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Guillain-Barre Syndrome

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Introduction

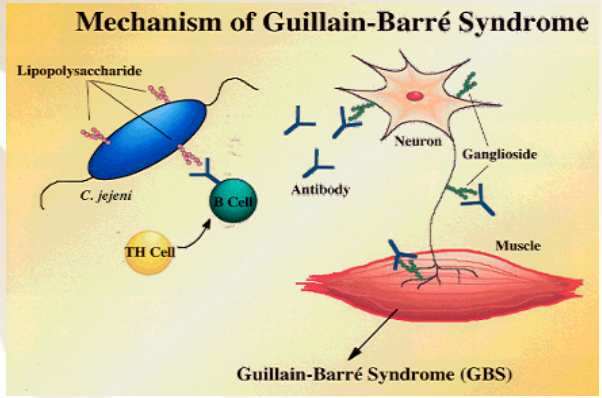
Guillain-Barre Syndrome consists of a group of neuropathic conditions characterized by progressive weakness and diminished or absent myotatic reflexes. The estimated annual incidence in the United States is 1.65 to 1.79 per 100,000 persons (Walling & Dickson, 2013). Understanding how this condition progresses and the affects it has on a patient will assistant in providing optimal patient care as a future nurse anesthetist. As stated by Turakhia, P., Barrick, B., and Berman, J. (2013) "the anesthetic implications for the various comorbidities are varied and can be profound" (p. 1). An individual who suffers from Guillain-Barre is a patient who certainly requires critical care management in order to avoid complications associated with the disease. Being aware of the pathophysiology, risk factors, signs and symptoms, and treatment involved in caring for Guillain-Barre Syndrome can better prepare the advanced practicing nurse for patients suffering from this condition.

Signs & Symptoms

The first symptoms include varying degrees of weakness or tingling sensations in the legs, that can spread to the upper body. This can progress to almost complete paralysis. Common symptoms are: Facial droop, diplopias, dysarthria, dysphagia, ophthalmoplegia, pupillary disturbances, dyspnea on exertion, shortness of breath, difficulty swallowing, slurred speech. Autonomic Symptoms include: Tachycardia, bradycardia, facial flushing, paroxysmal hypertension, orthostatic hypotension, diaphoresis, urinary retention. Required criteria for the diagnosis include: Progressive weakness of more than two limbs, areflexia, progression for no more than four weeks (Sebastian, 2012). Supportive criteria include: Relatively mild sensory signs, raised protein in the cerebrospinal fluid (CSF), with a relatively normal cell count, neurophysiological evidence of conduction block, weakness is frequently proximal and distal, unlike dying back axonopathies, and respiratory involvement occurs in about a quarter of cases. The CSF protein may be normal in the first week of the illness, but may then rise to several g/dl. The CSF cell count usually remains below 500 cells/litre. Oligoclonal bands are sometimes found in the CSF. Routine blood tests sometimes reveal a raised sedimentation rate with hyponatraemia from inappropriate antidiuretic hormone release, and mild impairment of liver function tests is not uncommon (Winer, 2001).

Underlying Pathophysiology

Subtypes of GBS include AIDP, AMAN, acute motor and sensory axonal neuropathy (AMSAN), acute sensory axonal neuropathy (ASAN), and Miller–Fisher syndrome. Electrophysiology is an important clinical tool for distinguishing demyelinating and axonal subtypes, because it may reveal demyelination, loss of motor axons only, loss of sensory axons only, or mixed loss (Franssen & Straver, 2014). Proposed mechanism involves an antecedent infection leading to an autoimmune response reacting with peripheral nerve components. Most of the pathogens gains entry to the body through mucosal or gut epithelium and induce antibody production against specific gangliosides in myelin. Proposed mechanism involves an antecedent infection leading to an autoimmune response reacting with peripheral nerve components. Most of the pathogens gains entry to the body through mucosal or gut epithelium and induce antibody production against specific gangliosides in myelin. The immune response depends on bacterial factors, such as the specificity of lipo-oligosaccharide (LOS), and on host factors, such as genetic polymorphism and immune status. The presence of antibodies leads to activation of T cells and complements, leading to a cascade of inflammation and demyelination. The demyelination decreases the velocity of nerve conduction and slows the impulse transmission along the axons. The extent of nerve damage varies, with more damage seen in the intensely myelinated peripheral nerves, causing motor and sensory weakness (Sebastian, 2012). Based on human observational studies and data obtained from EAN models, it is hypothesized that CCR2 monocytes cross the brain nerve barrier (BNB) via interaction with Schwann-cell-secreted CCL2 expressed by the BNB endothelium. These monocytes differentiate into endoneurial macrophages that migrate to CCL2-expressing Schwann cells to induce demyelination. CCR1/CCR5 macrophages may migrate to axons expressing CCL5 to induce axonal degeneration. CXCR3 T cells may migrate across the BNB via interactions with BNB endothelium CXCL10 and directly contribute to nerve injury via cytokine secretion or expression of pro-inflammatory chemokines that further attract hematogenous monocytes and lymphocytes, enhancing the inflammatory process (Chiang & Ubogu, 2013).

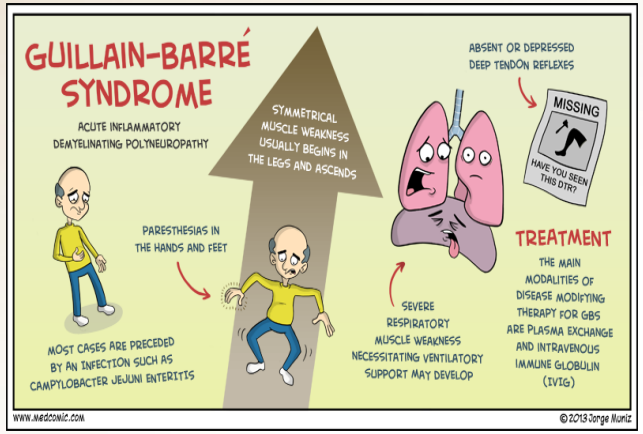


Significance of Pathophysiology

It is of utmost importance to understand the pathophysiology of Guillain-Barre Syndrome. Having an understanding of how it progresses and manifests itself can lead to quick diagnosis and intervention. Prevention of life-threatening complications remains the cornerstone of supportive care. Progressive demyelination of the phrenic nerve, innervating the diaphragm, may lead to acute respiratory muscle paralysis. Early detection of respiratory failure and anticipation of mechanical ventilation are crucial to avoid emergency intubation and cardiopulmonary arrest. Life-threatening episodes of hemodynamic instability related to autonomic dysfunction may occur in GBS patients. They should be admitted to the hospital for close monitoring of respiratory status, hemodynamic instability, and autonomic dysfunction (Sebastian, 2012).

Implications For Nursing Care

According to Dubey et al. (2016) "early identification of GBS may lead to earlier initiation of management, including immunomodulatory therapy, intensive care unit admission in selected patients, and multidisciplinary team involvement. The initial clinical diagnosis of GBS may be challenging. Lack of evaluation by a neurologist, neuropathic pain, preserved reflexes, and an atypical pattern of weakness were all associated with a delay in considering the diagnosis of GBS. The delay in diagnosis had a significant impact on outcome, as assessed by the clinical status at the time of discharge and the discharge disposition" (p. 386). It is of the utmost importance that nurses identify signs of a potential case of GBS as soon as possible in order to promote an optimal outcome for patients. The primary nursing management of a patient with GBS should be centered on problems with the airway related to respiratory muscle weakness or paralysis, decreased cough reflex, and immobilization. Due to the progressive muscle weakness, nurses should plan interventions that focus on preventing complications related to immobility, such as ensuring skin integrity. (Moore & Shepard, 2014).

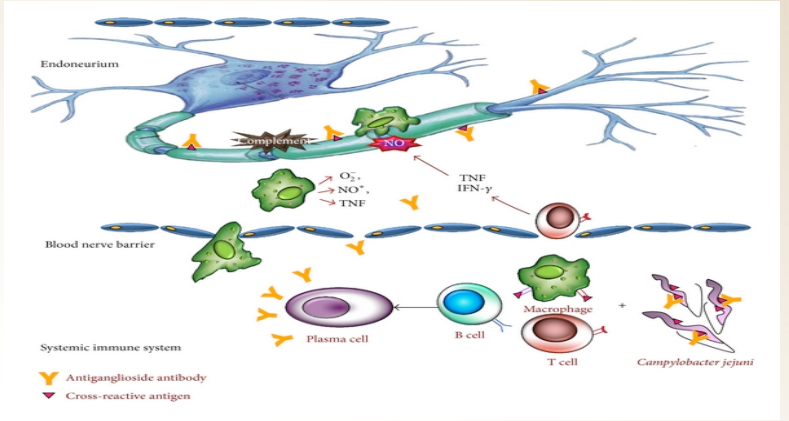


Treatment

Based on strong research evidence in adults, IVIG and plasma exchange hasten recovery from GBS in patients with impaired ability to ambulate (Rosen, 2012). Common treatment modalities include corticosteroids, intravenous immunoglobulin (IVIg), therapeutic plasma exchange (TPE), cerebral spinal fluid (CSF) filtration, and immunoadsorption. Treatment with intravenous immunoglobulin and plasma exchange reduces the time for recovery to occur, although some remain disabled (McNair, 2013).

Conclusion

Guillain Barre Syndrome can occur in any patient. It typically involves initial insult from injury or is preceded by an infection, leading to demyelination of the nerves. Understanding risk factors and how GBS presents itself can aid in early diagnosis. It is detrimental that those caring for patients be educated on the signs and symptoms, and be prepared to intervene as necessary to reduce the risk of complications. Interdisciplinary collaboration and careful management can help reduce the length of recovery and promote better outcomes for affected individuals.



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