Proceedings of ICEGEE 2026 Symposium: AI-Based Medicine and Biological Data Analysis
DOI: 10.54254/2753-8818/2026.AU32314

Dermatomyositis: Evaluating the Efficacy of Corticosteroids, Immunosuppressive Agents, and Intravenous Immunoglobulin (IVIG)

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Abstract. Dermatomyositis (DM) is an autoimmune disease characterized by heterogeneous cutaneous, muscular, and systemic manifestations associated with inflammation induced by the immune system attacking muscle tissues or body cells, with treatment efficacy varying by subtype and patient-specific factors. This research report evaluates three major therapeutic modalities for DM - corticosteroids, immunosuppressive agents, and intravenous immunoglobulin (IVIG) - to provide evidence-based guidance for patients and clinicians in treatment selection. A systematic analysis of each treatment's efficacy across DM subtypes is included among varied aspects, cost, accessibility, and safety profile. Results indicate that corticosteroids serve as first-line therapy for muscular inflammation but fail to resolve refractory cutaneous lesions or benefit clinically amyopathic dermatomyositis (CADM). Immunosuppressive agents act as steroid-sparing adjuvants, with subtype-specific efficacy. IVIG is a third-line option for refractory or high-risk cases, improving dual cutaneous/muscular symptoms in patients but with high cost and limited accessibility. Cost-wise, corticosteroids are the most affordable, while IVIG requires the highest expense. Safety profiles range from corticosteroids' cumulative toxicity to IVIG's mild infusion-related reactions. This evaluation emphasizes personalized treatment matching - aligning therapy with DM subtype, severity, comorbidities, and patient priorities - to optimize outcomes and reduce disease burden.

Keywords: Dermatomyositis, Corticosteroids, Immunosuppressive agents, Intravenous Immunoglobulin

1. Introduction

1.1. Introduction to Dermatomyositis (DM)

Dermatomyositis (DM) is an autoimmune disease that is rarely seen and is featured by both cutaneous findings and muscle involvement in some subtypes. As an idiopathic autoinflammatory disease, DM occurs more often among adults, with a female predominance and an annual incidence of 1-2 per 100,000 persons globally [1]. The presence of varied Myositis-specific autoantibodies (MSAs) triggers more specific symptoms and manifestations, such as malignancy and interstitial

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DOI: 10.54254/2753-8818/2026.AU32314

lung disease (ILD) - which only present in 5 - 35% of patients with DM – thereby bringing a higher risk of death [1,2].

1.2. Subtypes of dermatomyositis and autoantibody correlations

The clinical presentation of DM can be divided into two main subtypes, including Classic Dermatomyositis (CDM) and Clinically Amyopathic Dermatomyositis (CADM). CDM is the classic form of DM, where patients are suffering from both skin manifestations and muscle involvement. Whereas CADM is a subtype of DM where the primary feature is severe, dominant skin involvement, but there will not be visible or clinical muscle weakness [2].

Table 1. Clinical subtypes of dermatomyositis

Subtypes	Classic Dermatomyositis (CDM)	Clinically Amyopathic Dermatomyositis (CADM)	References
Key Features	Gottron's papules, heliotrope rash, V-sign.	Prominent, often severe, skin lesions (e.g., ulcers).	[1,2]
MuscleInvolvement	Proximal muscle weakness, elevatedcreatine kinase.	No clinical weakness. Subclinical inflammation may be detectedon MRI or labs.	[1,2]
Systemic Risks	Interstitial lung disease (ILD), dysphagia,malignancy.	High risk of rapidly progressive ILD, especially with antiMDA5 antibodies.	[1,2]

The disease phenotypes and associated systemic risks are strongly influenced by the types of myositis-specific autoantibodies (MSAs) present in each patient, as summarized in Table 2.

Table 2. Key myositis-specific autoantibodies (MSAs) and clinical correlations

Autoantibody Type	Cutaneous Manifestations	Systemic & Muscular Manifestations	References
Anti-Mi-2	Classic rash	Mild myositis; lower risk of ILD/malignancy.	[1-3]
Anti-MDA5	Ulcerations, painful palmar papules	High risk of rapidly progressive ILD; often in CADM.	[1-3]
Anti-TIF1γ	Psoriasiform lesions, ovoid palatal patches	Significantly increased malignancy risk.	[1-3]
Anti-NXP2	Calcinosis cutis	Severe dysphagia, myalgias; increased malignancy risk.	[1-3]
Anti-ARS	Mechanic's hands	Anti-synthetase syndrome: ILD, myositis, arthritis.	[1-3]
Anti-SAE	Classic rash, often severe	Dysphagia; mild ILD and malignancy risk.	[1-3]

1.3. Pathogenesis of dermatomyositis

DM pathogenesis involves immune-mediated microvascular injury, interferon-driven inflammation, and autoantibody-associated tissue damage, resulting in perifascicular muscle atrophy and cutaneous lesions. These mechanisms provide the rationale for immunosuppressive and immunomodulatory therapies used in current clinical practice [1,2,4-6].

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2. Literature review

2.1. Corticosteroids in the treatment of dermatomyositis

Two primary types of corticosteroids are used in treating DM, systemic and topical corticosteroids. Systemic corticosteroids are typically administered orally or by injection to affect the entire body; they work by reducing inflammation and suppressing the immune system, which can help manage the symptoms of dermatomyositis. While topical corticosteroids are applied directly to the skin in the affected areas, where they may help reduce inflammation and itching and allow the skin to heal; topical corticosteroids could be used to treat small areas of skin rashes or when steroids are too intense [7].

2.1.1. Mechanism of action

Systemic corticosteroids exert and expose their anti-inflammatory and immunosuppressive effects by regulating related gene expression, specifically inhibiting pro-inflammatory cytokines (e.g., TNF-α, IL-1), reducing T-cell activation and neutrophil migration to inflamed tissues. In DM, this reaction interrupts the immune-mediated cascade, causing muscle fiber damage and capillary inflammation [5,6,8].

Topical corticosteroids act locally by penetrating the epidermis, suppressing local immune responses by decreasing MHC I expression on muscle fibers, thus reducing the immune system's recognition of muscle tissue as "foreign", and limiting T-cell-mediated inflammation [5,6].

2.1.2. Efficacy in muscular and cutaneous disease

For muscular involvements, systemic corticosteroids (e.g., prednisone) are the first-line treatment choices for inducing remission in muscle disease, with 60–80% of patients experiencing improved proximal muscle strength within 2-4 weeks [6,8].

For cutaneous involvement, corticosteroids' responses are more varied; systemic corticosteroids often fail to fully resolve or cure skin lesions (e.g., Gottron's papules, heliotrope rash) [4]. Topical corticosteroids are more effective for localized disease: superpotent class I steroids (e.g., clobetasol) under occlusion to enhance the efficacy of high-potency topical corticosteroids (e.g., clobetasol propionate) in thickened lesions like scalp plaques or lichenified plaques, while low-potency agents (e.g., hydrocortisone) are preferred for thin skin (face, flexures) to minimize atrophy- skin thinning around the thinner skin areas with thinner stratum corneum and higher vascularity [4,9]. Notably, cutaneous DM associated with anti-MDA5 antibodies is frequently refractory and resistant to both systemic and topical steroids [7].

2.1.3. Side effects

The systemic side effects are more likely to be acute adverse events that include hyperglycemia, insomnia, as well as mood disturbances, while chronic use (over 3 months) risks osteoporosis, cataracts, and adrenal suppression [5,8]. For steroid-induced myopathy, it can be resolved only with dose reduction [8].

And in terms of the topical effects, there will possibly be local reactions, including skin atrophy, telangiectasias, and striae, particularly with high-potency agents or prolonged use [9]. Contact allergy (2–6%) is more common with non-fluorinated steroids (e.g., hydrocortisone) due to degradation into haptenic metabolites [9].

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2.2. Immunosuppressive agents in the treatment of DM

Immunosuppressive agents are crucial as second-line therapies in DM, primarily serving as steroid-sparing agents to reduce corticosteroid-related toxicity and manage disease refractoriness or efficacy enhancement to corticosteroids treatment. They have a well-defined safety spectrum, varying efficacy profiles, and mechanisms that target important immune pathways, all of which support their usage in a variety of therapeutic scenarios.

2.2.1. Mechanism of action

Immunosuppressive agents act on distinct components of the immune system to suppress the autoimmune cascade that drives DM pathogenesis.

Methotrexate (MTX) inhibits folate metabolism, impairing T-cell proliferation and pro-inflammatory cytokine production (e.g., IL-2, IFN-γ), while cyclosporine suppresses T-cell activation through calcineurin inhibition, reducing autoreactive T-cell infiltration into muscle and skin [10-12].

Rituximab selectively depletes CD20-positive B cells, leading to reduced autoantibody production and cytokine-mediated vascular inflammation; this mechanism is particularly effective in anti-TIF1γ-positive DM [12,14].

Azathioprine and mycophenolate mofetil (MMF) inhibit purine synthesis, suppressing the proliferation of rapidly dividing T and B lymphocytes and thereby dampening autoimmune activity [10,13].

Janus kinase (JAK) inhibitors such as tofacitinib block JAK/STAT signaling, reducing interferon-driven inflammation, which is especially relevant in anti-MDA5-positive DM and associated interstitial lung disease [14].

2.2.2. Efficacy

Immunosuppressive agents demonstrate variable efficacy across DM subtypes:

MTX is a first-line adjuvant treatment for cutaneous DM, inducing a ≥ 40% reduction in Cutaneous Dermatomyositis Area and Severity Index [CDASI] in 55% of patients, while improving muscle strength in 60–70% of cases [10,11].

MMF is superior drug for refractory cutaneous DM with 77% of patients achieving ≥ 40% CDASI improvement. It also shows efficacy in juvenile DM (JDM), allowing 75% of patients to reduce corticosteroids doses [13,14].

Azathioprine has a comparable efficacy to MTX in muscle disease (70–75% response rate) but is less effective for cutaneous involvement. It is commonly used for long-term management [10,12].

Rituximab is also beneficial in refractory cases, especially anti-TIF1γ-positive DM, with about 70% of patients meeting the improvement criteria of International Myositis Assessment and Clinical Studies Group (IMACS). Additionally, it also reduces relapse risk in JDM and is effective for severe JDM with calcinosis, reducing skin and muscle disease activity in 40% of cases [11,14].

2.2.3. Side effects

Immunosuppressive agents are associated with increased infection risk due to broad immunosuppression.

MTX is often associated with hepatotoxicity, bone marrow suppression, and gastrointestinal intolerance [10,11]. Azathioprine may lead to leukopenia, hepatotoxicity, gastrointestinal

DOI: 10.54254/2753-8818/2026.AU32314

disturbances, and some rare hypersensitivity reactions [12]. MMF frequently causes diarrhea and nausea and may predispose patients to viral infections [14]. Rituximab carries risks of infusion reactions and prolonged hypogammaglobulinemia, increasing infection risk [13]. Collectively, careful patient selection and routine laboratory monitoring are required.

2.3. Intravenous immunoglobulin (IVIG) in the treatment of dermatomyositis

Intravenous immunoglobulin (IVIG), a purified preparation of polyclonal immunoglobin G, has emerged as a valuable therapeutic option for DM, particularly in refractory cases unresponsive to conventional immunosuppressants.

2.3.1. Mechanism of action

For the mechanism of action, IVIG exerts its effects through multiple immunomodulatory pathways targeting the autoimmune pathogenesis of DM:

Firstly, IVIG blocks the formation and deposition of the complement membrane attack complex (MAC) on capillaries, preventing the vascular destruction and promoting neovascularization [15,16]. Then IVIG downregulates pro-inflammatory cytokines such as TGF-β and interferon-γ, reducing overall inflammation [6,15]. IVIG also suppresses T-cell activation and B-cell-mediated autoantibody production while enhancing regulatory T-cell function, thereby dampening the autoimmune cascade [6,17].

2.3.2. Efficacy

IVIG demonstrates significant efficacy in both cutaneous and systemic manifestations of DM, particularly in refractory disease:

Subtype-specific Responses: In anti-MDA5-positive DM, IVIG improves cutaneous ulcers and stabilizes pulmonary function, with case reports showing lung infiltrates can clear up within 28 weeks of treatment [6,15]. For malignancy-associated DM, 80% of patients experience reduced skin rash and muscle weakness. Importantly, it carries a low risk of infections related to immunosuppression, which is valuable for patients with cancer [6,17].

However, IVIG is not very effective for inclusion body myositis (IBM), a specific type of inflammatory myopathy. Only about 28% of IBM patients treated with IVIG experience even mild improvements in muscle strength, meaning most patients see little to no benefit from this treatment [6,15].

Juvenile DM (JDM): IVIG enhances muscle strength and reduces calcinosis in JDM, with 75% of patients achieving steroid sparing within 6 months. Long-term data from a 4-year follow-up of 9 JDM patients treated with IVIG showed long-lasting benefits, including sustained better muscle function and reduced disease relapses [6,15].

Refractory cutaneous disease: In patients unresponsive to common immunosuppressive agents, IVIG leads to cutaneous improvement in 83% of cases, including resolution of Gottron's papules, heliotrope rash and skin ulcers [16,17].

2.3.3. Side effects

IVIG is generally a well-tolerated treatment and has a potent steroid-sparing effect [6,17]. IVIG also does not cause broad immunosuppression, making it a safer option for high-risk patients, such as those with malignancy-associated DM or elevated infection risk [6,18]. Common infusion-related

DOI: 10.54254/2753-8818/2026.AU32314

reactions include headache (42%), pyrexia (19%), and nausea (16%), typically mild and managed with premedication [16,19]. Serious side effects are rare but include thromboembolic events (5–6%), often associated with high doses and rapid infusion rates, and aseptic meningitis (2%) [15,16,18].

3. Discussion

3.1. Overview of the efficacy of the three major treatments for DM

DM requires tailored or individualized therapeutic strategies for each distinct patient to meet the requirements of different individuals to best control the progression. The three core treatments exhibit distinct efficacy profiles that align with different disease severities, subtypes and clinical priorities.

3.1.1. Corticosteroids: foundational but subtype-limited efficacy

Corticosteroids are first-line therapy for DM patients, effective in controlling muscular inflammation. Systemic corticosteroids induce remission in approximately 60 to 80% of patients with muscular involvement within 2 to 4 weeks, particularly in classic CDM with proximal muscle weakness [5,6,9]. Topical corticosteroids are effective for localized cutaneous lesions [7].

However, their efficacy is limited: first, cutaneous manifestations often remain refractory to systemic corticosteroids; second, the amyopathic subtype (i.e., CADM) rarely benefits. Meanwhile, long-term high-dose usage will also result in various side effects [2,4].

3.1.2. Immunosuppressive agents: steroid-sparing and subtype-targeted efficacy

Immunosuppressive agents serve as the second-line or adjuvant therapies. They function as steroid-sparing drugs. Their efficacy varies by the agent and DM subtype, as summarized in Table 3.

Table 3. Profile of key immunosuppressive agents in dermatomyositis

Agent	Doses	Key Efficacy	Advantages	References
Methotrexate (MTX)	7.5-25 mg/week oral	Improves muscle strength in 60-70%; ≥40% CDASI improvement in 55%.	Rapid onset, low cost, proven in adults & JDM.	[12]
Mycophenolate Mofetil (MMF)	2-3 g/day oral	Superior for skin: 77% achieve ≥40% CDASI improvement; steroid-sparing in 75% of JDM.	Favorable long-term safety; best for refractory skin disease.	[13]
Azathioprine	2-3 mg/kg/day oral	Comparable to MTX for muscle (70-75% response); good for maintenance.	Well-tolerated long-term; lower hepatotoxicity risk.	[10]
Rituximab	Two 1g IV infusions 2 weeks apart	70% response in refractory/anti-TIF1γ+ DM per IMACS; reduces JDM relapse.	Highly effective for refractory disease and ILD.	[14]
Cyclophosphamide	500-1000 mg/m² IV monthly	For severe JDM with calcinosis; improves activity in 40%.	Targets severe, refractory inflammation.	[14]

DOI: 10.54254/2753-8818/2026.AU32314

3.1.3. IVIG: refractory and high-risk disease efficacy

IVIG is often used as the third-line therapy effectively for DM patients who are refractory to corticosteroids and immunosuppressants, with unique benefits in high-risk subgroups:

It improves both cutaneous and muscular manifestations in 79% of patients, resolving refractory lesions in 83% of cases [17,19]. In anti-MDA5-positive DM, IVIG stabilizes pulmonary function and clears lung infiltrates within 28 weeks [8,14]. For malignancy-associated DM, it reduces both rash and muscle weakness in 80% of patients without exacerbating or broadening immunosuppression [6,17]. On the other hand, in JDM, it enhances muscle strength and reduces calcinosis, enabling steroid sparing in 75% of patients within 6 months. Its primary limitation is poor efficacy in IBM, with a percentage of only 28% of patients experiencing mild muscle improvement [6,15].

3.2. Comparison of cost and accessibility

Cost and accessibility are practical barriers that significantly impact treatment choice.

Corticosteroids are the top choice as they are most accessible and affordable, crucial for initial management. IVIG is the least accessible due to high cost and logistical requirements, limiting its use to refractory or high-risk cases. Immunosuppressants occupy a middle ground in cost and accessibility but require regular monitoring [7,14,19].

Table 4. Comparison of the cost and accessibility of corticosteroids, immunosuppressive agents, and IVIG

Treatment Type	Cost Profile	Accessibility	References
Corticosteroids	Low: Oral prednisone costs ~$10–$50/month; topical clobetasol ~$20–$60/tube.	High: Widely available globally; no specialized administration required (oral/topical).	[12]
Immunosuppressive agents	Variable: MTX (~$50–$150/month) < MMF (~$300–$800/month) < Rituximab (~$5,000–$10,000 per 2-infusion cycle).	Moderate: MTX/MMF are oral and accessible; rituximab/cyclophosphamide require IV administration (hospital/clinic setting).	[13,14]
Intravenous immunoglobulin	High: ~$10,000–$20,000 per cycle (2 g/kg); maintenance (1 g/kg) adds ~$5,000–$10,000 every 4–6 weeks.	Low: Requires IV infusion under medical supervision; supply limited in low-resource regions due to plasma-derived production.	[17,19]

3.3. Comparison of safety and side effects

The safety profiles differ in the type, severity, and reversibility of side effects, as outlined below in Table 5. IVIG's key safety advantage is the lack of broad immunosuppression.

DOI: 10.54254/2753-8818/2026.AU32314

Table 5. Comparative Safety Profiles of corticosteroids, immunosuppressive agents, and IVIG

Treatment Type	Common Side Effects	Serious / Management-Intensive Side Effects	References
Corticosteroids	Systemic: Hyperglycemia, insomnia, mood swings. Topical: Skin atrophy, telangiectasias.	Chronic: Osteoporosis, cataracts, adrenal suppression. Monitoring: Taper to avoid relapse.	[5, 8]
Immunosuppressives	Varies by drug: Nausea/Diarrhea (MMF), Myelosuppression (MTX, AZA), Infusion reactions (Ritux).	Hepatotoxicity (MTX), Nephrotoxicity (Cyclosporine), Infertility/Bladder Ca (Cyclophos.), Herpes Zoster (Ritux). Monitoring: Regular CBC, LFTs, Renal Function.	[10-13]
IVIG	Infusion-related: Headache (42%), Fever (19%), Nausea (16%).	Thromboembolism (5-6%), Aseptic Meningitis (2%). Mitigation: Slow infusion rate, pre-hydration.	[6,15,16,18]

3.4. Clinical recommendations for individualized treatment selection

Personalized treatment selection should align with disease subtype, severity, comorbidities, and patient priorities. For different clinical scenarios, tailored treatments are recommended, as listed in Table 5.

Table 6. Individualized treatment recommendations for dermatomyositis subtypes

Clinical Scenario	First-Line / Initial Management	Second-Line / Refractory or Severe Disease	Key Considerations
CDM	Systemic CS: Oral prednisone (0.5–1 mg/kg/day). Topical CS: High-potency steroids for localized lesions.	Add Methotrexate (for mild-moderate disease). Switch to MMF (for severe cutaneous involvement).	Goal is rapid induction of remission followed by steroid taper. MMF is superior to MTX for skin.
CADM	Topical CS for localized skin lesions.	Switch to MMF for refractory skin disease.	Muscle involvement is absent; focus is on cutaneous and systemic (especially pulmonary) manifestations.
Anti-MDA5+ CADM (High ILD Risk)	Initiate IVIG (2 g/kg every 4 weeks).	Consider combination with Rituximab or Cyclosporine.	Avoid Methotrexate due to inefficacy and potential risk in severe ILD. Goal is to stabilize lung function rapidly.
Juvenile DM (JDM)	Mild JDM: MTX (1 mg/kg/week) + low-dose steroids.	Severe JDM/Calcinosis: IVIG + Cyclophosphamide.	MMF is a key steroid-sparing agent. IVIG and Cyclophosphamide are reserved for severe, refractory cases.
Malignancy-Associated DM	IVIG as first-line therapy.	Add Rituximab if anti-TIF1γ antibodies are present.	IVIG is preferred to avoid exacerbating immunosuppression in cancer patients.

DOI: 10.54254/2753-8818/2026.AU32314

Table 6. (continued)

Refractory DM (CS/Immunosuppressants)	IVIG (2 g/kg every 4 weeks) for all subtypes.	Add Rituximab if ILD is a prominent feature.	IVIG provides dual cutaneous/muscular improvement. Rituximab offers targeted efficacy in specific antibody profiles.

3.5. Limitations and future directions

Although this essay has evaluated the recommended use of drugs base on several criteria, it still with limitations: first, comparative data between treatments (e.g., IVIG vs. rituximab in anti-MDA5 DM) are scarce; the current data still rely on single-agent studies rather than head-to-head trials. Second, the long-term safety data (e.g., MMF's risk of infection over 5 years) are still limited, particularly in pediatric populations [1,10,14].

Future research priorities should include randomized controlled trials comparing treatments for subtype-specific DM (e.g., DM with the presence of MSAs anti-MDA5, anti-TIF1γ); The development of cost-effective IVIG alternatives should also be considered to improve accessibility while maintaining or improving the living/ life quality of DM patients. Furthermore biomarkers (e.g., interferon signature, MSA levels) may also be introduced to predict treatment responses and reduce trial-and-error in clinical practices [1,14].

4. Conclusion

Corticosteroids, immunosuppressive agents, and IVIG play distinct roles in DM management. Corticosteroids are foundational for muscular inflammation, immunosuppressants enable steroid sparing to reduce steroid dependency - alleviating side effects, and target specific subtypes, and IVIG addresses refractory and high-risk disease. By weighing efficacy, cost, accessibility, and safety against individual patient factors (subtype, comorbidities, priorities), clinicians can optimize treatment selection, ultimately improve quality of life, and reduce long-term disease burden for DM patients. This report provides a deepened understanding of DM and serve as a reference to help patients clinicians in selecting suitable treatments. Meanwhile, it also aims to raise public awareness of this condition.

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