parasite induce an immune-mediated inflammatory response. The enzyme-linked immunoelectrotransfer blot of serum (not obtained) is the serologic test of choice, with higher sensitivity and specificity than ELISA; however, both tests are unreliable in patients with 1 degenerating cysticercus (as in our patient) or calcified granulomas.

Urticarial vasculitis is an uncommon clinicopathologic entity distinct from acute urticaria based on dermatopathologic findings of vasculitis. Clinically, urticarial vasculitis presents with painful pruritus and erythematous wheals lasting longer than 24 hr, followed by a residual hyperpigmentation. Our patient’s differential diagnosis also included *Cysticercus cellulosae* cutis (painless subcutaneous nodules with larval cysts on biopsy) and Sweet syndrome (associated with pyrexia and neutrophilic infiltrate on histology without evidence of leukocytoclastic vasculitis). However, biopsy results combined with urticarial lesions on exam solidified a diagnosis of urticarial vasculitis.

To our knowledge, urticarial vasculitis associated with *T. solium* infection in which the urticarial vasculitis resolved after neurosurgical removal of the cyst and treatment with albendazole has not been reported. Physicians should continue to look for obscure causes of urticarial vasculitis.

Sheila Shaigany, BS, Ellen Dabela, MD, MBA, Andrew F. Teich, MD, PhD, Sameera Husain, MD, and Marc E. Grossman, MD, FACP

Department of Dermatology and Dermatology Consultation Service, Department of Neuropathology, and Department of Dermatopathology, Columbia University Medical Center, New York, New York

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Correspondence to: Sheila Shaigany, BS, New York Presbyterian/Columbia University Medical Center Department of Dermatology, 161 Fort Washington Avenue, 12th Floor, New York, NY 10032 E-mail: ss4172@columbia.edu

REFERENCES


Ulcerations within striae distensae associated with bevacizumab therapy

To the Editor: We present the case of a 29-year-old woman with glioblastoma multiforme (GBM) who
developed ulcers localized to corticosteroid-induced striae.

GBM in the setting of severe headaches and nausea was diagnosed. After complete resection of the mass, she was started on a course of dexamethasone to which daily temozolomide 75 mg/m² and every other week bevacizumab 10 mg/kg were added 4 months later. She also underwent localized radiation therapy. Soon after starting this combination regimen, she noted small tender ulcers on her abdomen and arms.

Physical examination revealed scattered saucer-shaped ulcerations confined to the striae on her abdomen and arms (Fig 1). Because bevacizumab-induced ulceration was suspected and given her degree of discomfort, bevacizumab was discontinued. The patient remained on temozolomide and dexamethasone. The ulcers significantly improved within 1 month with local wound care consisting of white petrolatum and nonstick dressings.

GBM is the most aggressive primary brain tumor in adults. Standard treatment for GBM after surgical resection includes radiotherapy in conjunction with temozolomide, followed by adjuvant temozolomide alone. Bevacizumab, a humanized monoclonal antibody against vascular endothelial growth factor (VEGF), received FDA approval in 2009 for the treatment of recurrent GBM.1

Delayed healing of surgical wounds is a well-known adverse effect of bevacizumab, in addition to other cutaneous adverse effects including painful hand-foot syndrome, exfoliative dermatitis, skin discoloration, and xerosis.2,3 There have been several reports4,5 of ulcer formation within corticosteroid-induced striae in patients receiving bevacizumab for advanced GBM. Striae often develop in the setting of chronic corticosteroid use secondary to epidermal atrophy and dermal changes from decreased collagen synthesis. While the exact mechanism of ulceration is unknown, we hypothesize that bevacizumab contributes by impairing striae healing through VEGF inhibition, rendering them persistently weakened and susceptible to ulceration from trauma. Additionally, bevacizumab may increase thrombotic events via endothelial cell disruption, leading to the formation of vascular microthrombi and regions of skin necrosis within striae.4 Striae are vascular in nature, making them more susceptible to these effects. Once an ulcer forms, bevacizumab inhibits healing through similar mechanisms.3,5

Proper identification and management is crucial once a patient presents with ulcerated striae. When possible, cessation of bevacizumab may lead to resolution of these areas. However, there are situations when its withdrawal is not feasible. In such cases, dermatologists must be aware of the implications of this regimen and how to prevent further ulcer formation in susceptible patients. In patients started on bevacizumab in the setting of preexisting striae, the striae should be kept moisturized and protected from trauma.3 Striae must be closely monitored for ulceration and when identified, early wound care is key. Several management techniques have been reported and include the use of silicone based or colloid dressings with the addition of silver foam or dressings if signs of infection are apparent.5 This case reinforces that ulcers localized to striae are a rare but possible side effect of combined therapy with dexamethasone and bevacizumab, and highlights the importance of early recognition and proper management in these patients.

Sara A. Farber, BA,a Sara Samimi, MD,b and Misha Rosenbach, MDb

Perelman School of Medicinea and Department of Dermatology, b University of Pennsylvania, Philadelphia

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Correspondence to: Misha Rosenbach, MD, Assistant Professor of Dermatology and Internal Medicine, University of Pennsylvania, Perelman School of Medicine, 3400 Spruce Street, 2nd Floor, Maloney Building, Philadelphia, PA 19104

E-mail: Misha.rosenbach@uphs.upenn.edu

REFERENCES
Psoriasis as a manifestation of HIV-related immune reconstitution inflammatory syndrome

To the Editor: Immune reconstitution inflammatory syndrome (IRIS) is a paradoxical exacerbation of a pre-existing condition or emergence of a previously unknown disease occurring in an HIV-infected patient after initiating combined antiretroviral therapy (cART). IRIS begins days to months after starting cART, and is associated with decreasing viral load.\(^1\)

The skin accounts for 52% to 78% of presentations, including a range of infectious, inflammatory, and neoplastic conditions.\(^2\) Inflammatory dermatoses such as eosinophilic folliculitis, seborrhea, and acne vulgaris have all been reported.\(^3,4\) Here we present, to our knowledge, the first report of paradoxical worsening of psoriasis as a presentation of IRIS.

A 63-year-old man with type 2 diabetes mellitus, hypertension, hepatitis C, HIV diagnosed in 1997, and a history of mild, untreated less than 1% total body surface area psoriasis presented to dermatology 1 month after starting a new cART, Stribild (elvitegravir, tenofovir, cobicistat, emtricitabine). The patient was switched 1 month prior from his previous cART regimen of Truvada (emtricitabine and tenofovir disoproxil fumarate), darunavir, and ritonavir, to Stribild, a once-a-day cART, because of concerns of medication noncompliance. His most recent CD4 count, 5 months before presentation, was 204 cells/µL and his viral load was 177,857 copies/mL. Skin examination demonstrated confluent erythema, edema, and focal areas of platelike desquamation over the palmar surfaces of both hands; thick sharply demarcated plaques with silvery scale over the elbows; and an eryhematous patch over the sacrum, with sparing of the feet and nails, affecting approximately 10% total body surface area. Despite reports of mild joint stiffness in his hands, plain film x-rays did not show signs of psoriatic arthritis. A repeated CD4 count was 138 cell/µL and viral load was 257 copies/mL. He met the proposed diagnostic criteria for IRIS, with worsening of a pre-existing condition (psoriasis) and a concomitant greater than log 10 reduction in viral load.\(^5\) He was started on topical psoralen plus ultraviolet A light 3 times per week and clobetasol ointment 0.05% twice daily, with improvement over the course of 2 months, eventually returning to less than 1% affected total body surface area. Of interest, 1 week before worsening psoriasis, he also developed herpes zoster involving the right leg and buttock, which was resolving at the time of his initial presentation to dermatology.

At the 12th World AIDS Conference in Geneva in 1999, French et al\(^6\) reported numerous conditions worsening after starting cART and introduced the term “immune restoration disease,” which is now more commonly termed “immune reconstitution inflammatory syndrome.” Over 15 years after the first reports, IRIS remains a well-described but poorly understood phenomenon. There is emerging evidence that one of the immunopathogenic mechanisms of IRIS involves the rapid and dysregulated shift from the T helper (Th)2-predominant state of advanced HIV infection to the Th1- and Th17-dominant state of immune recovery. It is intriguing that psoriasis, a Th1- and Th17-mediated inflammatory condition, has not, to our knowledge, until now been reported to undergo an IRIS reaction, despite being a relatively common HIV-associated dermatosis.

Shivani V. Tripathi, MD, Kieron S. Leslie, DTM&H, FRCP, Toby A. Maurer, MD, and Erin H. Amerson, MD

Department of Dermatology, University of California, San Francisco

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Correspondence to: Erin H. Amerson, MD, Department of Dermatology, University of California, San Francisco, 1701 Divisadero St, San Francisco, CA 94115

E-mail: amerson@derm.ucsf.edu

REFERENCES
