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

Patient Number	Age at Diagnosis /Sex	Diagnosis	Disease Duration	Failed Treatments	Response to Apremilast	Length of Apremilast Treatment	Adverse Effect from Apremilast	Treatment Limiting Toxicity? (Yes/No)	Denied Insurance Coverage? (Yes/No)
1	57/Female	Psoriasiform Dermatitis with Lichenoid Changes	5.5 years	Triamcinolone Acetonide cream, Tanning bed, Clobetasol 0.05% ointment and foam, Salicylic acid, Hydroxyzine, Methotrexate, Ultraviolet-B Phototherapy	No new lesions while on apremilast	27 months (ongoing) – started 10/1/2017	Minimal weight loss	No	No
2	46/Female	Lichenoid Dermatitis – with hand involvement	3 years	Clobetasol 0.05% ointment, Prednisone, Gloves, Vaseline, Methotrexate	Near complete remission – (<1% total body surface area involvement)	17 months (ongoing) – started 8/5/2018	Nausea, vomiting, diarrhea	No	No
3	61/Female	Interface Dermatitis	0.5 years	Ultraviolet-B Phototherapy, Clobetasol, Oxiconazole, Tazarotene cream, Betamethasone, Hydrocortisone Valerate, Hydroxychloroquine	No new lesions while on apremilast	5 months (ongoing) – started 8/23/2019	None	No	Yes – patient will go off Apremilast even though she is having a great response due to insurance denial of coverage
4	22/Female	Lichenoid Dermatitis	5 years	Prednisone, Triamcinolone	No new lesions while on apremilast	6 months – started 9/6/2017, stopped 2/20/2018	None	No	Yes – patient stopped Apremilast due to insurance denial of coverage
5	68/Female	Interface Dermatitis	1.5 years	Prednisone, Triamcinolone, Clobetasol, Methotrexate	Near complete remission – (<1% total body surface area involvement)	11 months – started 6/10/2018, stopped 7/4/2019	Diarrhea, nausea, upper respiratory illness	No (continued drug at 30 milligrams daily)	No

Fig 1. Characteristics of patients with superficial inflammatory dermatoses treated with apremilast.

or interface histology pattern confirmed on skin biopsy. A minimum follow-up time of 3 months after initiation of treatment with apremilast was necessary for inclusion. Before starting apremilast, attempted therapies included prednisone, clobetasol, methotrexate, betamethasone, azathioprine, and calcineurin inhibitors such as topical cyclosporine, with limited response to treatment.

Apremilast 30 mg twice daily was the starting dose for all 5 patients. Within 3 months of follow-up, all patients reported significant improvement in their skin lesions and associated symptoms. Three patients experienced no new lesions while taking apremilast, and 2 had near complete remission with less than 1% total body surface area involvement. Two of the 5 patients (patients 3 and 4) had a great response to apremilast but were forced to stop apremilast because of denial of insurance coverage. One of the 5 patients (patient 5) received apremilast for 11 months with an excellent response but experienced treatment-limiting toxicity (Fig 1).

Here, we have presented several cases of lichenoid and interface dermatoses that responded well to treatment with apremilast. In each case, patients tried several therapies, including those with an extensive and severe adverse effect profile. Several studies have reported off-label uses for apremilast, and it is hoped that the demonstration of its successful use in patients with these particular dermatoses can further treatment options for these patients. Limitations of our study include small sample size, lack of standardized scoring systems to assess treatment response, and limited follow-up time. Additional studies are needed understand the efficacy of apremilast in this patient population.

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Efficacy and safety of very-low-dose oral minoxidil 1.25 mg in male androgenetic alopecia

To the Editor: Oral minoxidil has been used with significant improvement in androgenetic alopecia in patients of either sex.[1-4] In men with androgenetic alopecia, oral minoxidil has been used alone and in combination with other therapies and in doses varying from 0.25 mg/day to 2.5 to 5 mg/day.[2,3] Some of the reported adverse effects of such lower doses of oral minoxidil include lower limb edema, generalized hypertrichosis, hair shedding, urticaria, and other allergic reactions.[1-4] In this case series, we share our experience of using oral minoxidil monotherapy at 1.25 mg/day in 32 patients with male androgenetic alopecia.

Thirty-two men (18-45 years; mean age 28.83 ± 7.12 years) with androgenetic alopecia (Hamilton-Norwood scale III-V) were treated with oral minoxidil monotherapy dosed at 1.25 mg/day for 24 weeks. Patients with severe hypertension, moderate hypertension on antihypertensive therapy, cardiovascular dysfunction, history of minoxidil hypersensitivity, or receiving treatment with any formulation of minoxidil, dihydroxytestosterone blockers, or other treatment(s) for androgenetic alopecia in the past 3 months were excluded. Of the included patients (n = 32), 18 (56.2%) were treatment naive, whereas 14 (43.8%) had failed previous monotherapy with either topical minoxidil 5% twice daily or oral finasteride received for at least 1.5 to 2 years. Efficacy outcome measures included global clinical photography and quantitative digital video trichoscopic assessment. Fig 1, *A*, schematically demonstrates the trichoscopy points that were evaluated (adopted from Leerunyakul and Suchonwanit[5]). Overall improvement was labeled "marked" if there was at least 1 grade improvement in the Hamilton-Norwood scale on global clinical photographic assessment; appreciable new hair growth not amounting to marked response was labeled as mild improvement. At each visit, patients

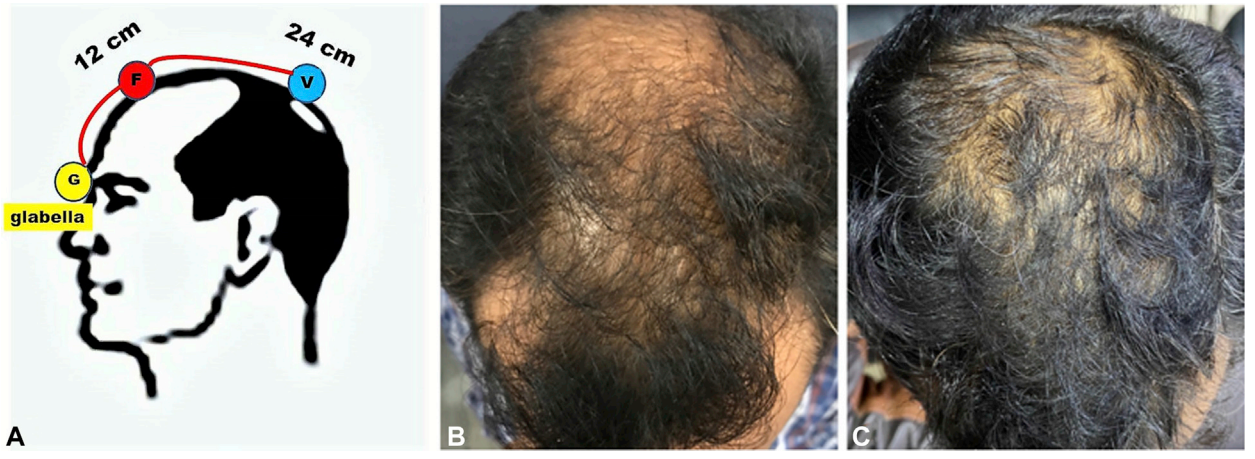

Fig 1. A, Schematic diagram of the human scalp (lateral view) demonstrating the points of trichoscopic analysis and their surface anatomy with respect to glabella (point G). Two sites on the scalp were measured at 12 and 24 cm from the glabella, identified as the frontal (point F) and vertex (point V) points, respectively. **B**, Baseline scalp clinical photograph of a patient with androgenetic alopecia Hamilton-Norwood scale V. **C**, Posttreatment clinical photograph showing significant improvement (Hamilton-Norwood scale III) after 6 months of oral minoxidil 1.25 mg/day.