

# Examining guillain barre syndrome



**ASSIGN  
BUSTER**

Guillain Barre Syndrome (GBS) is a rare immune mediated polyneuropathy that occurs in previously healthy individuals. The purpose of this paper is to provide readers with an understanding of Guillain Barre and conflictions GBS has with other medical resources and diseases. Included in this research paper are topics on origin, symptoms, treatments, medications and conflictions of medical resources with Guillain Barre.

Guillain-Barre Syndrome is an acute autoimmune disease that changes the peripheral nervous system and less commonly the motor or cranial nerves. GBS is random producing no warning and is an inflammatory condition that can lead to progressive muscle weakness and paralysis. It is a very rare sight in emergency departments and differentiating its early stages from common viral illnesses is also extremely difficult. Inflammation of the peripheral nerves affect the arms and legs resulting in impaired function, weakness, loss of feeling and limb paralysis with or without pain. “ Guillain-Barre´ syndrome (GBS) is an immune-mediated polyneuropathy with a worldwide incidence of 1-4 patients per 100 000 inhabitants” (European Journal of Neurology 2008, p. 1332).

#### Disease Name and Synonyms

“ The syndrome was named after the French physicians Guillain, Barre and Strohl, who were the first to describe it in 1916. It is sometimes called Landry’s paralysis, after the French physician who first described a variant of it in 1859.” (All about Guillain Barre Syndrome. (01-2009) symptoms. Retrieved from <http://www.jsmarcussen.com/gbs/uk/symptoms.htm>)

GBS is not just one disease the syndrome has several variations differentiated by their symptoms, the infections preceding it, the extent of the inflammatory phase, severity, and disorder site.

Common variations of the disorder are as follows: Acute Inflammatory Demyelinating Polyneuropathy (AIDP) which is the most frequent form of GBS in the Western part of the World. Acute Motor Axonal Neuropathy (AMAN), Acute Motor and Sensory Axonal Neuropathy (AMSAN) and the cranial nerve variant of GBS called Miller Fisher Syndrome (MFS) are all forms of GBS but are not as common as AIDP.

### Symptoms

Symptoms usually begin in the patient's feet, face or hands it spread to the arms or legs, it increase in potency as symptoms travel towards the midpoint of the body. The symptoms commonly play a part on both left and right sides of the body. GBS is so irregular that motor symptoms or interferences in the autonomous system may not be detected. ' It has been reported in rarer cases that GBS has affected an arm or leg without spreading to the rest of the individual's body.' (All about Guillain Barre Syndrome. (01-2009) symptoms. Retrieved from <http://www.jsmarcussen.com/gbs/uk/symptoms.htm>)

In some patients, the skin acquires hyperalgesia, or sensitivity to touch intensifies by bed sheets, socks and close-fitting shoes; in severe circumstances pain may limit walking. Patients with symptoms constrained to the feet and ankles may notice related symptoms in the fingertips; as the symptoms expand to the knees they possibly will extend to the wrists.

<https://assignbuster.com/examining-guillain-barre-syndrome/>

Seldom do these symptoms spread out beyond the knees into other parts of the body. Elevation of leucocytes and protein in the cerebrospinal fluid strongly indicates a diagnosis of GBS.

The patient loses the capacity to tell the difference amongst hot and cold, and may feel cold or may possibly start to sweat for no apparent reason. The patients may even receive injuries without noticing; their sense of taste can be affected; motor nerve fibers may be damaged as well.

The patient encounters a communication interruption between what he wants to perform and his ability to perform the desired act; because the motor nerves regulate movement, the damage inflicted to them triggers partial or complete blockage of the motor signals. The body surface affected by the damaged nerves drops its ability to function normally, causing reduced movement or coordination. The patient's muscles dwindle and waste; tendon reflexes are diminished or lost. An example of this is when slightly striking on the front of the patient's knee and that act not inducing a kick reaction.

Advanced weakening or paralysis could occur, on average arising in the feet, hands or face. The paralysis characteristically consists of more than one extremity, most frequently the legs. The paralysis is persistent and usually rising; expanding to the rest of the limb, and from there may extend to other extremities such as the legs, arms and the remainder of the body. Legs feel heavy; it becomes problematic to stand or climb flight of steps, or even to walk. The patient may struggle holding and manipulating objects, such as pins and buttons. Arms may seem weak and the patient will no longer be

able to lift heavy objects. The weakness may possibly be complemented by pain and involuntary muscle contractions. Constipation is more often a predicament, due to the condensed movement of the intestines, modification of diets, declining stomach muscles that contest the physical exertion by the individual to force out the intestinal contents.

Around 28% of patients with the syndrome endure and are able to walk unaided. In certain cases, the face could be affected when injury occurs to the cranial nerves. These nerves attach the brain en route to the muscles of the face, tongue and jaw, and also regulate the muscles that move the patient's head, neck and shoulders. While the paralysis evolves, all these regions may be paralyzed. The eyelids or one side of the face possibly will hang down resembling Bell's palsy; the face loses its ability to express emotions. The individual's voice may change given that the vocal chords are impaired. Speech may be incomprehensible, because the number of muscles required to form speech are declining. Deafness is rare but then again has been reported.

The progressive weakness has affected patients with varying intensities, and may be life threatening. The autonomic nerve system may be disrupted with the combination of pain, weakness, and sensory disruptions that are generally so frightening that the more inconspicuous alterations in the patient's autonomous nerve system might be unnoticed.

The autonomous nervous system controls the inner organs, the organ's functions are carried out automatically, examples of this is when the body secretes hormones, creates vision, urination, breathing, heartbeat, etc. It is

these functions that may be disrupted, which will result in arrhythmia, unstable blood pressure, blurred or double vision, vertigo, fainting spells, inability to regulate the body temperature, trouble breathing, reduced ability to control the function of the stomach, digestive system and bladder, loss of weight, vomiting after meals, reduced function of various glands, incontinence, impotency, and the bladder may feel as it is not being emptied no matter how many times it is expelled.

It is also very well noted that most patients have had a “ common” infection three weeks prior to GBS and it seems that the infection triggers the onset of GBS.

## Treatment

Treatment options for GBS focus on lessening the severity of the symptoms and accelerating recovery. Three main therapies are used to achieve this: intravenous immunoglobulin, plasma exchange and CSF filtration.

Intravenous immunoglobulin is understood to block the receptors on microphages preventing an attack on the Schwann cells and myelin. Plasma exchange works by circulating blood through a machine which removes antibodies, and replacing fluid loss with albumin. Cerebrospinal fluid filtration, which removes cells, including inflammatory mediators, is less commonly used. Research suggests that intravenous immunoglobulin and plasma exchange are the most common and effective treatment for GBS, when started within the first 2 weeks of syndrome’s onset. Quick intervention using either one of these treatments appears to be successful and may possibly reduce recovery time. Both treatments are very good and

neither is superior to the other, and there is no advantage to merging these treatments.

The main treatment for GBS is preventing and dealing with the complications (such as breathing complications or infections) and providing supportive care until symptoms begin to improve. This may include; reducing your breathing difficulties, sometimes with the help of a breathing machine, monitoring your blood pressure and heart rate is also good preventative care. Providing adequate nutrition if you have problems chewing and swallowing is also a key to overcoming this syndrome. The patient should attend physical therapy to help maintain muscle strength and flexibility. Preventing and treating complications such as pneumonia, blood clots in the legs, or urinary tract infections.

Other treatment of (GBS) depends on how severe your symptoms are. Careful monitoring is very important during the early stages of GBS because life threatening complications can occur within twenty four hours after symptoms first start.

#### Conflicts of Medical Resources with Guillain Barre

In 1976, vaccination against a new swine influenza A (H1N1) virus was linked to a substantial increased risk for GBS in the forty two days after vaccination (approximately 10 excess cases per 1 million vaccinations) considerations of ending the immunization program were taken into account despite the circumstantial severity of the influenza virus's transmission around the world.

There are certain circumstances in which immunizing individuals, particularly those with a prior history of GBS, may call for caution. However, the benefit of inoculations in averting disease and decreasing morbidity and mortality, particularly for influenza, needs to be weighed against the potential risk of GBS.

Destruction of the axonal or myelin membranes could presumably be mediated directly by vaccine virus or vaccine-associated products, or infection or damage of surrounding supporting cells by virus could lead to insertion of virus specified polypeptides into host cell membranes, resulting in a humeral or cell-mediated autoimmune response to the infected cell. Finally, axons or myelin cells could potentially be damaged by the introduction of sequestered myelin antigens into the circulation, inciting autoimmunity. Moreover, it is likely that host factors and genetic polymorphisms may result in a predisposition to GBS in some individuals. Several studies have suggested that various polymorphisms, including genes of the T-cell glycolipid.

### Recovery

Making a prediction about recovery is impossible. Recovery begins as abruptly and mysteriously as when GBS symptoms first started to appear. The symptoms fade gradually, but could take weeks, months or even years to finally get rid of. The development of the disease fluctuates for each patient. Recovery takes 3 to 6 months for most people, and only about two thirds of them ever recover completely.



As tingling, numbness and pain dissipates, strength comes back to the affected parts of the body, mostly in the reverse order of sequence as when the signs first appeared. This indicates that in most cases, the arms and fingers will regain their strength prior to the legs, however right handed patients may experience their muscle strength returning to their left hand before their right hand.

Axonal damage begins to be repaired; the axon grows little by little and is increasingly wrapped by myelin. The myelin sheath can grow outward in as little as a couple of days, while it could take longer for the body to repair a damaged axon. Example of this is a motor nerve that is regenerated at a rate of 1 mm/day, so it can take weeks if not months to restore a damaged nerve.

Demyelination is then repaired by the regeneration of the myelin sheath. The rate of regenerating myelination depends on the amount of damage. The sheath consists of multiple layers that grow back gradually; the myelin has to have a particular thickness prior to the nerve cells recapturing its ability to transmit impulses. The myelin sheath may never regain its normal thickness.

These facets decrease the nerve signal transmission speed forever, after the patient has recovered from GBS. Research on the use of treatments that speed up the growth of motor nerves is under way but no one will know when they will arrive or if they will ever arrive.

There is no feasible way in predicting which nerves will regenerate. Research states that damaged axons are not restored, and that the surrounding axons send branches out that take over the roles of the impaired nerves, in the

<https://assignbuster.com/examining-guillain-barre-syndrome/>

affected part of the body. The area could function again, and it may seem as if the muscle has regained full strength, but the muscle and nerves have to work harder to carry out the same job and they end up tiring faster than was the case prior to GBS.