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An Overview of Dermatomyositis

Srishti Deshpande[1*]; Neha Rangwar[2]; Aashutosh Sinwal[3]; Ishu[4];
Pooja Brahambhatt[5]; Aman Saini[6]
[1, 2]Malla Reddy College of Pharmacy, Maisammaguda, Hyderabad, Telangana, India- 500100
[3,4,5,6]Pharm D, School of Pharmaceutical Sciences, Jaipur National University, Jaipur, Rajasthan, India,302017

Corresponding Author: Srishti Deshpande[*]
Malla Reddy College of Pharmacy, Maisammaguda,
Hyderabad, Telangana, India- 500100

Abstract:- Dermatomyositis (DM) is an idiopathic inflammatory myopathy condition. It might be challenging to identify DM when the usual dermatologic symptoms or myopathy are not present. The onset of muscle involvement is often pain or myalgias, however, it can also be accompanied by proximal muscle weakening. The incidence rate was estimated to be 9.63 per 1,000,000 inhabitants in Olmsted County, Minnesota, according to a retrospective research that ran from 1967 to 2007. The exact reason(s) behind dermatomyositis are still a mystery. Environmental, immunological, and genetic variables may all have an impact, though. Diabetic myopathy (DM) is characterized by a progressive weakening of muscles, which might start mild and develop over a few weeks or months, or it can advance more rapidly. Typically, symmetric and proximal muscle involvement is the first to manifest, with distal muscle weakening developing later in the disease's progression. Muscle weakness, skin disease, and other underlying problems are the main focuses of dermatomyositis management. Systemic glucocorticoids, with or without immunosuppressants, are the initial line of defense against dermatomyositis-related muscle illness. Management relies heavily on physical therapy and rehabilitation. Active exercise programs should be advocated for patients with moderate illness.

Keywords:- *Dermatomyositis, Interstitial Lung Disease, Diabetes Mellitus, Immunosuppressants, Electromyography.*

I. INTRODUCTION

Distinct skin lesions and a clinically diverse constellation of systemic symptoms characterize dermatomyositis (DM), an idiopathic inflammatory myopathy (IIM). It might be challenging to identify DM when the usual dermatologic symptoms or myopathy are not present. To add insult to injury, "overlap" syndromes have been used to explain clinical variability in DM diagnoses, further complicating matters.[1] The onset of muscle involvement is often pain or myalgias, however, it can also be accompanied by proximal muscle weakening. There has been a description of an amyopathic variation that causes very little inflammation in the muscles. The link between DM and an elevated risk of internal cancer has long been recognized. The prognosis of dermatomyositis can be affected by the presence of an underlying cancer in a large percentage of individuals. Other forms of the disease do exist, albeit most patients present with skin and muscle symptoms. Interstitial lung disease (ILD) is another significant clinical characteristic of diabetes mellitus (DM). A seminal work by Bohan and Peter4 proposed criteria for the diagnosis and categorization of polymyositis (PM) and diabetic macular degeneration (DM) in 1975.[2] Progressive proximal symmetrical weakness, increased muscle enzymes, an abnormal electromyogram, and an abnormal muscle biopsy were the four muscle-related criteria, while the presence of concomitant cutaneous illness constituted the fifth. People used to think skin illness was the sole defining feature that set DM apart from PM. There has been significant debate in recent years regarding the exact mechanisms by which myopathy develops in diabetes mellitus (DM) and preeclampsia (PM). significant research has pointed to vascular inflammation as the pathogenetic basis for DM, while other research has focused on cytokines and found similarities between the two diseases. A resurgence of interest in the pathogenetic mechanisms of myopathy has been prompted by new research that has shown abnormalities in nitric oxide levels, tumor necrosis factor (TNF) receptors in the blood, soluble CD40 expression, and the expression of interleukin 1a and major histocompatibility complex class I in muscle. Little is known about the mechanisms that cause the cutaneous illness. These illnesses have an unknown origin.[1,2] It appears that DM and PM are associated with immunogenetic markers, and individuals with both conditions commonly have TNF-a polymorphisms. No one knows for sure what causes type 2 diabetes, pre-diabetes, or juvenile diabetes to start; however, some subgroups may have a seasonal start, and in children, the illness may develop after an infection. It follows that infections in people with an immunogenetic susceptibility to illness may have a role in the development of DM, PM, and/or juvenile DM as a result of environmental variables interacting with one another. [3]

- *Epidemiology*

Rare cases of dermatomyositis do occur. The incidence rate was estimated to be 9.63 per 1,000,000 inhabitants in Olmsted County, Minnesota, according to a retrospective research that ran from 1967 to 2007. The amyopathic

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subtype accounted for 21% of all cases, according to the same research. The average age of diagnosis for Dermatomyositis is 44.0 ± 18.3 years, and it usually strikes people in their 40s and 50s. With incidence rates of 3.98 and 4.68 per 1,000,000, respectively, the disorder is more frequent in women than in males.[4]

Dermatomycosis is more common in Southern Europe than in Northern Europe across Europe. Researchers in Quebec found that dermatomyositis was more common in city dwellers. Additionally, a cohort study in Pennsylvania found that areas with high levels of airborne pollution had clusters of clinically amyopathic dermatomyositis (CADM). According to their research, environmental variables may have a role in bringing on the illness.[5]

- *Etiology*

The exact reason(s) behind dermatomyositis are still a mystery. Environmental, immunological, and genetic variables may all have an impact, though. One result is that people with the condition had a higher frequency of specific genetically determined HLAs, also called "human leukocyte antigens," which may indicate underlying genetic and immunological processes. Important proteins in the immune system, HLAs also seem to affect a person's susceptibility to particular illnesses and the success of transplants. Both adults and children with dermatomyositis seem to have a higher frequency of particular HLAs, according to the available evidence. Nevertheless, the precise consequences of these discoveries remain unclear. [6] It is believed that dermatologtomyositis is one of several autoimmune illnesses, which occur when the body's immunological defenses mistakenly target healthy tissues. Dermatomyositis is characterized by an aberrant immune response that causes obstructive inflammatory changes in blood vessels within muscles and other tissues. Other symptoms may include patchy degeneration, atrophy, and regeneration of muscle fibers, as well as thinning of the epidermis and other skin layers.[7]

There have been reports that point to specific infectious organisms as possible causes of dermatomyositis. These agents include coxsackie virus, parvovirus, echovirus, HIV, human T-cell lymphotropic virus Type 1, Toxoplasma, and Borrelia species. Those who are genetically prone to dermatomyositis may develop the inflammatory changes and muscle tissue damage that are observed when antibodies are produced in response to foreign viral proteins (antigens) that are similar to some of the body's proteins. These antibodies may mistakenly "cross-react" or attack the body's cells, such as intramuscular blood vessels. There is evidence that environmental variables contribute to the onset or worsening of juvenile dermatomyositis in April and May. Furthermore, certain adult cases of dermatomyositis have been linked to malignancies, which raises the possibility of an aberrant autoimmune reaction targeting a shared antigen between the tumor and the muscle. Some cases of dermatomyositis have also been linked to specific vaccines or pharmaceuticals, such as quinidine, phenylbutazone, penicillamine, and statins. Injections of collagen or silicone into the breasts can cause or worsen dermatomyositis. However, further evidence is needed to confirm the link, and the consequences of these discoveries are yet unclear. [8,9]

II. CLINICAL MANIFESTATIONS

Diabetic myopathy (DM) is characterized by a progressive weakening of muscles, which might start mild and develop over a few weeks or months, or it can advance more rapidly. Typically, symmetric and proximal muscle involvement is the first to manifest, with distal muscle weakening developing later in the disease's progression. Common challenges for patients include getting out of a chair, navigating stairs, moving heavy things, and even shampooing their hair. When distal muscles are involved, it might be difficult to grip and manipulate items.[8] Muscle weakness might come on before, just after, or simultaneously with the DM rash. A defining indication of DM is Gottron's papules. Plaques and papules ranging from redness to purpleness cover the extensor surfaces of the metacarpophalangeal and interphalangeal joints, making up the majority of the lesions.[10] Active lesions often go away with depigmentation, atrophy, and scarring; these lesions may come with scales and occasionally form ulcerations. Less definitive evidence of DM can be found using Gottron's sign, which is characterized by erythematous macules and patches covering the knees and/or elbows. Diabetic reticulopathy can manifest as ulceration, telangiectasia, or livedo reticularis. Rough and cracked hyperkeratotic "dirty" lines across the palmer and lateral portions of the fingers are characteristic of this hand disease, which gives the impression of "mechanics" hands. Additionally, you could notice an abnormality on the nail plate or cuticular overgrowth. In diabetic individuals, vascular instability may manifest as palmar and plantar erythema, which may or may not be speckled. On top of that, the palmar hands' joint wrinkles might be accompanied by elevated, sensitive papules or plaques. These lesions show dermal mucin accumulation on biopsy.[7,11]

- *Pathophysiology*

A humoral-mediated assault on the endothelium of arterioles and muscle capillaries is believed to be the cause of dermatologomyositis. Initiation occurs when completer factor-3 (C3) is activated, leading to the formation of C3b and C4b. After that, the C5b-C9 membrane attack complex (MAC) and the neoantigen C3bNEO are formed. Inflammation is caused by the membrane's attack on complicated deposits on vascular walls. Muscle fibers, especially those located at the periphery and thus far from the vascular supply, atrophy as a result of hypoxic damage. Necrosis and degeneration of muscle fibers begin when capillary density decreases with time. [4,12]

- *Diagnosis*

In 1975, Bohan and Peter established the most widely used criteria for the diagnosis and categorization of DM and PM. In typical type 2 diabetes, patients often have aberrant levels of muscle enzymes such as creatine kinase (CK), aldolase, aspartate aminotransferase (AST), alanine aminotransferase (ALT), and lactate dehydrogenase (LDH). Additionally, patients may also experience proximal muscle

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weakness and dermatological symptoms. Early on in the course of the disease, electromyogram (EMG) examinations reveal abnormalities in 70–90% of patients; however, these studies are not disease-specific and can reveal other muscle disorders as well. [13] Positive sharp waves, complex repetitive discharges, early recruitment, tiny polyphasic motor till potentials, enhanced spontaneous and insertional activity with fibrillation potentials, and other typical EMG findings are listed below. If you suspect myositis and see any swelling in your muscles, a sensitive imaging method is magnetic resonance imaging (MRI). When fat is suppressed or short tau inversion recovery sequences are used, T2-weighted images become sharper, revealing hyperintense areas of inflammation. Loss of muscle mass is a hallmark of advanced illness. To confirm a diagnosis and eliminate other possible diseases, biopsies of the affected area (muscles and/or skin) are necessary. Muscles that are somewhat weak on physical examination, MRI-identified inflammatory regions, or muscles on the opposite side of abnormalities on electromyography (EMG) should be used for the muscle biopsy. [14]

III. COMPLICATIONS

- *Respiratory Disease*

Hypoventilation, aspiration pneumonia, and interstitial lung disease are three forms of pulmonary involvement. Having antibodies against histidine transfer ribonucleic acid synthetase is significantly linked to interstitial lung disease (ILD), which affects around one-third of dermatomyositis patients. When the muscles that control breathing become weak, a potentially fatal condition known as aspiration pneumonia can set in. [9,10]

- *Malignancy*

Malignancies occur in 24% of instances in patients with dermatomyositis, putting them at a higher risk. In a population-based investigation involving patients from Finland, Sweden, and Denmark, the standardized incidence ratio was 3.0. Factors that increase the likelihood of cancer include advanced age, lack of interstitial lung disease, extensive skin involvement (also known as the shawl sign), antibodies against 155/140 or NXP2, antibodies against myositis, treatment resistance, and a history of cancer with recurrence. Ovarian, lung, pancreatic, stomach, and colon adenocarcinomas, together with non-Hodgkin lymphoma, constituted the bulk of the cancer cases. During the first year of the condition, the risk of malignancy is greatest and stays high for up to five years. A higher-than-average risk of cancer persisted even after this time had passed. [15]

- *Heart Disease*

Cardiac involvement in dermatomyositis is typically not noticeable to the naked eye. Arrhythmias and irregularities in conduction can be revealed by electrocardiography (ECG). Myocarditis, congestive heart failure, and coronary artery disease are the three main forms of cardiac involvement. [16]

- *Esophageal Disease*

Weakness in the muscles that contract the oropharynx and esophagus can cause dysphagia in patients. Malnutrition and an increased risk of aspiration are possible outcomes of these diseases. [4,11] Calcinosis, muscular atrophy, and contractures are further potential consequences. [2]

IV. TREATMENT

Muscle weakness, skin disease, and other underlying problems are the main focuses of dermatomyositis management.

Systemic glucocorticoids, with or without immunosuppressants, are the initial line of defense against dermatomyositis-related muscle illness. The fundamentals of treatment for dermatomyositis remain the same, even if there is no established systemic steroid regimen. In the beginning, substantial dosages of prednisolone are administered for the first several months until the levels of muscle enzymes decrease and strength improves. It takes around six weeks for muscle enzymes to normalize, therefore patients should be monitored often to make sure they're responding well during this period. [17,6] Additionally, the improvement of muscular weakness might take up to three months. Systemic steroid dosing is progressively reduced if a satisfactory response has occurred. Systemic steroid treatment often lasts for nine to twelve months in total. You should know that glucocorticoid myopathy can develop if you provide high-dose glucocorticoids for longer than six weeks. [18]

If the patient does not improve after starting systemic steroids, the doctor should look for alternative reasons for myopathy. Hypothyroidism, glucocorticoid myopathy, inclusion body myositis, and underlying cancer are among them. It is possible to add steroid-sparing immunosuppressants after other possible diagnoses have been exhausted.

Long-term systemic steroids can have harmful consequences, however, immunosuppressants can help reduce those effects. Osteoporosis, heightened infection risk, cushingoid symptoms, and secondary diabetes are all potential side effects. In situations of severe myopathy, chronic medical problems (such as diabetes), and significant extra-muscular consequences (such as interstitial lung disease or esophageal dysfunction), immunosuppressant medication might be started in addition to steroids.[19] Methotrexate and azathioprine are first-line agents. Numerous parameters, such as dosage frequency, systemic involvement, side effects, and alcohol usage, influence the selection of an immunosuppressant. Patients who cannot refrain from alcohol, have interstitial lung disease, or have liver involvement should preferably be administered an immuno-suppressant such as azathioprine. The advantage of methotrexate is that it only has to be taken once every seven days.

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A patient is said to be resistant if they do not react well to steroid and azathioprine or methotrexate treatments.[20] Intravenous immunoglobulin (IVIG), cyclophosphamide, calcineurin inhibitors, rituximab, mycophenolate mofetil, and other similar treatments are available for resistant instances. If the patient is resistant to other treatments, the first line of defense should be rituximab, an anti-CD 20 drug. The second line of defense, in the event of a failure, might consist of intravenous immunoglobulin or a mixture of azathioprine and methotrexate. In circumstances when other treatments have failed, such as when interstitial lung disease is present, tacrolimus plus mycophenolate mofetil might be helpful. When interstitial lung disease is progressing quickly, cyclophosphamide is the drug of choice.[21]

The potential side effects of immunosuppressants must be closely monitored in patients. Stomatitis, hepatotoxicity, and leucopenia are some of the side effects that patients taking methotrexate should keep an eye out for. These side effects can be lessened by co-treating with folic acid or leucovorin. It may be necessary to discontinue therapy with azathioprine if it causes a flu-like response. Additionally, it might lead to pancreatitis and myelosuppression. Unless all other treatment options have been exhausted, cyclophosphamide should not be administered because of the increased risk of cancer.

Management of skin disease in dermatomyositis involves a combination of medical treatment, physiotherapy, and general treatments. Sun protection measures, such as staying out of the sun as much as possible, wearing clothes that block the sun's rays, and using sunscreen with an SPF of 30 or greater, should be a part of these efforts. Local treatments (such as pramoxine, menthol, or camphor) or oral medications (such as sedating antihistamines, amitriptyline, or gabapentin) can be used to alleviate the debilitating pruritus that can be caused by skin diseases. Topical agents and systemic drugs are also part of the medical treatment for skin diseases. [22,23] Corticosteroids and calcineurin inhibitors are examples of topical agents. Systemic medication is necessary for the management of skin disease in the majority of patients. Systemic medications such as methotrexate and hydroxychloroquine are the gold standard for treating skin diseases. Systemic glucocorticoids are useful for managing musculoskeletal disorders, but they have little effect on cutaneous disorders. [25]

While the discomfort of calcinosis complications is real, it may be lessened with prompt diagnosis and treatment. Calcinosis has shown improvement in therapy with the use of calcium channel blockers, particularly non-dihydropyridines like diltiazem. Surgical removal of tender lesions caused by calcinosis is an option, particularly in cases where the lesion is localized. The combination of surgery and medication (diltiazem) successfully reduced dystrophic calcinosis, according to a retrospective investigation of small patient groups. Diltiazem and other calcium channel blockers help alleviate calcinosis, a complication that is more common in children with dermatomyositis. Calcinatory nodules may necessitate surgical excision in certain instances. [24]

Management relies heavily on physical therapy and rehabilitation. Active exercise programs should be advocated for patients with moderate illness. One way to avoid contractures is to practice range of motion exercises. Speech therapy consultations and aspiration prevention strategies may be necessary for patients with esophageal dysfunction. Some ways to prevent aspiration include lifting the head off the bed, making feeds thicker, and, in some cases, feeding through gastric tubes. [26]

To avoid osteoporosis, individuals using long-term systemic corticosteroids may benefit from anti-resorptive medication. Preventative treatment against Pneumocystis jirovecii with trimethoprim and sulfamethoxazole should be explored for patients on high-dose systemic glucocorticoids or immunosuppressants. Finally, before using immunosuppressants, all patients should have the right vaccines.[27]

V. CONCLUSION

Dermatomyositis (DM) is characterized by distinct skin lesions and systemic symptoms, with muscle involvement being a key feature. The disease is associated with an elevated risk of internal cancer and is believed to have autoimmune origins, with genetic, immunological, and environmental factors playing a role in its development. The main focus of dermatomyositis management is to address muscle weakness, skin disease, and other underlying problems. Treatment typically involves systemic glucocorticoids as the initial line of defense, with or without immunosuppressants, and monitoring the patient's response closely. Long-term systemic steroids can have harmful consequences, so immunosuppressants may be added to reduce side effects. If a patient does not improve with systemic steroids, alternative reasons for myopathy should be explored. Physical therapy and rehabilitation are important components of management, along with preventative measures for complications such as osteoporosis and Pneumocystis jirovecii infection.

- *Conflict of Interest*

Nil

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