**Table I.** Patient demographics, efficacy, and safety outcomes

| Variable | Value |
|---|---|
| Sex, n (%) | |
| Male | 29/41 (70.7) |
| Female | 12/41 (29.3) |
| Mean age, y ± SD | 45.0 ± 11.0 |
| No. of previously failed biologic therapies, mean ± SD | |
| PASI 75 responders at wk 52 | 2.2 ± 1.4 |
| Non–PASI 75 responders at wk 52 | 2.5 ± 1.1 |
| Efficacy | |
| Wk 52 outcomes, n (%) | |
| ≥PASI 75 or PGA 0 or 1 | 28/41 (68.3) |
| <PASI 75, PGA >1, or discontinued | 13/41 (31.7) |
| Discontinuation before wk 52, n (%) | 11/41 (26.8) |
| Lack of efficacy | 10 (24.4) |
| Intolerance | 1 (2.4) |
| Mean treatment duration, wk ± SD | 40.0 ± 7.1 |
| Safety | |
| No. of patients with ≥ 1 AE, n (%) | 7/41 (17.1) |
| Reported AEs, n (%) | |
| Diarrhea | 2 (4.9) |
| Nasopharyngitis | 1 (2.4) |
| Bronchitis | 1 (2.4) |
| Cutaneous candidiasis | 1 (2.4) |
| Superficial skin bacterial infection | 1 (2.4) |
| Fever | 1 (2.4) |
| Abdominal pain | 1 (2.4) |
| Vomiting | 1 (2.4) |
| Bloody stool | 1 (2.4) |
| Neutropenia | 1 (2.4) |

*AE*, Adverse event; *PASI*, Psoriasis Area and Severity Index; *PGA*, Physician Global Assessment; *SD*, standard deviation.

elicit or worsen inflammatory bowel disease symptoms, both patients are continuing to receive treatment and responding exceptionally well, with near skin clearance at week 52. Discontinuation because of intolerance was uncommon, with just 1 patient stopping treatment between weeks 12 and 52 because of an episode of nasopharyngitis lasting several weeks.

Overall, our results suggest that fewer patients with psoriasis in real-world clinical practice maintain efficacious outcomes at week 52 than those enrolled in RCTs, with the majority of these patients discontinuing secukinumab treatment around week 40 because of lack of efficacy.

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*Jorge R. Georgakopoulos, BSc,[a] Arvin Ighani, BMSc,[b] Michelle Phung, BMSc,[c] and Jensen Yeung, MD, FRCPC[b,d,e,f]*

*Schulich School of Medicine and Dentistry, Western University, London, Ontario, Canada[a]; Faculty of Medicine[b] and Department of Pharmacology and Toxicology, University of Toronto, Toronto, Ontario, Canada[c]; Sunnybrook Health Sciences Centre, Toronto, Ontario, Canada[d]; Women's College Hospital, Toronto, Ontario, Canada[e]; and Probity Medical Research Inc., Waterloo, Ontario, Canada[f]*

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*Correspondence to: Jensen Yeung, MD, FRCPC, Department of Dermatology, Women's College Hospital, 76 Grenville St, Fifth Floor, Toronto M5S 1B2, Ontario, Canada*

*E-mail: jensen.yeung@utoronto.ca*

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## Effectiveness of low-level laser therapy in lichen planopilaris

*To the Editor:* Lichen planopilaris (LPP) is a lymphocytic scarring alopecia of unknown origin in which the hair follicle stem cells are destroyed by an autoimmune mechanism.[1,2] This condition tends to be progressive, and the most effective

**Table I.** Characteristics of patients with lichen planopilaris treated with low-level light therapy

| Pt | Age, y (mean 61.9 [range 46-79]) | Sex | Fitzpatrick skin phototype | Personal history | Time with disease, mo (mean 44.25 [range 24-60]) | Involvement of other areas | Other alopecias | Previous topical treatments | Previous systemic treatment | LPPAI pretreatment (mean 3.35) | LPPAI 3 months (mean 2.417) | LPPAI 6 months (mean 0.865) | THT pretreatment (mean 71.7 μm) | THT 3 months (mean 105.57 μm) | THT 6 months (mean 82.4 μm) | PGA pretreatment (mean 2.75) | PGA 3 months (mean 4.28) | PGA 6 months (mean 4.375) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 47 | M | IV | — | 36 | axilla | — | Mometasone solution | Hydroxychloroquine | 2.50 | 1.50 | 0.33 | 77.80 | 104.00 | 81.00 | 3 | 4 | 4 |
| 2 | 52 | M | III | — | 48 | — | MAGA-I | Clobetasol foam | Finasteride, azathioprine | 3.33 | 7.50 | 0.33 | 68.00 | 103.00 | 81.25 | 3 | 3 | 5 |
| 3 | 46 | F | IV | Hyperlipidemia | 24 | — | — | — | Pioglitazone | 2.50 | 1.50 | 0 | 66.40 | X | 78.00 | 3 | X | 4 |
| 4 | 69 | F | III | Hypertension, Hallux valgus | 60 | — | FAGA-I | Clobetasol foam | — | 4.16 | 3.33 | 3.00 | 60.00 | 112.8 | 89.00 | 3 | 3 | 3 |
| 5 | 65 | F | III | Hypothyroidism | 24 | — | — | Clobetasol foam | — | 2.50 | 1.75 | 0.66 | 89.00 | 103.00 | 83.00 | 3 | 5 | 5 |
| 6 | 72 | M | II | Hypertension, Kaposi sarcoma | 48 | — | — | — | — | 4.16 | 1.00 | 0.66 | 60.00 | 111.00 | 85.00 | 3 | 5 | 5 |
| 7 | 79 | F | III | Hypertension | 60 | — | — | Clobetasol foam | — | 4.50 | 2.75 | 2.00 | 80.00 | 100.00 | 83.00 | 3 | 5 | 4 |
| 8 | 65 | F | III | Chronic urticaria | 54 | — | FAGA-I | Clobetasol foam | — | 3.16 | 1.00 | 0.00 | 72.00 | 105.2 | 79.00 | 1 | 5 | 5 |
| *P* value* | | | | | | | | | | | .161 (Basal > 3 mo) | **.012** (Basal > 6 mo) | | **.018** (Basal < 3 mo) | **.035** (Basal < 6 mo) | | .141 (Basal < 3 mo) | **.016** (Basal < 6 mo) |
| *P* value† | | | | | | | | | | | **.012** (3 mo > 6 mo) | | | **.018** (3 mo > 6 mo) | | | **.012** (3 mo < 6 mo) | |

Significant values are in bold.

*FAGA*, Female androgenetic alopecia; *GHT*, global hair thickness; *LPPAI*, Lichen Planopilaris Activity Index; *MAGA*, male androgenetic alopecia; *PGA*, Patient Global Assessment; *X*, missing data; *THT*, terminal hair thickness; —, none.

*Compared with pretreatment.

†Comparing 3 mo versus 6 mo.

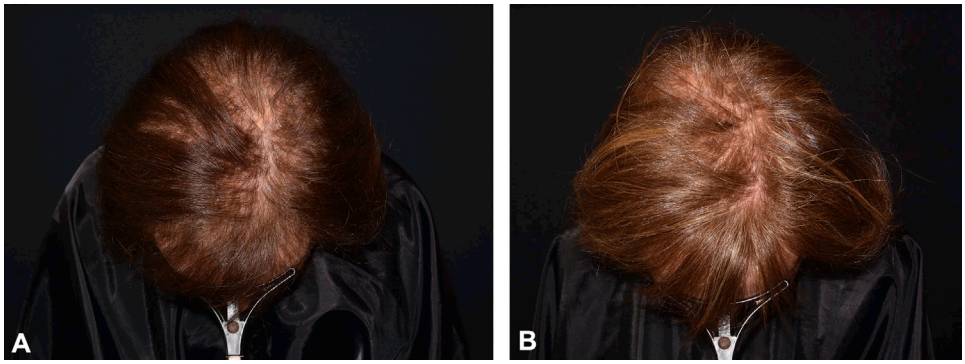


**Fig 1.** Diffuse lichen planopilaris treated with low-level laser therapy in patient 8. **A**, At baseline there is a marked diffuse loss of hair. **B**, Progressive growth of hair after month 6, which was a cosmetic improvement.

treatments have response rates of only 10%.[1] Low-level laser therapy (LLLT) is an emerging light therapy, with a reported effectiveness in several inflammatory diseases, such as lichen planus, although results are sometimes biased.[3] Our objective was to evaluate the potential usefulness of LLLT in patients with LPP.

A 6-month prospective interventional study was performed including patients with clinically and histologically diagnosed LPP. This research was approved by our Human Research Subjects Committee (227-16). The main clinical variable was the Lichen Planopilaris Activity Index (LPPAI),[2] with a final score of 1-10. Vidix polarized video dermatoscope (Medici Medical, Modena, Italy) was used for mapping 5 specific hair fields located at the same sites (3 frontal and 2 on scarring plaques) during each visit to evaluate trichoscopic images and to assess inflammation ( perifollicular erythema and hyperkeratosis) and terminal and global hair follicle thickness. Both videodermatoscopic mapping and cutaneous marks were used to identify hair fields. Standardized pictures were obtained at each visit.

A total of 8 white patients (5 women [62.5%] and 3 men [37.5%]) of a median age of 65 (range 46-79) years with LPP diagnoses were included. Mean time with LPP was 44.25 (range 24-60) months. Baseline mean LPPAI was 3.35 (range 2.50-4.50), and mean terminal hair thickness was 71.7 $\mu$m (Table I). Ledsmedical devices with high-powered unfocused 246 red LED (Skymedic, Barcelona, Spain), at a wavelength of 630 nm and a fluence of 4 J/cm$^2$, were used 15 minutes daily for 6 months.

All patients responded with a global reduction of symptoms, erythema, and perifollicular hyperkeratosis (Fig 1, *A* and *B*), with a mean decrease of 0.87 in LPPAI score after 6 months of intervention ($P = .012$) but not at the 3-month follow-up visit. Terminal hair thickness after 3 months (111.0 $\mu$m vs 71.7 $\mu$m, $P = .018$) and 6 months (82.4 $\mu$m vs 71.7 $\mu$m; $P = .035$) was greater than that at baseline. Other clinical data is shown in Table I.

Severe cases of LPP might be resistant to classical drugs.[2] This is the first study showing the potential effectiveness of LLLT in scarring alopecias. Interestingly, there are preliminary reports indicating that light therapies (low-dose 308-nm excimer laser) reduce inflammation in LPP patients.[4] Remarkably, a significant decrease in inflammation was observed in our cohort of LPP patients after 6 months of LLLT. A significant hair thickening was also detected, maybe related to its positive effect on the follicular cycle[5]; however, a narrowing was noted during the last 3 months of therapy, plausibly caused by tachyphylaxis. Recently, a malfunction in peroxisome proliferator-activator receptor $\gamma$ (PPAR-$\gamma$) has been described in LPP, which is leading to new therapies such as PPAR-$\gamma$ activators or thiazolidinediones.[6] Interestingly, LLLT has been demonstrated to increase PPAR-$\gamma$ expression in murine models of inflammation.[7] The limitations of our study include the small sample size, the lack of a control group, and the absence of histologic data.

*Pablo Fonda-Pascual, MD, Oscar M. Moreno-Arrones, MD, David Saceda-Corralo, MD, Ana R. Rodrigues-Barata, MD, Cristina Pindado-Ortega, MD, Pablo Boixeda, MD, PhD, and Sergio Vaño-Galvan, MD, PhD*

*From the Dermatology Department, Ramón y Cajal Hospital, Madrid, Spain*

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*Correspondence to: Pablo Fonda-Pascual, MD, Dermatology Department, Ramon y Cajal Hospital, Carretera Colmenar Viejo km 9.100, 28034 Madrid, Spain*

*E-mail: pfondap@gmail.com*

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## A cross-sectional survey of long-term outcomes for patients with diffuse capillary malformation with overgrowth

*To the Editor:* Diffuse capillary malformation with overgrowth (DCMO) was recently described as a distinct subtype of vascular anomaly characterized by extensive capillary malformations (CMs) involving >1 anatomic region.[1] Most patients exhibit proportionate soft tissue or bony overgrowth that does not correlate with CM location or intensity.[1] Somatic mutations in *GNA11* have been identified in DCMO, suggesting that aberrant mitogen-activated protein kinase signaling contributes to pathogenesis.[2] Because little is known about long-term outcomes in DCMO, we conducted a Boston Children's Hospital Institutional Review Board–approved anonymous REDCap survey.[3] Sixty-nine patients >5 years of age previously given a DCMO diagnosis and registered in the Vascular Anomalies Center database were contacted.[1] Thirty-one patients or parents of patients (45%) completed questionnaires about the vascular anomaly, overgrowth, and physical and psychological outcomes.

Table I summarizes patient demographics. The mean age was 14.2 years. CMs were most commonly located on the posterior trunk and lower extremities, and overgrowth most frequently involved the extremities.

Table II summarizes long-term outcomes. Nineteen of 31 (61%) patients, including 12 of the 13 with facial involvement, had laser therapy; 95% of the patients who had laser therapy reported response to treatment. Leg-length discrepancy (LLD) affected 19 of 31 (61%) patients; 63% of these individuals received treatments such as orthotics, surgical interventions, and physical therapy. Although no patient in the original cohort had true varicosities,[1] which would support an alternative diagnosis, 18 of 31 (58%) follow-up patients reported prominent veins or other venous abnormalities, including varicose veins, and 2 were treated with compression stockings or partial venectomy. No patient reported treatment for arm-length discrepancy, macrodactyly, or syndactyly; ability to perform daily tasks was largely unaffected.

We inquired about clinical features associated with other vascular anomalies that manifest over time (Table I).[1,4] One patient's diagnosis was subsequently changed to Sturge-Weber syndrome[4] because of the development of glaucoma with a facial CM. Scoliosis affected both sexes equally and did not correlate with LLD (Fisher's exact test, $P = .4472$), suggesting improper management of LLD was not responsible. No patient reported lipomas, hypotonia, seizures, lower extremity thrombosis, or malignancy. Ten (32%) patients reported psychosocial difficulties, eg, low self-esteem, anxiety, and social discomfort, with no sex predilection. Psychosocial distress most often manifested in early adolescence but was also reported in patients as young as 6 years of age while undergoing laser therapy. Of note, 7 (23%) patients or parents of patients self-reported no overgrowth. Very mild asymmetry can be difficult to ascertain and might have been deemed unreportable, or perhaps soft tissue overgrowth improved with time.

An infant without obvious overgrowth should be given a diffuse capillary malformation diagnosis and evaluated closely for development of overgrowth until adulthood. We found no evidence