A two tailed p-value<0.05 was considered statistically significant.

## RESULTS

Total 46 patients were included in the study, 32 male (69.6%) and 14 female (30.4%); mean age of patients was 36.5±16.2, range = 16years to 80 years. IVIg was given in 14 male patients (61%) and 9 female patients(39%) as shown in Table 1. Facial nerve was the most common cranial nerve to get involved in GBS (56.5%), unilateral in 3 patients and bilateral in 23 patients. Among patients with absent facial weakness, only 4 of them (17.4%) were treated with IVIg, and rest 16 patients (69.6%) weren't treated with IVIg($p<0.05$). Facial weakness was seen in 19 axonal variants (60%), 17 of them were having bilateral weakness, and 7 demyelinating variants(50%) had facial weakness, being bilateral in 1. Patients with bilateral facial weakness were given IVIg in 16 of them, while 7 patients weren't given ($p<0.05$). No difference in weakness pattern has been found between with or without IVIg patients. Hospital admission duration was found to be 21.0±11.6 days in patients with IVIg and 10.4±4.0 days in patients without IVIg ($p<0.001$).

**Table 1: Baseline demography of GBS patients**

| Clinical Characteristics | IVIg (n=23) | Without IVIg (n=23) | Total |
|---|---|---|---|
| **Age (mean ± SD) years** | 36.3±14.2 | 36.7.7±18.2 | 36.5±16.1 |
| **Sex** | | | |
| Male | 14 (61%) | 18 (78.3%) | 32 |
| Female | 9 (39%) | 5 (21.7%) | 14 |
| **Facial Weakness** | | | |
| Absent | 4(17.4%) | 16(69.6%) | 20[b] |
| Unilateral | 3(13.0%) | 0 | 3 |
| Bilateral | 16(69.6%) | 7(30.4%) | 23[b] |
| **Weakness Pattern** | | | |
| Ascending | 20(87.0%) | 21(91.3%) | 41 |
| Descending | 2(8.7%) | 0 | 2 |
| Together | 1(4.3%) | 2(8.7%) | 3 |
| **Nadir Duration** (days) | 10.4±4.2 | 11.1±4.2 | 10.7±4.2 |
| **Hospital Admission (days)** | 21.0±11.6 | 10.4±4.0 | 15.7±10.2[a] |
| **Mechanical Ventilation needed** | 8 (25.0%) | 4 (17.3%) | 12 |
| **Variants** | | | |
| **Axonal** | 17 (73.9%) | 15(64.8%) | 32 |
| AMAN | 10(43.5%) | 8(34.8%) | 18 |
| AMSAN | 7(30.4%) | 7(30.4%) | 14 |
| **AIDP** | 6(26.1%) | 8(34.8%) | 14 |

Data are expressed as n (%) or mean ± standard deviation(SD); Acute inflammatory demyelinating polyneuropathy; AMAN: Acute motor axonal neuropathy; AMSAN: Acute motor and sensory neuropathy; URTI: Upper respiratory tract infection, AGE:acute gastroenteritis; ODSS: Overall disability sum score; GBS: Guillain Barre Syndrome; IVIg: Intravenous Immunoglobulin; a: P-value<0.001, b: P-value<0.05

Thirty-two patients (70%) were axonal type, out of which 18 patients were AMAN and 14 patients were AMSAN. 14 patients (30%) were demyelinating type, out of which 11 patients had both motor and sensory features, and only 3 patients were pure motor demyelinating type. Patient with facial weakness were found to have higher ODSS at nadir(8.0±1.0 vs 6.0±2.0; p=0.043) and discharge(5.4±0.5 vs 4.0±1.0; p=0.009). But no statistical significance was found between these groups according to GBS disability score at nadir (3.4±0.5 vs 2.8±0.9; p=0.174) and discharge (2.57±0.5 vs 2.1±0.4; p=0.109).

Patients with IVIg were found to have higher ODSS at Nadir (9.3±1.8 vs 6.9±1.9; p <0.001) and discharge than patients without IVIg treatment (6.2±1.7 vs 5.0±1.6; p=0.001) as shown in Table 2. At Nadir, Patients with IVIg were found to have higher GBS disability score (4.1±0.7 vs 3.2±0.9; p<0.095), whereas no significant difference was found between with or without IVIg group in GBS disability score during discharge (2.9±0.8 vs 2.5±0.8; p<0.095). In IVIg group, Axonal variants were found to have higher ODSS score(9.6±1.9 vs 8.2±0.9, p=0.027) and GBS disability score (4.2±0.7 vs 3.5±0.5; p=0.019) at nadir than demyelinating group (Table 3), whereas no significant difference was found between these groups at discharge.

**Table 2: Clinical Severity according to ODSS and GBS disability score in IVIg and without IVIg group**

| Severity score | IVIg (n=23) | Without IVIg (n=23) | p value |
|---|---|---|---|
| **ODSS at Nadir** | 9.3±1.8 | 6.9±1.9 | <0.001 |
| **ODSS at Discharge** | 6.2±1.7 | 5.0±1.6 | 0.001 |
| **GBS Disability score (Nadir)** | 4.1±0.7 | 3.2±0.9 | <0.05 |
| **GBS Disability score (Discharge)** | 2.9±0.8 | 2.5±0.8 | 0.095 |

Data are expressed as mean ± standard deviation(SD); ODSS: Overall disability sum score; GBS: Guillain Barre Syndrome.

Total duration of hospital stay was higher in axonal variant in IVIg group, but no statistical difference was found. In without IVIg group (Table 4), no significant differences was found in ODSS between axonal and demyelinating variants at nadir(7.3±1.8 vs 6.1±1.8, p=0.152), ODSS at discharge (5.4±1.7 vs 4.1±1.0; p=0.057), GBS disability score at nadir (3.4±0.9 vs 2.8±0.8; p=0.019) and GBS disability score at discharge ( 2.7±0.9 vs 2.2±0.5; p=0.166). In IVIg group, no significant improvement was found in ODSS (3.1±1.0 vs 2.6±1.0; p=0.375) and GBS disability score (1.2±0.5 vs 1.0±0.6; p=0.510) difference in nadir and discharge between axonal and demyelinating variants (Table 5). In IVIg group, there was no significant improvement was found in ODSS (3.1±1.0 vs 2.6±1.0; p=0.375) and GBS disability score (1.2±0.5 vs 1.0±0.6; p=0.510) difference in nadir and discharge between axonal and demyelinating variants (Table 5). In without IVIg group, there was also no significant

**Table 3: Clinical Severity according to ODSS and GBS disability score in IVIg group patients**

| Severity score | axonal (n=17) | Demyelinating (n=6) | p value |
|---|---|---|---|
| **ODSS at nadir** | 9.6±1.9 | 8.2±0.9 | 0.027 |
| **ODSS at discharge** | 6.5±1.9 | 5.5±0.5 | 0.058 |
| **GBS disability score (nadir)** | 4.2±0.7 | 3.5±0.5 | 0.019 |
| **GBS disability score (discharge)** | 3.1±0.8 | 2.5±0.5 | 0.090 |

improvement was found in ODSS (1.8±0.9 vs 2.0±1.2; p=0.768) and GBS disability score (0.7±0.5 vs 0.6±0.5; p=0.850) difference in nadir and discharge between axonal and demyelinating variants (Table 6)

**Table 4: Clinical Severity according to ODSS and GBS disability score in without IVIg group patients**

| Severity score | axonal (n=15) | Demyelinating (n=8) | p value |
|---|---|---|---|
| **ODSS at nadir** | 7.3±1.8 | 6.1±1.8 | 0.152 |
| **ODSS at discharge** | 5.4±1.7 | 4.1±1.0 | 0.057 |
| **GBS disability score (nadir)** | 3.4±0.9 | 2.8±0.8 | 0.190 |
| **GBS disability score (discharge)** | 2.7±0.9 | 2.2±0.5 | 0.166 |

Data are expressed as mean ± standard deviation (SD); ODSS: Overall disability sum score; GBS: Guillain Barre Syndrome.

**Table 5: Clinical Severity according to ODSS and GBS disability score in IVIg group patients**

| Severity score difference (Nadir – Discharge) | axonal (n=17) | Demyelinating (n=6) | p value |
|---|---|---|---|
| **ODSS (IVIg group)** | 3.1±1.0 | 2.6±1.0 | 0.375 |
| **GBS Disability score (IVIg group)** | 1.2±0.5 | 1.0±0.6 | 0. 510 |

Data are expressed as mean ± standard deviation (SD); ODSS: Overall disability sum score; GBS: Guillain Barre Syndrome.

**Table 6: Clinical Severity according to ODSS and GBS disability score in without IVIg group patients**

| Severity score difference (Nadir – Discharge) | axonal (n=15) | Demyelinating (n=8) | p value |
|---|---|---|---|
| **ODSS (IVIg group)** | 1.8±0.9 | 2.0±1.2 | 0.768 |
| **GBS Disability score (IVIg group)** | 0.7±0.5 | 0.6±0.5 | 0. 850 |

Data are expressed as mean ± standard deviation(SD); ODSS: Overall disability sum score; GBS: Guillain Barre Syndrome.

IVIg course was repeated in 1 patient due to clinical fluctuation. Twelve patients (26.1%) needed mechanical intubation, 8 patients were IVIg group and 4 patients were without IVIg . Mortality was reported in 2 patients (4%), one during recovery phase due to dysautonomia and another in intensive care unit due to pneumonia and sepsis. No severe adverse reactions of the drug were seen in our patients during IVIg infusion period except low grade fever in 2 patients and mild headache in 2 patients.

## DISCUSSION

This is the first study from Nepal to evaluate the GBS patients who were treated with IVIg. Among 46 included GBS patients, 50% of them were treated with IVIg. In a recent study in India, only 16% elderly and 6% adult GBS patients were treated with IVIg infusion, whereas 84% elderly and 92% adult GBS patients received PE.[6] In low economic countries, use of plasma exchange is high due to the cost factor where the facility of low volume plasma exchange is available.[12] Variation in the number of patients treated with IVIg, PE or symptomatic management has been found in different studies.[6,12] In our study, 50% of the patients presented at our center just had mild symptoms at presentation, didn't progress much to nadir, and earlier improvement was seen. We suppose variation of patients with clinical severity is seen in neurology department of general hospitals where clinically severe patients are likely to be referred to neuro-centers.

Bilateral Facial Palsy is a common finding in GBS, which can be a manifestation of severe form.[15] Previous study has reported Facial palsy is associated with severity in demyelinating, but not in axonal variant.[16] In contrast to this, our study has found facial weakness was common in axonal variants and are clinically severe at nadir and discharge according to ODSS severity scale. In a north Indian study, axonal variants were found to be associated with poor functional outcome and patients with facial weakness were likely to need mechanical ventilation.[17]

Prevalence of predominant variants of GBS varies with studies, however demyelinating variants were common in most studies in western countries.[18,19] In our study, axonal variants were predominant as well as clinically severe, and most of them needed IVIg therapy. Recovery in axonal variants were found to be delayed in study by Feasdy et al.[20] Our study is in accordance with most Asian studies those have reported axonal variants being predominant and severe.[21] But there are also Asian studies with higher demyelinating variants[6] which shows frequency of variants might not be confined to specific geographical location. Our study also showed that no significant difference was found in the improvement in disability between axonal and demyelinating variants both in IVIg and without IVIg group.

Treatment relapses with IVIg therapy has been reported in previous GBS studies[22], but Romano et al didn't find any support for this statement.[23] In our study, 1 patient developed fluctuation in the course after first course of IVIg therapy and second course was needed to be given. In a study by Castro et al, a higher incidence of treatment related fluctuations with IVIg was reported and most of them improved with PE.[24]

Mortality and morbidity in GBS patients was comparatively high without the facility of intensive care unit.[4] Respiratory failure, Dysautonomia, sepsis and myocardial infarction were the common causes of mortality.[6,18,25] Since IVIg is given in patients with severe Weakness with or without respiratory muscles weakness, it is likely that these patients are at higher risk of complications. One of our patients died in ICU due to sepsis and another due to dysautonomia in recovery phase. Delay in the treatment initiation could also be a factor for high morbidity and mortality.[18]

IVIg therapy is comparatively safe and usually has only few and mild adverse events like headache fever, chills, myalgia, chest discomfort and nausea.[26] Rare serious reactions are stroke, pulmonary embolism, severe headache, anaphylactics and renal tubular necrosis.[26,27] IVIg infusion was fairly completed in all patients without interruption, 2 patients had low grade fever and 2 had mild headache during the course.

## CONCLUSIONS

IVIg is easier to administer and is safe with less adverse effects. Although expensive, it is an effective treatment option in resource limited center. Axonal variants are clinically severe and likely to need IVIg therapy. Since IVIg is usually used in moderate to severely disabled patients with or without bulbar or respiratory involvement, delay in disease progression or improvement can be observed within few days of initiation of treatment. In situation of Treatment relapses with IVIg, second course of IVIg can be given if PE is not available. GBS patients with severe weakness with respiratory involvement or dysautonomia are likely to have high risk of mortality and should be managed with high care and carefully monitored during the hospital stay.

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