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Original Research Article

An audit on the clinical and laboratory profile of patients with different variants of Guillain-Barre syndrome and effect of various treatment strategies on their recovery

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ABSTRACT

Background: Guillain-Barre syndrome (GBS) is an immune-mediated disorder of the peripheral nervous system, causing muscle weakness, paralysis, and sensory deficits. Its treatment mainly involves supportive care, immunomodulatory therapies such as intravenous immunoglobulin (IVIg) and plasma exchange (PE), and rehabilitation. Several randomized controlled trials (RCTs) and meta-analyses have evaluated the efficacy and safety of apheresis in GBS, but the results have been conflicting and limited by methodological issues.
Methods: This is a retrospective study with a sample of 30 patients carried out at neurology OPD of tertiary care centre in Pune, Maharashtra over a period of 32 months from July 2020 and February 2023. Patients were followed up for six months, and their outcomes were compared in terms of the improvement of clinical disability scores, the need for mechanical ventilation, and the time to recovery of walking ability and other functional outcomes.
Results: Apheresis treatment significantly improved the clinical disability scores and NCV recovery of patients with GBS in comparison to IVIg and corticosteroids. Moreover, patients who received apheresis treatment showed a shorter time to recovery of walking ability and other functional outcomes than those who did not. Symptomatic differences were seen between patients with different subtypes of GBS, but there was no difference in the response to apheresis or IVIg between subtypes.
Conclusions: Treatment with apheresis should be considered in patients not responding to conservative management. Earlier treatment with apheresis has shown to have good clinical and electrophysiological outcomes regardless of the GBS subtype.

Keywords: GBS, Demyelination, Apheresis, IVIg

INTRODUCTION

Guillain-Barre syndrome (GBS) is an acute, immune-mediated disorder of the peripheral nervous system, characterized by rapid-onset, progressive muscle weakness and paralysis associated with areflexia. It can affect the respiratory and autonomic nervous systems, and occasionally the cranial nerves like facial nerve, lower cranial nerves can get involved.[1] GBS is the most common cause of acute flaccid paralysis worldwide, with an annual incidence of 0.6 to 4 cases per 100,000 population.[2] Although the precise cause of GBS is unknown, it is thought to be brought on by previous infection like viral or bacterial or post vaccination. It is thought to be triggered by molecular mimicry between infectious agents and peripheral nerve antigens, leading to an autoimmune response that damages the myelin and axons of peripheral nerves.[3]

The diagnosis of GBS is mainly based on clinical features, such as the rapid progression of weakness, areflexia, and sensory deficits, and supportive laboratory

findings, such as reduced nerve conduction velocity with prolonged distal latencies on electrophysiological study, elevated protein levels in the cerebrospinal fluid (CSF) and the presence of specific antibodies, such as anti-ganglioside antibodies.[4,5] Electrophysiological tests can differentiate between the different subtypes of GBS and show PNS impairment. Acute inflammatory demyelinating polyneuropathy (AIDP), acute motor axonal neuropathy (AMAN), acute motor and sensory axonal neuropathy (AMSAN) and Miller Fisher syndrome (MFS) are a few subtypes of GBS, each with unique clinical characteristics and therapeutic options.

The treatment of GBS is mainly supportive and includes close monitoring of respiratory and autonomic function, immunomodulatory therapies, such as IVIg and PE, and rehabilitation.[6] IVIg and PE are considered first-line therapies for GBS, but their efficacy and optimal timing of administration remain controversial.[7]

Apheresis is a blood purification technique that removes plasma, including damaging antibodies and other potentially harmful substances and inflammatory mediators that leads to nerve injury from the patient's blood and replaces it with a replacement fluid, such as albumin or Fresh frozen plasma.[8] Apheresis can be performed by different methods, including PE, double-filtration plasmapheresis (DFPP), and immunoadsorption (IA), and can be used to treat various autoimmune and haematological disorders.[9] The rationale for using apheresis in GBS is to remove the pathogenic antibodies and other immune mediators that contribute to nerve damage and to restore balance of the immune system.[6]

RCTs and meta-analyses have evaluated the efficacy and safety of apheresis in GBS, but the results have been conflicting and limited by methodological issues, such as small sample size, heterogeneous patient populations, and variations in the apheresis techniques and protocols.[7,8] Moreover, most of the studies have focused on the short-term outcomes, such as the improvement of clinical disability scores and the need for mechanical ventilation and have not adequately addressed the long-term effects of apheresis on nerve conduction velocity (NCV) and electrophysiological recovery, which are important prognostic factors for GBS.[5]

Therefore, the present study aims to evaluate the effects of apheresis on the clinical and electrophysiological recovery of patients with various subtypes of GBS, using a longitudinal design that follows up the patients for up to 6 months. The specific objectives of the study were to compare the outcomes of apheresis, IVIg, and corticosteroids in patients with GBS, in terms of the improvement of clinical disability scores, the need for mechanical ventilation, and the time to recovery of walking ability and other functional outcomes in patients with different subtypes of GBS, and to compare the recovery of patients with different subtypes of GBS who received apheresis treatment versus those who did not.

METHODS

Study design

This is a retrospective cohort study that was conducted at the neurology OPD of a tertiary care hospital. The study will retrospectively follow patients for period of 6 months to assess effects of apheresis treatment on clinical and NCV recovery in patients with various subtypes of GBS.

Setting

The study was conducted at a single centre, in the neurology OPD of a tertiary care hospital in the city of Pune, Maharashtra over a period of 32 months from July 2020 and February 2023.

Participants

Participants will be recruited from GBS patients coming for follow-up in the neurology OPD of the hospital. Eligible participants will be patients who are diagnosed with GBS and meet the inclusion criteria, which include age 18 years or older, diagnosis of GBS, and availability for follow-up.

Sample size

Study included 30 patients who diagnosed with GBS during study duration who fulfilled inclusion criteria.

Inclusion criteria

Patients of age >18 years, patients who received treatment for GBS and patients who had confirmed variants of GBS with NCV and CSF examination were included.

Exclusion criteria

Patients who were diagnosed with any other condition can cause neuropathy or neurological symptoms. Patients who were not treated for GBS. Known allergy to treatment (Apheresis, IVIg), patients who had incomplete medical records or missing data. Patients who received treatment for GBS at another healthcare facility before coming to the tertiary care centre were excluded.

Data collection

Baseline demographic and clinical data, including disease subtype, severity, and NCV, will be collected for all eligible participants. Clinical and NCV assessments will be performed at baseline and at regular intervals during follow up for a period of 6 months.

Statistical analysis

Data are summarized as medians (interquartile ranges) for continuous variables and as frequencies (percentages) for

categorical variables. Evaluation of the association between continuous variables is done using the Wilcoxon rank sum test (for two groups) and the Kruskal Wallis test (for more than two groups), while the chi-square of the Fisher's exact tests is used for categorical variables as appropriate. Kaplan-Meier analyses are performed to evaluate the time to recovery. The logrank test is used to compare recovery times between the groups.

P values less than 0.05 are considered to be statistically significant. All hypotheses are formulated using 2 tailed alternatives against each null hypothesis. Data analysed using R, ver 4.2.3 (R Project for statistical computing).

Ethical considerations

The study will be conducted in accordance with the declaration of Helsinki and approved by the institutional review board. Informed consent will be obtained from all participants prior to enrolment, and patient confidentiality will be maintained throughout the study. Adverse events will be monitored and reported to the appropriate authorities as necessary.

RESULTS

This study included total of 30 patients with GBS of various types. Among them, 15 patients had acute motor axonal neuropathy (AMAN), 8 had acute motor sensory axonal neuropathy (AMSAN), and 7 had other types of GBS, including acute inflammatory demyelinating polyneuropathy (AIDP), acute pandysautonomia (APRN), and Miller Fisher syndrome (MFS).

There were no significant differences in the age and sex distribution among the three GBS types (Table 1). However, there was a significant difference in the distribution of weakness, paraesthesia's, bladder control, and sensory abnormalities. Asymmetric weakness was more common in AMSAN (75%) than in other GBS types, while symmetric weakness was more common in AMAN (100%). Paraesthesia's were more common in AMSAN (62%) and less common in AMAN (0%). Bladder control was more frequently affected in other GBS types (57%), while it was preserved in most AMAN and AMSAN cases. Sensory abnormalities were more common in other GBS types (88%) than in AMAN (0%).

Other clinical features, such as the presence of a preceding lower respiratory tract infection, diarrhoea, COVID-19 infection, pain, dysphagia, dyspnoea, dysarthria, duration of symptoms, power of upper and lower limbs, and deep tendon reflexes, did not differ significantly among the three GBS types.

Patients who were given IVIg had a significantly lower duration of symptoms (4 days as opposed to 9 days in those who weren't given IVIg, p=0.025) (Table 2). The recovery duration in patients who underwent apheresis was also significantly lower (median of 7 days vs median of 10 days in those who weren't, $p_{logrank}$=0.0089). (Figures 1-3).

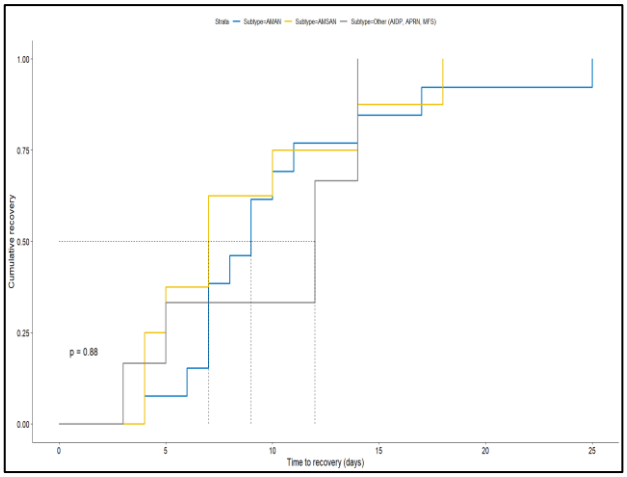

Figure 1: Kaplan-Meier plot with the logrank test showing time to recovery for GBS subtypes.

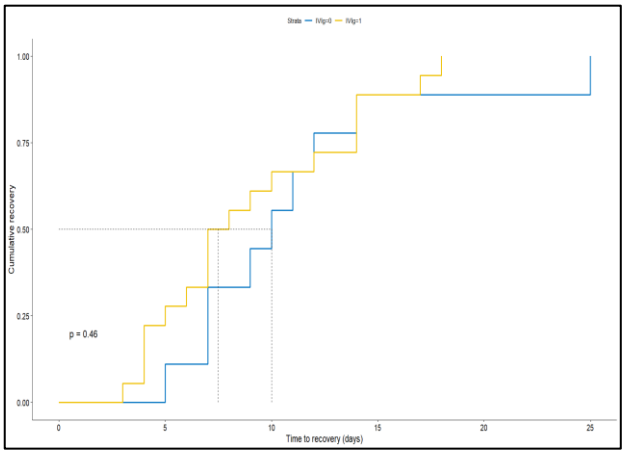

Figure 2: Kaplan-Meier plot with the logrank test showing time to recovery for patients given versus not given IVIg.

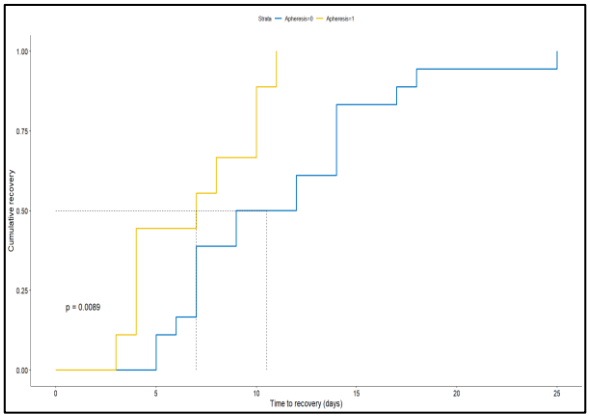

Figure 3: Kaplan-Meier plot with the log rank test showing time to recovery for patients given versus not given apheresis.

Table 1: Overall and subtype specific characteristics of GBS.

Variables	Overall, n=30[1], (%)	GBS type, n (%)			P value[2]
		AMAN, n=15[1]	AMSAN, n=8[1]	Other (AIDP, APRN, MFS), n=7[1]	
Characteristic					
Age (Years)	37 (25, 52)	38 (21, 51)	39 (33, 49)	33 (27, 54)	>0.9
Sex					
Female	6 (20)	3 (20)	3 (38)	0 (0)	0.3
Male	24 (80)	12 (80)	5 (62)	7 (100)	
Past history					
LRTI					
Absent	20 (67)	12 (80)	4 (50)	4 (57)	0.3
Present	10 (33)	3 (20)	4 (50)	3 (43)	
COVID					
Absent	29 (97)	15 (100)	8 (100)	6 (86)	0.2
Present	1 (3.3)	0 (0)	0 (0)	1 (14)	
Diarrhoea					
Absent	22 (73)	9 (60)	7 (88)	6 (86)	0.4
Present	8 (27)	6 (40)	1 (12)	1 (14)	
Other					
Absent	28 (93)	14 (93)	7 (88)	7 (100)	>0.9
Present	2 (6.7)	1 (6.7)	1 (12)	0 (0)	
Symptoms					
Weakness					
Asymmetric	8 (27)	0 (0)	6 (75)	2 (29)	<0.001
Symmetric	22 (73)	15 (100)	2 (25)	5 (71)	
Coordination					
Normal	30 (100)	15 (100)	8 (100)	7 (100)	
Sensory					
Paraesthesia’s					
Absent	24 (80)	15 (100)	3 (38)	6 (86)	0.002
Present	6 (20)	0 (0)	5 (62)	1 (14)	
Pain					
Absent	10 (33)	3 (20)	4 (50)	3 (43)	0.3
Present	20 (67)	12 (80)	4 (50)	4 (57)	
Bladder control					
Absent	3 (10)	0 (0)	0 (0)	3 (43)	0.009
Present	27 (90)	15 (100)	8 (100)	4 (57)	
Dysphagia					
Absent	27 (90)	13 (87)	7 (88)	7 (100)	>0.9
Present	3 (10)	2 (13)	1 (12)	0 (0)	
Dyspnoea					
Absent	25 (83)	13 (87)	6 (75)	6 (86)	0.8
Present	5 (17)	2 (13)	2 (25)	1 (14)	
Dysarthria					
Absent	29 (97%)	15 (100%)	7 (88%)	7 (100%)	0.5
Present	1 (3.3%)	0 (0%)	1 (12%)	0 (0%)	
Duration of symptoms (days)	5 (3, 9)	4 (3, 9)	8 (4, 12)	5 (4, 6)	0.4
Clinical examination					
Power upper limb	4 (2, 5)	3 (2, 4)	4 (3, 5)	4 (3, 5)	0.3
Power lower limb	3 (2, 4)	3 (2, 4)	3 (2, 3)	3 (2, 3)	0.8
DTR					
Absent	18 (60)	9 (60%)	6 (75%)	3 (43%)	0.4
Decreased	7 (23)	2 (13%)	2 (25%)	3 (43%)	
Present	5 (17)	4 (27%)	0 (0%)	1 (14%)	

Continued.

Variables	Overall, n=30[1], (%)	GBS type, n (%)			P value[2]
		AMAN, n=15[1]	AMSAN, n=8[1]	Other (AIDP, APRN, MFS), n=7[1]	
Clinical examination					
Sensory					
Absent	7 (23)	1 (6.7)	5 (62)	1 (14)	0.012
Present	23 (77)	14 (93)	3 (38)	6 (86)	
Investigations					
CSF protein	3.00 (1.00, 5.75)	5.00 (1.50, 5.00)	1.50 (0.63, 3.25)	6.00 (0.75, 7.00)	0.3
NCV					
AIDP	3 (10)	0 (0)	0 (0)	3 (43)	<0.001
AMAN	15 (50)	15 (100)	0 (0)	0 (0)	
AMSAN	8 (27)	0 (0)	8 (100)	0 (0)	
APRN	2 (6.7)	0 (0)	0 (0)	2 (29)	
MFS	2 (6.7)	0 (0)	0 (0)	2 (29)	
Treatment					
IVIG					
Absent	9 (30)	4 (27)	4 (50)	1 (14)	0.3
Present	21 (70)	11 (73)	4 (50)	6 (86)	
PE apheresis					
Absent	19 (63)	9 (60)	5 (62)	5 (71)	>0.9
Present	11 (37)	6 (40)	3 (38)	2 (29)	
Steroids					
Absent	15 (50)	6 (40)	5 (62)	4 (57)	0.6
Present	15 (50)	9 (60)	3 (38)	3 (43)	
Recovery	27 (90)	13 (87)	8 (100)	6 (86)	0.6
Recovery duration (Days)	9 (6, 13)	9 (7, 11)	7 (5, 11)	12 (7, 14)	0.7
Unknown	3	2	0	1	

[1]Median (IQR); n (%), [2]Kruskal-Wallis rank sum test; Fisher's exact test.

Table 2: Overall and treatment specific characteristics of patients with GBS.

Variables	Overall, n=30[1] (%)	IVIg, n (%)			Apheresis, n (%)			Corticosteroids, n (%)		
		Absent, n=9[1]	Present, n=21[1]	P[2]	Absent, n=19[1]	Present, n=11[1]	P[3]	Absent, n=15[1]	Present, n=15[1]	P[3]
Characteristic										
Age (Years)	37 (25, 52)	26 (22, 38)	49 (29, 54)	0.051	36 (24, 50)	44 (26, 56)	0.5	29 (24, 40)	50 (32, 58)	0.029
Sex										
Female	6 (20)	3 (33)	3 (14)	0.3	2 (11)	4 (36)	0.2	3 (20)	3 (20)	>0.9
Male	24 (80)	6 (67)	18 (86)		17 (89)	7 (64)		12 (80)	12 (80)	
Past history										
LRTI										
Absent	20 (67)	5 (56)	15 (71)	0.4	12 (63)	8 (73)	0.7	8 (53)	12 (80)	0.12
Present	10 (33)	4 (44)	6 (29)		7 (37)	3 (27)		7 (47)	3 (20)	
COVID										
Absent	29 (97)	9 (100)	20 (95)	>0.9	18 (95)	11 (100)	>0.9	14 (93)	15 (100)	>0.9
Present	1 (3.3)	0 (0)	1 (4.8)		1 (5.3)	0 (0)		1 (6.7)	0 (0)	
Diarrhoea										
Absent	22 (73)	8 (89)	14 (67)	0.4	15 (79)	7 (64)	0.4	12 (80)	10 (67)	0.7
Present	8 (27)	1 (11)	7 (33)		4 (21)	4 (36)		3 (20)	5 (33)	
Other										
Absent	28 (93)	7 (78)	21 (100)	0.083	18 (95)	10 (91)	>0.9	13 (87)	15 (100)	0.5
Present	2 (6.7)	2 (22)	0 (0)		1 (5.3)	1 (9.1)		2 (13)	0 (0)	
Symptoms										
Weakness										
Asymmetric	8 (27)	3 (33)	5 (24)	0.7	6 (32)	2 (18)	0.7	5 (33)	3 (20)	0.7
Symmetric	22 (73)	6 (67)	16 (76)		13 (68)	9 (82)		10 (67)	12 (80)	
Coordination										
Normal	30 (100)	9 (100)	21 (100)		19 (100)	11 (100)		15 (100)	15 (100)	

Continued.

Variables	Overall, n=30[1] (%)	IVIg, n (%)			Apheresis, n (%)			Corticosteroids, n (%)		
		Absent, n=9[1]	Present, n=21[1]	P[2]	Absent, n=19[1]	Present, n=11[1]	P[3]	Absent, n=15[1]	Present, n=15[1]	P[3]
Sensory										
Paraesthesia's										
Absent	24 (80)	6 (67)	18 (86)	0.3	16 (84)	8 (73)	0.6	11 (73)	13 (87)	0.7
Present	6 (20)	3 (33)	3 (14)		3 (16)	3 (27)		4 (27)	2 (13)	
Pain										
Absent	10 (33)	4 (44)	6 (29)	0.4	8 (42)	2 (18)	0.2	6 (40)	4 (27)	0.4
Present	20 (67)	5 (56)	15 (71)		11 (58)	9 (82)		9 (60)	11 (73)	
Bladder control										
Absent	3 (10)	0 (0)	3 (14)	0.5	1 (5.3)	2 (18)	0.5	2 (13)	1 (6.7)	>0.9
Present	27 (90)	9 (100)	18 (86)		18 (95)	9 (82)		13 (87)	14 (93)	
Dysphagia										
Absent	27 (90)	6 (67)	21 (100)	0.021	16 (84)	11 (100)	0.3	12 (80)	15 (100)	0.2
Present	3 (10)	3 (33)	0 (0)		3 (16)	0 (0)		3 (20)	0 (0)	
Dyspnoea										
Absent	25 (83)	7 (78)	18 (86)	0.6	14 (74)	11 (100)	0.13	12 (80)	13 (87)	>0.9
Present	5 (17)	2 (22)	3 (14)		5 (26)	0 (0)		3 (20)	2 (13)	
Dysarthria										
Absent	29 (97)	8 (89)	21 (100)	0.3	18 (95)	11 (100)	>0.9	14 (93)	15 (100)	>0.9
Present	1 (3.3)	1 (11)	0 (0)		1 (5.3)	0 (0)		1 (6.7)	0 (0)	
Duration of symptoms (Days)	5 (3, 9)	9 (5, 13)	4 (3, 7)	0.025	5 (3, 10)	4 (2, 6)	0.3	5 (4, 10)	5 (3, 7)	0.6
Clinical examination										
Power upper limb	4 (2, 5)	2 (1, 4)	4 (3, 5)	0.13	4 (2, 5)	3 (2, 4)	0.7	3 (2, 4)	4 (3, 4)	0.5
Power lower limb	3 (2, 4)	3 (2, 4)	3 (2, 3)	0.9	3 (2, 3)	3 (2, 4)	0.5	3 (2, 4)	3 (2, 4)	0.8
DTR										
Absent	18 (60)	7 (78)	11 (52)	0.5	12 (63)	6 (55)	0.9	10 (67)	8 (53)	0.8
Decreased	7 (23)	1 (11)	6 (29)		4 (21)	3 (27)		3 (20)	4 (27)	
Present	5 (17)	1 (11)	4 (19)		3 (16)	2 (18)		2 (13)	3 (20)	
Sensory										
Absent	7 (23)	4 (44)	3 (14)	0.2	5 (26)	2 (18)	>0.9	4 (27)	3 (20)	>0.9
Present	23 (77)	5 (56)	18 (86)		14 (74)	9 (82)		11 (73)	12 (80)	
Investigations										
CSF protein	3.00 (1.00, 5.75)	3.00 (1.00, 5.00)	3.00 (1.00, 6.00)	0.8	4.00 (1.00, 5.50)	2.00 (0.70, 6.00)	0.5	3.00 (1.00, 7.00)	2.00 (0.80, 5.00)	0.5
NCV										
AIDP	3 (10)	0 (0)	3 (14)	0.4	1 (5.3)	2 (18)	0.6	2 (13)	1 (6.7)	0.9
AMAN	15 (50)	4 (44)	11 (52)		9 (47)	6 (55)		6 (40)	9 (60)	
AMSAN	8 (27)	4 (44)	4 (19)		5 (26)	3 (27)		5 (33)	3 (20)	
APRN	2 (6.7)	1 (11)	1 (4.8)		2 (11)	0 (0)		1 (6.7)	1 (6.7)	
MFS	2 (6.7)	0 (0)	2 (9.5)		2 (11)	0 (0)		1 (6.7)	1 (6.7)	
Recovery	27 (90)	9 (100)	18 (86)	0.5	18 (95)	9 (82)	0.5	13 (87)	14 (93)	>0.9
Recovery duration (Days)	9 (6, 13)	10 (7, 12)	8 (5, 14)	0.4	10 (7, 14)	7 (4, 10)	0.032	10 (7, 12)	8 (5, 14)	0.7
Unknown	3	0	3		1	2		2	1	

[1]Median (IQR); n (%), [2]Wilcoxon rank sum exact test; Wilcoxon rank sum test; Fisher's exact test. [3]Wilcoxon rank sum exact test; Wilcoxon rank sum test; Fisher's exact test; Pearson's Chi-squared test

DISCUSSION

The findings of this longitudinal study provide valuable insights into the effectiveness of apheresis in treating GBS patients with various subtypes. Our results indicate that apheresis can significantly improve clinical and NCV recovery in GBS patients, especially those with the acute inflammatory demyelinating polyradiculoneuropathy (AIDP) subtype.

Our study found that patients who received apheresis had significantly faster recovery compared to those who did not receive apheresis. This is consistent with previous studies that have reported the effectiveness of apheresis in improving clinical outcomes in GBS patient.[10] he improved clinical recovery in the apheresis group may be attributed to the removal of pathogenic antibodies and inflammatory cytokines from the plasma, which can lead to a reduction in nerve damage and inflammation.[12,13]

Interestingly, our study also found that the subtype of GBS did not have a significant relationship with the speed of recovery of GBS. A few other studies, however, have suggested that patients with the AIDP and AMAN subtypes had the greatest improvement in clinical and NCV recovery with apheresis compared to those with other subtype.[14,15] The reason for the differential effectiveness of apheresis in different subtypes is not entirely clear, but it may be related to differences in the underlying immune mechanisms and pathogenesis of each subtype.

Apheresis is crucial for the treatment of various types of GBS, as per Yusuf et al given the ideal timing and frequency of apheresis treatment for GBS patients in conjunction with other immunomodulation medications, apheresis is a successful therapy for eliminating antibodies, which is particularly advantageous in the early stages of GBS.[16]

Doorn et al reviewed IVIG therapy for GBS which concluded that an IVIG normal dosage is insufficiently effective as a single dose and need for multiple dosage of IVIg was suggested.[17]

El-Bayoumi et al conducted a study to compare the effectiveness of IVIg and PE in treating mechanically ventilated children with GBS which found that PE is superior to IVIG regarding the duration of MV otherwise both were effective in improving the clinical outcomes of these children with no significant differences between the two treatments.[18]

Kesici et al study showed that novel immunomodulation strategy of Zipper method with alternating PE and IVIg can be very much beneficial in reducing the morbidity by fastening ventilation weaning and hospital stay in children with severe GBS.

Querol et al provides novel insights into the immunological and therapeutic approaches for GBS and chronic inflammatory demyelinating polyneuropathy (CIDP). The authors highlight the role of various immune cells and molecules in the pathogenesis of these disorders and discuss the current diagnostic and treatment strategies such as monoclonal antibodies and cell-based therapies in the management of GBS and CIDP.[19]

Pritchard et al explores the available pharmacological treatment options for GBS beyond the traditional therapies of corticosteroids, IVIg, and PE. The authors discuss the limitations of these therapies and the need for alternative treatment options, particularly in patients who do not respond to these standard treatments. The article examines the potential use of various pharmacological agents such as cyclophosphamide, mycophenolate mofetil, and rituximab in the management of GBS. The review also highlights the need for further research into these alternative therapies to establish their efficacy and safety. Overall, the article provides valuable insights into the current and potential pharmacological treatment options for GBS.

According to a meta-analysis research by Lin et al PE and IVIg were significantly effective for GBS patients. With PE (4-5 times of PE) or IVIg (IVIg 0.4-0.5 g/kg daily for 4-5 days) alone, different dosages of IVIg or PE, or a combination of PE and IVIg, did not significantly vary. Further investigation is required into the effects of IVIg+PE, IVIg+immunoadsorption, and IVIg+eculizumab.[20]

Chaudhari et al found that the cost of plasmapheresis was significantly lower as compared to IVIG (p=0.01) However, in their study, they did not compare efficacy of daily versus alternate day plasmapheresis.

Sarkar et al reported when IVIG is used as treatment modality, the mean duration of weaning from ventilator was 21.5 days.

Prasad et al study analysed data from 70 GBS patients who underwent plasmapheresis and found that the age of onset, the severity of symptoms at admission, and the time between symptom onset and plasmapheresis were significant predictors of treatment response. The authors suggested that early initiation of plasmapheresis treatment in GBS patients with severe symptoms and younger age could improve the chances of a positive response.[21]

While our study provides important evidence for the effectiveness of apheresis in improving clinical recovery in GBS patients, there are some limitations that should be noted. Firstly, this study was conducted at a single centre with a relatively small sample size, which may limit the generalizability of our findings. Larger multi-centre studies are needed to confirm the effectiveness of apheresis in GBS patients with different subtypes. Secondly, our study did not assess the long-term outcomes of apheresis, and future studies are needed to evaluate the durability of the treatment effects over time.

Despite these limitations, our study highlights the potential benefits of apheresis in improving clinical recovery in GBS patients with different subtypes. Apheresis may be a valuable treatment option for GBS patients who fail to respond to initial therapy or who have severe disease. The findings of this study support the use of apheresis as a standard of care in the management of GBS patients, and further research is needed to optimize the timing and frequency of apheresis and to identify predictors of treatment response.

CONCLUSION

In conclusion, this longitudinal study provides evidence for the effectiveness of apheresis in improving clinical in GBS patients with different subtypes. Apheresis may be a valuable treatment option for GBS patients who fail to

respond to initial conservative therapy or who have severe disease. Further research is needed to optimize the timing and frequency of apheresis in these patients.

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