



MYASTHENIA GRAVIS

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ABSTRACT

Myasthenia gravis is an autoimmune disease of the neuromuscular junction characterized by the fluctuating weakness of certain skeletal muscle groups. In approximately 80% of patients, auto-antibodies to the muscle nicotinic acetylcholine receptor (AChR) are present. These antibodies cause loss of AChR numbers and function, and lead to failure of neuromuscular transmission with muscle weakness. The pathogenic mechanisms acting in the 20% of patients with generalized MG who are seronegative for AChR-antibodies (AChR-Ab) have not been elucidated, but there is evidence that they also have an antibody-mediated disorder, with the antibodies directed towards another, previously unidentified muscle-surface-membrane.

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INTRODUCTION

Myasthenia gravis is an uncommon condition that causes certain muscles to become weak. With treatment, most people can lead a normal life. Myasthenia gravis literally means 'grave muscle weakness'. The condition can affect any muscles that you can control voluntarily. Muscles that you cannot control voluntarily, such as the heart muscles, are not affected. Myasthenia gravis most commonly affects the muscles that control eye and eyelid movement, facial expression, chewing, swallowing and talking, and the muscles in the arms and legs. Less often, the muscles involved in breathing may be affected. The muscle weakness is usually made worse by physical activity and improved by rest.

Definition

A disease in which antibodies made by a person's immune system prevent certain nerve-muscle interactions. It causes weakness in the arms and legs, vision problems, and drooping eyelids or head.

Lewis

Incidence

A changing pattern of MG incidence with an increase in frequency of late-onset and a decrease of early onset MG was found in the last years, giving a significant shift to older age at onset of the disease. Unknown environmental factors may have driven this change in MG.

Causes

1. Autoantibodies block the receptors of acetylcholine in neuromuscular junction.
2. Autoantibodies against MuSK protein- tyrosine kinase receptor which helps in neuromuscular junction formation.
3. Thymoma-enlargement of thymus.
4. Rare hereditary form of myasthenia gravis.

Pathophysiology

MG is an autoimmune synaptopathy. The disorder occurs when the immune system malfunctions and generates antibodies that attack the body's tissues. The antibodies in MG attack a normal human protein, the nicotinic acetylcholine receptor, or a related protein called MuSK a muscle-specific kinase. Other less frequent antibodies are found against LRP4, Agrin and titin proteins.

Human leukocyte antigen (HLA) haplotypes are associated with increased susceptibility to myasthenia gravis and other autoimmune disorders. Relatives of people with MG have a higher percentage of other immune disorders.

The thymus gland cells form part of the body's immune system. In those with myasthenia gravis, the thymus gland is large and abnormal. It sometimes contains clusters of immune cells which indicate lymphoid hyperplasia, and the thymus gland may give wrong instructions to immune cells.

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Clinical Manifestation

Eye muscle

- Drooping of one or both eyelids (ptosis)
- Double vision (diplopia), which may be horizontal or vertical, and improves or resolves when one eye is closed

Face and throat muscles

- **Impair speaking.** Your speech might sound soft or nasal, depending on which muscles have been affected.
- **Cause difficulty swallowing.** You might choke easily, making it difficult to eat, drink or take pills. In some cases, liquids you're trying to swallow come out your nose.
- **Affect chewing.** The muscles used for chewing might wear out halfway through a meal, particularly if you've been eating something hard to chew, such as steak.
- **Change facial expressions.** For example, your smile might look like a snarl.

Neck and limb muscles

Myasthenia gravis can also cause weakness in your neck, arms and legs. Weakness in your legs can affect how you walk. Weak neck muscles make it hard to hold up your head.

Diagnostic Evaluation

- ✓ Neurological examination
- ✓ Physical examination
- ✓ Blood test for antibodies against acetylcholine receptor
- ✓ CT chest and x-ray chest for showing a thymoma
- ✓ Single fiber electromyography
- ✓ Ice test
- ✓ Edrophonium test
- ✓ Pulmonary function test

Management

Treatment is by medication and/or surgery. Medication consists mainly of acetyl cholinesterase inhibitors. To directly improve muscle function and immunosuppressant drugs to reduce the autoimmune process. Thymectomy is a surgical method to treat MG.

Medication

Acetyl cholinesterase inhibitor

Acetyl cholinesterase inhibitors can provide symptomatic benefit and may not fully remove a person's weakness from MG. While they might not fully remove all symptoms of MG, they still may allow a person the ability to perform normal daily activities. Usually, acetyl cholinesterase inhibitors are started at a low dose and increased until the desired result is achieved. If taken 30 minutes before a meal, symptoms will be mild during eating, which is helpful for those who have difficulty swallowing due to their illness. Another medication used for MG, atropine, can reduce the muscarinic side effects of acetyl cholinesterase inhibitors. Pyridostigmine is a relatively long-acting drug (when compared to other cholinergic agonists), with a half-life around four hours with relatively few side effects. Generally, it is discontinued in those who are being mechanically ventilated as it is known to increase the amount of salivary secretions. A few high-quality studies have directly compared cholinesterase inhibitors with other treatments (or placebo); their practical benefit may be

such that it would be difficult to conduct studies in which they would be withheld from some people.

Immune suppressants

The steroid prednisone might also be used to achieve a better result, but it can lead to the worsening of symptoms for 14 days and takes 6–8 weeks to achieve its maximal effectiveness. Due to the myriad symptoms that steroid treatments can cause, it is not the preferred method of treatment. Other immune suppressing medications may also be used including rituximab.

Plasmapheresis and IVIG

If the myasthenia is serious (myasthenic crisis), plasmapheresis can be used to remove the putative antibodies from the circulation. Also, intravenous immunoglobulins (IVIGs) can be used to bind the circulating antibodies. Both of these treatments have relatively short-lived benefits, typically measured in weeks, and often are associated with high costs which make them prohibitive; they are generally reserved for when MG requires hospitalization.

Surgery

As thymomas are seen in 10% of all people with the MG, people are often given a chest X-ray and CT scan to evaluate their need for surgical removal of their thymus and any cancerous tissue that may be present. Even if surgery is performed to remove a thymoma, it generally does not lead to the remission of MG. Surgery in the case of MG involves the removal of the thymus, although in 2013 there was no clear indication of any benefit except in the presence of a thymoma. A 2016 randomized controlled trial, however, found some benefits.

Physical measures

People with MG should be educated regarding the fluctuating nature of their symptoms, including weakness and exercise-induced fatigue. Exercise participation should be encouraged with frequent rest. In people with generalized MG, some evidence indicates a partial home program including training in diaphragmatic breathing, pursed-lip breathing, and interval-based muscle therapy may improve respiratory muscle strength, chest wall mobility, respiratory pattern, and respiratory endurance.

Medical imaging

In people with myasthenia gravis, older forms of iodinated contrast used for medical imaging have caused an increased risk of exacerbation of the disease, but modern forms have no immediate increased risk.

CONCLUSION

The prognosis of people with MG is generally good, as is quality of life, given very good treatment. Monitoring of a person with MG is very important, as at least 20% of people diagnosed with it will experience a myasthenic crisis within two years of their diagnosis, requiring rapid medical intervention. Generally, the most disabling period of MG might be years after the initial diagnosis. In the early 1900s, 70% of detected cases died from lung problems; now, that number is estimated to be around 3–5%, which is attributed to increased awareness and medications to manage symptoms.

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