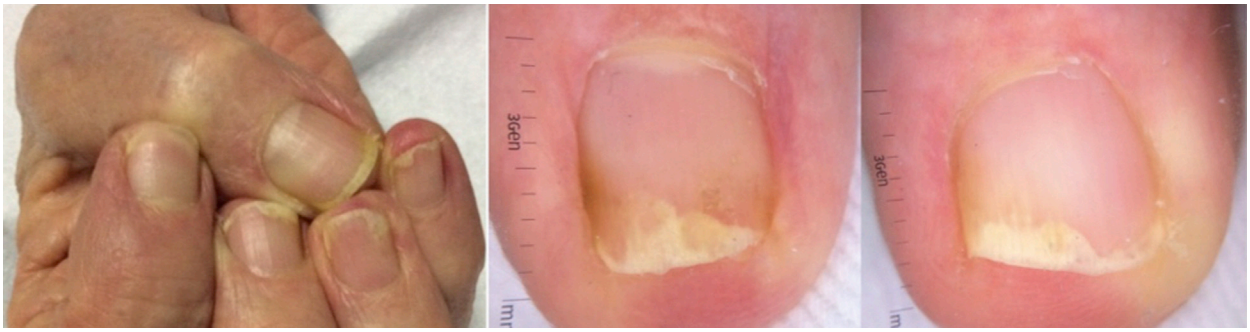

Fig 1. Patient 14 presented with mild nail signs at clinical examination, without subungual hyperkeratosis and paronychia. Dermoscopy of the nail plate showed a slight onycholysis with a slightly dented onycholytic border and no erythematous border.

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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

Table I. Disease history before initiation of tofacitinib therapy

Patient no.	Sex	Age at diagnosis, years	Disease variant	Diagnostic method	Previous treatment duration, years	Previous therapies
1	F	61	FFA, classic LPP	Biopsy	1	Doxycycline, dutasteride, HCQ, pioglitazone, tacrolimus ointment, topical steroids
2	F	69	Classic LPP	Biopsy	9	Doxycycline, dutasteride, finasteride, HCQ, ILK, laser cap, minoxidil, pimecrolimus cream, topical steroids
3	F	63	FFA	Clinical	1	Finasteride, minoxidil, MPA, topical steroids
4	F	49	Classic LPP	Biopsy	3	HCQ, ILK, naltrexone, pimecrolimus cream, topical steroids
5	F	63	FFA, classic LPP	Clinical	2.5	Dutasteride, finasteride, HCQ, ILK, naltrexone, pimecrolimus cream, pioglitazone, topical steroids
6	M	33	Classic LPP	Biopsy	2	HCQ, ILK, laser cap, naltrexone, pimecrolimus cream, pioglitazone, topical steroids
7	F	67	FFA	Clinical	1	Dutasteride, naltrexone, pimecrolimus cream, pioglitazone, topical steroids
8	F	61	FFA, classic LPP	Clinical	2.5	Dutasteride, excimer laser, HCQ, naltrexone, pimecrolimus cream, pioglitazone, prednisone
9	F	59	FFA, classic LPP	Biopsy	6	Cyclosporine, dutasteride, excimer laser, HCQ, ILK, ketoconazole cream and shampoo, laser cap, leflunomide, MPA, naltrexone, pimecrolimus cream, prednisone, tacrolimus ointment, topical steroids

F, Female patient; *FFA*, frontal fibrosing alopecia; *HCQ*, hydroxychloroquine; *ILK*, intralesional triamcinolone; *LPP*, lichen planopilaris; *M*, male; *MPA*, mycophenolic acid.

Table II. Treatment course of tofacitinib

Patient no.	Route	Dose	Time to initial response, months	Initial response*	Total treatment duration, months	Longitudinal response*	Concurrent therapies
1	Topical	2% cream BID	3	+1	17	+1	Dutasteride, ILK, naltrexone, pimecrolimus ointment
2	Topical	2% cream BID	5	+1	11	−2	Finasteride, ILK, laser cap, minoxidil, pimecrolimus cream, topical steroids
3	Topical	2% cream BID	3	+2	10	+3	Dutasteride, laser cap, minoxidil, MPA, naltrexone, pimecrolimus cream
4	Oral	5 mg BID	7	+3	15	+3	Naltrexone, pimecrolimus cream, topical steroids
5	Oral	5 mg BID	2	+2	10	0	Dutasteride, laser cap, minoxidil, naltrexone, pimecrolimus cream, topical steroids
6	Oral	5 mg BID, TID	1	+3	12	+2	ILK, naltrexone, pimecrolimus cream, topical steroids
7	Oral	5 mg BID, TID	4	+1	7	+2	Dutasteride, NAC, naltrexone, pimecrolimus cream, topical steroids
8	Oral	11 mg QD	5	+3	13	+3	Dutasteride, minoxidil
9	Topical	2% cream BID	1	−2	1	NA	Dutasteride, laser cap, naltrexone,
	Oral	5 mg BID	1	+1	4	+2	tacrolimus ointment, topical steroids

BID, Twice daily; *ILK*, intralesional triamcinolone; *MPA*, mycophenolic acid; *NA*, not applicable; *NAC*, *N*-acetylcysteine; *QD*, once daily; *TID*, 3 times daily.

*Treatment responses were graded relative to baseline disease severity: −3, much worse; −2, moderately worse; −1, mildly worse; 0, no change in disease activity; +1, mildly improved; +2, moderately improved; and +3, greatly improved.

daily after 10 and 7 months of treatment, respectively, to achieve greater disease control. Two patients showed improvement in a concomitant condition (erythema dyschromicum perstans and unspecified arthritis). Two other patients had accompanying diagnoses of rheumatoid arthritis and vitiligo, but treatment responses were not recorded.

Complete blood cell counts and comprehensive metabolic panels were available for review for all but 1 patient. Patient 5 experienced mild, transient hemoglobin and creatinine abnormalities. Lipid panels were available for review in 4 patients. Mildly elevated triglyceride and cholesterol levels were noted in 2 patients, but neither required treatment. No other adverse events were reported.

Both topical and oral formulations were effective in achieving a positive clinical response. Although oral tofacitinib led to more pronounced and sustained improvement, topical therapy may be considered a feasible alternative in some patients.[3] Tofacitinib was well tolerated in our patient cohort, with only minor laboratory abnormalities noted.

This study is limited by the small sample size, and 4 subjects received a diagnosis clinically. Because of the retrospective design, determining the direct clinical response to tofacitinib is difficult, especially with concurrent therapies. In addition, potential expectations for favorable treatment outcomes may introduce bias. Generalization of these findings to less refractory disease is uncertain. Providers should interpret these results accordingly.

In conclusion, this study supports further studies of tofacitinib's efficacy in refractory lichen planopilaris and suggests the therapeutic potential of topical formulations.

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Apremilast for the off-label treatment of lichenoid and interface dermatoses

To the Editor: Lichenoid and interface dermatoses are common in the field of dermatology and correlate with a large spectrum of complex dermatologic conditions. These superficial, inflammatory changes in the skin can be representative of many severe diseases, including systemic lupus erythematous, graft-versus-host disease, erythema multiforme, lichen planus, mycosis fungoides, and others.

Apremilast is a phosphodiesterase-4 inhibitor that is currently approved by the US Food and Drug Administration for psoriatic arthritis and plaque psoriasis. However, multiple studies have emerged in the literature suggesting that apremilast may have a role in the treatment of dermatologic disorders beyond what is approved by the US Food and Drug Administration.[1] It is postulated that apremilast could be effective in the treatment of lichenoid dermatoses such as lichen planus.[2,3] Apremilast inhibits the production of cytokines, including interferon gamma, tumor necrosis factor alpha, interleukin (IL) 2, IL-5, IL-8, and IL-12, which leads to the activation of cytotoxic T cells causing basal keratinocyte apoptosis, the underlying mechanism of lichen planus.[4] Furthermore, successful use of apremilast for interface dermatitis in the form of recurrent erythema multiforme and other drug eruptions has also been previously described.[5] However, literature describing the use of apremilast in patients with lichenoid and interface dermatitis still remains incomplete. Here, we describe an effective use of apremilast in 5 patients with lichenoid and interface dermatoses: 1 patient with psoriasiform dermatitis with lichenoid change, 2 with lichenoid dermatitis, and 2 with interface dermatitis (Fig 1).

A retrospective review of our institution's records was conducted to identify patients with a lichenoid