OCULAR MYASTHENIA GRAVIS

A Weakening of the Eye and Eyelid Muscles

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Ocular myasthenia gravis can occur as an isolated autoimmune disorder although most people with ocular symptoms go on to develop generalized MG within the first two years.

Ocular Myasthenia Gravis

Ocular myasthenia gravis is an autoimmune eye disorder that can occur alone or as a feature of generalized myasthenia gravis. In fact, eye changes related to weakened eye muscles or eyelid muscles are one of the first symptoms to occur in more than two-thirds of all patients with myasthenia gravis (MG). From 70 to 80 percent of patients with isolated ocular myasthenia gravis go on to develop generalized myasthenia gravis within the first two years.

Who is Affected?

Although three times as many women as men develop myasthenia gravis, slightly more men than women develop ocular myasthenia gravis. And while the mean age for generalized MG is 33 years, the mean age for ocular myasthenia is 38 years. The highest incidence of ocular myasthenia gravis occurs among the Chinese. Patients with ocular myasthenia gravis often have thyroid antibodies or autoimmune thyroid disease. Patients with generalized myasthenia gravis, in contrast, are more likely to be Italian and rarely develop autoimmune thyroid disease.

Symptoms of Ocular MG

Symptoms of ocular MG are very similar to those seen in thyroid eye disease, a disorder that is often confused with ocular MG. Common symptoms include diplopia (double vision), ptosis (drooping eyelids), light sensitivity, weakness of lid closure, nystagmus (constant involuntary movements of the eyeball in any direction) visual disturbances, and eyelid retraction, which causes an incomplete eye closure.

Disease Course

Similar to other autoimmune diseases, ocular MG is characterized by periods of variable symptoms alternating with periods of remission. When ocular symptoms alone persist for 3 years, patients are unlikely to develop generalized myasthenia gravis. Symptoms are often mild after periods of rest and intensify with activity. Symptoms of ocular MG also are unlikely to worsen after the first three years of the disease. Spontaneous remissions are also known to occur and can persist for years.
Environmental Factors

Symptoms of ocular MG tend to worsen with exposure to bright sunlight and during periods of emotional stress, viral illness, surgery, menstruation, pregnancy, and following immunizations.

Diagnosis

Test to confirm the diagnosis of ocular MG include repetitive nerve stimulation, and single-fiber electromyography (EMG) of the frontalis muscle. In addition, the eye muscles are examined for fatigue and weakness and the Tensilon test is usually performed. In the Tensilon test, a small amount of edrophonium is injected intravenously. If myasthenia gravis is present, the eye muscles will show a positive response and symptoms of muscle weakness and ptosis will improve dramatically. Eye muscle function may also be assessed before and after a 30-minute eye rest. In MG, improvement is seen after the period of rest.

Blood tests for blocking acetylcholine receptor antibodies are also used to help diagnose ocular MG although patients with ocular MG alone are less likely to have acetylcholine receptor antibodies than patients with generalized MG. About 70 percent of patients with ocular MG have these antibodies compared to 90 percent of patients with generalized MG.

An MRI or CT scan can be used to rule out other causes of eye symptoms such as tumors, and thyroid function tests are used to differentiate ocular MG from thyroid eye disease.

Treatment

Treatment for ocular MG is similar to treatment for generalized MG. Treatments include cholinesterase inhibitors such as pyridostigmine (Mestinon), prednisone and azathioprine. In severe cases, plasmapheresis or intravenous immunoglobulin (IVIG) therapy may be used.

For eyelid drooping, eyelid tape and wire eyelid crutches are often used. Eye patches and prisms are used to reduce symptoms of double vision.

Resources:


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