Using a keyword search within inpatient consult notes, we identified 1023 patients from the inpatient database with documented differential diagnoses containing micronutrient deficiencies (vitamins A, B3, B6, and C; copper; and zinc or corresponding named syndromes such as acrodermatitis enteropathica, scurvy) during hospitalization. Patients were validated by laboratory evidence of micronutrient deficiency within 1 year of hospitalization discharge date. There were 90 cases of micronutrient deficiencies in 65 unique patients (deficiencies in vitamins A [n = 9], B3 [n = 7], B6 [n = 8], and C [n = 10]; copper [n = 10]; and zinc [n = 46]). There were 18 patients with evidence of more than 1 micronutrient deficiency. Control individuals were matched by age ± 3 years, sex, and comorbidity index in a 1-to-1 manner (n = 89).

Demographic characteristics of patients with micronutrient deficiencies were well matched, including age, sex, Charlson comorbidity index, insurance status, and specific organ system comorbidities. Body mass index differed significantly between case patients (27.7 kg/m^2) and control individuals (34.1 kg/m^2). Cases with micronutrient deficiencies were associated with significantly increased length of stay (LOS) (21 vs 8.3 days, $P < .0001$), 30-day mortality rates (23.3% vs 7.9%, $P < .05$), and inpatient dermatology consults (98.9% vs 40.4%, $P < .001$) (Table I). It is possible that these patients had more prominent skin disease. Alternatively, dermatology consultation may more accurately identify micronutrient-related cutaneous disease, consistent with reports of increased diagnostic accuracy and reduced LOS when dermatologists care for hospitalized patients with skin findings.[4,5] Diagnosis of micronutrient deficiencies increased 3-fold (320%) compared with a modest increase in dermatology consult volume (142%) between 2012 and 2017 (Fig 1). Anecdotally, we attribute this to increased diagnostic awareness and value in the dedicated dermatologic inpatient consultation services established in 2014.

This study had multiple limitations. The small number of patients in each micronutrient deficiency subgroup may have affected the analysis. Data were obtained from a single institution and may lack external validity. Future prospective studies should evaluate the prevalence of micronutrient deficiencies among hospitalized patients with skin disease and corroborate the associations described. In conclusion, this study found associations between micronutrient deficiencies in inpatients with dermatologic conditions and poor outcomes, including substantially increased LOS and mortality rates. Micronutrient deficiencies are likely underdiagnosed, and given the differences in outcomes, increased awareness and testing should be considered in inpatients with dermatologic conditions.

Rachel L. Marsh, BA, John Trinidad, MD, MPH, Sabrina Shearer, MD, and Benjamin H. Kaffenberger, MD

From the Department of Internal Medicine, Division of Dermatology, The Ohio State University Wexner Medical Center, Columbus.

Funding sources: None.

Disclosure: Dr Benjamin Kaffenberger is an investigator for Biogen, Celgene, and Eli Lilly and is funded by the Dermatology Foundation. Ms Marsh and Drs Trinidad and Shearer have no conflicts of interest to declare.

IRB approval status: Reviewed and approved by The Ohio State University IRB (study ID no. 2014H0351).

Reprints not available from the authors.

Correspondence to: Rachel L. Marsh, BA, OSU Dermatology, 1328 Dublin Rd, Ste 100, Columbus, OH, 43215

E-mail: rachel.marsh@osumc.edu

REFERENCES

1. Waitzberg DL, Caiaffa WT, Correia MI. Hospital malnutrition: the Brazilian national survey (IBRANUTRI): a study of 4000 patients. *Nutrition*. 2001;17:573-580.
2. Falanga V, Schachner LA, Rae V, et al. Dermatologic consultations in the hospital setting. *Arch Dermatol*. 1994;130:1022-1025.
3. Hu L, Haynes H, Ferrazza D, et al. Impact of specialist consultations on inpatient admissions for dermatology-specific and related DRGs. *J Gen Intern Med*. 2013;28:1477-1482.
4. Kroshinsky D, Cotliar J, Hughey LC, et al. Association of Dermatology consultation with accuracy of cutaneous disorder diagnoses in hospitalized patients: a multicenter analysis. *JAMA Dermatol*. 2016;152:477-480.
5. Milani-Nejad N, Zhang M, Kaffenberger BH. Association of Dermatology consultations with patient care outcomes in hospitalized patients with inflammatory skin diseases. *JAMA Dermatol*. 2017;153:523-528.

https://doi.org/10.1016/j.jaad.2019.10.057

Response of alopecia areata of the beard to oral tofacitinib

To the Editor: Alopecia areata of the beard (BAA) affects 28% of men with alopecia areata.[1] Janus kinase inhibitors have recently emerged as a promising targeted treatment for alopecia areata. Although most studies have focused on scalp hair regrowth, there is, to our knowledge, only 1 case report

Table I. Patient demographics and clinical characteristics (N = 45)

Findings	n (%)	Mean ± SD	Median (range)
Age, y		38.6 ± 12.8	36.0 (20.0-67.0)
AA subtype			
Solitary patch	6 (13.3)		
Multiple patches	13 (28.9)		
Diffuse	8 (17.8)		
AT	4 (8.9)		
AU	14 (31.1)		
SALT score before tofacitinib		62.0 ± 38.9	69.0 (2.0-100.0)
SALT score after tofacitinib		41.7 ± 38.5	29.0 (0.0-100.0)
BAA subtype			
Solitary patch	2 (4.5)		
Multiple patches	24 (53.3)		
Total beard loss	19 (42.2)		
Duration of BAA, mo*		61.2 ± 74.1	28.0 (3.0-324.0)
Dose of oral tofacitinib, mg		7.2 ± 4.0	7.5 (1.0-20.0)
Duration of oral tofacitinib treatment, mo		15.5 ± 13.8	13.0 (3.0-86.0)

AA, Alopecia areata; *AT,* alopecia totalis; *AU,* alopecia universalis; *BAA,* alopecia areata of the beard; *SALT,* Severity of Alopecia Tool; *SD,* standard deviation.
*Duration of current episode of BAA not documented in 6 patients.

Table II. Response to treatment with oral tofacitinib (N = 45)

	Degree of beard regrowth		
	None	Partial	Complete
Patients, n (%)	16 (35.6)	19 (42.2)	10 (22.2)
BAA subtype, n (%)			
Solitary patch	2 (4.5)	0 (0.0)	0 (0.0)
Multiple patches	8 (17.8)	9 (20.0)	7 (15.6)
Total beard loss	6 (13.3)	10 (22.2)	3 (6.7)
Degree of scalp hair regrowth, n (%)			
None	14 (31.1)	3 (6.7)	1 (2.2)
Partial	2 (4.5)	15 (33.3)	3 (6.7)
Complete	0 (0.0)	1 (2.2)	6 (13.3)
Duration of BAA (months), mean ± SD	58.1 ± 78.5	45.1 ± 58.1	93.2 ± 72.5

BAA, Alopecia areata of the beard; *SD,* standard deviation.

documenting beard regrowth with ruxolitinib in a patient with alopecia universalis.[2]

We retrospectively reviewed the records of all male patients with scalp alopecia areata (SAA) who were treated with oral tofacitinib at a specialist hair clinic between July 2016 and August 2019. The inclusion criteria for this study were age of 18 years or older, BAA, and treatment with oral tofacitinib for at least 3 months. The Severity of Alopecia Tool score was used to quantify scalp hair loss. Beard regrowth (none, partial, or complete) was measured by an independent observer evaluation of global photographs. Data entry and analysis were performed with SPSS Statistics, version 24 (IBM, Armonk, NY).

A total of 45 patients met the inclusion criteria (Table I). Nineteen men had total beard loss, 24 had multiple discrete patches, and 2 had a solitary patch of BAA. Ten men achieved complete beard regrowth after 5.0 to 28.0 months of treatment (mean, 16.0 months) (Table II). The mean disease duration among complete beard responders was 93.2 months (range, 12.0-252.0 months). Overall, 60% of men who achieved complete beard regrowth also achieved complete scalp hair regrowth. Of the 19 patients with partial beard regrowth, 15 achieved partial and 1 achieved complete scalp hair regrowth. Of the 16 patients with no beard regrowth, 14 had no regrowth and 2 had partial regrowth of scalp hair.

There were no serious adverse events. Mild adverse events were seen in 10 patients and included upper respiratory infections, elevated liver

transaminases, fatigue, and acne. None required treatment cessation or dose modification.

BAA is associated with a high prevalence of anxiety and depressive symptoms.[1-3] Some religions, including Islam, Orthodox Judaism, and Sikhism, require men to grow a full beard.[3] One of our patients regularly competes in beard championships, and the loss of more than 50% of his beard as a result of BAA caused significant distress (Supplemental Figure 1; available at https://data.mendeley.com/datasets/nh2h672rhc/1).

Our results show a strong correlation between the extent of beard and scalp hair regrowth, suggesting that these hair-bearing sites may respond similarly to oral tofacitinib. In contrast to studies of SAA that showed an inverse association between disease duration and treatment response,[4,5] no such association was observed in our BAA cohort. On the contrary, patients with complete beard regrowth had a longer mean disease duration. Patient age, BAA phenotype (patchy or total beard loss), and duration of treatment did not influence the degree of beard response. As with our BAA cohort, age did not predict improvement in Severity of Alopecia Tool score in 90 patients with severe SAA treated with oral tofacitinib.[4]

Our findings suggest that oral tofacitinib might be a promising therapeutic option for patients with BAA. We suggest that prospective clinical trials evaluating oral Janus kinase inhibitors in alopecia areata should seek to include beard assessment in their protocols to determine their effectiveness for the treatment of BAA.

Karolina Louisa Suzanna Kerkemeyer, MBBS (Hons), BHlthSci, Jared Marc John, MBBS, MPH, Rodney Sinclair, MBBS, MD, FACD, and Bevin Bhoyrul, MBBS, MRCP

From Sinclair Dermatology, East Melbourne, Victoria, Australia.

Funding sources: Dr Bhoyrul was supported by The Geoffrey Dowling Fellowship from the British Association of Dermatologists.

Disclosure: Dr Sinclair has professional services associated with Leo Pharma, Amgen, Novartis, Merck & Co, Celgene, Coherus BioSciences, Janssen, Regeneron, MedImmune, GlaxoSmithKline, Cutanea, Samson Clinical, Boehringer Ingelheim, Pfizer, MSD, Oncobiologics, Roche, Eli Lilly, Bayer, and Sun Pharma. Ms Kerkemeyer, Mr John, and Mr Bhoyrul have no conflicts of interest to declare.

IRB approval status: Not applicable.

Reprint requests: Karolina Louisa Suzanna Kerkemeyer, MBBS (Hons), BHlthSci, Level 3, 2 Wellington Parade, East Melbourne, Victoria 3002, Australia

E-mail: Karolina.Kerkemeyer@sinclairdermatology.com.au

REFERENCES

1. Cervantes J, Fertig RM, Maddy A, et al. Alopecia areata of the beard: a review of the literature. *Am J Clin Dermatol*. 2017;18:789-796.
2. Ramot Y, Zlotogorski A. Complete regrowth of beard hair with ruxolitinib in an alopecia universalis patient. *Skin Appendage Disord*. 2018;4:122-124.
3. Saceda-Corralo D, Grimalt R, Fernandez-Crehuet P, et al. Beard alopecia areata: a multicentre review of 55 patients. *J Eur Acad Dermatol Venereol*. 2017;31:187-192.
4. Liu LY, Craiglow BG, Dai F, et al. Tofacitinib for the treatment of severe alopecia areata and variants: a study of 90 patients. *J Am Acad Dermatol*. 2016;76:22-28.
5. Phan K, Sebaratnam DF. JAK inhibitors for alopecia areata: a systematic review and meta-analysis. *J Eur Acad Dermatol Venereol*. 2019;33:850-856.

https://doi.org/10.1016/j.jaad.2019.10.058

The correlation of immune status with ultraviolet radiation–associated mutations in cutaneous squamous cell carcinoma: A case-control study

To the Editor: There are an estimated 1 million cases of cutaneous squamous cell carcinoma (SCC) per year in the United States.[1] Ultraviolet (UV) radiation contributes to the pathogenesis of SCC and causes characteristic pyrimidine-pyrimidine dimer mutations.[2] Pickering et al[3] performed whole-exome sequencing of 39 aggressive SCCs and found 65% of mutations to be UVB associated. In addition to UV radiation, immunosuppression increases SCC risk. Patients with organ transplants are 100 times more likely to develop SCC secondary to underlying immunosuppression and toxicity from chemotherapy.[4] However, the pathogenesis of SCC in immunocompromised patients remains to be fully elucidated. We hypothesized that there are fewer UVB-associated mutations in immunocompromised patients than in immunocompetent patients with SCC.

We performed next-generation sequencing using a hotspot mutation panel covering 76 cancer-associated genes (Vela Diagnostics, Singapore) in a cohort of 20 patients with high-risk SCC (Table I). We categorized mutations as being caused by UVA radiation, UVB radiation, reactive oxygen species