prevalence of rates reported. Other factors that also influence SSI rates (eg, prophylactic antibiotics, anatomic location, wound care practices, and patient comorbidities) were not assessed in this review, thus limiting the ability to draw conclusions in this regard.

Although widespread adoption of the CDC definition is a reasonable suggestion for future studies, this definition permits surgeons to ultimately use their own clinical judgment in defining SSI, inherently limiting its generalizability. Next steps should focus on developing a clear definition of SSI, including length of follow-up, that is not dependent on a surgeon's subjective diagnosis. Once established, future studies in the field of dermatology should use this standardized definition to allow for accurate comparisons of SSI rates among studies.

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Autoantibody profiles in patients' sera associated with distribution patterns of dermatomyositis skin symptoms

To the Editor: Myositis-specific autoantibodies (MSAs) were recently reported to be associated with distinctive clinical features of dermatomyositis (DM). For example, anti–transcriptional intermediary factor 1 (TIF1) γ antibody positivity is highly associated with malignancy, and serum anti-MDA5 antibody is a known marker of rapidly progressive interstitial lung disease (ILD).[1] To decide treatment approaches for patients with DM, identifying serum MSAs or myositis-associated autoantibodies (MAAs) is quite important, although convenient kits for measuring many kinds of MSAs/MAAs are not always available. Some cutaneous symptoms are known to be associated with certain MSAs/MAAs. For example, mechanic's hands and reverse Gottron sign are related to anti–aminoacyl transfer RNA synthetase (anti-ARS) and anti-MDA5 antibodies, respectively; however, they are not always apparent. We tried to find a simple new method for predicting MSA/MMA profiles in patients with DM using cluster analysis of skin symptom distribution patterns.

Skin symptoms and clinical data were retrospectively collected by unified questionnaires from 198 Japanese patients who fulfilled the criteria of Bohan and Peter[2] or those for clinically amyopathic DM.[3] Each MSA (anti–Mi-2, anti-TIF1γ, anti–MDA-5, anti-MJ, or anti-SAE) or MAA (anti-PM/Scl) was detected by in-house enzyme-linked immunosorbent assay (ELISA), and the results of the ELISA were confirmed by immunoprecipitation with recombinants.[4] Anti-ARS was tested by ELISA kits (MBL, Nagoya, Japan). Positive skin symptoms associated with each MSA/MAA are shown in Table I. We grouped skin symptoms with localization to body sites: face/scalp (heliotrope rash, facial erythema, alopecia), trunk/neck (V-neck/shawl, flagellate erythema), and extremities (Gottron sign/papule, mechanic's hands, nailfold erythema, digital ulceration). We analyzed DM skin symptoms by hierarchical clustering using an averaging method and applying the Gower distance to find similar skin symptoms or pattern groups (Supplemental Fig 1; available via Mendeley at https://doi.org/10.17632/nzsjnstn6w.1) by combinations of skin symptom–positive parts and numbers of skin symptoms. This study was approved by the Ethics Committee of our university.

The 198 patients were divided into 5 groups: clusters 1 to 5 (C1-C5), defined as in Fig 1.

Table I. Skin manifestations in the groups of MSA-/MAA-positive patients with DM, n (%)

Cutaneous signs	TIF1γ (n = 41)	Mi-2 (n = 13)	PM/Scl (n = 6)	ARS (n = 27)	MDA5 (n = 47)	SAE (n = 7)	MJ (n = 11)	None (n = 46)
Gottron sign/papules	35 (85)	11 (85)	6 (100)	20 (74)	42 (89)	7 (100)	6 (55)	28 (61)
Nailfold erythema	33 (80)	13 (100)	3 (50)	16 (59)	36 (77)	5 (71)	7 (64)	20 (43)
Digital ulceration	1 (2)	1 (8)	0	0	8 (17)	0	2 (18)	1 (2)
Mechanic's hands	0	1 (8)	2 (33)	7 (26)	11 (23)	1 (14)	0	2 (4)
Heliotrope rash	28 (68)	7 (54)	1 (17)	6 (22)	31 (66)	2 (29)	6 (55)	23 (50)
Facial erythema	20 (49)	5 (38)	3 (50)	3 (11)	17 (36)	3 (43)	4 (36)	13 (28)
Alopecia	1 (2)	1 (8)	0	0	5 (11)	1 (14)	1 (9)	1 (2)
V-neck/shawl sign	27 (66)	6 (46)	3 (50)	3 (11)	22 (47)	4 (57)	5 (45)	8 (17)
Flagellate erythema	8 (20)	1 (8)	0	0	3 (6)	1 (14)	1 (9)	4 (9)

ARS, Aminoacyl transfer RNA synthetase; *MAA*, myositis-associated autoantibodies; *MDA5*, melanoma differentiation-associated gene 5; *MSA*, myositis-specific autoantibodies; *PM/Scl*, polymyositis/scleroderma; *SAE*, small ubiquitin-like modifier activating enzyme; *TIF1*, transcriptional intermediary factor 1.

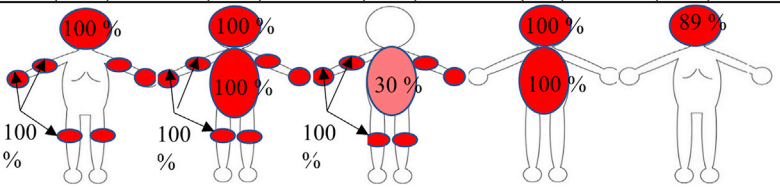
	C1 (n=63)	C2 (n=60)	C3 (n=53)	C4 (n=4)	C5 (n=18)	P value
Definition of the clusters from distribution of skin symptoms						
Antibody profile	Anti-MDA5-dominant pattern	Anti-TIF1γ-dominant pattern	Anti-ARS-dominant pattern	Rare pattern	Autoantibody is not likely detected	
Skin symptoms (No average (SD))	3.1 (1.0)	4.4 (1.0)	2.1 (0.9)	2.5 (0.6)	1.1 (0.6)	
age (years, median (range))	51.0 (1-85)	60.5 (4-84)	58 (27-79)	64.5 (30-79)	50.0 (4-76)	0.177
sex (f) (n (%))	46 (73%)	42 (70%)	36 (68%)	3 (75%)	12 (67%)	0.971
cancer (n (%))	12 (19%)	21 (35%)	10 (19%)		2 (11%)	0.071
autoantibody (n (%))						<.001
TIF1γ (41)	10 (16%)	24 (40%)	5 (9%)		2 (11%)	
Mi-2 (13)	5 (8%)	6 (10%)	2 (4%)			
PM/Scl (6)	2 (3%)	1 (2%)	3 (6%)			
ARS (27)	7 (11%)	1 (2%)	17 (32%)	1 (25%)	1 (6%)	
MDA5 (47)	19 (30%)	17 (28%)	9 (17%)	1 (25%)	1 (6%)	
SAE (7)	3 (5%)	1 (2%)	3 (6%)			
MJ (11)	3 (5%)	4 (7%)	2 (4%)		2 (11%)	
None (46)	14 (22%)	6 (10%)	12 (23%)	2 (50%)	12 (67%)	
myositis (n (%))	40 (63%)	30 (50%)	30 (57%)	1 (25%)	12 (67%)	0.326
ILD (n (%))	32 (51%)	25 (42%)	33 (62%)	3 (75%)	5 (28%)	0.032*
CK (IU/l, median (range))	288.0 (24-12522)	232.5 (23-9760)	428.0 (39-10000)	176.0 (63-272)	193.0 (12-6587)	0.239*

Fig 1. Definition of the clusters from distributions of skin symptoms and clinical characteristics of patients with DM in the skin symptom distribution clusters. C1 is a group of 63 patients with DM whose skin lesions were seen on both the face/scalp and the extremities but not on other body sites. C2 is a group of 60 patients who all had skin lesions on the face/scalp, trunk/neck, and extremities. C3 is a group of 53 patients whose skin lesions manifested on the extremities (100%) and, less frequently, on the trunk/neck (30%). C4 is a group of rare patients (4 patients) whose skin lesions were seen on both the face/scalp and the trunk/neck but on no other body sites. C5 consists of patients who had skin lesions only on the face/scalp (89%) and patients without any skin lesions (11%). *Nine patients in the ILD analysis and 7 patients in the CK analysis were excluded because of missing data. *ARS*, Aminoacyl transfer RNA synthetase; *C*, cluster; *CK*, creatine kinase; *DM*, dermatomyositis; *f*, female; *ILD*, internal lung disease; *MDA5*, melanoma differentiation-associated gene 5; *PM/Scl*, polymyositis/scleroderma; *SAE*, small ubiquitin-like modifier activating enzyme; *SD*, standard deviation; *TIF1*, transcriptional intermediary factor 1.

We compared clinical and laboratory findings among the cluster group by Kruskal-Wallis or chi-square test ($P < .05$). There were differences between the profiles of existing MSA/MAA in each group ($P < .001$). ILD showed a slight difference ($P = .032$) (Fig 1). Of our 198 patients, 40% of the anti-MDA5−positive patients (19/47), 59% of the anti-TIF1γ−positive patients (24/41), and 63% of the anti-ARS−positive patients (17/27) were included in C1, C2, and C3, respectively. No autoantibody was detected in most patients in C5 (67%, 12/18). This suggests we should check serum anti-TIF1γ and anti-ARS antibodies in patients with widespread skin lesions and with an extremities-dominant rash, respectively. More than half of the patients with skin symptoms on only the face/scalp had neither MSAs nor MAAs.

Our cluster groups did not have clinically distinct phenotypes. As DeWane et al.[1] mentioned, cutaneous manifestations sometimes vary and may or may not parallel myositis and/or systemic involvement. For example, skin ulceration is associated with ILD in anti-MDA5−positive patients with DM, but this relationship is not seen in anti-MDA5−negative patients with DM.[5]

Skin symptom distributions may give us clues to narrow down candidate patients with DM for MSA/MAA positivity, but they are not directly helpful for predicting clinical features.

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Low CD4$^+$ T-cell nadir as a preoperative risk factor for squamous cell carcinoma with larger surgical margins during Mohs micrographic surgery among patients infected with HIV

To the Editor: Patients with HIV are uniquely immunosuppressed and have a significantly increased risk of nonmelanoma skin cancer compared with the general population[1,2] despite antiviral therapy.[3] Preoperative prognostic risk factors that may predict the need for extensive Mohs micrographic surgery (MMS) for nonmelanoma skin cancer are not known. The objective of this study was to determine whether a CD4$^+$ T-cell count nadir of less than 200 cells/μL (CD4<200) is an independent risk factor for larger surgical margins for the treatment of squamous (SCC) and basal cell carcinomas (BCC) in patients with HIV.

A case-control retrospective chart review study was conducted on patients with HIV treated with Mohs micrographic surgery (MMS) from 2009 to 2019 at our single tertiary care institution with University of California−San Diego institutional review board approval. Two hundred patients with HIV with BCC or SCC were included in the study. The main outcome measures included final surgical margin and number of MMS stages.

Of the 200 patients in the study, 46 patients had both SCCs and BCCs; 98 had only BCCs, and 56 had only SCCs. Of the patients who had SCCs, 52 of 102 had CD4<200 (51%). There were no differences