www.jcimcr.org

JCIMCR OPEN ACCESS Journal of Clinical Images and Medical Case Reports

ISSN 2766-7820

Review Article

Open Access, Volume 4

Benefit of inpatient rehabilitation care in management of acute demyelinating syndrome in children

Anand Prakash Datta[1]*; Richa Chaudhary[2]

[1]NMC, DMIMS (DU), India.

[2]Department of Children, JNMC, DMIMS (DU), India.

***Corresponding Author: Anand Prakash Datta**
JNMC, DMIMS (DU), India.
Tel: 8825104787; Email: apd.97doc@gmail.com

Received: Mar 09, 2023
Accepted: Mar 23, 2023
Published: Mar 30, 2023
Archived: www.jcimcr.org

DOI: www.doi.org/10.52768/2766-7820/2349

Abstract

Background: Guillian Barre Syndrome (GBS) is the most common cause of minor flaccid paralysis in the world and post-polio eradication is the most common cause on the Indian subcontinent as well. It affects 0.6-4 people per 1 lakh people per year. Guillian-Barre syndrome should be distinguished by a variation associated with inflammation of the cortical area of brain of chronic inflammatory polyneuropathy predicting prognosis and clinical course. Children have a better prognosis than adults and usually recover after a different period. However, in the Pressing event of the disease, the damage can be severe and can lead to long-term residual disability which can hamper quality of life especially in young population. In such scenarios, use of early rehabilitation may prove to be of good effect. This article gives an effective over view of the effect of the physical therapy in improving quality of life. The objective of this article was to perform a summary of the current Knowledge-base on outcome and its determinants in using early rehabilitation programme as a part of GBS management.

Methods: Relevant Prospective literature was reviewed through a PUB med Search of English-language articles published up to June 2022 using advanced database with keywords mentioning “gbs”, “acute Demyelinating neuropathies”, ”rehabilitation”, ”physical therapy”, paediatric population” using AND OR as needed.

Conclusion: Information regarding various modalities adopted by various hospitals in acute and low setting environment in regards to physical therapy and occupational therapy gives us clear insight into benefit of early rehabilitation of children with GBS will serve as a guide for your treating pediatrician to plan a treatment plan and will also help them explain their predictions to parents.

Keywords: GBS; Demyelinating disease; Inpatient care; Rehabilitation; Physical therapy.

Introduction

Guillaine Barré Syndrome (GBS) is an acute, usually Post infectious neuropathy of common occurrence with a yearly incidence rate between 1.1 and 1.8 per 100,000 [1]. More recent data from a meta-analysis of 13 epidemiological studies from Europe and North America refined the estimated yearly crude incidence as lying between 0.81 and 1.89 per 100,000 [2]. GBS incidence increases exponentially with age, with age-specific rates increasing from 0.62 per 100,000 among 0e9-year-olds to 2.66 per 100 000 among 80e89-year-olds. Male subjects are mmor commonly affected with an RR of 1.78 [2]. The most Common preceding infection causing GBS has been shown to be Campylobacter jejuni enteritis. Other Incriminated infectious agents include cytomegalovirus, Epsteine Barr virus, Mycoplasma pneumonia and Haemophilus influenza [3]. In its typical form, GBS causes rapidly progressive diffuse proximal and distal weakness of the four limbs, sensory loss and symptoms and are flexia. By definition, the maximal weakness is reached within 4 weeks. However, in the majority of cases, nadir is attained within 2 weeks. Facial, bulbar and respiratory muscle Weakness is frequent, and autonomic involvement well described [3].

It is recognized as a heterogeneous syndrome with several variants, most common type being acute inflammatory demyelinating polyradiculoneuropathy [4]. A cute inflammatory polyneuropathy variants (≤4 Weeks progressive phase). Guillain–Barré Syndrome (GBS); Acute Inflammatory Demyelinating Polyradiculoneuropathy (AIDP); Acute Motor Axonal Neuropathy (AMAN); Acute Motor And Sensory Axonal Neuropathy (AMSAN); Miller Fisher syndrome (cranial nerve involvement) Miller Fisher/GBS overlap syndrome Acute sensory demyelinating neuropathy, Acute pandysautonomia [5]. Guillain Barre Syndrome is immune based –Often acute fulminant demyelinating inflammatory poly neuropathy. Sensitisation of T Lymphocytes to protein in the myelin sheath Is necessary for disease induction [6]. Patchy Areas of demyelination occur along peripheral Nerves, nerve roots and myelin sheaths as a Result of lymphocytic infiltration, causing Impaired conduction of action potential Leading to slow conduction velocity and conduction blocks [7]. In axonal neuropathies, the conduction velocity is normal, but the number of functional motor units is decreased [4]. Cerebrospinal Fluid (CSF) protein Levels are elevated in the second week of Illness. Within 2–3 weeks of the demyelination process, the inflammation resolves and re-myelination commences. Existing body of evidence has emerged that the disorder is mainly a humorally-mediated, rather than T-cell-mediated disorder, at least in the progressive Phase of nerve injury. The extent to which T cells might be involved in the induction phase of the disease, during which the immune response is generated, remains Uncertain, and continues to be explored in new models [8]. Acute motor axonal neuropathy is thought of as an antibody-mediated attack on the nerve axolemma driven by molecular mimicry between microbial and axolemmal surface molecules [9]. The molecular mimics are glycans (ie, sugars) expressed on Lipo Oligo Saccharides (LOS) of preceding infectious organisms, notably C jejuni, that are capable of inducing antibody responses to these carbohydrate antigens [10]. Anti-carbohydrate antibody responses are believed to be largely independent of T cells. Anti-LOS antibodies can then bind to structurally identical glycans present on nerve gangliosides. Anti-ganglioside Antibodies in acute motor axonal neuropathy are complement-fixing, of IgG1 and IgG3 subclass, and mainly bind to GM1 and GD1a gangliosides [11]. In animal models, they induce axonal injury by fixing complement, recruiting macrophages, and depositing membrane attack complex in the axolemmal membrane [12]. This immunological cascade disrupts the anatomical and physiological integrity of exposed nerve membranes in nerve terminals and nodes of ranvier, causing a nerve conduction blockade that is either reversible or, in severe cases, results in severe, widespread axonal degeneration with poor recovery. A similar model is proposed for Miller Fisher syndrome associated with anti-GQ1b antibodies [13]. In which GQ1b ganglioside is the antigenic target, and is disproportionately enriched in the motor nerves that innervate extra ocular muscles [14].

Understanding immunological cascade involved in acute inflammatory demyelinating polyneuropathy is less known owing to 2 main reasons, first being in a wide array of immunological stimulant and secondly, failure to characterize specific antibody markers.

High proportion of antibodies against moesin, a component of the ezrin–radixin–moesin cytoplasmic complex in schwann-cell microvilli that surround the nodal axolemma, have been reported in cases of acute inflammatory demyelinating poly neuropathy triggered by CMV infection, although this result has not been Replicated [15]. Nerve glycolipids expressed in glial membranes, including myelin, are prime candidates as important antigens in acute inflammatory demyelinating polyneuropathy [16]. Antibodies against the glycolipid LM1, sulphoglucuronosyl paragloboside, galactocerebroside, and sulfatide are found in a small proportion of patients with acute inflammatory demyelinating polyneuropath [17]. In addition to being present in axonal membranes, some gangliosides (including GM1 and GQ1b) are expressed in glial membranes at the node of ranvier, where they Might mediate paranodal demyelination that causes the pathophysiological features of acute inflammatory demyelinating polyneuropathy [18].

As noted previously, the nodal area is richly decorated with potential antigens, including proteins and glycolipids, and is functionally very sensitive to pathological perturbations induced by antibody deposits, complement activation, and macrophage recruitment. Nodal conduction block, of glial or axonal origin, can arise quickly, but functionality can be restored in equally short time periods through local repair of injured membranes. Conversely, complete axonal transaction (which is always followed by wallerian degeneration of the distal stump), especially if proximally located in the nerve roots at a long distance from the innervation target, will be a permanent irreparable injury because regeneration cannot effectively occur over long distances. Although these considerations have clinical relevance, prediction of how they might affect outcome in individual cases is difficult, and there are no specific therapeutic implications at present. Guillain-Barré syndrome is a potentially life-threatening disease. Both general medical care and immunological treatment are essential. Meticulous attention to supportive care is needed to prevent or to manage complications [19]. Measures include monitoring of respiratory function by frequent measurement of vital capacity and other clinical outcomes, and timely transfer to ICU when needed. To help this decision making process, the Erasmus Gbs Respiratory

Insufficiency Score (EGRIS) can be used on hospital admission, because it determines the chance a patient will need artificial Ventilation [20]. Among the other issues that need attention are cardiac and hemodynamic monitoring (autonomic dysfunction), prophylaxis for deep vein thrombosis, management of possible bladder and bowel dysfunction, early initiation of physiotherapy and rehabilitation, and psychosocial support. Two-thirds of patients with Guillain Barré syndrome have pain, which can be very severe and persist for many months [21]. However, not enough evidence exists to support the use of any specific pharmacological Intervention in these patients [22]. Several Randomized Controlled Trials (RCTs) studying the effect of immunotherapy in Guillain-Barré syndrome have been done in the past few decades. IVIg and plasma exchange have proven to be effective [23]. Although IVIg and plasma exchange have proved effective, many patients with Guillain-Barré syndrome still develop severe weakness and have a long disease Course, often with incomplete recovery, pain, and Fatigue. A better treatment is therefore needed [24]. This review aims at providing an existing overview of existing evidences for the effectiveness of rehabilitation intervention in GBS.

Methods

A comprehensive search of peer reviewed literature was conducted using electronic databases, PubMed, Cochrane library. A search for review article filter using PubMed database was conducted for identifying literature published till June 2022 Medical Subject Heading (MESH) search terms were used for all the database and keyword search was used if the Mesh term was not available which included: "AIDP", OR "GBS", OR "Guillian Barre syndrome" or "demyelinating neuropathies" combined using AND "Rehabilitation" OR "Physical therapy" AND "paediatric population". Systematic review, metanalysis, case report was given high priority. Descriptive studies were also scrutinised to identify gaps in service provision.

Results

GBS in children is one of the leading causes of acute flaccid paresis. The overall result are better than the that for adult patient but GBS places significant physical, mental, and financial burden on the patient ,family and community.

Kalra V. Et al. [30]. Studied 52 children with GBS and long-term data were obtained from 40 children. In the 1-year follow-up study, 87.5% of children recovered completely or had minor symptoms, after one year this increased to 95%. Only 2 of the 40 had symptoms for more than 1 year at the last follow-up visit. They found that factors most closely related to adverse effects were the need for adherence therapy, sensory sensitivity during nerve conduction tests, and delays in autonomic mobility. In a study by Briscoe et al. she studied 22 children during a seven-year follow-up period in those presented in 1970-85 and 9-7 years in those presented in 1970-80. 18 was normal in neurologic examinations and had no complications. Two others had chronic illnesses and one had a chronic illness. Of the 19 children who experienced full recovery at the clinic, all initially presented well, and the three who performed well had complex outcomes.

Many previous studies have reported GBS disease in children. A study by Akbayram et al. reported paralysis in 34/36 (94.4%) children with GBS [31]. The majority of young patients in the Pi-Lien et al. study were placed in [32]. Akbayram's reported death was 8.9%, and Salehiomran reported that no deaths were reported in the study. In a study by Korinthenberg et al. [33] total recovery was 92% with Maneesh Kumar et al. poll is 82.4%.

Connors et.al. [34] reported a case report that highlighted treatment strategies users to rehabilitation. An 61 year old admitted to LTACH following COVID 19 induced GBS and intravenous immunoglobulin treatment. On admission, patient's functional status was evaluated using modified functional independence measure ranging from dependant to independent. AM-PAC, a standardised assessment tool was used, with patient scoring 10 on mobility segment during initial physical therapy evaluation, indicating a 77% impairment. Patient was made to participate in 5-6 months, 30 minute long treatment blocks each day. These blocks constitute of combination of individual physical therapy and occupational therapy session, as ot\ pt co-treatment session, lower extremity and upper extremity exercises, fine motor coordination group session. By the time of discharge, patient scored 20 on AM-PAC indicating 36% impairment, which was 41% improvement from admission.

Sharma et al. [35] reported a case report of 7 year old boy with normal birth and development associated with no prior illness presented with tetroplgia, facial palsy and opthamoplegia with posies and sernegative anti-GQ1b antibody was started on intravenous immunoglobulin showed inadequate response and referred for inpatient rehabilitation. Rehabilitation was focused with goal to prevent complication and maximize Functional ability. Physiotherapy was focused on active assistive range of motion exercises of all limb joint, stretching and strengthening exercises. For respiratory muscles deep breathing and incentive spirometry included. Occupational therapy was mainly focussed on bed mobility, upper limb mobility, functional mobility, hand function activities, age appropriate activity and modification. Eye care was done with night patching and four hourly application with artificial tear drop after 3 week of inpatient rehabilitation, Barthel index Score at time of admission 20/100 improved to 60/100 showing significant functional recovery ($p \leq 0.001$).

Total of 62 GBS children patients admitted to our hospital from June 2014 to December 2018 were selected and divided into control group (n = 30) and experimental group (n = 32) according to the order of admission. The children patients in the control group received physical therapy combined with occupational therapy (PT + OT), while based on the treatment in the control group, the experimental group children patients were treated with electromyography biofeedback therapy. After that, the recovery of nerve and muscle at different time points, muscle strength score, Gross Motor Function Measure (GMFM) score, and Barthel Index (BI) score of the children patients before and after treatment were compared between the two groups. There were no significant differences in the recovery of nerve and muscle of the children patients between the two groups at T 0 and T 1 ($P > 0.05$), and the recovery of nerve and muscle of the children patients in the experimental group was significantly better than that in the control group at T 2, T 3, and T 4 ($P < 0.001$); the muscle strength score, GMFM score, and BI score of the children patients in the experimental group were significantly better than those in the control group after treatment ($P < 0.001$) [36].

A 5-year old female presented with acute tetraparesis and are flexia (Radha et al). Initial imaging and cerebrospinal fluid analysis were suggestive of Acute Disseminated Encephalomyelitis (ADEM). Minimal clinical response with intravenous steroids prompted further work up. Limited nerve conduction studies suggested possible acute motor-sensory axonal neurop-

athy, a rare variant of Guillain-Barré syndrome (GBS). Repeat imaging was compatible with polyradiculopathy indicating concomitance of ADEM and GBS. The patient suffered severe motor deficits and neuropathic pain. Slow but significant functional recovery was noted after intensive inpatient rehabilitation followed by continued rehabilitation via home health services [32].

A retrospective analysis was carried out on patients with GBS who needed to continue rehabilitation after hospitalization and admitted to the Neurological Department of La Spezia from 2003 to 2017 (Prada et al). MRC and GBS-Disability scale (GBS-DS) were performed at the time of greatest clinical disability, after medical therapy, and at the end of the overall FKT. The final outcome evaluation was based on the ability to walk with or without support. ANOVA with Bonferroni post-test were used to compare MRC and GBS-DS. Ninety-six patients were admitted, but only 51 satisfied inclusion criteria. Forty patients performed intensive treatment for an average of 60.95 days, and 31 of them, once discharged, are required to continue FKT as outpatients for a mean period of 96.45 days. The mean value of MRC and GBS-DS post-FKT improved significantly compared with the post-medical therapy. Concerning walking, among the 40 patients who did not walk before therapy, 8 need support after the medical therapy and 4 (11.76%) cannot walk independently at the last follow-up [36].

Many patients with Guillain-Barré Syndrome (GBS) and Chronic Inflammatory Demyelinating Polyneuropathy (CIDP) experience excessive fatigue, which may persist for years and reduce quality of life (Garssen et al). The authors performed a 12-week study of bicycle exercise training in 20 patients with severe fatigue, 16 with relatively good recovery from GBS, and 4 with stable CIDP. Training seemed well tolerated, and self-reported fatigue scores decreased 20% ($p = 0.001$). Physical fitness, functional outcome, and quality of life were improved [37].

In a systematic review including seven articles with one RCT showed that high-intensity relative to lower intensity exercise significantly reduced disability in patients with GBS, as measured with the FIM ($p<0.005$, $r=0.71$) (Simatos et al). Overall, various types of exercise programmers improve physical outcomes such as functional mobility, cardiopulmonary function, isokinetic muscle strength, and work rate and reduce fatigue in patients with GBS [38].

In a systematic review with one high quality randomized controlled trial demonstrated effectiveness of higher intensity multidisciplinary ambulatory rehabilitation in reducing disability in persons with GBS in the later stages of recovery, compared with lesser intensity rehabilitation intervention for up to 12 months (Khan et al). Four observational studies, further demonstrated some support for improved disability and quality of life following inpatient multidisciplinary rehabilitation up to 12 months. Evidence for unit-disciplinary rehabilitation interventions is limited, with 'satisfactory' evidence for physical therapy in reducing fatigue, improving function and quality of life in persons with GBS [39].

Reported in an observational study of twenty-seven patients admitted to Sir Charles Gardner Hospital (SCGH) with GBS between 1 May 2005 and 30 April 2010 were considered for inclusion (Dennis et al). Nineteen patients consented and a waiver of consent was granted for four other patients. Data were collected from case-note audit (n = 23) and telephone survey (n = 19) during June and July 2011. Participants receiving physiotherapy (n = 16) reported they were satisfied with management (87%), treatment frequency (88%), duration (94%), and timetabling (81%) of treatment and the professionalism and rapport (100%) of physiotherapists. Median length of hospital stay was 20 days (range 5-198) for 23 participants. Physiotherapists documented patient assessment within 2 days from admission (range 1-5). First functional improvements were documented on day 6 (median, range 2-34). Physiotherapists were most commonly first to mobilize patients to sit, stand, transfer, and walk (83%, 82%, 81%, and 90%, respectively). Twenty patients (87%) developed complications during their hospital stay, the most common being low back pain (61%). This study has demonstrated that GBS patients were satisfied with care provided by physiotherapy [40].

Discussion

Clinicians are the primary care giver in patient of Guillian Barre syndrome and their families in the community. The prognosis of GBS is good but recovery is prolonged. The effective remaining residual muscle weakness hampers the quality of life particularly in developing paediatric population which remains even after the intravenous immunoglobulin treatment or plasmapheresis which stills remain the mainstay of treatment. Evidence for unit-disciplinary rehabilitation interventions is limited, with 'satisfactory' evidence for physical therapy in reducing fatigue, improving function and quality of life in persons with GBS. Multidisciplinary rehabilitation is very effective in improving quality of life when used along with primary treatment. This article stresses on the very concept of holistic approach to be used for the management of GBS.

Conclusion

This review article aims at providing a systematic analysis of efficacy of rehabilitation and physical therapy in improving quality of life in paediatric population. But most of the systematic review, RCT, observational studies selected for reviewing were on adult population. Rehabilitation program for children with GBS still remains inconclusive. But it cannot be denied that combination therapy of intravenous immunoglobulin treatment and multidisciplinary rehabilitation can prove to be standard approach for the primary care practitioner for paediatric population.

References

1. Mc Grogan A, Madle GC, Seaman HE, et al. The epidemiology of Guillain-Barre´Syndrome worldwide. Neuroepidemiology 2009; 32: 150e63.
2. Sejvar JJ, Baughman AL, Wise M, et al. Population incidence of Guillain-Barre´Syndrome: a systematic review and meta-analysis. Neuroepidemiology 2011; 36: 123e33.
3. Rajabally YA, Uncini A, et al. Outcome and it's predictor in GBS. J neurol Neurosurg psychiatry 2012; 83: 711-718
4. Hughes RA, Wijdicks EF, Benson E, Cornblath DR, Hahn AF, Meythaler JM, et al. Supportive care for patients with Guillain-Barre syndrome. Arch Neurol 2005; 62: 1194-1198.
5. Hughes RA. Systematic reviews of treatment for inflammatory Demyelinating neuropathy. J Anat 2002; 200: 331-339.
6. Brosnan CF, Claudio L, Tansy FA, Martiney J.Mechanisms of autoimmune neuropathies. Ann Neurol 1990; 27: S75–S79.
7. Hallum A. Neuromuscular diseases. In: Neurological Rehabilitation. Umphred DA, ed.4th edn. St Louis: Mosby, 2001; 363–415.

8. Ropper AH. The Guillain Barre syndrome. NEngl J Med 1992; 326: 1130–1136.

9. Yuki N, Hartung HP. Guillain-Barré syndrome. N Engl J Med. 2012; 366: 2294-2304.

10. Hugh J Willison, Bart C Jacobs, Pieter A van Doorn, Guillain-BarrÃ© syndrome. The Lancet, Volume 388, Issue 10045, 2016, Pages 717-727, ISSN 0140-6736, (https://www.sciencedirect.com/science/article/pii/S0140673616003391)

11. Soliven B. Animal models of autoimmune neuropathy. ILAR J. 2014; 54: 282-290.

12. Susuki K, Yuki N, Schafer DP, Hirata K, Zhang G, et al. Dysfunction of nodes of Ranvier: A mechanism for anti-ganglioside antibody-mediated neuropathies. Exp Neurol. 2012; 233: 534-542.

13. Willison HJ. The translation of the pathological findings described in humans to experimental models of acute motor axonal neuropathy. J Peripher Nerv Syst. 2012; 17: 3-8.

14. Willison HJ, Goodyear CS. Glycolipid antigens and autoantibodies in autoimmune neuropathies. Trends Immunol. 2013; 34: 453-459.

15. Jacobs BC, Koga M, van Rijs W, Geleijns K, van Doorn PA, et al. Subclass IgG to motor gangliosides related to infection and clinical course in Guillain-Barré syndrome. J Neuroimmunol. 2008; 194: 181-190.

16. McGonigal R, Rowan EG, Greenshields KN, Halstead SK, Humphreys PD, et al. Anti-GD1a antibodies activate complement and calpain to injure distal motor nodes of Ranvier in mice. Brain. 2010; 133: 1944-19460.

17. Plomp JJ, Willison HJ. Pathophysio logical actions of neuropathy-related anti-ganglioside antibodies at the neuromuscular junction. J Physiol. 2009 Aug 15; 587: 3979-3999.

18. Liu JX, Willison HJ, Pedrosa-Domellöf F. Immunolocalization of GQ1b and related gangliosides in human extraocular neuromuscular junctions and muscle spindles. Invest Ophthalmol Vis Sci. 2009; 50: 3226-3232.

19. Nachamkin I, Liu J, Li M, et al. Campylobacter jejuni from patients with Guillain-Barré syndrome preferentially expresses a GD (1a)-like epitope. Infect Immun. 2002; 70: 5299-5303.

20. Willison H, Scherer SS. Ranvier revisited: Novel nodal antigens stimulate interest in GBS pathogenesis. Neurology. 2014; 83: 106-108.

21. Sawai S, Satoh M, Mori M, et al. Moesin is a possible target molecule for cytomegalovirus-related Guillain-Barré syndrome. Neurology. 2014; 83: 113-117.

22. Miyaji K, Shahrizaila N, Umapathi T, Chan YC, Hirata K, et al. Are ERM (ezrin/radixin/moesin) proteins targets for autoantibodies in demyelinating neuropathies? Hum Immunol. 2014; 75: 1089-1091.

23. Samukawa M, Hamada Y, Kuwahara M, et al. Clinical features in Guillain-Barré syndrome with anti-Gal-C antibody. J Neurol Sci. 2014; 337: 55-60.

24. Chiba A, Kusunoki S, Obata H, Machinami R, Kanazawa I. Serum anti-GQ1b IgG antibody is associated with ophthalmoplegia in Miller Fisher syndrome and Guillain-Barré syndrome: clinical and immunohistochemical studies. Neurology. 1993; 43: 1911-1917.

25. van den Berg B, Walgaard C, Drenthen J, Fokke C, Jacobs BC, van Doorn PA. Guillain-Barré syndrome: Pathogenesis, diagnosis, treatment and prognosis. Nat Rev Neurol. 2014; 10: 469-482.

26. Walgaard C, Lingsma HF, Ruts L, et al. Prediction of respiratory insufficiency in Guillain-Barré syndrome. Ann Neurol. 2010; 67: 781-787.

27. Ruts L, Drenthen J, Jongen JL, et al. Pain in Guillain-Barre syndrome: A long-term follow-up study. Neurology. 2010; 75: 1439-1447.

28. Liu J Wang LN McNicol ED Pharmacological treatment for pain in Guillain-Barré syndrome. Cochrane Database Syst Rev. 2015; 4 (CD009950.)

29. Raphaël JC, Chevret S, HughesKalra V, Sankhyan N, Sharma S, et al. Outcome in childhood Guillain-Barré syndrome. Indian J Pediatr. 2009; 76: 795-9.

30. Kalra V, Sankhyan N, Sharma S, Gulati S, Choudhry R, Dhawan B, et al. Outcome in childhood Guillain-Barre syndrome. Indian J Pediatr. 2009; 76: 795-799.

31. Akbayram S, Doğan M, Akgün C, Peker E, Sayın R, et al. Clinical features and prognosis with Guillain-Barré syndrome. Ann Indian Acad Neurol. 2011; 14: 98-102.

32. Hung PL, Chang WN, Huang LT, Huang SC, Chang YC, et al. A clinical and electrophysiologic survey of childhood Guillain-Barré syndrome. Pediatr Neurol. 2004; 30: 86-91.

33. Korinthenberg R, Mönting JS. Natural history and treatment effects in Guillain-Barré syndrome: a multicentre study. Arch Dis Child. 1996; 74: 281-287.

34. Connors C, McNeill S, Hrdlicka HC. Occupational and Physical Therapy Strategies for the Rehabilitation of COVID-19-Related Guillain-Barré Syndrome in the Long-term Acute Care Hospital Setting: Case Report. JMIR Rehabil Assist Technol. 2022; 9: e30794. Published 2022 Feb 10.

35. Sharma GS, Gupta A, K R, et al. Rare Clinical Presentation in a Case of Pediatric Guillain-Barré Syndrome and Rehabilitation Outcome. J Neurosci Rural Pract. 2021; 12: 435-437.

36. Liu Q, Xue J, Zhao P, et al. Effect of Electromyographic Biofeedback Therapy on Muscle Strength Recovery in Children with Guillain-Barré Syndrome. J Healthc Eng. 2021; 2021: 1220368. Published 2021 Dec 23.

37. Korupolu R, Ngo T, Hack N, Escott E, Salles S, et al. Rehabilitation outcomes after combined acute disseminated encephalomyelitis and Guillain-Barré syndrome in a child: A case report. J Pediatr Rehabil Med. 2014; 7: 267-272.

38. Prada V, Massa F, Salerno A, et al. Importance of intensive and prolonged rehabilitative treatment on the Guillain-Barrè syndrome long-term outcome: A retrospective study. Neurol Sci. 2020; 41: 321-327.

39. Simatos Arsenault N, Vincent PO, Yu BH, Bastien R, Sweeney A, et al. Influence of Exercise on Patients with Guillain-Barré Syndrome: A Systematic Review. Physiother Can. 2016; 68: 367-376.

40. Dennis D, Mullins R. Guillain-Barré syndrome patient's satisfaction with physiotherapy: A two-part observational study. Physiother Theory Pract. 2013; 29: 301-308.