COMMENTS AND OPINIONS

Terminology in Dermatology: Logical or Arbitrary?

In a recent article in the ARCHIVES about language and logic, Melski[1] cites the following lines by Lewis Carroll as an introduction: "When I use a word . . . it means just what I choose it to mean—neither more nor less." Though this should not be the case in scientific language, it seems to happen even to Melski, who certainly means "tick sting" when he writes "tick bite." Ticks have no jaws or pincers with which to bite; rather, they have highly specialized tools to sting and suck blood. Medicine (dermatology in particular) abounds with evidently wrong or even nonsense terms that have evolved throughout the history of science. However, when a new method is developed in our century of science, it should be termed correctly.

Luminescence is a physical phenomenon defined by emission of light when a substance or compound takes up any kind of energy. When a chemical reaction leads to emission of light it is called chemiluminescence; this is responsible for the light emitted by glowworms. This type of reaction is also used for highly sensitive biochemical tests. When a substance takes up energy and emits light for a longer time than the time it took to absorb the light, it exhibits phosphorescence. When a substance emits light immediately upon uptake of energy (in most cases by irradiation with a shorter-wavelength light) it exhibits fluorescence. However, when incident light is used for visualization of a surface or of subsurface structures, this is neither luminescence nor epiluminescence[2] but epi-illumination. Incident UV light is used for high-quality fluorescence microscopy, but the type of visible light used for dermoscopy does not elicit any visible luminescence (a collective term for fluorescence and phosphorescence). Even though some dermatologists may think it makes no difference,[3] I strongly believe in the advantage of using a correct term if it is available. This dermatologic procedure should therefore be called what it is: surface microscopy,[4] incident light microscopy,[5] epi-illumination microscopy, or derm(at)oscopy.[6]

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Contact Allergy From Temporary Tattoos

We read with great interest the article by Lyon et al,[1] in the January issue of the ARCHIVES about a case of allergic contact dermatitis reaction due to a henna tattoo. Several cases of contact allergy from temporary tattoos have been reported in the literature and in most, the patch tests showed a positive reaction to paraphenylenediamine.[2-6] We have now observed 6 cases of allergic contact reaction to temporary tattoos (*mehndi*) and in all of our cases the responsible agent was paraphenylenediamine. This is due to the presence of paraphenylenediamine in temporary tattoos, which is used to strengthen the color. In 2 of our 6 cases, the onset of the dermatitis a few weeks after the *mehndi* tattoo was applied indicates that sensitization to paraphenylenediamine probably occurred as a consequence of the tattoo.

Paraphenylenediamine is a strong sensitizer and its use in temporary tattoos is associated with a high risk of active sensitization. It is important to emphasize that the growing popularity of this fashion among young people will possibly result in an increase of paraphenylenediamine sensitization in the general populations of Europe and North America. The number of shops and beauty salons that offer application of temporary tattoos to patrons is presently growing in Italy and in the European Union countries and the *mehndi* tattoo can no longer be considered a merely exotic art.

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VIGNETTES

Long-term Efficacy of Tacrolimus Ointment for Recalcitrant Facial Erythema Resistant to Topical Corticosteroids in Adult Patients With Atopic Dermatitis

Recalcitrant facial erythema often occurs in adults with atopic dermatitis and resists common therapies, including topical corticosteroids.[1] Recently, several authors have reported that short-term use of tacrolimus (FK506) ointment, a newly developed immunosuppressant drug, shows excellent results with recalcitrant facial erythema.[2-5] In the present study, we analyzed the effects of long-term applications of topical tacrolimus on red face conditions.

Patients and Methods. After obtaining informed consent, 51 adult patients (age range,16-44 years) with atopic dermatitis, who had steroid-resistant diffuse indurated erythema on the face for 1 to 23 years (average, 5 years), were included in the study. All fulfilled Hanifin and Rajka's diagnostic criteria[6] for atopic dermatitis. Active skin lesions on the trunk and limbs were present on less than 10% of the total body surface in 27 patients, 10% to 50% of the body surface in 10 patients, and more than 50% of the body surface in 14 patients.

We prepared a 0.03% tacrolimus ointment by mixing an injectable form of tacrolimus (Prograf; Fujisawa Healthcare Inc, Deerfield, Ill) with white petrolatum. All of the patients applied only the tacrolimus ointment to their face twice daily; however, they were allowed to use topical corticosteroids on other body areas. All were observed weekly for the first 2 months, and then monthly for the following 10 months.

Facial lesions were assessed before treatment, after 2 weeks, after 3 months, and after 1 year, and were graded as greatly improved, fairly improved, or not improved. Adverse events were recorded at all study intervals.

Results. The proportion of patients whose symptoms were greatly or fairly improved (**Figure 1**) by the tacrolimus ointment was 94% (48/51), 82% (42/51), and 62% (32/51) after 2 weeks, 3 months, and 1 year of treatment, respectively. The proportion of the excellent responders was 81% (22/27) for those patients whose skin lesions were restricted mainly to the face, whereas the propor-

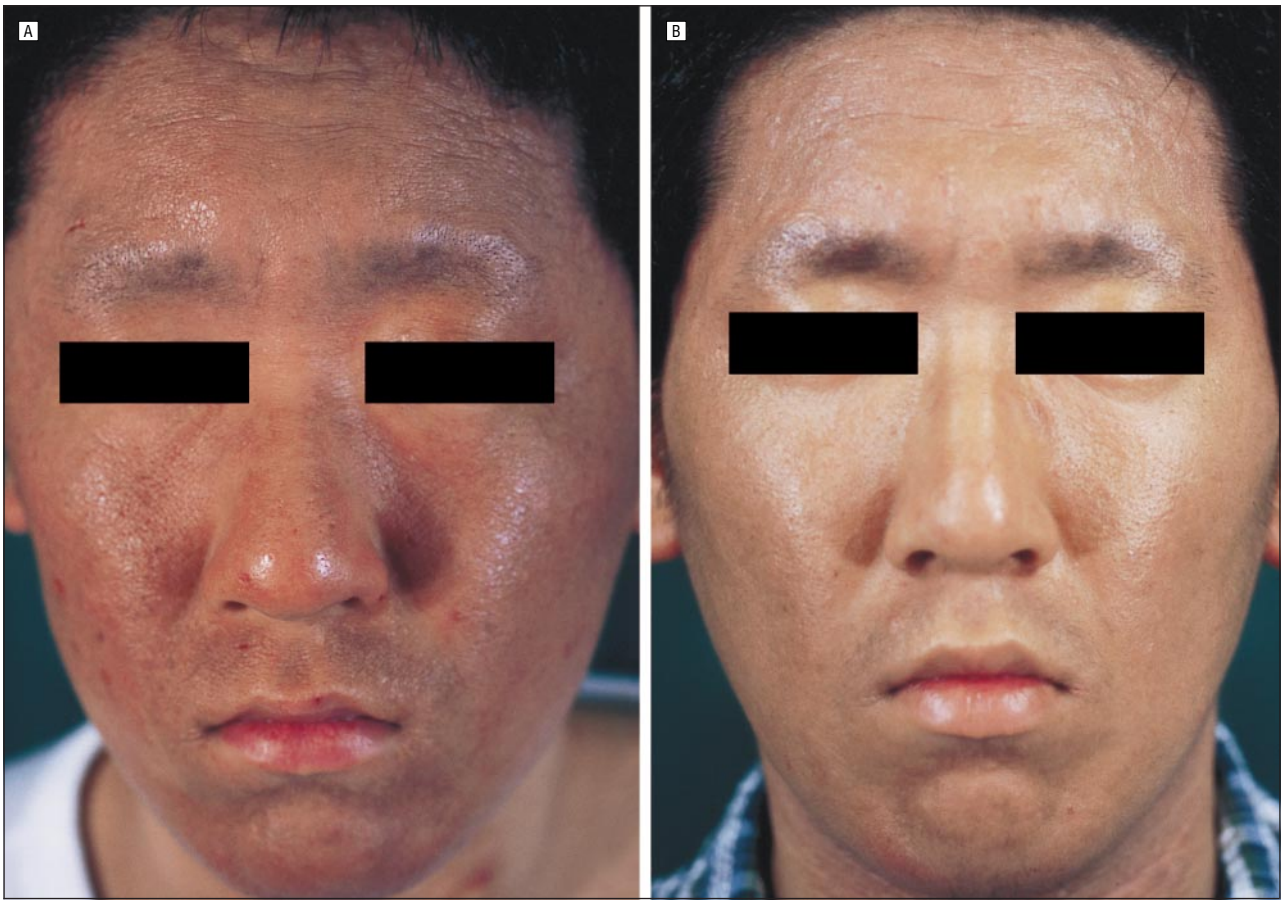

Figure 1. *Clinical features of recalcitrant facial erythema in adult patients with atopic dermatitis before (A) and 1 year after (B) treatment with 0.03% tacrolimus ointment. Diffuse indurated erythema resistant to 5 years of topical corticosteroid treatment was dramatically improved.*

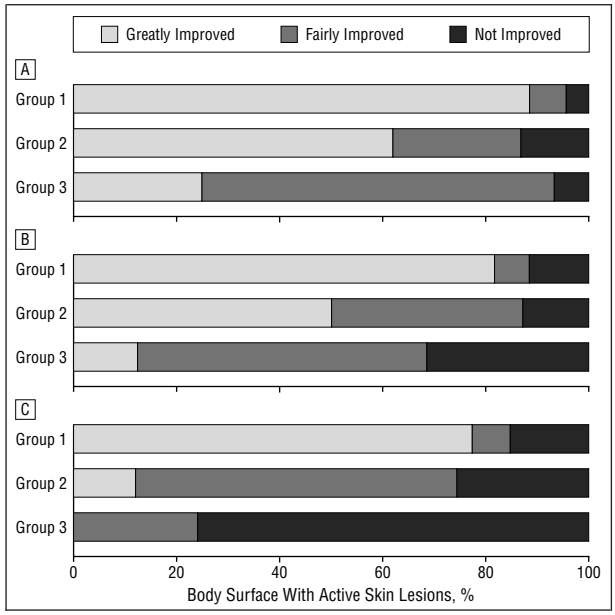

Figure 2. *Relationship between the distribution of active skin lesions and the efficacy of 0.03% tacrolimus ointment at 2 weeks (A), 3 months (B), and 1 year (C) of treatment. Group 1 consists of patients with active skin lesions on less than 10% of their total body surface; group 2, on 10% to 50%; and group 3, on more than 50%.*

tion was 21% (3/14) for those who had widespread skin lesions on the trunk and limbs (**Figure 2**). Mild episodes of herpes simplex (6 cases) and folliculitis (9 cases) occurred during the study, but no other severe adverse events were observed.

Comment. Our results confirmed previous reports[2-5] that short-term use of tacrolimus ointment shows excellent results with the recalcitrant facial erythema of atopic dermatitis. However, therapeutic efficacy was greatly reduced after continuous use of the ointment for 1 year. It is possible that prolonged application of tacrolimus ointment to the face often induces tachyphylaxis or contact hypersensitivity to the drug.

Long-term use of tacrolimus ointment for facial erythema was effective in patients who had sporadic skin lesions on the trunk, but ineffective in those with widespread lesions on the trunk. It is likely that in patients with extensive dermatitis, recurring episodes of the aggravating factors of the disease may affect both the trunk and face, resulting in a reduction in the therapeutic effect of tacrolimus ointment for facial dermatitis. Thus, in order to manage recalcitrant erythema on the face of patients with widespread atopic dermatitis, effective control of skin lesions on other body areas is mandatory.

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Pregnancy and Behçet Disease

Pregnancy is a physiological period during which various hormonal, metabolic, and immunologic changes occur. Behçet disease involves a course of systemic vasculitis and occurs in young adults.[1-5] For these reasons, this study was designed to observe 16 pregnant women with Behçet disease before, during, and after pregnancy. The health of the babies was also investigated.

Patients and Methods. The clinical features of the patients before pregnancy are summarized in the **Table**. It was the first pregnancy for 6 of the patients, the second for 3, the third for 3, and the fourth for 4. The patients were followed up with dermatologic, ophthalmic, gynecologic, and physical evaluations with routine laboratory examinations before, during, and after pregnancy. The newborns were evaluated with physical examinations.

Results. Activation of Behçet disease occurred in 4 cases before pregnancy, 9 cases during pregnancy, and 3 cases after pregnancy. Remission occurred in 12 cases before pregnancy, 7 cases during pregnancy, and 13 cases after pregnancy. The activation symptoms were as follows:

1. Before pregnancy there were oral ulcers alone (1 case), genital ulcers alone (2 cases), and oral ulcers with genital ulcers (1 case).
2. During pregnancy there were deep and frequent genital ulcers (7 cases), major oral ulcers (3 cases), papulopustular lesions (2 cases), and erythema nodosum (1 case). One case was diagnosed during pregnancy. No other organ attachments occurred, and 7 of 9 patients experienced remission before pregnancy.
3. After pregnancy there were frequent oral ulcers, genital ulcers, and erythema nodosum (1 case), frequent and deep genital ulcers (1 case), and uveitis (1 case). Two of these 3 cases were in remission before and during pregnancy. For all patients, pregnancy ended with normal delivery at term, and the babies were healthy.

Comment. The course of Behçet disease during pregnancy is variable. Remission,[1,2] activation,[4,5] or varying changes in each patient (and even in different pregnancies in the same patient)[3] have been reported. In our study, the number of patients in the activation period was greater than in the remission period. During pregnancy

Clinical Features of the Patients With Behçet Disease Before Pregnancy*

Patient No./ Age, y	Disease Duration, y	Clinical Features							
		OU	GU	EGU	EN	Tph	PpL	Pt	Other
1/19	5	+	+	–	–	–	+	+	–
2/20	3	+	–	–	–	–	+	–	Uveitis
3/24	1†	+	–	–	–	–	+	–	–
4/30	4	+	+	+	–	–	+	–	–
5/26	4	+	+	–	+	–	+	+	–
6/37	13	+	+	–	+	–	+	–	–
7/30	10	+	+	–	+	–	–	–	–
8/33	7	+	+	+	+	–	+	+	–
9/29	10	+	+	–	–	–	+	–	–
10/34	11	+	+	–	–	–	–	–	Uveitis
11/30	10	+	+	–	+	–	–	–	Uveitis
12/26	3	+	+	–	–	–	–	+	–
13/21	5	+	+	–	–	–	–	+	–
14/32	8	+	+	–	–	–	+	–	–
15/25	3	+	+	–	–	–	–	+	–
16/32	7	+	+	–	–	–	+	–	–

*OU indicates oral ulcer; GU, genital ulcer; EGU, extragential ulcer; EN, erythema-nodosum; Tph, thrombophlebitis; PpL, papulopustular lesions; Pt, pathergy; +, present; and –, absent.

†Patient 3 received her diagnosis during pregnancy when she had genital ulcers and a positive pathergy finding.

the characteristics of activation were mucocutaneous signs in most patients and arthritis, ocular findings, and thrombophlebitis in fewer patients.[3-5] Only mucocutaneous signs increased during pregnancy.

Medical abortion due to activation has only been reported in 1 study.[4] In our study, all pregnancies ended with normal delivery at term, as in other studies.[1-3,5] All of the reports, including ours, indicate that the newborn babies were healthy.[1-5]

In one study, it was reported that Behçet disease went into remission during pregnancy and reactivated after birth.[2] In another, it was found that the number of activations was the same during pregnancy as after, but the severity of the symptoms was worse during pregnancy (syndrome of vena cava inferior, Budd-Chiari syndrome, and ocular and articular symptoms).[5] In our study, fewer activations occurred after pregnancy than during pregnancy. In 1 case, ocular symptoms occurred after pregnancy, which never happened in any of the patients during pregnancy.

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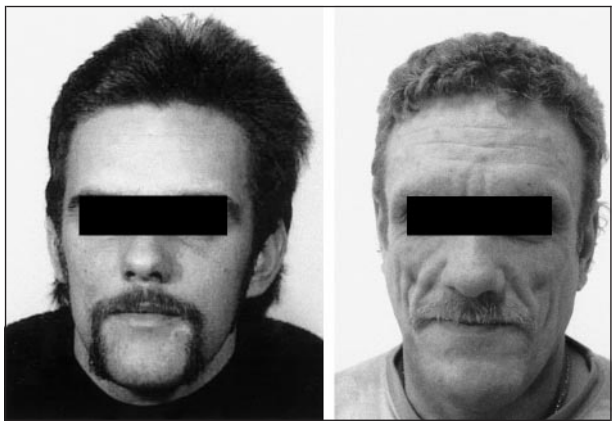
Curly Hair and Lipodystrophy as a Result of Highly Active Antiretroviral Treatment?

Different hair changes have been reported during human immunodeficiency virus infection. In patients with acquired immunodeficiency syndrome, hair may become softer, silky, discolored, and less curled.[1] We recently observed a patient with acquired immunodeficiency syndrome whose previously straight hair became curly during highly active antiretroviral treatment (HAART) about 4 months before developing a spinocellular epithelioma.

Case Report. A 48-year-old Belgian heterosexual truck driver was diagnosed with human immunodeficiency virus infection in 1991. His medical history revealed polyallergic asthma since childhood. In February 1993, his CD4 lymphocyte count was 130/µL and zidovudine monotherapy was started; in 1995, didanosine was added to his regimen. In October 1995, he developed severe pulmonary tuberculosis, complicated by adrenal insufficiency. He recovered after taking tuberculostatica with glucocorticoid and mineralocorticoid substitution.

In September 1996, HAART was initiated, including lamivudine, stavudine, and indinavir (a protease inhibitor). The glucocorticoid and mineralocorticoid substitution was continued. In February 1998, he noted that his previously straight hair had become curly (**Figure**). He also developed signs of peripheral lipoatrophy (Figure). His CD4 lymphocyte count was 294/µL, and he had an undetectable viral load. He took his antituberculous treatment until August 1998. In September 1998, findings from a biopsy specimen taken from an anal tumor revealed an invasive, moderately differentiated spinocellular epithelioma. After resection of the tumor, he was treated successfully with radiotherapy. In February 1999, he was free of complaints; his CD4 lymphocyte count was 181/µL; and his viral load remained undetectable. He still had curly hair.



Left, Straight hair, a few years before the diagnosis of human immunodeficiency virus infection was made. Right, Curled hair and signs of lipodystrophy during highly active antiretroviral treatment including a protease inhibitor.

Comment. A change from straight to curly hair has been observed during puberty, as a paraneoplastic symptom,[2] or in treatment with etretinate and isoretinoid.[3] So far, this phenomenon has not been reported in patients with acquired immunodeficiency syndrome. It is possible that indinavir may interfere with hair growth; indeed, indinavir penetrates into hair.[4] The change in hair pattern may also be associated with the HAART-associated lipodystrophy. Human immunodeficiency virus 1 protease, the target for protease inhibitors, shares sequence homology with cytoplasmic retinoic–acid binding protein type I (CRABP-1). Binding of protease inhibitors to CRABP-1 has been postulated as a mechanism causing HAART-associated lipodystrophy.[5] Protease inhibitors have been associated with retinoidlike adverse effects including hair loss, cheilitis, paronychia, and dry skin. Since isotretinoin has been associated with curly hair, the development of curly hair in our patient with HAART-associated lipodystrophy may be a manifestation of protease inhibition of CRABP-1.

The hair changes in our patient occurred during HAART, before his anal tumor became clinically apparent. However, it is also possible that these changes were an early paraneoplastic symptom. All patients developing curly hair should be examined for a possible early neoplasm before asserting that they present with a new adverse effect of protease inhibitors.

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