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Guillain-Barré- Adding Insult to Injury

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Guillain-Barré syndrome (GBS) is described as an acute peripheral neuropathy causing limb ataxia or weakness, that progresses over a short period of time (Lehman, Hughes, Charand, & Drenthen, 2014). GBS is thought to be immune mediated, generally triggered by a prior viral or bacterial infection (Blum & McCombe, 2014). Rapid diagnosis and treatment of any neurologic condition is paramount to recovery of the individual experiencing the condition. GBS was once thought as a singular disease, but has been subtended into many subgroups and with varying diagnostic criteria. The most common subgroups are acute inflammatory demyelinating polyneuropathy (AIDP) and acute motor axonal neuropathy (AMAN), and Miller Fisher Syndrome (MFS) (Blum & McCombe, 2014).

It is strongly suggested that the risk of developing GBS is increased by a host of factors, including iatrogenic, genetic, pathologic factors, particularly cystic fibrosis, chemical neuropathies (Reddel, 2013). Recent attention paid to GBS has been associated with the H1N1 vaccine. However, studies comparing time periods between 2015 and 2016, suggest an increased incidence of disease associated with the Zika virus (Kochar, et al., 2016). When the peripheral blood of patients with GBS have been examined, it revealed abnormalities in T cells, antibodies and gene expression (Blum & McCombe, 2014). Even with rapid treatment, there is a risk of long-term sequelae with severe disability (Kowabara, 2004).

**Signs & Symptoms**

Symptoms tend to appear within the first 2 to 8 days, depending on the precipitating event (Kopytko & Kowalski, 2014). Classic diagnostic criteria and clinical presentation is as follows:

- **Progressive motor weakness**
  - More than one limb
  - Relative symmetry
- **Over a period of days, up to 4 weeks**
- **Areflexia**
- **Universal**
  - Classic presentation
- **Dural**
  - Areflexia with intact/bare hypalgesia
- **Autonomic dysfunction**
  - Tachycardia
  - Postural hypotension
  - Hypertension
  - Vasomotor symptoms

**Underlying Pathophysiology**

Guillain-Barré syndrome is described as “an acquired neuropathy characterized by inflammatory perineurial nerves.” (Blum, Courches, Redolfi, Spies, & McCombe, 2013, p. 92). Varying subtypes are mediated by different inflammatory responses. The pathophysiology of GBS ranges from antibody mediated disease to macrophage and T cell initiated destruction of peripheral nerve cells (Blum, Courches, Redolfi, Spies, & McCombe, 2013). GBS are found on the surface of many NK cells, but also on CD4+ and CD8+ T lymphocytes (Blum, Courches, Redolfi, Spies, & McCombe, 2013). The T cell receptors recognize antigens presented by HLA molecules and immune reactions are influenced by variations in T-cell genetics (Blum & McCombe, 2014). Depending on the subtype of GBS, peripheral nerve cells are presented to T cells with the help of CD-1, CD-4 or CD-1a polymorphisms (Blum & McCombe, 2014).

Natural killer cells are lymphocytes that play an important role through chemokines and cytokines. Killer immunoglobulin-like receptors (KIR) are the largest group of natural killer (NK) receptors and exhibit roles in both inhibitory and activating processes (Blum, Courches, Redolfi, Spies, & McCombe, 2013). KIRs are found on the surface of many NK cells, but also on CD4+ and CD8+ T lymphocytes (Blum, Courches, Redolfi, Spies, & McCombe, 2013). The importance of an altered KIR/HLA combinations in GBS is the key to suggest that innate immunity plays a significant role in the disease process.

The symptoms of the pathophysiology vary by subtype. AIDP is the most common variant accounting for 85% of GBS cases (Blum & McCombe, 2014). The immune or inflammatory mediated macrophage attraction to the myelin sheet (Panesar, 2014) - ANM is most commonly seen following infection with campylobacter jejuni. It is associated with macrophage invasion of the node of Ranvier. (Panesar, 2014) - MSF cases present with positive anti-GQ1b antibodies 96% of the time but underlying pathophysiology is not completely understood (Panesar, 2014).

**Implications for Nursing**

Nursing can make a difference in the recovery of their patients by understanding the clinical presentation, anticipating potential complications and attending to the specialized needs of the patient during the acute and recovery phase of disease (Blum, Courches, Redolfi, Spies, & McCombe, 2013). Complications can include compromised skin integrity due to limited mobility, respiratory complications due to prolonged intubation coupled with impaired mediating, and manifestations of depression due to emotional distress. Mortality associated with GBS are generally secondary to respiratory problems.

While the clinical care a patient receives is important, emotional support and education regarding disease process are equally necessary. While motor function is significantly affected by the pathophysiology of the disease process, cognitive function is less affected. Communication to the patient regarding what care is being provided, why and what is extremely important to this patient population to support their emotional well being.

The recovery phase of disease can last up to 18 months, with some patients having permanent disability.

- **Appropriate Nursing Interventions**
  - Turning and repositioning at regular intervals
  - Coupled deep breathing exercises
  - Thorough skin care
  - Appropriate postural drainage
  - Passive range of motion
  - Provide emotional support as needed

**Significance of Pathophysiology**

Overall, GBS is thought to be disease due to immunity between pathogens present in antigens and those on peripheral nerves. The body identifies “self” as foreign due to recent exposure to an antigen with similar properties and initiates the immune response. With current research however, no specific HLA has been identified (Blum, Courches, Redolfi, Spies, & McCombe, 2013). In demyelinating chronic neuropathies antibodies tend to myelin, triggering complement activation and mobilization of macrophages (Pasanen, 2015). In axonal disorders, the immune response occurs at the nodes of Ranvier, rather than the myelin sheet (Pasanen, 2015). Gangliosides appear to be the primary target in axonal disorders, and specific ganglioside antibodies have been identified in as many as 80% of patients (Pasanen, 2015). Specific targets for demyelinating syndromes have been more difficult to identify due to the nonspecific nature of macrophages.

**References, cont.**


**Image 1. Anatomy of a nerve cell. (Charand, 2004)**

**Image 2. Pathophysiology of C. Jeantar associated GBS (van den Berg, et al., 2014)**

**Figure 1. Course of Guillain-Barré Syndrome (Williams, Jacobs, & van Doorn, 2016)**