LITERATURE REVIEW

Pulmonary Rehabilitation in Guillain-Barré Syndrome (GBS)

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ABSTRACT

GBS was first described by Landry in 1859, a post-infectious polyneuropathy primarily not only affecting the motor system, but also on the sensory nervous system and the autonomic nervous system. It is acute and known as an autoimmune disease. The disease is more common in male, 1.5 times higher than women in western countries, affects any ages, mostly adults. 60% of GBS patients will be preceded by an upper respiratory tract disease and 27% unidentified illness that preceded it. Started with lower limb weakness and progresses to the upper limb and eventually the bulbar muscles; known as Landry's Ascending Paralysis or rubbery legs. The diagnosis can be confirmed by history and physical examination. The prognosis depends on the subtype of GBS. For about 85% of patients will have functional recovery within a few months to a year. Rehabilitation in GBS management generally emphasis on immobilization and management of pneumonia and respiratory failure. GBS patients with respiratory dysfunction should get immediate rehabilitation and obtain a specific treatment program.

Keywords: *Guillain Barre Syndrome,Respirasi Rehabilitation,Breathing Exercise, Inspiratory muscle training*

INTRODUCTION

Guillain-Barré Syndrome (GBS)

Guillain-Barré Syndrome (GBS) was first described by Landry in 1859. Later in 1916 Guillain, Barre and Strohl found increased levels of protein and normal cell count in cerebrospinal fluid.[1] In 1949, Haymaker and Kernohan made clinical presentation in front of French armies about GBS and its histopathological changes in peripheral nerve inflammation that occurs in 50 cases of GBS. In 1980-1990, plasma exchange and intravenous immunoglobulin administration was identified as an effective therapy.[2]

In western countries, GBS is 1.5 times more likely in men than women. It affects all ages, mostly adults than children. The incidence of GBS in Australia is 1-2 cases per 100,000 population per year with a mortality rate of <1%.[1] Although GBS is said to have a good prognosis ("Maladie benigne et spontanement curable), but mortality may reach to 5% -10% of patients experienced a severe disability a year after the onset of neurologic deficit. The first team built to manage GBS patients in hospitals consisted of medical specialists and experts in

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intensive care.[1,5,6]

Guillain-Barre Syndrome (GBS) is a post infection polyneuropathy primarily affecting the motor, sensory and the autonomic nervous system. It is acute and recognised as an autoimmune disease. Most of the patients will exhibit demyelinating neuropathy, but in some cases the main abnormality is axonal degeneration.[5,7]

GBS is known as an acute inflammatory demyelinating polyneuropathy due to immunological reactions. Sensitization caused by T lymphocytes against proteins in the myelin sheath. Demyelination occurs intermittently along the peripheral nerves, nerve roots and the myelin sheath as a result of infiltration of lymphocytes, which eventually led to inhibition of action potential conduction. Thereby, reducing nerve conduction velocity and conductivity and causes blockade. In axonal neuropathy, nerve conduction velocity is normal, but the number of motor functional unit decreases. Within 2-3 weeks of demyelination, inflammation will stop and begin the process remielinisasi.[4,8] Referring to it, recovery can occur rapidly with the onset of remyelination. Axonal degeneration is associated with the deceleration of the recovery process and severe sequelae (Figure 1).[3,8]

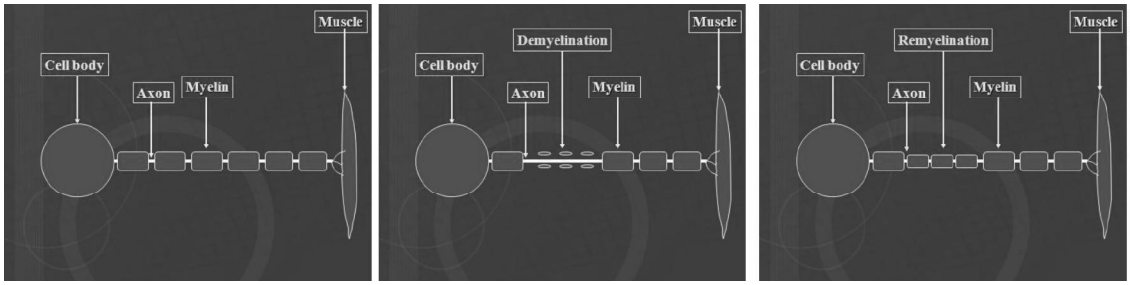

Figure 1. Demyelination and Remyelination[8]

Around 60% of GBS patients will be preceded by an upper respiratory tract disease, and 27% are unidentified illness. Viral infection (Epstein-Barr virus, Coxsackie, Influenza, Echovirus, cytomegalovirus, hepatitis viruses and HIV) is the most common preceding disease, although bacterial infection (Campylobacter jejuni, Mycoplasma pneumoniae) is also responsible less commonly.[3,8]

Based on the area of weakness, GBS is classified into (1) Acute inflammatory demyelinating polyradiculoneuropathy (AIDP), (2) Acute axonal neuropathy motors (SAFE), and (3) Acute sensory axonal neuropathy motors (AMSAN). Other classification includes Fisher syndrome and pure sensory neuropathy.[1,3,6]

GBS has a rapid development of the motor paralysis reflex with or without sensory loss. Paralysis occurs 10 days after the non-specific viral infection. The weakness starts at the lower limbs, and progresses to the upper limbs, and eventually, the bulbar muscles (known as Landry Ascending Paralysis or rubbery legs). The proximal and distal bilateral muscles are affected symmetrically, but asymmetrical weakness is found in 9% of cases. Symptoms develop progressively within few days or weeks and may cause inability to walk and tetraparesis accompanied by tingling sensation in the extremities. Lower limbs are more affected than the upper extremities. Facial paresis; pain in the neck, shoulder, and back; and weakness in extremities occur in 50% of cases. The pain can disappear by itself or can be treated by analgesics. Mild sensory deficits (pain and temperature), and severe symptoms in deep tendon reflex and proprioceptive may appear. When clinical symptoms entered the plateau period (4 weeks of onset), the deterioration will not occur.[2,3,7] Cranial nerves are frequently involved, causing paralysis with bulbar manifestation of airway clearance difficulty, such as respiratory failure, dysphagia and dysarthria. Facial muscles paresis with 50% of majority is bilateral VII nerve palsy. It interferes the eating process and increases the risk of aspiration. For about 40% of patients will experience respiratory dysfunction or oropharyngeal paresis. This

is due to the involvement of intercostal and phrenic nerves causing intercostal and main inspiratory (diaphragm) muscles paralysis. Thus, respiratory problems are inevitable.[3,4,5]

The diagnosis can be confirmed by medical history, physical examination, and classified based on GBS subtypes.

Cerebrospinal fluid is essential and the result is meaningful. Abnormal rise of protein concentration twice higher than normal level (1-10 g/L or 100-1000 mg/dL) without the presence of pleocytosis, and normal glucose level are indication. The electrodiagnostic testing (electromyography) exhibits significant decrease in motor nerve conduction velocity and sensory nerve conduction until the acute denervation occurs. The magnetic resonance imaging (MRI) of brain and spine should be performed to exclude other diseases.[2,7] Moreover, pulmonary function tests to perform include measurement of maximum inspiratory pressure, vital capacity neuromuscular respiratory function and prediction of the strength of the diaphragm. Maximum expiratory pressure also reflects the strength of the abdominal muscles. Routine evaluation should be performed at the bedside to monitor respiratory status and the need of ventilation assistance. Respiratory assistance should be considered when expiratory vital capacity decreased into <18mL/kg or constant oxygen saturation (PaO_2 <70 mm Hg).[10]

Differential diagnoses of GBS consist of transverse myelitis, myasthenia gravis, and vasculitis neuropathy.

Referring to an article written by Blocka K and Yelland J in 2011, there are three stages of GBS treatment. First, treatment modification to ensure adequate management that will shorten the acute phase includes administration of Plasma Exchange (PE), Intravenous Immunoglobulin (IVIG) and corticosteroids. Second, life support treatment at critical acute phase includes hemodynamic control and admission to the ICU. Lastly, palliative treatment to not merely reducing the symptoms, but it's priority is to assure patient's well-being, such as breathing exercise, physical therapy and pain control.[11]

In approximately 85% of patients will have functional recovery within a few months to a year. With the morbidity rate less than 5%, the most common cause of mortality is the pulmonary complication.[3,7] Factors that aggravate the prognosis are old age, respiratory dependency, abnormal function of the peripheral nerves and axonal degeneration, disease progression into quadriplegia, *campylobacter jejuni* infection, and pleteau condition in the past 3 weeks.[5,7,12]

There are five GBS recovery phases, include experiencing ADL dependency, helplessness, curiosity about GBS, discovering inner strength, and regain independency. GBS support groups play an important role in recovery process of hospitalized patients, both for patients and their families. GBS has a serious long-term impact on work and personal life, even 3-6 years after the occurrence of the disease. Patients and families should know and understand about the GBS progression and recovery rates to keep on being optimistic.[2]

Rehabilitation Management of GBS

Meanwhile, other literatures indicated that proper rehabilitation in according to the stage of GBS where the presences of weakness are troublesome would maximize the potential physical and functional activities of patients. During the acute phase, a prompt physical therapy to prevent immobilization and respiratory rehabilitation are effective. When the respiratory and motor functions have recovered, patients begin the recovery phase. In this phase, physical therapy focuses on the restoration of respiratory function. Once patients entering the long-term rehabilitation phase, the recovery process will be diverse. In this phase, rehabilitation process will focus on maintaining respiratory function, strengthening and re-educating motor function and sensory awareness.[13]

Many also say that patients with severe GBS will need hospital-based rehabilitation for 3-6 weeks followed by a community and home-based rehabilitation programs for 3-4 months.[4]

Pulmonary Rehabilitation in Patients with GBS

Rehabilitation management in GBS patients emphasizes in immobilization and management of pneumonia and respiratory

failure which commonly occur.[3,14] According to American Thoracic Society (ATS) and European Respiratory Society (ERS), pulmonary rehabilitation is defined as a comprehensive and multidisciplinary individually tailored therapy based on accurate diagnosis that comprises emotional and educational support; stabilizing or restoring physical and psychopathology of lung disease in effort to regain and prolong the highest functional lung capacity and in disabled situations. The exercise programs include respiratory muscle exercises, strength exercises and aerobic exercises. Education about pathology of the disease, management of disease, physical therapy, relaxation, skill, drugs, use of oxygen are also important.[15]

Chest therapy is a therapy to enhance the function of respiratory system. It is performed to expand the thorax cavity with eased and controlled breath to remove secretions from the lungs that restrain breathing capacity. Not only during the use of a ventilator, but it can also be used during the recovery process to prevent pneumonia. Therapies include postural drainage, chest percussion, breathing exercises (deep breathing), cough, chest vibration and chest mobility exercises.[16]

Postural Drainage is aimed at maintaining airway clearance and preventing respiratory complications. It is performed by positioning the chest to support secretions drainage and also to facilitate proper air entry to the lung lobes. Periodic suction with hygiene and nebulization, percussion, vibration, and mobilization of secretions can be carried out.[10,16]

Chest mobility exercise is an exercise that utilizes active movements of extremities along with deep breathing. This is performed to increase shoulder and chest wall mobility in facilitating breathing. In some cases, patients with trunk muscles stiffness are not able to expand the chest cavity optimally during respiration. Chest therapy exercises that will stretch these muscles with deep breathing, increases the airflow and causes improved ventilation. These are required to strengthen the expiration control. One way to do this is by putting the hip forward and trunk flexion during expiration. This movement is effectively expanding the abdominal viscera and pushes the diaphragm and facilitates expiratory easily.[16]

In addition, Active Cycle of Breathing Technique (ACBT) can be applied to mobilize sputum and provides good breathing techniques in patients with respiratory disorders. These are the ACBT techniques, breathing control, deep breathing exercise (DBE), huffing (forced expiratory technique/ FET).[17]

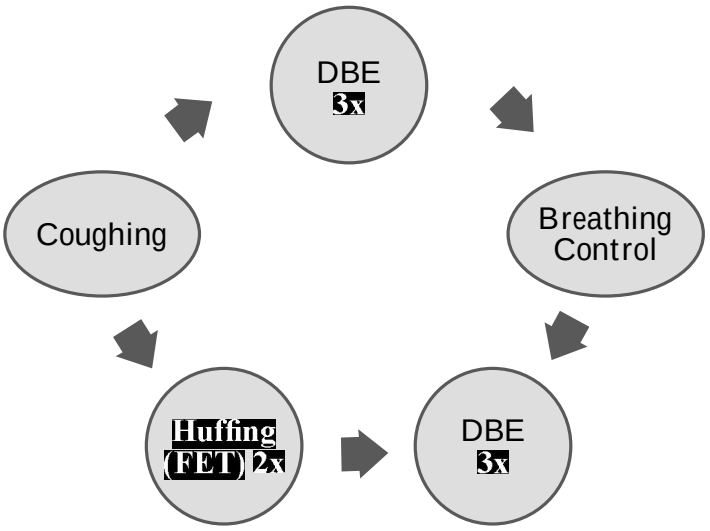

Figure 2. Active Cycle of Breathing Technique (ACBT)[17]

DISCUSSION

Pulmonary Rehabilitation Based on GBS phases

According to Firoz Ahmed, 2011, rehabilitation management of GBS patients falls into three phases. First phase is management during the acute phase when the diseases is in the acute progressive stage. The main treatment goal in this phase is airway clearance support, followed by preventing pulmonary infections, and maintaining peripheral circulation with a variety of physical techniques, e.g. chest percussion therapy, breathing exercises, restrictive inspiration workout for airway and lungs clearance and breathing support. Secondly, the recovery phase is when patients are able to maintain their own airway and ventilation. Breathing exercises and inspiratory muscle training are performed with tapering dose. Thirdly, the long-term rehabilitation phase focuses on airway maintenance and ventilation capacity with techniques of breathing and effective coughing exercises with frequency based on correct instruction.[12]

Patients with neuromuscular disorders such as GBS, show positive results in chest physical therapy.[6,8] In GBS, regaining an optimum respiratory function is a priority. Whether it is caused by pneumonia infection due to immobilization, prolonged ventilator usage; or respiratory muscle weakness itself.[1] GBS with abnormalities in respiratory system can be the major cause of death. GBS is one of the diseases that require ICU care in acute phase. Indications of ICU care are respiratory failure, severe weakness of bulbar system, unstable autonomous system, and aspiration.[13]

Neuromuscular respiratory insufficiency occurs about 17-30% cases, and the utilization of mechanical ventilation is 20-30%. The progression of respiratory insufficiency is visible and not few of GBS patients require mechanical ventilation due to high risk for complications such as pneumonia, tracheobronchitis, pulmonary embolism, or bacteremia (Table 1). In patients with GBS, mechanical ventilation is indicated involving the severity of bulbar dysfunction, increased risk of aspiration, and gas exchange abnormalities. It is advisable to make an assessment of vital capacity every 12 hours, respiratory rate every 2-4 hours, swallowing function (N.VII) every day, and if one of assessments deteriorated, then it is an indication of mechanical ventilation usage. Early expression of this disorder occurs in arterial blood gasses, which is a mild decrease in PaO_2.

The involvement of accessory respiratory muscles, tachypnea and dyspnea, and weakening of expiratory muscle strength decrease the coughing reflex, so reduce the ability to clear the bronchial and tracheal secretions and thus increase the risk of aspiration (pneumonia). Bulbar weakness can impair patient's ability to protect the airway. Decreased muscle tone of the tongue and oropharynx predispose to airway obstruction, especially at night, when the muscle tones of vocal cords decreases. Meanwhile, due to oropharyngeal muscles weakness, the tongue and face muscles may interfere the protective reflex (i.e. cough) and result in aspiration. The progression and pattern of the damage to nerve signal conduction also compromise the respiratory function (Table 1).[8,12]

Table 1. Risk Factors of Mechanical Ventilation and Predisposing Factors of Respiratory Failure[13]

Risk Factors of Mechanical Ventilation	Predisposing Factors to Respiratory Failure
Quick & Progressive Tachypnea	Tachypnea
Inadequate cough	Tachycardia
Bilateral facial weakness	Perspiration
Inability to stand	Supported by accessory muscles of respiration
Inability to lift elbow	Asynchronous chest and abdominal movements
Inability to lift head	Decrease in vital capacity <50% from baseline (15ml / kg)
Rise in liver enzymes	Pressure Inspiratory maximal (PImax) <30cmH_2O
Radiographic abnormalities	Pressure Expiratory maximal (PEmax) <40cmH_2O
Vital capacity <20 ml / kg	

According to Ted M. Burns, 2008, the average duration of mechanical ventilation usage is 2-6 weeks. The ventilator weaning process is carried out in a case of increase serial pulmonary function and strength tests.[2] The following figure is the examples of GBS patients with diaphragm paralysis.

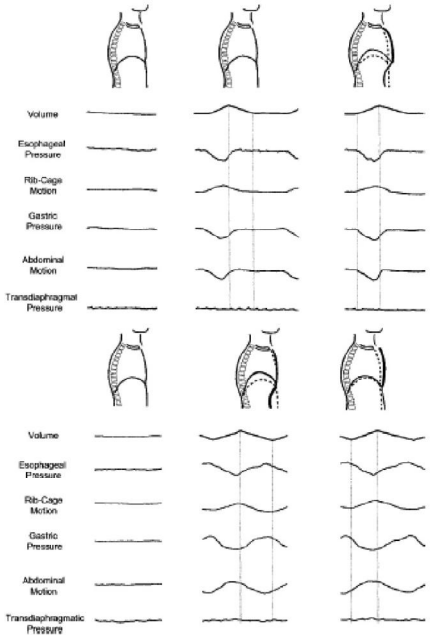

Figure 3. Case Representation[20]

Laghi F (2003) explains the difference in Figure 3 as follows: both GBS patients are apneustic (left image), expiratory (middle), and during inspiration (right). In the first patient, while apneustic, respiratory muscles and all other pressures are at the point of Functional Residual Capacity (FRC). We know that the FRC is the final state of expiration at rest. During expiration, the first patient has his breathing muscles in relaxation state; and volume lines, esophageal pressure, movement of the chest cavity, gastric pressure, abdominal movement and pressure and transdiaphragm will keep returning to relaxed position. At inspiration, muscles surround chest cavity contracts, esophagus shows a negative pressure, outward movement of the chest cavity and increase lung volume. Negative pressure also occurs in the diaphragm and abdominal at resting state, intrathoracic expands superiorly that causes negative pressure in gastric cavity and abdominal movement paradoxically. Transdiaphragm pressure remains unchanged. Whereas the second patient, an apnea patient has similar circumstances: relaxation state and at the point of FRC as in the first patient. When the expiratory muscles relax, chest wall move inward, abdominal muscle contracts and lung volume go under the FRC at the end of expiration. The lowest pulmonary pressure reduces the elastic recoil of esophagus and FRC point below the current state. During inspiration, chest cavity muscles contraction and move outward. Meanwhile, the expiratory muscles relaxation results in rapid rise of gastric pressure and the abdomen moves outwards (no paradoxical movement) whereas the transdiaphragm pressure remains constant.[20]

A case report by Narasimman S et al. recommends early physical therapy intervention for rapid recovery in GBS patients. Respiratory complications are often influenced by the acute phase of GBS, therefore early physical therapy is highly required when patients in ICU. The report indicates that patient with signs of tachypnea, grade 3 abdominal muscle strength, inspiratory mouth pressure of 30 cmH_2O, and cough and functional motor weaknesses, should be given immediate treatment on the second

day after hospital admission. The recommended treatment dose is 3 times daily of deep breathing exercise, segmental expansion exercise, assisted cough techniques, inspiratory muscle and active-assisted ROM of the extremities training. Inspiratory muscles training (IMT) is started with the resistance of 7 mm and the resistance is then increased gradually. Therapy sessions continued with the same dose until the next 3 days despite of the ongoing progression of the disease. Active ROM exercises are performed every day for 3 sessions, followed by an effective coughing exercise along with the progressive IMT. At day 6, mild strengthening exercises are initiated with minimal resistance in a sitting position height. Examination of inspiratory muscle strength using maximal inspiratory pressure (MIP) shows mild improvement ($40cmH_2O$), the abdominal muscle and diaphragm strengths on grade 3-4. Patient still undergoes 3 times daily of therapy as the last 5 days and remains under supervision. On the 7^{th} day, training sessions continue as before with strengthening exercise, IMT exercise, and assisted cough exercise.[21]

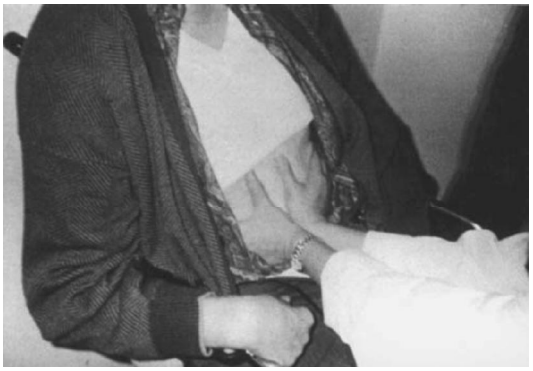

Figure 4. Assisted Cough Techniques[14]

In the second case report, Narasimman S, et al. described a patient who was immediately treated in the ICU with ventilator after admission, with clinical manifestations of tachypnea, sputum retention, areflexia, decrease motoric function, grade 3 abdominal muscle strength, the mouth inspiratory pressure of $30cmH_2O$, and cough weakness. Management delivered were postural drainage, percussion, vibration and suction also passive ROM exercises during the acute phase. Post-intubation hemodynamic was stable, followed by bed reclining and gradually increased up to sitting position (leaning). It is performed 3 times a day. If intubation and sputum retention occur, postural drainage, gentle suction with deep breathing and active ROM exercise were given. Post extubation, MIP was measured through an Endotracheal tube (ETT).[21]

Figure 5. Inspiratory Muscles Training (IMT)[21]

According to the explanation above, early therapy is important in GBS recovery and inspiratory muscle training shows to be effective. Lack of inspiratory muscle strength in any neuromuscular disease will cause alveolar ventilation reduction, micro-atelectasis and ventilation perfusion changes. The inspiratory muscles weakness will change the thoracic compliance, which in turn increase the work of breathing. Respiratory muscles strengthening is proven to increase isometric pressure generated by inspiratory muscles (PiMax). With these physiological principles, patients were given non-resistance training of threshold IMT. IMT is aimed at increasing inspiratory muscles strengths thus the training intensity should be adjusted. On the first case, IMT is considered as a precaution to prevent mechanical ventilation. If the patient is cooperative, IMT is recommended. On the other hand, the second case shows that IMT is impossible to begin immediately after initial physical therapy evaluation due to respiratory failure. PiMax is measured using a simple manometer and significant PiMax increase will be recorded. IMT is considered as a stage of pulmonary rehabilitation in GBS.[21]

Rabinstein A. stipulates that hypercapnia, evaluated from arterial blood gases is the end phenomenon of the disease. However, mild hypoxia initially occurs as a consequence of micro atelectasis caused by diaphragm weakness. Patients in supine position tend to develop pulmonary atelectasis. This creates an imbalance between ventilation and perfusion and it is responsible for the occurrence of mild hypoxia. Nevertheless, this situation tends to be mild in the early stage of respiratory insufficiency. Meanwhile, oxygen saturation may remain within normal range and identified as the earliest evidence of hypoxia. In addition to the patient's thoracic expansion, as well as the intensive use of incentive spirometry and chest physical therapy.[19]

After respiratory muscle weakness reaches a critical point where hypercapnia develops rapidly followed by hypoxia, thus immediate intubation is required. Sudden development of hypercapnia may also cause aspiration, an obstruction by mucous or upper airway obstruction due to bulbar muscle weakness. Once the patient is intubated, mechanical ventilation should be initiated. The most widely used mode of ventilation in patients with GBS is Synchronized Intermittent Mandatory Ventilation (SIMV). This mode is convenient for patients, guaranteeing a minimum level of ventilation per minute and allows minimal respiratory assistances that are variable. Pressure support ventilation is often combined with SIMV to reduce the breathing effort during spontaneous breathing and to minimize ventilator desyncronization. At the beginning of mechanical ventilation, some patients feel more comfortable with the Assist-control mode. It is important for patients with GBS, especially those who require emergency intubation, to rest on the first 24 to 48 hours. To achieve this goal, breathing should be minimized by providing full mechanical ventilation support. Ventilation arrangements should include adequate levels of support pressure, Positive End Expiratory Pressure (PEEP) to prevent further atelectasis, sufficient up and down volume to achieve lung expansion. Tidal volume of 8-12 ml/kg is common in patients with neuromuscular respiratory failure.[19]

Steinberg J. suggests that if the respiratory muscles are weak, tracheostomy would be required and connected to a mechanical ventilator. Various methods are necessary to clear the lungs from secretions and prevent pneumonia. Mechanical ventilation support is continued until muscle strength is better.[22]

A study by Orsini M et al. describes four conditions of respiratory failure patients with GBS: (1) compromised upper airway because of oropharyngeal and laryngeal muscles weakness that raise the risk of aspiration since the swallow and secretion clearance are disrupted; (2) inspiratory muscles weakness (diaphragm, intercostals and accessory muscles) results in restrictive impairment (low compliance) and micro atelectasis that cause impaired ventilation and lead to hypoxemia; (3) expiratory muscles weakness that can reduce the ability of cough, increasing the risk of respiratory infections; and (4) pulmonary complications, such as embolism and pneumonia that are associated with other conditions. The following assessments are needed to evaluate the ventilation support and

endotracheal intubation, include Vital Capacity (VC), Maximum Inspiratory Pressure (MIP) and Expiratory Pressure (MEP), signs of respiratory muscles fatigue (dyspnea and chest muscles cramp). Early risk identification of respiratory failure is associated with the "role 20/30/40 (VC <20 mL/kg, MIP <30 cmH_20, MEP <40 cmH_20). Patients with GBS is unlikely being a candidate for the use of non-invasive ventilation because the protective mechanism of upper airway, cough are insufficient and often disturbed in severe conditions.[1]

Ventilation support influences the functional outcome significantly. The most important GBS prognostic determinant factor is the severity of muscle weakness. The requirement and duration of ventilator are strongly associated with poor motor recovery.[24] Azim A et al. (2013) found that 64% of GBS patients need prolonged mechanical ventilator usage, which was more than 14 days. They discovered the positive results of mechanical ventilator usage in early supportive care in conjunction with chest physical therapy to prevent atelectasis.[24]

Chatterjee A et al. (2009) showed the advantageous analytic results of clinical and electrophysiological data of 34 GBS patients due to higher ability to ambulate without assistance. For about 56% of patients were male with an average age of 41 years, and 34 patients were mostly below 30 years old. Weakness (50%) was the primary complaint. Mechanical ventilation was required in 41.2% of patients with 7.1% of mortality. Despite of the prolonged severe GBS recovery, independent ambulation was regained in majority (61.8%, p <0.001) .[23]

Pitetti KH elaborated those post-GBS patients with mild neurological deficits who were given endurance exercises with doses of 3 times a weeks for 16 weeks, 30 minutes in duration per session, 75-80% of intensity from maximum Heart Rate (HR). Around 9-11% showed improvement from the top decision-O_2 on Schwinn-water-dyne ergometer (SAE) & Bicycle ergometer (BE). Regarding the peak ventilation, for about 23% changes were seen in SAE & BE. The arm-crank ergometer (ACE) showed an increase of 16% in peak ventilation, with no improvement in aerobic capacity. Total capacity of working in the BE had improved as much as 29% post exercise. At the end of this study, significant improvement in ADL was significant.[25]

In a literature by Sendhikumar R et al. (2013) demonstrates improvement in sleep quality and functional ability of post-GBS patients by therapeutic yoga pranayama and meditation on post-GBS patients. The given dose was 1 hour per session for 3 weeks, 1 session per day, 5 days per week including yoga relaxation, pranayama (breathing exercises).[26]

Another study by Ted Burns stated that patients with history of fatigue for 6 months to 15 years after GBS were also improved after 12 weeks of cycling. The program had positive effect on fatigue, as well as anxiety, functional ability and quality of life. The exercise was performed in three sessions and supervised each week for 12 weeks. Each session consisted of 5 minutes of warming-up, 30 minutes of cycling, and followed by 5-10 minutes of cooling down. As the result, 80% of patients were motivated to continue exercising.[2]

Orsini M. assessed the effects on physical fitness, muscle strength and function, anxiety and depression, disability, and quality of life by static cycle exercise in 20 severe fatigue patients for about 12 weeks. 16 experienced a relatively good and stable recovery and 4 with chronic inflammatory demyelinating polyneuropathy (CIDP). There were significant changes and they were well tolerated, fatigue scores decreased by 20%. Physical fitness, functional and quality of life were increased.[1] Bussmann et al., also provided a 12 weeks physical exercise program in patients with severe fatigue after GBS. Improvements in physical fitness, mobility, physical function and mental patients were obtained. An incentive training of spirometry was given to support the recovery process for it was believed to be able to clear the airway.[27]

Another study highlighted the improvement of muscle strength assessed by isokinetic and isometric exercises and the functional capacity assessed by Functional Independence Motor (FIM) in 6 patients with GBS. They were given extremity muscle strengthening exercises for about18 months after the onset. Muscle strength of knee extensors showed significant

improvement statistically during the first 6 months. In 6-18 months, muscle strength was improved gradually and statistically significant. Within 18 months post onset patients recovered completely. The exercise was also recommended within 24 months post onset in order to keep muscle strength. [27]

CONCLUSION

GBS patients with respiratory dysfunction should be immediately rehabilitated and obtained a specific program. Majority of patients are able to return to normal with different recovery period and rehabilitation therapy to gain support.

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