Overlap of dermatomyositis and cutaneous lupus erythematosus: A case series

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INTRODUCTION

Dermatomyositis (DM) is a chronic, systemic autoimmune disorder commonly characterized by classic cutaneous findings, which include Gottron's sign, Gottron's papules, and heliotrope rash. These lesions can occur independently, which would lead to classification as clinically amyopathic DM or in conjunction with muscle weakness, which would lead to classification as classic DM.[1] Several studies have indicated that idiopathic inflammatory myopathies, which includes DM, can overlap with other autoimmune connective tissue diseases.[2,3]

Cutaneous lupus erythematosus (CLE) with or without systemic manifestations is one such autoimmune disorder that can overlap with DM.[4] CLE is classified into 3 subtypes with distinct clinical presentations.[5] Acute CLE classically presents with a transient, nonscarring malar rash. Subacute CLE (SCLE) has erythematous, scaly annular plaques or papulosquamous lesions that are both nonscarring. Discoid lupus erythematosus is the most common chronic CLE and presents with well-defined, scaly, erythematous papules that can progress to infiltrated discoid plaques and lead to scarring.

In addition to genetic autoimmune predisposition, several external triggers, including medications and infections, have been implicated as triggers for new-onset DM or lupus.[6-12] Herein, we report a case series of 8 patients with an overlap of DM and CLE that developed in the context of background autoimmunity and/or after COVID-19 infection or vaccine, tumor necrosis factor (TNF)-α inhibitor use, and anti-CD20 agent use. These findings suggest the potential role of external factors in modulating these diseases and present the complex management of multiple multisystem autoimmune disorders.

Abbreviations used:

CLE:	cutaneous lupus erythematosus
DM:	dermatomyositis
SCLE:	subacute lupus erythematosus
SLE:	systemic lupus erythematosus
TNF:	tumor necrosis factor

CASE SERIES

The demographic and clinical features of all patients are summarized in Table I. Table II summarizes the prior laboratory and additional investigations for all patients. The mean age was 54.3 years, and all patients were females and most were Caucasian (n = 6). The majority of patients had a history of lupus (n = 7) before developing DM. Among these patients, 3 developed DM symptoms following identifiable triggers, which included COVID-19 infection and/or vaccine (n = 3, patients 2,3,4) and the TNF-α inhibitor adalimumab for concomitant rheumatoid arthritis (n = 1, patient 4).

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Table I. Patient demographics and disease characteristics

Patient	1	2	3	4	5	6	7	8
Age, y	34	42	62	59	55	71	67	45
Sex	Female	Female	Female	Female	Female	Female	Female	Female
Race	White	Hispanic/Latino	Black	White	White	White	White	White
Overlap disease timing	2020: SCLE/SLE 2022: CADM	2001: ACLE/SLE 2021: CDM	2007: DLE 2021: SCLE and CDM	1980s: SCLE/SLE 2016: CDM	2007: ACLE/SLE 2018: CDM	2015: CDM 2020: SCLE	1997: ACLE/SLE 2007: CDM	2006: ACLE/SLE 2019: SCLE 2020: CADM
Overlap disease triggers	N/A	DM rash after COVID-19 infection and 9 mo later, muscle symptoms after bivalent vaccine	DM after first COVID-19 vaccine	DM rash after starting adalimumab* and 6 y later, muscle symptoms after COVID-19 infection	N/A	SCLE after receiving RTX†	N/A	N/A
Other concomitant autoimmune diseases	N/A	APLS, Relapsing polychondritis	Scleroderma	Rheumatoid arthritis, secondary Sjogren's syndrome	Sjogren's syndrome	N/A	Sjogren's syndrome	Cutaneous vasculitis, Sjogren's syndrome
Clinical manifestations								
Lupus	Erythematous papules on arms and trunk, photosensitivity, arthritis	Malar rash, serositis, arthritis, Raynaud's phenomenon, oral ulcers	Hyperkeratotic and atrophic papules in conchal bowls, hyperpigmented macules and patches on arms, legs, and back	Photoexposed annular lesions, seizures, fatigue, low-grade fevers, polyarthritis	Photosensitive rash, alopecia, oral/nasal ulcers, arthritis, serositis, Raynaud's phenomenon, lymphopenia	Erythematous plaques on the back, arms, and chest	Photosensitive rash, oral ulcers, fatigue, arthralgias, Raynaud's phenomenon, anemia	Malar rash, alopecia, oral ulcers, migraine, nephritis, arthritis, erythematous scaly papules on chest, back, arms, and hairline, thrombocytopenia
DM	Malar erythema, Gottron's sign, itchy rash on arms	Periorbital erythema and edema, malar erythema, Gottron's sign, dysphagia, myalgia	Erythematous V-of the neck, arms, abdomen, and back, myalgia, proximal weakness	Scaly erythema on the scalp with alopecia, erythema on ears, arms, and knees, Gottron's sign, periungual erythema with nailfold capillary changes, myalgia, proximal weakness, ILD	Periorbital lichenification, erythema over elbows, Gottron's sign, proximal nailfold changes, myalgias	Erythema and scale on the scalp with thinning of hair, facial erythema, dysphagia, proximal weakness	Erythema on hands, elbows, hips, and shoulders, Gottron's sign/papules, papules on elbows, mechanic's hands, proximal weakness	Periorbital erythema, Gottron's sign, proximal nailfold changes
Complete topical treatment history	Clobetasol 0.05%, Triamcinolone 0.1%	Clobetasol 0.05%	Clobetasol 0.05%, Tacrolimus 0.1%, Triamcinolone 0.1%	Betamethasone 0.05%, Clobetasol 0.05%, Pimecrolimus 1%, intralesional Triamcinolone 5 mg/mL	Clobetasol 0.05%, Tacrolimus 0.1% Triamcinolone 0.1%	Clobetasol 0.05%, Triamcinolone 0.1%, Tacrolimus 0.1%	Triamcinolone 0.1%	Pimecrolimus 1%, Tacrolimus 0.1%, Triamcinolone 0.1%
Complete systemic treatment history	HCQ, MMF, MP, MTX	Anakinra, AZA, CTX, dapsone, HCQ, MMF, MP, MTX, QC	Apremilast, HCQ, IVIG, MMF, prednisone	Chloroquine, HCQ, IVIG, MTX, prednisone, QC, TFB‡	BEM, HCQ, MMF, MTX, prednisone	HCQ, IVIG, MMF, prednisone	AZA, chloroquine, HCQ, MTX, prednisone, QC	CTX, HCQ, MMF, prednisone
Systemic treatment changes at onset of overlap Disease	Continued on HCQ and MMF	Continued on HCQ and MP; Added MMF and QC	Continued HCQ; Added MMF and prednisone	Switched adalimumab to TFB; Added HCQ	Continued on HCQ, MMF and prednisone; Discontinued BEM	Continued on HCQ	Discontinued HCQ, MTX, and quinacrine; Started AZA, chloroquine, and prednisone	Continued on HCQ and MMF

Current regimen	Triamcinolone 0.1%, HCQ 400 mg/d, MMF 2000 mg/d	Anakinra 100 mg, HCQ 400 mg/d, CTX every 2 wk, MP 32 mg/day, QC 100 mg/d	Clobetasol 0.05%, IVIG 2 g/kg monthly, MMF 3000 mg/d, prednisone 5 mg/d	Betamethasone 0.05%, Clobetasol 0.05%, Pimecrolimus 1%, IVIG 2 g/kg every 5 wk, MTX 0.6 mL/wk, TFB 11 mg/d	Clobetasol 0.05%, Tacrolimus 0.1% Triamcinolone 0.1%, HCQ 300 mg/d, MTX 20 mg/wk	Clobetasol 0.05%, Triamcinolone 0.1%, Tacrolimus 0.1%, HCQ 400 mg/d, IVIG monthly	Triamcinolone 0.1%, Chloroquine 250 mg/d for 4 d/wk, prednisone taper, MTX 20 mg/wk	Pimecrolimus 1%, Tacrolimus 0.1%, Triamcinolone 0.1%, HCQ 300 mg/d, prednisone taper, MMF 2000 mg/d
Response to current regimen	Continues to experience arthritis and transient itchy rash on the chest and forehead	Improving facial erythema and swelling, and arthritis	Continues to experience pain in lower extremities and skin erosions	Improved muscle weakness but continues to experience erythema on the scalp/face and alopecia	Improved rashes with continued mild Gottron's sign	Improved muscle symptoms and skin activity but continues to have mild facial erythema in the malar area and erythematous papules/ plaques on the trunk	Continues to experience weakness in lower extremities and itchy rash on ears and arms	Continues to have facial erythema

ACLE, Acute cutaneous lupus erythematosus; *APLS*, antiphospholipid syndrome; *AZA,* azathioprine; *BEM,* belimumab; *CADM*, clinically amyopathic dermatomyositis; *CDM*, classic dermatomyositis; *CTX,* cyclophosphamide; *DLE*, discoid lupus erythematosus; *DM,* dermatomyositis; *HCQ,* hydroxychloroquine; *ILD*, interstitial lung disease; *IVIG,* intravenous immune globulin; *MMF*, mycophenolate mofetil; *MP,* methylprednisolone; *MTX,* methotrexate; *QC,* quinacrine; *RTX,* rituximab; *SCLE,* subacute cutaneous lupus erythematosus; *SLE,* systemic lupus erythematosus; *TFB*, tofacitinib.
*Adalimumab was used for treatment of rheumatoid arthritis.
†Rituximab was used for treatment of brain lymphoma since 2017.
‡Tofacitinib replaced adalimumab in the setting of new-onset DM.

Table II. Summary of prior laboratory findings and investigations

Patient	1	2	3	4	5	6	7	8
ANA	Negative	1:1280 homogenous	1:160 nucleolar	1:640 homogenous	1:80 diffuse	1:2560 speckled	1:160 diffuse	1:80 speckled and 1:1280 SSA/Ro
Lupus								
Anti-dsDNA	Negative	Positive (105 IU/mL)	Negative	Negative	Negative	Negative	Negative	Positive (elevated to >300 IU/mL)
Anti-Smith	Negative	Negative	Negative	Negative	Negative	Negative	N/A	N/A
Anti-SSA	Negative	Negative	Negative	Positive (>8.0)	Negative	Positive (elevated to 3.80)	N/A	Positive (>8.0)
Anti-SSB	Negative	Negative	Negative	Negative	Negative	Positive (elevated to 6.4)	N/A	Positive (no titer)
Antiphospholipid antibodies	Negative	ACL: IgM 14.7 and IgG 16 units B2GP: negative LAC: positive	N/A	N/A	ACL: Negative B2GP: N/A LAC: N/A	Negative	N/A	ACL: IgG 19 units B2GP: N/A LAC: N/A
C3	Normal	Normal	Normal	Normal	Decreased to 85 mg/dL	Normal	Normal	Decreased to 24 mg/dL
C4	Normal	Normal	Normal	Normal	Normal	Normal	Decreased to 13 mg/dL	Decreased to <2 mg/dL

Continued

Table II. Cont'd

Patient	1	2	3	4	5	6	7	8
Skin biopsy	Superficial and deep perivascular and periadnexal dermatitis	Multiple biopsies showing thrombotic vasculopathy	Interface dermatitis and focally increased dermal mucin deposition	N/A	N/A	N/A	Interface dermatitis associated with cutaneous horn and features of perforating disorder; perivascular lymphocytic infiltrate and focal papillomatosis	N/A
DM								
CK	N/A	Normal	Elevated to 2757 U/L	Normal	Normal	Elevated to 1167 U/L	Elevated to 158 U/L	Normal
Aldolase	N/A	Elevated to 8.6 U/L	Elevated to 40.8 U/L	Normal	Normal	Elevated to 14 U/L	Normal	Normal
Myositis panel	Negative	Negative	Anti-Ku positive, anti-RNP positive	Negative	Negative	Anti-RNP positive	N/A	Negative
Skin biopsy	N/A	Multiple biopsies showing thrombotic vasculopathy	Dermal mucin deposition	N/A	Mild interface dermatitis	Interface dermatitis	N/A	N/A
Other tests	PFTs: normal	Flouroesophagram: nonspecific esophageal dysmotility	MRI muscle: diffuse, severe soft tissue and muscle edema	PFTs: decreased DLCO and TLC; restrictive lung disease EMG: abnormal spontaneous activity showing mild myopathy CT chest: basilar predominant reticulation and groundglass opacity	PFTs: normal MRI Muscle: negative for myositis	PFTs: normal MRI Muscle: very mild myopathic changes in right vastus medialis	PFTs: mildly reduced DLCO Muscle biopsy: mild myositis and multifocal fiber atrophy MRI muscle: negative for myositis	MRI muscle: negative for myositis
Malignancy work-up	N/A	Mammogram: within normal limits Ultrasound pelvis: multiple simple ovarian cysts, endometrial polyp, and complex free fluid in pelvis CT abdomen and pelvis: subacute bilateral pulmonary emboli, parastomal hernia	Colonoscopy: consistent with diverticulosis Mammogram: within normal limits	Mammogram: new calcifications in the upper inner quadrant of the left breast* CT abdomen and pelvis: normal	Colonoscopy: normal Mammogram: within normal limits	CT chest: right lower lobe lung nodule† MRI head: right frontal lobe mass‡ CT abdomen and pelvis: normal	CT abdomen and pelvis: suggestive of liver hemangioma MRI Abdomen: hemangiomas in the right and left hepatic lobes Colonoscopy: one small rectal polyp Mammogram: within normal limits	Mammogram: within normal limits

ACL, Anticardiolipin antibodies; *ANA*, antinuclear antibodies; *Anti-dsDNA*, anti-double stranded DNA antibodies; *B2GP*, beta-2-glycoprotein; *CK*, creatinine kinase; *CT*, computed tomography; *EMG*, electromyography; *LAC*, lupus anticoagulant; *MRI*, magnetic resonance imaging; *PFT*, pulmonary function testing; *RNP*, ribonucleoprotein; *SSA*, Sjogren's-syndrome-related antigen A; *SSB*, Sjogren's-syndrome-related antigen B.

*Diagnosed with stage 1A breast cancer in 2019.

†Diagnosed with grade 1 neuroendocrine tumor in 2023.

‡Diagnosed with diffuse large B-cell lymphoma in 2017.

Patient 6 had a history of DM and then developed lupus in the setting of receiving the anti-CD20 agent rituximab for brain lymphoma. Overall, the mean time to presentation of disease overlap was 12.8 (range 2 to 36) years. In addition to DM and lupus, 6 patients had a past medical history of other autoimmune connective tissue disease-related manifestations that included Sjogren's syndrome (n = 4), relapsing polychondritis (n = 1), antiphospholipid syndrome (n = 1), scleroderma (n = 1), rheumatoid arthritis (n = 1), and cutaneous vasculitis (n = 1). Seven of the 8 patients had completed malignancy screenings, and 2 patients (patients 4 and 6) had a documented history of cancer. Patient 4 had a history of stage 1A left breast cancer. Patient 6 had a history of diffuse large B-cell brain lymphoma and grade 1 neuroendocrine tumor of the lung.

Six patients had systemic lupus erythematosus (SLE) with skin manifestations whereas 2 had CLE only. Five patients had SCLE, 4 patients had acute CLE, and 1 had discoid lupus erythematosus. SLE manifestations predominantly presented with joint involvement (n = 6), oral ulcerations (n = 4), and hematologic abnormalities (n = 3). SCLE lesions most commonly presented as erythematous papules on the arms and trunk (n = 3). Discoid lupus erythematosus lesions in the 1 patient were limited to the conchal bowls. Previous laboratory testing has shown that 2 patients had anti-double stranded DNA antibodies, 3 patients had anti-Sjogren's-syndrome-related antigen A antibodies, 2 patients had anti-Sjogren's-syndrome-related antigen B antibodies, 2 patients had antiphospholipid antibodies, and 3 patients had hypocomplementemia. Of the 4 patients with available skin biopsy reports of a clinically suggestive CLE lesion, 2 were consistent with the diagnosis.

Six patients presented with both skin and muscle DM symptoms, while 2 had only skin involvement. Rashes predominantly presented on dorsal joints of the hand (n = 6), face and periorbital region (n = 5), and arms including elbows (n = 5). Muscle involvement largely presented as proximal muscle weakness in the arms and legs (n = 4) and dysphagia (n = 2). Previous laboratory testing has shown elevated muscle enzymes in 4 out of the 6 patients that exhibited muscle symptoms. Four out of the 6 patients with muscle symptoms underwent muscle magnetic resonance imaging (MRI) of which 2 patients had findings consistent with active muscle disease. One out of the 6 patients with muscle symptoms had a negative muscle MRI but biopsy-proven muscle disease. One out of the 6 patients with muscle symptoms had electromyography findings consistent with myositis. The myositis panel was positive in only 2 patients for anti-Ku and anti-ribonucleoprotein antibodies. Four patients had available skin biopsies of a clinically suggestive DM rash, of which 3 showed features consistent with the diagnosis (dermal mucin and interface dermatitis). One patient also had a diagnosis of interstitial lung disease confirmed with computed tomography chest findings and pulmonary function testing.

All patients had received topical steroids and/or immunomodulators, systemic antimalarials, systemic steroids, and immunosuppressive agents for their lupus and/or DM. Agents included mycophenolate mofetil (n = 6), methotrexate (n = 5), intravenous immune globulin (n = 3), azathioprine (n = 2), cyclophosphamide (n = 2), anakinra (n = 1), dapsone (n = 1), apremilast (n = 1), tofacitinib (n = 1), and belimumab (n = 1). Of note, at the time of disease overlap, 5 patients underwent changes to their treatment regimen, and 3 patients continued on their existing regimen. Patients were treated most commonly with hydroxychloroquine (n = 8), mycophenolate mofetil (n = 5), and steroids (n = 4). Four patients were noted to have improvement in symptoms with their current regimens.

DISCUSSION

Seven patients had a history of CLE with or without systemic manifestations and subsequently developed DM. The criteria used to diagnose DM in these patients included development of characteristic skin rashes (Gottron's sign, periorbital erythema, facial erythema, V-neck erythema, and/or periungual erythema and changes) with or without concomitant muscle weakness. One patient had a history of DM before developing CLE. A diagnosis of CLE was made in this patient due to the development of erythematous plaques. It is also important to note that in these patients, only 2 out of the 4 available CLE biopsies were consistent with the diagnosis, and only 2 out of the 4 classic DM patients who underwent muscle MRIs had positive findings. This points to the difficulty in establishing these diagnoses with currently available tests, and highlights the importance of following clinical findings and the need for more definitive diagnostics.

Additionally, while DM can overlap with other immune-mediated connective tissue diseases due to an autoimmune predisposition, several of the patients in this case series also developed disease overlap in the context of identifiable external triggers.

DM skin has predominantly myeloid dendritic cells, while CLE skin contains plasmacytoid dendritic cells, with CLE also involving myeloid dendritic cells in quinacrine responders.[13-15] These cell types, in addition to macrophages, other inflammatory cells,

and keratinocytes, contribute to elevated type 1 interferon-regulated gene expression in the peripheral blood of SLE/CLE patients and in DM patients.[13,14] Having a predisposition of an elevated type 1 interferon signature with a diagnosis of either DM or lupus could explain the increased likelihood of developing the other disease type and presenting with an overlap. Furthermore, using immunohistochemistry or transcriptomics to differentiate between myeloid dendritic cells and plasmacytoid dendritic cells as drivers of the type 1 interferon signatures in these disease states may be useful for diagnosis given the current difficulties with definitive diagnostic testing. We would also like to highlight that of the CLE subtypes, interestingly SCLE was most commonly seen in the overlap with DM.

Previous studies have implicated TNF-α inhibitors in the onset and exacerbation of DM symptoms.[6,7] TNF- α inhibition increases type 1 interferon signaling, which plays a key role in DM pathogenesis through activation of antigen-presenting cells and autoantigen production, leading to autoantibody production.[8] Another working hypothesis is that TNF regulates B cell lymphopoiesis through induction of apoptosis in B cell precursors, and TNF inhibition would allow for more B cell proliferation and autoantibody generation.[9] Patient 4 had a history of SCLE/SLE and was treated with adalimumab for concomitant rheumatoid arthritis, after which a rash consistent with DM developed. With the rising use of TNF-α inhibitors in the treatment of dermatologic and rheumatologic diseases, providers must consider the possibility of DM development when prescribing these agents, especially in patients with an existing autoimmune connective tissue disease history.

Recent studies have also indicated a temporal association between new-onset and exacerbation of DM to COVID-19 infection and vaccination.[10,11] Viral infections in general are known triggers for DM. Specifically with COVID-19, exact mechanisms are unclear, but studies have suggested that both infection and vaccine can upregulate type 1 interferon pathways and autoantibody production.[10] In this case series, 3 patients with an initial diagnosis of lupus developed DM symptoms temporally associated with either the COVID-19 vaccine or COVID-19 infection. Given the similarity between pathways involved in COVID-19 and those in the pathogenesis of DM and lupus, it is not surprising that COVID-19 infection and vaccination can also play a role in the development of DM and lupus overlap. Providers should be aware of this potential association when counseling patients with an autoimmune connective tissue disease background so they can optimize medical management.

CD20-inhibitors like rituximab have been found to play a role in flares of SCLE.[12] Suggested mechanisms involve the proinflammatory effects of B cell lysis or the increased homing of T cells to the skin due to decreased B cell-mediated regulation of interleukins 10 and 12.[12] Patient 6 developed SCLE overlapping with DM in the setting of rituximab treatment. Though this patient did not receive rituximab as treatment for DM, rituximab is an available therapeutic option for DM patients. In DM patients being treated with rituximab, developing concomitant SCLE should be considered.

In conclusion, this case series presents patients with DM and lupus overlap, and suggests the role that external triggers like TNF-α inhibitors, COVID-19 vaccine/infection, and anti-CD20 agents can play in the presentation of disease overlap. Providers should be aware of these possible associations to recognize treatment-related risks when managing patients with multiple systemic autoimmune disorders.

Conflicts of interest

VPW has grants from Pfizer, Corbus, Priovant, and CSL Behring, and has consulted for Pfizer, Janssen, Bristol-Myers Squibb, Octapharma, CSL Behring, Corbus, Galderma, Novartis, and Rome Pharmaceuticals.

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