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Research Article

A Comparison of Clinical Profile and Short-Term Outcome of Demyelinating and Axonal Subtypes of Guillain-Barré Syndrome in Children

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Abstract

Introduction: Studies comparing the Demyelinating GBS (Dmy-GBS) and axonal GBS (Ax-GBS) subtype in children are lacking.

Methods: In this hospital based, prospective and observational study, consecutive children with GBS were studied to compare the clinical profile and outcome among the subtypes.

Results: Among 9847 children admitted to the emergency, 95 had acute flaccid paralysis,57 of whom had GBS. Electrophysiologic studies were completed in 56, of whom 20 each had Dmy-GBS and Ax-GBS (19 motor axonal), 12 had non-reactive nerves, and 5 unclassifiable findings. Mean age of onset in Dmy-GBS was 55 months while Ax-GBS occurred later at 84 months. More children in Ax-GBS group had preceding gastroenteritis (4 vs 2), while Dmy-GBS had upper respiratory infections (12 vs 7). Mean time from onset of symptoms to hospital admission was more in Dmy-GBS 18 days to 8 days in Ax-GBS. Ataxia was only seen in Dmy-GBS while wrist drop, foot drop and hyperreflexia were seen only with Ax-GBS. Asymmetry of motor findings was more likely in Ax-GBS (10 vs 4 P = 0.048). Respiratory muscle involvement (6 vs 3) and artificial ventilation (5 vs 2) was more in Ax-GBS. The average duration of hospital stay was more in Ax-GBS 16 days to 11 days in Dmy-GBS. Children with Ax-GBS less likely to be non ambulant at discharge (12 vs 6, p = 0.036). Mean disability scores at hospital discharge (4.9 ± 1.2 vs 4 ± 0.9, p = 0.015) and at last follow up (0.7 ± 1.01 vs 0.05 ± 0.2, p = 0.016) were higher in Ax-GBS. Children with Dmy-GBS were more likely to achieve normalcy on follow up (19 vs 12, p = 0.023). IVIg was the treatment modality and was tolerated well with no side effects reported with no relapse of symptoms after treatment.

Conclusion: Axonal and demyelinating subtypes of GBS are equally common in children of North India. Children with axonal GBS have severe clinical course and more short term morbidity and slower recovery.

Keywords: *Acute Inflammatory Demyelinating Polyneuropathy; Acute Motor Axonal Neuropathy; Clinical Features; Guillain-Barré Syndrome; Neurophysiology*

Introduction

Guillain-Barré syndrome (GBS) is an acute onset, usually monophasic immune-mediated disorder of the peripheral nervous system. The term GBS is often considered to be synonymous with acute inflammatory demyelinating polyradiculoneuropathy (AIDP), but with the increasing recognition of variants over the past few decades, the number of diseases that fall under the rubric GBS have grown to include axonal variants and more restricted variants, such as Miller Fisher syndrome (MFS) [1]. During the past 15 years, neurophysiologic, pathologic and immunologic observations have shown that GBS is divided into the two major subtypes: acute inflammatory demyelinating polyneuropathy (AIDP) and acute motor axonal neuropathy (AMAN) [2-4]. However different subtypes of GBS have different incidence

rates in different parts of the world. In Europe and North America AIDP is dominant contributing to 90% of the cases. In contrast in China and Japan AMAN being the most common subtype [5]. In Indian series the incidence of AIDP and AMAN are virtually equal although AMAN is more common in younger patients [6]. In western countries, GBS is common in the 5th decade [7] but in India it occurs more commonly at a younger age [8,9]. GBS is equally common in men and women and can occur at any age.

Although the pathogenesis of GBS remains unclear, there are increasing indications that it is an autoimmune disease, often triggered by a preceding infection [9]. AMAN is a pure motor disorder and is frequently associated with serum antibodies against gangliosides GM1, GM1b, GD1a, or GalNAc-GD1a and antecedent *Campylobacter jejuni* enteritis. Another axonal subtype of GBS is acute motor and sensory axonal neuropathy (AMSAN), in which neurophysiologic and pathologic findings indicate axonal degeneration of both motor and sensory nerves. Each subtype of GBS presumably has an independent immunopathogenesis and constitutes subgroups with similar but somewhat different clinical features.

It usually requires hospitalization for early detection and treatment [10,11]. The reported incidence rates range from 0.6 to 4.0/100,000 population [11,14]. Despite effective therapies, such as IV immunoglobulins (IVIg) and plasma exchange (PE), acute mortality remains relatively high and about 20% of hospitalized patients may have a long-term disability [12].

GBS is a severe disease for which optimization of treatment is clearly needed. The clinical severity should be lessened, the plateau phase shortened and complications minimized. Current treatment for GBS hastens recovery, but still 20% needs artificial ventilation and outcome after 6 or 12 months, however, has not or only marginally improved. Currently, IVIg has replaced PE as the preferred treatment in many centers mainly because of its greater convenience and availability. The combination of PE followed by IVIg was not significantly better than PE or IVIg alone.

There is paucity of data regarding clinical phenotype, electrophysiological feature and response to treatment between different types of GBS in children from this part of the world so our study was an attempt to compare the clinical profile and short term outcome of children with axonal and demyelinating subtypes of Guillain Barre syndrome.

Methodology

We conducted prospective, cross-sectional, observational study at a tertiary care centre over 18 mo from Nov 2013 through April 2015. We included all children between 6 months to 12 years with clinical suspicion of Guillain Barre syndrome or any of its variants, and presentation within 1 month of the onset of illness. Children with hemodynamic instability; or those with a pacemaker, vagus nerve stimulator or any other device interfering with electrophysiological testing were excluded. The clinical details and demographic profile were recorded into an Epi Info 2003 database software (Center for Disease Control and Prevention, Atlanta, GA, USA). Nerve conduction studies were performed using the conventional procedures on the Synergy two-channel model electromyography system. Motor nerve conduction studies (NCS) were performed on the median, ulnar, common peroneal and posterior tibial nerves unilaterally. Sensory NCS included stimulation of median nerve (orthodromic) and sural nerve (antidromic). F responses were studied in median and posterior tibial nerves. The H reflex of the posterior tibial nerve was studied from the gastrocnemius muscle. Varying on the tolerance and stability, not all patients could undergo all the testing. Skin temperature was maintained at > 32°C. The nerve conduction values were defined as abnormal if they were beyond 2.5 standard deviations of the pediatric standard data [13]. Children were classified into different subtypes based on electro diagnostic criteria modified from Hughes., *et al.* [14,15] (Table 1).

Children with an alternative diagnosis were excluded. The degree of disability was assessed on admission and thereafter till discharge, to determine the peak disability. Time taken to regain independent walking was assessed in non-ambulant children. Functional status was recorded at admission, during hospital stay, and thereafter till discharge. The disability grading score suitable for GBS used was: 0- normal, 1- slight gait problem, 2 -moderate gait problem, 3- walks holding furniture, 4-walks holding person, 5-not able to walk, 6- not able to

sit upright, 7 tetraparesis, 8- tetraplegia, 9-ventilated and 10-death [16]. The study was approved by the Institutional Ethics Committee. An informed consent was obtained from the parents or the guardians.

AIDP	At least one of the following in each of at least two nerves, or at least two of the following in one nerve if all others inexcitable and dCMAP ≥ 10% LLN: 1. Motor CV • < 90% LLN • < 85% LLN if CMAP amplitude < 50%LLN 2. Distal motor latency (DL) • > 110% of ULN • > 120% of ULN if CMAP amplitude < 100% of LLN 3. Absent F waves or prolonged minimum F wave latency • 120% of ULN 4. Conduction block • < 0.5 Prox-dist amp ratio and dCMAP>20%LLN
AMAN	1. None of the above features of demyelination in any nerve (except one demyelinating feature allowed in one nerve if dCMAP <10% LLN) 2. dCMAP <80% LLN in at least two nerves
AMSAN	1. No evidence of demyelination, 2. distal CMAP amplitude <80% of lower limit of normal (as in AMAN) 3. and 4. Reduction of sensory nerve action potential amplitude (SNAP) < 50% of lower limits of normal in at least two nerves
In excitable	dCMAP absent in all nerves (or present in only one nerve with d CMAP < 10% LLN)
Unclassifiable	Does not exactly fit criteria for any other group

Table 1: *Diagnostic criteria for GBS subtypes.*

AIDP: Acute Inflammatory Demyelinating Polyneuropathy; AMAN: Acute Motor Axonal Neuropathy; AMSAN: Acute Motor Sensory Axonal Neuropathy; dCMAP: Compound Muscle Action Potential Amplitude After Distal Stimulation; LLN: Lower Limit of Normal; pCMAP: Compound Muscle Action Potential Amplitude After Proximal Stimulation; ULN: Upper Limit of Normal.

The purposive sampling method of non-probability sampling was used. All consecutive children reporting to authors' centre were screened for inclusion. Distribution of the continuous variables was ascertained using Bartlett's test. The Mann- Whitney U test, Unpaired t-test and Fischer's exact test were used when appropriate. P value of ≤ 0.05 was considered significant.

Results

Demography

During the study period of 18 months 9847 children were admitted in pediatric emergency. AFP reporting of 95 children was done from the hospital. 60 children were enrolled in our study and 3 were excluded because of alternative diagnosis. The total numbers of children enrolled and analyzed were 57. There were 46 boys, with a male: female ratio of 4.1:1. The median age of the children was 52 mo (IQR 32 - 164.5). Forty-one (72%) children were admitted during the winter months of October-March (Figure 1).

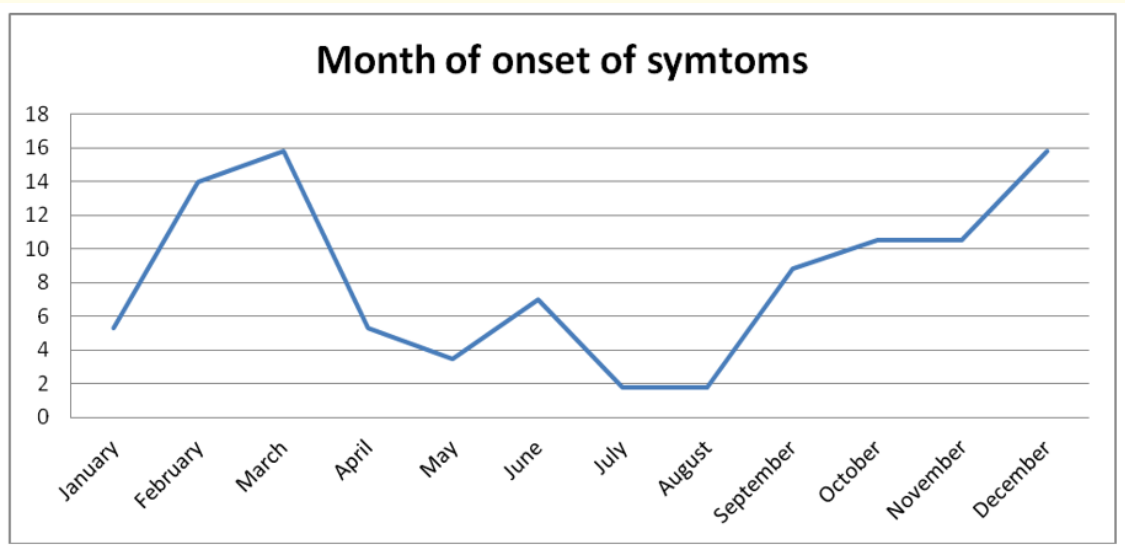

Figure 1: *Month of onset of symptoms of the study population.*

Nerve conduction study was done in 57 patients. Subtype classification, based on Hughes criteria, revealed AIDP in 20 children (35%); AMAN in 19 children (33%); twelve children (21%) had in excitable nerves; one child had AMSAN; and five remained unclassifiable (Figure 2).

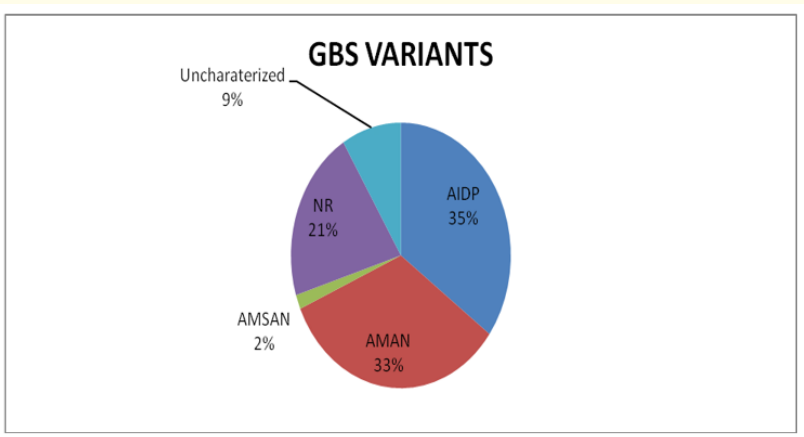

Figure 2: *Types of GBS variants in the studied children.*

Clinical profile

The mean time from onset of symptoms to hospital admission was 8days IQR 2 to 10 days. The most common presenting symptoms was lower limb weakness, present in 55 out of 57 (96.5%). Other symptoms at presentation were upper limb weakness in (56%), myalgias (49%), difficulty swallowing (33%), facial weakness (11%) and unsteadiness (7%). Thirty five percent had difficulty in breathing at admission (Table 2).

Variable	Total (n=57)	AIDP (n=20)	AMAN (n=19)
Male	46	14 (70%)	17 (89%)
Age at onset (months)	68 ± 40	55 ± 33	84 ± 42
Days from Onset to admission	8 ± 7	18 ± 6	7.5 ± 7
Antecedent Episode URTI Diarrhoea	27, 8	12 (60%) 2 (10%)	7 (37%) 4 (21%)
Upper Limb Weakness	35	10 (50%)	11(58%)
Lower Limb Weakness	53	19(95%)	18 (94%)
Facial Weakness	21	7 (35%)	5 (26%)
Neck Weakness	13	6 (30%)	4 (21%)
Bulbar Weakness	20	7 (35%)	6 (32%)
Poor swallow	19	7 (35%)	7 (37%)
Intercostal Weakness	19	3 (15%)	5 (26%)
Diaphragm Weakness	18	1 (5%)	6 (32%)
Truncal Weakness	32	9 (45%)	11 (58%)
Asymmetric Weakness	17	4 (20%)	10 (53%)
Cranial Nerve Deficit	6	2 (10%)	1 (5%)
Areflexia	53	19 (95%)	17 (89%)
Hyperreflexia	2	0	2 (10%)
Meningeal Signs	32	10 (50%)	10 (52%)
Ataxia	2	2 (10%)	0
Clawing	3	0	2 (10%)
Mechanical Ventilation	19	2 (10%)	6 (32%)
Ventilation Duration in days	43 ± 30	32 ± 31	30 ± 35
Dysautonomia	22	6 (30%)	5 (26%)
Mean CSF protein	97 ± 74	103 ± 72	106 ± 92
IVIg Therapy	49 (86%)	17 (85%)	15 (79%)

Table 2: *Clinical features of GBS and comparison with demyelinating and axonal variants.*

Forty seven (82.4%) children had antecedent illness: Upper respiratory tract infection in 27 (47.4%), non localizing fever in 11 (19.3%), diarrhea was present in 8 (15.8%) and only 1 had dysentery. Only one child developed GBS after vaccination. The most common examination finding was paraparesis, it was found in 53 (93%). asymmetrical weakness was seen in 17%. Respiratory compromise were present in 19 (33.3%), requiring ventilation. Children with non reactive nerves or uncharacterized NCV findings had the maximum duration of ventilation. Autonomic dysfunction was recorded in 22 (38.6%) cases predominantly affecting BP and abnormal sweating.

The average duration of symptoms prior to admission in AIDP group was 8.1 ± 5.9 days while in AMAN group 7.47 ± 8 days. Both group had lower limb weakness as predominant symptoms 95%in AIDP and 94% in AMAN group. The average duration of hospital stay was 11.3 ± 12.3 days in AIDP group while in AMAN group had average hospital stay of 15.8 ± 22 days. Ten percent children in AIDP required ventilation while 26% AMAN required ventilation. The average duration of ventilation was 32 ± 31days in AIDP and in AMAN 30.2 ± 34.7 days (Table 2).

Outcome

The average duration of hospital stay was 23 ± 29 days with IQR between of 6.5 to 27.5 days. Majority of patients in Non reactive (35.5 ± 25.5) and uncharacterized (53 ± 57) variants had longer hospital stay. At discharge 25 (45%) could walk with or without support. None of the patients were normal at discharge. The mean duration of follow up was 11.4 ± 8 months median of 9 months and interquartile range of 5 to 14 months. At last follow-up, 41 (72%) of children were symptom free normal gait and able to run. Most of the children became normal within 1 month of discharge. A total of 4 children of GBS died in our study group one child died during the height of illness and 1 died due to withdrawal of life support as the parents were not willing for treatment. Two children died during follow up.

At discharge majority of AIDP 70% were able to walk with support. In AMAN group 36.8% was not able to walk 15.8% could not sit upright and 2 children could not lift their leg from the bed. A total of 53 children were followed up after discharge on OPD basis. The mean duration of follow up was 11.4 ± 8 months median of 9 months and interquartile range of 5 to 14 months.

The mean duration of follow-up in AIDP was 14 mo while in AMAN was 10.5 mo. At last follow-up, 19 of the 20 AIDP children regained the ability to walk and run independently while 12 in AMAN group were able to walk. The mean disability scores at last follow-up was significantly higher in the AMAN group (0.7 ± 1.01 vs. 0.05 ± 0.2, p = 0.016). Children with AIDP were more likely to achieve normalcy on follow-up (19 vs. 12, p = 0.02) (Table 3).

Variables	AIDP n=20	AMAN n=19
Duration Of Hospital Stay	11 ± 12	16 ± 22
Mean Duration of Follow-Up (months)	14 ± 9	10.5 ± 7.5
Ambulatory at discharge	14 (70%)	7 (37%)
Gait Abnormality at follow up	1 (5%)	7 (37%)
Peak Disability	5.75 ± 1.8	6.3 ± 1.9
Mean Disability score*	4.9 ± 1.2	4 ± 0.9
Mean Disability score Follow-up**	0.7 ± 1	0.05 ± 0.2

Table 3: *Comparison of Outcomes of AIDP and AMAN.*
**P = 0.015, **P = 0.016 other comparison are not statistically significant.*

Discussion

GBS is an acute, monophasic, symmetrically progressive, peripheral ascending demyelinating polyneuropathy characterized by rapidly evolving symmetrical limb weakness, areflexia, absent or mild sensory signs, and variable autonomic disturbances**.** Although the occurrence of GBS in children is relatively rare, it is the most common cause of acute flaccid paralysis in infants and children during the post-polio eradication era.

In the current study it was found that, the pure motor form of GBS (54%) is the most frequent type of GBS in children. Of the electrophysiological subtypes, AIDP (35%) and AMAN (33%) were of near equal frequency. There has been conflicting reports on the dominant

subtype of GBS in children in India. The study from North India, revealed that 38 of the total 65 children (58%) had pure motor involvement [17]. In a study by Kalita., *et al.* AIDP was the predominant subtype in a study of 140 children with GBS the authors reported that 95 of the 140 children had AIDP (68%) and 33 had AMAN (24%), respectively [18]. However, AMAN emerged as the most common subtype of childhood GBS in two previously reported prospective study from North India [17,19]. The exact cause for the difference in subtypes are still not clear. The heterogeneity of the antecedents and the indigenous risk factors may play a role in this, but it is still speculative. However possible reasons could be due to regional variations in the predominant subtypes of different part of the world. Examples include the predominant subtype of AMAN in Mexico [20] and AIDP in the neighboring El Salvador [21]; AMAN in major parts of China and AIDP in Changchun province of China [22].

In our study our aim was to study the clinical profile and electrophysiological profile of GBS and compare clinical profile and short term outcome of the different variants of GBS.

In our study the mean age of onset was 68 months with majority being in age group 3-9 years with male preponderance. It was consistent with the study of Koul., *et al.* and Dhadke., *et al.* and Nachamkin., *et al.* It was believed to be due to exposure to several infection, toxins and increased susceptibility of young myelin to demyelination. In our study majority of cases had occurrence of disease during winter months (Oct-March) which was consistent with the study done by Korinthenberg., *et al.* [16] and Sankhyan., *et al* [17]. In study from North India there were two peak of presentation -rainy season and winter season. AIDP occurred in winter peak whereas AMAN was present in both peaks. AIDP and AMAN occurred year round in Mexico city. In our study there was association of antecedent risk factors of URTI with AIDP while Acute gastroenteritis was associated with AMAN. It was consistent with findings from North India, Mexico, Japan and China [20-22].

The clinical profile of 2 major subtypes was generally comparable and mean duration of symptoms to hospital admission were similar too, though we found Ataxia only with patients with AIDP. The occurrence of ataxia in AIDP is possibly secondary to involvement of sensory limb of the reflex arc. This also manifests with loss of position sense in some of the patients. Since AMAN affects only motor nerves it explains the lack of ataxia in children with AMAN. We also noticed that respiratory muscle involvement is nearly twice as common in AMAN as AIDP though no statistical significant difference was found. This also led to a severer peak disability in AMAN group as compared to AIDP. A striking difference in severity of subtypes was reported in a Japanese study, 27% of AMAN children required assisted ventilation compared to none in AIDP group [23]. Similar results were observed in a study from Mexico, 19.6% of AMAN children required respiratory support as compared to 12% with AIDP [20]. In a Chinese study, 91% AMAN were found to be severe than 75% in AIDP [22]. It appears that AMAN in childhood is a more severe GBS subtype than AIDP. In contrast to these studies, a study from Northern India reported higher incidence of mechanical ventilation in AIDP (13%) compared to 3.7% in AMAN group [17] possibly, due to milder cohort of AMAN patients encountered by the authors.

Involvement of cranial nerves was more common in AIDP than in AMAN (35% to 26%). This may be clinically relevant thought not statistically significant. In keeping with this finding, in a study from North India, 67% of children with AIDP and 44% with AMAN group had facial weakness [17]. A much more remarkable difference was reported in a study from Japan in which 91% in AIDP to 53% had cranial nerve deficits ($p = 0.04$) [23]. Interestingly we observed asymmetry of motor findings in nearly half of patients of AMAN compared to one fifth of patient in AIDP ($p = 0.048$). Certain signs were exclusively seen with AMAN like wrist drop, foot drop and hyperreflexia.

In contrast to AIDP, children with AMAN have been described to have a more rapid progression and higher frequency of ventilator dependence with less frequent cranial nerve and autonomic involvement than AIDP. The pattern of recovery in AMAN is also distinctive. While the speed of recovery in AIDP is relatively uniform, both rapid and slow patterns have been noted in AMAN [24]. The two children with hyperreflexia did not have other bipyramidal signs to merit neuroimaging.

Outcome

It was found in our study that peak disability was higher in AMAN group than AIDP group though not statistically significant, it appears to be clinically relevant. It was seen that AMAN children tend to recover slowly than AIDP. There was statistically significant difference in mean discharge disability score in AMAN group compared to AIDP (p = 0.025). At follow up the current disability was higher in AMAN than AIDP and it was statistically significant too (p-0.031). In our study, the proportion of children ambulant at discharge and normal at follow up was significantly higher in the AIDP group compared to AMAN group. A retrospective multicentre study on the natural history of childhood GBS from Europe recounted complete recovery in 92% of children by six months [24]. A prospective study from India on long term outcome of GBS at 12 mo in 40 children, by Kalra., *et al.* described full recovery in 70% of children [19]. However, both the studies were not aimed at accurate electrophysiological subtype classification of childhood GBS. A 12-month follow-up study of 26 children by Nagasawa., *et al.* from Japan revealed complete recovery in 100% of the AIDP group and 86% of AMAN respectively [23]. The present results on follow-up are congruent with Nagasawa., *et al.* with complete recovery in 95% AIDP and 60% AMAN.

Conclusion

In conclusion, this study revealed that axonal and demyelinating subtypes of GBS are equally common in children of north India. AIDP are more likely to have preceding URTI, symmetrical weakness, cranial nerve palsy and ataxia whereas AMAN has more association with preceding gastroenteritis, asymmetric limb weakness, foot drop or wrist drop. Children with AMAN are more likely to have respiratory muscle weakness hence more likely to require assisted ventilation. Many of the above difference were not statistically significant because of less number of patients in each group. It was found that more severe form of GBS was AMAN, manifesting as higher discharge disability score and also slower recovery.

Limitation of the Study

The present study had certain limitations. Firstly, serial electrophysiological evaluations could have better delineated the various subtypes of GBS. Changes in subtype classification from AIDP and equivocal groups to AMAN variant on repeat assessment has been observed in studies using serial electrophysiological assessments. Serial assessment was done in very few patients in present cohort. On the other hand, a recent study has suggested that with new criteria, a repeat NCS study may not yield any additional yield. The present study was not adequately powered to detect the individual differences between the two subtypes. A larger sample and longer follow-up would have better defined the true impact of subtypes of GBS on functional outcome.

Conflict of Interest

None.

Source of Funding

None.

Bibliography

1. Levin KH. "Variants and mimics of Guillain-Barré syndrome". *Neurology* 10.2 (2004): 61-74.
2. Griffin JW., *et al.* "Pathology of the motor-sensory axonal Guillain-Barré syndrome". *Annals of Neurology* 39.1 (1996): 17-28.
3. Kuwabara S. "Guillain-Barré syndrome. Epidemiology, pathophysiology and management". *Drugs* 64.6 (2004): 597-561.
4. Hughes RA and Corblath DR. "Guillain-Barré syndrome". *Lancet* 366 (2005): 1653-1666.

5. McKhann GM., *et al.* "Acute motor axonal neuropathy. A frequent cause of acute flaccid paralysis in China". *Annals of Neurology* 33.4 (1993): 333-342.

6. Ogawara K., *et al.* "Axonal Guillain-Barré syndrome. Relation to antiganglioside antibodies and Campylobacter jejuni infection in Japan". *Annals of Neurology* 48.4 (2000): 624-631.

7. Kaur U., *et al.* "Guillain-Barré syndrome - a clinical electrophysiological and biochemical study". *Acta Neurologica Scandinavica* 73.4 (1986): 394-402.

8. Gupta SK., *et al.* "Acute idiopathic axonal neuropathy (AIAN) - a clinical and electrophysiology observation". *Acta Neurologica Scandinavica* 89.3 (1994): 220-224.

9. Hughes RAC and Rees JH. "Clinical and epidemiologic features of Guillain- Barré syndrome". *The Journal of Infectious Diseases* 176.2 (1997): S92-S98.

10. Jiang G-X., *et al.* "Guillain-Barré syndrome in south-west Stockholm, 1973-1991, 1. Quality of registered hospital diagnoses and incidence". *Acta Neurologica Scandinavica* 91.2 (1995): 109-117.

11. Koobatian TJ., *et al.* "The use of hospital discharge data for public health surveillance of Guillain-Barré syndrome". *Annals of Neurology* 30.4 (1991): 618-621.

12. The Italian Guillain-Barré syndrome Study Group. "The prognosis and main prognostic indicators of Guillain-Barré syndrome: a multicenter prospective study of 297 patients". *Brain* 119 (1996): 2053-2061.

13. Darras BT., *et al.* "Neuromuscular disorders of infancy, childhood, and adolescence: a clinician's approach". *Indian Journal of Pediatrics* (2015).

14. Van der Meché FG., *et al.* "FG GBS-consensus group of the Dutch Neuromuscular Research Support Centre. Diagnostic and classification criteria for the Guillain-Barré syndrome". *European Neurology* 45.3 (2001): 133-139.

15. Hughes RAC and Cornblath DR. "Guillain-Barré syndrome". *Lancet* 366.9497 (2005): 1653-1666.

16. Korinthenberg R and Mönting JS. "Natural history and treatment effects in Guillain-Barré syndrome: a multicentre study". *Archives of Disease in Childhood* 74.4 (1996): 281-287.

17. Sankhyan N., *et al.* "Childhood Guillain- Barré syndrome subtypes in northern India". *Journal of Clinical Neuroscience* 21.3 (2014): 427-430.

18. Kalita J., *et al.* "Prospective comparison of acute motor axonal neuropathy and acute inflammatory demyelinating polyradiculoneuropathy in 140 children with Guillain-Barré syndrome in India". *Muscle Nerve* 57.5 (2018): 761-765.

19. Kalra V., *et al.* "Outcome in childhood Guillain-Barré syndrome". *Indian Journal of Pediatrics* 76.8 (2009): 795-799.

20. Nachamkin I., *et al.* "Patterns of Guillain-Barré syndrome in children: results from a Mexican population". *Neurology* 69.17 (2007): 1665-1671.

21. Ramírez-Zamora M., *et al.* "Guillain-Barré syndrome in the paediatric age: epidemiological, clinical and therapeutic profile in a hospital in El Salvador". *Revista de Neurología* 48.6 (2009): 292-296.

22. Ye Y-Q., *et al.* "Clinical and electrophysiologic features of childhood Guillain-Barré syndrome in Northeas China". *Journal of the Formosan Medical Association* 113.9 (2014): 634-639.

23. Nagasawa K., *et al.* "Electrophysiological subtypes and prognosis of childhood Guillain-Barré syndrome in Japan". *Muscle Nerve* 33.6 (2006): 766-770.

24. Kuwabara S and Yuki N. "Axonal Guillain-Barré syndrome: concepts and controversies". *The Lancet Neurology* 12.12 (2013): 1180-1188.

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