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ASSESSMENT OF COGNITIVE FUNCTION IN MYASTHENIC PATIENTS (LITERATURE REVIEW)

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Abstract:

Myasthenia gravis is an autoimmune disease caused by destruction of nicotinic acetylcholine receptors on the motor endplates of the transverse striated muscles. The incidence ranges from 1 to 9 cases per million in the general population. The prevalence of MG ranges from 15 to 179 cases per million population worldwide. Although it is predominantly a muscular disease, cognitive impairment in patients with myasthenia gravis has been discussed in the literature. Some studies have found cognitive decline in attention memory, executive functions, verbal fluency, and planning tasks. However, there are other studies that have found no differences in the cognitive functions of myasthenia gravis patients compared to healthy controls.

Keywords: myasthenia gravis, neuropsychological condition, muscle weakness, acetylcholine receptors

INTRODUCTION. Myasthenia gravis (MG) is an autoimmune disease caused mainly by autoantibodies to skeletal muscle nicotinic acetylcholine receptors (nAChRs) at the postsynaptic membrane, resulting in depletion of ACh at the neuromuscular synapse. MG is rare with a prevalence of (25-125)/106. The disease tends to affect women more often than men (3: 2) in the second and third decades of life. The main symptoms of myasthenia gravis are fatigue and weakness of skeletal muscles with repetitive or prolonged exertion during the day, but decreasing at rest. The eye muscles are initially affected in about 2/3 of patients, then spread to the bulbar muscles and limb muscles. Approximately 85% of patients develop generalized weakness. In many patients, the disease progresses from mild to severe, and if respiratory muscle weakness becomes so severe that artificial ventilation is required, the patient is said to be in crisis. Spontaneous remissions are very rare and last for various periods, which mostly occur during the first 3 years of the disease. In adults, the thymus gland is abnormal in 90% of people with myasthenia gravis, about 70% have hyperplasia of the thymus gland, and 10-20% have benign tumors of the thymus gland or thymoma. Current treatments for myasthenia gravis include acetylcholinesterase inhibitors (AChE-I) (as pyridostigmine), immunopharmacological drugs (as prednisone, azathioprine, cyclosporine, mycophenolate mofetil, cyclophosphamide, tacrolimus and rituximab, plasmapheresis, intravenous immunoglobulins (IVIG) and thymectomy. Subjective impairment of memory and other cognitive functions in MG patients is very common, but previous studies that have examined cognitive function in such patients have shown conflicting results. Some reported memory and other cognitive dysfunctions and electroencephalographic (EEG) abnormalities. In contrast, others reported no neuropsychological abnormalities and normal intelligence, attention, memory, and motor activity in MG.

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azathioprine, cyclosporine, mycophenolate mofetil, cyclophosphamide, tacrolimus and rituximab, plasmapheresis, intravenous immunoglobulins (IVIG) and thymectomy. Subjective impairment of memory and other cognitive functions in patients with MG is very common, but previous studies that have examined cognitive function in such patients have shown conflicting results. Some have reported memory and other cognitive impairments as well as electroencephalographic (EEG) abnormalities. In contrast, other researchers have reported no neuropsychological abnormalities and normal measures of intelligence, attention, memory, and motor performance in MG.

Edrophonium test. This test uses injections of edrophonium chloride to provide short-term relief of weakness in people with myasthenia gravis. The drug blocks the breakdown of acetylcholine and temporarily increases acetylcholine levels at the neuromuscular synapse. It is commonly used to test for ocular muscle weakness.

Blood tests - Most people with myasthenia gravis have abnormally elevated levels of acetylcholine receptor antibodies. A second antibody, called MuSK antibody, has been found in about half of myasthenic patients who do not have acetylcholine receptor antibodies. Blood tests can also detect this antibody. However, some people with myasthenia gravis do not have either of these antibodies. These people are said to have seronegative (antibody negative) myasthenia gravis. Electrodiagnostics - Diagnostic tests involve repetitive nerve stimulation, which repeatedly stimulates a person's nerves with small electrical impulses to fatigue certain muscles. Muscle fibers in myasthenia gravis, as well as in other neuromuscular disorders, do not respond as well to repetitive electrical stimulation as muscles in healthy individuals. Single fiber electromyography (EMG), considered the most sensitive test for myasthenia gravis, detects impaired nerve-muscle transmission. EMG can be very useful in diagnosing mild cases of myasthenia gravis when other tests cannot detect abnormalities.

Diagnostic imaging. Diagnostic chest imaging using computed tomography (CT) or magnetic resonance imaging (MRI) can detect the presence of a thymoma.

Pulmonary function study. Measurement of respiratory force can help predict whether breathing may stop and lead to myasthenic crisis. The mission of the National Institute of Neurological Disorders and Stroke (NINDS) is to gain fundamental knowledge about the brain and nervous system and to use that knowledge to reduce the burden of neurological disease. NINDS is part of the National Institutes of Health (NIH), the world's leading proponent of biomedical research. Although there is no cure for myasthenia gravis, treatment for the disorder has improved over the past 30 years. Understanding of the causes, structure and function of the neuromuscular synapse, fundamental aspects of the thymus gland and autoimmunity has improved. Technological advances have led to more timely and accurate diagnosis of myasthenia gravis, and new and improved treatments have improved treatment options. Researchers are working to develop better drugs, identify new ways to diagnose and treat people, and improve treatment options.Some people with myasthenia gravis do not respond well to available treatment options, which usually involve long-term suppression of the immune system. New drugs are being tested either alone or in combination with existing medications to see if they are more effective in affecting the causes of the disease. In addition to developing new drugs, researchers are trying to find better ways to diagnose and treat the disorder. For example, NINDS-funded researchers are studying the assembly and function of connections between nerves and muscle fibers to understand the fundamental processes of neuromuscular development. This research may reveal new treatments for neuromuscular diseases such as myasthenia gravis. Researchers are also exploring better ways to treat myasthenia gravis by developing new tools to diagnose people with undetectable antibodies and identify potential biomarkers (traits that can help diagnose or measure disease progression) to predict individual response to immunosuppressants. Because weakness is a common symptom of many other diseases, myasthenia gravis is often not diagnosed or is diagnosed with a delay (sometimes up to two years) in people who experience mild weakness or in those whose weakness is limited to only a few muscles. Myasthenia gravis can generally be controlled today. There are several treatments that help reduce and improve muscle weakness.

Thymectomy. This surgery to remove the thymus gland (which is often abnormal in people with myasthenia gravis) can reduce symptoms and may cure some people, possibly by rebalancing the immune system. An NINDS-funded study has shown that thymectomy is beneficial for both people with thymoma and those without signs of a tumor. Drugs to treat this disorder include anticholinesterase agents such as mestinone or pyridostigmine, which slow down the breakdown of acetylcholine at the neuromuscular synapse and thereby improve neuromuscular transmission and increase muscle strength.

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Immunosuppressants These drugs improve muscle strength by inhibiting the production of abnormal antibodies. They include prednisolone, azathioprine, mycophenolate mofetil, and tacrolimus. The drugs can cause serious side effects and must be closely monitored by a physician.

Plasmapheresis and intravenous immunoglobulin . These treatments may be options in severe cases of myasthenia gravis. People may have antibodies in the plasma (a liquid component of the blood) that attack the neuromuscular junction. These treatments remove the destructive antibodies, although their effectiveness usually lasts for weeks to months.

CONCLUSIONS: With treatment, most people with myasthenia gravis can significantly reduce muscle weakness and lead normal or nearly normal lives. In some cases of myasthenia gravis, remission - temporary or permanent - may occur and muscle weakness may disappear completely, so that medication can be discontinued. Stable, long-term complete remissions are the goal of thymectomy and can occur in about 50 percent of people who undergo the procedure.

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