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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 (Lahmeyer, Hughes, & Hartung, 2010). 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 neuroligic condition is paramount to the recovery of the individual experiencing the ailment. GBS was once thought of as a singular disease, but has recently been distinguished 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 factors, particular pathogens, and certain immune dysregulations (Kleineczek et al., 2016). The most common pathogens associated with the Zika virus (Cao et al., 2016). When the peripheral blood of patients with GBS have been examined, it reveals abnormalities in T cells, antibodies and gene expression (Blum & McCombe, 2014). Even with rapid treatment, GBS patients can experience weakness and with severe disability (Korneva, 2004).

**Implications for Pathophysiology**

Overall, GBS is thought to be a disease due to immunity between pathogens expressed on 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 recent research however, no specific HLA has been identified (Blum, Courths, Reddal, Spies, & McCombe, 2013). In demyelinating diseases antibodies bind 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 sheath (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.

**Conclusion**

Guillain-Barré syndrome diagnosis, treatment and management of related sequelae requires a multidisciplinary approach, supportive care and early mobility (Pasanen, 2015). Rarely are common and frequently follow vaccinations or illness, however, GBS occurring after the initial insult (Kleineczek & Kouriwski, 2014). Despite significant advances that have been made in treatment, GBS mortality remains steady at 4%-15% and thus, early diagnosis, appropriate treatment and prevention of complications are of the utmost importance.

**References**


**Figure 1. Course of Guillain-Barré Syndrome (Williams, Jacobs, & van Dooren, 2014)**

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

**Significance of Pathophysiology**

Guillain-Barré syndrome, GBS is described as an acquired neuropathy characterized by inflammation of peripheral nerves (Blum, Courths, Reddal, Spies, & McCombe, 2013). Pathophysiology of GBS ranges from antibody mediated disease to macrophage and T cell induced destruction of peripheral nerve cells (Blum, Courths, Reddal, Spies, & McCombe, 2013). KIR/HLA receptors and exhibit roles in both inhibitory and activating processes (Blum, Courths, Reddal, Spies, & McCombe, 2013). KIRs are found on the surface of many NK cells, but also on B cells and T cells. T lymphocytes (Blum, Courths, Reddal, Spies, & McCombe, 2013). CD4+ T lymphocytes are important in the KIR/HLA combinations in GBS to the sequence that ingests immunity plays a significant role in the disease process.

The details of the pathophysiology vary by subtypes. AIDP is the most common variant accounting for 85% of GBS cases. 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 also impacted. Communication to the patient regarding what care is being provided, why and by whom is especially important to this patient population to support their emotional well-being. The recovery phase of GBS can last up to 18 months, with some patients having permanent disability.

- Appropriate Nursing Interventions:
  - Turning and positioning to prevent pressure areas
  - Consistently deep breathing exercises
  - Thorough skin care
  - Maintain a moist skin.
  - Passive range of motion
  - Provide emotional support as needed

**Definitions**

- AIDP: Acute inflammatory demyelinating polyradiculoneuropathy
- AMAN: Acute motor axonal neuropathy
- AIDP: Acute inflammatory demyelinating polyradiculoneuropathy
- CIDP: Chronic inflammatory demyelinating polyneuropathy
- GBS: Guillain-Barré syndrome
- MFS: Miller Fisher syndrome
- T cell: Lymphocytes of the immune system that play a role in the cellular immune response
- B cell: Lymphocyte of the immune system that plays a role in the humoral immune response
- NK cell: Natural killer cell
- Macrophage: Large immune cells that engulf and destroy cellular debris
- Antibody: Protein produced by the immune system in response to an antigen
- Complement: Sequence of plasma proteins that work together to destroy invaders

**References, cont.**


