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Trichoscopic findings of discoid lupus erythematosus alopecia: A cross-sectional study

To the Editor: Trichoscopy may allow us to diagnose alopecia caused by discoid lupus erythematous.[1] The aim of our study was to systematically describe the trichoscopic findings in a set of patients with discoid lupus erythematous on the scalp.

We designed an observational cross-sectional multicenter study approved by the local institutional review board. Inclusion criteria were patients with discoid lupus erythematous alopecia confirmed by skin biopsy. Patients with less than 1 year of follow-up were excluded. We collected epidemiologic data (age, sex, and autoimmune comorbidities), clinical presentation, duration of the disease (5 years or less, 6-10 years, and 11 years or more), and treatments received. Dry trichoscopy was performed with FotoFinder medicam 1000 (Fotofinder Systems Gmbh, Bad Birnbach, Germany). Vessels were evaluated with immersion fluid. For comparison between groups, we used the χ^2 test, as appropriate. All given P values are 2-tailed and $P < .05$ indicates statistical significance. All analyses were performed with SPSS (version 23.0) statistical software package.

Thirty-seven patients with a total of 55 lesions were analyzed. Thirty patients were women (89%) with a mean age of 54.4 years (range 25-90 years). The commonest Fitzpatrick's phototype was III (70.3%; range I-V). Thirteen patients had autoimmune conditions associated (35%), 12 patients had positive antinuclear antibody results (32.4%), and 4 patients had systemis lupus erythematosus (10.8%). The most frequent location of alopecia patches was the parietal region (19 patients; 45.2%), followed by the occipital and temporal region (10 [23.8%] and 9 patients [21.4%], respectively). The mean duration of the disease was 6.4 years (range 0.2-30 years). The treatments received were topical corticosteroids (18 patients; 48.6%), intralesional triamcinolone (10 patients; 27%), and oral hydroxychloroquine (4 patients; 10.8%). Three patients received all treatments simultaneously.

The trichoscopic findings are showed in Table I. Patients with 5 or less years' duration of discoid lupus erythematous presented incontinentia pigmenti signs ($P = .013$, Supplementary Figure 1, available on Mendeley at https://doi.org/10.17632/h69m9nwf5d.1) and thin vascular structures ($P = .002$) more frequently. Long-standing lesions presented an increased number of shiny white structures ($P = .01$). Patients with positive antinuclear antibody results showed a thin vascular pattern more frequently ($P = .02$). No statistically significant associations were found with the rest of the clinical variables or treatments received.

Trichoscopic findings of scalp discoid lupus erythematous have been described before in different groups of patients. Our study includes uncommon trichoscopic findings, such as red dots or "red spider on yellow dots,"[2,3] and establishes their frequency (14.5% and 7.3%, respectively).

Discoid lupus erythematous dermoscopic features have been previously correlated with the duration of the disease.[4] Telangiectatic vessels were found to be more common in older lesions. However, previous study included discoid lupus erythematous cases of less than 2 years of evolution, whereas our patients had a mean time of evolution of 6.4 years. In addition, our study was focused on trichoscopic findings of lupus alopecia, so data between studies are difficult to compare. We found a significant increase of shiny white structures in long-lasting discoid lupus erythematous lesions (chrysalides and rosettes) (Fig 1). They have been correlated to stromal alteration and fibrosis.[5] We suggest that they could be a diagnostic clue for long-

Table I. Trichoscopic findings from patches of alopecia of discoid lupus erythematous distributed by the time of evolution of the disease (n = 55)

Trichoscopic findings, no. (%)	5 years or less	6 to 10 years	More than 10 years	Total	*P*
Hair shafts and follicular openings					
Pili torti	2 (3.6)	1 (1.8)	1 (1.8)	4 (7.3)	>.05
Broken hairs	2 (3.6)	3 (5.4)	3 (5.4)	8 (14.5)	>.05
Black dots	3 (5.4)	8 (14.5)	4 (7.3)	16 (29.1)	>.05
Yellow dots	4 (7.3)	4 (7.3)	3 (5.4)	11 (20)	>.05
Red dots	5 (9.1)	3 (5.4)	0	8 (14.5)	>.05
"Red spider on yellow dots"	3 (5.4)	0	1 (1.8)	4 (7.3)	>.05
Keratotic follicular plugs	5 (9.1)	8 (14.5)	6 (10.9)	19 (34.5)	>.05
Perifollicular and interfollicular findings					
Perifollicular erythema	3 (5.4)	2 (3.6)	3 (5.4)	8 (14.5)	>.05
Perifollicular scaling	12 (21.8)	15 (27.3)	19 (34.5)	46 (83.6)	>.05
Incontinentia pigmenti signs	16 (29.1)	14 (25.5)	3 (5.5)	33 (60)	.013
Scattered brown discoloration	16 (29.1)	14 (25.5)	3 (5.5)	33 (60)	
Blue-gray dots	8 (14.5)	4 (7.3)	0	12 (21.8)	
White interfollicular scaling	14 (25.5)	9 (16.4)	12 (21.8)	35 (63.6)	>.05
Yellowish interfollicular scaling	0	1 (1.8)	2 (3.6)	3 (5.4)	>.05
Splinter hemorrhage	3 (5.4)	0	1 (1.8)	4 (7.3)	>.05
Erosions or ulcers	0	0	1 (1.8)	1 (1.8)	>.05
Vascular pattern					
Thin vascular pattern*	10 (18.2)	8 (14.5)	4 (7.3)	22 (40)	.002
Coiled capillary loops	1 (1.8)	0	1 (1.8)	2 (3.6)	
Elongated capillary loops	0	0	2 (3.6)	2 (3.6)	
Thin arborizing vessels	9 (16.4)	8 (14.5)	2 (3.6)	19 (34.5)	
Thick vascular pattern†	11 (20)	10 (18.2)	10 (18.2)	31 (56.4)	>.05
Giant capillaries	5 (9.1)	6 (10.9)	5 (9.1)	16 (29.1)	
Thick arborizing vessels	9 (16.4)	4 (7.3)	5 (9.1)	18 (32.7)	
Vascular network	1 (1.8)	0	1 (1.8)	2 (3.6)	>.05
Fibrotic structures					
White dots	14 (25.5)	10 (18.2)	16 (29.1)	40 (72.7)	>.05
White scarring areas	16 (29.1)	28 (50.9)	6 (10.9)	50 (90.9)	>.05
Milky red areas	14 (25.5)	16 (29.1)	10 (18.2)	44 (80)	>.05
Shiny white structures	4 (7.3)	13 (23.6)	15 (27.3)	28 (50.9)	.02
Chrysalides	3 (5.5)	9 (16.4)	6 (10.9)	18 (32.7)	
Rosettes	1 (1.8)	4 (7.3)	9 (16.4)	14 (25.5)	
Total				55 (100)	

*Thinner than hair shafts.
†Thicker than hair shafts.

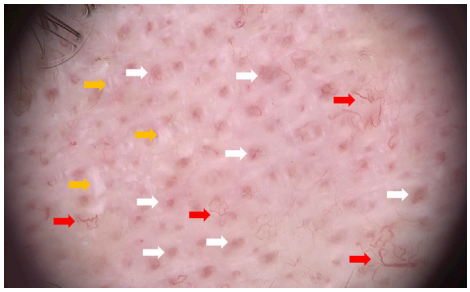

Fig 1. Trichoscopy of a patch of discoid lupus alopecia with immersion fluid (×20). Red follicular dots (white arrows) and thin arborizing vessel (red arrows) can be observed. There are also shiny white structures (yellow arrows) in the interfollicular area.

standing discoid lupus erythematous alopecia patches.

Limitations must be noted. Some trichoscopy features could have been influenced by local treatments. However, we did not find any differences between treated and untreated patients.

In conclusion, we have systematically described the trichoscopic features of discoid lupus erythematous alopecia. Shiny white structures are a novel trichoscopic finding in long-lasting lesions.

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Management of paronychia with pseudopyogenic granulomas secondary to epidermal growth factor receptor inhibitors: An assessment of topical timolol and the need for multiple medical and procedural therapies

To the Editor: Paronychia and pseudopyogenic granulomas (PGs) occur in 10% to 30% of patients receiving epidermal growth factor receptor (EGFR) inhibitor therapy and frequently affect patients' quality of life.[1] Recently in case series, topical beta-blockers have been explored as a noninvasive strategy to treat this nail toxicity.[1-4] However, their rate of efficacy varies widely across studies when used as monotherapy.

To assess our experience of topical timolol for this indication, we conducted a retrospective review of patients with EGFR inhibitor–related paronychia with PGs who were referred to the Oncodermatology Clinic at the Yale Cancer Center from 2018 to 2020. Patients treated with topical timolol (0.5% twice daily under occlusion) monotherapy were included. Timolol treatment response was recorded as either complete, partial, or no response at 4 weeks. Those who did not respond were treated according to recommendations for the management of EGFR inhibitor–associated paronychia.[5] In addition, patient characteristics; cancer treatment; lesion number and location; additional medical, physical, or surgical therapy; and impact on cancer therapy were recorded. All patients tolerated dermatologic treatment without significant adverse effects.

Table I summarizes patient characteristics and treatment response. There were 10 patients, including 5 men and 5 women (mean age, 60 years; range, 49-78 years) with lung (n = 7) and head and neck (n = 3) cancer treated with topical timolol. Associated EGFR inhibitors included afatinib monotherapy (n = 2), afatinib followed by osimertinib (n = 2), combination afatinib/cetuximab (n = 2), erlotinib monotherapy (n = 1), combination erlotinib/nivolumab (n = 1), and poziotinib monotherapy (n = 1).

The median time receiving EGFR inhibitor(s) before development of paronychia with PGs was 146 days. There were, on average, 2.3 PGs per patient, and 7 of 10 patients had both fingernail and toenail involvement. Topical timolol monotherapy resulted in complete response in 2 patients at 4 weeks (patient 8 and patient 10), of whom 1 experienced recurrent PGs that partially responded. Nine of 10 patients required additional therapy because of insufficient (partial or no) response. Adjunct medical treatment included topical antibiotics/antiseptics (n = 9), topical steroids (n = 5), and oral antibiotics after bacterial culture (n = 5). Adjunct procedural intervention included silver nitrate cauterization (n = 5), cryotherapy (n = 1), and partial or full nail plate avulsion (n = 3) by podiatry for recalcitrant cases. Overall, all patients showed a clinical response (n = 4 complete, n = 6 partial) to dermatologic therapy (Fig 1), and no patients required interruption of oncologic therapy. Concurrent cutaneous toxicities included acneiform