Use of platelet-rich plasma in lichen planopilaris and its variants: A retrospective case series demonstrating treatment tolerability without koebnerization

To the Editor: Platelet-rich plasma (PRP) is an autologous concentrate of platelet- and growth factor–rich plasma. Off-label use of PRP has emerged as a promising treatment in nonscarring alopecias including androgenetic alopecia (AGA). By contrast, the efficacy and safety of PRP in the treatment of cicatricial alopecias—including lichen planopilaris (LPP) and its variants, frontal fibrosing alopecia (FFA) and fibrosing alopecia of a pattern distribution (FAPD)—remain unknown. Only 4 reported cases exist describing clinical benefits of PRP in LPP; however, the associated risk of inducing new areas of disease (ie, koebnerization) remains unclear.[1-4] Because LPP koebnerization has previously been reported after more invasive facial and scalp surgeries, we sought to characterize outcomes and adverse events related to PRP in patients with LPP, FFA, and FAPD.[5]

An institutional review board–approved retrospective analysis of patients with LPP, FFA, or FAPD who presented to New York University Langone Health between 2007 and 2018 yielded 10 patients who received PRP injections. Patients were selected for PRP if they were not satisfied with the improvement achieved with previous therapies.

Demographics, comorbidities, concomitant therapies, and adverse reactions were recorded (Table I). Alopecia progression was evaluated using hairline measurements from fixed points, trichometric measurements, and photography. Inflammation was assessed on trichoscopy. Disease status was determined at the initial PRP visit or within 6 weeks prior. Posttreatment status was assessed at the most recent PRP visit or 1 to 3 months after the final PRP injection. Clinical improvement was defined by disease stabilization and/or attenuated symptoms.

The majority of patients were female, with an average age of 57.4 years; 8 of the 10 patients had concomitant AGA. After an average of 4 treatments, 4 of the 10 patients showed improvement of FAPD and FFA, 3 of 10 with LPP and FFA showed neither improvement nor worsening, and 1 in 10 showed LPP disease progression. Two of the 10 patients were of indeterminate status because of inconsistent follow-up or documentation focusing on AGA. The 3 improved patients with FFA showed decreased hair loss and/or inflammation without hair regrowth at the frontal hairline. Seven of 10 patients discontinued PRP after an average of 5 treatments, most commonly because of patient preference and minimal trichometric improvement.

The exact mechanism of action of PRP remains unknown; it is theorized that platelets, growth factors, and anti-inflammatory mediators may promote hair growth. However, concerns exist regarding potential disease exacerbation from proinflammatory mediators in PRP. One patient exhibited LPP progression with increased inflammation and shedding in pre-existing alopecic areas, although this may be attributed to natural disease progression and/or inconsistent follow-up for intralesional triamcinolone injections.

Although 4 patients showed clinical improvement, the relative therapeutic contribution of PRP remains unclear given multitherapy regimens (Table I). Although future prospective studies are necessary to clarify the utility of PRP in cicatricial alopecias, benefits are documented in AGA, a diagnosis also carried by the majority of our patients. Although limited by sample size, our case series highlights the overall tolerability of PRP in LPP and its variants without koebnerization. Therefore, PRP need not necessarily be avoided in patients with concomitant AGA if careful follow-up and monitoring are ensured.

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Table I. Summary of patients with LPP, FFA, and FAPD receiving PRP injections

Patient	Age, y	Sex	Scarring alopecia	Concomitant alopecia	Previous discontinued treatments	Concomitant treatments	Total PRP treatments, n	Disease status at/before initial treatment*	Disease status at/after final treatment*	Treatment discontinuation	Reason for discontinuation	Koebnerization
1	44	M	LPP	AGA	Doxycycline, minocycline, pioglitazone, tacrolimus 0.3% in CC	Finasteride, ILTAC, minoxidil 5% solution, clobetasol 0.05% solution, clobetasol shampoo, ketoconazole shampoo, hydroxychloroquine, naltrexone	4	Active or progressive at first PRP	Active or progressive at 6 weeks after final PRP	Yes	Continued disease progression	None
2	59	F	FFA	AGA	Finasteride, betamethasone 0.1% lotion	Doxycycline, ILTAC, hydrocortisone butyrate 0.1%, minoxidil 5% foam, tacrolimus 0.3% in CC	3	Active or progressive at 6 weeks before PRP	Stable at third PRP	No	Not applicable	None
3	73	F	FAPD	AGA	Doxycycline, ILTAC, hydrocortisone butyrate 0.1%, clobetasol 0.05% solution	Minoxidil 5% foam	3	Stable at 1 month before PRP	Stable at 1 month after final PRP	Yes	No statistically significant increase in hair density	None
4	40	F	LPP	None	Doxycycline, ILTAC, tacrolimus 0.3% in CC, clobetasol 0.05% solution, mycophenolate mofetil, naltrexone	Finasteride, minoxidil 5% solution, hydroxychloroquine, pioglitazone	1	Minimally active at first PRP	Indeterminate	Yes	Patient did not follow up for 1 year and did not continue PRP	None
5†	27	M	LPP	AGA	Doxycycline, ILTAC, minoxidil 5% solution, clobetasol 0.05% solution and foam, ketoconazole shampoo, naltrexone, pioglitazone, fluocinolone acetonide 0.05%	Finasteride, dutasteride, tacrolimus 0.3% in CC, excimer laser, minoxidil 10% solution, spironolactone 5% solution, topical naltrexone	2	Indeterminate	Indeterminate	Yes	Patient preference	None

Continued

Table I. Cont'd

Patient	Age, y	Sex	Scarring alopecia	Concomitant alopecia	Previous discontinued treatments	Concomitant treatments	Total PRP treatments, n	Disease status at/before initial treatment*	Disease status at/after final treatment*	Treatment discontinuation	Reason for discontinuation	Koebnerization
6	79	F	FFA	None	ILTAC	Minoxidil 5% solution, clobetasol 0.05% solution, ketoconazole shampoo	5	Minimally active at 1 month before PRP	Stable at 1 month after final PRP	Yes	Patient preference after improvement of symptoms	None
7	67	F	FAPD	AGA, TE	Doxycycline, clobetasol 0.05% solution	Finasteride, ILTAC, minoxidil 5% solution, tacrolimus 0.3% in CC, fluocinolone acetonide 0.05%	7	Active or progressive at first PRP	Stable at final PRP	No	Not applicable	None
8	50	M	LPP	AGA	None	Finasteride, ILTAC, minoxidil 5% solution, oral minoxidil, tacrolimus 0.3% in CC, clobetasol 0.05% solution, naltrexone	2	Minimally active at first PRP	Active or progressive 3 months after final PRP	Yes	Increased shedding and inflammation in pre-existing disease areas	None
9	63	F	FFA	AGA	None	ILTAC, minoxidil 5% solution, oral minoxidil	2	Stable first PRP	Stable 1 month after final PRP	Yes	No statistically significant increase in hair density	None
10	72	F	FFA	AGA	None	Finasteride, doxycycline, ILTAC, topical minoxidil 5% solution or foam, oral minoxidil, tacrolimus 0.3% in CC, clobetasol 0.05% solution, hydroxychloroquine, pioglitazone	10	Active or progressive at first PRP	Stable at final PRP visit	No	Not applicable	None

AGA, Androgenetic alopecia; *CC*, Cetaphil cleanser (Galderma Laboratories, Fort Worth, TX); *FAPD*, fibrosing alopecia in a pattern distribution; *FFA*, frontal fibrosing alopecia; *ILTAC*, intralesional triamcinolone; *LPP*, lichen planopilaris; *PRP*, platelet-rich plasma; *TE*, telogen effluvium.

*Active or progressive: The presence of subjective measures and objective measures showing inflammation with or without increased hair loss. Minimally active: The presence of subjective measures and/or objective measures showing mild inflammation in the setting of stable hair loss. Stable: No or mild subjective measures plus no objective measures of inflammation seen on trichoscopy in the setting of stable hair loss. Complete remission or resolution: Complete remission of both subjective measures (scalp pruritus, pain, or burning) and objective measures (perifollicular scale, erythema) in the setting of stable hair loss for 4 months or longer.

†Patient 5's visits focused on androgenetic alopecia rather than cicatricial alopecia and showed increased hair density on the midcrown region and decreased hair density on the parietal region of the scalp.

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Factors associated with insurance coverage of tofacitinib for alopecia areata: A retrospective review from an academic institution

To the Editor: It has become increasingly recognized that JAK inhibitors (JAKis) have substantial efficacy in the treatment of alopecia areata (AA).[1] JAKis approved by the US Food and Drug Administration (FDA) are currently under patent in the United States and can be expensive. The high cost of medication is a significant obstacle for patients, and, in the United States, the vast majority rely on insurance coverage to defray costs. AA is an off-label indication for JAKis, often complicating prescription plan coverage. We were interested in investigating the initial and postappeal rates of insurance plan coverage of JAKis for AA at our academic specialty hair clinic.

We conducted a retrospective review of our electronic medical records for patients seen with AA between 2017 through the end of 2019 in the Hair Disorders Clinic in the Department of Dermatology at the University of Iowa who had been prescribed tofacitinib, the most well-studied JAKi for AA,[2-4] over this period of time. Our query showed 42 patients who met these criteria (Table I). One patient was initially authorized for prescription plan coverage of tofacitinib; this patient carried the diagnosis of rheumatoid arthritis, for which tofacitinib is FDA approved. Of patients who were initially denied coverage, 5 patients either did not start the appeal process or stopped the process before a final, definitive decision. Of patients who completed the appeal process, 20 of 36 (55.6%) patients were provided insurance plan coverage after the first appeal, and 2 (5.6%) patients were provided insurance plan coverage after the second appeal. An external review/appeal by an independent physician was offered to those denied coverage after a first or second appeal. Six of the 9 cases externally reviewed (66.7%) were approved for coverage. In total, 29 of 42 patients (69%) received some amount of coverage.

We further examined cases in which patients were unable to obtain insurance coverage. We found that government-sponsored plans were associated with an increased final denial rate (Table I). It is noteworthy that an external review was not available for our patients with Medicaid plans. Excluding patients with pending coverage, patients who halted or did not start the appeal process, and patients with an FDA-approved indication, 60% (3 out of 5) of patients with government-sponsored plans (Medicaid or Medicare) were denied coverage, whereas 7.1% (2/28) of patients with private insurance were denied coverage ($P = .0165$, Fisher's exact test).

Overall, we report here that most private insurance companies will agree to provide some amount of coverage when presented with the growing amount of efficacy data and the risk/benefit profile for tofacitinib for AA if the appeal process options are pursued. A template for our appeal letters is provided in the supplemental materials (available via Mendeley at https://doi.org/10.17632/27dfnj844b.1). Limitations of our study include the focus on a single academic specialty clinic supervised by a sole provider and the limited number of patients. Of note, baricitinib, a JAKi that had previously been reported as a treatment for AA,[5] was recently granted breakthrough status by the FDA and may therefore benefit from an accelerated time frame for an AA indication and, possibly, lower prescription plan denial rates.

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