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Review Article

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Updates in Dermatomyositis: Newer Treatment Options and Outcome Measures From Dermatologic Perspectives

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ABSTRACT

Dermatomyositis (DM) is a rare autoimmune connective tissue disease with characteristic skin manifestations and possible muscle involvement. Recent advances in classification system to include skin-predominant subtypes, understanding underlying pathogenic mechanisms and the relationship between clinical phenotypes and myositis-specific autoantibodies have led to development of novel therapeutic options. This corresponds with efforts to develop better outcome measures to accurately catch the patients' current disease status and treatment-induced improvements. This report will review the updates in newer treatments and outcome measures of DM, specifically from a dermatologic point of view.

Keywords: Dermatomyositis; Outcome measures; Treatment

INTRODUCTION

Dermatomyositis (DM) is an idiopathic inflammatory myopathy (IIM) with an autoimmune pathogenesis. It classically features characteristic cutaneous manifestations, with a diverse systemic involvement, particularly muscle and lung[1-3]. Ongoing efforts to develop new therapies in DM have been hindered by lack of well-established classification system and outcome measures. Correct diagnosis and evaluation of treatment effects is crucial not only for an optimal patient management but also for better clinical trials to develop new medications.

EVOLUTION OF CLASSIFICATION

Categorizing DM as one of the IIMs implicitly implies that myositis is necessary for the diagnosis, as the original diagnostic criteria of Bohan and Peter suggests[4]. Characteristic skin manifestations with symptomatic myositis defines "classic DM". DM was previously thought to represent one end of the DM/polymyositis (PM) spectrum, involving both skin and muscle, while PM was the other muscle-limited end of the spectrum. This concept caused most of older clinical trials or epidemiologic studies to group DM and PM as one disease without clear distinction, complicating analyses[5].

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However, current understanding is that DM and PM are pathogenically distinct entities. Moreover, DM include the skin-predominant forms, also known as clinically amyopathic DM (CADM), encompassing hypomyopathic DM and amyopathic DM (ADM). In hypomyopathic DM, at least one muscle test, such as imaging, muscle biopsy, or muscle enzymes, is abnormal without clinically apparent myositis, whereas all the tests are normal in ADM. When typical DM skin lesions exist for at least 6 months without clinical evidence of muscle weakness by history or physical examination, CADM can be diagnosed[6]. Although 20%–30% of DM patients are clinically amyopathic[7,8], CADM has historically been ignored in most classification systems. The longstanding inappropriate exclusion of CADM from the IIM classification system caused misclassification in 55.6% of patients, with a median time of delay to proper diagnosis of about 15 months and a substandard screening for DM-associated findings, such as interstitial lung disease (ILD) or cancer[9,10]. It is only recently that CADM has gained recognition by medical fields other than dermatology, such as neurology and rheumatology[11,12]. Thus, recent classification schemes are trying to include CADM[11,14], and most include heliotrope sign, Gottron's papules and Gottron's sign as skin items. Unfortunately, a subsequent study found that this still missed 26% of ADM patients because the misclassified patients had other types of DM skin manifestations[15]. Thus, there remains a need for more sensitive criteria. Another latest movement is to incorporate the role of autoantibody-based serology[13]. Some myositis-specific autoantibodies (MSAs) have been shown to be associated with distinct DM skin features[16,17]. In 2018, The European Neuromuscular Center system suggested to subclassify the patients with a DM-specific autoantibody according to that autoantibody, whereas the patients who had DM without a DM-specific autoantibody were subclassified as having "autoantibody negative DM." Ulcer on the hand extensor joints, as seen in anti-melanoma differentiation-associated gene 5 (MDA5) DM, were considered equivalent to Gottron's papules, enhancing the diagnostic strength. This classification is meaningful because it clearly stipulated that a diagnosis of DM cannot be made without DM skin features.

Currently, there is an ongoing effort to develop a more inclusive skin-focused classification criteria with refined variables to distinguish cutaneous DM from mimickers[18]. A list of 25 potential criteria was generated in the categories of distribution, morphology, symptomatology, pathology and contextual factors. This system is not limited to the skin symptoms, but also includes DM-specific myositis antibodies, and contextual factors of ILD on CT and muscle weakness. To minimize ambiguity and increase precision, clinical descriptions replaced eponyms or signs, with the aid of pictures. For example, clinical descriptions of 'erythematous papules and/or plaques that are often flat topped, with or without scale, over the dorsal metacarpophalangeal (MCP) and/or interphalangeal (IP) joints' and 'macular erythema over the dorsal MCP and/or IP joints,' replaced the popular terms Gottron papule and Gottron sign, respectively. Presence of MSA was not specified to include all the pertinent antibodies, such as anti-Mi-2, anti-Jo-1, anti-MDA5, anti-nuclear matrix protein 2, and anti-small ubiquitin-like modifier activating enzyme. The criteria are undergoing validation in an international study with prospective enrollment of DM patients and mimicker controls. This study will hopefully allow inclusion of additional variables that can improve the classification of skin-predominant DM, and interdigitate with the European League Against Rheumatism and American College of Rheumatology criteria, which themselves are undergoing further evaluation.

OUTCOME MEASURES AND SCORES

Well-validated outcome measures are crucial in the optimal assessment and management of diseases (**Table 1**). The Cutaneous Dermatomyositis Disease Area and Severity Index (CDASI) was developed and refined to determine the cutaneous severity of DM in the aspects of activity (A) and damage (D); the skin disease activity is evaluated according to erythema, scale, and erosion/ulceration, with a range of 0–100; while the damage is scored by poikiloderma and calcinosis, with a range of 0–32[19-21]. It has been proven to have excellent correlation with other measurement scales like physician global assessment, and a good inter-rater and inter-disciplinary reliability[22,23]. The CDASI detects improvements in skin disease that are significant to patients, and a higher CDASI activity score correlated well with poorer quality of life (QoL)[24,25]. In patients with an initial CDASI-A score > 14 points, a 40% change in the CDASI-A score was found to indicate a meaningful change in QoL[26].

Another outcome measure to be increasingly used in various myositis clinical trials is the Total Improvement Score (TIS), with a range of 0–100[27,28]. Six core set measures (CSM) of myositis disease activity include Physician and Patient Global Activity on a 10-cm Visual Analogue Scale (VAS), muscle strength measured by manual muscle testing, physical function measured by the Health Assessment Questionnaire, Extramuscular Global Activity measured by the physician on a 10-cm VAS, and the most abnormal serum muscle enzyme. The TIS was calculated based on the levels of improvement and relative weights of each CSM. The skin activity is evaluated within the 'extramuscular global activity,' and thus the global score only indirectly measures the impact of cutaneous activity. Despite its usefulness to discriminate responses in active myositis, its applicability and sensitivity in skin-predominant DM is less straightforward. In a study to evaluate the sensitivity of various outcome measures, the CDASI activity score was more sensitive across time points than other outcome measures, including

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Table 1. Outcome measures and scoring system of dermatomyositis

Scoring system	Components of the measures	Skin-specific element
Cutaneous Dermatomyositis Disease Area and Severity Index (CDASI)	Calculated by sum of each domain 1) Total activity score - Each anatomical location: erythema; scale; erosion/ulceration - Gottron's - hands: erythema (double score if papules are present); ulceration - Periungual changes - Alopecia 2) Total damage score - Each anatomical location: Pokiloderma (dyspigmentatino or telangiectasia); calcinosis - Gottron's - hands: damage (dyspigmentation, scarring)	Yes
Total Improvement Score (TIS)	Calculated based on the levels of improvement and relative weights of 6 core set measures of myositis disease activity 1) Physician global activity 2) Patient global activity 3) Manual muscle testing 4) Health assessment questionnaire 5) Extramuscular global activity 6) Serum muscle enzymes	No
Dermatomyositis Outcomes for Muscle and Skin (DMOMS)	Improvement from baseline in 1) Physician global activity 2) Patient global activity (50% increase in weight) 3) Manual muscle testing 4) CDASI-activity	Yes

IGA and TIS[29]. Although the levels of improvement captured by TIS was proportionate to the degree of patient- and physician-reported improvement in skin disease, it was not specific to improvement in DM skin disease, since it is a composite measure. Another measure, the DM Outcomes for Muscle and Skin (DMOMS), allows equal weighting of skin and muscle disease and incorporates many aspects of the TIS used in the intravenous immunoglobulin (IVIG) trial. The DMOMS measure has performed better in muscle responders and skin responders in a phase 3 trial relative to the TIS[30].

TREATMENT

Conventional anti-inflammatory treatment

When it comes to deciding treatment modalities, many factors come into play: the degree of skin or muscle involvement, other systemic manifestations, and associated malignancy. The treatment responses of myositis and skin manifestations are often discordant, and skin disease is more recalcitrant[3,31]. Skin signs/symptoms and pruritus, especially on the scalp frequently persist even once myositis calms down. Unfortunately, potent topical treatments together with systemic therapy are frequently inadequate[1].

Photoprotective measures, such as broad-spectrum sunscreens and sun avoidance during peak ultraviolet hours are prudent[32]. Topical treatments are necessary, but insufficient to completely control any but the most the mild lesions. Topical corticosteroids can help control skin symptoms, such as erythema, and pruritus, especially for mild symptoms or when used in addition to systemic agents. Although high potency steroid solutions are commonly tried for scalp pruritus, it frequently is disappointing. Topical calcineurin inhibitors, such as tacrolimus 0.1% or pimecrolimus 1% could have comparable effects to mid-potency corticosteroids without concern for steroid-associated atrophy or hypopigmentation.

Although systemic corticosteroids are the first-line treatment of choice for myositis, it is not recommended as a long-term monotherapy to control skin symptoms, given that they often prove to be unimpressive, and the frequent significant steroid-related side effects[33,34]. Sometimes, even when the myositis is responsive to the systemic corticosteroids, the skin symptoms still remain. Systemic corticosteroids are usually indicated for muscle, joints, or lung involvement, and steroid-sparing agents such as oral immunosuppressives should often be combined with the corticosteroids.

Antimalarials, particularly hydroxychloroquine (HCQ), has been the treatment of choice for skin symptoms in DM. However, the benefit is underwhelming compared to that in LE, with modest efficacy in 15%–25% of patients[33]. Interferon (IFN)-β production by myeloid dendritic cells correlated with HCQ refractoriness[35]. When HCQ monotherapy is not sufficient, addition of quinacrine or substitution with chloroquine, or combination with other immunosuppressives or IVIG therapy may be necessary[33]. Adverse reactions, such as drug reactions to HCQ, are more common than in LE[36]. About 20% of DM patients experience skin eruptions from HCQ, and thus have stopped the medication. They frequently need short term corticosteroids to treat the adverse reaction. Although HCQ may be useful for mild inflammatory arthritis, it usually is not

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effective for non-cutaneous symptoms in DM[37,38]. Considering the relatively favorable safety profile of antimalarials, they can be used as primary baseline therapy, often with other systemic therapies.

Low dose methotrexate (MTX) is effective to control skin symptoms in DM[39-41]. It could also be helpful for myositis and accompanying arthritis, although these conditions might require higher doses or MTX could be given for steroid-sparing effects[42,43]. If pulmonary involvement is suspected, it would be best to choose other agents, such as mycophenolate mofetil, given the potential of MTX to induce pneumonitis[44]. Mycophenolate mofetil is another option for skin symptoms and myositis in DM, and is the treatment of choice when ILD is present[45-49].

Novel therapeutic approaches

1) IVIG and fragment cystallizable receptor (FcRn) inhibitors

The use of IVIG is not entirely new (**Table 2**). In 1993 its outstanding effects for myositis and skin symptoms were noted in a randomized controlled trial (RCT)[50], multiple single institution-based case series reported improvements in refractory cutaneous DM, associated with 60%–80% response rates and the ability to decrease or discontinue systemic corticosteroid use[51-53]. In one study, a second course of IVIG was still successful even after relapse in 53% of the patients 6 months after the last injection[51]. The mechanism of action of IVIG in autoimmune diseases is still not clearly elucidated. It is thought that IVIG has diverse immunomodulatory effects. One important trait of many autoimmune diseases is the appearance of autoantibodies, particularly immunoglobulin (Ig) G isotype[54]. The neonatal FcRn is responsible for the long half-life of IgG, by protecting IgG from intracellular degradation and recycling it back to the circulation[55,56]. Both IVIG and pathogenic IgG autoantibodies compete for an access to IgG receptors, including FcRn. Thus, high-dose IVIG saturates FcRn binding sites, causing loss of FcRn protection for pathogenic autoantibodies, followed by their accelerated degradation[57,58]. IVIG works not only through neutralization of pathogenic autoantibodies, but also through competitive inhibition of reticuloendothelial immune system through large immune complexes, clearing complement and inhibiting B cells, and expanding regulatory cells[59]. The standard dosing regimen is 2 g/kg monthly divided over 2 to 5 days. The therapeutic effect may be as early as within one month, but it may not be perceived until the second or third dose.

In 2022, a placebo-controlled phase 3 trial of IVIG exhibited a significant difference in the TIS score in 95 randomized patients (79% vs. 44%, 95% confidence interval, 17 to 53; $p<0.001$) treated with placebo or IVIG at a dose of 2.0 g/kg every 4 weeks for 16 weeks[60]. Headache was the most common side effects (42%), followed by pyrexia (19%) and nausea (16%). Six thromboembolic events were the most notable serious adverse events, leading to changes in the rate of infusion of IVIG. Recently, a post-hoc analysis of the trial to determine the efficacy of IVIG versus placebo for controlling of the cutaneous aspect of DM showed that at Week 28, more than 70% of patients experienced at least a 35% improvement in CDASI-A score, a threshold which is associated with a meaningful change in QoL[26,61]. Different efficacy measures had good correlations; at week 16, the decrease in cutaneous disease activity score was strongly correlated with the improvement in extramuscular global assessment with TIS, suggesting that both the myopathic and cutaneous aspects of DM improve similarly with IVIG therapy.

This was the key trial for the US Food and Drug Administration (FDA) approval of IVIG for the treatment of DM. However, because the patients with skin-predominant disease, including those with resolved muscle disease were excluded from the trial, there have been problems getting insurance approval for IVIG for the skin-predominant subgroup[62]. Since FDA recommends using a non-validated IGA instead of the fully validated CDASI score, this could have precipitated this shift of focus to myopathic DM patients[29]. This emphasizes the need for inclusion of the entire spectrum of the disease for clinical trials.

Considering the potential mechanism of action of IVIG involves an inhibition of FcRn, specifically targeting FcRn could have favorable effects for DM. Efgartigimod is a human IgG1 Fc fragment mutated to improve its affinity for FcRn, while Nipocalimab is a fully human aglycosylated IgG1 monoclonal

Table 2. Novel therapeutics available or in development for dermatomyositis

Name	Class	Target	Current availability
Intravenous immunoglobulin	Pooled antibody	IgG receptors (FcγRs)	Available
Efgartigimod	IgG1 antibody Fc fragment	FcRn	On trial
Nipocalimab	Monoclonal antibody	FcRn	On trial
Rituximab	Monoclonal antibody	CD20 on B cells	Available
Dazukibart	Monoclonal antibody	Interferon-b	On trial
Ruxolitinib	Small molecule inhibitor	JAK1 and 2	Available (off-label)
Tofacitinib	Small molecule inhibitor	JAK1, 2, and 3	Available (off-label)
Brepocitinib	Small molecule inhibitor	TYK2 and JAK1	On trial
Enpatoran	Small molecule inhibitor	Toll-like receptors 7/8	On trial
Lenabasum	Selective receptor agonist	Cannabinoid receptor 2	On trial

IgG: immunoglobulin G, Fc: fragment cystallizable, FcγRs: Fc gamma receptor, FcRn: neonatal Fc receptor, JAK: Janus kinase, TYK: tyrosine kinase.

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antibody against FcRn[63,64]. Currently, Efgartigimod PH20 SC (NCT05523167)[65] and Nipocalimab (NCT05379634)[64] are undergoing phase 2/3 and phase 2 trials, respectively, to evaluate their efficacy and safety versus placebo in active IIMs, including DM, PM, and anti-synthetase syndrome.

2) Depletion of B cells

Rituximab is a chimeric monoclonal antibody against CD20 on B cells[66]. Considering that immature transitional B cells were found to expand significantly and correlated with the pro-inflammatory type 1 IFN signature in JDM, depleting B cells could be useful for managing DM[67]. Despite its reported efficacy with disease modifying effects in other autoimmune diseases such as pemphigus vulgaris, its effect in cutaneous DM is debatable. A meta-analysis of 26 studies to evaluate the efficacy of rituximab in IIMs, including DM, showed that 65% of the patients were responsive to rituximab, with a 45% of complete response rate[68]. Subgroup analysis for showed that 68% of patients with refractory DM or PM were responsive to rituximab, and the overall efficacy rate for skin involvement was 81%. In a systematic review regarding rituximab use in the treatment of ILD related to anti-MDA5 DM, 71.4% (25) of the 35 included patients responded to treatment. Nineteen patients (54.3%) had improvements in skin involvement as well, including reduced degree and size or healing of skin ulcers. The most common side effects were infections (37.1%)[69]. In a post-hoc analysis of an RCT of 120 refractory DM patients (72 adult DM and 48 JDM), the skin disease activity improved from baseline in both adult DM and JDM patients[70]. In adult DM patients, there was a significant decrease in frequency of erythroderma, erythematous rash without secondary changes of ulceration or necrosis, heliotrope, Gottron sign/papules, periungal erythema, diffuse alopecia and mechanics hands, whereas there was no improvement in cutaneous ulceration, panniculitis, erythematous rash with ulceration or necrosis, focal alopecia, calcinosis, cutaneous scarring or atrophy, poikiloderma, or lipodystrophy. Adult DM patients receiving rituximab earlier in the trial tended to respond faster than those who received rituximab later. In a case series of the patients with recalcitrant skin symptoms despite good control of muscle symptoms, cutaneous lesions were well managed after completion of rituximab therapy with a benefit of discontinuing concomitant DM therapies[71]. On the other hand, in an open-label pilot trial, skin disease severity score at week 24 did not change from baseline, with 3 of 8 participants exhibiting partial remission of muscle strength deficits, although peripheral B cells were adequately depleted[72]. Further well-designed skin-oriented investigations are warranted.

As another measure to target B cells, chimeric antigen receptor (CAR)-T cells that recognize CD19 on B cells have recently gained interest. Although not specifically in DM, there have been a few case reports on the patients with multi-drug resistant refractory anti-synthetase syndrome, who were responsive to CD19 CAR-T cell therapy[73,74]. With B cell depletion confirmed in peripheral blood, they experienced clinical improvements of reduced myalgia and regained muscle strength, and decreased disease-associated serum markers, including anti-Jo-1 antibody levels. In a recent case series about CD19 CAR-T cell therapy in various autoimmune diseases, all the patients with IIM had an ACR–EULAR major clinical response during the median follow-up of 15 months after a single infusion, and immunosuppressive therapy was completely stopped[75]. There currently are early-phase ongoing clinical trials about CAR-T cell therapy in DM. An open-label phase 1/2 trial is investigating CABA-201, an investigational fully human CD19-CAR T cell therapy, in patients with several subtypes of IIMs, including DM, immune-mediated necrotizing myopathy and anti-synthetase syndrome (NCT06154252)[76]. Another Phase I single arm study is evaluating the efficacy and safety of CD19 targeted CAR-T cells therapy for patients with refractory autoimmune diseases, including systemic lupus erythematosus and DM (NCT06056921)[77].

3) Targeting IFN pathways

Since type I IFNs play crucial roles in DM pathogenesis, a potent, selective, humanized IgG1 neutralizing antibody against IFN-β, dazukibart (PF-06823859), has been developed[78]. A recent double-blind, placebo-controlled, phase 2 study (NCT03181893) enrolled the patients with skin- or muscle-predominant refractory DM who had failed at least one standard of care systemic treatment. In skin-predominant group, the treatment effect was noticeable as early as week 4, for not only skin symptoms evaluated by CDASI-A, but also for QoL and mental component of the disease.

Alternatively, the blockade of type I IFNs using Janus kinase (JAK) inhibitors could also be a suitable step. Ruxolitinib, a JAK-1 and -2 inhibitor, abolished the *in vitro* type I IFN-induced pathogenic effects on myoblasts and vascular network, and improved skin lesions, muscle weakness, and reduced serum type I IFN levels and IFN-inducible gene scores in refractory DM patients[79]. Small cases series also reported the usefulness of tofacitinib in the patients with recalcitrant cutaneous DM symptoms[80-82]. Brepocitinib, a tyrosine kinase 2 and JAK1 inhibitor, is under phase 3 trial for DM (NCT05437263)[83].

Based on the contribution of activated toll-like receptors (TLRs) 7 and 8 to type I IFN production in dendritic cells and monocytes[84], a phase 2 trial in DM and PM to evaluate the efficacy and safety of enpatoran (M5049), a TLR7/8 inhibitor, is also in progress (NCT05650567)[85].

4) Others

Lenabasum is a non-psychoactive selective agonist for cannabinoid receptor 2, which is expressed on activated immune cells, highest in DCs followed by B cells, T cells, and macrophages in DM

skin lesions[86,87]. It has anti-inflammatory attributes; it suppressed secretion of TNF-α, type I IFNs, IL-4 and IL-31 from immune cells of patients with DM *in vitro*[88,89]. A double-blind, randomized, placebo-controlled phase 2 trial tested the safety and efficacy of lenabasum in 22 skin-predominant DM patients with refractory skin activity of CDASI ≥ 20 despite the use of immunosuppressive drugs[90]. Twelve weeks of lenabasum treatment decreased the protein expression of IFN-β, IFN-γ, and IL-31, and the number of CD4+ T cells in DM skin[86,90]. The mean difference of CDASI improvement from baseline between two groups became noticeable (=−4.5, p=0.0857) by 2 weeks after the dose was increased from 20 mg daily to 20 mg twice daily. The effect was statistically significant (−6.5, p=0.0382) on day 113, 4 weeks after lenabasum discontinuation, suggesting its long-lasting immunomodulatory effects. The following multicenter phase 3 trial evaluated the efficacy at 28 weeks and 52 weeks after lenabasum treatment (20 mg twice daily vs placebo) by TIS[91]. The trial was discontinued after all the participants completed week 28, and some had completed week 52 by then. The primary endpoint at week 28 was not met (TIS, 28.3±19.75 vs. 27.2±19.23; p=0.3311), and the secondary endpoint at 52 weeks was not either (TIS, 40.6±16.88 vs. 34.8±19.94; p=0.2290). However, subgroup analysis of patients with and without muscle weakness, better captured improvements; in subjects with muscle weakness, lenabasum-treated group achieved a greater TIS score and treatment difference at week 40 (p=0.0172), but the change was not statistically significant at the predetermined endpoint at week 28; in subjects without muscle disease, mean CDASI activity score change was significantly greater in lenabasum group than placebo group at week 28 (p=0.0461) and week 52 (p=0.0056).

CONCLUSION

There have been significant advances in the classification of IIMs, with increasing awareness of skin-predominant types of DM, novel findings of disease phenotypes associated with MSAs, and development of newer treatment modalities. However, there still remain demands for validated classification criteria and skin-predominant DM-inclusive clinical trials for better management of challenging patients. Diagnosis, evaluation, and management of DM often requires multidisciplinary efforts.

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The authors have nothing to disclose.

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