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Myasthenia: Issues of Pathogenesis and Treatment

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Abstract: Myasthenia, characterized by periodically occurring excessive weakness and fatigue of striated muscles, was classified as a nosological entity more than 100 years ago. Despite the long period since its recognition, a number of fundamental issues concerning the etiology, pathogenesis, clinical presentation, and treatment of this disease require further detailed study and investigation.

Keywords: myasthenia, etiology, pathogenesis, clinical diagnosis, treatment.

The first mentions of symptoms similar to myasthenia can be found in the works of ancient Greek and Roman physicians. Although the term "myasthenia" itself was introduced only in the 19th century, descriptions of conditions resembling myasthenia already appear in ancient sources [1].

- Hippocrates (circa 460–370 BCE) – In his works, he refers to diseases accompanied by muscle weakness, which could be closely related to the modern understanding of myasthenia.
- Galen (circa 129–200 CE) – A Roman physician and philosopher, described various diseases of the nervous system, including disorders such as muscle weakness and fatigue, which may have been similar to myasthenia.

However, these ancient references do not reflect a complete understanding of myasthenia as a disease, since its causes and mechanisms became clear only in the 20th century. The term "myasthenia" as a nosological concept was introduced in 1877 by the English neurologist Thomas Williams. He used the term to describe a condition characterized by muscle weakness and fatigue. Later, in 1934, French physician Jean-Louis Benjamin first described myasthenia as an independent disease, and in the 1940s, the immunological mechanisms associated with the development of myasthenia were established [2].

This disease is a rare autoimmune disorder that causes weakness and fatigue of skeletal muscles. Typically, it is associated with impaired transmission of nerve impulses from nerve cells to muscles due to the production of antibodies that block or damage acetylcholine receptors on the surface of the muscles.

Prevalence of Myasthenia

1. Global Prevalence:

Incidence Rate: According to various studies, myasthenia gravis occurs in approximately 14–20 cases per 100,000 people in the general population. In some countries, such as the USA or Japan, this figure may be slightly higher or lower depending on diagnostic methods and the populations studied.

Geographic Variation: The prevalence of myasthenia can vary by geographic region, which is associated with different factors including genetic predisposition, availability of medical care, diagnostic approaches, and clinical traditions.

2. Types of Myasthenia:

Myasthenia Gravis (MG): The most common form, associated with autoimmune attack on acetylcholine receptors, leading to impaired neuromuscular transmission.

Neonatal myasthenia – a form that occurs in newborns if the mother has myasthenia, as antibodies can be transferred through the placenta.

Postprandial myasthenia – a rare form associated with nervous system dysfunction, more commonly observed in individuals with certain conditions such as cancer or infectious diseases.

3. Geographic Differences:

In some countries, such as the USA, myasthenia gravis occurs at a rate of approximately 15–20 cases per 100,000 people.

In Asian countries, such as Japan, the prevalence may be slightly lower.

In Europe, for example in the United Kingdom, the incidence is also about 15–20 cases per 100,000 people, but statistics in developing countries may be less accurate due to limited access to diagnostics and treatment [3].

Age Distribution of Myasthenia

1. Young Age (20–40 years):

- Myasthenia is most frequently diagnosed in young women aged 20 to 40 years, accounting for 50–70% of cases in this age group.
- In women, myasthenia typically has an autoimmune nature and usually presents with weakness in facial and ocular muscles (ptosis, diplopia), as well as difficulty swallowing and speaking.
- At this stage, patients may experience mild to moderate symptoms, which can worsen throughout the day and improve with rest.

2. **Middle Age (40–60 years):**

- Although myasthenia can still be diagnosed in this age group, cases are less frequent.
- However, disease severity may be greater in this age group, with more pronounced muscle weakness.
- Women still show a high incidence rate, but men in this age group may also develop myasthenia, particularly in cases of secondary myasthenia associated with thymic tumors (thymoma).

3. **Older Age (over 60 years):**

- Among individuals over 60, myasthenia occurs more often in men, and the clinical manifestations may be more varied.
- In this age group, the disease may develop in the context of secondary conditions, such as cancer (especially thymoma) or other autoimmune disorders.
- In older individuals, myasthenia often begins gradually with less prominent early symptoms, but it can lead to more serious respiratory and motor impairments if not treated in time.

Summary Table: Myasthenia Prevalence by Age Group

Age Group	Predominant Characteristics	Prevalence
20–40 years	Predominantly affects women, autoimmune form	High among women, less in men
40–60 years	Potentially more severe course, secondary forms (e.g., thymoma)	Moderate, more common in women
60+ years	Predominantly affects men, often associated with thymic tumors	Lower in women, higher in men

Characteristics of the Disease Course by Age:

- In young patients, myasthenia generally has a milder course, but it may be prone to progression.
- In elderly individuals, the symptoms may develop more slowly, but the disease can be more severe, especially if the respiratory muscles are involved.

Thus, age is an important factor influencing the manifestation of the disease and the choice of treatment.

Myasthenia Gravis: Etiology and Pathogenesis

Myasthenia gravis is a chronic autoimmune disease characterized by progressive weakness of skeletal muscles, caused by a disruption in nerve impulse transmission at the level of neuromuscular synapses. The etiology and pathogenesis of myasthenia involve a complex interaction of genetic, immunological, and possibly environmental factors.

Etiology of Myasthenia

1. Autoimmune Mechanism

The primary cause of myasthenia lies in an autoimmune reaction in which the immune system produces antibodies against acetylcholine receptors (**AChR**) or other components involved in neuromuscular transmission.

- Antibodies to acetylcholine receptors (**AChR**):

These antibodies, produced by the immune system, block or damage the acetylcholine receptors on the surface of muscle cells. Acetylcholine is a neurotransmitter essential for transmitting signals from nerve cells to muscles. When antibodies bind to these receptors, they impair their function, resulting in disrupted neuromuscular transmission.

- Antibodies to muscle-specific kinase (**MuSK**):

In 20–30% of myasthenia cases, antibodies are found that target muscle-specific kinase (**MuSK**) — a protein critical for the formation and maintenance of the neuromuscular junction. These antibodies interfere with normal synaptic function, also leading to impaired nerve signal transmission.

- Antibodies to **LRP4** (low-density lipoprotein receptor-related protein 4):

In some cases of myasthenia, antibodies to **LRP4** are detected. **LRP4** is a receptor that participates in the binding of acetylcholine to muscle receptors.

2. Immune Disorders

Myasthenia can develop against a background of immune system dysfunctions, such as:

- Increased T-cell activity:

Some studies show that patients with myasthenia exhibit increased T-lymphocyte activity, which plays a crucial role in triggering autoimmune responses.

- Role of the thymus:

The thymus gland, where T-lymphocytes mature, is key in the etiology of myasthenia. In most patients, thymic abnormalities such as hyperplasia (enlargement) or thymoma (thymic tumor) are observed. These changes contribute to the production of autoantibodies.

3. Genetic Predisposition

Although myasthenia is not considered a hereditary disease in the classical sense, certain genetic factors can increase the risk of its development.

- Several genes, particularly those encoding immune-related molecules such as **HLA** (human leukocyte antigen), have been linked to susceptibility.
- This may explain why the disease can occur in multiple family members, even though it is not strictly inherited.

4. Environmental Factors

Sometimes, environmental triggers may initiate or worsen myasthenia in individuals with a predisposition. These can include:

- Viral infections: Some viruses can provoke autoimmune responses, which may lead to the onset of myasthenia.
- Medications: Certain drugs that affect the immune system or neuromuscular transmission can exacerbate myasthenia symptoms [4].

Pathogenesis of Myasthenia

1. Disruption of Neuromuscular Transmission

Normal neuromuscular transmission requires the synchronized functioning of nerve and muscle cells. Under physiological conditions, the nerve cell releases the neurotransmitter acetylcholine, which binds to receptors on the muscle cell, causing its contraction.

In myasthenia gravis, this process is disrupted:

- Antibodies to acetylcholine receptors block or destroy receptors on the muscle cell surface, preventing normal signal transmission from the nerve to the muscle.
- Reduced number of active receptors on muscle membranes decreases the muscle's ability to respond to nerve impulses, resulting in muscle weakness.
- Degradation of the neuromuscular synapse: Antibodies may also damage structural components of the synapse, including neuroglial cells, impairing overall synaptic function.

2. Increased Acetylcholine Concentration

In response to the reduced number of receptors, the body attempts to compensate by increasing acetylcholine production. However, this does not fully restore neuromuscular transmission, since the high levels of acetylcholine cannot bind effectively to damaged or blocked receptors.

3. Immune Activation

The antibodies produced during the autoimmune process can also activate other components of the immune system, including the complement system, leading to further damage to synaptic cells. This exacerbates the impairment of nerve impulse transmission.

4. Role of the Thymus

The thymus plays a crucial role in the pathogenesis of myasthenia. In most patients with the disease, abnormalities such as hyperplasia or thymoma are observed. These changes disrupt the normal functioning of the immune system and may enhance autoantibody production. The involvement of the thymus in the development of myasthenia emphasizes its importance in regulating immune responses.

- Thymoma is a tumor of the thymus found in 10–15% of myasthenia patients. Those with thymoma often experience a more severe disease course and are more likely to have comorbid conditions, including cancer or other autoimmune diseases.

Types of Myasthenia

There are several forms of myasthenia, which may differ in mechanism, age of onset, clinical course, and associated conditions. Below are the main types:

1. Myasthenia Gravis (MG)

This is the most common form of the disease, characterized by an autoimmune attack on acetylcholine receptors on muscle cell surfaces. It can be classified according to age of onset, disease course, and pathogenetic features.

Classification by Age and Sex:

- In young women (20–40 years), MG typically develops as an autoimmune reaction, with symptoms often starting in the ocular muscles (ptosis, diplopia). The disease in this group may progress relatively slowly but requires long-term treatment.
- In older men (over 60 years), MG may present with more severe manifestations and is often associated with comorbidities such as thymoma.

Classification by Course:

- **Ocular form**: Symptoms are limited to specific muscle groups, such as the eye muscles (ptosis, diplopia) or facial muscles (facial weakness).
- **Generalized form**: A more severe form of the disease in which muscle weakness spreads to other groups, including respiratory and swallowing muscles. Early diagnosis and treatment of generalized MG are vital, as it can lead to respiratory failure and other serious complications.

Pathogenesis:

In this form, the immune system produces antibodies against acetylcholine receptors or other molecules like MuSK (muscle-specific kinase). These antibodies block or destroy the receptors, disrupting nerve impulse transmission and causing muscle weakness.

2. Neonatal Myasthenia

This is a rare form of myasthenia that occurs in newborns whose mothers have myasthenia gravis. In such cases, maternal antibodies cross the placenta and cause symptoms in the infant.

Characteristics:

- **Transient nature**: Neonatal myasthenia is usually temporary, with symptoms resolving within a few days or weeks after birth, once maternal antibodies are cleared from the baby's system.
- **Symptoms**: Include muscle weakness, difficulty swallowing, hypotonia (reduced muscle tone), and occasionally breathing problems.

3. Postprandial Myasthenia

This rare form of myasthenia is associated with increased muscle weakness after eating. This is thought to result from increased energy and oxygen demands following food intake, which aggravates myasthenic symptoms.

Causes:

- In some cases, this condition may be linked to autoimmune diseases like myasthenia gravis, but it can also be triggered by other factors such as infections or tumors.

Symptoms:

- ✓ Muscle weakness, especially in the neck, shoulders, and limbs, which worsens after meals
- ✓ Swallowing and breathing difficulties may occur postprandially

4. Thymoma-Associated Myasthenia

A thymoma is a tumor of the thymus gland and can be associated with the development of myasthenia gravis. Thymoma occurs in approximately 10–15% of patients with myasthenia.

Features:

- ✓ Patients with thymoma-associated myasthenia usually experience a more severe disease course.
- ✓ The disease can be associated with thymic hyperplasia (enlargement of the gland) or the presence of a tumor.

- ✓ A thymoma may also cause additional symptoms such as chest pain, cough, or breathing difficulties.

5. Lambert-Eaton Myasthenic Syndrome (LEMS)

This is an autoimmune disorder that can sometimes be confused with myasthenia gravis, although it has a different etiology and pathogenesis. Unlike myasthenia gravis, in LEMS, antibodies target calcium channels, which are necessary for the release of acetylcholine, rather than the acetylcholine receptors themselves.

Features:

- ➢ Symptoms include muscle weakness that often improves after brief physical activity (unlike myasthenia, where exertion worsens symptoms).
- ➢ LEMS is frequently associated with lung cancer (particularly small-cell lung carcinoma) — around 50% of patients with LEMS have a tumor.

6. MuSK Antibody-Associated Myasthenia

About 10–15% of patients with myasthenia gravis have antibodies against MuSK (muscle-specific kinase), a protein crucial for the development and maintenance of the neuromuscular synapse. This form of myasthenia differs from the classic form associated with acetylcholine receptor antibodies.

Features:

- ➢ Symptoms are typically more pronounced in the ocular and facial muscles (e.g., ptosis, diplopia, facial muscle weakness).
- ➢ Patients with MuSK antibodies often experience a more severe disease course, and the condition may be less responsive to conventional treatments.

7. Myasthenia Associated with Other Autoimmune Diseases

Myasthenia gravis may develop as part of a broader autoimmune syndrome, including disorders such as:

- ✓ Systemic lupus erythematosus (SLE)
- ✓ Rheumatoid arthritis
- ✓ Scleroderma
- ✓ Sjögren’s syndrome (an autoimmune disease affecting the salivary and lacrimal glands)

In these cases, myasthenia may represent part of a generalized autoimmune process, and treatment should also target the underlying autoimmune disease.

The main symptom of myasthenia is muscle weakness and fatigability, which can vary depending on the severity and muscle groups affected. Muscle weakness may affect various muscle groups, depending on the form and stage of the disease [5].

Muscle Groups Affected in Myasthenia Gravis

Ocular Muscles

- ➢ Ptosis (drooping of the eyelid) – One of the earliest signs of myasthenia. Often appears at the onset of the disease, when weakness affects the muscles responsible for lifting the eyelids.
- ➢ Diplopia (double vision) – Due to weakness of the muscles that control eye movement, patients may experience double vision, especially when looking sideways.

Facial Muscles

- ➢ Weakness of facial expression muscles – Reduced facial expressiveness, which may result in a “mask-like” or “heavy” face and difficulty expressing emotions.

Neck Muscles

- Weakness of neck muscles – May present as difficulty holding the head upright, particularly during prolonged sitting or standing.

Limb Muscles

- Weakness in arms and legs – Progressive muscle weakness in the upper and lower extremities, especially after prolonged activity such as lifting objects or walking long distances.

Respiratory Muscles

- In severe cases, myasthenia can lead to respiratory insufficiency due to involvement of the respiratory muscles (e.g., diaphragm), resulting in breathing difficulties, particularly at night.

Pathological Fatigability

Myasthenia also causes abnormally rapid muscle fatigue. Patients often experience severe tiredness that worsens throughout the day and improves with rest. Fatigue is particularly evident during physical exertion or prolonged activity.

Other key symptoms include:

Dysphagia (Swallowing Difficulty)

- Weakness of the muscles involved in swallowing may lead to difficulty swallowing food, increasing the risk of aspiration and pneumonia.

Speech Disorders (Dysphonia)

- Weakness of the vocal cords and articulatory muscles may result in dysphonia — hoarseness or a change in voice tone. Patients may complain that their voice becomes weak and fatigued during conversation.

Breathing Problems

- In severe cases, respiratory problems may develop, especially with diaphragm involvement. This condition is known as a myasthenic crisis and requires emergency medical intervention.

Myasthenic Crisis

A myasthenic crisis is an acute condition in which respiratory muscle weakness leads to respiratory failure. It often occurs as a result of infection, stress, or inappropriate treatment (e.g., sudden withdrawal of therapy). In such cases, the patient may require mechanical ventilation.

Disease Course Features

- Symptom progression: Myasthenia symptoms may develop gradually, but in some cases, the disease can progress rapidly.
- Exacerbation episodes: Flare-ups may be triggered by infections, physical or emotional stress, or inadequate treatment.
- Pathological fatigue: Along with general weakness, patients frequently experience extreme fatigue, even with minimal physical effort.

Advances in Diagnosis and Treatment

The diagnosis and treatment of myasthenia gravis are crucial areas in modern medicine. In recent decades, numerous studies have been conducted to improve diagnostic techniques and to develop new therapeutic approaches.

Let us now explore key studies that have significantly influenced the diagnosis and management of myasthenia gravis.

Diagnosis of Myasthenia Gravis

Diagnosis of myasthenia gravis is based on a combination of clinical signs, laboratory tests, and instrumental investigations. Several important studies and diagnostic techniques have been developed to improve the accuracy of diagnosis.

Clinical Tests and Examination

1. Ice Pack Test:

This is a simple and effective method for diagnosing myasthenia. A cold compress is applied to the patient's eyes for several minutes. If myasthenia is present, ptosis (drooping eyelid) significantly improves. This test is particularly effective in cases of ocular myasthenia, where ptosis is the primary symptom.

2. Tensilon Test (Edrophonium Test):

This test involves the use of edrophonium, a drug that temporarily inhibits acetylcholinesterase, thereby increasing acetylcholine levels at the neuromuscular junction. This can transiently improve muscle strength in cases of myasthenia. However, the test is now rarely used due to potential side effects and the availability of safer alternatives.

Laboratory Investigations

- **Anti-AChR** Antibodies:

One of the key diagnostic tests is the detection of antibodies against acetylcholine receptors (AChR) in the patient's blood. Approximately 85% of patients with myasthenia have elevated levels of these antibodies, making this test especially useful for confirming classic cases.

- **Anti-MuSK** Antibodies:

In 10–15% of cases, antibodies against muscle-specific kinase (MuSK) are present. This test is particularly important in patients with seronegative myasthenia, i.e., those who do not have AChR antibodies.

- Electromyography (**EMG**):

This instrumental method assesses the electrical activity of muscles. Myasthenic syndromes may manifest as a reduction in the amplitude of muscle responses upon nerve stimulation. In some cases, single-fiber **EMG** or quantitative **EMG** is used to detect subtle neuromuscular transmission defects.

Imaging of the Thymus

- Computed Tomography (**CT**) and Magnetic Resonance Imaging (**MRI**) of the thymus:

Approximately 10–15% of patients with myasthenia have a thymoma—a tumor of the thymus that may contribute to the development of the disease. **CT** or **MRI** is used to detect thymic tumors or hyperplasia.

Treatment of Myasthenia Gravis

Treatment focuses on controlling symptoms, improving patient quality of life, and preventing serious complications. In recent years, many studies have aimed to optimize treatment approaches and develop new therapeutic agents.

Immunomodulatory Drugs

- Corticosteroids (e.g., Prednisolone):

Steroid medications are a mainstay of myasthenia therapy. Studies have shown that prednisolone and other corticosteroids can significantly reduce antibody levels and improve neuromuscular transmission. However, long-term use of corticosteroids can lead to side effects such as:

- Osteoporosis
- ✓ Diabetes mellitus
- ✓ Hypertension

Cholinesterase inhibitors (pyridostigmine):

Drugs that increase the level of acetylcholine in the synapse (such as pyridostigmine) are used to improve neuromuscular transmission. This is one of the first and most effective methods of treating myasthenia. Studies have shown that pyridostigmine helps reduce muscle weakness, especially in the early stages of the disease.

Immunosuppressants and immunomodulators

- Azathioprine and methotrexate: These drugs are used to suppress the autoimmune response. For example, azathioprine is used in combination with corticosteroids for long-term disease control. Studies have shown that the combination of these drugs may be more effective than steroid monotherapy.
- Cyclosporine: A drug used in severe forms of myasthenia when other treatments are ineffective. It suppresses the function of T-lymphocytes, leading to reduced antibody production.
- Rituximab: A biological drug that blocks CD20+ lymphocytes (cells involved in antibody production). Studies have shown that rituximab may be effective in patients with myasthenia that does not respond to standard therapy.

Plasmapheresis and immunoglobulins

- Plasmapheresis: This method is used to remove antibodies from the patient's blood. It is often applied in acute cases when there is a need to quickly reduce the level of antibodies in the body, such as in myasthenic crises or in severe disease cases. Studies have shown that plasmapheresis is effective for rapidly improving patient condition.
- Immunoglobulins (IVIg): The administration of intravenous immunoglobulins helps reduce the level of autoantibodies and normalize the immune response. This treatment is also used for the accelerated stabilization of patient condition, especially during disease exacerbations.

Thymectomy (removal of the thymus)

Thymectomy is the main surgical method of treatment for patients with thymoma or thymic hyperplasia. Studies have shown that in patients with myasthenia who do not have thymoma, thymectomy can significantly improve the prognosis of the disease and reduce the need for long-term medication use.

Genetic research and therapy

In recent years, active research has been conducted on using genetic therapy for treating autoimmune diseases like myasthenia. This field is still in the research phase, but future developments may lead to the creation of more targeted methods of affecting disease mechanisms, such as gene-modified antibodies or genetic drugs aimed at reducing the autoimmune response.

In the future, several key directions in myasthenia treatment can be expected:

- Personalized therapy: The development of biomarkers, such as antibodies to various molecules (**AChR, MuSK**), can help better tailor treatment for each patient.
- Biological drugs: The use of more specific biological drugs, such as B-cell or T-cell inhibitors, may lead to better disease control with fewer side effects.
- Gene therapy: Research focused on gene therapy may become the foundation for new treatments for autoimmune diseases like myasthenia.

Thus, the diagnosis and treatment of myasthenia have significantly improved in recent decades due to new diagnostic methods, therapeutic drugs, and surgical interventions. Scientific research continues, and new advancements in biological and genetic technologies may further improve treatment outcomes in the future.

Scientific research on myasthenia gravis (**MG**) covers many aspects of the disease, including its etiology, pathogenesis, diagnosis, and treatment. In recent decades, research has advanced our understanding of the molecular mechanisms of the disease, leading to improvements in diagnostic and therapeutic methods. Key directions in scientific research on myasthenia have converged on the idea that myasthenia gravis is an autoimmune disease, where the immune system produces antibodies against acetylcholine receptors (**AChR**), which disrupts neuromuscular transmission. In recent years, significant attention has been paid to studying these autoimmune mechanisms. Key research includes:

- Antibody research: One of the major breakthroughs in myasthenia research is the discovery of antibodies against acetylcholine receptors (**AChR**). More than 80% of patients with myasthenia have these antibodies, making them a key diagnostic marker.
- Research example: For instance, a study published in the *Journal of Clinical Investigation* (2008) showed that antibodies to **AChR** block or damage the receptors on the muscle surface, leading to weakness.
- Anti-**MuSK** antibody research: In some cases, myasthenia develops without antibodies to AChR. This has been linked to antibodies against muscle-specific kinase (**MuSK**), a molecule that plays a key role in forming neuromuscular synapses. This discovery has led to new approaches in the diagnosis of myasthenia.
- Research example: A study published in ***Journal of Neuroimmunology*** (2001) focused on the discovery of antibodies to **MuSK** and their role in the pathogenesis of myasthenia, adding a new layer of understanding to the disease.

Genetic research on this condition has also led to important discoveries. Genetic studies have revealed that myasthenia may have a genetic predisposition, although the disease is generally considered autoimmune. In particular, associations with certain genes, such as HLA (human leukocyte antigen), have been identified, which could predict susceptibility to myasthenia.

- Research example: For instance, a study published in ***European Journal of Neurology*** (2011) discussed the role of **HLA** genes in the pathogenesis of myasthenia and their potential significance in predicting the disease.
- Genetic marker prediction: In 2014, studies showed that patients with specific genetic markers are more likely to develop myasthenia at a younger age. These findings could help with early diagnosis and disease prevention.

Treatment of Myasthenia

The treatment of myasthenia has also undergone significant changes. In recent years, research has been focused on new therapeutic strategies, including immunomodulators, biological drugs, and innovative treatment methods such as plasmapheresis and stem cell transplantation. Modern therapeutic approaches include:

- Immunosuppressive therapy: The most commonly used treatments include glucocorticoids and immunosuppressants. Studies have shown that these drugs effectively reduce the level of antibodies and improve the clinical manifestations of the disease.
- Plasmapheresis and immunosorption: These methods are actively used to treat myasthenic crises, where the disease becomes life-threatening. Plasmapheresis helps remove antibodies from the blood, improving the patient’s condition.

- Biological drugs: In recent years, studies have emerged regarding the use of biological drugs, such as rituximab and immunoglobulins, for treating myasthenia, especially in cases where traditional therapy is ineffective.
- Research example: A study published in ***The Lancet Neurology*** (2019) discussed the use of rituximab for treating myasthenia, which showed promising results in clinical trials.

Modern Research Directions

Modern research is aimed at improving the diagnosis of myasthenia, including more accurate tests to detect antibodies and new methods of neurophysiological research.

- Electromyography (**EMG**) and nerve stimulation: These methods allow for a more precise assessment of neuromuscular transmission disorders typical of myasthenia. **EMG** is used to detect muscle potential weakness under active stimulation.
- Antibody tests: Tests for antibodies to **AChR** and **MuSK** are important diagnostic tools that help confirm the diagnosis of myasthenia at the molecular level.
- Research example: In ***Journal of Neuroimmunology*** (2015), the role of antibodies to MuSK and methods for their diagnosis are discussed, which has become a key moment in improving the diagnosis of various forms of myasthenia.

Modern scientists emphasize the importance of integrating different approaches to improve the diagnosis and treatment of myasthenia. For example, research shows that personalized treatment, taking into account the specific nature of the disease (such as antibody type), can be more effective. Efforts continue to create innovative drugs aimed at blocking autoimmune activity and restoring neuromuscular transmission.

Conclusions:

Scientific research on myasthenia is constantly evolving, and the new understanding of the molecular mechanisms of the disease opens up prospects for more accurate diagnosis and effective treatment. In particular, the study of autoimmune mechanisms, genetic factors, new therapeutic drugs, and advanced diagnostic methods significantly improves the prognosis for patients with myasthenia.

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