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Quinidine-induced psoriasis

To the Editor:

Quinidine is associated with many skin reactions, but an association with psoriasis has been reported only rarely. Recently a patient with an apparent exacerbation of psoriasis while on quinidine was seen and is now reported.

On May 3, 1982, a 64-year-old white male retired railroad conductor experienced acute anterior chest pain due to an acute myocardial infarction and was admitted to the hospital. On May 11 he underwent coronary artery bypass surgery. Prior to admission, during his recovery period, and throughout his posthospital convalescent period, his chronic psoriasis vulgaris of 25 years remained in remission. He had received once-weekly ultraviolet light therapy (PUVA, 8 joules) prior to his hospitalization but received no treatments after May 3. On June 29, he was begun on quinidine for an arrhythmia that had developed. Within 72 hours he noted slight scaling of his scalp, which progressed to include psoriatic plaques on the scalp, trunk, hands, elbows, and knees. He was restarted on twice-weekly PUVA therapy on August 6. He was begun at 1.5 joules and increased 0.5 joule per treatment. Despite PUVA therapy and topical steroid creams, the psoriasis continued to worsen. In October, 1982, the patient asked that the quinidine be stopped because he was convinced that it made his psoriasis worse. His physician reviewed the available literature, did not find an association between quinidine and psoriasis, and advised him not to stop the quinidine. However, the patient decided to stop the quinidine despite this advice without telling the physician. The psoriasis began to clear 1 week after stopping the quinidine, and all psoriatic lesions were gone within 2 weeks. He has remained free of psoriatic lesions since that time but has continued once-weekly PUVA therapy.

A more detailed search of the literature revealed two previously reported cases of quinidine[1,2] exacerbating psoriasis. In addition to quinidine, other drugs reported as exacerbating psoriasis include the structurally related antimalarials, such as chloroquine,[3-5] adrenergic agents such as propranolol,[6,7] and clonidine[7]; agents which stimulate leukocytes, such as iodine,[8] lithium,[9-11] and the prostaglandin inhibitor, indomethacin.[12]

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Depression—a side effect of 13-*cis*-retinoic acid therapy

To the Editor:

Reported side effects of 13-*cis*-retinoic acid (isotretinoin) have included cheilitis, xerosis, skin fragility, bone aching, headache, stomach upset, alopecia, and corneal abnormalities.[1] We now wish to report another side effect recently observed by us—depression.

Six of 110 patients (5.5%) with acne or keratinizing disorders treated with 1.0 to 2.0 mg/kg body weight/day experienced depressive symptoms while on the drug. The depression was present in five patients with acne and in a single patient with palmar-plantar keratoderma associated with hypohidrotic ectodermal dysplasia. Four patients were women and two were

...he mean age of affected patients was 28.5, with ...ge from 20 to 42 years. One patient had a previous ...ry of depression. In a 21-year-old man, symptoms ...depression and forgetfulness were severe enough to ...use withdrawal of the drug. The other five patients ...continued with the drug despite feelings of depression.

All patients experienced depressive symptoms, manifested by crying spells (3/6), malaise (3/6), or forgetfulness (1/6), within 2 weeks of starting the drug. Symptoms rapidly resolved on discontinuing the drug.

Meyskens[2] noted similar psychologic changes in patients with cancer treated with 3 mg/kg/day of 13-*cis*-retinoic acid. Peck,[3] however, saw psychologic improvement in his patients with acne treated with isotretinoin. It was not reported whether any of their patients had a previous history of psychiatric disorder.

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Stanford study of Sézary syndrome

To the Editor:

Stanford University Medical Center is opening a study for the treatment of mycosis fungoides (especially with Sézary syndrome). This is an experimental protocol using monoclonal antibodies which may be combined with interferon in some patients. Requirements for this study include: Karnofsky status greater than 70% (cares for self but unable to carry on normal activity or do active work), white blood cell count greater than 3,500, platelet count greater than 100,000, and off all treatment for 1 month prior to treatment. The patient should not have renal, hepatic, or cardiac dysfunction that would intefere with treatment. Additionally, the patient must be willing to commute to Stanford for a 4- to 8-week treatment program which requires two to three visits per week.

If you have a patient who meets these general eligibility requirements, please contact Linda Rich, R.N. (address and phone number below). Thank you for your interest.

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Reproduction of hydroa vacciniforme with UVA

To the Editor:

We are pleased that Halasz et al (J Am Acad Dermatol 8:171-176, 1983) confirmed our observation that repetitive exposures to ultraviolet A (UVA) light reproduce the lesions of hydroa vacciniforme to clinical and histologic examination.[1] We constructed a dose-response relationship, showing that even suberythemogenic doses of UVA, when given repetitively at 48-hour intervals, could reproduce the papulovesicles of hydroa vacciniforme, whereas a single dose of up to 160 joule./cm^2 was ineffective in producing lesions other than erythema. Pathologic responses were neither produced nor enhanced by exposures to UVB light, either alone or in combination with UVA light. We observed that the antimalarial hydroxychloroquine sulfate, when given in doses of 200 mg by mouth daily, was dramatically effective in diminishing our patient's photosensitivity both subjectively and objectively. The number of repetitive applications of UVA light, the dose of UVA light per application, and the total dose of UVA light applied which were necessary to reproduce lesions of hydroa vacciniforme were all elevated after hydroxychloroquine sulfate therapy. In addition, the UVA minimal erythema dose rose from 12 joules/cm^2 to 20 joules/ cm^2, both within the normal range. Indomethacin in doses of 200 mg by mouth daily did not affect the patient's photosensitivity.

We have further observed a low level of the third component of complement before therapy with hydroxychloroquine sulfate, with a return to the normal range after treatment and after cessation of disease activity.

It would be interesting to assess Halasz et al's patient in a similar manner with respect to therapy with antimalarials to confirm the usefulness of hydroxychloroquine sulfate in the treatment of hydroa vacciniforme. Photo-