IJACT, Volume 3, Issue 1, 2025 ISSN: 2995-5378
http://medicaljournals.eu/index.php/IJACT

INTERNATIONAL JOURNAL OF ALTERNATIVE AND CONTEMPORARY THERAPY

Complications, origin and diagnostics of myasthenia gravis in a modern interpretation

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Abstract: Sometimes patients complain of muscle weakness, which reduces their quality of life. A person cannot work at a computer for a long time, because due to muscle weakness the upper eyelid droops, or he notices that his speech changes, becomes slow and nasal during a long conversation. Such symptoms require a full examination to exclude serious diseases, one of which is myasthenia gravis. This disease is manifested by objective pathological muscle weakness. Myasthenia gravis comes from the Greek. myasthenia - muscle + impotence, gravis - heavy, serious. In this article we will tell you what myasthenia gravis is, the symptoms of the disease and methods of treatment. This article is advisory in nature. Treatment is prescribed after consulting a specialist.

Keywords: Myasthenia gravis, Myasthenia gravis chronic autoimmune disease, effects on neuromuscular junction, effects on muscle weakness and fatigue

The neuromuscular junction is the specific connection between a nerve and a muscle where the conversion of a nerve impulse into muscle movement occurs. With myasthenia gravis, this conversion is difficult: because antibodies attack the receptors that transmit nerve impulses to the muscles. This leads to muscle weakness and other characteristic symptoms.

The neuromuscular junction consists of the following parts:

Axon of a motor neuron. This is the process of a nerve cell that contacts a muscle fiber and through which a nerve impulse is transmitted.

Presynaptic membrane. At the end of the axon there is a thickening covered with a special membrane, in which there are a large number of vesicles, vesicles containing the substance acetylcholine.

Synaptic cleft. This is the space between the presynaptic and postsynaptic membranes where acetylcholine is released.

The synaptic cleft contains acetylcholinesterase, which destroys acetylcholine residues.

Postsynaptic membrane. This is a section of muscle tissue where acetylcholine binds to receptors, which activates special ion channels through which sodium ions flow, thereby stimulating muscle motor activity.

Structure of the neuromuscular synapse

Acetylcholine is a chemical that is released from the presynaptic membrane as a nerve impulse travels along an axon. It is this that enables muscle contraction and movement.

Antibodies play a major role in the development of myasthenia gravis. These are proteins of the immune system that are produced to fight pathogens such as viruses. In autoimmune diseases, such as

myasthenia gravis, antibodies are directed against healthy cells of the body, destroying and disrupting their function. Myasthenia gravis antibodies block acetylcholine receptors in 85% of cases. Thus, acetylcholine cannot bind to the receptors and is destroyed by acetylcholinesterase located in the synaptic cleft. As a result, neuromuscular transmission is disrupted.

In rare cases, antibodies are directed not against acetylcholine receptors but against muscle tyrosine kinase (MuSK), a specific enzyme that is also necessary for the transmission of nerve impulses to muscles.

Myasthenia: causes of occurrence and mechanism of development in humans

The reasons for the development of synapse attack are not fully known, but there is convincing evidence of the influence of the thymus gland or thymus on the development of myasthenia gravis.

The thymus is an organ of the immune system responsible for the formation and "training" of immune cells. It is well developed in children. With age, the thymus decreases in size and is replaced by fatty tissue, which is called thymic involution. In 65% of patients with myasthenia gravis, an enlarged thymus is its hyperplasia, and in 10%, a tumor of the thymus is called thymoma. It is most often benign, but sometimes it is malignant. In other cases of myasthenia gravis, changes in the thymus gland are not detected.

Based on these results, it is believed that under such conditions, the thymus gives incorrect "instructions" to the immune system, and it begins to attack itself.

Myasthenia gravis is divided into the following depending on the development:

Congenital myasthenia gravis

This form is hereditary and results from mutations in genes responsible for the structure and function of the neuromuscular synapse.

Acquired myasthenia gravis

This is the most common form of the disease. It is autoimmune and is associated with an enlarged thymus gland or a tumor called a thymoma.

Small cell lung cancer has a condition called Lambert-Eaton syndrome, which is very similar to myasthenia gravis. It is also caused by an autoimmune attack on calcium ion channels, which reduces or completely blocks the release of acetylcholine into the synaptic cleft. The symptoms of true myasthenia gravis are different from Lambert-Eaton myasthenic syndrome, which is characterized by:

Weakness of the arm and leg muscles - they are affected first

Pain in the upper arms and legs

Dry mouth

Drooping eyelids

The disease begins at a young age, before the age of 40. For every sick man, there are three women. After the age of 50, symptoms of myasthenia gravis in women often manifest themselves in the form of weakness of the eyes, face and chewing muscles. Men suffer from myasthenia gravis less often and usually after the age of 60. Symptoms can be divided into groups depending on the form of the disease. There are 3 forms of myasthenia gravis:

a) Eye

b) Bulbul

c) Generalized

Classic symptoms of the ocular form of myasthenia gravis:

Drooping of the upper eyelid - ptosis. Occurs due to weakness and rapid fatigue of the muscle that lifts the upper eyelid

Double vision - diplopia. It is caused by weakness of the extraocular muscles.

Symptoms worsen after prolonged eye strain: watching TV, reading, driving, or working on a computer.

Ptosis is the most prominent sign of myasthenia gravis.

Symptoms of bulbar myasthenia gravis:

Change in facial expression. A sad look on the face is obtained due to weakness of the facial muscles. Possible sagging of the lower jaw due to the inability of the facial muscles to hold it

Difficulty chewing. The process takes a long time due to weakness and rapid fatigue of the chewing muscles

Swallowing disorder - dysphagia. Due to weakness of the pharyngeal muscles, the movement of the food bolus is impaired, food can enter the nose, which leads to choking.

Speech disorder - dysarthria. During a long conversation, speech gradually slows down, becomes slurred, and becomes nasal.

Myasthenia gravis, the common form, is characterized by:

Weakness of the muscles of the extremities, manifested by difficulty climbing stairs, getting out of bed, out of a chair, or raising the arms overhead.

Weakness and fatigue of all muscles gradually increase, usually the eye muscles are affected first, then the muscles of the face, pharynx and limbs. Possible progressive damage to the respiratory muscles, which requires urgent medical attention

Symptoms in all forms decrease after rest and increase after muscle tension, as well as in a cold room.

Factors that trigger the symptoms of myasthenia gravis:

a) stress

b) infectious diseases

c) operation

d) pregnancy

Separately, it is worth considering myasthenic crisis - this is a life-threatening complication of the disease. The condition occurs in an acute form and is characterized by weakness of the respiratory muscles, which leads to severe shortness of breath, respiratory failure and even respiratory arrest. Myasthenic crisis requires emergency care and artificial ventilation.

Treatment of myasthenia gravis

Myasthenia gravis cannot be cured. There are drugs that stabilize the condition and prevent the development of symptoms. The question of how to treat myasthenia gravis is most often answered by neurologists. After all, it is they who treat myasthenia gravis and have conservative methods in their arsenal, and in some cases it is necessary to resort to a surgeon.

The medical approach includes:

Acetylcholinesterase inhibitors. Acetylcholinesterase is an enzyme that breaks down acetylcholine residues in the synaptic cleft. Reducing its activity helps acetylcholine to penetrate more and bind to muscle receptors, thereby preventing the rapid development of weakness and fatigue. Usually prescribed for mild myasthenia gravis and the ocular form of the disease.

Immunosuppressive drugs - glucocorticosteroids are prescribed for severe bulbar myasthenia gravis. It should be remembered that dexamethasone, a drug from the glucocorticosteroid group, should be taken with caution, as it inhibits the release of acetylcholine, which increases muscle weakness in the first days of treatment.

Cytostatic drugs, like glucocorticosteroids, slightly inhibit the activity of the immune system, thereby inhibiting the release of antibodies to receptors.

Plasmapheresis. In plasmapheresis, blood plasma containing large amounts of antibodies directed against acetylcholine receptors is passed through a special machine that "cleans" the plasma of abnormal antibodies.

Intravenous immunoglobulin. Immune system proteins are injected into the blood and bind to "harmful" antibodies, thereby preventing their interaction with acetylcholine receptors. Plasmapheresis and infusions are often used for exacerbations of myasthenia gravis.

Monoclonal antibodies. These are a newer class of drugs that directly affect the immune system and are used for severe generalized forms of myasthenia gravis.

Eculizumab is also known as Soliris and Elizaria. The drug contains antibodies that interact with the immune system. By binding to one of its components, the drug prevents the breakdown of the neuromuscular junction that occurs in myasthenia gravis. It is important to remember one serious side effect of this drug - a decrease in immunity, which makes the body susceptible to infections, including meningococcal infections. Therefore, the drug is prescribed under the strict supervision of a doctor and only if the patient has been vaccinated against meningococcal infection.

Efgartigimod is under the trade name "Vivgart". The newest drug for the treatment of myasthenia gravis. Its mechanism of action is similar to plasmapheresis. This is a type of plasmapheresis in a vial. The drug blocks the receptor responsible for the circulation of immunoglobulin G in the blood, thereby reducing not only the total amount of this immunoglobulin, but also the abnormal antibodies produced in myasthenia gravis.

Monoclonal antibody drugs are expensive and are only purchased and sold by the government. These drugs are prescribed only for myasthenia gravis with antibodies to the acetylcholine receptor, and their use is strictly monitored by a doctor to avoid serious side effects;

Surgical treatment consists of removing the thymus gland - thymectomy. This is one of the most effective methods of treating myasthenia gravis in young women with a short-term course of the disease, less than 1 year, and high titers of antibodies to the acetylcholine receptor. Removal of the thymus is also indicated in progressive forms of myasthenia gravis. Regardless of the antibody titer and symptoms, removal of the thymus is indicated in the presence of thymoma - its tumor.

Diagnosis of myasthenia gravis

Diagnosis begins with a conversation with the patient, the doctor determines his complaints and determines the severity of the symptoms of the disease. Next, he conducts an examination with a mandatory examination of the neurological status: assessment of muscle strength, muscle tone, reflexes, sensitivity and coordination of movements.

Functional tests

To identify pathological muscle fatigue, the following is done:

Ice test for diagnosing myasthenia gravis. The patient is placed on the overhanging eyelid with an ice pack for 2 minutes, and the test is considered positive if the eyelid rises 2 mm or more after the ice is removed. This occurs because exposure to cold in myasthenia gravis reduces muscle weakness

To diagnose the general form, perform a fist test. After several repeated fists, the patient will feel weakness in the hand.

Instrumental diagnostics

The most important instrumental method for diagnosing myasthenia gravis is electroneuromyography, namely its special "reduction-test" mode. In this mode, electrical impulses emitted by the device cause muscle contractions at a high frequency. With myasthenia gravis, the force of contractions decreases due to progressive fatigue of muscle fibers.

Computed tomography allows you to detect an enlarged thymus or its tumor - thymoma.

Respiratory function should be assessed using spirometry. This is necessary to analyze the work of the respiratory muscles, whether they are affected or not, their function is affected.

Laboratory research

A serological blood test, known as an enzyme-linked immunosorbent assay (ELISA), is used to detect antibodies to the acetylcholine receptor and MuSK protein. In addition, a study may be performed to look for antibodies to skeletal muscle, but their detection is not specific, as they can be detected in other autoimmune diseases and in the presence of thymic tumors without myasthenia gravis.

To rule out myasthenic syndrome in small cell lung cancer, blood is tested for antibodies to calcium channels.

To exclude other pathologies that are manifested by muscle weakness, for example: myositis - inflammation of the muscles, thyrotoxic myopathy in thyroid diseases, myotonia - additional laboratory tests are used. This is a biochemical blood test to determine the level of creatine phosphokinase and potassium, which increase with myositis. Determination of the level of thyroid hormones, their increase may be accompanied by pseudomyasthenic symptoms.

Consequences of myasthenia gravis

In the conditions of modern medical care, the prognosis of myasthenia gravis is favorable, even in bulbar and generalized forms. To maintain health, it is necessary to constantly monitor the course of the disease and start treatment in a timely manner. This will ensure long-term remission.

Monitoring the condition is important because 20% of patients with myasthenia gravis will experience a myasthenic crisis within 2 years of diagnosis, which requires immediate medical attention due to the risk of respiratory arrest and cardiac arrest.

A note about contraindications for myasthenia gravis

Certain activities and medications can aggravate myasthenia gravis and increase its symptoms. Therefore, patients should be aware of what restrictions to follow, what medications to take with caution, and what to avoid altogether:

a) Avoid excessive exercise.

b) Limit sun exposure

c) Avoid magnesium preparations - magnesia, panangin, asparkam

d) Avoid taking muscle relaxants.

e) Neuroleptics and tranquilizers are also contraindicated - benzodiazepines, lithium preparations

f) All diuretics except spironolactone are contraindicated.

Some antibiotics, such as aminoglycosides and fluoroquinolones, and antimalarials, such as quinine, should be avoided. Myasthenia gravis cannot be prevented.

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