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Onychoscopy of allergic contact dermatitis caused by artificial nails: A double-center retrospective study on 34 patients

To the Editor: Artificial nails have gained popularity among women in recent years. They are associated with a high risk of developing allergic contact dermatitis because of the acrylates contained in the formers.[1-3] Artificial-nail-induced allergic contact dermatitis usually presents with acute dystrophic nail changes, often misdiagnosed as psoriasis, that occur at removal of the artificial nails. To date, no information about dermoscopy of this condition has been published, to our knowledge. A double-center retrospective study included 34 patients affected by allergic contact dermatitis caused by artificial nails, confirmed by patch-testing positivity. Demographic data, history, and clinical and dermoscopic features were collected (Table I). Age at presentation was variable, ranging from 26 to 80 years, with a peak incidence at aged 50 to 59 years. Median duration of the disease was 20 days.

Clinical examination showed nail color changes and onycholysis associated to some extent with subungual hyperkeratosis and paronychia as a sign of nail bed and periungual reaction. Onychoscopy was exhibited in all the affected patients as white discoloration, appearing as a distal color change of the nail plate in most cases (Fig 1). The presence of onycholysis was confirmed by dermoscopy, with a slightly dented onycholytic border that was more common than the linear one. An interesting finding was the low rate of an erythematous border of onycholysis, different from that commonly observed in nail psoriasis.[4]

Main clinical signs of nail bed psoriasis are onycholysis with erythematous border and salmon oil spot, whereas nonspecific signs are splinter hemorrhages and subungual hyperkeratosis. Onychoscopy is helpful in patients with fingernail onycholysis secondary to psoriasis, allowing visualization of the erythematous border surrounding the distal edge of the detachment. This is the most typical symptom of psoriasis and it appears as a bright orange-yellow border surrounding the distal edge of the detachment, which is observed as a slightly dented margin.[5] In our patients, subungual hyperkeratosis was commonly observed, but in most cases the extent was mild. Less than half the patients presented with a variable periungual tissue damage, ranging from mild paronychia to excoriations, thus probably indicating that in the remnant the allergic reaction was limited to nail tissue. The detection of splinter hemorrhages in a minority of patients made the differential diagnosis with nail psoriasis more challenging because these are nonspecific signs of nail psoriasis. Patch-testing results were similar to previously reported ones,[1] indicating that methacrylate was the most common allergen responsible for allergic contact dermatitis caused by artificial nails. The low rate of erythematous border and additional signs such as splinter hemorrhages, the mild hyperkeratosis, the history of a use of artificial nails, positivity of patch testing, and the absence of history for psoriasis could make the diagnosis of allergic contact dermatitis caused by artificial nails more likely.

Nail dermoscopy (onychoscopy) is used by experts in nail diseases to magnify the specific alterations on all visible parts of the nail unit, but also to provide important diagnostic information.

To our knowledge, our study was the first to describe specific onychoscopic features of allergic contact dermatitis caused by artificial nails. We believe our findings will help physicians in determining differential diagnoses of other common nail diseases such as psoriasis, which is often confused with allergic contact dermatitis. Dermoscopic findings were derived from a pilot double-center retrospective study, whose results should be confirmed by further studies.

Table I. Summary of patients' characteristics and dermoscopy

				Dermoscopic features				
Patient no.	No. of digits involved	Duration, days	Clinical features	Surface	Border of onycholysis	Subungual hyperkeratosis	Periungual tissues involvement	Specific signs
1	10	10	WD, OL, SH	Whitish DD	LN	Yes	Yes	Splinter hemorrhages
2	10		WD, OL, mild PN	Red-purple DD	SD	Yes	Yes	Splinter hemorrhages
3	10		WD, OL	Whitish DD	LN and SD	No	Yes	No
4	20		WD, OL, SH	Yellow DD	LN and SD	Yes	Yes	Blood spots
5	10		WD, OL	Whitish DD	LN	No	No	No
6	10		WD, OL, SH, PN	Whitish DD, BL	SD with erythematous border	Yes	Yes	Splinter hemorrhages
7	10		WD, OL, SH, mild PN	Whitish DD	LN	Yes	Yes	Absence of nail plate because of file on the first digit of left hand
8	10		WD, OL	Whitish DD	LN	No	No	Blood spots at the distal margin in a few digits
9	10	15	WD, OL, mild PN	Whitish DD	SD	Yes	Yes	Splinter hemorrhages
10	10	10	WD, OL, SH, PN	Whitish DD	SD with erythematous border	Yes	Yes	Splinter hemorrhages
11	10	15	WD, OL, PN	Whitish DD	SD with erythematous border	Yes	Yes	No
12	10	7	WD, OL, PN	Whitish DD	LN	No	Yes	No
13	10	20	WD, OL	Whitish DD	SD	Yes	No	No
14	10	20	WD, OL	Whitish DD	SD	Yes	Yes	No
15	10	15	WD, OL, SH, PN	Whitish DD, BL	SD with erythematous border	Yes	Yes	Splinter hemorrhages
16	10	20	WD, OL, mild PN	Whitish DD	SD	Yes	Yes	No
17	10	20	WD, OL	Whitish DD	SD	Yes	No	Splinter hemorrhages
18	10	20	WD, OL	Whitish DD	SD	Yes	No	No
19	10	20	WD, OL	Whitish DD	SD	Yes	No	Splinter hemorrhages
20	10	20	WD, OL	Whitish DD	SD	Yes	No	Splinter hemorrhages and nail red in a few digits
21	10	20	WD, OL	Whitish DD	SD	Yes	No	Pitting and splinter hemorrhages
22	10	20	WD, OL, PN	Whitish-yellowish DD	SD	No	No	BL
23	10	30	WD, OL	Whitish DD	SD	Yes	No	Longitudinal fissuring
24	10	20	WD, OL	Whitish DD	SD	Yes	No	No
25	10	20	WD, OL	Whitish DD	SD	Yes	No	No
26	10	20	WD, OL	Whitish DD	SD	Yes	No	No
27	10	20	WD, OL	Whitish DD	SD	Yes	No	No
28	10	20	WD, OL	Whitish DD	SD	Yes	No	No
29	10	20	WD, OL	Whitish DD	SD	Yes	No	No
30	10	15	WD, OL	Whitish DD	SD	Yes	No	Splinter hemorrhages
31	10	10	WD, OL, PN	Whitish-yellowish DD	SD	Yes	Yes	Splinter hemorrhages
32	10	20	WD, OL	Whitish DD	SD	Yes	No	No
33	10	15	WD, OL	Whitish DD	SD	Yes	No	Splinter hemorrhages
34	10	15	WD, OL, SH, PN	Whitish to yellow DD	SD	Yes	Yes	Splinter hemorrhages

BL, Beau lines; *DD*, distal discoloration; *LN*, linear; *OL*, onycholysis; *PN*, paronychia; *SD*, slightly dented; *SH*, subungual hyperkeratosis; *WD*, white discoloration.

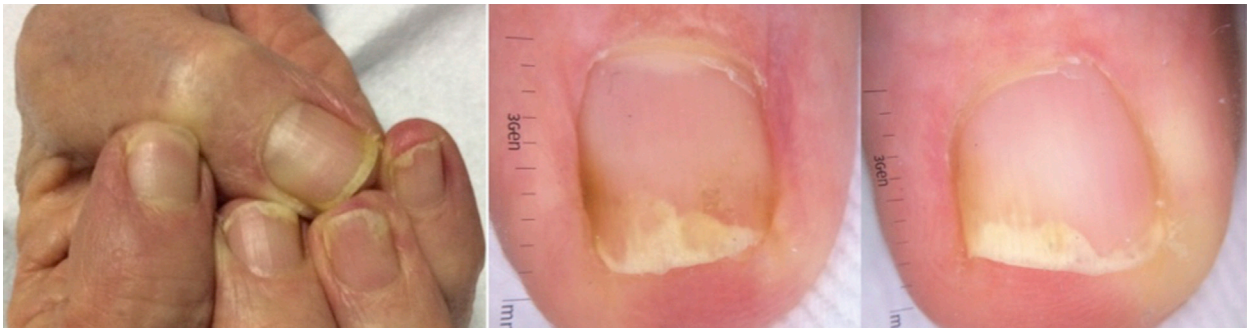

Fig 1. Patient 14 presented with mild nail signs at clinical examination, without subungual hyperkeratosis and paronychia. Dermoscopy of the nail plate showed a slight onycholysis with a slightly dented onycholytic border and no erythematous border.

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Tofacitinib in the treatment of lichen planopilaris: A retrospective review

To the Editor: Tofacitinib is a Janus kinase inhibitor currently approved for the treatment of rheumatoid arthritis, psoriatic arthritis, and ulcerative colitis.[1] Topical and systemic forms have also been used off label to treat a variety of dermatologic conditions, including psoriasis and vitiligo.[1-3] Lichen planopilaris is an immunomediated cicatricial alopecia that is often treatment refractory, but limited evidence supports the therapeutic potential of oral tofacitinib.[4,5] The aim of this study is to further investigate the efficacy of both topical and oral tofacitinib in the treatment of recalcitrant lichen planopilaris.

A retrospective review was conducted with approval of the institutional review board at the Medical University of South Carolina. From May 2012 through March 2020, 9 patients treated with tofacitinib for lichen planopilaris were identified (Table I). All patients were white and had failed extensive therapies.

Tofacitinib was used adjunctively in all patients (Table II). In both topical and oral forms, the median time to initial treatment response was 3 months. Through use of patient-reported symptoms and physical examination findings (affected area, erythema, and presence of follicular spines), treatment response was graded relative to baseline disease severity at the initiation of tofacitinib therapy. Three of 4 patients receiving topical therapy achieved a positive initial response, and 2 patients exhibited sustained clinical improvement. Patient 9 exhibited a negative clinical response to topical therapy and was converted to systemic therapy after 1 month. All patients receiving systemic therapy demonstrated a favorable initial response, and this was maintained in all but 1 patient. In patients 6 and 7, oral dosing was increased to 3 times