Discussion | The term “saxophone penis” generally describes a physical examination finding of pronounced curvature and deformity of the penis along its longitudinal axis. Another synonym is “ram horn penis.” The exact mechanism of this deformity is unknown and may vary depending on acute or chronic occurrence. One proposed explanation describes contraction, and in some cases fibrosis, of the connective tissue on the dorsal side, creating a dependent ventral side, which has richer vascularity and can lead to edema and exaggerated dorsal curvature.

While there are many potential causes of saxophone penis findings, they are generally limited to conditions affecting penile lymphatics or vessels. Infectious causes include lymphogranuloma venereum, S aureus, and mycobacterial infections. Other potential causes may include primary lymphedema and, theoretically, trauma secondary to penile fracture. Although our patient had superficial infection with S aureus, his dramatic improvement despite lack of antimicrobial treatment would support an inflammatory reaction to imiquimod as opposed to a primary bacterial infection.

Imiquimod, 5%, cream is a topical immunologic therapy approved for the treatment of external genital warts, superficial basal cell carcinomas, and actinic keratosis. A wide spectrum of cutaneous adverse effects have been associated with topical imiquimod through an increase in T helper 1 cytokines, including hypopigmentation and vitiligo, lichen planopilaris, lupus erythematosus-like reactions, pemphigus-like skin lesions, urticaria, and angioedema.

Before initiating imiquimod therapy, physicians should thoroughly counsel patients about the potential adverse effects and should provide specific guidelines as to when to contact the prescribing physician. As with our patient, the use of imiquimod resulting in saxophone penis deformity may cause considerable physical discomfort and emotional distress and in some cases may be functionally incapacitating if left untreated.

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Telaprevir-Induced Acquired Perforating Dermatosis

Since its approval in 2011, a novel serine protease inhibitor, telaprevir, has been increasingly used in combination with pegylated interferon and ribavirin as an effective treatment for chronic hepatitis C virus (HCV) infection. However, it was found to cause cutaneous eruption, mostly pruritic eczematous dermatitis, in 56% of patients as opposed to 34% of patients taking peginterferon and ribavirin alone. Moreover, severe adverse cutaneous events were reported to be more frequent in telaprevir-treated patients (3.7% vs 0.4%). We report herein the first case to our knowledge of acquired perforating dermatosis (APD) induced by telaprevir in a patient with HCV infection.

Report of a Case | A man in his 50s with HCV infection was referred for pruritic ulcerated papules on the lower legs. The lesions appeared 3 weeks after the patient started treatment with telaprevir (2250 mg/d orally) (Janssen-Cilag) in combination with ribavirin (1200 mg/d orally) (Hoffman-La Roche) and pegylated interferon alfa-2a (180 µg/wk subcutaneously) (Hoffman-LaRoche). He had previously been treated with peginterferon and ribavirin without cutaneous adverse effects. Blood test results for human immunodeficiency virus (HIV) were negative. Serum α-fetoprotein level was normal, and abdominal computed tomography did not show evidence of hepatocellular carcinoma. He had no diabetes mellitus or chronic kidney failure and was taking no other medication.

Physical examination revealed extensive xerosis and ulcerated papular and nodular lesions on the lower legs, each with an inflammatory border and a central keratotic plug (Figure 1). Histologic examination of a lesion specimen revealed a focal epidermal ulceration covered by a hyperkeratotic crust containing necrotic debris and inflammatory cells. Collagen bundles and elastic fibers oriented perpendicularly to the surface were extruded through the epidermis (Figure 2). A diagnosis of APD was rendered. Telaprevir therapy was discontinued, and the patient was treated with 1 application per day of betamethasone dipropionate and petroleum jelly (Vaseline; Uniliver), with slow but progressive improvement observed within 2 weeks and no new lesions. All lesions entirely resolved within 2 months, leaving pigmented and atrophic scars.

Discussion | In our observation, APD was quite likely secondary to telaprevir treatment, given the delayed onset of lesions, the improvement after treatment discontinuation, and the prior benign treatment course with pegylated interferon alfa-2a and ribavirin.

Acquired perforating dermatosis has been associated with several diseases, including diabetes mellitus, chronic renal failure, malignant conditions and AIDS. Rare observa-
tions of APD associated with HCV infection have been reported, but in all cases, the eruption was related to either renal failure or hepatocellular carcinoma.2,3 Drug-induced APD is exceptional and has mostly been described for new biologics like natalizumab, gefitinib, infliximab, etanercept, bevacizumab, erlotinib, and sorafenib.4 Although telaprevir-induced APD has never been described, 2 cases of APD induced by indinavir, another protease inhibitor, were described in patients with HIV in the absence of diabetes mellitus or chronic kidney failure.5

The etiopathogenic mechanisms involved in APD are uncertain. However, it is widely believed that in susceptible individuals, APD is a cutaneous response to superficial trauma caused by scratching. Hypoxia, accumulation of fibronectin, and increased levels of matrix-degrading metalloproteins may also contribute to the transepidermal elimination of dermal collagen.4 We suggest that in susceptible individuals, APD is a potential severe cutaneous adverse effect of telaprevir brought on by the itching and repeated scratching often caused by this protease inhibitor.

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COMMENT & RESPONSE

Balancing the Cardiovascular Risk and Dermatologic Hazard in Patients With Hypertension

To the Editor We greatly appreciated the article by Wu et al,1 which showed the independent association between long-term hypertension, long-term administration of β-blockers, and incident psoriasis in a large prospective cohort. However, although the study presented new evidence of the association, no management advice was proposed. How should patients with hypertension, who have the propensity to have psoriasis, be treated?

Psoriasis most often arises in people younger than 40 years.2 Hypertension in young patients is characterized by sympathetic activation; thus, β-blockers provide satisfactory clini-