School of Medicine at Quinnipiac University, North Haven, Connecticut.[c]

Funding sources: None.

Disclosure: Dr Armstrong has served as an investigator and consultant to AbbVie, Janssen, Lilly, Leo, Novartis, UCB, Ortho Dermatologics, Dermira, Sanofi Genzyme, Regeneron, Bristol-Myers Squibb, Dermavant, and Modernizing Medicine. Ms Sierro, Ms Young, Ms Kassabian, and Mr Wu have no conflicts of interest to declare.

IRB approval status: Reviewed and approved by the University of Southern California IRB (HS-18-00868).

Reprints not available from the authors.

Correspondence to: April W. Armstrong, MD, MPH, University of Southern California, 1975 Zonal Avenue, KAM 510, MC 9034, Los Angeles, CA 90089

E-mail: armstrongpublication@gmail.com

REFERENCES

1. DeBord LC, Patel V, Braun TL, Dao H Jr. Social media in dermatology: clinical relevance, academic value, and trends across platforms. *J Dermatolog Treat*. 2019;30:511-518.
2. Ross NA, Todd Q, Saedi N. Patient seeking behaviors and online personas: social media's role in cosmetic dermatology. *Dermatol Surg*. 2015;41(2):269-276.
3. About Instagram. Available at: https://www.instagram.com/about/us/. Accessed May 2, 2019.
4. About YouTube. Available at: https://www.youtube.com/user/YouTube/about. Accessed May 2, 2019.
5. About Twitter. Available at: https://about.twitter.com/en_us/lets-go-twitter.html. Accessed May 2, 2019.

https://doi.org/10.1016/j.jaad.2020.03.001

Analysis of clinical characteristics of drug-induced cutaneous lupus erythematosus in men

To the Editor: Cutaneous lupus erythematosus (CLE) can be divided into acute CLE, subacute CLE, and chronic CLE. Pathogenesis for CLE includes genetic predisposition, autoimmunity, and drug exposure.[1] Although previous studies have evaluated the incidence and clinical characteristics of CLE,[2-4] to our knowledge, none has focused on CLE in men and, in particular, on the incidence of drug-induced CLE (DICLE) in men.

This retrospective study describes the clinical characteristics of men diagnosed with CLE and systemic lupus erythematosus with cutaneous features at Duke University Medical Center between 2007 and 2017 and specifically compares the patients with DICLE to those with non-DICLE. Clinical characteristics and laboratory test results were obtained by chart review. Statistical analyses were performed with JMP, version 13.0 (SAS, Cary, NC). Duke University's institutional review board approved this protocol (no. 00084622).

In all, 31 men were diagnosed with CLE (Table I). Nine out of 31 patients (29%) were diagnosed with DICLE based on disappearance of symptoms after discontinuation of the offending drugs, including antihypertensive medications and proton pump inhibitors. The mean age at onset of CLE overall was 43 years (range, 8-75 years) but was higher among patients with DICLE compared to patients with non-DICLE (62 vs 36 years old; $P < .0001$). Men with DICLE were more likely to be white ($P = .036$) and to have subacute CLE and bullous CLE ($P = .020$ and $P = .022$, respectively). No African American men in our cohort had DICLE but comprised the majority (55%) of non-DICLE male patients. The mean drug-to-symptom onset time among DICLE patients was 6.5 weeks (range, 3.8-12.5 weeks). The histopathology in DICLE was less likely than in non-DICLE to show interface dermatitis ($P = .027$). There was no significant difference between the 2 groups in areas of involvement, systemic features, or autoantibody positivity.

There are limitations to our study. Our sample size is small because CLE occurs less frequently in men. Second, the diagnostic criteria of DICLE are not clearly defined. In our cohort, the diagnosis was based on symptomatic improvement after drug withdrawal, potentially underestimating the true incidence of DICLE in men. Finally, there is heterogeneity in histologic features and autoantibodies tested during the study period.

The incidence of DICLE in our cohort is 29%, which is higher than rates previously reported in both sexes, including an incidence of 12% by Marzano et al.[4] This may in part be due to men having less likelihood of idiopathic autoimmunity than women. In addition, elderly men frequently take antihypertensive medications.[1] Our report is consistent with previous studies that showed that, when compared with patients with non-DICLE, patients with DICLE had the following features: (1) older age at onset; (2) median drug-to-onset time of approximately 4-8 weeks; and (3) no specific autoantibody pattern associated with DICLE.[2-4] Moreover, our cohort suggests that interface dermatitis is observed less commonly in DICLE than in non-DICLE. Furthermore, in the southeastern United States, the overall CLE incidence is 3- to 5-fold higher in African American patients than in white patients of both sexes.[5] Interestingly, African American men in our cohort had only non-DICLE.

Table I. Comparison of clinical characteristics of men with DICLE versus non-DICLE who were seen in the dermatology and rheumatology clinics at the Duke University Medical Center between November 1, 2007, and October 31, 2017

Metric	Full cohort (N = 31)	DICLE (n = 9)	Non-DICLE (n = 22)	*P* value*
Age at onset, y, mean (range)	43 (8-75)	62 (36-75)	36 (8-66)	**<.0001**[†]
Percentage of total	—	29	71	
Mean time to onset, y, mean (range)	—	6.5 (3.8-12.5)	—	
Offending agents, n (%)				
Anti-HTN medications	—	7 (78)[‡]	—	
PPIs	—	2 (22)	—	
Race/ethnicity, n (%)				
White	15 (48)	7 (78)	8 (36)	**.036**
African American	12 (39)	0 (0)	12 (55)	**.0047**
Hispanic	4 (13)	2 (22)	2 (9)	.32
Areas of involvement, n (%)				
Sun exposed	7 (23)	2 (22)	5 (23)	.96
Widespread	9 (29)	2 (22)	7 (32)	.60
Head and neck	8 (26)	1 (11)	6 (27)	.38
Upper limbs	5 (16)	2 (22)	4 (18)	.81
Lower limbs	1 (3)	1 (11)	0 (0)	.11
Back	1 (3)	1 (11)	0 (0)	.11
Subtypes, n (%)				
SCLE	14 (45)	7 (78)	7 (32)	**.020**
Discoid	13 (42)	0 (0)	13 (59)	**.0025**
Tumid	2 (7)	0 (0)	2 (9)	.32
Bullous	2 (7)	2 (22)	0 (0)	**.022**
Systemic symptoms, n (%)				.15
With systemic symptoms[§]	13 (42)	2 (22)	11 (50)	
Without systemic symptoms	18 (58)	7 (78)	11 (50)	

	Full cohort (n = 21)	DICLE (n = 9)	Non-DICLE (n = 12)	
Histologic features, n (%)				
Interface dermatitis	8 (38)	1 (11)	7 (58)	**.027**
Lichenoid dermatitis	6 (32)	4 (44)	2 (17)	.16
Spongiotic dermatitis	2 (10)	2 (22)	0 (0)	.086
Lymphocytic infiltrate	3 (14)	0 (0)	3 (25)	.11
Subepidermal bullae	2 (10)	2 (22)	0 (0)	.086

	Full cohort	DICLE	Non-DICLE	
Autoantibody panel, n positive/n total (%)				
ANA$^+$	20/27 (74)	5/7 (71)	15/20 (75)	.85
Anti-Ro/SS-A$^+$	8/20 (40)	1/5 (20)	7/15 (47)	.29
Anti-La/SS-B$^+$	2/20 (5)	1/5 (20)	1/15 (7)	.39
Anti-SM$^+$	5/20 (25)	0/5 (0)	5/15 (33)	.14
Anti-RNP$^+$	7/21 (33)	0/5 (0)	7/16 (44)	.07
Anti-dsDNA$^+$	7/20 (35)	1/5 (20)	6/15 (40)	.42

ANA, Antinuclear antibody; *DICLE*, drug-induced cutaneous lupus erythematosus; *dsDNA*, double-strand DNA; *HTN*, hypertensive; *PPI*, proton-pump inhibitor; *RNP*, ribonucleoprotein; *SCLE*, subacute lupus erythematosus; *Sm*, Smith.

*Bold type indicates statistical significance.

[†]Student *t* test. All other statistical comparisons were Pearson chi-square test.

[‡]Anti-HTN medications include ACE inhibitors (captopril and enalapril), 4 of 7; beta blockers (sotalol and labetalol), 2 of 7; and calcium channel blocker (amlodipine), 1 of 7.

[§]Systemic symptoms include nephritis, joint involvement, pericarditis, and thrombocytopenia.

In sum, physicians should remain vigilant about DICLE, particularly when elderly white male patients present with CLE.

Amy J. Petty, BS,[a] *Adela R. Cardones, MD, MHSc,*[b,c] *and Anne L. Marano, MD*[b]

From the School of Medicine, Duke University, Durham, North Carolina[a]*; Department of Dermatology, Duke University, Durham, North Carolina*[b]*; and Department of Dermatology, Durham Veterans Affairs Medical Center, North Carolina.*[c]

Funding sources: None.

Disclosure: Dr Anne Marano is a principal investigator for a study for Viela Bio, a phase I trial of VIB7734 in patients with systemic lupus erythematosus, cutaneous lupus erythematosus, Sjögren syndrome, systemic sclerosis, polymyositis, and dermatomyositis. MS Petty and Dr Cardones have no conflicts of interest to declare.

IRB approval status: Reviewed and approved by the Duke University IRB (approval 00084622).

Reprints not available from the authors.

Correspondence to: Anne L. Marano, MD, 234 Crooked Creek Parkway, Ste 300, Durham, NC 27713

E-mail: Anne.marano@duke.edu

REFERENCES

1. Hejazi EZ, Werth VP. Cutaneous lupus erythematosus: an update on pathogenesis, diagnosis and treatment. *Am J Clin Dermatol*. 2016;17:135-146.
2. Guicciardi F, Atzori L, Marzano AV, et al. Are there distinct clinical and pathological features distinguishing idiopathic from drug-induced subacute cutaneous lupus erythematosus? A European retrospective multicenter study. *J Am Acad Dermatol*. 2019;81:403-411.
3. Laurinaviciene R, Sandholdt LH, Bygum A. Drug-induced cutaneous lupus erythematosus: 88 new cases. *Eur J Dermatol*. 2017;27:28-33.
4. Marzano AV, Lazzari R, Polloni I, Crosti C, Fabbri P, Cugno M. Drug-induced subacute cutaneous lupus erythematosus: evidence for differences from its idiopathic counterpart. *Br J Dermatol*. 2011;165:335-341.
5. Drenkard C, Parker S, Aspey LD, et al. Racial disparities in the incidence of primary chronic cutaneous lupus erythematosus in the southeastern US: the Georgia Lupus Registry. *Arthritis Care Res (Hoboken)*. 2019;71:95-103.

https://doi.org/10.1016/j.jaad.2020.03.002

A cross-sectional report on melasma among Hispanic patients: Evaluating the role of oral tranexamic acid versus oral tranexamic acid plus hydroquinone

To the Editor: Oral tranexamic acid (TA) has been described as a game changer as a solo agent in the treatment for refractory moderate to severe melasma.[1] Its therapeutic mechanism has been postulated to address the vascular component in melasma. However, optimal dose, duration, and studies on depigmenting creams are limited.[2] A retrospective treatment outcome analysis, at a single center over a 1-year period (June 2018 to June 2019), of patients with melasma receiving oral TA 650 mg daily ± hydroquinone (HQ) 4% cream was performed. This report describes the main clinical characteristics and impact on patients' quality of life (QOL). The primary outcome was the modified Melasma Area and Severity Index (mMASI) score, and impact on QOL was assessed using the Spanish Melasma on Quality of Life questionnaire (Sp-MELASQOL). Patients were evaluated to exclude the risk of thrombosis. The mMASI was calculated by 2 dermatologists blinded to treatment groups. Pearson correlation, paired *t* test, and unpaired *t*-test with Welch's correction were used. A *P* value of less than .05 was considered statistically significant.

Fifty-three patients' charts with sufficient documentation on progress and the degree of improvement at weeks 8 and 20 were included. The main background clinical characteristics are summarized in Table I. Twenty-seven patients (50.94%) received oral TA 650 mg daily (mean baseline mMASI score, 8.25), and 26 (49.05%) received oral TA 650 mg daily plus HQ 4% cream (mean baseline mMASI score, 8.20). At week 20 of treatment, there was a 46% reduction in mMASI score in the TA group versus 61% in the TA plus HQ 4% cream group; this difference was significant ($P = .048$). Mild adverse effects to TA were reported in 7 patients. Notably, there was a 49% reduction in the Sp-MELASQOL score in the combined group compared with 29% with oral TA alone. The overall clinical response rate observed aligns with that reported in previous studies. Zhu et al,[3] in a randomized study, reported no significant differences in the MASI and melanin index among 500; 750; 1,000; or 1,500 mg oral TA. Del Rosario et al,[2] in a randomized study of 39 patients taking 250 mg TA twice daily, reported a 49% reduction in mMASI score compared with 18% in the control placebo group. To our knowledge, this is the first treatment outcome analysis among Hispanic patients. Our data indicated that combined therapy appears superior to oral TA alone. Padhi and Pradhan reported a more significant improvement in MASI score with oral TA 250 mg twice daily in conjunction with a triple combination depigmented cream compared with the triple combination alone,[4] and Karn et al,[5] in a prospective randomized controlled trial, found a significantly higher clinical improvement with oral TA 250 mg twice daily combined with topical HQ versus topical HQ alone. Our data support the use of oral TA plus HQ 4% as a combined therapy for moderate to severe melasma with better clinical and QOL results.

Ileana E. Arreola Jauregui, MD,[a] Gabriel Huerta Rivera, MD,[a] Manuel Soria Orozco, MD,[b] Silvia