

Benefit of Inpatient Rehabilitation Care in Management of Acute Demyelinating Syndrome in Children

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Abstract

Background

Guillain 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. Guillain-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 overview 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 PUBmed Search of English-language articles published upto 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; Acute Demyelinating neuropathies; rehabilitation; physical therapy

Introduction

Guillain-Barré syndrome (GBS) is an acute, usually postinfectious 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 0-9-year-olds to 2.66 per 100 000 among 80-89-year-olds. Male subjects are more 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, Epstein-Barr virus, *Mycoplasma pneumoniae* and *Haemophilus influenzae*.³ In its typical form, GBS causes rapidly progressive diffuse proximal and distal weakness of the four limbs, sensory loss and symptoms and areflexia. 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⁾

Acute 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 lipooligosaccharides (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 extraocular 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 characterise 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,50 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 polyneuropathy.(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 transection (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 haemodynamic 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 GuillainBarré 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 randomised 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.

Method

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 were given high priority. Descriptive studies were also scrutinised to identify gaps in service provision.

Result

GBS in children is one of the leading cause 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 a77% 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 cotreatment 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 tetropgia ,facial palsy and opthamoplegia with ptosis 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 maximise Functional ability. Physiotherapy was focused on active assisstive 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 electromyographic 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)

Radha et al. A 5-year old female presented with acute tetraparesis and areflexia. 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 neuropathy, 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)

Prada et al. 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. 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)

Garssen et al. 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. 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)

Simatos et al. 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 ($p < 0.005$, $r = 0.71$). Overall, various types of exercise programmes improve physical outcomes such as functional mobility, cardiopulmonary function, isokinetic muscle strength, and work rate and reduce fatigue in patients with GBS.(38)

Khan et al. In a systematic review with One high quality randomised 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. Four observational studies, further demonstrated some support for improved disability and quality of life following inpatient multidisciplinary rehabilitation up to 12 months. Evidence for uni-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)

Dennis et al. Reported in an observational study of Twenty-seven patients admitted to Sir Charles Gairdner Hospital (SCGH) with

GBS between 1 May 2005 and 30 April 2010 were considered for inclusion. 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 uni-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 alongwith 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.

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