treatment, including drugs targeting the clonal cell population, are warranted.

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Funding sources: None.

Conflicts of interest: None disclosed.

IRB approval status: Not applicable.

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https://doi.org/10.1016/j.jaad.2020.09.089

Choosing between isotretinoin and acitretin for epidermal growth factor receptor inhibitor and small molecule tyrosine kinase inhibitor acneiform eruptions

To the Editor: We read with great interest Andrews et al's[1] article on the therapeutic benefits of oral retinoids on acneiform eruptions secondary to epidermal growth factor receptor (EGFR) inhibitors and mitogen-activated protein kinase inhibitors, and want to reinforce their findings.

After approval by the Mayo Clinic's institutional review board, a search of Mayo Clinic Rochester, Arizona, and Florida was performed for patients who were prescribed isotretinoin or acitretin and concomitant EGFR inhibitors or small molecule tyrosine kinase inhibitors or who were diagnosed with an acneiform eruption. A total of 766 possible cases were returned. Chart review was performed, and 6 patients met the inclusion criteria of isotretinoin or acitretin for the treatment of an EGFR inhibitor/small molecule tyrosine kinase inhibitor acneiform eruption. Information regarding demographics and treatment course were obtained. Estimated rash improvement was based on clinical notes and clinical photographs. Minimal improvement was defined as 1% to 35% improvement in body surface area, moderate improvement was defined as 36% to 65% improvement, and significant improvement was defined as >66% improvement.

Of the 6 patients, 4 were treated with isotretinoin, 1 with isotretinoin and subsequently acitretin, and 1 with acitretin (Table I). Patients were initially treated with topical antibiotics, topical steroids, and oral tetracyclines. Two patients remained on oral tetracyclines concomitantly with oral retinoids. Retinoid doses ranged from 10 mg to 60 mg daily for isotretinoin and 10 mg daily or 25 mg every other day for acitretin. Duration of treatment ranged from 1 month to >1 year. At least some clinical improvement was noted in every patient. Both acitretin patients had minimal improvement; 20% and 80% of isotretinoin patients had moderate or significant improvement, respectively. One isotretinoin patient developed

Table I. Acneiform cases, their chemotherapy, retinoid, and rash improvement

Age/sex	Chemotherapy drug	Initial reaction grade	Retinoid	Duration of retinoid therapy	Rash improvement	Concomitant tetracycline use	Reduction in chemotherapy drug dose
70/F	Erlotinib	Grade 2	Isotretinoin 20 mg daily or qod	7-8 months	Moderate	No	No
59/M	Cetuximab	Grade 3	Isotretinoin 20 mg daily	6 months	Significant	Yes	No
62/M	Cetuximab	Grade 3	Isotretinoin 60 mg daily	3 months	Significant	No	Yes
37/M	Cetuximab	Grade 3	Isotretinoin 10 mg daily	1-2 months	Significant	No	Yes
73/M	Erlotinib	Grade 3	Isotretinoin 40 mg daily	10 months	Significant	No	No
	Cetuximab and irenotecan	Grade 2	Acitretin 25 mg qod	6 months	Minimal	No	No
67/M	Afatinib	Grade 3	Acitretin 10-25 mg daily	7 months	Minimal	Yes	Yes

F, Female; *M*, male; *qod*, once every other day.

retinoid dermatitis, treated with topical corticosteroids. There were no other significant adverse effects associated with retinoid use in these patients.

In our retrospective study, treatment with both isotretinoin and acitretin led to clinical improvement in acneiform eruptions secondary to EGFR inhibitors, similar to the experience by Andrews et al[1] and others.[2-4] However, isotretinoin led to greater improvement in clinical outcomes compared with acitretin, although our sample size is small. If this observation is validated by others, an explanation might be the striking difference in sebostatic activity with isotretinoin compared with other synthetic retinoids.[5] This study further reinforces the use of oral retinoids in treating EGFR and small molecule tyrosine kinase inhibitors.

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Funding sources: None.

Conflicts of interest: None declared.

IRB approval status: Reviewed and approved by Mayo Clinic IRB approval #19-006401.

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https://doi.org/10.1016/j.jaad.2020.09.090

An open-label study evaluating the efficacy of abatacept in alopecia areata

To the Editor: Alopecia areata (AA) is a form of nonscarring autoimmune hair loss. Our previous genome-wide association study revealed strong genetic susceptibility at the cytotoxic T lymphocyte–associated protein 4 (CTLA4) locus in patients with AA.[1] CTLA4 is an immune checkpoint receptor that has emerged as a clinically important target controlling immune responses in both cancer and autoimmunity. Abatacept is a CTLA4–immunoglobulin