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REVIEW ARTICLE

Guillain-Barré Syndrome: Review and Summary

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ABSTRACT

Guillain-Barré Syndrome is a life-threatening, demyelinating, autoimmune condition in which the body's immune system attacks the myelin of the peripheral nervous system. Guillain-Barré Syndrome is characterized by ascending motor weakness and acute flaccid paralysis. Demyelination results in nerve inflammation, numbness, tingling, muscle weakness, structural damage to the myelin sheath, and possible respiratory system complications. The annual incidence rate is 1.1 to 1.8 per 100,000 persons worldwide. Guillain-Barré Syndrome is thought to be triggered by an antecedent infection such as a viral, gastrointestinal, or bacterial infection, food poisoning, or reaction to a vaccine. Approximately 9-11% of cases result in severe disability or death. The acute phase can vary in length from a few days to several months, although over 90% of patients begin rehabilitation within four weeks. Patient care involves a team of neurologists, physiatrist, internist, nurses, physical, occupational, and speech therapists, social worker, psychologist and family physician. Elevated cerebrospinal fluid protein, symmetrical muscle weakness, the rate and order at which symptoms appear, and the absence or prolonged latency of reflexes are hallmarks for diagnosing Guillain-Barré Syndrome. A lumbar puncture to test for protein levels in the brain and spinal cord, and nerve conduction velocity test may aid in proper diagnosis, critical for optimizing treatment options and minimizing further progression. Although there is no cure, treatment may consist of plasmapheresis, typically performed four times during hospitalization, or intravenous immunoglobulin. Intravenous immunoglobulin combined with plasmapheresis should be avoided. Although glucocorticoids could repair damage to the blood-nerve barrier, oral corticosteroids could delay recovery.

INTRODUCTION

Guillain-Barré Syndrome is a potentially life-threatening, inflammatory, demyelinating condition of the peripheral nervous system, characterized by ascending motor weakness and acute flaccid paralysis. Guillain-Barré Syndrome has been classified as an autoimmune disease in which the body's immune system mistakenly attacks its own myelin sheath, a protective coating of proteins and phospholipids that insulates nerve fibers and increases the rate of nerve impulses [1,2]. Demyelination results in nerve inflammation, numbness, tingling and muscle weakness, and structural damage to the myelin. One quarter of patients diagnosed with Guillain-Barré Syndrome develop respiratory system complications, requiring mechanical ventilation [3]. Among the most common causes of neuromuscular paralysis in the world, the annual incidence rate for Guillain-Barré Syndrome is 1.1 to 1.8 per 100,000 persons worldwide [4]. Guillain-Barré Syndrome is believed to be triggered by an antecedent infection^{1,6} such as a viral infection (especially influenza, COVID-19, and Zika virus) [5-10], a gastrointestinal-related infection, bacterial infection such as *Campylobacter jejuni* [11-13], food poisoning, or from a reaction to a vaccine [14]. Approximately 9-11% of cases result in severe disability or death [12,15,16].

The acute phase of Guillain-Barré Syndrome can vary in length from a few days to several months, with over 90% of patients moving into the rehabilitation

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phase within four weeks. Patient care typically involves a team of neurologists, physiatrist, internist, nurses, physical therapist, occupational therapist, speech therapist, social worker, psychologist and family physician [3].

GUILLAIN-BARRÉ SYNDROME DEVELOPMENT

Although *Campylobacter jejuni* from undercooked poultry is thought to be the most common cause of Guillain-Barré Syndrome, it has been suggested that influenza and the Zika virus could play more important roles than previously suspected [7,9,17,19]. Although this is a new research area, current literature shows that infection with COVID-19, as well as the COVID-19 vaccine, could also play a triggering role in Guillain-Barré Syndrome development in some patients. The Janssen COVID-19 vaccination fact sheet was recently updated by the Food and Drug Administration to include a warning about Guillain-Barré Syndrome associated with its vaccine [16,20–27].

UNDERLYING RISK FACTORS

While studies are ongoing, a myriad of genes, gene networks and canonical pathways, including transcription factors and inflammatory cytokines, have been revealed in Guillain-Barré Syndrome pathogenesis. A study by Chang et al. found 256 genes and 18 gene networks significantly associated with Guillain-Barré Syndrome [28], including four significantly upregulated functional genes, FOS, PTGS2, HMGB2 and MMP9, involved in inflammatory response, infectious and respiratory diseases. Associated gene networks included hub genes MMP9, PTGS2 and CREB1. The gonadotrophin-releasing hormone pathway, corticotrophin-releasing hormone pathway and ERK/MAPK were the most significant canonical pathways involved in Guillain-Barré Syndrome. Molecular and cellular functions associated with Guillain-Barré Syndrome included cellular development, movement and cell death. Hematological system development and function, immune cell trafficking and organismal survival were the most significant functions involved in Guillain-Barré Syndrome physiological development. A DNA analysis from 2002 revealed a deletion at chromosomal locus 17p12, typical of Hereditary Neuropathy with Liability to Pressure Palsies (HNPP) [1]. HNPP has been shown to be an autosomal dominant neuropathy presenting in Guillain-Barré Syndrome. In 2008, molecular analysis further revealed this duplication at chromosome [29] 17p11.2–12, a known genetic risk factor for Charcot-Marie-Tooth disease type 1A, of which symptoms are similar to Guillain-Barré Syndrome. The study concluded that this genotype may comprise a previously unrecognized risk factor for Guillain-Barré Syndrome. Another study from 2006 showed that subjects with genotype CD1E*01/01 were two-and-a-half times more likely to develop Guillain-Barré Syndrome and claimed that Guillain-Barré Syndrome susceptibility was related to CD1E

and CD1A gene polymorphisms [30]. Studies from 2010 and 2012 revealed genetic variations of Tumor Necrosis Factor (TNF) and found that TNF- α 308 G>A polymorphism was significantly associated with an increased risk of Guillain-Barré Syndrome in the overall population [31]. Among 140 Guillain-Barré Syndrome patients tested in 2010, patients with A and T alleles had higher TNF- α serum levels. Studies concluded that TNF- α 308 A allele may be a moderate risk factor for Guillain-Barré Syndrome. Another study from 2012 examined Toll-Like Receptors (TLR) in 21 Guillain-Barré Syndrome patients and found that expressions of TLR2, 4 and 9 were higher among Guillain-Barré Syndrome patients compared with healthy controls [32,33]. This study showed that Guillain-Barré Syndrome patients' disability scores had a strong correlation with higher levels of these receptor molecules and suggested the TLR signaling pathway could be involved in the pathogenesis of Guillain-Barré Syndrome. A 2009 study examined the Glucocorticoid Receptor (GR) gene in 318 patients and found that Single Nucleotide Polymorphisms (SNPs) in the GR gene influenced sensitivity to glucocorticoids and are related to both microbial colonization and an increased susceptibility to developing Guillain-Barré Syndrome. Furthermore, this study found that haplotypes carrying Bc1I minor allele polymorphisms were related to Guillain-Barré Syndrome phenotypes and outcomes [34].

Molecular mimicry is also thought to play a role in the development of Guillain-Barré Syndrome. During the antecedent infection, the patient's immune system produces antibodies against peripheral nerve gangliosides [4,12]. The antibodies bind to gangliosides and microbial antigens, which penetrate the blood-nerve barrier by inducing B cells to produce an anti-ganglioside immune response. Endoneurial macrophages release proinflammatory cytokines and free radicals, which invade and degrade the myelin and/or axons of nerves. This process activates T cells to release more cytokines, causing further damage to myelinated Schwann cells [35].

Furthermore, although the majority of autoimmune disorders are more prevalent among females, Guillain-Barré Syndrome has been shown to be increased in males, providing evidence for male and female differences in the ability of a target organ to withstand damage. Sex hormones are believed to have immune modulating properties such as an influence on innate and adaptive immune cells, the number of B and T cells, antigen presentation and cytokine secretion and to provide cellular protection against tissue damage [36]. Therefore, sex hormones may offer a therapeutic benefit in several autoimmune conditions. A 2009 study suggested that sex plays a vital role that must be taken into consideration for all immunological studies, including at all levels of biological research [36,37].

RESULTS OF THE DISORDER

The blood-nerve barrier is considered the gatekeeper that

regulates functionality of peripheral nerves connecting the central nervous system to muscles of the limbs and sensory organs. The blood-nerve barrier is important for regulating proper nerve function by enabling necessary serum nutrients to flow in while keeping unwanted material out. Breakdown of the blood-nerve barrier plays a key role in Guillain-Barré Syndrome disease progression [38]. The disruption of diffusion barriers at Guillain-Barré Syndrome onset increases exposure of spinal roots and peripheral nerves to possible pathogenic macromolecules, allowing elevated protein concentrations to enter the cerebrospinal fluid [39]. Glucocorticoids, any of a group of anti-inflammatory and immunosuppressive corticoids, have been shown to repair damage to the blood-nerve barrier [39-41].

SCREENING FOR GUILLAIN-BARRÉ SYNDROME

Elevated total protein concentration with a normal cell count in cerebrospinal fluid is a hallmark for Guillain-Barré Syndrome diagnosis. A lumbar puncture, or spinal tap, can be performed by inserting a needle into the patient's lower back for the withdrawal of cerebrospinal fluid to test for elevated protein levels in the brain and spinal cord [41]. Other signs and symptoms can also present in Guillain-Barré Syndrome, including symmetrical muscle weakness occurring on both sides of the body, the rate and order at which symptoms appear (Guillain-Barré Syndrome symptoms typically develop over a period of days, beginning in the fingertips and toes and spreading up the arms and legs), accompanied by the absence or prolonged latency of reflex responses. A conduction block in the most proximal reflexes of the peripheral nervous system is the most sensitive parameter in diagnosing early Guillain-Barré Syndrome onset. Therefore, a nerve conduction velocity test may aid in Guillain-Barré Syndrome diagnosis [42]. Rapid and proper diagnosis of Guillain-Barré Syndrome is critical to optimize treatment options and decrease the likelihood of further immediate progression [43,44].

GUILLAIN-BARRÉ SYNDROME TREATMENT

Although there is no known cure for Guillain-Barré Syndrome, treatment may consist of blood purification by means of plasmapheresis or intravenous immunoglobulin. Plasmapheresis has been used since the 1950s to separate plasma from other components of blood cells. Once separated, the plasma is returned to the patient, avoiding the loss of vitamins, coagulation proteins and antibodies, while reducing the risk of further infection. This procedure is optimally performed four times during hospitalization, although two treatments may suffice in mildly affected patients [45]. Intravenous immunoglobulin products from human plasma were first used in 1952 to treat immune deficiencies [46]. Intravenous immunoglobulin contains pooled immunoglobulin from approximately 1,000 or more

plasma donors. Intravenous immunoglobulin is administered through an IV and has been shown to be an effective treatment for Guillain-Barré Syndrome. Intravenous immunoglobulin combined with plasmapheresis, however, should be avoided as a treatment for Guillain-Barré Syndrome [47]. Also, although glucocorticoids could repair damage to the blood-nerve barrier, a study showed that treatment involving glucocorticoids did not significantly hasten recovery from Guillain-Barré Syndrome or affect long-term outcomes, and oral corticosteroids could delay the recovery process [48,49].

After hospitalization from Guillain-Barré Syndrome, patients are transferred to inpatient rehabilitation, where treatment and recovery focus on physical, occupational and speech therapies. Rehabilitative therapies should resemble those performed for spinal cord injuries and post-polio syndrome. However, these therapies should not be performed to the point of motor unit exhaustion, which could cause paradoxical weakening in rehabilitating Guillain-Barré Syndrome patients [50].

The length of stay for inpatient rehabilitation can be predicted by factors such as whether and how long the patient required ventilator support during hospitalization, and dysautonomia, cranial nerve involvement and other medical complications during the acute phase. During the early stages of recovery, some complications could interfere with the rehabilitation program, including deafferent pain syndrome, deep venous thrombosis, joint contractures, hypercalcemia of immobilization and bedsores. Anemia is also a concern during the early stages of recovery after Guillain-Barré Syndrome [50].

For long-term maintenance after Guillain-Barré Syndrome, one systematic review of seven articles showed that high-intensity exercise could significantly reduce disability, and various forms of exercise could improve overall mobility, muscle strength and cardiopulmonary function and reduce fatigue in Guillain-Barré Syndrome patients [51].

GUILLAIN-BARRÉ SYNDROME MORTALITY RATE

The mortality rate in patients diagnosed with Guillain-Barré Syndrome varies between 1-18%. Death may result from pneumonia, sepsis, Adult Respiratory Distress Syndrome (ARDS), autonomic dysfunction or pulmonary embolism. In a 2011 study, case records were analyzed for 273 Guillain-Barré Syndrome patients (190 men and 83 women) who required medical ventilation between 1984-2007. Of those ventilated patients, records showed a 12.1% mortality rate. Factors that determined mortality from Guillain-Barré Syndrome included elderly age, autonomic dysfunction, pulmonary complications, hypokalemia (low potassium levels) in the blood and bleeding complications. The risk of mortality increased 4.69-fold when pneumonia

was present. Higher age was associated with a higher risk of mortality [52].

GUILLAIN-BARRÉ SYNDROME PROGNOSIS

Prognosis of Guillain-Barré Syndrome is generally good. Eighty percent of Guillain-Barré Syndrome patients recover completely within 3–6 months, while the remaining survivors suffer residual neurological disability. Early diagnosis is important to ensure a favorable outcome. After following 29 Guillain-Barré Syndrome patients for 2 weeks to 10 years, a 2012 study found that facial paralysis in 5 participants (17%) was still present after 10 years [53]. Eleven participants (38%) continued to experience paresthesia, a sensation of numbness, tingling, pins and needles and prickling, usually felt on the skin. Six patients (21%) had limitations using their arms, and 15 (52%) had limitations in walking. Decreased health-related quality of life in comparison with the general public was seen at 10 years post-Guillain-Barré Syndrome [54].

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