Guillain-Barré Syndrome - a patient guide and nursing resource

By Michael Kehoe

Abstract
Guillain-Barré Syndrome (GBS) is an illness characterized by acute neuromuscular paralysis. A review of the history, course of the disease, current treatments, and nursing interventions, as well as excerpts from a patient teaching guide developed by the author for patients with GBS is included in this paper. The objectives are to present information about GBS, first at a level of understanding appropriate for patients and their families, and then to provide a more indepth discussion for health care providers. Despite the potential severity of GBS, the expected outcomes are encouraging, GBS affects 1-2.73 individuals per 100,000/year (Hahn, 1998). The symptoms can range from numbness and tingling with mild weakness to total paralysis requiring mechanical ventilation. Once diagnosed, patients are usually treated with intravenous immune globulin (IV Ig), which significantly reduces the duration of the illness (Hughes, 1997; Guillain-Barré Syndrome Study Group, 1985). Neuroscience nurses can make a difference in the recovery of their patients by anticipating potential complications and attending to their special needs during the acute and recovery phases of their illness. Aside from physical care, being able to support and teach the patient and family about GBS is crucial. Use of a patient and family teaching guide is one strategy for providing education and support.

Introduction
The first time the majority of patients hear the words Guillain-Barré Syndrome (GBS) is when they receive the diagnosis. This is the beginning of their quest for information about the syndrome. After their initial contact with the admitting physicians, a major source of information will be the nursing staff providing their care. Patients and their families are scared and in need of answers. Much of the information available is of a technical nature, written at a level beyond the comprehension of many patients, and fails to address the questions of patients and their families. At the stressful time of diagnosis, a simplified source of information that would provide a starting point for answering their questions was needed. This resulted in the development of the “Patient Guide, Guillain-Barré Syndrome” for the Queen Elizabeth II Health Sciences Centre in Halifax, Nova Scotia.

It is not the intent of this author to engage in an indepth discussion of guidelines for developing patient teaching information, as that is beyond the scope of this paper. However, it is worth making explicit the process that was followed in developing the “Patient Guide, Guillain-Barré Syndrome” for the Queen Elizabeth II Health Sciences Centre. The hospital’s patient and family learning centre provides a handbook entitled: “Developing & Revising Patient/Family Education Print Material”. This guide outlines the steps to take in the development of educational materials. It is stated in the guide that: “When used effectively, print materials can help staff maximize limited teaching time and enable patients to better manage their health. It is important to remember that print materials are only a tool for accomplishing an overall

Résumé
Le syndrome de Guillain-Barré (SGB) est une maladie qui se caractérise par une paralysie neuromusculaire sévère. Vous trouverez dans cette présentation une revue de l’histoire de la maladie, le processus de développement, les traitements utilisés, les interventions en nursing ainsi que des extraits du guide d’information pour le patient atteint du SGB, tel que développé par l’auteur. L’objectif est de procurer au patient et à sa famille des renseignements compréhensibles sur le syndrome de Guillain-Barré et de plus, fournir aux professionnels de la santé une information plus approfondie. Malgré les effets potentiellement néfastes de cette maladie sur les patients, nous prévoyons maintenant obtenir des bons résultats suite au traitement. Le SGB atteint une à deux personnes sur 100,000 par année (Hahn, 1998).

Les symptômes peuvent se manifester sous forme d’engourdissements, de picotements ou de faiblesses légères jusqu’à la paralysie totale. Dans certains cas, il est nécessaire d’avoir recours à la ventilation artificielle. Lorsque le diagnostique est établi, les patients sont généralement traités avec une solution d’immunoglobuline (IG, i.v.) administrée par voie intraveineuse, laquelle réduit de façon importante la durée de la maladie (Hughes, 1997; Neurology, 1985). Les infirmières en neurosciences peuvent influencer le rétablissement de leurs patients en surveillant l’apparition possible de complications et en prodiguant les soins appropriés de la phase critique à la phase de rétablissement. En plus des soins au patient, la capacité de donner du support et de l’information à la famille du patient est d’une grande valeur. L’utilisation du guide de renseignements pour le patient et sa famille est un bon moyen d’atteindre cet objectif.
The maximum degree of weakness is reached in less than eight cases, the patient will be placed on a ventilator. If the condition starts in the feet and works its way up. In the most severe cases, a group of common causes and symptoms. It is diagnosed by (Fanion, 1998; Hahn, 1998). It is called Guillain-Barré Syndrome.

The patient education coordinator reviewed the initial draft and applied a readability formula to ensure that the material was written at a grade six level and conformed to the hospital guidelines. The guide was then reviewed by fellow staff nurses, the nurse educator, patients with GBS, their families, and finally by the head of the neurology department. Each review resulted in revisions until the final version was reached. The resulting pamphlet provides basic information and answers to common questions patients and their families are likely to ask their caregivers.

It is essential that nurses have a thorough understanding of Guillain-Barré Syndrome prior to utilizing the pamphlet to teach patients and their families. Throughout the remaining sections of this paper, a review of GBS is presented for nurses. Text boxes are used to highlight information in the patient guide as an introduction to each topic.

What is Guillain-Barré Syndrome (GBS)?

It is an illness of the nervous system. The symptoms range from mild weakness to severe paralysis. Most people have never heard of this syndrome until someone they know or they have been diagnosed with it. This can be a very frightening time for you and your family. This guide provides basic information to help you understand what is happening and what to expect while in hospital.

Since the introduction of the polio vaccine, GBS has become the most common cause of acute neuromuscular paralysis (Fanion, 1998; Hahn, 1998). It is a syndrome that occurs before the specific disease-causing agent is known. It is diagnosed by a group of commonly occurring signs and symptoms. Classically, GBS is an ascending symmetrical paralysis that starts in the feet and works its way up. In the most severe cases, the patient will be placed on a ventilator. If the maximum degree of weakness is reached in less than eight weeks, it is GBS. In 90% of the cases, maximum weakness will be reached within four weeks. Those with symptoms lasting longer than eight weeks are classified as suffering from chronic inflammatory demyelinating polyneuropathy (CIDP), and are no longer considered to have GBS (Barohn, 1998).

GBS affects the peripheral nervous system (PNS), which is composed of 31 pairs of spinal nerves arising from the spinal cord and 12 pairs of cranial nerves that originate in the brainstem, except the first nerve that originates in the olfactory bulb. The cranial nerves carry impulses to and from the brain. Peripheral nerves that send information toward the central nervous system (CNS) are afferent or sensory nerves. Those that transmit information from the CNS are efferent or motor nerves. The efferent system is subdivided into the somatic nervous system (SNS) that conveys information to the skeletal muscles and the autonomic nervous system (ANS). The ANS conveys information to smooth muscles, cardiac muscles, and glands (Phipps, Long, Woods, & Cassmeyer, 1991; Tortora & Anagnostakos, 1990).

GBS is thought of as an acute inflammatory demyelinating polyradiculoneuropathy (AIDP). AIDP makes up 85-90% of all GBS cases in western countries (Hahn, 1998). The autoimmune response is usually triggered by a bacterial or viral infection. The antibodies produced in response to the infection cause segmental demyelination of the peripheral nerves. Macrophages penetrate the Schwann-cell surface membrane, leading to the breakdown of the myelin sheath (Murray, 1993). The unprotected nerve becomes inflamed, resulting in a slowing of nerve conduction and conduction block, causing muscle weakness and paresthesiae. The demyelination of nerve fibres is self-limiting; once the immune reaction has stopped remyelination occurs in reverse, from top to bottom (Worsham, 2000). In severe cases, the inflammation and swelling caused by the demyelination can lead to secondary damage to the axon. The degree of axonal loss has a direct bearing on the prognosis for recovery (Hahn).

Another variant to classic GBS is acute motor-sensory axonal neuropathy (AMSAN). It is associated with the sudden onset of paralysis within two to four days of the onset of symptoms following an episode of a diarrhea or flu-like illness. The paralysis is the result of an autoimmune attack occurring at the motor and sensory nerve axons, with little demyelination of the peripheral nerves. There is also another axonal variant known as acute motor-axonal neuropathy (AMAN). It is strongly associated with Campylobacter jejuni (C. jejuni) infections. In these cases, axonal degeneration occurs primarily at the motor-nerve terminals, with normal action potentials being seen in the sensory nerves. Certain strains of C. jejuni have been associated with the Miller Fisher Syndrome (MFS) variant. The antibodies produced affect the nodal regions of the oculomotor nerves, the dorsal-root ganglion cells and cerebellar neurons (Hahn, 1998).

The incidence of GBS was 1 to 2.73 per 100,000/year during 1991-93 (Hahn, 1998). Males are at a slightly higher risk than females, at a ratio of 1.5:1. GBS is uncommon in children under the age of two, which may be associated with an immature immune system. It is rare that someone will have a
recurrence of the syndrome, although it occurs in 2-5% of cases. There are bimodal peaks with respect to age. The first peak occurs between the ages of 15-35, the second between 50-75 years of age (Fanion, 1998). The incidence in those 70 and over was 8.6 per 100,000 individuals (Hahn). A review of 98 patients admitted to the QEII from 1993-1999 yielded similar information. Males had a higher incidence than females with a ratio of 1.33:1. Table One, which displays the incidence according to age at QEII, reflects the bimodal age peak, with those over the age of 55 making up 46% of all admissions.

The first written description of the key clinical features of GBS is attributed to a French scientist named Jean-Baptiste-Octave Landry in 1859. His superior had diagnosed a patient, who had presented with vague sensory changes and mild weakness, as being hysterical. The patient died after developing respiratory difficulties as Landry had predicted. Landry later found several other reports of similar cases in the literature. These patients were then said to be suffering from “Landry’s ascending paralysis” (Winer, 1995). The function of the peripheral nerves was not understood at this time, so he believed that the problem was arising within the central nervous system.

In 1916, three French doctors, Georges Guillain, Jean-Alexandre Barré, and André Strohl, wrote a report describing paralysis in soldiers after the Battle of the Somme. They recorded a delay in muscle reflex movements, and that the cerebral spinal fluid (CSF) showed elevated protein levels without an increase in white cell count. They concluded that the primary problem involved the peripheral nerves. The syndrome was called Landry-Guillain-Barré-Strohl syndrome, but it was too lengthy and simply became known as Guillain-Barré syndrome (Winer, 1995).

### What causes Guillain-Barré Syndrome?

At present, the cause is not known. It often appears several days to weeks after a viral infection. The body’s own defence against infection, the immune system, turns against itself and starts to attack the protective covering around the nerve fibres. As the disease progresses, the speed at which signals travel along the nerves becomes slower. This results in weakness that may lead to paralysis. There is no evidence that it is contagious, even to people exposed to the same viral infection.

There are a number of potential events that can precede the onset of GBS. An upper respiratory infection with + cytomegalovirus (CMV) titers (18%) is more common in younger patients and results in more sensory loss and cranial nerve involvement. In this group, respiratory insufficiency is more common, affecting up to 65% of patients. They may also take longer than average to regain independent locomotion. GBS following gastrointestinal infections generally occurs with + C. jejuni titers (28%) (Neuromuscular, 2000). C. jejuni infection is the most common cause of gastroenteritis in the developed world. It is associated with predominantly motor involvement and a more severe outcome. Estimates indicate that one in 1058 persons will develop GBS following infection with C. jejuni; the rate increases to one in 158 with serotype O19 (NIAID Workshop). Other infections that are possible precursors to GBS include Epstein-Barr virus, HIV, varicella zoster virus, and Mycoplasma pneumoniae (Hahn, 1998). There have been isolated cases reported following surgery, hepatitis A infection, and during the first two weeks of the postpartum period (Neuromuscular, 2000).

Outbreaks of GBS have been associated with vaccinations. The most notable event that occurred in North America was during the 1976-1977 swine flu vaccination initiative. It caused an additional 10 cases per million individuals vaccinated. This has resulted in an over-estimation of the increased chance of developing GBS following the influenza vaccination. Studies of the 1992-1993 and 1993-1994 influenza vaccines have shown that there may be slightly more than one additional case of GBS per one million persons vaccinated. Thus the benefits versus the risks associated with vaccination in a patient with a pre-existing neurological illness must be carefully weighed. Vaccination of patients with CIDP may be contra-indicated and should only be considered if other underlying disease processes are at work (Lasky et al., 1998). A review of the admission dates at the QEII between 1993 and 1999 indicates a possible seasonal component to the incidence of GBS, with 30% of admissions occurring in May and June. This data is shown in Table Two. However, this increase is too remote from the key influenza vaccination time of September and October to have been a direct precursor to GBS at QEII.

### What are the symptoms of Guillain-Barré Syndrome?

- The first symptom may be a feeling of numbness before any weakness is noted. This usually starts in the feet and legs but can also start in the head and arms. Both sides of the body are affected at the same time.
- The symptoms can occur rapidly over a few hours or slowly over several weeks.
- There may be pain in the legs and lower back.
- There may be shortness of breath if the breathing muscles are affected.

In 50% of patients, the first symptoms of GBS are sensory. Paresthesiae, such as numbness, prickling, burning and tingling, will be experienced to varying degrees by 70% to 90% of patients during the course of their illness (Neuromuscular, 2000). This is the result of demyelination of
the sensory fibres of the PNS. Typically, these sensory symptoms are followed closely by a progressive symmetrical ascending weakness starting in the lower extremities. The weakness is caused by a delay in the conduction velocities of the motor fibres as demyelination occurs. If the course of GBS leads to a total conduction block, it will result in paralysis (Murray, 1993). In extreme cases, peak weakness may be reached within several hours of the onset of symptoms, although for most it is reached within two weeks. In other cases, the ascending weakness may continue for up to four or five weeks. The severity of the weakness can vary from mild leg weakness to quadriplegia requiring ventilation (Fanion, 1998; McLeland, 1995; Neuromuscular, 2000). Pain in the lower back and buttock is also a common complaint, as well as pain in the shoulders and thighs (Fanion).

The cranial nerves (CN) are involved in 45% to 70% of cases. The facial nerve CN VII is the most commonly involved and can lead to facial weakness. In the early phases, the facial weakness is symmetrical and may become asymmetrical even if other weaknesses are resolving (Neuromuscular, 2000). Involvement of other cranial nerves can lead to dysphagia and dysarthrias (Fanion, 1998). In 10% of cases, there will be paralysis of the extraocular muscles causing ptosis and double vision (McLeod, 1995). Some patients develop acute opthalmoplegia (eye paralysis), sluggish pupillary reflexes, ataxia and areflexia (Hahn, 1998). This is the MFS variant of GBS. MFS often mimics a brainstem lesion. Patients with a pure form of MFS do not experience generalized weakness. In many cases, they develop neuropathies that overlap with GBS (Guillain-Barré Syndrome Support Group of the United Kingdom, 1999).

**How is the diagnosis made?**

- The doctor will check for changes in reflexes, sense of touch and muscle strength.
- An EMG (electromyograph) will show how well the nerves conduct signals.
- If needed, a sample of spinal fluid may be taken during a lumbar puncture to measure the protein level.

GBS is a potential medical emergency. Accurate and timely diagnosis is essential, but difficult due to the variety of symptoms, especially in the early stages. Symptoms are often blamed on anxiety when apparently healthy patients describe vague sensory disturbances. This is particularly true of patients who have a slow progression of the syndrome. The diagnosis is more readily made in those who develop a sudden onset of symmetrical weakness.

The history and physical examination are the first steps in arriving at a diagnosis. Many patients report a recent history of viral infection or gastrointestinal illness. This can be followed several weeks later by sensory changes and/or symmetrical weakness. In 70% of GBS patients, deep tendon reflexes, such as knee jerks, are absent or greatly reduced. There is a progressive loss of these reflexes over the first week. The biceps jerk is the least likely to be affected (Neuromuscular, 2000). Tests for muscle strength show weakness, while tests of sensation reveal deficits.

A lumbar puncture can be performed to analyze the cerebrospinal fluid. Guillain and Barré, as part of their diagnostic procedure, first used this. In 90% of GBS patients, there is an elevation in CSF protein greater than 400mg/L with no increase in white cell counts (Fanion, 1998; Worsham, 2000). Within the first and second days of onset, 85% of the patients will show normal levels of CSF proteins. However, within the first and second week of onset, 66% and 82% of the patients, respectively, exhibit elevated protein levels (Neuromuscular, 2000). Protein levels will reach their peak in four to six weeks and this is thought to be due to a release of plasma proteins from the inflammation, degeneration and damage to the nerve roots (Doenges, 1988).

Nerve conduction studies can be performed to test for a measurable slowing of nerve
conduction velocities, prolongation of distal and F-wave latencies and conduction block (Hahn, 1998). It may take two to three weeks for the conduction changes to develop (Fanion, 1998). Electromyography (EMG) is used to record muscle activity. In GBS patients, single muscles are activated rather than groups of muscle fibres (Worsham, 2000).

Patients should be admitted to hospital when GBS is suspected. Patients can then be monitored for rapid onset of respiratory paralysis and autonomic changes, and can then receive early treatment to help shorten the duration of the illness.

How will it be treated?

You may be treated with one or both of the following treatments:

• Intravenous immunoglobulin (IVIG). This is a product that concentrates the part of the blood that fights infection (antibodies). It is usually given over a five-day period and may need to be repeated.

• Plasmapheresis. This is a process that withdraws some of the patient’s blood and removes the antibodies before the blood is returned to the patient.

Over the years, a number of treatments have been used with varying degrees of success. One of the first treatments used by Landry in 1859 was injection of strychnine, which proved to be unsuccessful. Later Guillain and Barré thought that they had had some success by providing their patients with “chops and Bordeaux wine” as several patients responded well and improved (Winer, 1995). Neither of these treatments is used today. Since GBS is an autoimmune disease where inflammatory cells cause myelin and/or axon damage, treatment with corticosteroids has been tried. Studies have indicated that corticosteroids alone are not effective in treating GBS (Hahn, 1998), but are a useful treatment in patients with CIDP (Steinberg; Barohn, 1998).

To date only two treatments, through randomized trials, have proven successful in reducing the length and severity of GBS (Hughes, 1997). The first treatment used successfully was plasmapheresis. It is a process that uses centrifugation of the patient’s blood to remove some of the white cells and abnormal proteins. The plasma exchange of four or five treatments is carried out over a seven- to 10-day period. It has been shown to reduce the recovery time by up to 50% (Fanion, 1998; Guillain-Barré Syndrome Study Group, 1985). Similar results have been achieved with the use of intravenous immune globulin (IV IG) (Hughes; Neuromuscular, 2000). IV IG is a concentrated blood product of the immunoglobulin IgG. It is the major antibody in the secondary humoral response of immunity. The treatment dose is typically 0.4g/kg/d given daily for five days (Fanion; Hahn, 1998; Hughes). With either treatment there is a 10% chance that the symptoms will worsen within one to two weeks of the initial treatment and a second course may be required (Hahn).

The attending physician will determine which treatment will be best for the patient. IV IG is available in areas that lack access to plasma exchange and can be administered to patients with poor venous access. The most common side effects include chills, fever and headache. They are mild and transitory and can be eliminated by slowing the rate of infusion. IV IG has been reported to be associated with renal dysfunction, acute renal failure and osmotic nephrosis. IV IG should be avoided in patients predisposed to acute renal failure. This would include those with “pre-existing renal insufficiency, diabetes mellitus, age greater than 65, volume depletion, sepsis, paraproteinemia, or patients receiving known nephrotoxic drugs” (Bayer Inc., 2000). Plasmapheresis is used with patients having good venous access and/or patients with a history of allergic reactions to IV IG. It is the preferred treatment for pregnant patients, and patients suffering from either congestive heart failure or renal insufficiency (Neuromuscular, 2000). Controlled trials have shown that the benefits of plasma exchange are ineffective when started later than two weeks from onset of symptoms (Hahn, 1998).

Your hospital stay

Depending on the severity of your illness and how fast it progresses, you may need to be admitted to either the neurology unit or intensive care unit. It is not unusual for the person with Guillain-Barré Syndrome to experience fear, anxiety and/or depression. These are all normal feelings and the nurses are there to help you work through them on your way to recovery. Sometimes the symptoms will get worse before they get better. You will be observed for:

• Changes in heart rate and blood pressure.

• Increasing paralysis from just the legs to the whole body. In severe cases a machine that breathes for you (a ventilator) may be needed until you are able to breathe on your own again.

• Problems passing urine. A tube may be needed for a short time to drain your bladder.

• Trouble swallowing. You may need to eat food that is easier to swallow. In severe cases, a feeding tube may be needed until you can eat on your own.

• Difficulties having a good bowel movement (constipation). Laxatives and stool softeners may be ordered.

During your hospital stay you and your family will have the support of a number of health professionals.

Nurses play an important role in the care and management of GBS patients. Their ongoing assessment skills are used to revise the patient care plan and modify it according to the changes in the patient’s condition. Being aware of potential complications associated with the progression of the disease and close observation of the GBS patient can help avoid a potential crisis. Nursing priorities are to maintain and support respiratory function, fluid and electrolyte balance, body function, nutrition, and elimination. Nurses also assist the patient and significant others in dealing with psychological aspects of the situation (Doenges, 1988).

Respiratory insufficiency can develop as quickly as 24 to 72 hours after the initial onset of neurological symptoms.
There is a high risk of respiratory failure related to progressive weakness or paralysis of the intercostal and diaphragmatic muscles. Interventions include assessing airway patency, breath sounds, respiratory rate and effort, skin colour, chest movement and serial vital capacities every two hours (Holloway, 1993). An early indicator of the approach of respiratory failure is ascending losses of sensation to light touch from the iliac crest up to the shoulders. This is usually followed by muscle weakness. If sensation loss is at the level of the T8 dermatome, anticipate intercostal muscle involvement. Also monitor for upper arm and shoulder weakness that often precedes respiratory failure (Swearingen & Ross). The nurse should watch for an increase in heart rate, a change in mental status, a respiratory rate greater than 30 breaths per minute along with paradoxical movement of the chest wall and abdomen (Murray, 1993). Forced vital capacities should be monitored on a regular basis. If the vital capacity is less than one litre, the patient should be admitted for observation in an ICU as their condition can deteriorate rapidly. (Tintinalli, Kelen, & Staplzynski, 2000). Intubation is indicated if the vital capacity is less than 12-15 ml/kg, if hypoxia develops with a PaO2 < 80 mm Hg or if the patient has difficulty with secretions. Approximately 33% of patients will require intubation (Neuromuscular, 2000).

An aspect of GBS that may not receive the attention it is due is autonomic dysfunction. With the improvements in care of patients on ventilators, autonomic cardiac abnormalities are a major cause of death in GBS patients (Fanion, 1998). Nurses must be aware that patients with GBS display markedly labile vital signs. They can experience cardiac arrhythmias, bradycardia, tachycardia, hypotension, hypertension and hypothermia. The blood pressure fluctuations may be caused by unopposed sympathetic outflow or loss of outflow to the PNS causing changes in vascular tone (Swearingen & Ross, 1999). The same patient can experience each of these at different times during the course of the illness. Due to the lability of the autonomic abnormalities, short-acting treatments are used. Patients with second and third degree heart block may require temporary pacing (Fanion, 1998). Many patients experience tachycardia, which is not usually treated but may act as an early warning sign of more life-threatening autonomic dysfunction. Tachycardia may also be caused by involvement of cranial nerve X (vagus) due to loss of parasympathetic inhibition (Murray, 1993). About 20% will develop more serious cardiac arrhythmias (Parry, 1998).

Mild hypoxia can induce or exacerbate cardiac arrhythmias and may be the first signs of respiratory failure. Intubation itself can cause arrhythmias, thus care should be taken to avoid emergency intubation. If possible, the use of Succinylcholine, a muscle relaxant commonly used in preparation for intubation, should be avoided as it can induce cardiac arrhythmias (Parry, 1998). Some findings suggest that skin sympathetic nerve activity in GBS patients during the acute phase may contribute to some of the autonomic nerve symptoms (Yamamoto, et al, 1997). A less life-threatening autonomic dysfunction involves the bladder resulting in urinary retention. Studies indicate that up to 25% of GBS patients will develop some form of micturitional disturbance. They tend to be more common in patients with a severe weakness (Sakakibara, et al 1997). Care should be taken when assisting patients to void at the bedside as urinary hesitancy combined with orthostatic hypotension and leg weakness can lead to falls.

Patients need to be assessed for cranial nerve involvement, which can lead to a number of complications. The earliest sign of cranial nerve involvement is an inability to wrinkle the forehead, close the eyes or smile. This indicates that CN VII (facial) has been affected. The potential for aspiration and risk for nutritional deficits arises if CN IX (glossopharyngeal) and XII (hypoglossal) are involved. Patients will exhibit slurred speech, a loss of taste and dysphagia. The patient will also have difficulty chewing if cranial nerve V (trigeminal) is affected. If CN X (vagus) is involved there may be difficulty with mucociliary clearance (Murray, 1993). Bedside swallowing assessments should be conducted as the patient’s condition changes to reduce the chance of aspiration by selecting the appropriate consistency of their diet. If the patient is unable to eat because of cranial nerve involvement or an endotracheal tube is present, enteral feeding will be required. Either a nasogastric or gastrostomy tube can be used to deliver a high-protein, high-calorie enteral formula to maintain basal metabolic rate and prevent muscle wasting. If the patient is unable to tolerate enteral feeding, then total parenteral nutrition will be required (Worsham, 2000).

Pain is often underestimated and under-treated while more life-threatening problems are dealt with. Some degree of pain has been reported in up to 80% of patients (Parry, 1998). The pain is often described as a deep aching or cramping in the lower back, hips, buttocks and shoulders. It may become a stabbing pain with movement. In many patients, it is a minor discomfort. In those with a more rapid progression of the syndrome, the pain may become severe. For patients on a ventilator, it is very important that they be asked about their level of pain and be treated accordingly. As patients recover, the type of pain may change to a burning, stabbing or shooting pain that responds poorly to narcotic analgesics. They may be hypersensitive to even the lightest touch. This neuropathic pain may respond better to medications like amitriptyline or carbamazepine. Patients must be made aware that these drugs take time to be effective and may not totally relieve their pain. The pain can also interfere with rehabilitation, as exercise and weight-bearing may exacerbate the symptoms (Parry). Nurses can help the patient cope with pain by assessing the need for analgesics on a regular basis. Frequent repositioning, every 30-45 minutes for some, can decrease muscle tension and fatigue (Worsham, 2000). Passive range of motion can help reduce muscle stiffness. Patients with muscle tenderness may respond well to moist heat or warm baths. If the patient can tolerate light touch, then massage can be considered (Swearingen & Ross, 1999).
The syndrome of inappropriate antidiuretic hormone (SIADH) can occur in GBS patients (Fanion, 1998; Holloway, 1993). It results in a total body water increase because of water retention. Unlike congestive heart failure, edema does not occur, as the volume expansion results in natriuresis and hyponatremia. Stressors such as fear, acute infections, pain and anxiety can bring on SIADH, all of which can be experienced by the GBS patient. Daily monitoring of serum electrolytes can identify changes in sodium levels. Water restriction, as low as 500 cc per day, is the first step in treating SIADH. Nurses can help by monitoring daily intake of fluids, spacing them out over the day, offering ice chips to relieve thirst, and providing frequent mouth care (Phipps et al., 1991).

GBS patients require care similar to other patients with mobility problems. It can be expected that up to 30% of patients will progress to quadriplegia and another 30% will be bed-bound during their admission (Neuromuscular, 2000). Many of the complications of immobility can be prevented by informed nursing care. The chance of thrombosis from venous pooling can be reduced by the use of antiembolism stockings and/or the administration of low dose heparin as ordered. Avoid the use of knee gatch, leg crossing or pillows placed directly under the popliteal area (Holloway, 1993).

Immobile patients are at an increased risk for impaired skin integrity related to impeded capillary flow, altered sensation or venous stasis. Turn and reposition the patient every one to two hours using careful turning technique. When providing care, use lotions sparingly, avoiding harsh soaps, and clean and dry the skin thoroughly and gently. In some instances, the situation can be complicated by nutritional deficits, obesity, diabetes mellitus, and incontinence. Additional precautions may include the use of convoluted foam mattress, sheepekins, air mattresses or kinetic beds as the condition indicates (Holloway, 1993). The risk of contractures and muscle atrophy can be reduced by range of motion exercises (ROM). These can be active to the extent that the patient is able to participate, or passive (Murray, 1993). ROM can be combined with the patient’s regular turning schedule.

The nurse should determine what the patient’s normal bowel elimination pattern is and develop a bowel routine to maintain regular function and avoid constipation. This can be achieved by encouraging adequate fluid intake, unless contraindicated, and the use of a high-fibre diet as tolerated. Aids such as laxatives, suppositories, enemas and stool softeners may be prescribed on consultation with doctors (Holloway, 1993). The potential for constipation increases with progressive weakness, ANS involvement, and the use of narcotics for pain control.

As GBS progresses, many patients feel increasingly powerless and display ineffective coping skills. Nurses help their patients cope by providing emotional support and frequent factual information about the course of the disease. Prepare the patient and family for potential problems of the acute phase. Emphasize that the effects of GBS are temporary and explain the potentially reversible nature of the disorder. Patients can be overwhelmed by the sudden loss of independence and the total reliance on others for their every need. It is not unusual for patients to react with feelings of anger, depression, grief, and even paranoia. Encouraging ventilation of feelings can facilitate healthy coping behaviour. Cultivating an attitude of acceptance of these feelings helps to achieve this goal. Manipulative behaviour may be an attempt to regain some control over their situation. It is important to allow the patient to make choices regarding care whenever possible. Allowing choices, even in small matters, can increase a patient’s sense of self-control (Holloway, 1993). Besides dealing with the course of the disorder itself, there are other stressors. Financial stressors can be caused by loss of income, loss of employment, the potential need for a career change, or inadequate insurance to cover the costs of care and rehabilitation. Family dynamics can be affected by separation and role changes (Murray, 1993).

Nurses can assist by identifying the patient’s special needs and ensuring that appropriate referrals are initiated. Social workers may be able to intervene to help resolve or lessen financial and social concerns. Psychologists assist the patient and family to deal with the emotional reactions resulting from the suddenness and severity of the illness. A recent study in the Netherlands utilizing the Sickness Impact Profile looked at how GBS affected the psychosocial factors of communication, alertness behaviour, emotional behaviour and social interaction. Many patients can experience some degree of psychosocial dysfunction even three to six years after the illness, no matter its severity or residual effects (Bernsen, Jacobs, de Jager, & van der Meche, 1997).

One question most patients will ask is, “How long will I be here?” There is no simple answer to this question as each case is unique. A review of the QEI data indicates that 57% of patients are discharged from the acute care facility in less than three weeks (Table Three). The longest stay was 445 days, with two others lasting 206 and 240 days.

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<th>Weeks in acute care</th>
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Table Three: Length of stay 1993-1999
Rehabilitation

Rehabilitation begins while you are in hospital. A physiotherapist will work with you to help maintain and strengthen your muscles. An occupational therapist will help you to resume normal activities such as bathing and dressing. Depending on the individual case, some may require additional recovery time at the Nova Scotia Rehabilitation Hospital.

Rehabilitation requires a team approach involving nurses, speech language pathologists (SLP), physiotherapists (PT), occupational therapists (OT), psychologists and doctors. The PT and OT will work to strengthen the body and regain maximal use of weak muscles to perform activities of daily living. The SLP will plan exercises to help improve speech and the ability to swallow. Nurses are involved in all aspects of care and coordinate and implement the restorative care plan 24 hours a day, seven days a week.

The rehabilitation process begins in the acute care setting. In severe cases, the treatment may start with passive range of motion exercises to help maintain flexibility. Strength usually returns in the opposite direction that the weakness occurred. If there was an ascending paralysis, strength will return in a descending pattern with the arms showing improvement before the legs. Moderation is advised with respect to exercise. Pushing to fatigue is not recommended due to the damaged nerves. Excessive exercise may result in cramps, pain and early exhaustion.

Prognosis

The prospects for recovery are good. A full recovery can be expected in 50-95% of patients with 10-40% experiencing some neurologic sequel (Fanion, 1998; Steinberg, n.d.). Patients who experience a slow or incomplete recovery may have either primary or secondary axonal damage. The return of motor function is dependent on the motor nerve regenerating at a rate of 1mm/day and reaching its target muscle (Ho, 1998). The recovery period can take from six months to two years. The mortality rate is between 5-10%. Death is usually related to a severe autonomic instability, and complications from prolonged intubation and paralysis (Fanion, 1998).

A recent British study showed that after one year 62% of the patients had made a complete or almost complete recovery; 17% were able to mobilize independently but unable to run; 9% required assistance in walking; and 4% were bed-bound or ventilator-dependent. The 8% of patients who died were all over 60 (Rees, Thompson, Smeeton, & Hughes, 1998).

Conclusion

GBS is a potential medical emergency. Patients newly diagnosed with GBS require close observation for changes in
respiratory status and autonomic dysfunction. Patient outcomes for survival are promising, but there is a degree of morbidity and mortality associated with the syndrome. Neuroscience nurses make a difference in patient outcomes by anticipating potential complications, by educating and supporting the patients and their families, providing support and encouragement, and attending to the physical and emotional needs of patients diagnosed with Guillain-Barré Syndrome.

About the author

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References


