2023

The Progression, Treatment, and Rehabilitation of Guillain Barré Syndrome: Case Report

Hannah M. Shaffer
University of North Dakota

The Progression, Treatment, and Rehabilitation of Guillain Barré Syndrome: Case Report

by

Hannah May Shaffer

A Scholarly Project

Submitted to the Graduate Faculty of the

Department of Physical Therapy

School of Medicine and Health Sciences

University of North Dakota

in partial fulfillment of the requirements for the degree of

Doctor of Physical Therapy

Grand Forks, North Dakota

May
2023

This Scholarly Project, submitted by Hannah Shaffer in partial fulfillment of the requirements for the Degree of Doctor of Physical Therapy from the University of North Dakota, has been read by the Faculty Advisor and Chairperson of Physical Therapy under whom the work has been done and is hereby approved.

DocuSigned by:
Meridee Danks
A9A5B2E0CBF5413...
(Graduate School Advisor)

DocuSigned by:
Cindy Blom-Meland
D0E1D60A5EE3422...
(Chairperson, Physical Therapy)

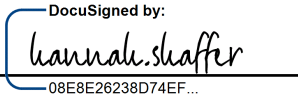
PERMISSION

Title: The Progression, Treatment, and Rehabilitation of Guillain Barré: Case Report

Department: Physical Therapy

Degree: Doctor of Physical Therapy

In presenting this Scholarly Project in partial fulfillment of the requirements for a graduate degree from the University of North Dakota, I agree that the Department of Physical Therapy shall make it freely available for inspection. I further agree that permission for extensive copying for scholarly purposes may be granted by the professor who supervised my work or, in her absence, by the Chairperson of the department. It is understood that any copying or publication or other use of this Scholarly Project or part thereof for financial gain shall not be allowed without my written permission. It is also understood that due recognition shall be given to me and the University of North Dakota in any scholarly use which may be made of any material in this Scholarly Project.

Signature 

Date 5/15/2023

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

ACKNOWLEDGEMENTS

I would like to express sincere appreciation to the patient featured in this case study. Her strength, perseverance, and overwhelming faith throughout her battle with Guillain-Barré Syndrome was awe-inspiring. I sincerely enjoyed getting to know throughout her rehabilitation journey and feel lucky to have crossed paths with such a courageous woman.

ABSTRACT

Background and Purpose: Guillain Barré Syndrome (GBS) is an acute demyelinating disease of the peripheral nervous system. It is characterized by sudden progressive weakness and paraesthesias of the limbs. This case study evaluates the effectiveness of physical therapy (PT) and the use of body-weight support gait training (BWST) in the return to function in GBS patients. **Case Description:** The patient was a 42-year-old female who presented to the acute care hospital with sudden onset paresthesias and weakness causing sudden loss of ambulation and functional mobility. **Interventions:** Due to decreased activity tolerance, initial PT treatment consisted of passive range of motion and dependent sitting balance in order to prevent contractures and other complications of prolonged bed rest. After three weeks of hospitalization, the patient began to make noticeable gains in muscular strength and functional mobility. At this time, the goal of PT became a return to prior level of function. Due to significant weakness, BWST was deemed appropriate. **Outcomes:** Following PT and the use of BWST, the patient was able to increase her strength and functional mobility. **Discussion:** The patient responded well to plasmapheresis and made a successful return to prior level of function. Additional research is required to further establish the benefits of PT and use of BWST in the GBS population.

KEYWORDS

"Overground BWST," "Guillain Barré Syndrome," "overground gait training"

CHAPTER I

BACKGROUND AND PURPOSE

Guillain Barré Syndrome (GBS) is an acute, immune mediated, demyelinating disease of the peripheral nervous system.[1] The disease is characterized by sudden progressive bilateral weakness and ascending paresthesias of the limbs.[2] GBS is the most common cause of acute paralysis affecting 1 in 100,000 people globally each year.[3] The disease course can include involvement of respiratory or cranial nerves and sensory disturbances, with 25% of severe cases requiring mechanical ventilation.[2] The involvement of the autonomic nervous system contributes to mortality which is estimated to be about 10% of all cases. The disease process is rapid and maximal disability is often reached within two to four weeks of onset.[3] After reaching nadir, patients plateau and remain at their maximum disability for several weeks or months, depending on each individual case. During this plateau phase, patients experience minimal improvement in symptoms but no further progression of symptoms.[2] Following the plateau phase, most patients begin to recover. Nearly 80% of patients regain the ability to walk independently within 6 months of initial onset although prognosis is often hard to predict due to the discrepancy between cases. GBS is a monophasic disease and relapse is rare occurring in only 2% of cases.[3]

Diagnosis of GBS can be difficult due to heterogeneity in presentation amongst patients. Difficulty of diagnosis is aided by the lack of highly specific diagnostic tools or biomarkers that are definitively present with each case meaning that clinical history and examination must be considered in order to make a diagnosis. The classic clinical pattern features weakness and

sensory loss beginning in the legs and progressing to the arms and cranial muscles.[3] The two most common criterion sets used to diagnose GBS are by the National Institute of Neurological Disorders and Stroke (NINDS) in 1990 and the Brighton Collaboration in 2011. NINDS criteria is considered to be more inclusive to GBS variants and atypical presentations and is more widely used. Two clinical features required for a diagnosis according to the NINDS criterion include: progressive weakness and diminished or absent reflexes. Other features that strongly support the diagnosis are autonomic dysfunction, relative symmetry of signs and symptoms, cranial nerve involvement, muscular or radicular back pain, electrodiagnostic features of motor or sensorimotor neuropathy, and increased protein levels in cerebrospinal fluid. Cerebrospinal fluid examination often helps rule out other causes of acute paralysis such as metabolic or electrolyte dysfunction, HIV, and Lyme disease.[1] Normative CSF protein levels do not rule out a GBS diagnosis as 30-50% patients show normal CSF protein levels within a week of onset.[4] Electromyography is also used as a diagnostic tool, in which features of demyelination can be seen such as prolonged distal motor latency, increased temporal dispersion, and conduction block.[1] GBS presents with an atypical pattern in which the sural sensory nerve action potential is unaffected and the median and ulnar sensory nerve action potentials are abnormal or absent.[1,4] Similarly to CSF protein levels, lack of evidence of neuropathy as seen on electrodiagnostic studies do not definitively rule out a diagnosis of GBS. EMG results are often normal in the early weeks of the disease process despite symptoms.[4] Nearly 50% of GBS cases experience cranial nerve involvement including facial weakness and/or difficulty swallowing. One third of GBS cases experience autonomic dysfunction such as tachycardia, hypertension, and bladder dysfunction. The presence of autonomic dysfunction suggests dysfunction of sympathetic and/or parasympathetic innervation, but specific mechanisms have yet to found and therefore cannot be

clinically predicted.[1] Once again the lack of these symptoms does not dismiss a GBS diagnosis. Overall further testing is required to further increase the sensitivity of diagnostic procedures of GBS.

The etiology of GBS is not yet fully understood. The current hypothesis states that GBS is due to an acute infectious process that causes an autoimmune response in the body triggering the attack of peripheral nerves.[4] Between 60 and 70% of GBS patients report symptoms associated with an infection three to six weeks prior to onset. [3,4] The pathogens that have been found to cause infections that most commonly precede GBS include C.jejuni, cytomegalovirus, Epstein-Barr virus, Mycoplasma pneumonia, Haemophilus influenzae and influenza A virus. Regardless of the association between specific pathogenic infections and GBS, the possibility of resulting GBS is quite rare, only 1 in 5,000 patients with campylobacter enteritis will be affected. C. jejuni has been found to be responsible for up to one third of GBS cases. It is thought that during the initial infective disease process, antibodies are generated that cross react with specific gangliosides triggering complement activation in susceptible individuals.[1] This immune activation could be influenced by genetic factors, however more research is required to fully understand this association.

Immunotherapy treatments have been proven most effective for GBS patients.[1] One of the top treatments for GBS includes intravenous immunoglobulin therapy (IVig).[1] It contains a purified plasma product that encompasses human plasma from thousands of donors.[5] IVig is thought to modulate the immune system in order to reduce the autoantibody production and inhibit complement activation and membrane attack complex formation. Although a distinct immune modulating mechanism of IVig has yet to be confirmed, the broad effect of this therapy is immunosuppressive and anti-inflammatory causing a reduction in the demyelination and

axonal injury that can contribute to the debilitating effects of GBS. By reducing these effects, the treatment is able to accelerate recovery and improve motor and sensorimotor complications.[6] The standardized dosage is 0.4 g/kg body weight daily over five consecutive days.[5] It is indicated when the patient is less than two weeks from weakness onset and is unable to walk independently.[1] The most common immunotherapy used is IVig, as it is readily available and easy to administer through and intravenous line in the peripheral veins.[7] As with any treatment or medication, adverse side effects can be experienced. Fatigue, nausea, fever, as well as allergic reactions and hemolytic anemia are possible complications of IVig treatment.[6]

Another immunotherapy treatment that has been proven clinically effective is plasmapheresis, or plasma exchange. The hypothesis behind plasma exchange treatment is thought to remove circulating neurotoxic antibodies, complement factors and other humoral mediators that can contribute to inflammation and immune response. Plasma exchange is found to have the greatest effect on clinical outcomes when it is started within 2 weeks of symptom onset, however it is shown to have a positive effect up to four weeks after onset.[1] The average amount is 200-250ml plasma per kg of body weight usually over five sessions. Evidence has yet to be established as to the superiority of plasmapheresis or IVig as a treatment for GBS. Additionally, plasma exchange followed by IVig is no more effective than each of them separately.[3] IVig is more broadly used due to its availability and ease of administration compared to plasma exchange.

Equally important in the treatment of GBS is rehabilitation and motor function restoration. A multidisciplinary approach has been proven most effective in the rehabilitation of GBS patients due to disability and prolonged hospitalization.[9] Nearly 90% of those affected by GBS make a full recovery within 1-2 years with the fastest rate of muscle strength recovery in

the first 6 months. The speed and extent muscle recovery in GBS has been correlated with duration of plateau phase and degree of motor paralysis at nadir.[10] Of key importance in the return to prior level of function is independent ambulation. Due to the disease's characteristic limb and trunk weakness, gait training GBS patients becomes a difficult task. Another barrier to gait training in this patient population is knee extensor strength recovery, which has been shown to correspond directly with duration of nadir.[10]

Body weight support training (BWST) is a common rehabilitation intervention for patients with neurological impairments and associated weakness. BWST allows a specific amount of the patient's body weight to be supported through a suspension system.[11] Through the partial unweighting of the lower extremities, weak muscles are able to demonstrate improved propulsion. Unweighting helps generate a more efficient gait pattern and discourage asymmetry and compensation due to weakness.[12] Despite the partial unweighting of limbs, it has been shown that muscle contractility and gait kinematics have been preserved in up to 30% bodyweight support allowing for greater functional carryover.[13] BWST has been proven to restore the gait pattern in acute, subacute, and chronic neurological patients while also requiring less input from a therapist.[14]

Ambulation is the outcome of complex interactions of multiple afferent and efferent signals in order to various muscles and body segments while maintaining equilibrium. Recent advances in technology have allowed the emergence of BWST devices that assist in gait training over ground. BWST over ground has been found to be more effective than on treadmill as it better increased neuronal plasticity and recovery of voluntary movements.[15] Repetitive motor task practice increases brain reorganization and neuroplasticity leading to enhance motor recovery.[16] Additionally, BWST overground increases the specificity of practice by requiring

active initiation and termination of gait and the inclusion of sensory feedback of walking on a natural surface.[15] This variability is desirable during gait practice and aids in greater degrees of carryover to independent walking. [17]

The superiority of BWST over conventional therapies such as the Bobath approach is owed to the higher intensity and task oriented repetition it allows.[16] The Bobath approach is one of the most popular neurorehabilitation approaches and is characterized by improving postural control through selective movements with carryover towards functional activities.[18] Studies have shown that BWST is able to be initiated earlier in the rehabilitation and leads to better locomotive outcomes such as increased gait speed and gait endurance when compared to the conventional Bobath approach.[19,20]

Much of the research on BWST has been conducted using stroke patients and their associated weaknesses. Minimal research is available on the use of BWST overground specifically for the GBS population, although similar outcomes can be hypothesized between the populations due to the shared neurological constraints in each. The purpose of this case study is to present the effectiveness of multi-disciplinary approach, including physical therapy and the use of BWST overground in the rehabilitation of GBS in the acute setting. It aims to discuss the outcomes of BWST in GBS and associated weaknesses, and the benefits of BWST on this patient population.

CHAPTER II

CASE DESCRIPTION

The patient described in this case report is a 42-year-old woman. Prior to her hospitalization, she was an active mother of 3 who enjoyed running, swimming, and biking. Additionally, she managed her own chiropractic business. She presented to her local emergency department (ED) August 21st with sudden onset paraesthesias in the upper and lower extremities and increased neck stiffness. The patient had a past medical history of cervical stenosis, disc herniations at C5 and C6, as well as a previous breast augmentation. When asked about any infections or illness within the last 4-6 weeks, she stated she experienced urinary tract infection symptoms, which resolved within 24 hours without treatment. Bloodwork and imaging proved unsignificant, and she was discharged home with the emergency medical team contributing her symptoms to chronic cervical changes causing nerve impingement. The patient returned to the emergency department the following day with sudden onset progressive lower extremity weakness, in addition to the continued parasethesias in the upper and lower extremities. Due to progressing weakness, the patient was unable to walk independently. She presented with areflexia and dysphagia. Cranial spinal fluid and electromyography (EMG) tests were done, to no significance. Due to her worsening condition, the patient was immediately admitted to the neuro intensive care unit with the diagnosis of "unspecified progressive weakness."

Examination, Tests, and Measures

Upon admittance, the patient immediately began receiving intravenous immunoglobulin treatment (IVig) despite the lack of a formal Guillain-Barré diagnosis. The patient received the standard dosage of Privigen, 0.4 gm/kg over the next 5 days. Orders were given to physical therapy to evaluate and treat. At the time of evaluation, the patient was non-ambulatory and was in 8/10 pain according to the Visual Analog Scale (VAS). Parasthesias in the arms and legs remained consistent from her ED arrival. They presented grossly in no dermatomal pattern. The patient reports the sensation similar to "pins and needles". Patient notably struggled to cough and clear oral secretions during the evaluation. Her lower extremity strength was tested during manual muscle testing (MMT) upon evaluation. All lower extremity motions were deemed 3/5 grossly, meaning she was able to move through the full range of motion against gravity, but unable to hold against resistance. Her right extremity was noted to be slightly weaker than her left. The patient required maximum assist of two for bed mobility initially and moderate assistance of one for sitting balance. The patient's problems were listed as extensive weakness, sensory disturbance, lack of functional mobility/capacity, and severe back pain.

The plan of care was determined and centered around maintaining functional capacity through strengthening and repeated task specific training such as practicing of transfers with varying levels of assistance. Repetitive motor practice forms the basis of motor learning by contributing to the recovery of neuronal pathways. The practice of functional tasks can reduce muscle weakness and spasticity associated with neuromuscular diseases.[21]

Within the following weeks, the patient received physical therapy every day, and she continued to physically deteriorate. The patient became completely dependent in all transfers and functional mobility. During each visit MMT was performed in order to document her strength.

Charting the progression or regression of strength and functional capacity was important to define capability, track progress of the disease, and accurately time treatments in patients with GBS.[10] (See Tables 1 and 2). She was unable to actively move her legs, and minimal muscle twitch could be felt upon quadriceps and hamstring contraction. She maintained minimal plantarflexor and dorsiflexor strength but fatigued quickly during ankle pumps. She was unable to perform more than 5 repetitions without significant straining. Her parasethsias had progressed, ascending to into to her trunk, up to the mammary line. She had begun to experience severe trunk and back pain.

Table 1. Progression of Strength

	August 23rd Initial Eval	**1 week later**	**1 month later**
Dorsiflexors	3/5	2/5	2-/5
Plantarflexors	3/5	2/5	3/5
Knee Extensors	3/5	1/5	2-/5
Knee flexors	3/5	1/5	3/5
Hip Flexors	3/5	1/5	2-/5

Table 2. Progression of Functional Mobility

	August 23rd Initial Eval	**1 week later**	**1 month later**
Bed Mobility	MaxA x 1	MaxA x 2	SBA
Sit to Stands	MaxA x 1	MaxA x 2	MinA
Sitting Balance	ModA x1	MaxA x 2	SBA
Ambulation	Unable	Unable	Mod A x 1 w/ FWW

Additionally, the patient began displaying increased blood pressure during this time, in the absence of a personal or familial history. Her blood pressure remained between 140-150/90.

Hypertension signals autonomic dysfunction and the involvement of the sympathetic and parasympathetic nervous systems. Autonomic dysfunction has been linked to higher incidence of mechanical ventilation and poorer long-term outcomes in GBS patients.[1] The patient was unable to tolerate sitting edge of bed dependently during therapy sessions due to significant "shooting" pain down her legs.

Despite any diagnostic evidence of GBS, this patient demonstrated typical GBS signs and symptoms. The classical presentation of GBS features weakness that occurs in a symmetric ascending pattern that can occur over days or weeks. It often begins distally, with the legs and arms being affected at onset and the weakness advancing proximally. Although often symmetric, weakness can present irregularly in almost 50% of cases.[22] A large amount of GBS patients also experience severe radicular pain, backache, and meningism.[1] Additionally, sensory impairments can be variable, rarely presenting with sensory level associations that would suggest myelopathy.[21] Sensory disturbances without pattern help contribute to the GBS diagnosis and rule out spinal cord compression or transverse myelitis.[1]

After 3 weeks of hospitalization, a further EMG study revealed abnormalities of nerves within the cauda equina, greatest along the ventral rootles, which is indicative of GBS. In GBS, antibodies are produced against human peripheral nerve gangliosides. These antibodies bind to nerve gangliosides and cause activated endoneural macrophages to release cytokines and free radicals, which can cause initial inflammatory endoneural oedma. This ultimately causes the characteristic Schwann cell damage, nerve conduction blocks, axonal generation, and the eventual demyelination that can appear on EMG with GBS.[22] Nerve conduction studies are essential in the diagnostic process of GBS, although the sensitivity is only around 20% during the first 4 days of progression. This is often why the initial testing comes back without

indications of demyelination or axonal degeneration and repeat testing during the disease course is imperative. Endoneural oedma is often present in the ventral rami of lumbar and cervical nerves during the initial stages of GBS. Figure 1 indicates evidence of endoneural oedma at the histological level in a patient with GBS. Figure 1B shows a semithin section of the L5 ventral root in which the myelin fiber density is preserved. Figure 1C shows a similar semithin section of the L5 ventral root in which endoneural oedma can be seen, as noted by the arrowheads. Endoneural oedma disperses the myelinated fiber leading to reduced density. Animal studies have found that axonal degeneration occurs at the peak of the development of endoneural oedma and may explain why this degeneration can not be seen in humans until the later stages of the disease.[23]

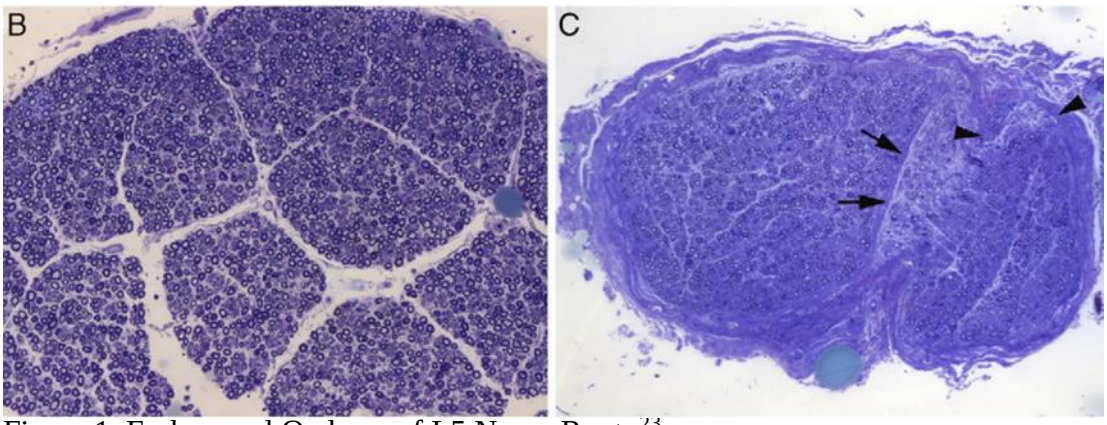


Figure 1. Endoneural Oedema of L5 Nerve Root. [23]

Clinical Impression: Intervention, Diagnosis, and Prognosis

At this point, the patient's symptoms had slowed in progression, but failed to improve at the end of IVig treatment. She continued to experience weakness, sensory loss, and high blood pressure. The patient had been receiving therapy every day for the past three weeks with no improvements. Her therapy sessions were focused on regaining functional strength and mobility by practicing bed mobility and transfers. She was unable to actively participate or tolerate sitting

due to severe pain during therapy sessions. Due to the lack of progress, the patient and therapist discussed reducing therapy to twice weekly and shifting the focus of the plan of care toe preventative measures. This approach concentrated on preventing further disability from prolonged bedrest. Immune decline, muscle atrophy, bone reabsorption, pneumonia, and deep vein thrombosis can result from prolonged bedrest, leading to increased mortality and disability rates of patients.[24] Additionally, limb contractures are an especially relevant concern for neuromuscular diseases. Prevention of contractures can be crucial to order to maintain function. In the absence of active standing or walking, passive range of motion has been shown to slow or prevent the formation of limb contractures. Therefore, during the bi-weekly therapy sessions, the therapist performed passive range of motion to all the joints of the lower extremity to patient tolerance.

Despite the grim reality of the patient's symptoms, her outlook remained positive. Due to her previously active lifestyle, her main goal was to return to prior level of function. Although the prognosis for GBS remains encouraging, the amount of motor recovery that returns and the pace at which it returns is case specific. Age of the patient, speed of progression, degree of motor paralysis, autonomic involvement, EMG evidence of demyelination, and duration of plateau phase all have been identified as prognostic factors in the motor recovery of GBS.[10] In this case, the patient rapidly deteriorated and demonstrated autonomic involvement such as dysphagia and hypertension. Additionally, she demonstrated severe weakness at nadir resulting in nearly total body paralysis. These factors would seem to predict a poorer prognosis; however, she was young, did not require mechanical ventilation and did not demonstrate widespread demyelination or axonal damage on EMG. Lack of widespread axonal damage can contribute to a faster motor recovery. These factors pointed to a favorable return to function for this patient[2,3]

Nearly 4 weeks after her initial evaluation, the patient had a central venous port placed and began receiving plasma exchange treatments. The plasmapheresis treatment was outlined for 200 ml plasma per kg of body weight for 8 treatments on alternating days. The patient received the first two treatments, in which minor improvements in wrist motion were observed, and she was able to perform all wrist motions actively. On the day of the third plasmapheresis treatment, the patient began experiencing low fibrinogen. Fibrinogen is a plasma product that helps in clotting formation.[26] Low fibrinogen counts greatly increase the risk of bleeding, which can lead to serious complications.[26] The plasmapheresis treatment was delayed for 5 days due to continued low fibrinogen levels, which contributed to slowed motor recovery.

One month after initial evaluation the patient had received the 3rd plasmapheresis treatment and began to experience some notable improvements in motor function. She was able to demonstrate active assist heel slides and muscle contraction that could be felt during quad sets. Despite these improvements, the patient maintained noticeably weaker left side as compared to the right side. In the wake of the 4th plasmapheresis treatment, the patient achieved a supine to sit transfer with maximum assistance of two, her first in over a month. The patient was able to horizontally abduct her shoulder to actively reach for the bed rail and flex her hips to a hooklying position prior to the transfer. Remarkably, she was also able to complete the transfer without pain. She was then able sit to stand transfer with maximum assistance (MaxA) of one and bilateral blocking of knees due to significant weakness of knee extensors. It was at this point that it was determined she was ready to initiate BWST.

Many different factors were taken into consideration regarding her eligibility for BWST. Initially, the patient was not a candidate for BWST. She was experiencing progressive weakness and severe pain. Additionally, she had limited tolerance to dependent edge of bed activities due

to pain. She also required MaxA x 2 for bed mobility and struggled to hold her own head up. BWST was only considered after a month of hospitalization when she started to see some return of motor function following plasmapheresis treatments. The primary factor in considering her appropriateness for BWST was muscular strength. Muscular strength recovery is variable in GBS. As demonstrated in this case, knee extensor weakness is correlated with length of plateau phase and can be slow to recover.[10] Directly prior to initiating BWST the patient grossly demonstrated 3-/5 strength of hip flexors, knee flexors, hip extensors and dorsiflexors. Her knee extensors and plantarflexors maintained 2+/5 grossly. Muscular strength is important in order to determine eligibility for gait training due to the muscle force required during specific phases of the gait cycle. For example, the patient required bilateral knee blocking during sit to stand transfers due to significant knee extensor weakness. During gait, the knee extensor strength is required in order to prevent knee collapse during stance phase.[27] Knee collapse during standing indicates that this patient would face considerable difficulties in attempting ambulation, which requires significant knee extensor strength to move through single leg stance. Additionally, hip flexors and dorsiflexors were key in allowing the patient to actively practice stepping during BWST. Active practice of gait has been shown to drive neuroplasticity of motor skills.[15] A MMT score of 3/5 was essential in allowing this patient to lift her leg and ankle against gravity in order to take steps.

Also of importance was the safety of the staff participating in gait training. Regular gait training would have been an immense undertaking due to the patient's significant weakness and balance impairment. It would have been extremely difficult to manually support the patient enough to allow her to take steps independently. It puts staff at risk for injuries and the patient at increased risk of falling. For this reason, BWST was the safer option for the patient and staff.

Patient cognition also played a role in the safety and effectiveness of gait training. Cognition helps a patient initiate active stepping and respond to cueing during BWST. Communication is key for the patient to be able to express any concerns or fatigue during the session.

CHAPTER III

INTERVENTION

The patient was seen every day for the initial 3 weeks of her hospitalization. Due to lack of tolerance and severe pain, therapy was moved to every other day. Once the patient began demonstrating improvements in pain and motor function following plasmapheresis treatments, therapy resumed daily. Prior to initiating gait training, the patient was able to perform bed mobility with MinA. She performed a sit to stand transfer with maximum assistance (MaxA) She was able to perform a 45 second stand with MaxA for balance and bilateral blocking of knees. The patient demonstrated 3/5 gross lower extremity strength, which allowed her to perform active stepping during BWST. This allowed for a safer option for gait training due to the extensive weakness and balance impairment of the patient. By reducing ground reaction forcing through unloading, the patient would be able to practice the motor pattern of ambulation in absence of full strength and balance. Additionally, EMG studies report minimal change in muscle activation even at high rates of unloading, suggesting that gait training with body weight support can also help strengthen the same muscle groups used in ambulation.[15]

The partial weight bearing suspension walker used was the Rifton TRAM. The TRAM is a transfer and mobility device that can be used for gait training and/or transfers. The TRAM features a harness support system with two belts and safety buckles. Additionally, the TRAM's light frame makes it easy to maneuver and adds minimal resistance to the patient. Figure 2 illustrates the TRAM.[28]

Figure 2. The Rifton TRAM.[28]

On the day of the intervention, the patient was informed of treatment plan and gave verbal consent to proceed. She was harnessed into the TRAM in a sitting position from edge of bed. Two straps were placed between her legs in order to support her body weight. Once fastened, the device was utilized to lift the patient from a sitting to a standing position. Upon standing, the weight was adjusted to the patient's preference, about 45% body weight. Once adjusted, the patient was instructed to begin taking steps. Assistance was required from the clinician to assist the patient in steering out of the narrow hospital room, but once in the hallway the patient was able to propel herself independently. The patient was able to walk 160 ft consecutively prior to fatigue. She was then lowered into a wheelchair utilizing the lif mechanism in the TRAM. From here, she was able to recover in a seated position. She was audibly short of breath and unable to continue ambulation training.

She required no cueing for stepping and only required occasional cues for steering. The patient reported no pain during gait training and was limited only by knee extensor weakness and

aerobic deconditioning. Following her gait training, the patient was wheeled back to her room in a wheelchair due to fatigue.

The following session, the patient reported mild fatigue but was ready to continue with her physical therapy session. During the session, she was able to complete bed mobility with SBA. Additionally, she performed a sit to stand transfer from the edge of bed with moderate assistance (ModA). This was greatly improved as compared to the session prior to BWST in which she required MaxA for a sit to stand and MaxA for bed mobility. Due to this drastic improvement, clinical experience was utilized in order to make the decision to attempt ambulation with a front wheeled walker (FWW). Despite being able to barely stand without knee buckling and MaxA prior to BWST, the patient was able to ambulate 20' with a FWW with ModA prior to fatigue. Table 3 demonstrates the significant difference in functional mobility directly before and after BWST.

Table 3. Comparison of Functional Mobility Before and After BWST

	Before BWST 9/15	**After BWST 9/17**
Bed Mobility	MaxA	SBA
Sit to Stand	MaxA	ModA
Ambulation	Unable	20' w/ FWW ModA

CHAPTER IV

OUTCOMES

Due to the extent of the patient's disability during her plateau phase, the outcomes in this case were highly favorable. Despite the extent of the patient's disability during nadir, the patient was able to make an impressive recovery. Objective and subjective outcome measures were used to measure the patient's response to physical therapy intervention. The objective outcome measures used were MMT, distance ambulated, and level of assistance needed for transfers. Manual muscle testing helped track the patient's motor function recovery throughout her rehabilitation. Level of assistance was utilized in order to help determine the patient's level of independence in functional tasks and help track her motor recovery. Table 4 illustrates a comparison of her functional mobility at initial evaluation and discharge. Over nearly 6 weeks of hospitalization, her functional status initially declined and then sharply increased in her final two weeks of hospitalization. The frequency of physical therapy sessions was then moved up to everyday following her rapid recovery of functional mobility. In total she received 38 physical therapy treatment sessions over the duration of her 6 week hospitalization, The improvement over her hospitalization in functional status is notable. Table 5 illustrates her improvement in motor function as demonstrated by MMT from nadir around 4 weeks to discharge at 6 weeks. It demonstrates the rapid return of muscle strength in a little over 2 weeks.

Table 4. Comparison of Functional Mobility.

	Initial	**Final**
Ambulation distance	Unable	75 feet w/ FWW ModA
Bed Mobility	MaxA x 1	SBA
Sit to Stands	MaxA x 1	MinA
Sitting Balance	MaxA x 1	SBA

Table 5. Comparison of MMT.

	At nadir	**Final**
Dorsiflexors	2/5	3/5
Plantarflexors	2/5	3/5
Knee Extensors	1/5	3/5
Knee Flexors	1/5	3/5
Hip Flexors	1/5	3/5

The patient had a favorable response to BWST. She was able to practice the motor pattern of ambulation without having to use strength to support her body weight. Additionally, she stated even performing the upright motion of ambulation in the TRAM improved her mental wellbeing and motivation following being bedbound for over a month. She felt as if she had "turned a corner" in her rehabilitation. After a single trial of BWST, she had progressed past the need for body-weight support and was able to ambulate with a FWW. It is likely due to the degree of motor return the patient experienced in a short amount of time, but BWST training played a pivotal role in that recovery of motor function.

The patient occasionally reported increased fatigue following her physical therapy interventions. She noted that when she pushed herself during therapy, she was often more tired the following day. A common symptom of GBS is fatigue, and it did not further benefit the patient or her recovery to exhaust herself daily.[2] This effect was combatted by alternating our ambulation days, as the patient was often unable to tolerate gait training back-to-back days.

Compliance during therapy sessions was never an issue, and the patient always strived to push herself. She remained motivated throughout her rehabilitation and was very pleased with her progress.

On the day of discharge, 6 weeks after initial evaluation, the patient was able to ambulate 75 feet with a front wheeled walker with ModA prior to fatigue. She was able to perform self-care tasks, bed mobility, and most transfers at that time. Continued inpatient therapy services were recommended due to her rapid recovery in order to further improve functional capacity. She received intense inpatient therapy services for up to 3 hours a day for another week following discharge from the hospital. Once home, she was referred to outpatient physical therapy and began to ambulate community distances (up to one mile) with a single point cane a month after discharge. Over the course of the next several months, she steadily progressed to independent ambulation. 6 months after discharge, she was able make a full return to prior level of function. In conclusion, she was able to return to her prior level of function following discharge through continued outpatient rehabilitation services and the BTWS was a critical role in facilitating that return to function.

CHAPTER V

DISCUSSION

The purpose of this case study was to explore the role of physical therapy and the usage of BWST in the GBS population. In this case study, the usage of BWST played a pivotal role in the return to independent ambulation for the patient. The patient presented to inpatient physical therapy with significant weakness, ascending paraesthesias, and severe back pain. The patient was unable to walk and became dependent in nearly all functional tasks. Through clinical experience and literature, an initial evidence-based treatment of preventing further disability from being bed bound was adopted. This consisted of dependent sitting balance and passive range of motion. After several weeks of hospitalization and a failed round of IVig, the patient began to notice returns in motor function following plasma exchange treatment. At this point, a physical therapy plan shifted to focus on facilitating a return to prior level of function. The primary physical therapy overseeing the case utilized clinical experience and judgement to initiate BWST in this patient. Following the BWST training, the patient was able to ambulate with a FWW with ModA much improved from being unable to ambulate days prior.

Due to the rarity of GBS, literature supporting BWST in the GBS population is minimal. The literature supporting overground BWST in post-stroke patients is abounding, and it is hypothesized to have comparable effects in the GBS population due to similarities in neuromuscular constraints. Over ground BWST demonstrates increased neuronal plasticity and recovery of motor function due to brain reorganization following repetitive task practice when

compared to conservative pre-gait strengthening.[15,16] It also demonstrates better carryover to independent gait than body weight support training performed on a treadmill.[7] Another benefit of BWST is that is can be initiated sooner in rehabilitation compared to traditional gait training as the patient is able to be supported completely by the device if necessary. Earlier initiation has been shown to lead to increased gait speed and gait endurance when compared to the conventional approach of pre-gait strengthening.[19,20] It has also been shown that unweighting aids to increased efficiency during gait despite muscle weakness. Additionally, BWST discourages compensation due to weakness.[12] Regardless of percentage of body unweighting, muscle contractility and gait kinematics are maintained throughout BWST suggesting strengthening and motor learning benefits.[13]

Many of the benefits of BWST studied in the post-stroke can be seen in this case study. Gait speed and gait endurance were unable to be measured during the course of this patient's rehabilitation due to her severe weakness. Prior to BWST, she was unable to walk during her hospitalization and was able to initiate ambulation following one session of BWST. This can also be attributed to the rapid rate of motor recovery often seen in GBS and more studies would be required to measure gait speed improvements from BWST in this population. In can be hypothesized that this patient received strengthening and motor functioning benefits as detailed in post-stroke BWST literature.[11-15] Similar evidence can be seen in the rapid increase of functional mobility in the 48 hours following BWST. She required significantly less assistance with bed mobility and sit to stand transfers. Additionally, her knee extensor strength had progressed so that she was able to maintain stance phase without collapse during ambulation. This increase in strength can also be attributed to the psychological boost given to the patient during BWST. The patient stated that she felt "renewed hope" in her recovery following her

BWST. Additionally, rapid motor recovery can be largely attributed to the effects of the plasmapheresis treatment, while BWST and physical therapy just helped to guide than motor recovery.

Reflective Practice

The main limitation of this case study was that due to the rapid recovery of motor function BWST was only performed during one session. Additionally, GBS often demonstrates significant heterogeneity between symptom presentation among cases making it difficult to standardize patients. If the study was to be repeated, it would be suggested that BWST be a more consistent tool utilized in physical therapy treatment rather than just during one session. Additionally, it would be beneficial to utilize other functional outcome measures other than just MMT and assistance during transfers. I think once again it would be beneficial to track motor function daily and compare it with the immunotherapy treatment they are receiving. In conclusion, Further research is required in order to establish the indications of physical therapy and BWST with acute GBS and the effects of physical therapy and regular BWST on the GBS population.

REFERENCES

1. van Doorn PA, Kuitwaard K, Walgaard C, van Koningsveld R, Ruts L, Jacobs BC. IVIG treatment and prognosis in Guillain-Barré syndrome. *J Clin Immunol*. 2010;30 Suppl 1(Suppl 1)

2. 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(8):469-82.

3. Leonhard SE, Mandarakas MR, Gondim FAA, et al. Diagnosis and management of Guillain-Barré syndrome in ten steps. *Nat Rev Neurol.* 2019;15(11):671-683.

4. Sudulagunta SR, Sodalagunta MB, Sepehrar M, et al. Guillain-Barré syndrome: clinical profile and management. *Ger Med Sci.* 2015;13:Doc16.

5. Hughes RA, Swan AV, van Doorn PA. Intravenous immunoglobulin for Guillain-Barré syndrome. *Cochrane Database Syst Rev*. 2014;2014(9):CD002063.

6. Liu S, Dong C, Ubogu EE. Immunotherapy of Guillain-Barré syndrome. *Hum Vaccin Immunother.* 2018;14(11):2568-2579.

7. Shang P, Feng J, Wu W, Zhang HL. Intensive care and treatment of severe Guillain-Barré Syndrome. *Front Pharmacol*. 2021;12:608130.

8. Chevret S, Hughes RA, Annane D. Plasma exchange for Guillain-Barré syndrome. *Cochrane Database Syst Rev*. 2017;2(2):CD001798.

9. Khan F, Ng L, Amatya B, Brand C, Turner-Stokes L. Multidisciplinary care for Guillain-Barré syndrome. *Eur J Phys Rehabil Med*. 2011;47(4):607-612.

10. El Mhandi L, Calmels P, Camdessanché JP, Gautheron V, Féasson L. Muscle strength recovery in treated Guillain-Barré syndrome: a prospective study for the first 18 months after onset. *Am J Phys Med Rehabil.* 2007;86(9):716-724.

11. Apte S, Plooij M, Vallery H. Simulation of human gait with body weight support: benchmarking models and unloading strategies. *J Neuroeng Rehabil.* 2020;17(1):81.

12. Burgess JK, Weibel GC, Brown DA. Overground walking speed changes when subjected to body weight support conditions for nonimpaired and post stroke individuals. *J Neuroeng Rehabil.* 2010;7:6.

13. Richter C, Braunstein B, Staeudle B, et al. Gastrocnemius Medialis Contractile Behavior Is Preserved During 30% Body Weight Supported Gait Training. *Front Sports Act Living.* 2021;2:614559.

14. Park BS, Kim MY, Lee LK, et al. Effects of conventional overground gait training and a gait trainer with partial body weight support on spatiotemporal gait parameters of patients after stroke. *J Phys Ther Sci*. 2015;27(5):1603-1607.

15. Awai L, Franz M, Easthope CS, Vallery H, Curt A, Bolliger M. Preserved gait kinematics during controlled body unloading. *J Neuroeng Rehabil.* 2017;14(1):25.

16. Mao YR, Lo WL, Lin Q, et al. The Effect of Body Weight Support Treadmill Training on Gait Recovery, Proximal Lower Limb Motor Pattern, and Balance in Patients with Subacute Stroke. Biomed Res Int. 2015;2015:175719.

17. Barela AMF, Gama GL, Russo-Junior DV, Celestino ML, Barela JA. Gait alterations during walking with partial body weight supported on a treadmill and over the ground. *Sci Rep.* 2019;9(1):8139.

18. Díaz-Arribas MJ, Martín-Casas P, Cano-de-la-Cuerda R, Plaza-Manzano G. Effectiveness of the Bobath concept in the treatment of stroke: a systematic review. *Disabil Rehabil.* 2020;42(12):1636-1649

19. Visintin M, Barbeau H, Korner-Bitensky N, Mayo NE. A new approach to retrain gait in stroke patients through body weight support and treadmill stimulation. *Stroke.* 1998;29(6):1122-1128.

20. Mehrholz J, Thomas S, Elsner B. Treadmill training and body weight support for walking after stroke. Cochrane Database Syst Rev. 2017;8(8):CD002840.

21. French B, Thomas LH, Coupe J, et al. Repetitive task training for improving functional ability after stroke. *Cochrane Database Syst Rev.* 2016;11(11):CD006073.

22. Rodríguez Y, Rojas M, Pacheco Y, et al. Guillain-Barré syndrome, transverse myelitis and infectious diseases. Cell Mol Immunol. 2018;15(6):547-562.

23. Berciano J. Axonal pathology in early stages of Guillain-Barré syndrome. *Neurologia* (Engl Ed). 2022;37(6):466-479.

24. Ding R, Zhang H. Efficacy of very early mobilization in patients with acute stroke: a systematic review and meta-analysis. *Ann Palliat Med*. 2021;10(11):11776-11784.

25. Skalsky AJ, McDonald CM. Prevention and management of limb contractures in neuromuscular diseases. Phys Med Rehabil Clin N Am. 2012;23(3):675-687.

26. Jagdish K, Jacob S, Varughese S, et al. Effect of double filtration plasmapheresis on various plasma components and patient safety: A prospective observational cohort study. *Indian J Nephrol.* 2017;27(5):377-383.

27. Sylvester AD, Lautzenheiser SG, Kramer PA. Muscle forces and the demands of human walking. *Biol Open*. 2021;10(7):bio058595.

28. TRAM; Three powerful functions in one device. Rifton.com. Published January 2023. Accessed February 6, 2023. https://www.rifton.com/products/lift-and-transfer-devices/rifton-tram