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Case Report

# A Case Report on Dermatomyositis Highlighting Muscle Weakness and Rash

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**ABSTRACT**

The disorder is known as Dermatomyositis, a rare autoimmune condition characterized by inflammation of the muscles and specific types of skin rashes. The case report presents a 33-year-old woman with hypothyroidism that developed progressive muscular weakness, mobility problems and new rashes around her eyes. Her major symptoms included difficulty in walking without assistance and general body weakness. Physical examination at admission showed significant truncal weakness as well as reduced reflexes. Lab tests indicated increased TSH levels, CKMM, as well as low vitamin D3 levels; muscle biopsy confirmed dermatomyositis. This was further proved by other examinations including MRI and nerve conduction studies. Systemic glucocorticoids, methotrexate, Intravenous Immunoglobulin (Ivlg) and supportive therapies were given to the patient leading to remarkable improvement in muscle power and the disappearance of lesions on her skin. This particular case stands out because it involves both hypothyroidism and dermatomyositis concurrently indicating that some overlapping autoimmune diseases might be present in this patient population. This underscores the importance of an integrated diagnostic approach involving biopsy of muscles, enzyme assays and imaging for accurate diagnosis and management of dermatomyositis. The treatment plan for refractory cases that consisted of IVIG demonstrated positive results thus implying individualized interventions are effective. Therefore, this case illustrates the significance of a multidisciplinary approach to managing dermatomyositis, especially in patients with other autoimmune diseases and underlines the need for close observation, personalized therapy schedules and supportive measures to optimize results.

**Keywords:** Case report, Dermatomyositis, Muscle weakness, Antinuclear antibody Immunosuppressants.

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## INTRODUCTION

Dermatomyositis is an uncommon acquired autoimmune disorder involving muscle inflammation and skin rashes. This idiopathic inflammatory myopathy is characterized by its scaly alopecia, photo-distributed erythema or poikiloderma, heliotrope rash, periungual telangiectasia and Gottron's papules, among other prominent cutaneous signs. Besides affecting the skin and muscles most commonly, dermatomyositis is a systemic condition that can result in joint pain, joint inflammation, esophageal disorders and heart failure in those affected.[1]

The specific etiology of dermatomyositis is yet unclear, although it is thought to result from hereditary, immunologic and environmental causes. In Dermatomyositis, there is likely an inappropriate activation of complement proteins targeting the Perimysium of the skeletal muscle tissue. It is thought that there are antibodies that accidentally target the perimysium or there are dermatomyositis-specific antibodies that one has that target the perimysium and blood vessels. These antibodies activate the complement system of specialized proteins that promote the inflammatory response. This results in blood vessel inflammation causing ischemia or infraction to muscle tissues. This leads to muscle inflammation and muscle atrophy.[2]

Muscle enzyme tests, such as those using Creatine Phosphokinase (CPK), aldolase, LDH, AST and ALT, might be used to diagnose it. Most individuals with dermatomyositis have Antinuclear Antibodies (ANA), yet these do not aid in the diagnosis. Electromyography aids in determining which muscle groups are most impacted and offers direction on which muscles should be biopsied. Chest radiography, Magnetic Resonance Imaging (MRI) and a muscle biopsy can also be done.[3]

Dermatomyositis patients require systemic glucocorticoids, with or without immunosuppressants, as their first-line therapy for muscle illness. Prednisone is administered daily at a dose of

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0.5-1.5 mg/kg until blood creatine phosphokinase levels return to normal, then gradually weaned down over the next year. An alternative treatment plan involves split doses of 40-60 mg of prednisone daily until the CPK level returns to normal, then a single daily dosage, which can be decreased by one-fourth every three to four weeks, until reaching a daily maintenance dose of 5-10 mg. Methotrexate is the primary treatment for individuals with resistant dermatomyositis, starting with an oral dosage of 7.5-10 mg/week and increasing to 25 mg/week. Children can receive 1 mg/kg. Intravenous immunoglobulins have multiple roles in dermatomyositis, including cytokine inhibition, competition with autoantibodies, complement deposition inhibition and interference with antigen recognition by sensitized T-cells. Intravenous immunoglobulin-treated patients showed statistically marked improvement in muscle strength and active skin lesions after receiving monthly cycles. Furthermore, it is also used in patients with refractory dermatomyositis who don't tolerate or are contraindicated to immunosuppressant therapy. Non-pharmacological therapy includes sun avoidance and sunscreen usage.[4,5]

## CASE REPORT

A female patient, age 33, with a history of hypothyroidism presented to a multispecialty hospital with chief complaints of progressive weakness and difficulty in mobility. Her principal complaints included inability to walk unaided and general weakness from the last 4 months, difficulty in standing from a squatting position since 3 months, her inability to sit since the past 15 days and 7 days before admission, she developed dry rashes around her eyes. She was on Tab. Thyroxine 50 mcg daily. The patient did not have any significant family or social history. On admission, vital signs were stable: afebrile, BP 110/70 mmHg, oxygen saturation 96% on room air, heart rate 76 bpm and respiration rate 20 breaths/min. Physical examination showed the tone is lowered in the lower limbs and normal in the upper limbs for motor functions. Power assessment shows apparent truncal weakness with no neck folding. The reflexes for the biceps, triceps and knee are +1 and the plantar reflex is reduced. Laboratory investigations revealed TSH of 12.8 µIU/mL, CKMM 45 IU/L and vitamin D3 at 15.93 ng/mL. Specific diagnostic tests, including a muscle biopsy and Antinuclear Antibody (ANA) test, were conducted. The muscle biopsy showed relatively preserved fascicular architecture with adipocytic infiltration, mild variation in fiber size, atrophic angulated fibers, myonuclear clumps, regenerating fibers, multifocal endomysial infiltrates of lymphocytes, focal myophagocytosis and myonecrosis. No distinct perifascicular atrophy or vacuoles were observed. Nerve biopsy indicated moderate to severe nonuniform loss of myelinated nerve fibers with occasional regenerating clusters, without acute axonal breakdown. Immunohistochemistry revealed a positive ANA (1:100) with fine speckled and cytoplasm granular patterns and the presence of antibodies against Ro-52. MRI of the lumbar spine indicated mild bulges at L4-L5 and L5-S1 with compression on thecal sac and nerve roots, extensive abnormal signals in paraspinal and pelvic muscles indicating myositis and mild inflammatory edema in subcutaneous fat of lower spine and pelvic regions. Nerve conduction studies showed evidence of distal and proximal demyelination.

The patient was hospitalized for five days during which she received the following therapy - Tab. Prednisolone (40 mg)-8 tablets of 5 mg each were administered with milk before breakfast, Tab. Methotrexate was given weekly 15 mg, Tab. Amoxicillin+Clavulanic Acid 625 mg BD, Tab. Thyroxin 50 mcg OD before breakfast, Tab. Folic acid 5 mg OD, Tab. Calcium+Vitamin D3 500 mg BD, Vitamin D3 sachet 60000 IU once weekly, Inj. IvIg 20g 24 hr, Tab. Pantoprazole 40 mg OD and Inj. Optineuron 2A 12 hr was given. The administration of prednisone and methotrexate aims to reduce inflammation and modulate the immune response, while IvIg is particularly useful in refractory cases and for skin manifestations and the inclusion of supportive therapies such as calcium and vitamin D3 supplementation treats the patient's deficiency and is crucial for overall bone health and muscle function. The administration of folic acid helps mitigate the side effects of methotrexate and pantoprazole is used to prevent gastrointestinal complications. The antibiotic therapy (amoxicillin+clavulanic acid) was likely prescribed to address any potential or existing infections, ensuring the patient's comprehensive care.

## DISCUSSION

Dermatomyositis is an idiopathic inflammatory myopathy that generally manifests as cutaneous symptoms. The progression of proximal symmetrical weakening is a hallmark of this uncommonly acquired immune-mediated muscle disease. The only perceived difference between polymyositis and dermatomyositis was the existence of a cutaneous disease. It affects adults and children equally, with a higher incidence rate in women than in men. Every year, there are 6-7 cases per 100,000 individuals. Although the precise etiology of the condition is yet unclear, environmental, immunological and genetic variables contribute to its development. Symptoms may include myalgias, weariness, weakness and muscle soreness. The pharynx or upper esophagus may be involved in striated muscle involvement and dysphagia or dysphonia may indicate a fast-moving course and potentially a dismal outcome.[6]

Treatment with high-dose prednisolone is continued until remission of both biochemical and clinical activity. Intravenous immunoglobulin is also utilized to decrease skin manifestations and improve muscle strength. Steroid-sparing agents, most commonly immunosuppressive agents and/or methotrexate are potentially useful for early intervention in the induction and maintenance of remission. Each patient will achieve some degree of response with improvements in the strength of the response,

status of muscle enzyme, or decrease in ranking in corticosteroid dose as it is given in 50-75% of the patients.[7]

## CONCLUSION

The case discussion features a female patient of age 33, who had been diagnosed with Dermatomyositis, an unusual ailment. To investigate this case, muscle biopsy, muscle enzyme concentration, antinuclear antibody and nerve conduction were performed and provided an influential clue for the condition. This case underscores the importance of a multidisciplinary approach in managing dermatomyositis, particularly in patients with concurrent conditions such as hypothyroidism. The combination of pharmacological and non-pharmacological treatments, along with vigilant monitoring and supportive care, is essential for optimizing patient outcomes and managing the systemic effects of the disease.

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## CONFLICT OF INTEREST

The authors affirm that they have no competing interests.

## ABBREVIATIONS

**MRI:** Magnetic Resonance Imaging; **MAC:** Membrane Attack Complex; **CK MM:** Creatinine Kinase-Muscle; **TSH:** Thyroid Stimulating Hormone; **T3:** Triiodothyronine; **T4:** Tetraiodothyronine; **LL:** Lower Limb; **UL:** Upper Limb**; ANA:** Antinuclear antibody; **NCV:** Nerve Conduction Velocity; **CPK:** Creatine phosphokinase; **LDH:** Lactate dehydrogenase; **AST:** Aspartate aminotransferase; **ALT:** Alanine aminotransferase.

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