Letter to the Editor

Abatacept-Induced Psoriasis

To the Editor:

A 63-year-old woman presented with a history of rheumatoid arthritis of 7 years' duration that was treated with abatacept after treatment failure with adalimumab and etanercept, respectively. The patient presented to our clinic with a history of a rash involving the palms of her hands and soles of her feet of 4 months' duration (Figure). Of note, she was started on abatacept approximately 2 months prior to the development of the rash and had been off tumor necrosis factor α (TNF- α) inhibitors for approximately 6 months. She was seen by a physician who assumed it was tinea pedis and was treating it with topical antifungal medication. She denied having lesions anywhere else on her body. She initially treated the lesions with hydrocortisone but

switched to triamcinolone because hydrocortisone was ineffective. The patient received a biopsy consistent with pustular psoriasis showing hyperkeratosis with parakeratosis and epidermal acanthosis with elongation of rete ridges.

Psoriasis is a chronic inflammatory skin disease thought to be driven by T cell–mediated cytokine production leading to keratinocyte proliferation and angiogenesis. The development of psoriasis has been well-described in the literature with the use of TNF- α inhibitors. 1,2 Additionally, the development of psoriasis has been reported in patients with TNF- α inhibitors and subsequent abatacept. 3,4 We suspect that by attenuating the second signal in regulatory T cells in individuals with a genetic predisposition, such as patients with polymorphisms in the genes



Side of the foot (A), sole of the foot (B), palms of the hands (C), and tops of the feet (D) 8 weeks after treatment with abatacept was initiated.

encoding IL-23R and IL-12B, an imbalance of $T_{\rm H}17$ helper T cells may occur, promoting development of psoriasis through cytokine production,⁵ likely similar to what has been described in TNF- α mouse models with an increase in $T_{\rm H}17$ cells leading to an increase in IL-22.^{6,7} Our patient's psoriasis was recalcitrant to topical therapy and she decided to stay on abatacept for her rheumatoid arthritis because her disease activity responded extremely well.

Sincerely, David Silverman, MD Alyce Oliver, PhD, MD

From the Department of Internal Medicine, Georgia Health Sciences University, Augusta.

The authors report no conflict of interest.

REFERENCES

1. Wollina U, Hansel G, Koch A, et al. Tumor necrosis factor-alpha inhibitor-induced psoriasis or psoriasiform

- exanthemata: first 120 cases from the literature including a series of six new patients. Am J Clin Dermatol. 2008;9:1-14.
- Ko JM, Gottlieb AB, Kerbleski JF. Induction and exacerbation of psoriasis with TNF-blockade therapy: a review and analysis of 127 cases. J Dermatolog Treat. 2009;20:100-108.
- 3. Jost C, Hermann J, Caelen Lel-S, et al. New onset psoriasis in a patient receiving abatacept for rheumatoid arthritis [published online ahead of print March 6, 2009]. BMJ Case Rep. pii:bcr09.2008.0845.
- 4. Brigant F, Clavel G, Chatelain D, et al. A case of generalized guttate psoriasis induced by etanercept with relapse after abatacept [letter]. *Dermatol Online J.* 2011;17:11.
- Liu Y, Helms C, Liao W, et al. A genome-wide association study of psoriasis and psoriatic arthritis identifies new disease loci. PLoS Genet. 2008;4:e1000041.
- Ma HL, Napierata L, Stedman N, et al. Tumor necrosis factor alpha blockade exacerbates murine psoriasis-like disease by enhancing Th17 function and decreasing expansion of Treg cells. Arthritis and Rheum. 2010;62:430-440.
- 7. Ouyang W, Kolls JK, Zheng Y. The biological functions of T helper 17 cell effector cytokines in inflammation. *Immunity*. 2008;28:454-467.

118 CUTIS® WWW.CUTIS.COM