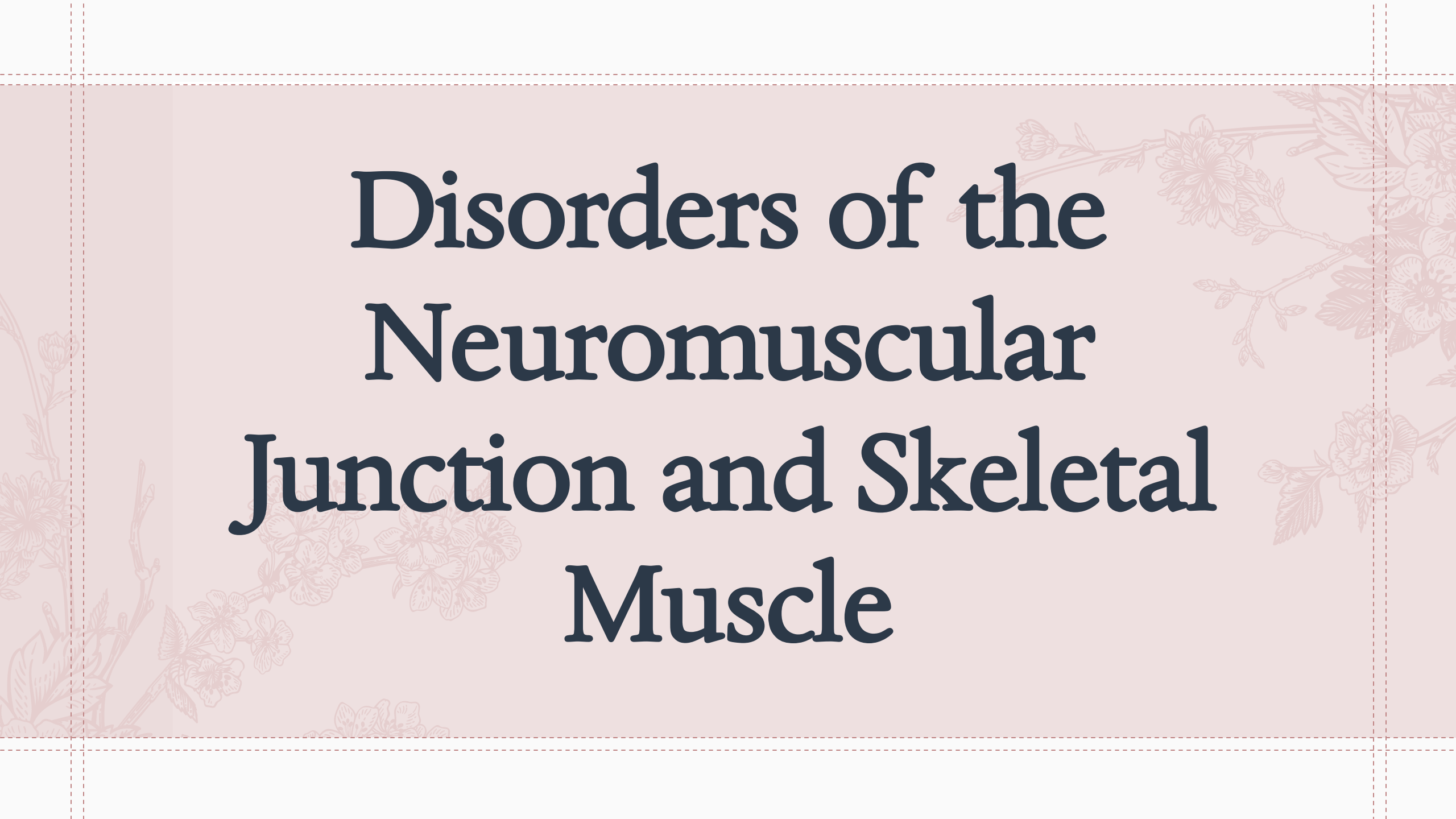
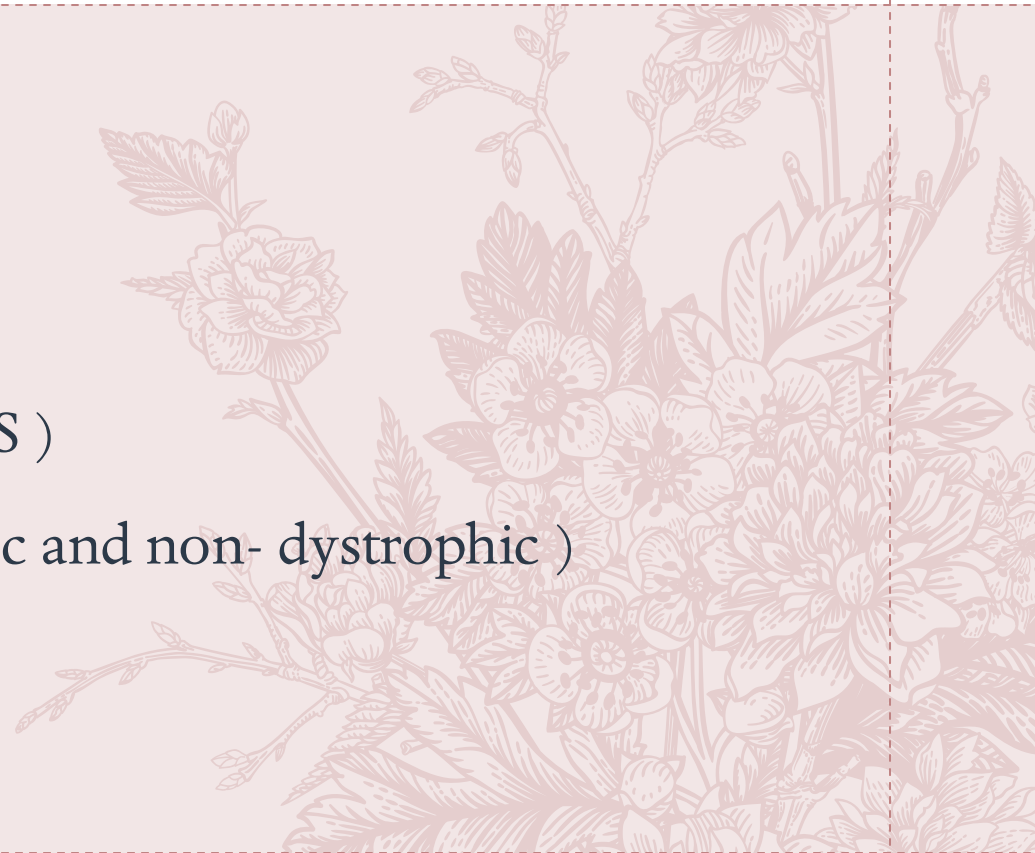


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Disorders of the Neuromuscular Junction and Skeletal Muscle

Main topics:

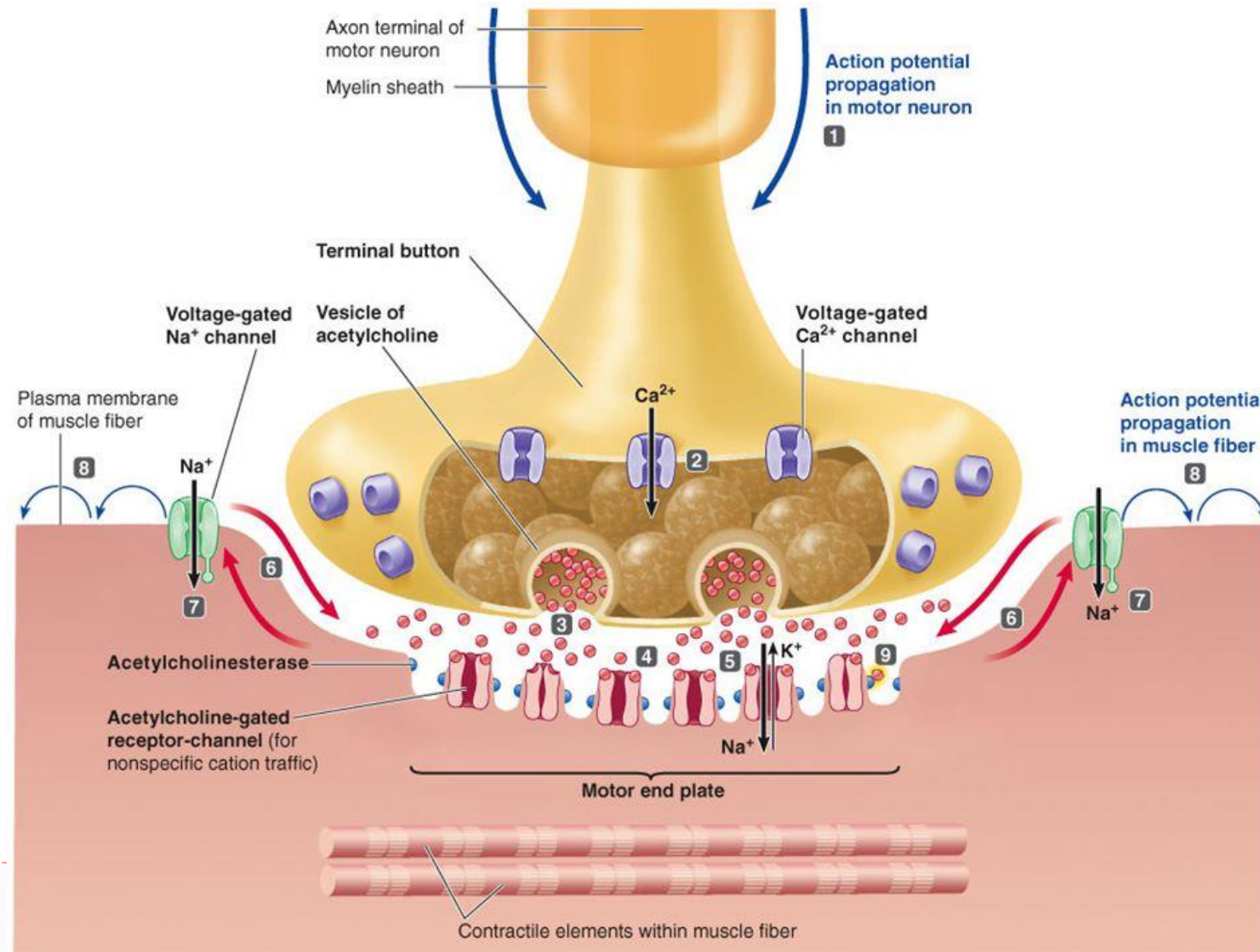
- Describing the neuromuscular junction
- Disorders of the neuromuscular junction (MG , LEMS)
- Disorders of muscle (Acquired , Congenital dystrophic and non- dystrophic)



DESCRIBING THE NEUROMUSCULAR JUNCTION

By Bara'ah Rasheed (C12)

Neuromuscular Junction



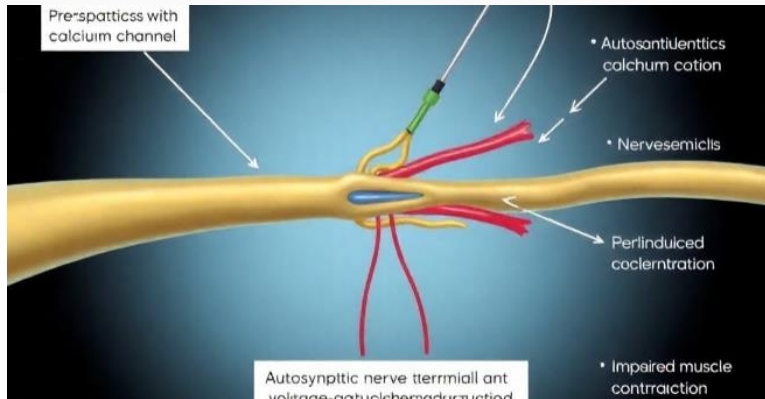
- NMJ disorders are classified as presynaptic, synaptic, and postsynaptic.
- Some NMJ diseases are congenital, immune-mediated (LEMS, MG) or due to toxic and metabolic causes.

Myasthenia Gravis

- autoimmune disease in which the patients have developed antibodies that block or destroy their own nicotinic ACh-receptor proteins at the postsynaptic neuromuscular junction.
- Virtually any skeletal muscle may be affected, notably those of the eyes and face, swallowing muscles, and respiratory muscles.
- In severe cases, the patient may die of respiratory failure as a result of severe weakness of the respiratory muscles.
- Anticholinesterase drugs

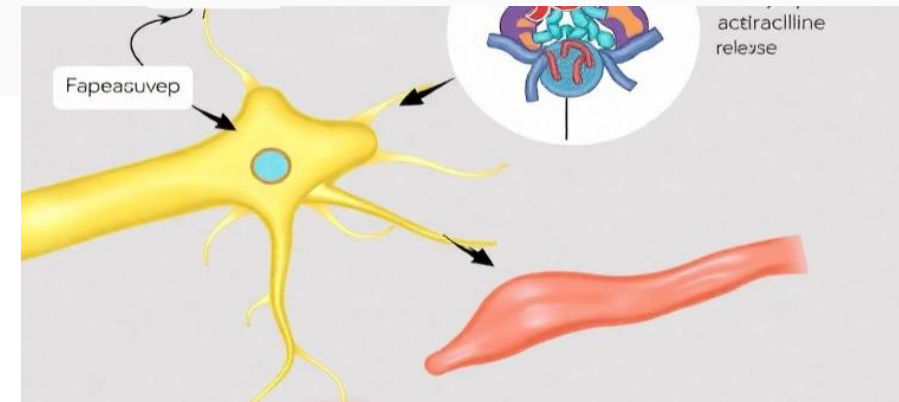
Introduction to Lambert-Eaton Syndrome

By Abdallah Dardas (C12)



Overview of Lambert-Eaton Syndrome (LES)

Lambert-Eaton Syndrome (LES) is a rare autoimmune disorder that affects the neuromuscular junction, causing muscle weakness and other neurological symptoms. It is characterized by the body's immune system mistakenly attacking calcium channels in the nerve cells, leading to impaired communication between the nerves and muscles.



Pathophysiology of LES

The pathophysiology of Lambert-Eaton Syndrome involves the production of antibodies that target voltage-gated calcium channels (VGCCs) in the presynaptic nerve terminals. These autoantibodies disrupt the release of acetylcholine, a neurotransmitter responsible for transmitting signals from the nerves to the muscles. As a result, muscle contraction is impaired, leading to weakness and other symptoms associated with LES.

Causes of Lambert-Eaton Syndrome

Possible Underlying Factors Leading to LES: The exact cause of Lambert-Eaton Syndrome is not fully understood. However, it is often associated with an underlying malignancy, particularly small cell lung cancer. In some cases, LES can occur without an identifiable malignancy, and it may be classified as idiopathic. Other possible underlying factors include autoimmune disorders and certain infections, such as human immunodeficiency virus (HIV) and Epstein-Barr virus (EBV).

Symptoms of Lambert-Eaton Syndrome

Common Signs and Symptoms Experienced by Individuals with LE Syndrome can vary in severity and may include:

Muscle weakness, particularly in the limbs, which may improve with repetitive use

Fatigue and exhaustion

Difficulty walking or climbing stairs

Impaired coordination and balance

Autonomic symptoms, such as dry mouth, constipation, and erectile dysfunction

Diagnosis of Lambert-Eaton Syndrome

Diagnostic Techniques Used to Identify LES

Diagnosing Lambert-Eaton Syndrome involves a combination of clinical evaluation, electromyography (EMG), and blood tests. EMG helps assess the electrical activity of the muscles and can detect the characteristic pattern of muscle fatigue seen in LES. Blood tests may be performed to detect the presence of autoantibodies against voltage-gated calcium channels (VGCCs).

Importance of Thorough Medical History and Neurological Examination

A thorough medical history and neurological examination are crucial in the diagnosis of Lambert-Eaton Syndrome. The healthcare provider will assess the pattern and progression of muscle weakness, review the patient's medical history for any underlying malignancies or autoimmune disorders, and perform a comprehensive neurological examination to evaluate muscle strength, reflexes, and coordination.

Treatment of Lambert-Eaton Syndrome

Management Approaches for LES, Including Medications and Therapies: The treatment of LambertEaton Syndrome aims to improve muscle strength and function, as well as manage symptoms.

Medications: Calcium channel blockers, such as 3,4-diaminopyridine (3,4-DAP), can help enhance nerve-to-muscle communication. Immunosuppressive drugs, such as corticosteroids and azathioprine, may be prescribed to reduce immune system activity and prevent further damage.

Therapies: Physical and occupational therapy can play a vital role in managing muscle weakness and improving mobility. These therapies focus on strengthening muscles, improving coordination, and enhancing daily functioning.

Multidisciplinary Care: The management of Lambert-Eaton Syndrome often requires a multidisciplinary approach, involving neurologists, oncologists (if an underlying malignancy is present), physical and occupational therapists, and other healthcare professionals.

Conclusion

In conclusion, Lambert-Eaton Syndrome is a rare autoimmune disorder characterized by muscle weakness and impaired neuromuscular communication. It is important to understand the differences between Lambert-Eaton Syndrome and Myasthenia Gravis to ensure accurate diagnosis and appropriate treatment. The causes of LES can vary, with an underlying malignancy being a common association. Recognizing the symptoms and conducting thorough diagnostic evaluations are essential in providing effective management. Treatment approaches include medications, therapies, and a multidisciplinary care approach.

DISORDERS OF MUSCLES (Acquired)

By Suzan Al-Zaidanin (C12)

Patients may report “weakness,” a term used to describe a variety of symptoms ranging from fatigue to limited mobility (e.g., due to stiffness). It is, therefore, important to elucidate if the symptom actually reflects a loss of power

- Asking what specific activities pose a challenge (e.g., standing from low chairs, moving in or out of cars, maintaining the arms above shoulder level) can be helpful
- The exam may help characterize the distribution of affected muscle groups further
- Additional information may be gained through laboratory studies, including a creatine kinase (CK) level, and electrodiagnostic studies
- In certain instances, muscle ultrasound and MRI may show pattern-specific abnormalities or help guide the choice of a biopsy site, although biopsies are becoming increasingly rare with advances in genetic testing.

Creatine kinase (CK) is an enzyme that mainly exists in your heart and skeletal muscle, with small amounts in your brain. The cells release CK into your blood when they're damaged.

- Trauma or strenuous exercise can cause CK elevations in the absence of an underlying disease. Conversely, muscle disorders can be discovered incidentally (e.g., through CK or liver function test (LFT) abnormalities) during routine screening

There is an important relationship between liver function tests (LFTs) and muscle disorders, as certain liver enzymes, specifically aspartate aminotransferase (AST) and alanine aminotransferase (ALT), are also found in muscle tissues. Muscle damage can lead to elevated levels of these enzymes, which may sometimes be misinterpreted as liver dysfunction.

While ALT is more specific to the liver, AST is also present in significant amounts in muscle tissues (particularly in skeletal and cardiac muscle).

GGT (Gamma-Glutamyl Transferase): This enzyme is specific to the liver, so normal GGT levels in the presence of high AST/ALT suggest that the source is more likely muscle than liver.

Infectious

- Myalgias: This refers to general muscle aches or soreness, which are common during or after viral infections. Myalgias are typically mild and self-limited
- Rhabdomyolysis: a condition in which muscle fibers break down, releasing myoglobin into the bloodstream. This can lead to complications such as kidney damage and electrolyte imbalances.
- In the United States, enteroviruses and influenza A and B are most frequently linked to self-limited viral myositis
- These viruses typically cause transient inflammation of the muscles, leading to symptoms like muscle pain and tenderness. In most cases, the myositis resolves without significant intervention, as the viral infection clears.
- HIV is linked to a more chronic myopathy, which means muscle inflammation and weakness persist for longer periods, often becoming a lasting issue.
- HIV-related myopathy can develop as a direct result of the virus itself, or as a consequence of the immune system dysfunction caused by HIV. Additionally, certain medications used to treat HIV, such as zidovudine (AZT), can cause muscle damage and contribute to myopathy.

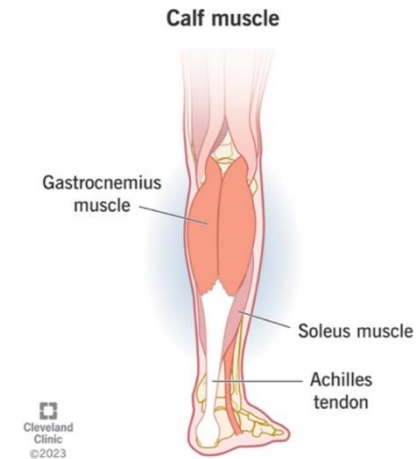
Toxic

1. Acute Alcoholic Myopathy:

Excessive alcohol use can be associated with both acute and chronic myopathies. The former usually occurs after a binge and results in diffuse muscle tenderness, cramps, swelling, and weakness that may be most pronounced in the calves

Malnourished women who suffer from alcohol dependence are most susceptible to the chronic form of myopathy which is gradual in onset and proximal in distribution

- Mechanism: Alcohol interferes with muscle metabolism, causing a breakdown of muscle fibers. This damage can result in the release of creatine kinase (CK) and other muscle breakdown products, potentially leading to rhabdomyolysis in severe cases.
- Recovery: Symptoms typically resolve once the person stops drinking alcohol, although severe cases may require medical intervention for rhabdomyolysis and kidney protection.



- Lipid-lowering and antiretroviral drugs and glucocorticoids can cause myopathies. At doses above 40 to 60 mg per day, prednisone can lead to detectable weakness within 2 weeks. Proximal limb weakness is most common. Muscle enzyme assays and electromyographies (EMGs) are usually normal. Although not typically performed in this setting, a biopsy would be likely to show atrophy of type II fibers. Strength can improve when the glucocorticoid dose is lowered

neuroleptic malignant syndrome can develop because of withdrawal from dopamine agonists used to treat Parkinson disease but is more commonly caused by the use of neuroleptic agents

This muscle stiffness is generalized and can be so intense that it leads to complications like rhabdomyolysis (breakdown of muscle tissue), which can result in kidney failure

NMS is a consideration when mental status change is accompanied by a temperature above 38°C, generalized muscular rigidity (with CK elevations >1,000 µ/L).

Discontinuing the culprit medication is one of the most important steps to take. Sometimes drugs such as dantrolene and bromocriptine are given.

Endocrine

- In hypothyroidism, muscle symptoms often develop gradually and include muscle weakness, stiffness, cramps, and aches. These symptoms are due to slowed metabolic processes in muscle tissue, leading to reduced energy availability for muscle function.

delayed relaxation phase of deep tendon reflexes (DTRs). This means that after the reflex (such as the knee-jerk reflex) is triggered, the muscle takes longer to return to its resting state

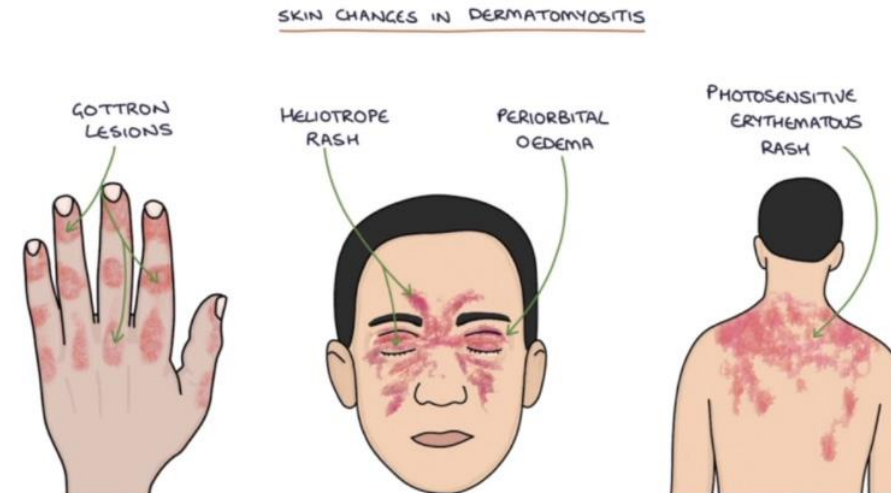
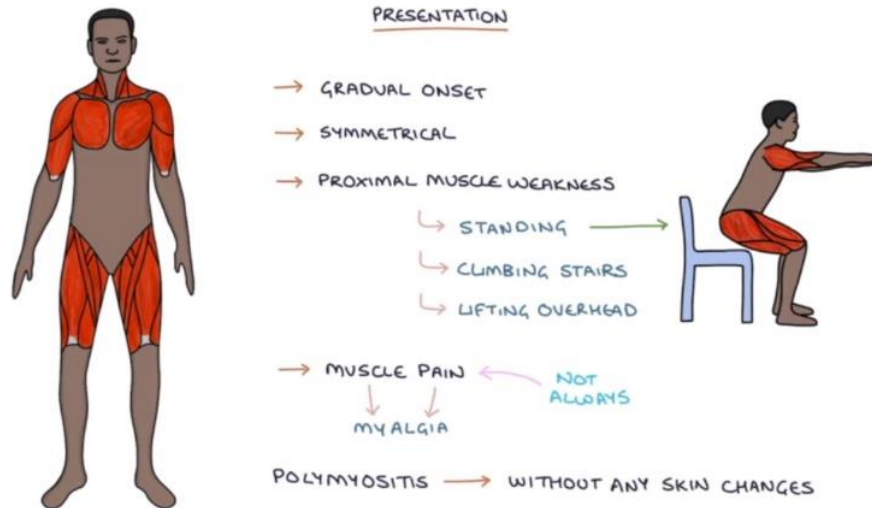
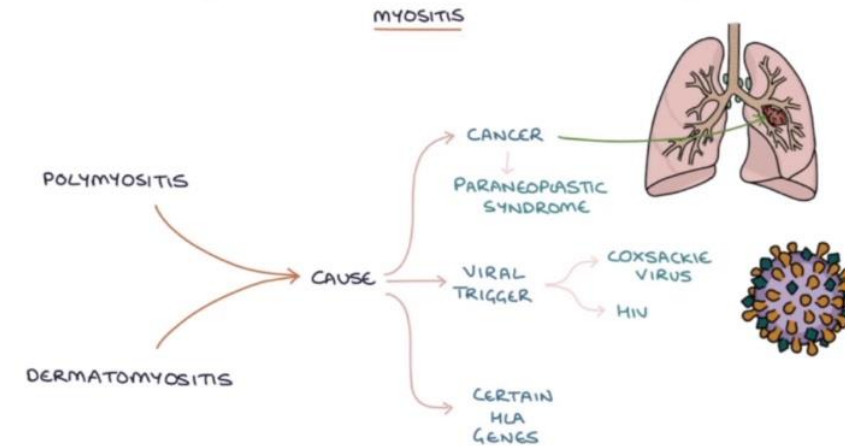
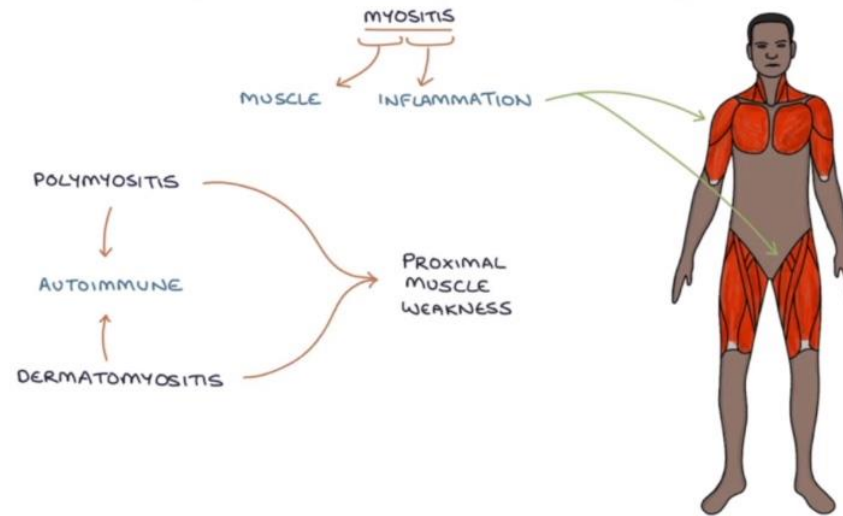
CK levels are often elevated. This is because muscle cell membranes become more permeable in the hypothyroid state,

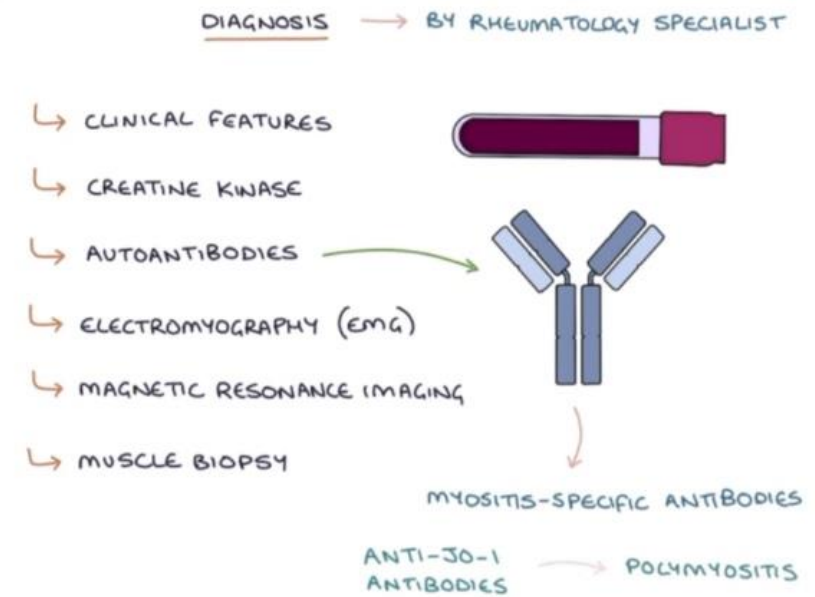
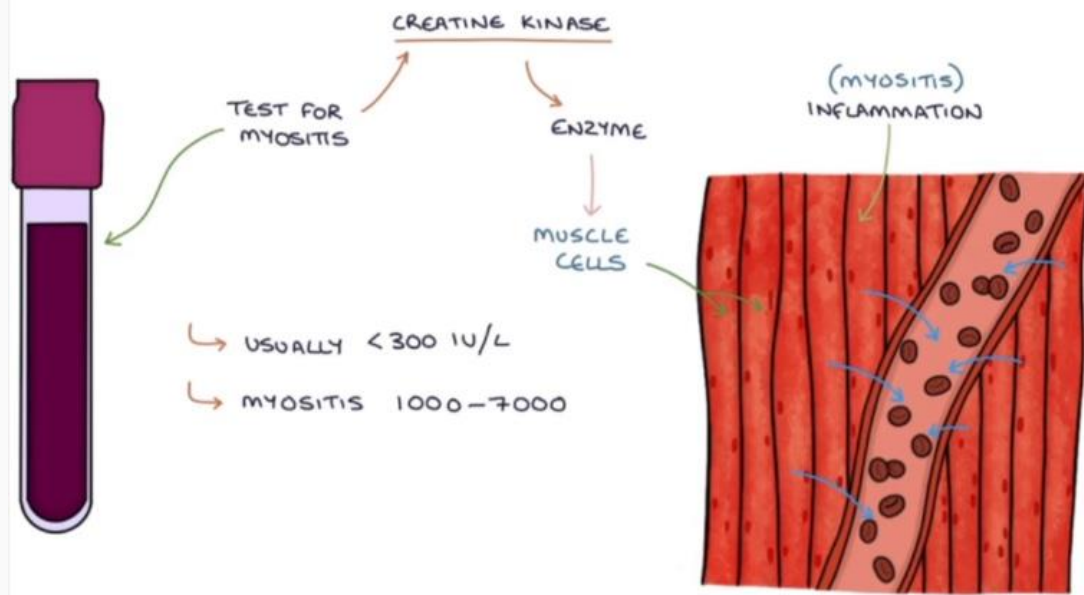
- In hyperthyroidism, muscle symptoms may also include muscle weakness, but they tend to present more acutely or subacutely. Muscle weakness is primarily due to catabolism (breakdown of muscle proteins), as the excess thyroid hormone accelerates metabolism, causing muscle wasting.

hyperactive or increased DTRs. This occurs because the nervous system becomes more excitable

CK levels are typically normal in hyperthyroidism.

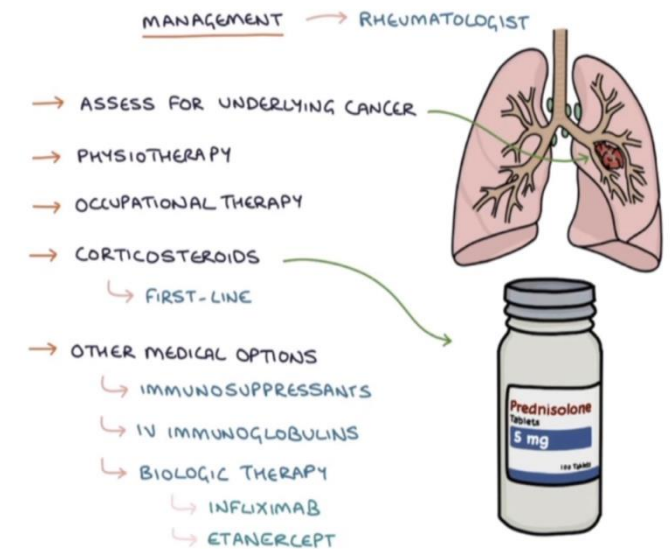
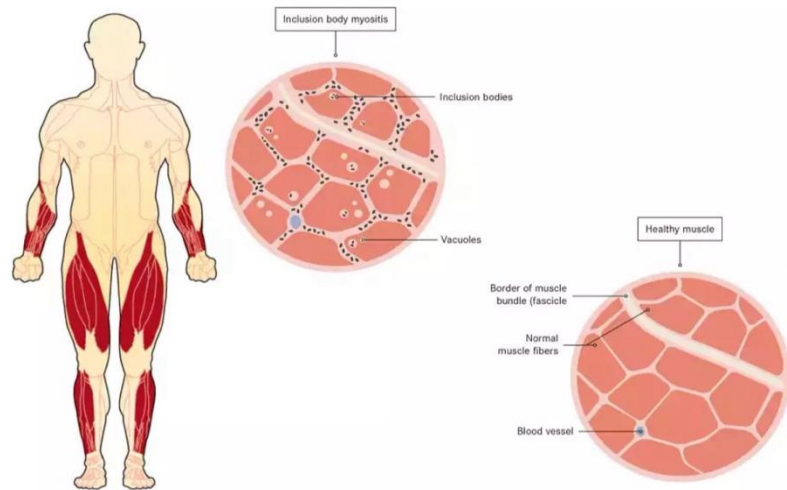
Polymyositis and dermatomyositis





inclusion body myositis

affect proximal legs and forearm flexors.



DISORDERS OF MUSCLES (CONGENITAL NON- DYSTROPHIC)

Metabolic myopathies

By Maram Bashayreh (C12)

Metabolic myopathy present as proximal myopathies, one element of a more diffuse neurologic syndrome with central nervous system dysfunction, or as intermittent myoglobinuria and cramps. The hallmark of metabolic myopathies is exertion-related symptoms, including the development of muscle pain and cramps during exercise. These processes reflect dysfunction in lipid or glycogen metabolism

Mitochondrial myopathies

An important group of metabolic myopathies is the mitochondrial myopathies, which are related to abnormalities in the oxidative phosphorylation pathway and they have several form

Forms

One form is a myopathy associated with short stature and other multisystem abnormalities. An example of this form is mitochondrial encephalomyopathy with lactic acidosis and stroke-like episodes (MELAS)

It is a rare genetic disorder that affects the mitochondria, the parts of cells responsible for producing energy.

Key features of MELAS:

1. **Mitochondrial Encephalomyopathy:** Affects muscles (myopathy) and the brain (encephalopathy) due to problems with energy production in the mitochondria.
2. **Lactic Acidosis:** A buildup of lactic acid in the body, causing the blood to become too acidic. This happens because the mitochondria cannot make enough energy, forcing cells to use an alternative method that produces lactic acid.
3. **Stroke-like episodes:** Episodes that mimic a stroke, causing temporary paralysis, speech issues, or other neurological problems.

Another mitochondrial myopathy is chronic progressive external ophthalmoplegia, a syndrome of bilateral ptosis and weakness of extraocular muscles that worsens over time. A third type is limited to the skeletal muscle; it presents with muscle aches, fatigue with exercise, elevated CK, and sometimes, rhabdomyolysis.

Physiological stressors

In each of these cases, symptom exacerbations can occur because of physiologic stressors such as:

- Cold exposure
- Fasting
- Heavy exercise

Channelopathies

By Bara'ah Nassar (C12)

What is channelopathies?

- channelopathies are a group of disorders caused by dysfunction in ion channels, which are proteins that help regulate the flow of ions (like sodium, potassium, and calcium) across cell membranes. These channels play a crucial role in maintaining the electrical activity of neurons and muscle cells.
- A channelopathy may cause an abnormal gain of function (such as myokymia, myotonia, and epilepsy) or an abnormal loss of function, (such as weakness or numbness) depending on whether loss of channel function leads to excessive membrane excitability or to membrane inexcitability.

hyper- and hypokalemic periodic paralysis (PP):

- In both of these rare conditions, patients have bouts of painless weakness, most pronounced in proximal limbs.
- Bulbar and respiratory muscles are generally not affected, and consciousness is preserved.
- During symptoms, a chemistry panel may demonstrate a high or a low potassium level.

Hyperkalemic periodic paralysis:

- The condition is often caused by mutations in the SCN4A gene, which encodes a sodium channel critical for muscle function.

Symptoms:

- Muscle Weakness: Sudden, episodic weakness, often affecting the limbs, which may vary in severity.
- Paralysis: In more severe cases, temporary paralysis can occur, particularly in the arms and legs.
- Muscle Cramps: Painful muscle cramps or spasms may precede or accompany weakness.
- Triggered Episodes: Attacks can be triggered by factors such as rest after exercise, high potassium meals, or stress.
- Recovery: Weakness typically resolves on its own, although it may take time for full recovery.

Hypokalemic pp:

- hypokalemic periodic paralysis results from mutations in the gene coding for the $\alpha 1$ subunit of a skeletal muscle calcium channel.

Symptoms:

- Weakness or paralysis: Typically occurs in the arms and legs but can affect other muscle groups.
- Duration: Episodes can last from minutes to several days.
- Common triggers include: High carbohydrate meals ,Strenuous exercise followed by rest , Stress or anxiety, Changes in temperature (heat or cold).

Diagnosis

- ♦ Blood Tests: Measure serum potassium levels during an episode to confirm hyperkalemia. Check for other electrolyte imbalances.
- ♦ Electromyography(EMG):May be conducted to evaluate muscle response and assess for abnormalities during attacks.
- ♦ Genetic Testing :Testing for mutations in genes associated with ion channels (e.g., SCN4A) to confirm the diagnosis.

treatment:

- ♦ Abortive treatments include calcium or beta-adrenergic agonists (for hyperkalemic PP) or potassium chloride without dextrose (for hypokalemic PP).
- ♦ Prophylactic therapies include acetazolamide (carbonic anhydrase inhibitors), but attacks may also be avoided by limiting exercise and carbohydrate intake.

Important notes:

Whenever PP is a diagnostic consideration, obtaining an ECG is important because the channelopathies and associated electrolyte abnormalities can predispose to arrhythmias, including with a prolonged QTc. Caution with general anesthesia, and avoidance of depolarizing agents, is recommended to avoid paralysis and complications with extubation.

DISORDERS OF MUSCLES (CONGENITAL DYSTROPHIC)

Duchenne and Becker Muscular Dystrophies

By Marwan Al-Shalash (C12)

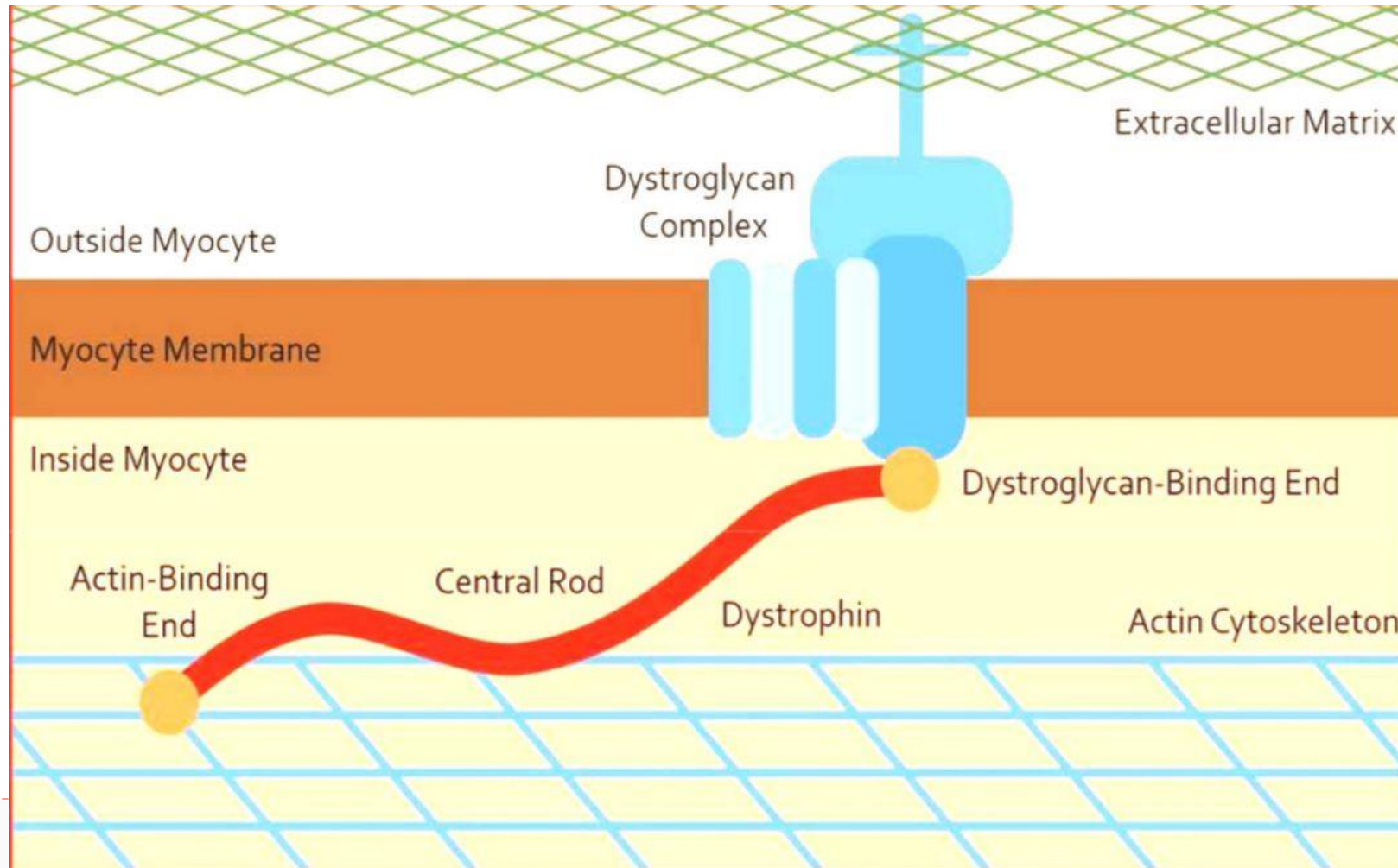
CONGENITAL MUSCLE DISORDERS

- NONDYSTROPHIC
- DYSTROPHIC
 - 1) Duchenne (DMD) and Becker Muscular Dystrophies (BMD)
 - 2) Emery–Dreifus Muscular Dystrophy (EDMD)
 - 3) Myotonic Dystrophy
 - 4) Facioscapulohumeral Muscular Dystrophy
 - 5) Limb-girdle Muscular Dystrophy
- Main symptom: progressive muscle weakness

THE DYSTROPHIN-GLYCOPROTEIN COMPLEX IN SKELETAL MUSCLE

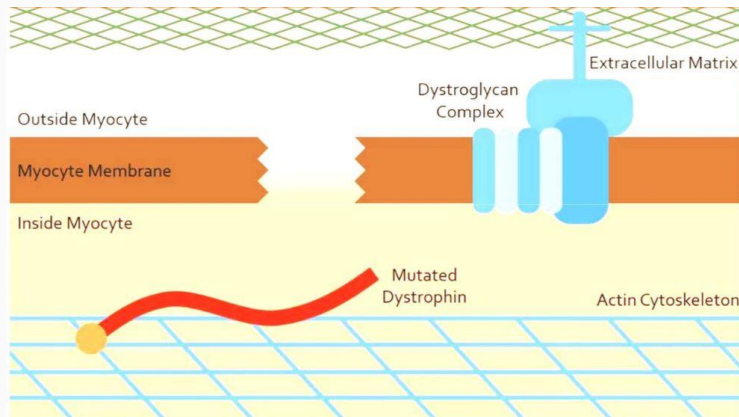
- The dystrophin-glycoprotein complex (DGC) is a multi-subunit complex within and across the membranes of cardiac and skeletal muscle cells as well as vascular smooth muscle cells.
- The complex mediates interactions between the cytoskeleton, Membrane, and the extracellular matrix.
- The complex is composed of up to 15 different proteins. The major intracellular protein is the large dystrophin protein.
- Dystrophin protein is integral to the structural stability of the myofiber. Without dystrophin, muscles are susceptible to mechanical injury and undergo repeated cycles of necrosis and regeneration.
- Ultimately, regenerative capabilities are exhausted or inactivated.

- The dystrophin gene (one of the largest human genes described to date) occupies almost 2% of the X chromosome and nearly 0.05% of the entire genome, **WHICH MEANS THAT THE DYSTROPHIN GENE IS MORE SUSCEPTIBLE TO MUTATION.**

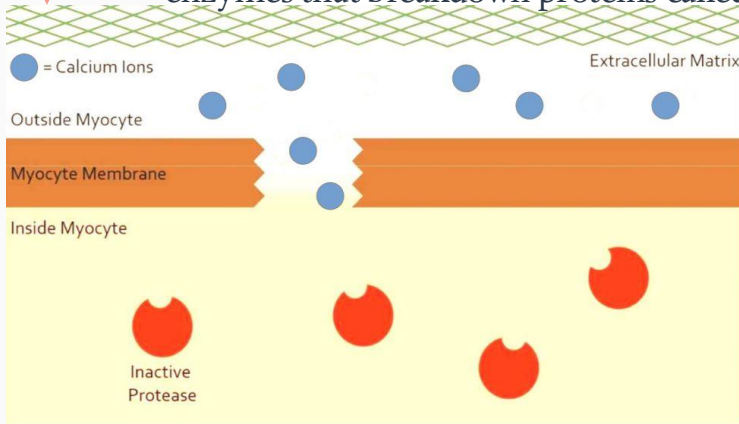


1) Duchenne (DMD)

Caused by **absent** dystrophin gene due to **nonsense / frameshift** mutation



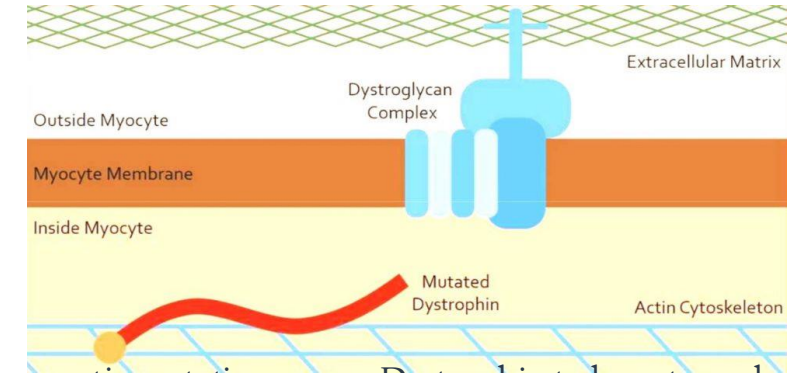
3) These rips allow Ca ions found outside the myocytes flow in through these small rips and activate Ca dependent intracellular enzymes that breakdown proteins called protease



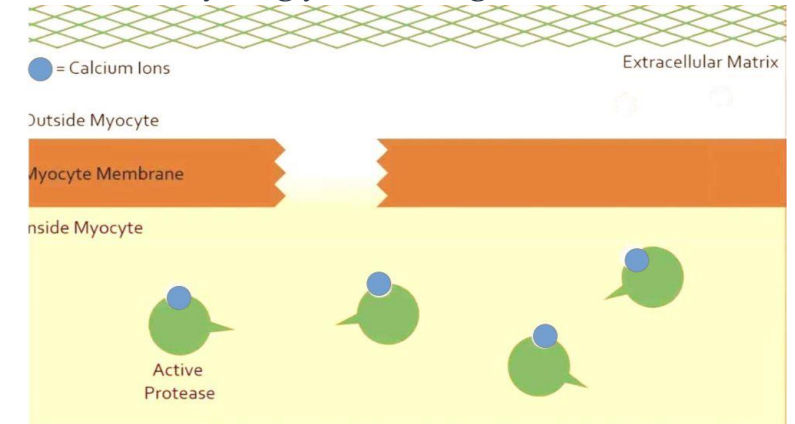
and

Becker (BMD)

Caused by **misshapen** dystrophin gene due to **missense** mutation
Some functioning protein



1) a genetic mutation causes Dystrophin to be extremely short lacking the dystroglycan binding end.



2) Because of this every time the muscle contracts, small rips appear in the membrane

4) extremely high ca level activates too many of proteases which break down important functional proteins, killing the myocyte in the process

- Another important molecules that diffuse through the rips is creatine kinase which leaks out of the cell and eventually into the blood (increased level of creatine kinase in the blood), No energy stores which weakens the muscle and replacment of the muscles tissue with fatty tissue

1) Duchenne (DMD)

and

Becker (BMD)

• *PATHOLOGY & CAUSES*

Caused by **absent** dystrophin gene due to **nonsense/frameshift** mutation

X-linked recessive inheritance pattern
mostly male patients

• *SIGNS & SYMPTOMS*

MNEMONIC: DMD
Cause of Duchenne Muscular
Dystrophy
Doesn't
Make
Dystrophin

MNEMONIC: BMD
Cause of Becker's Muscular
Dystrophy
Badly
Made
Dystrophin (truncated protein)

• *PATHOLOGY & CAUSES*

Caused by misshapen dystrophin gene due to missense mutation (Some functioning protein)

X-linked recessive inheritance pattern mostly
male patients

• *SIGNS & SYMPTOMS*

- 1) Milder form, later onset than Duchenne muscular dystrophy
- 2) Symptoms appear 10–20 years old
- 3) Intellectual disability not as common/severe as Duchenne muscular dystrophy



- 1) Affected boys typically develop proximal weakness in early childhood; weakness usually occurs 2–3 years old
- 2) Walking begins later in childhood; may have slow, ungainly run; difficulty jumping.
- 3) Proximal-limb muscle weakness before distal, lower extremities before upper decreased reflexes
- 4) Gowers' sign
- 5) Waddling gait
- 6) Calf muscles pseudohypertrophy (replacement of muscle fibres with fatty connective tissue)
- 7) Decreased mobility, May lead to independent ambulation impairment, wheelchair-use by 12 years old (usually)
- 8) Primary dilated cardiomyopathy
- 9) The disease progresses relentlessly, with death usually from cardiac and respiratory complications before the age of 20, though with better supportive care some patients are now surviving into early adult life.
- 10) **Scoliosis**, **kyphosis** and **lordosis** and abnormal seating postures are almost invariable features in patients affected by neuromuscular disorders, of which Duchenne muscular dystrophy (DMD) is the most common.

1) Duchenne (DMD)

and

Becker (BMD)

• COMPLICATIONS

- 1) Wheelchair needed for mobility
Scoliosis → poor pulmonary function
- 2) Weak diaphragm → respiratory failure (may develop)
- 3) Fibrosis progression in dilated cardiomyopathy → mitral regurgitation (may develop)
- 4) Dilated cardiomyopathy (late stages) → heart failure, arrhythmias (may develop)
- 5) Falling → fractures
- 6) Respiratory insufficiency/cardiomyopathy
- 7) death (late teens, early twenties)

• DIAGNOSIS

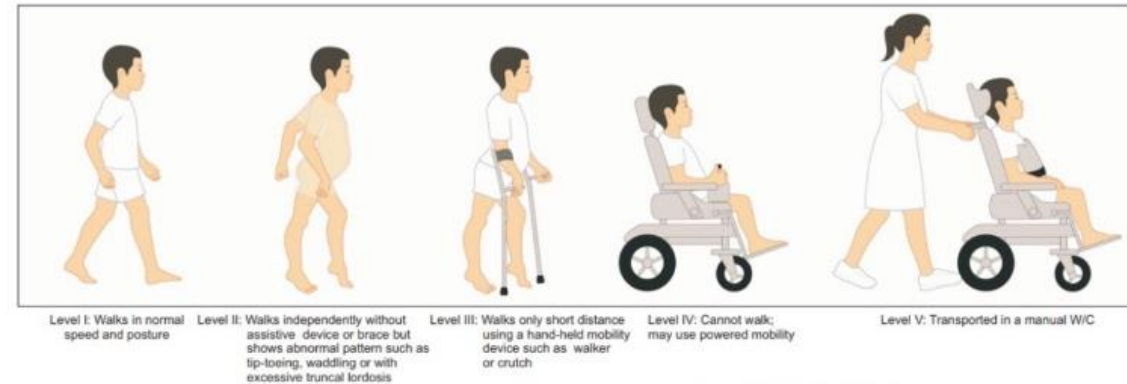
- 1) Genetic testing is key in establishing a diagnosis
- 2) ↑ serum creatine kinase
- 3) Muscle biopsy: Stain for dystrophin

• TREATMENT

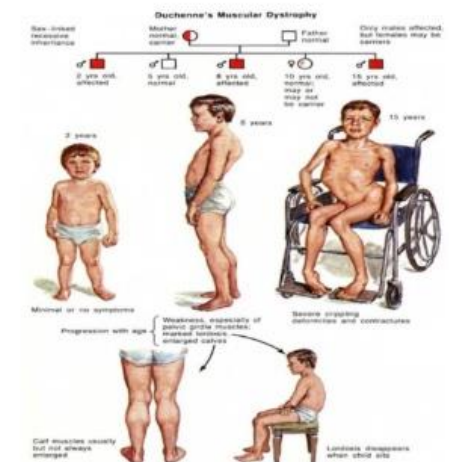
- 1) No cure, gene therapy is promising path
- 2) MEDICATIONS: Glucocorticoids to slow muscle degeneration
- 3) OTHER INTERVENTIONS: Vitamin D, calcium supplements support bone health, Physical therapy and Complication management

• COMPLICATIONS

- 1) *same but in milder form*
- 2) *can live beyond 30 years of age*



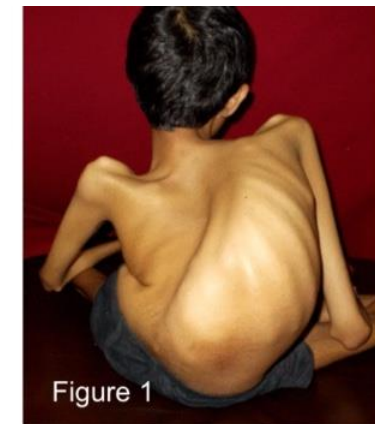
Gowers' sign



Case for reading

A 15-year-old boy presented with progressive proximal weakness of the lower limbs starting at 4 years of age followed by involvement of the upper limbs. He is the product of a consanguineous marriage; he had a family history of similar disease in a second-degree cousin and also had a history of delayed motor developmental milestones since birth. Clinically, he had flaccid quadriparesis with wasting and contractures without any sensory or neurological involvement. His weakness worsened leading to an inability to walk without support by the age of 9 and total wheelchair dependence by the age of 12. He was frequently admitted to hospital with chest infections.

The patient's creatine kinase was 2600 IU/L (normal 50–150 IU/L) and muscle biopsy from left quadriceps showed rounded small muscle fibres with evidence of degeneration and an absence of dystrophin protein. He was diagnosed as a case of duchenne muscular dystrophy. He is presently bed bound with weakness and contractures of all limbs and spinal deformities as shown in figure 1. He developed scoliosis at the age of 12 which has gradually worsened to the present state as shown in figures 2 and 3.



Emery-Dreifus Muscular Dystrophy and Myotonic Dystrophy

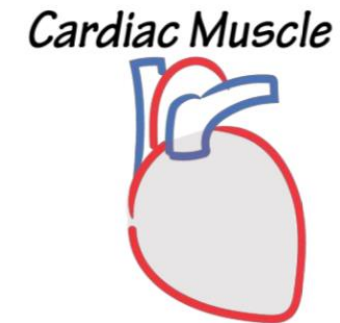
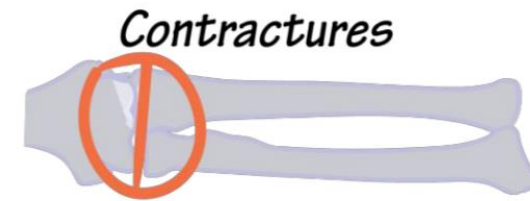
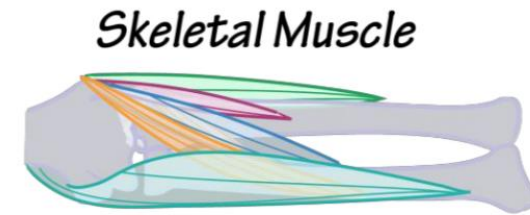
By Deema Bzour (C12)

Emery-Dreifus Muscular Dystrophy

- Genetic degenerative disease primarily affect voluntary muscles
- Its inheritance pattern is variable
- Mutation in the genes that produce nuclear membrane proteins of muscle cell:
 - In X-linked EDMD → defect of emerin protein
 - In AD and AR → defect of lamin A and lamin C
- The onset of symptoms is usually by 10-15



- EDMD characterized by clinical triad :
 1. Slowly progressive **muscle weakness** begins in humeroperoneal distribution , with involvement of biceps, triceps, and tibialis anterior
 2. **Contractures**(stiff joints) of the Achilles tendons(toe-walking),elbow flexors and neck presented very early
 3. **Cardiac involvement**-conduction defects, arrhythmias and dilated cardiomyopathy
- Genetic testing can provide a definitive diagnosis
- Treatment is supportive (physical therapy, anti arrhythmic drugs , cardiac pacemaker)



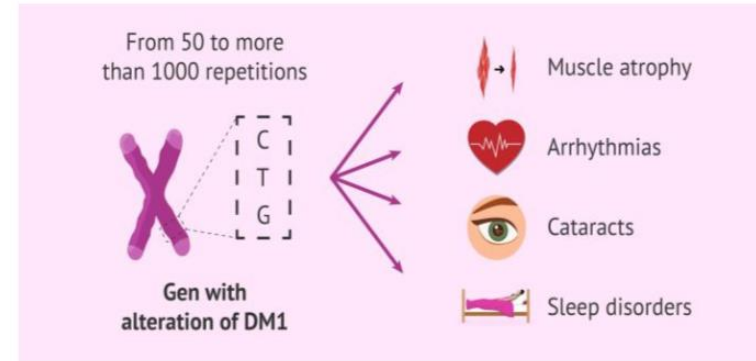
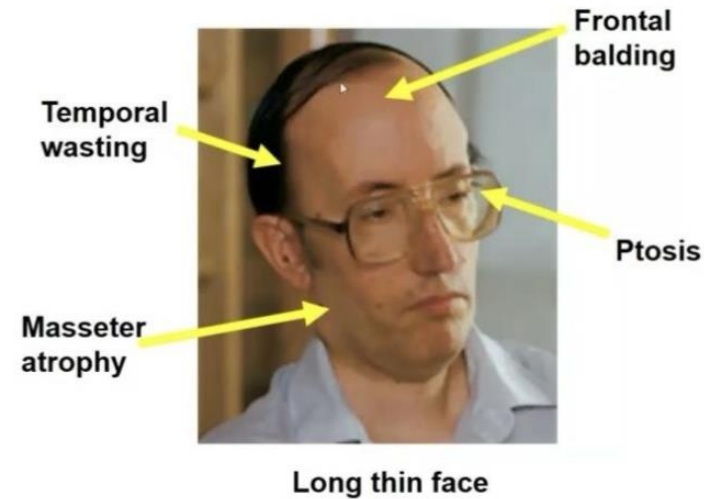
Myotonic Dystrophy

- Is an autosomal dominant multisystem disorder that affects muscles and other organs in the body
- Characterized by muscle weakness and delayed relaxation of skeletal muscles after voluntary contraction or myotonia
- It's a nucleotide repeat expansion disease :
 - DM 1:mutation in DMPK(dystrophia myotonica protein kinase)gene / CTG
 - DM 2:mutation in CNBP(CCH-type zinc finger ,nucleic acid binding protein)gene / CCTG

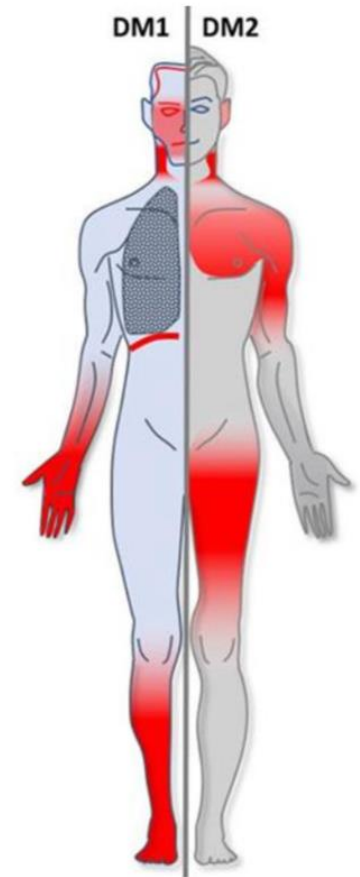


Clinical features:

- Narrow, elongated face, frontal balding, temporal wasting, ptosis, and horizontal Smile
- Sleep disorder
- Endocrine disturbances
- Cataract
- Cardiac conduction abnormalities



- the two forms of the disorder can be distinguished by the time of symptom onset, the distribution of weakness, and the presence of pain
- Patients with DMI usually present in their late teens with distal weakness and Wasting
- Whereas DMII usually presents in adults in their 40s or older with proximal Weakness
- Pain in the arms, thighs, and back is more frequent in DMII



(FSHD) and Limb-girdle muscular dystrophy

By Reem Al-Sbaihat (C12)

Facioscapulohumeral muscular dystrophy (FSHD)

It's an AD gene mutation in **DUX4**.

It presents with **facial weakness**, **scapular winging**, **upper arm weakness**, and also leg and abdominal muscular weakness/aches.



Limb-girdle muscular dystrophy

It's a group of disorders characterized by progressive loss of power and muscular bulk in **shoulder and pelvic girdle (proximal)**.

They differ in age, onset, severity, and **gene mutations**. (sarcoglycan, calpain, caveolin, and dysferlin)

>>It may affect muscles of the heart and respiration.

>>Diagnosis is done by genetic testing, labs (CK elevation) .

Thank You

Resources:
Blueprints Neurology 5th edition

