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Original Research Article

Factor affecting recovery after guillain-barre syndrome: A narrative review

Chirag Solanki[1],*, Hetvi Chitroda[1], Hetvi Jethloja[1], Fenisha Parmar[1], Radhika Raichura[1], Janvi Rana[1], Priyanka Unadkat[1]

[1] *Dept. of Physiotherapy, School of Physiotherapy, R K University, Rajkot, Gujarat, India*

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ABSTRACT

Background: Guillain-Barre syndrome (GBS) is a set of clinical syndromes with a common pathophysiological basis, and is usually considered to be an immune-mediated disorder of the peripheral nervous system. GBS is usually characterized by symmetrical flaccid paralysis with areflexia, which usually reaches a maximum severity within four weeks. To identify the predictive factors associated with prognosis in the Guillain-Barré syndrome (GBS), which can be helpful to fully evaluate the disease progression and provide proper treatments. GBS is usually self-limiting, and most patients either recover completely or only retain minor residual symptoms. But there are still several patients who may face severe out-comes including death. In our study, GBS prognostic factor are studied and relevance is found regarding the disorder.

Materials and Mehthods: Literature was searched using many electronic databases. Additionally, reference list of most prominent articles were searched to increase the search accuracy, as much as possible. Studies which are evaluating the factors which are affecting recovery after GUILLAIN-BARRE SYNDROME.

Conclusion : This suggests that there are certain infections causing GBS into the patient, also it occurs by a weak immune response to illness.

1. Introduction

Guillain-Barré syndrome is found in Landry's report on 10 patients with "ascending paralysis" in 1859. In 1916 Guillain, Barré, and Strohl described two French soldiers with motor weakness, areflexia and "albuminocytological dissociation" in the cerebrospinal fluid. Subsequently several cases with similar manifestations were reported and this clinical entity was named after Guillain and Barré. Later, different types of the syndrome with characteristic clinical features were identified. This distinction is possible today on the basis of clinical features, aetiology and electrophysiological characteristics.

Guillain-Barré syndrome is an autoimmune disorder encompassing a heterogeneous group of pathological and clinical entities. Antecedent infections are thought to trigger an immune response, which subsequently cross reacts with nerves leading to demyelination or axonal degeneration.[1]

According to WHO overall incidence of GBS is 0.4 to 4.0 people per 100 000 per year. People of all ages can be affected, but it is more common in adults and in males.

Symptoms typically last a few weeks, with most individuals recovering without long-term, severe neurological complications.

1. The first symptoms of Guillain-Barré syndrome include weakness or tingling sensations. They usually start in the legs, and can spread to the arms and face.

* *Corresponding author.*
E-mail address: Chirag.solanki@rku.ac.in (C. Solanki).

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2. For some people, these symptoms can lead to paralysis of the legs, arms, or muscles in the face. In 20%–30 % of people, the chest muscles are affected, making it hard to breathe.
3. The ability to speak and swallow may become affected in severe cases of Guillain-Barré syndrome. These cases are considered life-threatening, and affected individuals should be treated in intensive-care units.
4. Most people recover fully from even the most severe cases of Guillain-Barré syndrome, although some continue to experience weakness.
5. Even in the best of settings, 3%–5% of Guillain-Barré syndrome patients die from complications, which can include paralysis of the muscles that control breathing, blood infection, lung clots, or cardiac arrest.[2]

The etiology of GBS is not completely understood. It is not a contagious disorder. About two-thirds of people with GBS develop it soon after they have been suffered with diarrhea or a respiratory infection. This suggests that the disorder may be triggered by an improper immune response to the previous illness.

Infections that may trigger GBS include:

1. Campylobacter jejuni which can cause gastrointestinal infection. This bacterium is one of the most common risk factors for GBS.
2. Cytomegalovirus (CMV)
3. Epstein Barr virus
4. Cytoplasm
5. Varicella zoster virus
6. Human immunodeficiency virus (HIV), dengue, or influenza
7. Less commonly vaccinations, surgical procedures, and trauma have been reported to trigger the development of GBS.

GBS can affect person of any age group, however the incidence of GBS increases with age. People older than 50 years are at greatest risk for developing GBS.

1.1. Variants of GBS are

1. Acute Motor Axonal Neuropathy - This variant of GBS initially was recognized as epidemics of paralysis in children in rural Northern China. It does not affect sensory nerves and patient more often requires ventilator support.
2. Miller Fisher Syndrome - It consists of the triad of areflexia (loss of deep tendon reflexes, such as the knee and ankle jerk), external ophthalmoplegia (weak eye muscles that cause double vision), and ataxia (poor balance and coordination with sloppy or clumsy walking).
3. Chronic Inflammatory Demyelinating Polyneuropathy (CIDP) - CIDP is a chronic variation to Guillain-Barré syndrome, and is characterized by symmetrical weakness and sensory changes. In comparison to GBS, breathing, swallowing and speaking are rarely affected.
4. Multifocal Motor Neuropathy (MMN) - It is a rare chronic inflammatory neuropathy characterized by episodes of right and/or left-sided asymmetric, distal limb weakness, of the upper limbs more often than the lower limbs.

Most people recover fully from GBS, but some people have long-term nerve damage. Residual weakness can be seen in 30% of GBS cases after three years. About three percent of cases may suffer a relapse of muscle weakness and tingling sensations many years after the initial attack. 3%-5% of GBS patients may die from complications, which include;

1. Paralysis of the muscles that control breathing,
2. Sepsis (blood infection),
3. Pulmonary embolism (lung clots)
4. Cardiac arrest

Guillain-Barré syndrome is difficult to diagnose because the symptoms are similar to other neurological disorders. Diagnosis is based on symptoms, findings on neurological examination including diminished or loss of deep-tendon reflexes.

1. Routine blood tests are done, to exclude other diseases with similar symptoms and for better assessment of functional status and prognosis.
2. Specific tests are required to identify the cause of trigger of GBS.

The following tests are used to confirm a diagnosis of GBS-

1. Lumber puncture (Spinal tap) - People with Guillain-Barré syndrome has higher-than-normal levels of protein in their cerebrospinal fluid without an elevation in white blood cells. The increase in cerebrospinal fluid (CSF) protein reflects the widespread inflammation of the nerve roots.
2. Electromyography — An electromyography is a nerve function test. It reads electrical activity from the muscles and distinguishes whether muscle weakness is caused by nerve damage or muscle damage.
3. Nerve Conduction Tests — Nerve conduction studies may be used to test how well nerves and muscles respond to small electrical stimuli.
4. Imaging studies, such as magnetic resonance imaging (MRI) and computed tomography (CT) scanning of the spine may be helpful in excluding mechanical causes of myelopathy.[3]

2. Materials and Methods

2.1. Does type of GBS affects recovery process?

Little is known about the long-term prognosis for patients that the severe acute motor axonal neuropathy (AMAN)

form of Guillain–Barré syndrome (GBS).

Generally, it is believed that no further recovery can be expected two to three years after GBS,30 but follow up has mainly been limited to six months or one year in most previous studies, which may be the reason for the overly pessimistic outlook for long term recovery from severe GBS.[4]

2.2. Does coma affects GBS recovery?

Electroencephalogram (EEG) tracings typically identify alpha rhythm activity unresponsive to painful and auditory stimulation during fulminant GBS, but other tracings have also been reported (sleep, responsive or the so-called "alpha-delta" stage of sleep). Fulminant GBS with brain-death presentation is rare but deserves medical knowledge and awareness. Its diagnosis leads to a well-established treatment that reduces long-term disability.

This case reminds us of the importance of electrophysiological investigations during clinical brain-death states with no definite cause. Finally, long-term physiotherapy and specific rehabilitation programs appear essential to improve recovery.

Diagnosis can be very difficult when the patient is seen during the coma period with no previous case history, where the patient's Glasgow coma scale score was 3/15 and when there is nonreactive bilateral mydriasis. The presence of bilateral mydriasis has rarely been described in GBS.[5]

2.3. Does length of stay in hospital affects the GBS recovery?

Despite improved recognition and treatment, GBS continues to be a severe disease. One-quarter of patients will require mechanical ventilation for respiratory failure or airway protection, 5-7 and 3-11% will die of GBS-related complications.

Although most patients make substantial recoveries, 20% to 38% experience residual disability,5,9 and more than one-third are forced to make adjustments to their work and social lives.

Because most patients require hospitalization, GBS results in more than 6000 hospital admissions annually in the United States alone. Recent trends in the treatment of GBS are resulting in fewer patient transfers to tertiary care centers, broadening the number of hospitals and neurologists who need familiarity and expertise in the management of GBS. This review aims to provide guidelines for the inpatient care of GBS, focusing first on treatment of the underlying disease process and then turning to the prevention and management of potential complications.[6]

2.4. Does body mass index (BMI) affects the GBS recovery?

There was one study done by Ming Ding et al., where they included three large ongoing cohort studies involving 252,980 participants followed for 27–34 years and from that 328 incident GBS cases showed that higher BMI and WC were associated with higher risk of GBS.

These data from large cohorts showed that higher BMI and WC jointly were associated with higher risk of GBS. Their study highlighted the importance of maintaining a normal body weight and waist circumference in prevention of GBS.[7]

2.5. Does family history affects the GBS recovery?

NO family history does not because, the illnesses are neither hereditary nor contagious.

2.6. Does specific gender is being affected in GBS?

Young or old, male or female. The illnesses are neither hereditary nor contagious. GBS affects about 1500 people every year in the United Kingdom. It can occur at any age from infancy onwards but is slightly more common in the old; it is more common in men than in women; it is not hereditary; it is neither passed onto children nor is it infectious and it is not caught from or transmitted to anybody else. However, it does often develop a week or two after a throat or intestinal infection.

2.7. Does side of body involved affects GBS?

A study stated that the mildest cases of GBS includes weakness may arrest and cause only moderate difficulty in walking, requiring sticks, crutches or a walking frame.

In some cases, the weakness progresses and leads to complete paralysis of the legs, the arms may also be affected. In a quarter of cases the paralysis progresses up the chest and the patient are unable to breathe on his or her own and needs to rely on a mechanical breathing machine (ventilator).[8]

2.8. Does alcohol consumption affects the GBS recovery?

Johannes et al. they did a study on alcohol related acute axonal polyneuropathy they concluded that the patients the combination of alcohol abuse and malnutrition caused severe acute axonal polyneuropathy. It's distinction from Guillain-Barre syndrome is important because treatment requires balanced diet, vitamin supplementation, and abstinence from alcohol, while immunotherapy may not be indicated.[9]

2.9. Does diabetes affects the GBS recovery?

The effects of diabetes on patients with GBS could be explained in several ways. Some patients with diabetes may have pre-existing nerve injury, so further injury from GBS makes things worse (Berciano et al., 2000; Bae et al., 2016).

Reduced rates of nerve regeneration were found early in diabetes even before symptoms and signs of neuropathy appeared, while presence of diabetic neuropathy was associated with a further decrease in a capacity to regenerate.

Diabetes may also increase inflammation in GBS since both diseases are associated with the systemic inflammation including increased level of different cytokines (Berciano et al., 2000; Hartge et al., 2007; Lu and Zhu, 2011; Bae et al., 2016). It is also interesting that our GBS patients with diabetes were prone to develop the axonal form of the disease.[10]

2.10. Does hypertension affects the GBS recovery?

Cardiovascular dysautonomy in GBS is often present in patients with extensive motor involvement, and may be present both during the late progressive phase of the disease and during the plateau phase.

Tachycardia tends to abate as weakness progresses, but in some cases, hypertension and tachycardia may persist until the onset of recovery.

In this case, hypertension and tachycardia started before the onset of motor weakness and persisted until the beginning of the recovery phase, but was well controlled with antihypertensive medication.

Hypertension occurs in 60% to 70% of patients with GBS,9 and marked fluctuation of BP is a bad prognostic sign. Eiben et al" detected hypertension in 60% of all patients with GBS and in 95% of the patients who required respiratory support.[11]

3. Discussion

The purpose of this article was to review and evaluate the existing scientific literature for predictive factors affecting after GBS.

Yitao Zhang et al stated that Diabetes, high fasting blood glucose level and high blood pressure at admission, urosepsis, abnormal body temperature, requiring ventilator support, disorder of consciousness, no preceding upper respiratory tract infection, low level of blood sodium and albumin, high white blood cell count, high fibrinogen level, and abnormal hepatic and renal function were demonstrated as poor prognostic factors. Hyponatremia is so common in GBS patient, despite that it is not a classical manifestation of GBS; however, there are series in which are described to be present in 21.5 to 48% of the cases; in our review, it was found in 21.6% of our patients, which is similar to the Northern China study and the British study. Hyponatremia is also a predictor of poor prognosis.

The elevated liver enzyme level indicates poorer prognosis as well. There are two possible reasons. First, liver damage conditions such as infection with hepatitis virus; alcohol abuse; hepatotoxic drugs; recent surgery and so on may influence the systemic health condition significantly, and those who with liver damage may recover more slowly compared with those who have normal hepatic function. poor nutrition condition such as low serum albumin level, high coagulation state as high fibrinogen level, and infection at early stage of GBS such as high white blood cell count, are also predictors of poorer prognosis.[12]

Hypertension occurs in 60% to 70% of patients with GBS, and marked fluctuation of BP is a bad prognostic sign. Eiben et al" detected hypertension in 60% of all patients with GBS and in 95% of the patients who required respiratory support. They also noted that patients with hypertension have a higher.[13]

Acute motor axonal neuropathy is subtype of GBS in northern china as stated by jing tian. Acute motor axonal neuropathy patients had significantly higher prevalence of antecedent diarrhoea, longer duration of hospitalization, and slightly slower recovery than those with acute inflammatory demyelinating polyneuropathy (AIDP). Based on multivariate regression analysis, acute motor axonal neuropathy patients with antecedent diarrhoea or conduction blocks (CBs) had dramatically better short-term prognosis.[14]

Ming Ding el al. stated that in total, 328 GBS cases were documented during 5,422,788 person years of follow-up in the three cohort's incident rate of GBS was higher in men than women. Compared to participants with BMI <25kg/m2, of GBS was 1.34 for participants with overweight (25kg/m2≤BMI<30 kg/m2), and 1.68 (95% CI: 1.21, 2.35) for participants with obesity (BMI≥30 kg/m2).[15]

Naglaa Mohamed El-Khayat in his article stated that Cranial nerve involvement was found in 12 patients and the most cranial nerve involved was facial nerve, most studies report cranial nerve involvement ranging from 30-60 % with facial and bulbar nerves being commonly involved, 40% patients had cranial nerve affection in one study and one third of patients had cranial nerve involvement in another study.[16]

4. Conclusion

There are different factors that are responsible for the trigger of GBS. Certain infections are causing the patient to have GBS. This suggest that the disease occurs by a weak immune response to illness.

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7. Conflict of Interest

None.

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Author biography

Chirag Solanki, Assistant Professor

Hetvi Chitroda, Student

Hetvi Jethloja, Student

Fenisha Parmar, Student

Radhika Raichura, Student

Janvi Rana, Student

Priyanka Unadkat, Student

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