Further-more, the propensity score–matching analyses were performed to eliminate potential bias. This study was exempted from full review by the Tri-Service General Hospital Institutional Review Board.

Among the total selected patients with psoriasis, the mean and median time intervals from cohort entry date to psoriasis incidence date were 850 and 829 days, respectively. The log-rank test revealed that long-term BZRA users had a higher likelihood than short-term BZRA users and nonusers in receiving a psoriasis diagnosis ($P < .001$; Fig 1). The adjusted hazard ratio (aHR) for psoriasis for long-term BZRA users was 1.48 (95% confidence interval 1.34-1.63) compared with nonusers (Table I), whereas the aHR for psoriasis in short-term BZRA users was 1.23 (95% confidence interval 1.17-1.29) compared with nonusers. In addition, Table I summarizes the HRs of psoriasis incidence among BZRA users and their matched nonusers. The findings of this study demonstrate that long-term BZRA users were 1.50 times more likely to develop psoriasis than their propensity score–matched nonusers after adjustments were made, whereas short-term BZRA users had a significantly greater risk of psoriasis than their matched nonusers (aHR 1.30).

Nevertheless, several limitations should be taken into account in this study. First, the data sets used provide no information regarding genetic factors, sunlight exposure, body mass index, or smoking habits. Second, the National Health Insurance program database had no records regarding the Psoriasis Area Severity Index. Therefore, we could not estimate the influence of psoriasis severity.

In conclusion, this study shows that long- and short-term BZRA use could increase the risk of psoriasis. Consequently, clinicians should assess the risks and benefits of BZRA use. More direct biologic evidence is required to validate the connection between BZRA use and psoriasis.

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Clinicopathologic comparison of Rowell syndrome, erythema multiforme, and subacute cutaneous lupus erythematosus

To the Editor: Rowell syndrome is characterized by erythema multiforme–like lesions with serologic and historical evidence of lupus erythematosus (LE).[1] Classification of Rowell syndrome remains controversial, given overlapping clinical features of erythema multiforme and cutaneous LE (CLE).[2] Our objective was to identify histologic and immunohistochemical findings that support classification of Rowell syndrome because current definitions lack these criteria.[3]

Table I. Comparison of RS, SCLE, and EM by clinical, serologic, histologic and immunohistochemical findings

Characteristic or finding	RS (5 patients; 8 biopsies)	SCLE (4 patients; 7 biopsies)	EM (5 patients; 5 biopsies)	RS and SCLE, *P* value	RS, SCLE, and EM, *P* value
Age, y, mean ± standard deviation	53 ± 23.15	62.8 ± 16.44	52.8 ± 16.07		
Female sex	80 (4/5)	100 (4/4)	80 (4/5)	1	1
History of LE*	100 (4/4)	75 (3/4)	0 (0/5)	1	**.0047**
Targetoid lesions†	100 (4/4)	0 (0/4)	100 (5/5)	**.0286**	**.0028**
Mucosal involvement	0 (0/4)	0 (0/4)	40 (2/5)	1	.2821
Implicated medication	0 (0/4)	0 (0/4)	20 (1/5)	1	1
Speckled antinuclear antibody pattern	50 (2/4)	67 (2/3)	0 (0/2)	1	.5238
Lesional direct immunofluorescence‡	100 (1/1)	100 (1/1)	0 (0/1)	1	1
Full-thickness epidermal necrosis	50 (4/8)	0 (0/7)	80 (4/5)	.077	**.017**
Parakeratosis	25 (2/8)	85.7 (6/7)	20 (1/5)	**.041**	**.034**
Dermal eosinophils	0	0	20 (1/5)	1	.250
Periadnexal lymphocytic infiltrate§	87.5 (7/8)	85.7 (6/7)	0	1	**.003**
≥10% $CD123^+$ cells¶	62.5 (5/8)	57.1 (4/7)	20 (1/5)	1	.395
Dermal-predominant CD123 pattern	42.9 (3/7)	83.3 (5/6)	40 (2/5)	.266	.292
Intraepidermal $CD123^+$ plasmacytoid DCs	100 (7/7)	66.7 (4/6)	100 (5/5)	.192	.163
Periadnexal $CD123^+$ plasmacytoid DCs	100 (7/7)	66.7 (4/6)	40 (2/5)	.192	**.049**

Values are % (n/total) except where indicated. Significant values are in bold.
DCs, Dendritic cells; *EM*, erythema multiforme; *LE*, lupus erythematosus; *RS*, Rowell syndrome; *SCLE*, subacute cutaneous lupus erythematosus.
*History of systemic or cutaneous LE.
†≥2 zones of color.
‡Granular deposition of IgG in the lower epidermis along the basement membrane zone, with an antinuclear antibody pattern in keratinocytes. Granular deposition of IgM, IgA, and complement C3 and C5b-9 along the basement membrane zone.
§Perifollicular or perieccrine.
¶Percentage of inflammatory cells positive for antibody staining in a single histologic section.

CD123, which labels pathogenic plasmacytoid dendritic cells in LE, might be useful in this context.[4,5]

After institutional review board approval, the archives of the University of Florida and Inform Diagnostics were queried for diagnoses of Rowell syndrome (8 biopsies from 5 patients), subacute CLE (SCLE, 7 biopsies from 4 patients), and erythema multiforme (5 biopsies from 5 patients) rendered during November 2016-June 2018; an additional single indeterminate case was also identified. Clinical features, serology, histology, and CD123 antibody staining for plasmacytoid dendritic cells were compared between Rowell syndrome, SCLE, and erythema multiforme cases after a blinded retrospective histologic review. A Fisher's exact test was used to compare histologic variables and CD123 staining patterns among Rowell syndrome, SCLE, and erythema multiforme cases. Two-tailed *P* values $<.05$ were deemed statistically significant.

Rowell syndrome and SCLE were characterized by female predominance, antecedent history of LE, lack of causative medication, absence of mucosal involvement, positivity for rheumatoid factor, and speckled antinuclear antibody pattern with anti-Ro (SS-A) positivity (Table I). Targetoid plaques were observed in Rowell syndrome (4/4) but not SCLE (0/4). Conversely, anti-La (SS-B) positivity was observed in SCLE (2/3) but not in Rowell syndrome (0/4), which might reflect variable assay sensitivity for the detection of this autoantibody.[6] Direct immunofluorescence was positive in Rowell syndrome (1/1) and SCLE (1/1) but negative in erythema multiforme (0/1). Summarized in Table I, the common and unifying histologic features of Rowell syndrome (Fig 1) and SCLE (Figure 2; available at https://data.mendeley.com/datasets/bwswc4h9yb/1) include periadnexal lymphocytic infiltrates ($P = 1$), absence of dermal eosinophils ($P = 1$), and CD123 positivity of ≥10% of the inflammatory infiltrate ($P = 1$). Distinction of Rowell syndrome from erythema multiforme (Figure 3; available at https://data.mendeley.com/datasets/bwswc4h9yb/1) can be supported by periadnexal lymphocytic infiltrates ($P = .003$) and periadnexal $CD123^+$ plasmacytoid dendritic cells ($P = .049$). Using these features, the indeterminate case was reclassified as Rowell syndrome. Several features did not aid classification ($1 > P > .05$), including interface tissue reaction subtype (lichenoid or cell poor), deep perivascular lymphocytic infiltrates, periadnexal plasmacellular aggregates, mucin deposition (by Alcian blue staining), and the predominant CD123 staining pattern (epidermal or dermal).

This series is limited by its small sample size, retrospective nature, and lack of complete clinical data for all patients. Previously dependent

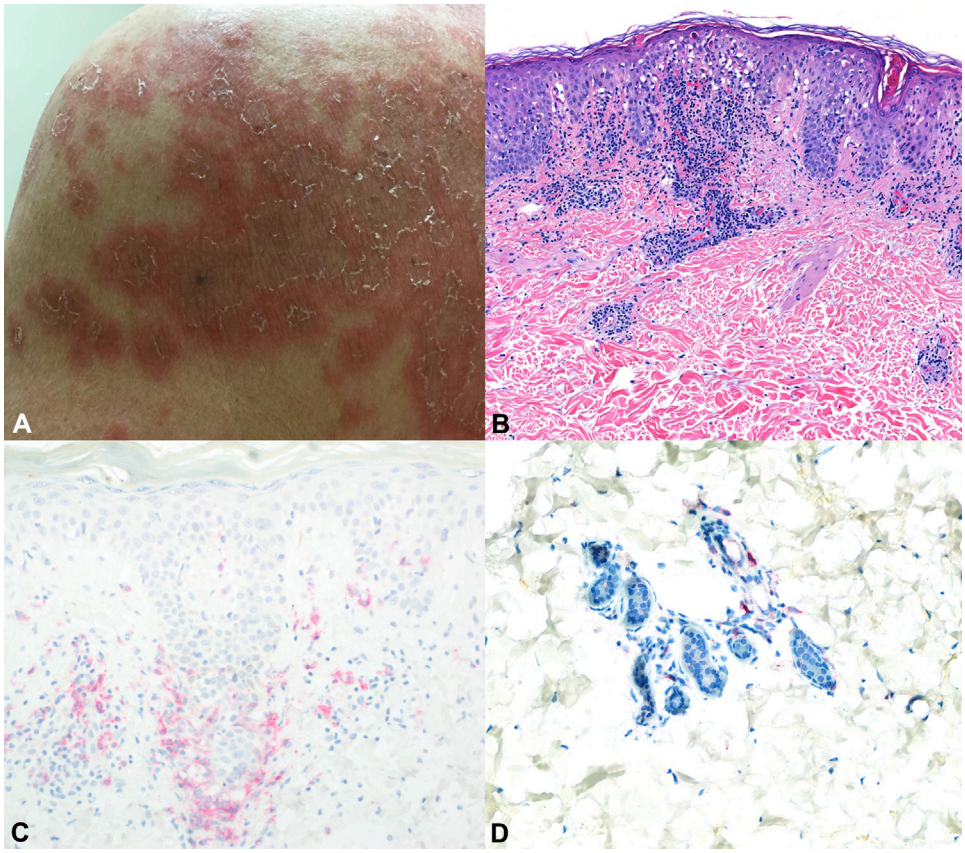

Fig 1. Rowell syndrome. **A**, Targetoid plaques with dusky centers and peripheral erythema. **B**, Lichenoid interface dermatitis with keratinocyte necrosis, perivascular lymphocytic infiltrates, and mucin. **C**, Perifollicular and intraepithelial plasmacytoid dendritic cells, representing >10% of the inflammatory infiltrate. **D**, Perieccrine plasmacytoid dendritic cells. (**B**, Hematoxylin-eosin stain; original magnification: ×100; **C** and **D**, CD123 stain with red chromagen; original magnification: ×200.)

on clinical history, serology, and nonspecific clinical morphology, diagnosis of Rowell syndrome might be improved by histologic detail and CD123 staining. Unifying features of Rowell syndrome and SCLE include antecedent history of LE, absence of mucosal involvement, serology (antinuclear antibody, anti-Ro, and rheumatoid factor), direct immunofluorescence positivity, periadnexal lymphocytic infiltrates, absence of dermal eosinophils, and $CD123^{+}$ inflammatory cells only when comprising ≥10% of the inflammatory infiltrate. In the context of targetoid clinical morphology, this feature aids in the distinction of Rowell syndrome from erythema multiforme. These clinicopathologic findings support inclusion of Rowell syndrome in the spectrum of CLE and provide therapeutic or prognostic benefit to patients with diagnoses previously identified as Rowell syndrome or erythema multiforme.

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Increased interest in sunless tanning versus tanning beds in the United States: A Google Trends analysis

To the Editor: Tanning became fashionable in 1920s, when Coco Chanel had a suntan after returning from vacation, and it still remains popular. Despite strong evidence for its association with skin cancer and aging, altering the perception of tanning and its dangers has been difficult so far. Therefore, the American Academy of Dermatology has suggested the use of artificial self-tanning products containing dihydroxyacetone for individuals who want to achieve a bronze look.[1] We aimed to evaluate how this suggestion and the warnings about the dangers of UV tanning have changed the interest in 2 tanning methods and sunscreen use by using Google Trends (Google, Mountainview, CA). Google Trends has been used for describing interest in search terms.[2,3] We extracted and quantified searches for *tanning bed*, *spray tan*, and *sunscreen* to indicate popular trends. Queries from the United States for years 2004 through 2019 were included in the analysis. Data presented as search volume index, ranging from 0 (for no searches) to 100 (for the peak of the most populous search). Every year, interest in sunscreen peaked in June. Interest in sunscreen increased during the period studied (β coefficient, 0.05; 95% confidence interval [CI], 0.00-0.10; $P = .057$). As in prior literature, the 2 tanning-related searches peaked in spring, suggesting a greater interest in the prospect of summer.[3] This seasonal trend was also noted in southern US states with continuous sunlight. The search volume index was higher for *tanning bed* than for *spray tan* in 2004 (Fig 1). Regression analysis showed a significant increase over time for the term *spray tan* ($\beta = 0.11$; 95% CI, 0.09-0.13; $P = .001$) but no change for *tanning bed* ($\beta = 0.01$; 95% CI, 0.00-0.03; $P = .108$). This resulted in a greater mean number of searches for *spray tan* after 2011. Being tan is often misunderstood as being less sun-sensitive, even when the tan is due to the application of dihydroxyacetone (which provides a sun protection factor of 3 on the day of application).[1,4] Emphasis should be given to the fact that a spray tan does not prevent sun damage and sunburns. Regular consultations about photoprotection, including sunscreen use, staying in shade, and wearing sun-protective clothing all year long, are necessary to achieve compliance with dermatology recommendations.

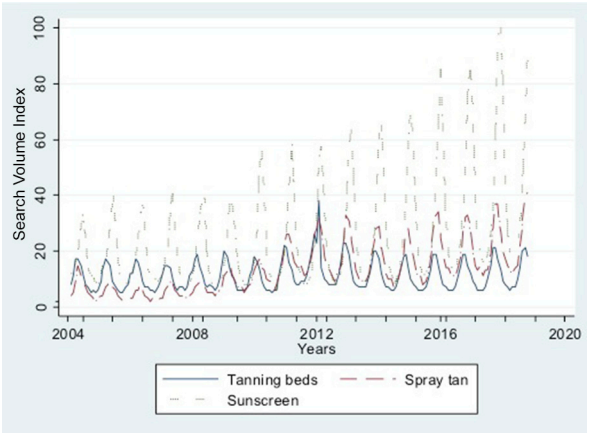

Fig 1. Google Trends for *tanning beds*, *spray tan*, and *sunscreen*, 2004 through 2019 in the United States. Data are presented as search volume index, ranging from 0 (for no searches) to 100 (for the peak of the most populous search).

Based on these metrics, the popularity of looking tan has not decreased. This is reasonable because beauty standards are affected by the media, which still promotes a bronzed appearance.[5] Considering how the tanning trend began, the media might be a way for people to start loving their natural skin color. However, given the fact that light-skinned people are still pursuing a tan appearance, the greater interest in alternatives that do not involve ultraviolet radiation could be a positive step.

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