it is reversible. The principle approach is to avoid or substitute the drugs wherever possible, and to maintain strict dental hygiene regimes. Antibiotics can also be used (metronidazole, clarithromycin, azithromycin), and in resistant cases a gingivectomy may be performed by means of scalpel, electrosurgery, cryosurgery, or carbon dioxide laser, although the condition tends to recur within 3 to 12 months.[1,2]

Everolimus is a new sirolimus derivative immunosuppressant with a better bioavailability and shorter half-life. It is a potent proliferation signal inhibitor operating via the m-TOR receptors. It is used in prophylaxis to prevent rejection of solid organ transplants in adult recipients with a low to moderate immunological risk.[5]

The most common adverse reactions associated with this group of drugs are hyperlipidemia, thrombocytopenia, delayed healing, delayed recovery from acute tubular necrosis in kidney transplantation, reduced testosterone levels, increased proteinuria, pneumonitis, headache, asthenia, joint pain, lymphocele and, in combination with cyclosporine, an increase in hemolytic uremic syndrome, nephrotoxicity, and systemic hypertension.

Use of the drug has also been associated with adverse skin reactions: mouth ulcers (60%), gingivitis (20%), chronic labial fissures (11%), epitaxis (60%), acneiform rashes (46%), folliculitis of the scalp (26%), suppurative hidradenitis (12%), chronic edema and sclerodermiform changes (55%), angioedema (15%), nail disorders (74%), and periungual infections (16%).[6]

We consider this case worthy of discussion as we have found no other reports of gingival hyperplasia secondary everolimus amongst the many descriptions of such drug reactions in our review of the literature.

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S. Pérez-Barrio,* M.R. González Hermosa, and J.L. Díaz-Pérez

Servicio de Dermatología, Hospital de Cruces, Baracaldo, Vizcaya, Spain

*Corresponding author.
E-mail address: spbarrio@hotmail.com (S. Pérez-Barrio).

Contact Allergic Dermatitis to Quinine in an Anti-hair Loss Lotion

Dermatitis alérgica de contacto a quinina por una loción capilar anticaída

To the Editor:

The use of anti-hair loss lotions for treating androgenic alopecia can occasionally cause pruritus and desquamation of the scalp. The most common causes include irritant contact dermatitis, allergic contact dermatitis, and even exacerbation of seborrheic dermatitis. Allergic contact dermatitis caused by anti-hair loss lotions has been widely described in the literature and is generally associated with the use of minoxidil or propylene glycol used as excipients, the latter being the causative allergen in most cases.[1] Allergic contact dermatitis to the quinine contained in anti-hair loss lotion, as in the present case, is much less common.

The patient was a 73-year-old woman with no relevant personal history, referred by her health-area dermatologist for a highly pruritic and impetiginous papulovesicular dermatitis of the scalp, ears, and face that had begun several days earlier (Figure 1). The patient reported the topical application of Bio-anagenol shampoo, Lacovin (2% minoxidil), and Kavel anti-hair loss lotion over a period of 13 years for androgenic alopecia. She denied having used other topical, cosmetic, or therapeutic products, did not associate her symptoms with any occupational activity, and had no history of atopy or other skin diseases. Treatment was prescribed with corticosteroid solution (clobetasol propionate twice a day), prednisone (30 mg/d), antihistamines, and oral antibiotics for a week, leading to complete resolution of the symptoms. An open test subsequently performed on the patient's forearm with Lacovin (2% minoxidil) was negative, while Kavel anti-hair loss lotion was positive (++) on the third day. A patch test with Kavel anti-hair loss lotion applied to the back was positive (++). The other patch tests with a standard series from the Spanish Contact Dermatitis

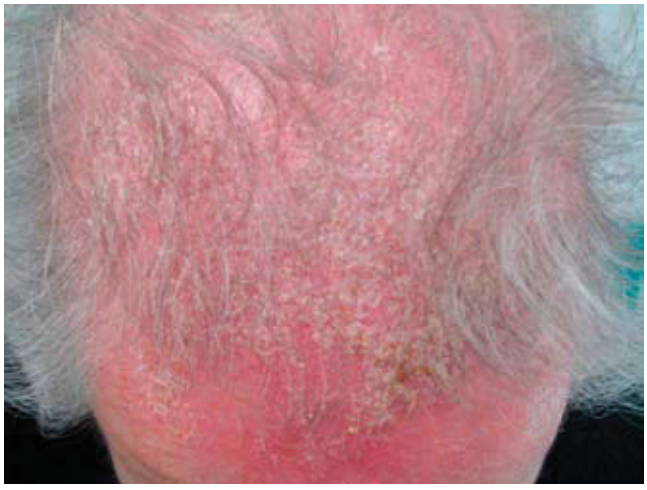

Figure 1 Erythematous plaque with desquamation and honey-colored crusts on the scalp.

Figure 2 Patch test (++++) for 1% quinine hydrochloride at 48 h.

Table Active Ingredients Tested

Butylene glycol, 1% aq	(−)
Cegaba liquid, 1% aq	(−)
Dekaben-Phenonip, pure aq	(−)
DL Panthenol, 1% aq	(−)
Disodium edetate, 1% aq	(−)
Denatured ethanol, 1% aq	(−)
Hydrolite 5, 1% aq	(−)
Quinine hydrochloride, 1% aq	(+ + + +)
Sephora root powder, 1% aq	(−)
Taurine, 1% aq	(−)
Transcutol CG 1% aq	(−)
Zinc gluconate, 1% aq	(−)

Abbreviation: aq, aqueous solution.

and Skin Allergy Research Group (GEIDAC), 2% minoxidil and 5% propylene glycol, both in petroleum jelly, were negative at 48 and 96 hours. The manufacturer was contacted and provided the individual components of Kavel anti-hair loss lotion, with which further patch tests were performed (Table). Patches were placed on the back, removed after 48 hours, and read following the recommendations of the International Contact Dermatitis Research Group (ICDRG). Positivity (++++) for quinine hydrochloride, 1% aqueous solution, was observed at 48 and 96 hours (Figure 2). This allergen was negative in 20 controls. The patient remained asymptomatic after avoiding contact with Kavel lotion.

Quinine is an alkaloid obtained from the bark of several species of cinchona tree[2] and was already part of the first patch-test standard series proposed by Poul Bonnevie in the early 20th century. It was later withdrawn due to scant usage and low frequency as an allergen.[3] Quinine was used therapeutically for a variety of conditions, such as myotonia, nocturnal cramps, and some cases of chloroquine-resistant falciparum malaria.[2]

Allergic contact dermatitis to the quinine contained in anti-hair loss lotions has been previously reported in the literature, despite being infrequent and occasionally difficult to demonstrate.[4] This is the first documented case at the Skin Allergy Unit of the Dermatology Department at Hospital General Universitario de Valencia (Spain) with experience in over 10 000 patients who have undergone patch testing. In addition to sensitization after using anti-hair loss lotions containing quinine, there have been reports of other forms of contact sensitization to this allergen either after applying chemical contraception containing quinine as a spermicide or after the handling of this substance by employees of the pharmaceutical industry.[2] Quinine may cause sensitization after exposure for 2 or 3 months, or even after many years of use as is the case here.[5] Photoallergic reactions can also occur.[6] The possibility of such reactions in this patient was not ruled out due to the intensely positive results of the patch tests and because systemic reactions have been observed in previously sensitized individuals after the consumption of small amounts of quinine, such as the content of a tonic water. There is even an anecdotal case of toxic epidermal necrolysis after drinking a gin and tonic.[2,7]

In conclusion, the case reported here indicates that quinine must be borne in mind as an “old allergen” in patients with eczema of the scalp using anti-hair loss products for cosmetic reasons. The collaboration of the pharmaceutical industry is crucial in these cases in order to establish a definitive etiological diagnosis.

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P. Hernández-Bel,* J. de la Cuadra-Oyanguren, L. Martínez, J. López, A. Agustí, and V. Alegre

Servicio de Dermatología, Consorcio Hospital General Universitario de Valencia, Valencia, Spain

*Corresponding author.
E-mail address: pablohernandezbel@hotmail.com (P. Hernández-Bel).

Cutaneous Lupus Erythematosus and Vitiligo

Lupus eritematoso cutáneo y vitíligo

to the Editor:

Lupus erythematosus and vitiligo are diseases of autoimmune origin. Reports in the literature suggest patients are more likely to suffer more than 1 autoimmune disease (30% of patients with generalized vitiligo have another autoimmune disease).[1] However, there are few reports of cutaneous lupus erythematosus in association with vitiligo.

We describe a 42-year-old woman with no family history of autoimmune disease. In August 2008 she consulted for the appearance of erythematous papules on the arms, face, and upper trunk. The papules were slightly infiltrated, of variable size, and coalesced into larger plaques on hypopigmented skin; there was no desquamation (Figure 1). On suspicion of cutaneous lupus, a biopsy was performed, which showed vacuolar degeneration of the basal layer with a lymphocytic infiltrate in the dermis, dense perivascular lymphocytic cuffing, and focal deposits of mucin (Figure 2). Autoimmunity tests for antinuclear and anti-DNA antibodies were negative. Topical corticotherapy and sun protection measures were prescribed, and the patient improved.

The lupus lesions improved considerably within 3 months. However, the patient continued to present large hypopigmented plaques on the forearms, face, and upper trunk where the lupus lesions had been. Achromotrichia, not present at the first visit, was also observed in the eyebrows, scalp hair, and eyelashes (Figure 3). The lesions were suggestive of vitiligo, even though most were in areas that had previously presented lupus lesions. The differential diagnosis was with postinflammatory lesions secondary to lupus. The patient came to follow-up visits every 6 months; no new lupus lesions were observed, but the achromotrichia continued to spread. Our patient was therefore diagnosed with subacute cutaneous lupus with no systemic involvement (in remission at the time of writing) and vitiligo.

Reports of the coexistence of 2 autoimmune pathologies such as lupus erythematosus and vitiligo are rare in the literature. The earliest articles usually mention the differential diagnosis between the residual hypopigmented lesions of cutaneous lupus and vitiligo.[2] In 1981, Forestier et al[3] described 2 patients, 1 with discoid lupus who developed vitiligo-like lesions and 1 with vitiligo whose clinical course was complicated by the appearance of lesions of discoid lupus. Both patients presented elevated antinuclear antibodies, while all other autoantibodies were negative. Postinflammatory lesions were also considered in our patient, but the course of the lesions and, in particular, the achromotrichia supported the diagnosis of vitiligo. Other authors have subsequently reported further, isolated cases of patients with lupus who developed vitiligo over the course of their disease.[2,4,5]

A genetic explanation for the association between lupus erythematosus and vitiligo has recently been attempted. In a study of 16 European families, Nath et al[6] found that the *SLVe1* gene on chromosome 17 may explain the relationship between systemic lupus erythematosus and vitiligo. Rahner et al[7] related various mutS homolog 6 gene mutations (present in hereditary nonpolyposis colorectal cancer) with the presence of both autoimmune processes. However, both authors base their findings on a small number of patients. More studies are needed in patients with systemic lupus erythematosus and in patients with cutaneous lupus with no systemic involvement.

Based on the greater predisposition to the association of autoimmune conditions, isolated cases of patients with more than 1 autoimmune disease have been reported by several authors, such as Johnson et al,[1] who reported a case of vitiligo associated with type 1 diabetes mellitus and Callen,[8] who described a patient with discoid lupus erythematosus and autoimmune thyroiditis with high