unresponsive to LDN, doses of 25 or 50 mg/d were prescribed, as previously described.[4] Follow-up and laboratory monitoring were performed according to clinical response.

No standardized assessment scale has been validated to date for HHD. The disease was considered severe if the patient had a body surface area (BSA) ≥5% or a dermatology life quality index (DLQI) ≥11, moderate if BSA was 3% to 4% or DLQI was 6 to 10, and mild if BSA was <3% or DLQI was ≤5. The Clinician Global Impression of Change (CGIC) and Patient Global Impression of Change (PGIC) were performed when the patient discontinued the treatment or at the cutoff point of February 2019. Patient response was classified as having no improvement, initial improvement but with relapse during follow-up, or sustained improvement (BSA <3% or DLQI ≤5 for ≥6 months).

The study included 14 patients with a median age of 56.5 years (interquartile range, 52-68 years). Patient baseline characteristics and response to therapy are summarized in Table I. Disease was severe in 8 patients. The median follow-up time was 33.6 weeks (interquartile range, 15-54 weeks). Six patients had a follow-up of >1 year and 3 of >6 months. Six patients showed no improvement with LDN, and 6 had an initial improvement but relapsed. Two patients had a sustained response of >1 year. Six patients discontinued the treatment, 4 because of ineffectiveness and 2 because of adverse events.

Most publications of HHD treated with LDN describe a reduced number of patients with variable follow-up time. In our study, the largest series described to date, we observed a lack of response or an initial response with subsequent relapse in most patients. In patients who had a relapse, there was a mild to moderate improvement with the treatment, according to the CGIC and PGIC, probably as a result of a decrease in the intensity and duration of flares. Only 2 patients, with follow-up of >1 year, showed sustained improvement. Interestingly, these 2 patients are sisters. Further studies are required to determine whether certain mutations[5] can be associated with favorable responses to LDN. Patients showed a response to treatment mainly on doses of 3.0 and 4.5 mg/d. Increasing the dose of naltrexone to 25 or 50 mg/d, did not seem to offer better results in our patients.

LDN may be an alternative in patients with refractory HHD, although with lower response rates than those described, with frequent relapses.

We thank our patients and their families, who are the main reason for our studies, and the Hospital Clínic de Barcelona Pharmacy Department, especially Dr M. Carmen Lopez and Dr. Nuria Socoro, for their contribution.

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Funding sources: None.

Conflicts of interest: None disclosed.

Reprints not available from the authors.

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https://doi.org/10.1016/j.jaad.2019.04.024

A pilot study of 2% tofacitinib cream with narrowband ultraviolet B for the treatment of facial vitiligo

To the Editor: Current treatments for vitiligo are limited in efficacy, often producing suboptimal results. Recent studies have established that $CD8^+$ T-cell and interferon γ signaling, mediated by the Janus kinase (JAK)−signal transducer and activator of transcription pathway, contribute to the pathogenesis of vitiligo.[1] JAK inhibitors block this pathway and are currently Food and Drug Administration approved for autoimmune diseases,

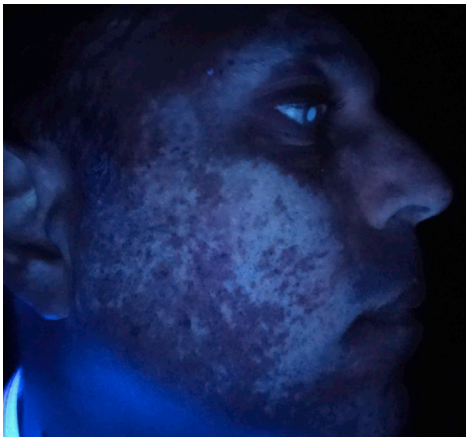

Fig 1. Nonsegmental vitiligo in a white man at baseline.

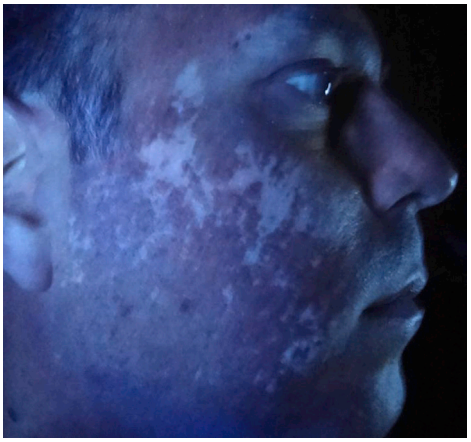

Fig 2. Nonsegmental vitiligo after 4 months of treatment with 2% tofacitinib cream twice daily and narrowband ultraviolet B phototherapy 3 times weekly.

including psoriatic arthritis, rheumatoid arthritis, ulcerative colitis, and myeloproliferative disorders.[1]

Recently, treatment with oral tofacitinib, a JAK 1 and 3 inhibitor, was shown to improve vitiligo disease severity in case reports and a small series. Unfortunately, this medication carries risks of systemic side effects, including infections, malignancies, and cytopenias, and is expensive, with monthly costs >$4000 USD for sixty 5-mg tablets.[2] Given these limitations, topical JAK inhibitors could serve as a safer, more cost-effective treatment for vitiligo. We report the use of topical 2% tofacitinib cream in a small series of patients with facial vitiligo.

In this institutional review board–approved study, 11 patients with vitiligo seeking treatment at the Pigmentary Disorders Clinic of the University of Texas Southwestern Medical Center were treated with 2% tofacitinib cream twice daily in conjunction with narrowband ultraviolet B (NB-UVB) therapy thrice weekly over a period of 3 ± 1 months. NB-UVB dosing was performed as recommended by the Vitiligo Working Group consensus guidelines.[3] All patients had previously tried ≥3 months of treatment with topical corticosteroids or calcineurin inhibitors in addition to NB-UVB phototherapy or sunlight exposure 3 times weekly without improvement of their disease. The Vitiligo Area Severity Index (VASI) was used to measure the amount of depigmentation on each patient's face, also called facial VASI, before and after 3 ± 1 months of treatment.[4]

Six patients were non-Hispanic white, 4 were Asian, and 1 was Hispanic. The male-to-female ratio was 5:6, and mean age was 44 years. The mean facial VASI was 0.80 (range 0.1-2.25) at baseline and 0.23 (range 0.03-0.75) at follow-up (Figs 1 and 2), which is a mean improvement of 70% (range 50%-87%). Mean time to follow-up was 112 (range 84-154) days. Because of the small area of involvement, a 30-g tube of tofacitinib 2% cream lasted each patient an average of 90 days and cost $320 USD. The cream was obtained from a compounding pharmacy in Pennsylvania and mailed to the patient. There were no reported side effects.

Recent studies have shown better repigmentation in lesions exposed to sunlight compared to unexposed lesions in patients treated with oral and topical JAK inhibitors.[1,5] The improvement seen in our patients with combination topical tofacitinib and phototherapy suggests a synergistic relationship. Repigmentation of facial vitiligo lesions was good to excellent in all 11 patients. The face was chosen in this pilot study because this region has the best response in patients with vitiligo, but future studies should include treatment of nonfacial areas. Other limitations of this pilot study are small sample size and lack of controls. Future controlled studies with larger sample sizes and long-term follow-up should be performed. If they are confirmatory, topical tofacitinib could become a useful addition to our therapeutic armamentarium for vitiligo.

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Funding sources: None.

Conflicts of interest: Dr Pandya is a consultant for Pfizer, maker of tofacitinib, and serves as the principal investigator for a vitiligo study with a new drug funded by Pfizer. Dr McKesey has no conflicts of interest to disclose.

Reprints not available from the authors.

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https://doi.org/10.1016/j.jaad.2019.04.032

Effectiveness and safety of low-dose oral minoxidil in male androgenetic alopecia

To the Editor: Low-dose oral minoxidil (OM) has been successfully used in female hair loss (0.25-1.25 mg daily),[1-3] but there are scarce reports in male androgenetic alopecia (MAA).[1,4] The objective of our study was to evaluate the effectiveness and safety of low-dose OM (2.5-5 mg daily) in men with MAA.

We retrospectively reviewed male patients who had MAA diagnosed clinically and by trichoscopy and were receiving OM in monotherapy or as an additional therapy. Of the patients receiving other concomitant therapies, only those without treatment modifications in the 12 months before minoxidil therapy were included. OM, 2.5 or 5 mg daily, was given for a minimum of 6 months. This dosage was based on a previous report by Lueangarun et al.[4] Therapeutic response was assessed by comparison of pretreatment and post-treatment clinical images by 3 independent dermatologists with expertise in hair disorders (D.S.C., R.R.B., and S.V.G.), using a 4-point scale (worsening, stabilization, mild improvement, or marked improvement). An improvement of 1 grade or more on the Norwood-Hamilton scale was defined as marked improvement.

A total of 41 men with a mean age of 33.3 years (range, 20-55) were included. They received OM at a daily dose of 2.5 mg (10 patients) or 5 mg (31 patients). In all, 25 patients (61%) had previously undergone other therapies for a mean of 18 months (range, 12-48): oral dutasteride (18 patients), mesotherapy with dutasteride (9 patients), oral finasteride (3 patients), topical minoxidil (2 patients), and topical finasteride (1 patient). A total of 16 patients (39%) received OM as monotherapy (Table I). Clinical improvement was observed in 37 patients (90.2%), with 11 of these patients (26.8%) presenting a marked improvement. Four patients (9.8%) showed stabilization, and none of them worsened. All those in the subgroup of 16 patients receiving OM as monotherapy presented clinical improvement, with 6 patients (37.5%) showing marked improvement (Fig 1, *A* and *B*). Adverse effects were detected in 12 patients (29.3%): hypertrichosis in 10 patients (24.3%), lower limb edema in 2 patients (4.8%), and shedding in 1 patient (2.4%). All of the adverse effects were mild and well tolerated. Only 1 patient discontinued the treatment, because of pedal edema. These adverse effects appeared with the dose of 5 mg daily, except in 2 patients with slight hypertrichosis and 1 patient with shedding (2.5 mg daily).

OM, 0.25-1.25 mg daily, has been used for the treatment of female androgenetic alopecia, traction alopecia, and telogen effluvium, showing improvement in 61 to 86% of patients and a good safety profile.[1-3] There are few articles describing the effectiveness of OM in MAA.[1,4] A previous study reported improvement in 30 men (100%) with MAA who were taking OM, 5 mg, with a higher rate of adverse events than in our cohort: 93% of patients had hypertrichosis, 10% had edema, and 10% had an electrocardiogram alteration.[4]

The study's retrospective design and low number of patients are limitations.

In conclusion, OM at a dose of 5 mg daily was effective and presented an acceptable safety profile in our cohort of male patients with MAA. The optimum dose needs to be delineated in future controlled studies.

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Funding sources: None.

Conflicts of interest: None disclosed.