

DUCHENNE MUSCULAR DYSTROPHY: FROM MOLECULAR MECHANISMS TO CLINICAL MANAGEMENT

Mussa Vaishnavi*, Syeda Nishat Fathima

Department of Pharmacy Practice, Jayamukhi College of Pharmacy, Narsampet, Warangal-506332,
Telangana, India

ABSTRACT

Duchenne muscular dystrophy (DMD) is a severe, progressive X-linked recessive neuromuscular disorder caused by mutations in the dystrophin gene, resulting in absence of dystrophin protein. It predominantly affects males and is one of the most common inherited myopathies, with an incidence of approximately 1 in 3,500–5,000 live male births. The disease is characterized by progressive muscle degeneration, initially involving proximal muscles and later affecting cardiac and respiratory muscles. Clinical manifestations typically begin in early childhood and include delayed motor milestones, muscle weakness, Gowers' sign, and pseudohypertrophy. Diagnosis is primarily confirmed by genetic testing, supported by elevated creatine kinase levels and, in some cases, muscle biopsy. Although no definitive cure exists, management focuses on slowing disease progression and improving quality of life through corticosteroid therapy, multidisciplinary care, and emerging gene-based treatments. Despite advances, DMD remains a life-limiting condition with significant morbidity and mortality due to cardiopulmonary complications.

Keywords: Duchenne muscular dystrophy; dystrophin; X-linked recessive disorder; muscle degeneration; cardiomyopathy; neuromuscular disorder; creatine kinase; paediatric myopathy

INTRODUCTION:

Duchenne muscular dystrophy Duchenne muscular dystrophy (DMD) is a severe, progressive neuromuscular disorder caused by mutations in the dystrophin gene located on chromosome Xp21. These mutations result in the absence or near absence of dystrophin, a crucial structural protein responsible for maintaining the integrity of muscle cell membranes. The lack of dystrophin leads to membrane instability, muscle fiber damage, and progressive degeneration. DMD follows an X-linked recessive inheritance pattern and primarily affects males, while females are typically carriers, although some may exhibit mild clinical manifestations due to skewed X-chromosome inactivation. DMD is recognized as one of the most common and severe inherited muscular dystrophies, with onset usually occurring in early childhood. Clinical symptoms typically begin around the age of four years and include delayed motor milestones, progressive muscle weakness, and difficulty in activities such as running, climbing stairs, and rising from the floor. The disease initially affects the proximal muscles of the pelvis and thighs and gradually progresses to involve the upper limbs, respiratory muscles, and cardiac muscle. By early adolescence, most affected individuals lose the ability to walk and become wheelchair-dependent. Characteristic features such as pseudohypertrophy of calf muscles, scoliosis, and muscle contractures are commonly observed as the disease advances. At the molecular level, DMD is caused by mutations or deletions in any of the 79 exons of the dystrophin gene, leading to disruption of the dystrophin protein and subsequent muscle fiber degeneration. Approximately two-thirds of cases are inherited from carrier mothers, while the remaining one-third arise from de novo mutations. The progressive muscle weakness ultimately leads to severe disability, with patients becoming dependent on assistance for daily activities. In later stages, involvement of respiratory and cardiac muscles results in life-threatening complications such as respiratory failure and cardiomyopathy, which are the leading causes of mortality. Despite significant advances in diagnostic techniques and supportive management, DMD remains an incurable disorder with substantial clinical

burden. Current treatment strategies primarily focus on slowing disease progression and improving quality of life through glucocorticoid therapy, physiotherapy, and multidisciplinary care. The aim of this review is to provide a comprehensive overview of Duchenne muscular dystrophy, including its epidemiology, etiology and genetics, pathophysiology, clinical manifestations, diagnostic approaches, and current management strategies. [1]

EPIDEMIOLOGY

Duchenne muscular dystrophy is the most common form of muscular dystrophy and shows a clear male predominance due to its X-linked recessive inheritance pattern. Global epidemiological data from systematic reviews indicate that the pooled prevalence of DMD is approximately 7.1 cases per 100,000 males and about 2.8 cases per 100,000 individuals in the general population. The global birth prevalence is estimated to be around 19.8 per 100,000 live male births, reflecting its relatively high occurrence among inherited neuromuscular disorders. The incidence of DMD is commonly reported as 1 in 3,500–5,000 live male births, making it one of the most frequent genetic disorders affecting boys. Approximately one-third of cases arise from de novo mutations, while the remaining cases are inherited from carrier mothers with relatively consistent distribution across different geographic regions. Although DMD is rare in females, they may act as carriers and, in rare circumstances, can manifest symptoms. [2]

AETIOLOGY AND GENETICS:

Duchenne muscular dystrophy is caused by mutations in the dystrophin (DMD) gene located on the short arm of the X chromosome at position Xp21 and is inherited in an X-linked recessive pattern, although nearly one-third of cases arise due to spontaneous (de novo) mutations during germline transmission. The dystrophin gene is one of the largest in the human genome, spanning approximately 2.5 megabases and containing 79 exons, and it encodes dystrophin, a large cytoskeletal protein (~427 kDa) essential for maintaining muscle cell integrity. Dystrophin forms a critical link between the intracellular actin cytoskeleton and the extracellular matrix via a multi-protein complex, thereby stabilizing the sarcolemma during muscle contraction. In the absence or severe reduction of dystrophin, the muscle cell membrane becomes fragile, allowing excessive calcium influx into the cell, which leads to muscle fiber damage, repeated cycles of necrosis and regeneration, and eventual replacement of muscle tissue with fat and fibrous connective tissue.

Mutations in the dystrophin gene give rise to a spectrum of disorders known as dystrophinopathies, including Duchenne muscular dystrophy, Becker muscular dystrophy, and intermediate phenotypes, with disease severity depending on the type of mutation and residual dystrophin production. The majority of mutations are deletions (60–70%) and duplications (5–15%), while point mutations and small insertions or deletions account for approximately 20% of cases. These mutations commonly occur in hotspot regions of the gene, particularly within exons 45–55 and exons 3–9. In addition to these common mutations, rare genetic mechanisms such as large chromosomal rearrangements and X-autosome translocations have been reported, which may lead to disease expression even in females due to non-random X-chromosome inactivation.

Although DMD predominantly affects males, it can rarely occur in females (approximately 1 in 50 million births), particularly in cases involving Turner syndrome, X-autosome translocations, or skewed X-inactivation. Female carriers are usually asymptomatic; however, about 2.5–20% may exhibit clinical manifestations such as mild muscle weakness or cardiomyopathy, explained by the Lyon hypothesis, where preferential inactivation of the normal X chromosome results in expression of the mutated allele. Additionally, daughters of an affected father and a carrier mother have a 50% probability of being affected and a 50% chance of being carriers. De novo mutations and germline mosaicism contribute significantly to disease occurrence, and even mothers without detectable mutations may have an increased recurrence risk, with mosaicism reported in up to 14% of germ cells. Dystrophin is primarily expressed in skeletal and cardiac muscle but is also present in the brain and retina, which explains the associated cognitive and neurological manifestations observed in some patients. [3]

PATHOPHYSIOLOGY:

Duchenne muscular dystrophy is characterized by a progressive degeneration of skeletal and cardiac muscle resulting from the absence of dystrophin, a key cytoskeletal protein essential for maintaining muscle cell stability. Dystrophin plays a fundamental role in linking the intracellular actin cytoskeleton to the extracellular matrix through the dystrophin–glycoprotein complex, thereby preserving the structural integrity of the sarcolemma during cycles of muscle contraction and relaxation. In healthy muscle fibers, this connection acts as a mechanical stabilizer that distributes contractile forces evenly across the cell membrane. However, in DMD, the absence of dystrophin disrupts this critical linkage, leading to destabilization of the sarcolemma and increased susceptibility to mechanical stress-induced injury.

One of the earliest and most significant consequences of dystrophin deficiency is membrane instability. The sarcolemma becomes fragile and prone to microtears during normal muscle activity, allowing the uncontrolled entry of extracellular calcium ions into the muscle fibers. This abnormal influx of calcium disrupts intracellular calcium homeostasis, which is tightly regulated under physiological conditions. Elevated intracellular calcium levels activate calcium-dependent proteases such as calpains, which degrade essential structural and regulatory proteins within the muscle cell. In addition, calcium overload adversely affects mitochondrial function, leading to impaired ATP production and opening of the mitochondrial permeability transition pore, further contributing to cellular injury and energy depletion. Mitochondrial dysfunction is a central component of DMD pathophysiology. Excess intracellular calcium accumulation within mitochondria disrupts oxidative phosphorylation, reduces energy availability, and promotes the generation of reactive oxygen species (ROS). These ROS cause oxidative damage to cellular proteins, lipids, and DNA, exacerbating muscle fiber degeneration. Normally, a balance exists between ROS production and antioxidant defenses; however, in DMD, this balance is disrupted, leading to oxidative stress and further weakening of muscle cells. The combination of calcium dysregulation and oxidative damage accelerates the degeneration of muscle fibers and contributes to disease progression.

Another key pathological feature of DMD is chronic inflammation. Muscle fiber damage leads to the release of intracellular components that act as damage-associated molecular patterns, triggering an immune response. Infiltration of inflammatory cells such as macrophages and T lymphocytes occurs in affected muscle tissue. While acute inflammation is essential for muscle repair, persistent inflammation in DMD becomes detrimental, as it perpetuates tissue damage and interferes with effective regeneration. Pro-inflammatory cytokines further amplify muscle injury and contribute to the progressive decline in muscle function. Initially, muscle fibers attempt to regenerate through activation of satellite cells, which are responsible for muscle repair. However, repeated cycles of degeneration and regeneration eventually exhaust the regenerative capacity of these cells. As the disease progresses, muscle tissue is progressively replaced by fibrous connective tissue and adipose tissue, a process known as fibrosis and fatty infiltration. This replacement not only reduces muscle mass but also impairs contractile function, leading to the characteristic muscle weakness observed in DMD patients. Fibrosis also increases muscle stiffness and further compromises functional capacity.

The involvement of cardiac and respiratory muscles significantly contributes to morbidity and mortality in DMD. In cardiac muscle, dystrophin deficiency leads to progressive cardiomyocyte damage, fibrosis, and the development of dilated cardiomyopathy. Similarly, respiratory muscles, particularly the diaphragm, become weakened, resulting in reduced ventilatory capacity and increased susceptibility to respiratory infections. These complications are major determinants of disease severity and patient survival. In addition to muscular involvement, dystrophin is also expressed in the brain, albeit at lower levels. Its deficiency has been associated with cognitive and behavioral abnormalities in some patients, including learning difficulties and neurodevelopmental disorders. This highlights the multisystem nature of DMD beyond skeletal muscle pathology. [4]

CLINICAL MANIFESTATIONS:

Duchenne muscular dystrophy is a progressive neuromuscular disorder characterized by early-onset muscle weakness and multisystem involvement affecting skeletal, cardiac, and respiratory systems. Affected individuals typically appear normal at birth, and symptoms usually become evident in early childhood, often before the age of five years. Initial manifestations commonly include delayed motor milestones, such as late walking (often after 18 months), and difficulty in performing age-appropriate physical activities. As the child begins to walk, characteristic abnormalities in gait become apparent, including a waddling, wide-based gait, toe walking, and frequent falls due to proximal muscle weakness, particularly involving the pelvic girdle muscles.

The disease primarily affects voluntary muscles, with early involvement of the hips, thighs, pelvis, and calves. Progressive weakness leads to difficulty in climbing stairs, running, or rising from the floor. One of the hallmark clinical signs of DMD is the Gowers' sign, in which the child uses the hands to "walk up" the thighs in order to stand from a sitting or lying position, reflecting proximal muscle weakness. Muscle fatigue is common, and affected children often display an awkward or clumsy walking pattern. As the disease progresses, weakness extends to the shoulder girdle, upper limbs, and neck muscles, followed by involvement of respiratory muscles.

A characteristic physical finding is pseudohypertrophy, particularly of the calf muscles, where enlargement occurs due to infiltration of fat and connective tissue rather than true muscle hypertrophy. Similar changes may also be observed in the tongue, buttocks, and shoulder muscles. Over time, muscle fiber degeneration leads to contractures, especially of the Achilles tendon and hamstrings, resulting in reduced joint mobility and further impairment of function. Skeletal deformities such as lumbar hyperlordosis, scoliosis, anterior pelvic tilt, and chest wall abnormalities may develop as compensatory mechanisms for muscle weakness and postural instability.

The progression of DMD leads to a gradual loss of ambulation, typically occurring between 7 and 13 years of age, after which patients become wheelchair-dependent. As mobility declines, complications such as joint contractures and neuromuscular scoliosis become more pronounced. In advanced stages, most patients experience severe physical disability, often described as near-complete paralysis of voluntary muscles by early adulthood.

Cardiac involvement is a major feature of DMD and includes the development of dilated cardiomyopathy, arrhythmias, and, in some cases, congestive heart failure. Cardiomyopathy is often detectable during adolescence and progressively worsens with age. Respiratory complications also play a critical role in disease progression, as weakness of the diaphragm and intercostal muscles leads to restrictive lung disease, reduced vital capacity, and ineffective cough. This predisposes patients to recurrent respiratory infections, pneumonia, and respiratory failure. Additionally, obstructive sleep apnea and nocturnal hypoventilation may occur in later stages.

DMD also has non-musculoskeletal manifestations due to the expression of dystrophin in the brain. Affected individuals may exhibit cognitive and neurobehavioral abnormalities, including learning disabilities, reduced intelligence quotient, attention-deficit/hyperactivity disorder, and difficulties with short-term memory and language processing. Although these cognitive impairments are generally non-progressive, they contribute to the overall disease burden.[5]

DIAGNOSIS:

The diagnosis of Duchenne muscular dystrophy involves a combination of clinical evaluation, laboratory investigations, genetic testing, and, in selected cases, muscle biopsy. Early and accurate diagnosis is essential to initiate appropriate management, provide genetic counseling, and improve patient outcomes. DMD is often suspected in young boys presenting with delayed motor milestones, progressive muscle weakness, frequent falls, and characteristic signs such as Gowers' sign. Elevated serum enzyme levels, particularly creatine phosphokinase, may be an early indicator and often prompt further diagnostic evaluation.

Genetic testing is the cornerstone of diagnosis and can confirm DMD in the majority of cases. A blood-based DNA test is used to identify mutations in the dystrophin gene, which consists of 79 exons. Modern molecular techniques such as multiplex polymerase chain reaction, multiplex ligation-dependent probe amplification, and gene sequencing allow detection of deletions, duplications, and point mutations with high accuracy, often exceeding 95%. These tests not only confirm the diagnosis but also help determine the specific mutation, which is important for prognosis, genetic counselling, and eligibility for mutation-specific therapies. Prenatal diagnosis is also possible through techniques such as chorionic villus sampling, amniocentesis, and non-invasive testing using cell-free fetal DNA, especially when the mother is a known or suspected carrier.

In cases where genetic testing does not identify a mutation, a muscle biopsy may be performed. This involves obtaining a small sample of muscle tissue, usually from the vastus lateralis, for histopathological and protein analysis. Key diagnostic techniques include immunohistochemistry, immunocytochemistry, and immunoblotting to assess the presence, absence, or quantity of dystrophin protein. The absence of dystrophin confirms DMD, while reduced or partially functional dystrophin suggests milder dystrophinopathies such as Becker muscular dystrophy. Histological examination typically reveals variation in muscle fiber size, degeneration and regeneration of fibers, central nuclei, and progressive replacement by adipose and connective tissue in advanced stages.

Laboratory studies play a supportive role in diagnosis. Serum CPK levels are markedly elevated, often 50–300 times above normal in early disease, reflecting muscle breakdown. Other enzymes such as transaminases, lactate dehydrogenase, and aldolase may also be elevated, although they are less specific and can be influenced by liver function. Importantly, CPK levels tend to decline as muscle mass decreases in later stages of the disease.

Imaging modalities such as ultrasonography are increasingly used as non-invasive tools to assess muscle involvement. Affected muscles show increased echogenicity due to fatty infiltration and fibrosis, making ultrasound useful for both diagnosis and disease monitoring. Electromyography may demonstrate myopathic changes, such as short-duration, low-amplitude motor unit potentials; however, it is not specific for DMD and is less commonly used with the availability of genetic testing.

Cardiac and respiratory assessments are also essential components of the diagnostic workup. Electrocardiography may reveal abnormalities such as right ventricular strain, tall R waves, deep Q waves, and T-wave inversions, indicating early cardiac involvement. Pulmonary function tests help evaluate respiratory muscle strength and detect restrictive lung disease. These assessments are particularly important for preoperative evaluation and long-term management. [6-7]

DIFFERENTIAL DIAGNOSIS:

The differential diagnosis of Duchenne muscular dystrophy (DMD) includes several neuromuscular disorders that present with progressive muscle weakness, particularly of proximal muscles. The most important condition to distinguish is Becker muscular dystrophy, which has a later onset, slower progression, and partial dystrophin production. An intermediate form may also occur with features between DMD and Becker muscular dystrophy. Myotonic dystrophy differs by its autosomal dominant inheritance and predominant involvement of distal muscles, with myotonia. Limb-girdle muscular dystrophy closely resembles DMD clinically but affects both sexes and shows variable progression. Congenital muscular dystrophy presents at birth or infancy and may be associated with brain abnormalities. Other conditions include Emery-Dreifuss muscular dystrophy, characterized by early contractures and cardiac involvement, and Spinal muscular atrophy, a neurogenic disorder with muscle wasting but without primary muscle pathology. Metabolic myopathies such as Pompe disease may also mimic DMD. [8]

TREATMENT / MANAGEMENT:

Duchenne muscular dystrophy has no definitive cure, and management is primarily supportive, aimed at slowing disease progression, preventing complications, and improving quality of life. A multidisciplinary approach involving neurologists, cardiologists, pulmonologists, physiotherapists, and genetic counselors is essential for optimal care. [9-11]

Glucocorticoid therapy remains the cornerstone of treatment. Corticosteroids such as prednisone and deflazacort are widely used to improve muscle strength and function, delay disease progression, and prolong ambulation. Prednisone is typically administered at a dose of 0.75 mg/kg/day or intermittently, while deflazacort is often preferred due to a more favorable side effect profile and is given at approximately 0.9 mg/kg/day. These agents help reduce muscle inflammation and necrosis, improve pulmonary function, delay scoliosis, and reduce the risk of cardiomyopathy, although long-term use is associated with side effects such as weight gain, osteoporosis, and growth suppression.

Cardiac management is critical, as cardiomyopathy is a major cause of morbidity and mortality. Early use of angiotensin-converting enzyme (ACE) inhibitors and beta-blockers is recommended to delay the onset and progression of cardiac dysfunction. In advanced cases, heart failure is managed with standard therapies including diuretics and digoxin. Regular cardiac surveillance with electrocardiography and echocardiography is essential for early detection and monitoring.

Respiratory care plays a vital role in management. Pulmonary function should be regularly assessed, especially after loss of ambulation. As respiratory muscles weaken, assisted ventilation, cough support techniques, and airway clearance methods become necessary. Early intervention helps prevent complications such as pneumonia and respiratory failure.

Orthopaedic management focuses on maintaining mobility and preventing deformities. Physiotherapy is essential to preserve muscle strength, flexibility, and joint range of motion. Stretching exercises, orthotic devices such as ankle-foot orthoses, and mobility aids like braces and wheelchairs are commonly used. Surgical interventions may be required to correct contractures or scoliosis, particularly in advanced stages, to improve posture and respiratory function.

Nutritional management plays a vital role, as patients are at risk of both obesity (due to reduced mobility and steroid use) and malnutrition in later stages. A balanced diet with appropriate caloric intake is essential. Supplementation with calcium and vitamin D is recommended to prevent osteoporosis, particularly in patients on long-term corticosteroids. Regular monitoring of bone health using DEXA scans is advised, along with early management of fractures if they occur.

Recent therapeutic advances have introduced mutation-specific and disease-modifying treatments. Exon-skipping therapies such as eteplirsen, golodirsen, viltolarsen, and casimersen are designed to restore partial dystrophin production in patients with specific genetic mutations. Ataluren is used in patients with nonsense mutations to enable read-through of premature stop codons. Givinostat, a histone deacetylase inhibitor, reduces inflammation and muscle degeneration and represents a non-steroidal therapeutic option. Gene therapy, such as delandistrogene moxeparvovec, offers a promising strategy by delivering a functional version of the dystrophin gene, potentially addressing the root cause of the disease.

Psychosocial care is also an important aspect of management. Patients and families often face significant emotional, social, and financial challenges. Psychological support, educational interventions, and social services help improve coping, enhance quality of life, and ensure better integration into society.

Exercise is encouraged but should be carefully supervised. Low-impact activities such as swimming and gentle recreational exercises help maintain muscle strength and prevent disuse atrophy. However, excessive or high-resistance exercise should be avoided, as it may lead to muscle damage. Activity levels should be adjusted based on symptoms such as muscle pain or myoglobinuria.

PROGNOSIS:

Duchenne muscular dystrophy remains an incurable, progressive disorder despite advances in gene therapy and molecular medicine. The prognosis is generally poor, with patients experiencing gradual loss of muscle function leading to wheelchair dependence by approximately 10–12 years of age. As the disease progresses, involvement of respiratory and cardiac muscles results in life-threatening complications. Most patients die in their teens or twenties, commonly due to respiratory failure or cardiomyopathy, while other causes of death include pneumonia, aspiration, and airway obstruction. However, improvements in multidisciplinary care have significantly extended survival. Current estimates suggest a life expectancy of around 25–30 years, with many patients living into their 30s with optimal management. Interventions such as ventilatory support, airway clearance techniques, and early cardiac treatment have played a major role in improving outcomes. In some cases, especially with advanced respiratory assistance and comprehensive long-term care, survival may extend into the fourth decade or beyond. Although rare, prolonged survival into the forties or even fifties has been reported with intensive supportive care, including mechanical ventilation, appropriate positioning, and cardiac management. [12]

CONCLUSION:

Duchenne muscular dystrophy is a progressive, life-limiting neuromuscular disorder with no definitive cure, and its management relies on early diagnosis, multidisciplinary care, and emerging therapies to improve quality of life and survival.

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