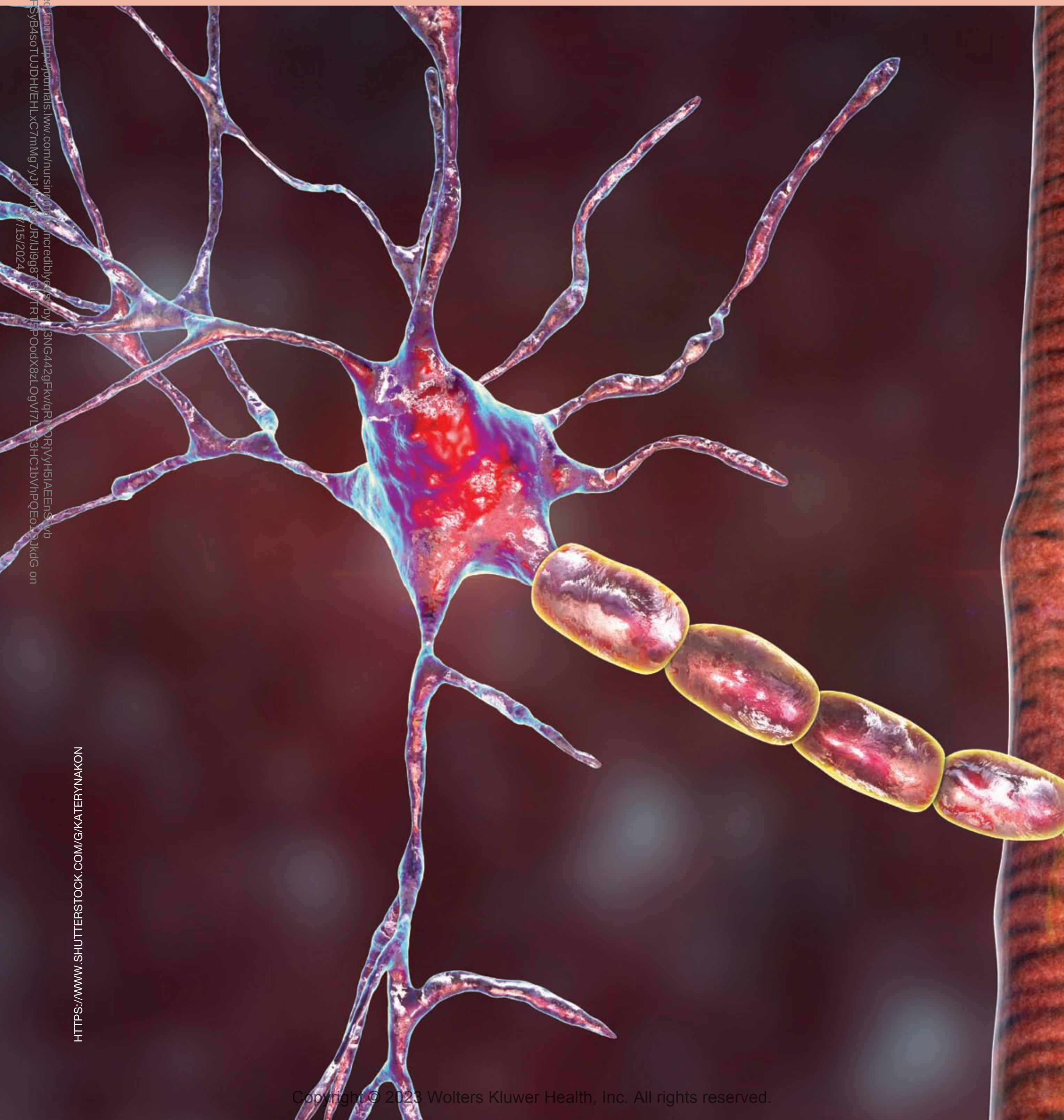

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The nurse's guide to myasthenia gravis

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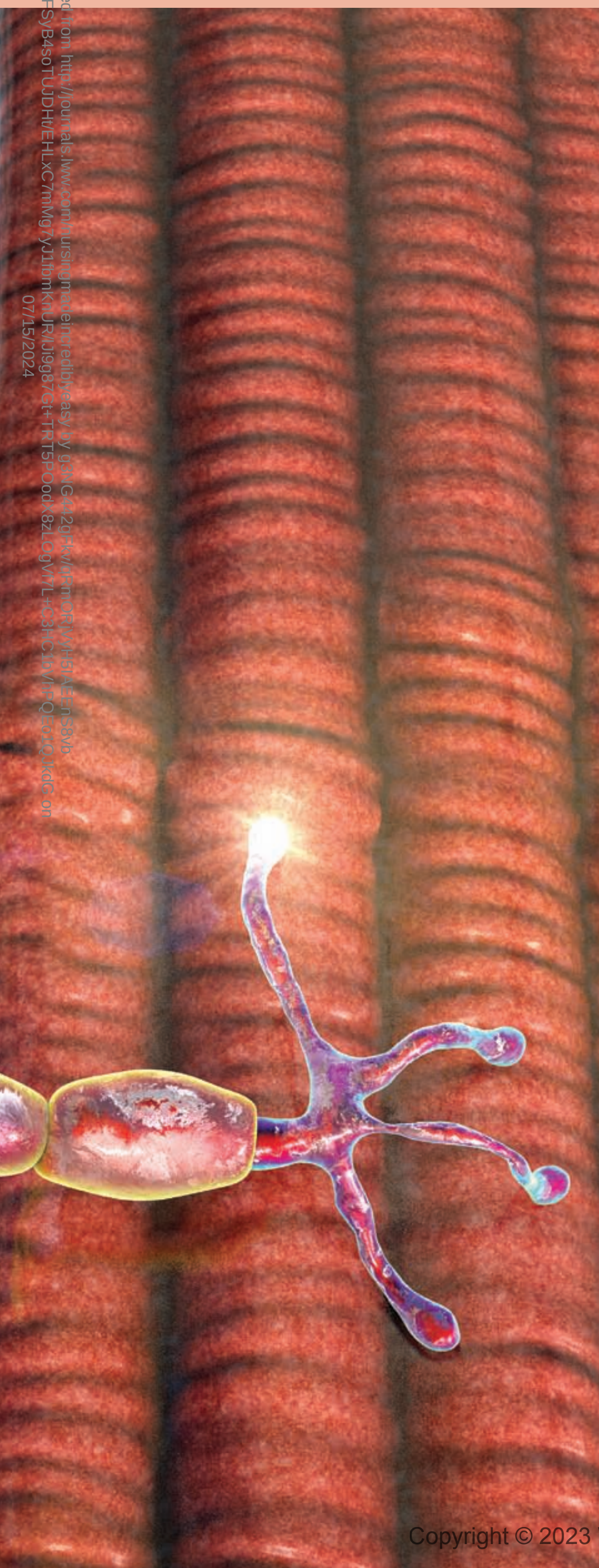


With treatment from an interprofessional team, most patients with MG are expected to have an active quality of life and a lifespan similar to people without the disease.

By Richard L. Pullen, Jr., EdD, MSN, RN, CNE, CNE-cl, ANEF, FAAN and Lori Hammond, DNP, MSN, RN, CNE-cl, GERO-BC

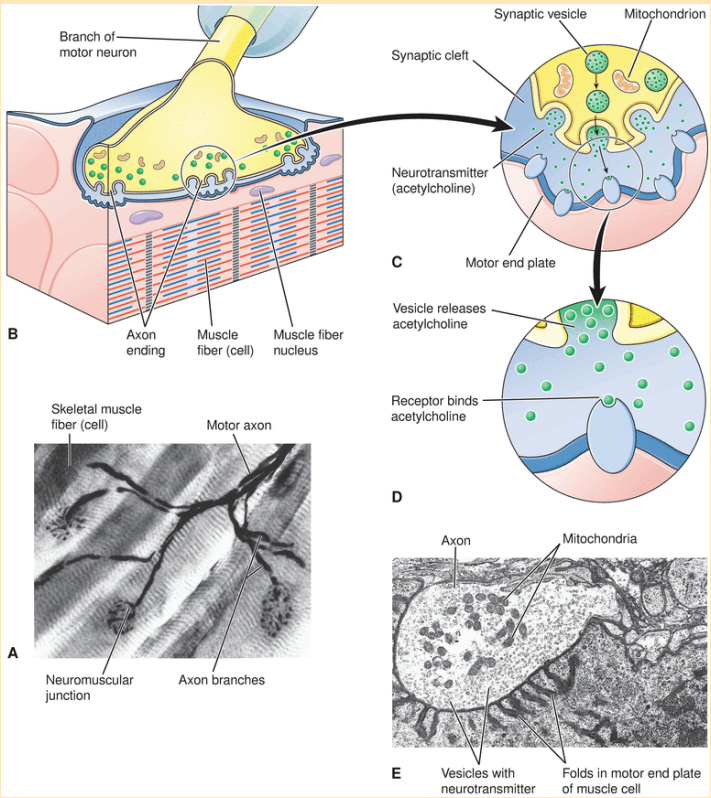
Myasthenia gravis (MG) is an autoimmune disease that causes a defect in the postsynaptic membrane in the neuromuscular junction (NMJ), resulting in excessive fatigue and fluctuating muscle weakness.[1] The affected muscles may include those that control eyelid motion, facial expression, chewing, swallowing, speaking, and the extremities. Weakness may worsen with activity and improve with rest. Symptoms may be mild but can become life-threatening if the disease affects respiratory muscles because this can result in respiratory failure.[1] This article presents the pathogenesis, signs, symptoms, and management of patients with MG.

Epidemiology and risk factors

In Latin and Greek, myasthenia gravis means "grave muscle disease," with the suffix "asthenia" meaning "weakness or fatigue."[2] MG affects an estimated 20 per 100,000 of the population, or 36,000 to 60,000 people in the US. The global prevalence is 150 to 200 people per 1,000,000.[3,4] MG occurs in all ages, across all racial and ethnic groups, and has a female-to-male ratio of 3:1 during childbearing years and closer to 1:1 after menopause.[3-5] Autoimmune diseases, including MG, are more common in females than males.[5,6] Higher estrogen levels among women put them at a higher risk for autoimmunity compared with men, whose androgens offer protection from autoimmune disease by suppressing the immune system.[5,6] Several genes, including the human leukocyte antigen, tumor necrosis factor alpha-induced protein 3, interacting protein 1, and tyrosine phosphatase nonreceptor 22 are implicated as the origin of pathogenesis.[3,7] Precipitating factors in genetically susceptible patients include but aren't limited to physical and emotional stress, infections, immunizations,

The NMJ

Motor neurons stimulate skeletal muscle cells at the NMJ. **A.** A motor axon branches to stimulate multiple muscle fibers (cells). **B.** An axon branch makes contact with the membrane of a muscle fiber (cell) at the NMJ. **C.** Enlarged view of the NMJ showing release of neurotransmitter (acetylcholine) into the synaptic cleft. **D.** Acetylcholine attaches to receptors in the motor end plate, which has folds that increase surface area. **E.** Electron microscope photograph of the NMJ.



Cormack DH. *Essential Histology*, 2nd Ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2001; Courtesy of A. Sima

pregnancy, childbearing, surgery, obesity, smoking, poor nutrition, and having other autoimmune diseases.[7,8]

Pathogenesis

The NMJ is a synaptic connection where impulses are transmitted from a motor neuron to skeletal muscle, causing muscle contraction (see *The NMJ*).[9] Motor neurons are separated by a tiny space called the synaptic cleft. The presynaptic motor neuron initiates an impulse; the postsynaptic motor neuron receives the impulse.[9]

When a person contracts a muscle, the body first sends a nerve impulse, which causes acetylcholine (ACh) neurotransmitters to release from a motor neuron and attach to nicotinic ACh receptors on the target muscle, enabling contraction.[9] However in patients with MG, B-lymphocyte-mediated autoantibodies, or anti-AChR antibodies, are produced to work against ACh receptors (AChRs).[7,9-11] These anti-AChR antibodies compete with ACh for control of a target muscle, by attacking the postsynaptic membrane at the NMJ and attaching to nicotinic AChRs, preventing ACh from binding to these receptors to stimulate muscle contraction.[7,9-11] Anti-AChR antibodies win the competition, resulting in rapid and excessive generalized or localized weakness and fatigue that worsens with activity. Approximately 85% of patients with MG have circulating anti-AChR antibodies.[7,9-11] Patients who don't have anti-AChR antibodies may possess lipoprotein-associated protein 4 (LRP4) or muscle-specific tyrosine kinase receptor (MuSK), both of which are crucial antibodies for proper NMJ function. Newborns of mothers with MG may have temporary muscle weakness that fades as the maternal hormones clear the infant's system.[7,9-11]

The thymus gland controls the maturation of immunologic function in early life and is associated with MG because it triggers and maintains the production of autoantibodies that attack the NMJ.[7,12] The thymus gland is composed of epithelial cells and lymphocytes. Normally, the thymus gland grows during puberty, gradually atrophies as a person ages, and is replaced with fatty tissue. Approximately 15% of patients diagnosed with MG have a tumor of the thymus gland called a thymoma. Conversely, about 50% of patients diagnosed with thymoma will develop MG.[7,12] A person's immune system response against the genetic makeup of a thymoma influences neurotransmission in the NMJ, causing MG.[7,9,12] Thymomas grow slowly and

can invade the surrounding chest cavity, including the pleura and pericardium. Therefore, removal of the thymoma (thymectomy) is always recommended because of the malignancy risk. Thymectomy in the absence of thymoma decreases the immunologic attack on the NMJ and can improve MG in 70% of the cases.[7,9,12]

Clinical presentation

Fluctuating strength of voluntary muscles is a defining feature of MG.[4,13] The severity of weakness depends on the degree of exertion to muscle groups, varies over time, and is influenced by stress, hormonal and immunologic factors, and unknown factors.[4,13] All voluntary muscles may be involved to some degree with relapse and remission (see *Adapted MGFA classification for MG*).[4,13]

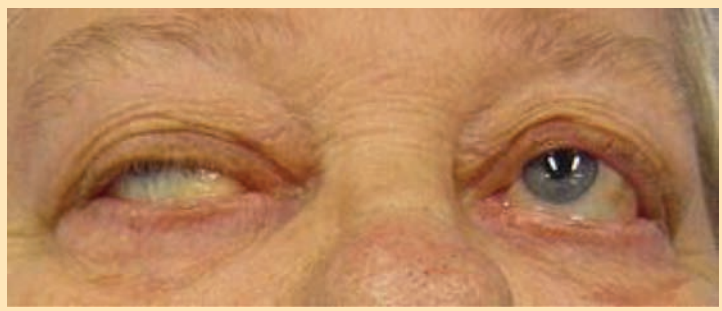
Ocular weakness is the most common initial symptom in MG with or without generalized weakness, manifesting as unilateral or bilateral upper eyelid weakness and drooping, or ptosis (see *Ptosis*).[13,15-17] Ptosis is unilateral in most cases and caused by impairment of the levator palpebrae superioris muscle secondary to oculomotor nerve (cranial nerve III) involvement.[13,15-17] Cranial nerve III pathology also results in diplopia and blurred vision in patients with MG. Instructing the patient to wear a patch over the affected eye can mitigate visual symptoms. Eyelid weakness in ptosis increases when a patient takes an upward gaze for 15 seconds.[13,15-17] The Cogan lid twitch may also be elicited in MG. This happens when the patient takes a downward gaze for 15 seconds and returns to the primary eye position. The eyelid overshoots or twitches before it returns to the normal ptotic position. Improvement in ptosis after applying an ice pack to the affected eye(s) for 5 minutes is a symptom of MG.[13,15-17]

Proximal muscle weakness is more common than generalized weakness in MG.[15-17] For example, the second most common manifestations are bulbar symptoms related to cranial nerves V (trigeminal), VII (facial), IX (glossopharyngeal), X (vagus), XI (accessory), and XII (hypoglossal).[15-17] Bulbar refers to the muscles innervated by motor neurons originating in the medulla or pons.[15-17] MG's most common bulbar sign is weakness of facial muscles, which may be asymmetrical and misdiagnosed as Bell palsy.[15-17] At rest, facial expression appears normal, but with emotions such as laughing, it doesn't. Patients may appear sad or angry from weak facial muscles and avoid social interaction. Speech may be affected, such as by having difficulty in articulation, having a nasal voice quality, lisping, and/or slurring words.[15-19] Differential diagnoses, such as amyotrophic lateral sclerosis, should be explored relative to the hypernasal speech quality.[15-19] Tongue weakness causes

Adapted MGFA classification for MG[14]

Class	Description
I	Ocular muscle weakness. Normal strength for all other muscles.
II	Mild muscle weakness other than ocular muscle weakness. Ocular muscle weakness may be present.
III	Moderate muscle weakness other than ocular muscle weakness. Ocular muscle weakness may be present.
IV	Severe muscle weakness other than ocular muscle weakness. Ocular muscle weakness may be present.
V	Requires invasive or noninvasive ventilation support.

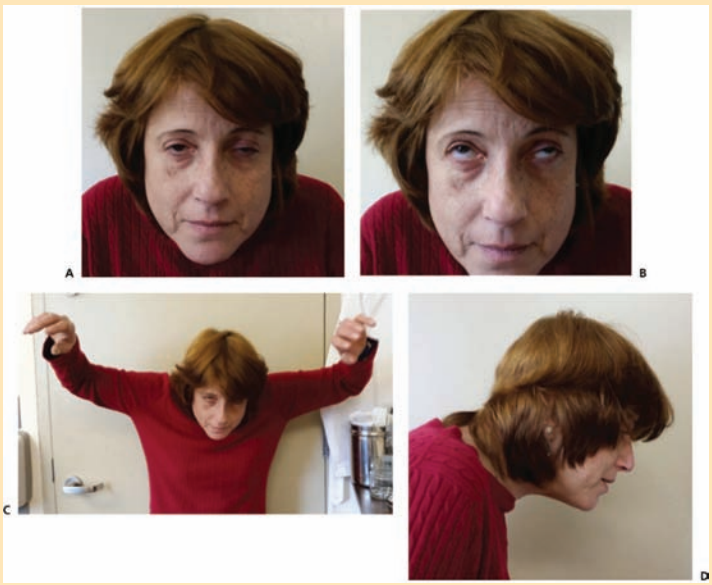
Ptosis



Source: Image created by James Heilman, MD and published on Wikimedia Commons under CC BY-SA 3.0.

Clinical features of MG

A. Bifacial weakness and asymmetric ptosis. **B.** Weakness of extraocular muscles causing dysconjugate gaze. **C.** Proximal arm weakness. **D.** Neck flexor weakness causing head drop.



Louis ED, Mayer SA, Rowland LP. *Merritt's Neurology*. 13th ed. Philadelphia, PA: Wolters Kluwer Health and Pharma, LWW; 2015. Courtesy of C.M. Ulane.

difficulty chewing food. Weakness in the lips, tongue, and pharyngeal muscle may cause difficulty swallowing, making the patient susceptible to choking.[15-19] Regurgitation of food and fluids through the nose indicates palatal muscle weakness.[15-19] Neck muscle weakness may cause difficulty balancing the head and create problems swallowing, breathing, and ambulating. Muscles of the arms, hands, fingers, legs, and for breathing are involved in generalized MG.[15-19] Patients may have difficulty performing activities of daily living, including opening a jar, hanging laundry, toileting, bathing, moving from sitting to standing, and walking. They may experience a feeling of profound heaviness when the arms and legs are affected.[15-19] Muscle atrophy and immobility contribute to weakness, fatigue, and environmental safety issues.[15-19] Myasthenic crisis (MC) and cholinergic crisis (CC) may occur when there are imbalances of ACh at the NMJ, causing severe muscle impairment requiring invasive or noninvasive ventilatory support.[15-19] (See *Clinical features of MG*.)

The presence of anti-AChR, anti-MuSK, and anti-LRP4 antibodies is highly indicative of MG. Approximately 5%-10% of patients are negative for these antibodies but still have the clinical manifestations of MG.[10,15-19] Repetitive nerve stimulation (RNS) and single fiber electromyogram of a motor neuron cause a muscle to release increasing amounts of ACh, resulting in muscle weakness.[10,15-19] Most patients with MG have a decremental response to RNS, a diagnostic feature of the disease. A decremental response means that the muscle becomes weaker each time it's stimulated because ACh is depleted; this indicates NMJ pathology.[10,15-19] A decremental response correlates with the patient's muscle weakness and fatigue.[10,15-19] Other conditions, including but not limited to botulism and Lambert-Eaton myasthenic syndrome (LEMS), impact the NMJ and should be included in the differential diagnosis.[1,20] LEMS results from an autoantibody attack of calcium channels in the presynaptic component of the NJM.[1,20] Chest X-ray, computed tomography, and MRI may indicate thymoma and exclude central and peripheral nervous system pathologies.[13] Edrophonium is a medication used to support a diagnosis of MG. Edrophonium is a synthetic acetylcholinesterase that increases the amount of ACh at the NMJ.[21,22] The patient's muscle strength dramatically improves within 1 minute of I.M. administration, lasting about 10 minutes.[21,22] (See *Summary of physical signs and symptoms*.)

Management

MG management focuses on reducing circulating pathogenic antibodies and improving signs and symptoms. MG has no cure, but treatment can induce remission and improvement of the patient's functional ability (see *Common medications in MG*).[23-25]

Selected treatment of MG

MC is a life-threatening complication of MG that manifests as severe weakness of the respiratory, upper airway, ocular, and proximal limb muscles.[1,19,26] Its pathology is a lack of ACh at the NMJ for muscle function. The patient in MC has difficulty speaking from the weakness of the mouth, tongue, throat, and vocal cords.[1,19,26] They may have trouble breathing, clearing their throat, coughing, and are prone to aspiration. They may experience bowel and bladder incontinence. Infections are the most common causes, although other factors such as stress, pregnancy, surgery, and environmental warmth are also a trigger.[1,19,26] Antibiotics in the aminoglycoside, macrolide, fluoroquinolone classifications, beta-blockers, and antiepileptic medications may trigger MC.[1,19,26] Local and general anesthesia should be used with caution. Patients with MC are managed in the ICU with noninvasive or invasive ventilation with close monitoring of arterial blood gases.[1,19,26] The administration of edrophonium, immunosuppressants, and intravenous immune globulin should improve the patient's symptoms.[1,19,26] Chest physiotherapy is instituted when a patient can't take deep breaths and cough to clear the airway. Enteral tube feedings may be prescribed when a patient can't swallow.[1,19,26]

CC is a rare life-threatening complication of MG from taking too much acetylcholinesterase inhibitors, such as pyridostigmine, resulting in excessive stimulation at the NMJ.[27] Patients experience muscle twitching (fasciculations), generalized muscle weakness, impairment of respiratory muscles, increased pulmonary secretions, increased lacrimation, pupillary constriction, bradycardia, nausea, vomiting, diarrhea, and abdominal cramps.[27] A diagnosis of CC is confirmed when symptoms worsen with the administration of edrophonium.[27] The administration of atropine may offset ACh activity.[27] Patients are managed in the ICU, similar to MC. Maintenance doses of medications, such as pyridostigmine, may need to be adjusted to prevent recurrences of MC or CC.[27]

Summary of physical signs and symptoms[13,15]

Chronic fatigue with minimal exertion	
Eye muscle symptoms (often first symptom)	• Ptosis can be unilateral or bilateral • Diplopia can improve by closing one eye
Face muscle symptoms	• Change in facial expressions–a smile may not look like a smile • Decrease in ability to chew–may tire quickly before the meal is consumed
Dysarthria-impaired speaking	• Low or weak voice
Neck/throat muscle symptoms	• Dysphagia–difficulty swallowing • Weakness in the neck making it difficult to hold up the head
Lung symptoms	• Dyspnea–difficulty breathing • Shortness of breath • Inability to clear mucus/phlegm from airways
Extremities	• Weakness of arms and/or legs • Difficulty walking

Nursing strategies

MG is a chronic disease that's generally managed on an outpatient basis. The nurse focuses patient care on energy conservation, medication management, prevention of complications, and strategies to help with ocular symptoms.[15] Therapeutic blood levels of acetylcholinesterase medication are crucial to prevent worsening of muscle weakness. Patients must take the medication as prescribed. The following are additional strategies for nurses:

1. Perform a head-to-toe physical assessment focusing on neuromuscular function. Ensure cardiopulmonary stability and the patient's safety relative to muscle weakness.[1-4]
2. Assess the presence of comorbidities, take a medication history, and perform a lifestyle assessment. This type of

Common medications in MG[23-25]

Pyridostigmine Inhibits acetylcholinesterase from breaking down ACh	Adverse reactions: Nausea, vomiting, diarrhea, increased saliva, increased tear production, increased sweating, muscle cramps, and visual disturbances
Efgartigimod Blocks fragment crystallizable (FC) neonatal receptors in adults who are anti-AChR antibody positive	Adverse reactions: Headache, urinary tract infections, respiratory tract infections, fatigue, and arthralgia
Corticosteroids Reduces immune system response to decrease antibody production	Adverse reactions: Infection, mental status changes, insomnia, osteoporosis, weight gain, increased appetite, sodium retention, hyperglycemia, hypertension, bruising, bleeding, glaucoma, and cataracts
Azathioprine Inhibits T-lymphocyte production, causing immunosuppression	Adverse reactions: Infection, bruising, bleeding, fatigue, arthralgia, hepatotoxicity, renal toxicity, photosensitivity, and increased susceptibility to malignancy
Mycophenolate mofetil Inhibits T-lymphocyte and B-lymphocyte production, causing immunosuppression	Adverse reactions: Infection, nausea, vomiting, diarrhea, hepatotoxicity, renal toxicity, bruising, bleeding, and anemia
Cyclosporine Inhibits production of T-lymphocytes, causing immunosuppression	Adverse reactions: Infection, hypertension, bruising, bleeding, anemia, hepatotoxicity, renal toxicity, tremor, swollen gums, neuropathy, blood glucose changes, and increased hair growth
Tacrolimus Inhibits production of T-lymphocytes causing immunosuppression	Adverse reactions: Infection, hypertension, anemia, bruising, bleeding, blood glucose changes, renal toxicity, neuropathy, nausea, diarrhea, headache, tremors, and hair loss
Other treatment strategies	
Intravenous immune globulin (IVIG) Modulates T-lymphocytes and B-lymphocytes and neutralizes pathogenic antibodies	IVIG is used in MG flares and MC to improve symptoms rapidly. Long-term IVIG is indicated in some patients. Adverse reactions include headache and fatigue.
Plasmapheresis (plasma exchange) Filters blood and removes pathogenic antibodies, which block neurotransmission to muscles	Plasmapheresis is used in MG flares to temporarily reduce the circulation of pathogenic antibodies, especially in MC. Adverse reactions include hypotension, dysrhythmias, and muscle cramps.
Thymectomy Surgical removal of the thymus gland suppresses pathogenic antibody production	Removing the thymus gland will likely improve MG symptoms even when the person has no thymoma.

assessment includes asking the patient questions such as: (1) Does the patient live alone or with someone? (2) What's the patient's financial status? (3) Is the patient's home environment safe and free from harm? For example, sufficient lighting (not bright lights), assistive devices for activities of daily living, and the absence of loose rugs.[8,13,19]

3. Encourage the patient to avoid hot environments to prevent MG exacerbation.[19]
4. Assess lab data indicating MG pathology, including but not limited to the presence of AChR, MuSK, or LRP4 antibodies.[19,25-27]
5. Assess complications of medication therapy. For example, infection,

hepatoxicity, renal toxicity, and bleeding. Fever, elevated white blood cells, erythrocyte sedimentation rate, and c-reactive protein may indicate infection. A reduction in RBCs may indicate anemia. High creatinine may indicate renal toxicity. High liver enzymes may indicate hepatotoxicity.[19]

6. Assess the patient's appearance: (1) Is the patient having trouble breathing? (2) Can the patient hold up their head? (3) Does the patient have a downward angle of the lips reflecting muscle weakness? (4) Does the patient's face look depressed? (5) Is ptosis present? Perform the ice-pack test to evaluate the improvement of ptosis and Cogan eye twitch strategy to evaluate worsening of ptosis.
7. Perform a comparative analysis of pupillary response to light stimulus.[19]
8. Perform the six conjugate eye movements (gazes) to assess eye muscle movement ability.[19]
9. Encourage the patient to wear an eye patch over the ptotic eye. Weak eye muscles in the ptotic eye are responsible for diplopia. Closing the ptotic eye or covering it with a patch reduces or eliminates diplopia.[19]
10. Encourage the patient to avoid bright indoor lights as well as looking directly at the sun to avoid eye irritation. Suggest that they wear sunglasses when out in the sun.[19]
11. Assess the weakness of oropharyngeal muscles by asking the patient to speak. Muscle weakness causes nasal voice, regurgitation of oral mucus secretions, and difficulty swallowing.[19]
12. Assess muscle strength and atrophy: (1) Perform a comparative analysis of hand grip strength and equality. (2) Perform a comparative analysis of muscle strength, and equality of the legs. (3) Evaluate balance and strength by asking the patient to stand and walk with the nurse at their side.[16-19]
13. Recommend interprofessional collaboration with the primary provider, neurologist, rheumatologist, speech therapist, nutritionist, physical therapist, and occupational therapist.[28-30]

A better quality of life

Muscle weakness generally peaks and stabilizes about 3 years after initial diagnosis, followed by periods of flares and remission.[3,4] With an interprofessional team approach to treatment, most patients with MG are expected to have an active quality of life and a lifespan similar to people without the disease.[3,4,19] The prognosis of patients with MG has significantly improved in the past 40 years with the widespread use of immunotherapy and enhanced care of patients with respiratory complications.[2] Nurses can provide care and patient education that will enable patients with MG to have an improved quality of life. With their knowledge of MG, RNs, APRNs, LPNs, nurse educators, and nurse researchers can ensure quality care. Nurses at all levels of educational preparation can be advocates for patients with this chronic illness. ■

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