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Myasthenia Gravis and implications for the Certified Registered Nurse Anesthetist

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Introduction

In healthcare, particularly in anesthesia, there are several considerations that require various careful assessments by the practitioner. One example of such a condition requiring special attention is myasthenia gravis. Myasthenia gravis is an autoimmune disease in which skeletal muscle weakness and rapid fatigability result from destruction of nicotinic acetylcholine receptors (7). The disease affects approximately 150 to 200 thousand people in the United States (7) and 20 to 60 per 100,000 worldwide (9). The mechanism of the management of those with this disease is often extremely serious in nature. Understanding the pathophysiology, various treatments (both surgical and medical), and anesthetic implications can result in better outcomes for individuals with myasthenia gravis that require surgery.

Case Study

Mr. Smith, a 52-year-old male, presents to his primary care physician having been evaluated elsewhere. He hasn’t seen a similar practitioner for nearly 10 years because not only does he need a physical before his hip replacement surgery planned for next month, but also because he has not been feeling well. Mr. Smith has been experiencing generalized muscle weakness at times, leading him to feel short of breath. In addition to this, he has been noticing ptosis of both his eyelids and drooping of his eyelids with rest. Mr. Smith has also been experiencing double vision, a blurring of his vision, heart difficulty, weakness, and drooping of his eyelids, perceived as negative, including normal glucose and thyroid panel, and a thorough neurological examination was performed. Mr. Smith’s physician suspected myasthenia gravis so he ordered a tensilon test, which confirmed the diagnosis of myasthenia gravis. Mr. Smith presents with classic symptoms of myasthenia gravis and is within the typical age range for disease presentation in men, which is between 50 and 60 years of age (8). Weakness commonly affects the muscles of one or both eyes, including eyelids, lips, and diaphragm (4) and the age of onset in men is typically Visual disturbance. This is seen in more than half of patients with myasthenia gravis. Fatigue that resolves with rest is often resistant to this medication, necessitating increased dosages to achieve acceptable paralyzation (4). Approximately 150 to 200 vesicles that contain nearly 10,000 molecules of acetylcholine (7). The rapid influx of sodium ions depolarizes the motor end plate and skeletal muscle, which allows for muscular contraction (9). The following scheme depicts how and acetylcholine’s role in action potential propagation.

Pathophysiology

To understand the pathophysiology of myasthenia gravis, one must first have an understanding of skeletal muscle depolarization and the receptors involved. When an electrical impulse reaches the presynaptic nerve terminal, an influx of calcium triggers the release of proxysmatic acetylcholine. Approximately 230 to 200 vesicles that contain nearly 10,000 molecules of acetylcholine each are released into the synaptic cleft and bind to nicotinic acetylcholine receptors. Nicotinic acetylcholine receptors are located on postticularic end plates, which are located in close proximity to skeletal muscle. The binding of acetylcholine to the alpha subunit of the nicotinic acetylcholine receptor causes a conformational change in the receptor, opening a channel that allows sodium ions to enter the synapse. The rapid influx of sodium ions depolarizes the motor end plate and skeletal muscle, which allows for muscular contraction (8). The following scheme depicts how and acetylcholine’s role in action potential propagation.

Significance of Pathophysiology

Most frequent symptom is muscle weakness.

- Weakness occurs when 70%-90% of nicotinic acetylcholine receptors are lost (7).
- Myasthenia gravis can be classified, or according to severity and muscles affected by the Osborn-Schlegel system shown below.

Nursing Implications

Increased numbers of nicotinic acetylcholine receptors and the associated skeletal muscle weakness have important implications for the nurse anesthetist caring for the patient with myasthenia gravis perioperatively. A medication type frequently utilized by the nurse anesthetist to facilitate tracheal intubation and provide a protected surgical field for the surgeon is neuromuscular blockers (NMBs). The two types of paralytics utilized by the nurse anesthetist are depolarizing NMBs and non-depolarizing NMBs (11). The only depolarizing paralytic NMB in today’s market is succinylcholine (6). Chemically, succinylcholine is composed of two molecules of choline and one molecule of succinic acid (5). Succinylcholine (a depolarizing paralytic) will propagate further action potentials from propagating it from depolarize in that it is not metabolized by pseudocholinesterase but rather by a non-specific esterase (8). Because of the similarities between succinylcholine and acetylcholine, it is not surprising that patients with myasthenia gravis are often resistant to this medication, necessitating increased dosages to achieve acceptable paralyzation (9). Approximately 230 to 200 vesicles that contain nearly 10,000 molecules of acetylcholine (known as diacetylcholine). As mentioned earlier, myasthenia gravis is an autoimmune disease in which antibodies result from autoantibodies against acetylcholine receptors and other proteins resulting in their destruction. Specifically, nearly 65% of people with myasthenia gravis have antibodies that target nicotinic acetylcholine receptors. Of the 15% that do not have anti-acetylcholine antibodies, 70% have antibodies targeting other proteins involved in neurotransmitter transmission that includes muscle-specific tyrosine kinase. Regardless of the type of autoantibody muscle membrane destruction is caused by antibody activation of complement, disintegration of the extracellular matrix, and deactivation of the postsynaptic fold (the primary location of nicotinic acetylcholine receptors). Investigation of thymus abnormalities (thymomas, thymus, or hyperplasia) is warranted in patients with myasthenia gravis because the thymus major site for antibody production (2). As mentioned earlier, myasthenia gravis is an autoimmune disease in which antibodies result from autoantibodies against acetylcholine receptors and other proteins resulting in their destruction. Specifically, nearly 65% of people with myasthenia gravis have antibodies that target nicotinic acetylcholine receptors. Of the 15% that do not have anti-acetylcholine antibodies, 70% have antibodies targeting other proteins involved in neurotransmitter transmission that includes muscle-specific tyrosine kinase. Regardless of the type of autoantibody muscle membrane destruction is caused by antibody activation of complement, disintegration of the extracellular matrix, and deactivation of the postsynaptic fold (the primary location of nicotinic acetylcholine receptors). Investigation of thymus abnormalities (thymomas, thymus, or hyperplasia) is warranted in patients with myasthenia gravis because the thymus major site for antibody production (2). Myasthenia gravis is a disease process that can cause profound muscle weakness both perioperatively and in day-to-day life. Understanding the pathophysiology of the disease, as in the case of Mr. Smith, can provide the nurse anesthetist with so as to optimally manage the anesthetic.

References