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Adverse cutaneous drug reactions with antimalarials in cutaneous lupus and dermatomyositis: A retrospective cohort study

To the Editor: Aminoquinoline antimalarials are first-line systemic treatment for cutaneous lupus erythematosus (CLE) and dermatomyositis (DM). Their mechanism of action relates to their immunomodulatory, anti-inflammatory, and ultraviolet protective properties. Antimalarials can improve pruritus, photosensitivity, and erythema and eliminate the need for corticosteroid/immunosuppressant treatment in some patients with DM and CLE.[1]

Antimalarials may cause adverse cutaneous drug eruptions (ACDRs). Such reactions are uncommon in patients with CLE, whereas the opposite has been observed in patients with DM, in whom they have been reported with an incidence of 13% to 39%.[2-4]

We performed a retrospective review of adult patients with DM and CLE who were seen at the University of Utah Department of Dermatology from January 2013 to January 2018 to evaluate the frequency of antimalarial ACDRs. We included patients who were taking hydroxychloroquine (HCQ) or chloroquine and had at least 3 months of follow-up data noting reaction timing and severity. ACDRs were diagnosed by the treating physician on the basis of history, physical examination, and/or skin biopsy.

We identified 180 patients with DM or CLE (Table I). Demographic characteristics were similar, with the exception of age (mean age, 54 years with DM vs 48 years with CLE [$P = .03$]). The frequency of ACDRs was similar in both groups: 2 of 44 in the group with DM (5%) versus 4 of 136 in the group with CLE (4%) ($P = .68$). All eruptions were associated with HCQ use. More patients with DM were taking immunosuppressants (75% of those with DM vs 26% of those with CLE [$P < .001$]); however, diagnosis did not make a difference in number of ACDRs, even accounting for concomitant immunosuppressive therapy ($P = .83$).

ACDRs occurred 5 to 14 days after initiation of HCQ treatment and were non–life-threatening. Eruptions were characterized as lichenoid, urticarial, or exanthematous and resolved after discontinuation of HCQ treatment. One patient required a brief prednisone taper. No patients were hospitalized.

Two patients were later rechallenged with HCQ without ACDR recurrence. Successful rechallenge has been reported in patients with mild ACDRs to HCQ.[5] There are also reports of successful chloroquine treatment in patients with ACDRs to HCQ, suggesting the feasibility of alternate antimalarial therapy.[1]

The incidence of antimalarial ACDRs may be overestimated in the literature, and it varies between institutions. One possible explanation for the discrepancies may be differences in chemical compositions of generic HCQ formulations. Alternatively, rates of ACDRs may be related to dermatomyositis-specific autoantibody profiles. Ethnic/geographic differences in the frequencies of such autoantibodies are well documented. One recent report suggests that patients with small ubiquitin-like modifier-1 activating enzyme autoantibodies might be at increased risk of antimalarial ACDRs.[4] Interestingly, there is no evidence that patients with DM are at greater risk of ACDRs to other drugs or to other side effects of antimalarials than are patients with CLE. Further epidemiologic study is warranted.

Limitations to our study include its retrospective nature, a small sample size owing to the rarity of these diseases, and a single-institution population consisting of a primarily white demographic. DM-specific autoantibody testing was not available during much of the study period and was therefore not assessed.

We did not observe an increased frequency of antimalarial ACDRs in our patients with DM compared with in our patients with CLE. Antimalarial drugs should be offered to patients with DM and CLE. In patients who develop mild ACDRs, a re-challenge should be considered.

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Table I. Demographics and clinical presentation of the cohort

Variable	All patients (N = 180)	Cutaneous LE (n = 136)	Dermatomyositis (n = 44)	*P* value
Sex, n (%)*				.56
Female	146 (81%)	109 (80%)	37 (84%)	
Male	34 (19%)	27 (20%)	7 (16%)	
Age at evaluation, y†				.03
Mean (SD)	50 (16)	48 (17)	54 (15)	
Min/max	(20, 88)	(20, 88)	(20, 84)	
Ethnicity, n (%)‡				.72
Hispanic/Latino	18 (10%)	15 (11%)	3 (7%)	
Not Hispanic/Latino	151 (84%)	112 (82%)	39 (89%)	
Unknown	11 (6%)	9 (7%)	2 (5%)	
Race, n (%)‡				>.99
White	145 (81%)	108 (79%)	37 (84%)	
Black or African American	1 (1%)	1 (1%)	0 (0%)	
Asian	7 (4%)	6 (4%)	1 (2%)	
American Indian and Alaska Native	3 (2%)	2 (1%)	1 (2%)	
Native Hawaiian and other Pacific Islander	1 (1%)	1 (1%)	0 (0%)	
Other	13 (7%)	10 (7%)	3 (7%)	
Unknown	10 (6%)	8 (6%)	2 (5%)	
Medication§				
Hydroxychloroquine‡	179 (99%)	135 (99%)	44 (100%)	>.99
Chloroquine‡	7 (4%)	6 (4%)	1 (2%)	>.99
Previous exposure to antimalarial, n (%)*	26 (14%)	21 (15%)	5 (11%)	.50
Concomitant immunosuppression, n (%)*	68 (38%)	35 (26%)	33 (75%)	<.001
Drug reaction‡	7 (4%)	5 (4%)	2 (5%)	.68
Drug reaction (controlling for concomitant immunosuppression)‖				.83

The cutaneous LE group was composed of 92 patients with discoid LE, 25 with subacute cutaneous LE, 3 with LE tumidus, and 16 with acute cutaneous LE. The dermatomyositis group consisted of 7 patients with clinically amyopathic dermatomyositis and 37 with classic dermatomyositis.

LE, Lupus erythematosus; *SD*, standard deviation.

*Chi-square test used.

†*t* Test used.

‡Fisher exact test used.

§Six patients who were first treated with chloroquine and were switched to hydroxychloroquine because of gastrointestinal upset, tinnitus, or a national shortage of chloroquine. Only 1 patient was treated with chloroquine exclusively.

‖Cochran-Mantel-Haenzsel test used.

Reprints not available from the authors.

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Post–nail procedure analgesia: A randomized control pilot study

To the Editor: For success in nail surgery, proper anesthesia is essential. Postoperative pain after nail surgery is a common complication. Lidocaine is the most widely used anesthetic in nail surgery because of its safety profile and faster onset of action.[1]