**Institution of affiliation**

**Guillain-Barre Syndrome**

Guillain-Barre syndrome is a disorder in which the body’s immune system attacks the nerves (Mayo, 2017). Among the first symptoms include tingling sensations in the leg and varying weakness in the body. The symptoms can easily spread throughout the body and paralyze it. The signs and symptoms include prickling sensations in fingers, toes, ankles and wrists, weakness in legs that spreads to upper body, unsteadiness in walking, difficulty with facial and eye movements, low or high blood pressure, severe pain that feels itchy or cramp like, difficulty in bladder control, rapid heart rate and difficulty in breathing (Mayo, 2017).

Guillain-Barre is rare, and according to the National Institute of Neurological Disorders and Stroke, it affects only 1 in 100,000 Americans (NINDS, 2017). There are several types of the disorder, but the most common form is the acute inflammatory demyelinating polyradiculoneuropathy (AIDP), which leads to damage to myelin (Mayo, 2017). Other types includes Miller Fisher Syndrome, which attacks the cranial nerves and acute motor axonal neuropathy (AMAN) (Mayo, 2017).

**History of first diagnosis**

It was first discovered by Georges Guillain, Jean Alexandre Barre and Andre Strohl when they diagnosed two soldiers with the illness in 1916 (Kusunoki, 2016). They described the key abnormalities, which was albuminocytological dissociation. The doctors were faced with three similar conditions with different definitions: Landry’s ascending paralysis, acute febrile neuropathy and radiculoneuritis. However, the term Guillain-Barre syndrome was used in 1927 at a presentation by Dragonescu and Claudian (Kusunoki, 2016).

**Causes of GBS**

The cause has not been established, but the disorder is always preceded by an infectious illness such as stomach flu and respiratory infection. Basing on data from Centers for Disease Control and Prevention (CDC), about 66% of victims develop the symptom after experiencing diarrhea or a respiratory infection after days or weeks (Mayo, 2017). There are also been reported cases following Zika virus infection. It’s therefore likely that the disorder may be caused by an improper immune response to a previous illness. Guillain-Barre syndrome has been associated with *Campylobacter jejuni* infection (NINDS, 2017). It’s a common risk factor for Guillain-Barre, and a widespread bacterial cause for diarrhea in the United States. The infection is found in undercooked food, especially poultry.

Other infections associated with the disorder includes influenza, cytomegalovirus, Epstein-Barr virus, mycoplasma pneumonia and HIV/AIDS (Mayo, 2017). In AIDP, the nerves protective covering is destroyed, which prevent nerves from sending signals to the brain, causing weakness, numbness and paralysis.

**Treatment and prevention**

The syndrome has no cure, but treatment can alleviate the severity of the symptoms and reduce the duration of the illness. Anyone with the symptoms of Guillain-Barre symptoms can be admitted to a hospital for diagnosis and observation. The symptoms can be fatal if left untreated. In extreme cases, full-body paralysis can affect the victims. The disorder can be life-threatening if paralysis affects the diaphragm or chest muscles, hindering breathing. The disorder can resolve on its own, hence it’s described as an autoimmune inflammatory process that’s self-limiting. The objective of the treatment is to reduce the severity of the immune attack and support body functions while the nervous system recovers (Mayo, 2017). Treatment options includes plasma exchange, intravenous immunoglobulin and pain relieve medication (Mayo, 2017). Plasma exchange removes the antibodies attacking the nervous system from your blood. Intravenous immunoglobulin helps to block the anti-bodies causing Guillain-Barre symptom. Physical therapy is also a treatment option, whereby caretakers will move your arms and legs to keep them flexible. Physical therapists also work on muscle strengthening and personal care activities.

**Myasthenia Gravis**

Myasthenia Gravis is a neuro-muscular disease that is characterized by varying degrees of weakness in skeletal muscle (Herndon, 2016). The commonly affected muscles are the ones in the eyes, face and throat. When the nerve cells and the muscles have impaired communication between them, and such condition prevents crucial muscle contractions, leading to muscle weakness. Basing on findings from Myasthenia Gravis Foundation of America, myasthenia gravis is the most common neuro-muscular disorder. It affects about 14 to 20 people per 100,000 people in the United States (F.Howard, 2015).

The main symptom is weakness in the voluntary skeletal muscles, but other symptoms includes speech problems, facial paralysis, difficulty in breathing, fatigue, drooping of eyelids and double vision (Herndon, 2016).

**History of first diagnosis**

Virginian chroniclers in 1664 was the first to describe it following the death of a Native American Chief Opechancanough who had shown symptoms relating to the disorder. He had excess fatigue, drooping eyelids, and loss of muscle control. In 1672, English physician Thomas Willis talked of a patient who had weakness in limbs and problematic speech. In the late 1800s, the name myasthenia gravis was coined by combining the Greek terms for muscle and weakness to yield myasthenia, and adding the Latin word gravis meaning severe.

**Causes of Myasthenia Gravis**

It is caused by an autoimmune problem, but the exact cause of the autoimmune attack is still not known. It occurs when communication between the nerve and muscle is interrupted at neuromuscular junction, where nerve cells connect with the muscles (Herndon, 2016). Neurotransmitters are chemicals that brain cells use to communicate information. When electrical signals goes through a motor nerve, the nerve endings release acetylcholine, which is a neurotransmitter (Herndon, 2016). Acetylcholine travels from nerve endings to the receptors on the muscle. The binding of the neurotransmitter to the receptor activates the muscle and causes it to contract. According to Muscular Dystrophy Association, it’s possible that certain viral or bacterial proteins may make the body to attack acetylcholine (MDA, N.A). In the disorder, the immune system produces antibodies that block the muscles’ receptor sites for a neurotransmitter called acetylcholine. As the receptor sites get fewer, the muscles receive fewer nerve signals, leading to weakness. The antibodies may also block the function of a protein called tyrosine kinase, which is involved in forming the nerve-muscular junction. As the antibodies block the function of this protein, it may lead to myasthenia gravis.

**Treatment and prevention**

Myasthenia gravis has no cure. However, treatment can help alleviate the signs and symptoms such as weakness of arms and leg muscles, double vision, drooping eyelids and difficulties in facial movements and body functions such as chewing, breathing and swallowing. The role of treatment is to alleviate the symptoms and control the reaction to the immune system. Treatment option includes medication, thymus gland removal, plasma exchange, intravenous immune globulin and lifestyle changes (Herndon, 2016). Medications such as corticosteroids and immunosupprssants can minimize the abnormal immune response. Removal of thymus gland has been observed and patients typically exhibit less muscle weakness. Plasma exchange removes harmful antibodies from blood, resulting in improvement in muscle strength. Intravenous immune globulin affects the creation and function of antibodies, hence essential in treatment of autoimmune myasthenia gravis. Lifestyle changes such as plenty of sleep and avoiding stress and exposure to heat will lessen the symptoms (Herndon, 2016).

**References**

F.Howard, J. (2015, June). *Clinical Overview of MG.* Retrieved from myasthenia.org: http://www.myasthenia.org/HealthProfessionals/ClinicalOverviewofMG.aspx

Herndon, J. (2016, February 24). *Myasthenia Gravis.* Retrieved from healthline: https://www.healthline.com/health/myasthenia-gravis

Kusunoki, S. (2016). History of Guillain–Barré syndrome. *Neuroimmunology*, 305-311.

Mayo. (2017, October 17). *Mayo Clinic.* Retrieved from Guillain-Barre syndrome: https://www.mayoclinic.org/diseases-conditions/guillain-barre-syndrome/symptoms-causes/syc-20362793

MDA. (N.A). *Myasthenia Gravis (MG).* Retrieved from mda.org: https://www.mda.org/disease/myasthenia-gravis/causes-inheritance

NINDS. (2017, May 10). *Guillain-Barré Syndrome Fact Sheet.* Retrieved from NINDS: https://www.ninds.nih.gov/Disorders/Patient-Caregiver-Education/Fact-Sheets/Guillain-Barr%C3%A9-Syndrome-Fact-Sheet