COMMENTS AND OPINIONS

Dermatologic Surgeons Will Determine What's in Their Name: Both High Quality and High Standards

Drs Harold J. Brody and William P. Coleman[1] conclude their editorial "What's in a Name? Are We Dermatologic Surgeons or Surgical Dermatologists?" with these words: "If dermatopathologists would be content to be called pathologic dermatologists, then we would consent to being called surgical dermatologists. Otherwise, dermatologic surgeon will do quite nicely, thank you." Why the pique that peeks through and between the lines written by Drs Brody and Coleman, who are identified at the end of their piece as "the president-elect and president, respectively, of the American Society for Dermatologic Surgery"? Such defensiveness does not comport with the assertion by them in the same last paragraph that "Within our specialty we know who we are."

Perhaps brief exegesis of 3 quotations from the essay can illuminate what seems to be nettling them and whether their vexation is justified. First,

> [W]hat we [dermatologic surgeons] call ourselves is of paramount importance.[1(p1406)]

A name is important, but even more important is the manner in which one does one's work and the standard to which one aspires. Drs Brody and Coleman want to be known as *dermatologic surgeons*, and that is fine (although the analogue of dermatopathologist is dermatosurgeon). But some colleagues disposed favorably to *dermatologic surgery*, I among them, are concerned about a distressing trend in that field toward the name *cosmetology* and to the failure of leaders like Drs Brody and Coleman to decry it. Dermatologic surgery performed well by a skilled and caring physician is an adornment to dermatology and to medicine. Support and succor for it is a responsibility of all of us who are proud to call ourselves dermatologists and physicians. We are not, however, obliged to condone or applaud behavior that sometimes seems shamelessly self-serving and injurious to the good name of our profession.

Second,

> Some dermatologists confuse the terms *cosmetologist* and *cosmetic dermatologic surgeon*: one is a hairstylist, the other a physician.[1(p1407)]

That the boundary between the terms *cosmetology* and *cosmetic dermatologic surgery*, as Drs Brody and Coleman choose to call it, has become blurred is everywhere evident, so much so that a plenary lecture at the World Congress of Dermatology in Sydney, Australia, in June 1997 by Dr Alistair Caruthers, a Canadian dermatologist and advocate for cosmetic dermatology, was titled "Dermatocosmetology: Where Are We at the End of the 20th Century?" If, for example, cosmetic dermatologic surgeons were to cut and style hair and perform manicures and pedicures, all the while proclaiming as they compete for "customers" with hairdressers and barbers, "We do it better than they because we know more about keratin," would Drs Brody and Coleman speak out forcefully against such practices? Currently, there are practices by some dermatologic surgeons that violate the Hippocratic oath, and yet those deviations are not acknowledged, let alone condemned, by the leaders of societies of dermatologic surgery. Such condemnation would be more constructive than silly nonarguments about what's in a name.

Last,

> We all teach primary care physicians how to do biopsies.[1(p1407)]

All too many dermatologic surgeons perform biopsies that produce woefully inadequate specimens, thereby making it impossible for dermatopathologists to serve either the surgeons or their patients satisfactorily. Anyone who has been engaged in the active practice of dermatopathology for the past 30 years will attest to the curious paradox of a plummet in the quality of performance of skin biopsies by dermatologists concurrent with the surge to dermatologic surgery for treatment. If the current practice of progressively more superficial shave biopsy procedures continues, including all too many of them for lesions suspected clinically of being melanoma, pathologists can expect to receive specimens that consist only of stratum disjunctum.

Dermatologic surgeons should be in the forefront of the advance of dermatology as it pertains to all surgical aspects, and that includes, among many other matters, (1) performing biopsies well and teaching residents and others to do the same; (2) ensuring that trainees in dermatologic surgery learn the rudiments of cutaneous embryology, histology, anatomy, and pathology, without which comprehension of cutaneous neoplasia is impossible, and learn as well how to differentiate by conventional microscopy between conditions that they deal with surgically (eg, between dermatofibroma with "monster cells" and malignant fibrous histiocytoma, desmoplastic trichoepithelioma and morpheiform basal-cell carcinoma, and desmoplastic Spitz nevus and desmoplastic melanoma); and (3) calling to task publicly those chairmen of departments of dermatology in university medical centers who employ dermatologic surgeons merely as "cash cows," without demanding that they do what everyone else in academic life is obligated to do, namely, advance the perimeter of the discipline by either clinical or basic science research.

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I have addressed these and other subjects that I deem important to the future of dermatology in various forums, including many lectures to major societies of dermatologic surgery in the United States, including the American Society for Dermatologic Surgery, and abroad. Parenthetically, I have been an even more rigorous critic of some developments in dermatopathology and in academe than I have been of some in dermatologic surgery. In my view, a strong opponent of all that weakens a field of human endeavor—and as strong a proponent of all that strengthens it—is precisely what a concerned citizen should be.

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In reply

Dr Ackerman ponders "Why the pique that peeks through and between the lines?" This is quite disingenuous because Dr Ackerman knows all too well that dermatologic surgeons are very concerned over his continued use of the word cosmetologist *in reference to cosmetic dermatologic surgery. This has occurred in private, in public, and in print. At the International Dermatology Retreat in Atlanta, Ga, in 1998, he used this disparaging term in front of a large audience of international dermatologists. He closed by stating "I don't care whose feelings I hurt." We immediately pointed out to him that the word* cosmetologist *refers to minimally trained individuals, usually with only a high school education. We also told him that we found the term highly insulting. This has been repeated to him again in print and through mutual friends. Using the word* cosmetologist *to refer to fellow dermatologists is simply a slur and will not be tolerated.*

One of the most wonderful things about being a dermatologist is the ability to choose subspecialty areas within this diverse field. Dr Ackerman refers to the oath of Hippocrates, which states "I will follow that system of regimen which, according to my ability and judgment, I consider for the benefit of my patients. . . . " Dermatologic surgeons have pioneered many of the cosmetic surgical procedures in practice today. On a daily basis we bring great joy and pleasure to our patients who elect to undergo these procedures. Dermatology is a small field, and we think Dr Ackerman will agree with us that our specialty will prosper best if we cooperate with each other in a collegial atmosphere of mutual respect and high ethical standards.

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Green Tea Polyphenols May Be Useful in the Treatment of Androgen-Mediated Skin Disorders

The timely review by Katiyar et al[1] on green tea and skin discusses the anti-inflammatory, anticarcinogenic, and antioxidant properties of green tea polyphenols. Another potential beneficial effect of green tea is worth noting. Liao and Hiipakka[2] have shown that the green tea catechins (-)epigallocatechin-3-gallate and (-)epicatechin-3-gallate are potent inhibitors of type 1,5α-reductase.[2] While controlled human studies have yet to be conducted to prove clinical efficacy, these findings suggest the potential for the use of green tea polyphenols in the treatment of androgen-mediated skin disorders such as androgenetic alopecia, hirsuitism, and possibly acne.

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On the Importance of Definition in Dermatology and All Fields of Human Endeavor

Dr Ackerman[1] did not go far enough in his reply to Dr Gniadecki.[2] He did not explain what definitions refer to and why they are so important to the human mind.

Dr Gniadecki's position on the status of definitions in dermatology is tantamount to an outright assault on knowledge itself.[2] When Dr Gniadecki likens definitions to descriptions, he undercuts the ability of a person to form a definition, because he makes no attempt to address the entities in nature that give rise to a concept, and in turn to the definition, which is "a statement that identifies the nature of units subsumed under a concept."[3(p.40)]

When Dr Gniadecki[2] avers (through a reference to the philosopher Wittgenstein) that "It is incorrect (but tempting) to think that there must be something in common to all individual forms and varieties of a particular disease, something that defines the disease," he implies that one can know what a disease is only by not knowing what a disease is—ie, by not knowing what the concept of a specific disease refers to in reality.

Valid definitions can be created only by reference to concepts, a concept being defined as a "mental integration of two or more units possessing the same distinguishing characeristic(s), with their measurements omitted."[4(p13)] In fact, definitions serve no other function than

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an epistemological one, and they cannot be used as isolated statements detached from the concepts they serve to define. If, however, a definition of psoriasis refers only to "a flexible family of resemblances," as stated by Dr Gniadecki,[2(p1271)] it is no wonder that a definition cannot be made using that approach.

Definitions do not refer to "family resemblances"; rather, they refer to a spectrum of actually existing entities (or entities that existed in the past or that will exist in the future) that contain similar fundamental units, identified as essential by morphologic observation, physiology, response to therapy, and so on, and that are united by a mental integration, with specific measurements of those observations omitted. The definition is merely the briefest statement of reference to the fundamental units involved to "distinguish a concept from all other concepts and thus to keep its units differentiated from all other existents."[3(p40)]

There is more: additional observations of the entities under a concept may add to the extended knowledge that may be discovered to be even more essential, requiring a change in or amendment to the definition, such as genetic information in some cases, just to name one example. However, in the context of the entities within the conceptual spectrum, all other observations are still true; they are just no longer essential and no longer definitional.

Thus, in contrast with Dr Gniadecki's statement that "Ackerman's 'Dictionary of Dermatology' is likely to halt progress in dermatology rather than to catalyze its development," I say just the opposite. Definitions are the second most fundamental epistemological exercise that an individual can undertake (second only to the inductive process of observation of entities that give rise to the concept), serving to focus the human mind to understand the spectrum of the concepts to which all those definitions refer.

Such a dictionary, if designed properly, should serve to help focus the minds of dermatologists and dermatopathologists, because a definition, to be established at all, must evolve inductively through observation of the entities within a conceptual spectrum. For a definition to be proven wrong, it must be only by reasoned argument based on evidence of the error. If the definition is correct, within a context of knowledge (nothing can be defined outside of a context), the definition will stand through vigorous observation and research; if not, it should and must be rejected and replaced by a valid one. Either way, human knowledge is advanced.

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Are Porokeratoses an Infection?

I read the communication from Hernandez and colleagues[1] in the December 2000 issue of the ARCHIVES in which they report a case of disseminated porokeratosis and add to the list of diseases associated with this unusual keratosis. Although textbooks have considered the disease to be a disorder of keratinization or a chromosomal defect,[2] I believe that it is clear at this point that porokeratoses are actually manifestations of viral infection.

What is the evidence? First, the associated diseases share the common feature of immunosuppression. Sunlight, renal disease, corticosteroid use, human immunodeficiency virus infection, organ transplantation, psoralen plus UV-A therapy, and cancer chemotherapy are all recognized to be immunosuppressive.[1,2] Second, autotransplantation experiments demonstrate that a fragment of porokeratosis will regenerate an entire round lesion, which strikes me as the sort of thing a wart might do.[3] Third, a lesion partially removed as a biopsy specimen will regrow its old border in a matter of weeks (personal observation) in the fashion of a partially excised wart.

What virus might be the etiologic agent? On a couple of occasions, I have persuaded a pathologist to look for papillomavirus in specimens of porokeratoses, but none was found using a panpapilloma probe (or more accurately, a pan–*known* papilloma probe). Perhaps Epstein-Barr virus might be involved; consider its effects on oral epithelium in hairy leukoplakia. Or perhaps human herpesvirus 8 is involved in this disease too.

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VIGNETTES

Dominant Dystrophic Epidermolysis Bullosa Associated With Pyloric Stenosis and Congenital Absence of Skin

The occurrence of pyloric atresia in association with congenital epidermolysis bullosa (EB) has been reported in more than 80 patients. Almost all of these cases are in patients with junctional EB of a special subset characterized by abnormalities of the α6β4-

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integrin by mutation of either the α6 or the β4 chain, resulting in both cases in a defective link between the basal keratinocytes and the anchoring filaments of the lamina lucida.[1] We present an uncommon case of congenital pyloric stenosis in a young patient associated with a dominant dystrophic form of EB and a congenital localized absence of skin (Bart syndrome).

Report of a Case. An 8-month-old boy was referred for mucous and acral blistering since birth. His familial background was remarkable for early and regressive acral trauma–induced blisters, atrophic scars on the dorsal aspects of feet and hands, cutaneous brittleness, and residual, long-lasting onychodystrophy of most fingers in his father, his paternal grandmother, and his great aunt. No significant morbidity occurred during pregnancy and delivery, but findings of clinical examination at birth revealed absence of skin on the anterior aspect of the right leg. Blisters with clear content soon developed on acral areas with minimal trauma and on the oral mucosa as well. Investigations into retardation of growth and frequent vomiting led to the discovery of a pyloric stenosis, which was surgically cured at age 3 months allowing growth to resume at a normal pace. Results of histologic and immunohistologic studies performed on a recent blister revealed a noninflammatory, subepidermal splitting under the lamina densa, with a sharp decrease of collagen VII. All the other investigated antigens of the basal membrane zone were normally present, including the α6β4-integrin. Together with the familial history, these data allowed the diagnosis of dominant dystrophic EB associated with pyloric stenosis and congenital localized absence of skin.

Comment. The association between pyloric atresia or stenosis (a minor form of atresia) and EB that differs from the junctional EB subset linked to the α6β4-integrin has been reported in only 4 cases, all of the dystrophic type (excluding cases also displaying a congenital absence of skin).[2-4] It must be pointed out that the level of split was measured only by electron microscopy (in 2 of 5 cases) and that none of these cases was investigated by the more reliable immunological or molecular methods. In our case, findings from immunological and molecular studies clearly demonstrated that it was a dystrophic form.

The triple association of EB, pyloric atresia or stenosis, and congenital absence of skin has been described in 33 patients, including an extended Bedouin kindred.[5,6] In all cases but 3, the bullous disease was clearly of the junctional type; 2 cases were suggestive of both junctional and dystrophic types, whereas a third case, reported as a simplex type, remained quite questionable because electron microscopy was performed after the death of the patient, which raises the possibility of a postmortem artifact affecting the true level of the split. The mode of inheritance and the nonrandom association between these 3 rare conditions suggest that this disorder is perhaps a distinct type of EB, somehow different from the EB–pyloric atresia subset without congenital absence of skin (linked, for instance, to different mutational patterns of the α6 or β4 chain genes). However, our observations in this case together with the findings from the 3 nonjunctional EB previous reports show that genetic heterogeneity exists among these patients as well.

This case (together with the earlier and perhaps more questionable ones) emphasizes the absence of strict correlation between pyloric abnormality and a specific subset of EB. Accordingly, immunological and in some cases molecular investigations remain mandatory in cases of EB, whatever the pyloric status.

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Imiquimod: A New Possibility for Treatment-Resistant Verrucae Planae

We report the successful treatment of multiple verrucae planae in a 42-year-old man with topical application of 5% imiquimod cream. Imiquimod is a new immune response modifier for local application that induces the release of cytokines including interferon alfa, tumor necrosis factor α, and certain interleukins by peripheral mononuclear blood cells and lymphocytes.[1]

Clinical trials have shown a greater efficacy and safety of imiquimod for the treatment of external genital warts induced by human papilloma virus and a lower recurrence rate compared with other treatment options.[2] Based on these findings and the similarity of the etiologic origin to verrucae planae, which show a close relationship

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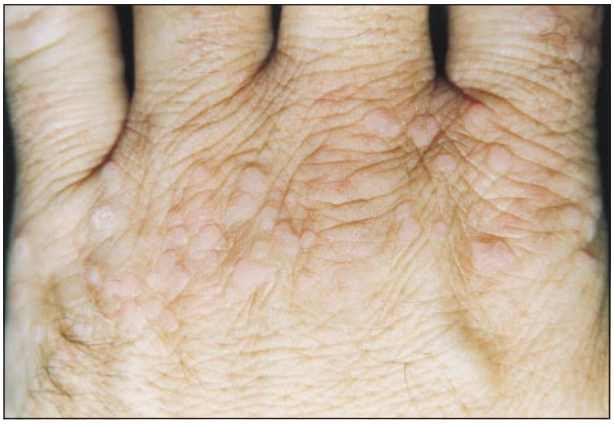

Figure 1. *Verrucae planae on the back of the left hand before treatment with imiquimod.*

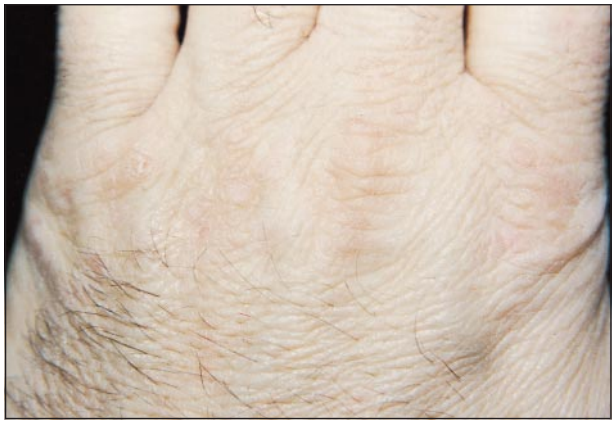

Figure 2. *Findings 6 weeks after treatment with imiquimod.*

to human papilloma virus types 3 and 10, we decided to treat this patient with imiquimod.

He had multiple verrucae planae on his fingers, the backs of his hands (**Figure 1**), and his lower arms for more than 2 years. His general condition was strongly affected so that various local (keratolytic agents, cryotherapy, and excochleation) and systemic (cimetidine and dimepranolinosin) treatment options had been tried. The number of warts was reduced for a short time, but they recurred within weeks. At the time the diagnosis was established, a consumptive disease and immunodeficiency had already been ruled out. The patient was advised about hand towels as a source of infection from the beginning, and he was exceptionally compliant.

Topical treatment with imiquimod was performed 3 times weekly and continued for 6 weeks. The cream was applied before bedtime without occlusion. After this period, the warts were no longer visible (**Figure 2**), and no adverse effects occurred.

Plane warts are benign lesions that occur predominantly in younger patients. They appear on fingers and hands, lower arms and legs, and the face. Spontaneous involution without scarring is frequent, although the natural course is not predictable.[3] Some patients are very difficult to treat because they are susceptible to recurrent warts. In these cases, plane warts can develop into a serious problem for patient and physician. Treatment options have to fulfill the following criteria: no scarring, no pain, and low cost. External treatment options such as physical destruction (cryotherapy, electrocoagulation, excochleation, and laser therapy), keratolytic agents, virostatic agents, and systemic options such as the hydrogen-receptor-antagonist cimetidine or the immunostimulant dimepranolinosin are well known and more or less common. But imiquimod acts on a different level than these therapies. It enables the immune response of the skin to destroy the warts. This mode of action shows a causative principle and makes imiquimod a possible future treatment option for verrucae planae.

This positive experience with imiquimod in the treatment of our patient requires confirmation on a larger scale. Similar to other virus-induced skin diseases in which imiquimod has been successfully applied on a trial basis (verrucae vulgares and mollusca contagiosa[4]), clinical trials must be conducted.

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Cutaneous Leishmaniasis Due to *Leishmania infantum* in the Northeast of Spain: The Isoenzymatic Analysis of Parasites

Human cutaneous leishmaniasis (HCL) has been known in Spain since 1914, when it was described in the Alpujarras region (Andalusia). The causative agent was characterized in 1986 for the first time by isoenzyme electrophoresis and identified as a zymodeme of *Leishmania infantum*.[1] Further studies showed the enzymatic polymorphism of *L infantum* in HCL, which we now report in Catalonia.

Ten *Leishmania* isolates from 8 patients with cutaneous and mucocutaneous leishmaniasis acquired in Catalonia were collected between 1984 and 1996 and identified by isoenzyme electrophoretic analysis in starch gel using 15 loci.[2] Characteristics of the patients and isoenzyme identification are given in the **Table**.

First reports on infraspecific identification of *L infantum* in the Mediterranean region revealed the existence of various zymodemes more or less related to clinical patterns of the disease. Therefore, *L infantum* zymodemes in the Mediterranean region were classified as dermotropic or viscerotropic. With the spread of human immunodeficiency virus (HIV) and the high inci-

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List of Strains, Characteristics of Patients, and Zymodemes Identified*

	Patients					
WHO Code	Age, y	Sex	HIV	Clinical Pattern	Topography	Zymodeme
MHOM/ES/1984/BCN-1	75	M	–	CL	Face	MON-29
MHOM/ES/1984/BCN-11	71	F	–	CL	Face	MON-29
MHOM/ES/1986/BCN-16	10	F	–	CL	Face	MON-1
MHOM/ES/1986/BCN-18	8	M	–	CL	Face	MON-33
MHOM/ES/1988/BCN-24	26	M	–	MCL	Bucconasal	MON-1
MHOM/ES/1994/BCN-113[a]	29	M	+	MCL	Palate	MON-1
MHOM/ES/1995/BCN-137[a]	30	M	+	MCL	Sublingual	MON-34
MHOM/ES/1996/BCN-144	40	M	+	CL	Hand	MON-33
MHOM/ES/1996/BCN-148[b]	30	M	+	MCL	Perianal	MON-1
MHOM/ES/1996/BCN-168[b]	30	M	+	MCL	Perianal	MON-1

**WHO indicates World Health Organization; HIV, human innumodeficiency virus. The WHO codes are read as follows: M indicates mammalia and HOM, the genus* Homo *(the host from which the strain was isolated); ES, Spain (country where the strain was isolated); indicated year, the year the strain was isolated; BCN, Laboratori de Parasitologia, Universitat de Barcelona, Barcelona, Spain (the laboratory where the strain was isolated). CL indicates cutaneous leishmaniasis; MCL, mucocutaneous leishmaniasis; and MON, the zymodeme code for Montpellier, France. Stocks indicated with superscripts* a *and* b *belong to repeated lesions in the same patients.*

dence of *Leishmania* HIV coinfections,[3] the spectrum of symptoms and our understanding of the etiology of the disease have changed. The infraspecific identification of *Leishmania* strains from HIV-coinfected patients showed that the parasite tropism is related to both the strain genotype and the host immune status.[4]

In the last 16 years, 10 *Leishmania* strains from patients with cutaneous or mucosal infections have been identified in our laboratory. This number does not reflect the extent of the disease in Catalonia, where the number of cases of cutaneous leishmaniasis, mainly in non-HIV patients, is unknown. By Spanish law, beginning in 1982, each case of leishmaniasis must be reported to the proper health authorities; nonetheless, almost all cases remain undeclared and the responsible organism is rarely isolated and identified. All the strains from tegumentary leishmaniasis that have been received at our laboratory for identification since 1988 were isolated from patients coinfected with HIV. This may be a consequence of the bias introduced during diagnosis of cutaneous leishmaniasis in these patients. The classic Oriental sore in non-HIV patients is often clinically diagnosed by dermatologists, and in these cases culture and strain isolation are rarely performed. The atypical lesions in HIV-coinfected patients and their poor response to treatment are some of the reasons for involving a laboratory in the diagnosis of the disease, and in these cases the culture of samples plays a fundamental role.

The 4 zymodemes found in the present study have been reported from human visceral leishmaniasis in Spain and France. Although few cases have been reported here, most of them belong to zymodeme MON-1 (World Health Organization code number), the most frequent zymodeme in human visceral leishmaniasis and only occasionally found in HCL.

The dog is regarded as the main reservoir of *L infantum*. However, from the 4 zymodemes found in our study, only zymodeme MON-1 has been found in dogs in Spain and in France, among hundreds of strains identified and reported.[5] The epidemiological cycle of these zymodemes (non–MON-1) and their anthroponotic or zoonotic characteristics must be studied further.

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Cutaneous Intolerance to Tattoos in a Patient With Human Immunodeficiency Virus: A Manifestation of the Immune Restoration Syndrome

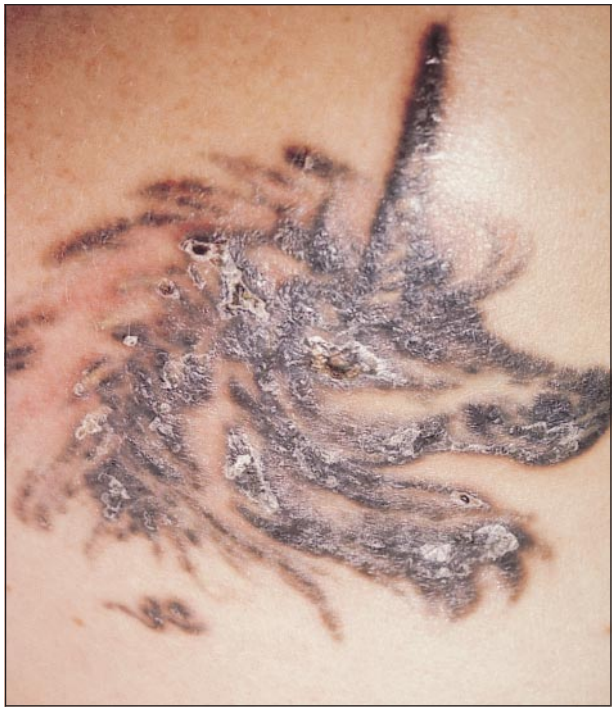
Cutaneous intolerance of the pigments used for tattooing has been known for a long time. This may present in different clinicohistologic forms, including eczema, photosensitivity, lichenoid reaction, granulomatous reaction, and pseudolymphoma.[1]

Cutaneous reactions to a tattoo tend to occur a few weeks or months after the tattoo has been done. Later appearances (after some years) have occasionally been described but are not common. This reaction may be due to retarded hypersensitivity to one of the pigments used and can be reproduced by epicutaneous tests with suitable allergens. On other occasions, the granulomatous reactions are of the foreign-body type or have sarcoid characteristics, and the findings of epicutaneous tests tend to be negative. However, since the delayed reaction to tattoos is also immunological, it is unlikely to be associated with delayed hypersensitivity type IV.

We had the chance to treat a patient with human immunodeficiency virus (HIV) whose cutaneous intolerance to tattoos was very delayed but coincided with an important improvement in his virologic and immunologic status after the introduction of highly active antirretroviral therapy (HAART). He was a 36-year-old man with advanced HIV who consulted us for itch and scabs that had appeared over a period of 1 month on 3 tattoos. The tattoos had been done 10 years previously and had caused no problems before. Two months before we saw him, he had begun HAART; by the time he came to us, his viral load was 1 800 000 copies/mL, and his CD4 count was 0.026×10^9 cells/L (26 cells/µL). On physical examination we found 3 tattoos, all done in several colors. There were scabs over the black color of all 3 (a sample is shown in the **Figure**). We performed a skin biopsy, the results of which showed a granulomatous and eczematous reaction. No microorganisms were detected with Giemsa and Ziehl-Neelsen stains. Radiographic energy–dispersive spectroscopy was performed on sections of the skin biopsy specimens, and no iron or titanium was detected. We asked for a blood test in which there was a rise in the CD4 T-cell count to 0.106×10^9 cells/L (106 cells/µL), and the viral load was undetectable. We carried out epicutaneous tests, using the standard European battery and several metals, with negative results.

Treatment was started with potent topical corticosteroids (clobetasol propionate), and the lesions all disappeared over a period of 2 weeks. Skin biopsy specimens of these areas showed a fibrous scar reaction. There were no signs of eczema or of sarcoid-type granulomas. The patient has remained symptom-free for the past 6 months.

The use of HAART has resulted in unprecedented reductions in viral load, increased blood CD4 T-cell count, and seems to have restored pathogen-specific immune responses. Since HIV-infected patients have been treated with HAART, a change in the natural history of some diseases has occurred. In this way, regression of Kaposi sarcoma[2] and resolution of recalcitrant warts[3] have been achieved. On the other hand, 1 case of sarcoidosis[4] and 3 cases of hepatitis C[5] have been reported after this treatment was begun. The appearance of these diseases after HAART has been started has been called *immune restoration syndrome*. We think that our patient's cutaneous reaction to the tattoos could be related to this immune restoration syndrome.



Scabs over the black color of one of the tattoos.

Intolerance to black coloring in tattoos is rare but has been described recently.[6-9] The black color in tattoos may be india ink (containing carbon particles), logwood wood (hematoxylin campechianum, which contains chromium), iron oxide, or titanium oxide. Carbon was the allergen involved in 2 cases,[7,8] and iron oxide was the allergen in the cases involving intolerance of cosmetic tattooing of the eyebrows.[9] We think that the organic carbon was the material used in our patient's tattoo because x-ray analysis did not detect specific elements.

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Efficacy and Safety of Topical Atropine in Treatment of Multiple Eccrine Hidrocystomas

The treatment of multiple eccrine hidrocystomas (MEHs) is complex and unsatisfactory. A few isolated cases have been reported in which treatment with oral and topical anticholinergic drugs has resulted in transitory resolution of the lesions, but sometimes with adverse effects to the vision.[1-5] We have performed the first prospective study to evaluate the efficacy and safety of topical atropine sulfate in the treatment of MEH.

Patients and Methods. Five women (aged 58-79 years) with symptomatic histologically confirmed MEH (40-100 lesions; mean number, 66) were enrolled in the study. Hidrocystomas were located on the forehead and cheeks (5 patients), lower eyelids (4 patients), nasal bridge (2 patients), and chin (1 patient). All the patients experienced either pruritus or burning pain (2-5 on an analog scale of 0-10; mean intensity, 3.4). They were treated with topical 1% atropine sulfate in aqueous solution, applied with a gauze or cotton ball, once a day, in the afternoon (0.4-0.6 mL/d, equivalent to 4-6 mg of atropine sulfate). The study was performed between July and September 1999 (the hot season) in Madrid, Spain. Follow-up included visits at 15 days, 1, 2, and 3 months after beginning the therapy. Before and during the follow-up, safety was measured by the appearance of anticholinergic adverse effects (blurred vision, inhibition of sweating, mouth dryness, thirst, palpitations, headache, difficulty in micturition, and reduced intestinal peristalsis); evaluation of heart rate; and a complete ophthalmologic examination that included best-corrected visual acuity (distance and near vision), anterior segment biomicroscopy, intraocular pressure readings, monocular and binocular amplitude of accommodation, and pupil evaluation (size and reaction to light). Informed consent was obtained from all the patients.

Results. The beginning of reduction or clearing of lesions after the treatment ranged from 4 to 7 days. The best results were obtained at 15 days, with a reduction of 50% to 100% (mean reduction, 77%) (**Figure 1** and **Figure 2**). Unfortunately, MEHs reappeared 1 to 3 days after the therapy was discontinued. The pruritus and burning pain disappeared from all patients within the first week.

Findings from ophthalmologic examination were within normal limits except for asymptomatic mydriasis (pupil size increased 0.75-2.75 mm; mean increase, 1.7 mm) in the 4 tested patients and a slight decrease of accommodation amplitude (both eyes) in 3 (asymptomatic in 2 of these). The fourth patient reported a slight blurred vision in 1 eye in the morning due to an abuse of monolateral application of the therapy; this blurred vision cleared once the atropine dose was reduced for that eye. All the patients had 20/20 near vision with appropriate correction. Intraocular pressure did not show significant changes (± 2 mm Hg). A local reduction of sweating occurred in all the patients. There was no significant difference in heart rate during the treatment.

Comment. Although MEHs are not a serious problem, they represent a troublesome and cosmetically unpleasant condition. In the present study we have shown the efficacy of 1% topical atropine in the treatment of MEH and a high degree of patient satisfaction. This therapy was safe, and the few adverse effects were either asymptomatic or well tolerated. Although more studies are neces-

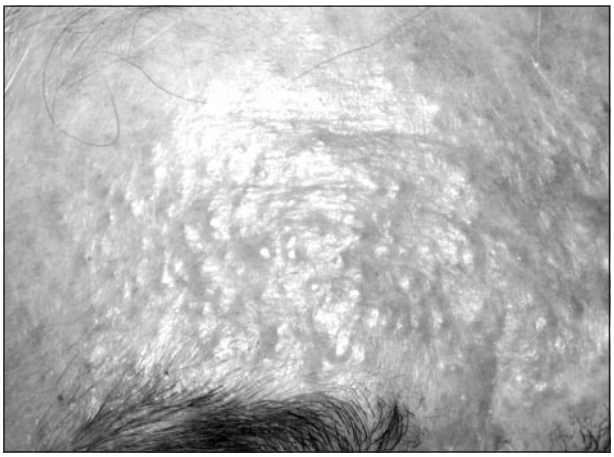

Figure 1. *Multiple eccrine hidrocystomas on the forehead before treatment with 1% topical atropine.*

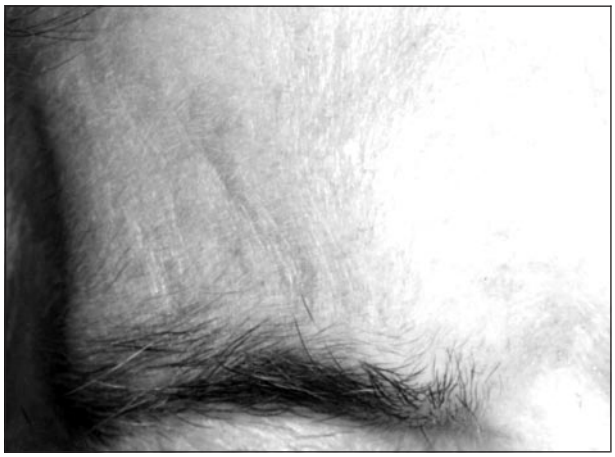

Figure 2. *Absence of lesions 2 months after the beginning of treatment to the same patient's forehead.*

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sary to confirm our results, we propose topical atropine as a first-line therapy for MEH during the hot season.

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Diabetic Foot Ulcers and *Chlamydia pneumoniae*: Innocent Bystander or Opportunistic Pathogen?

Chlamydia pneumoniae has not been searched for in cutaneous vessels and wounds although it induces vasculopathy and atherosclerosis in internal vessels. *Chlamydia pneumoniae* may play a role in the pathogenesis of chronic skin ulcers in diabetics,[1] atherosclerosis, and other vasculopathies,[2] autoimmune diseases such as multiple sclerosis[3] and Reiter disease.[4] We cultured and evaluated specimens of diabetic foot ulcers by immunohistochemistry to determine if *Chlamydia pneumoniae* was present and if specific antimicrobial therapy was helpful in patients with diabetic foot ulcers. *Chlamydia pneumoniae* was cultured from 4 of 9 patients, and immunostaining with anti–*C pneumoniae* antibodies detected intracellular inclusions in these samples from the 4 culture-positive diabetic foot ulcers. *Chlamydia pneumoniae* may be an opportunistic pathogen in chronic diabetic foot ulcers that leads to chronic inflammation, scarring, and poor wound healing.

Patient Reports. *Patient 1.* An 81-year-old white man with type 2 diabetes mellitus (DM) and severe peripheral vascular disease requiring an amputation above the right knee developed a decubitus ulcer on his left heel approximately 4 months prior to seeking treatment at the Diabetes Foot Center (Nashville, Tenn). The ankle-brachial index (ABI) was 0.54. The left heel ulcer was 4×4.5 cm and had peripheral maceration and a partially fibrotic ulcer bed with granulation tissue. At the time of the culture, the patient was receiving topical becaplermin and recombinant human platelet–derived growth factor, but no systemic or topical antibiotics. The ulcer debris was gently removed by curettage, placed in sterile saline for transport, and cultured for *C pneumoniae* on human lung carcinoma cells (HL) as previously described.[5]

Patient 2. A 61-year-old white man with type 2 DM developed a plantar ulceration of the first right metatarsal approximately 2½ months prior to presenting to the Diabetes Foot Center. He had had a coronary artery bypass graft in 1981 and had renal and venous insufficiency and coexisting stasis, and recurrent cellulitis in both lower extremities. Pedal pulses were palpable. Insensitivity to 5.07 monofilament up to the right ankle joint was detected. The 1.0×0.8-cm plantar ulcer was 0.4 cm deep with healthy-appearing granulation tissue. He received cephalosporin, 500 mg 4 times daily, and a reduced dosage of trimethoprim-sulfamethoxazole, 250 mg 4 times daily, to treat renal insufficiency. The ulcer debris was obtained and cultured as described above.

Patient 3. A 61-year-old white woman with type 1 DM, cardiomegaly, atrial fibrillation, and long-term tobacco addiction developed a plantar ulcer of the left hallux approximately 8 months prior to presenting to the Diabetes Foot Center. She had had a partial amputation of her left hallux because of osteomyelitis. She had a palpable posterior tibial pulse, a nonpalpable anterior tibial pulse, and insensitivity to 5.07 monofilament starting below the ankle. The left hallux ulcer was 0.6×1.5 cm and 3 to 4 mm deep and had not been treated with topical or systemic antibiotics prior to culture for *C pneumoniae*.

Patient 4. A 51-year-white woman with type 1 DM and hypertension developed incision dehiscence status after condylectomy for a nonhealing ulcer of the fifth metatarsal head. *Staphylococcus epidermidis* was cultured, and she was treated with intravenous vancomycin hydrochloride and rifampin. After 7 weeks of antibiotic therapy the ulcer was 1.2×0.8 cm with a partially granulated, fibrotic base and no bone exposure. A culture was taken and yielded *C pneumoniae*. The antibiotic therapy was continued and the ulcer healed with no signs of recurrence.

Comment. Cultures of curettage material from diabetic foot ulcers in 4 diabetic patients revealed viable *C pneumoniae* identifiable by immunoperoxidase and immunofluorescent microscopy of the cell cultures with anti–*C pneumoniae* antibodies. The **Figure** shows positive culture for *C pneumoniae* in patient 1. *Chlamydia pneumoniae* is difficult to detect unless special methods are used to isolate and propagate them in culture.[2,6] The *C pneumoniae* was detected in 4 of 9 cultures from patients with diabetic foot ulcers. In the Nashville Veterans Affairs Medical Center, serological and polymerase chain reaction evidence of *C pneumoniae* was detected in 74% (90/121) of randomly tested patients as compared with 21% (21/100) from the Nashville Red Cross in healthy blood donors (Mitchell et al, unpublished data). *Chlamydia pneumoniae* was identifiable by multiple methods serologically

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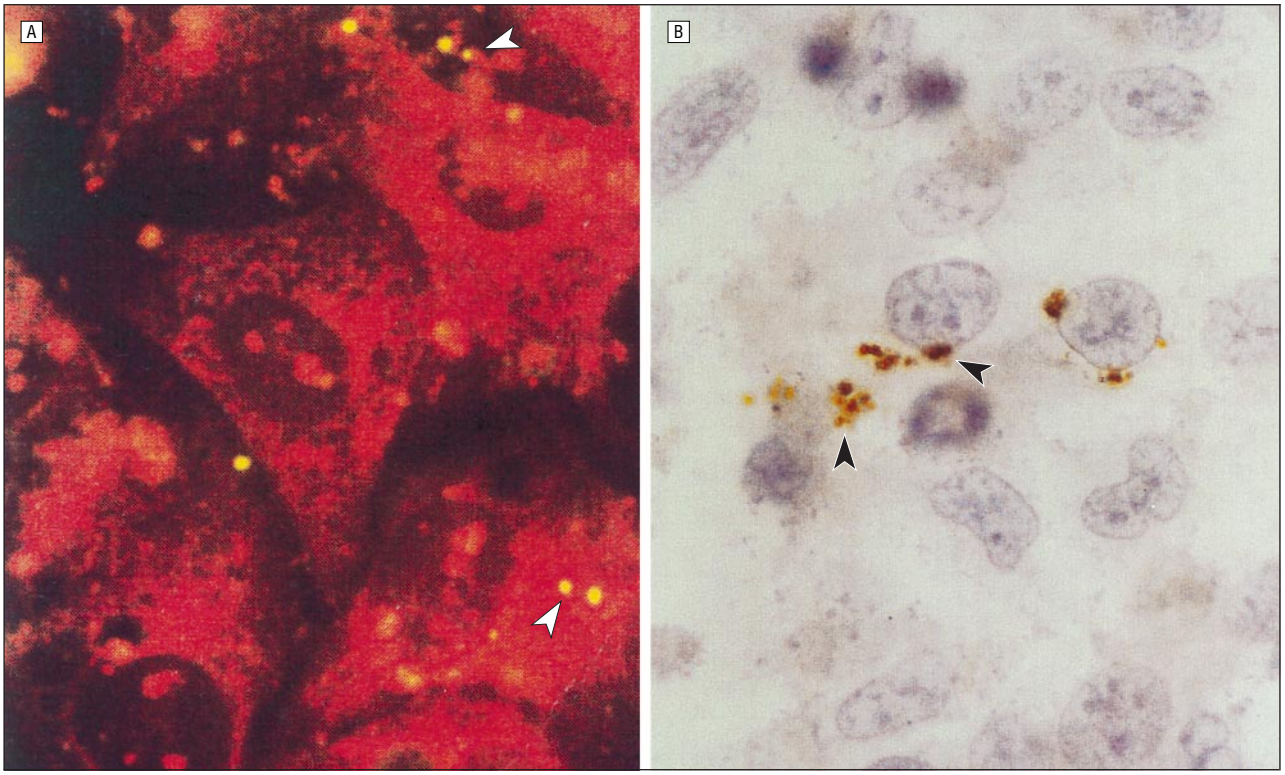

Representative culture evidence of Chlamydia pneumoniae *in curettage biopsy samples from diabetic foot ulcers from patient 1. A, Immunofluorescent detection of cytoplasmic inclusions of* C pneumoniae *(arrowheads) in samples was achieved using primary mouse monoclonal antibody RR402, which is specific for the major outer membrane protein antigen of* C pneumoniae *(Washington Research Foundation, Seattle) and a fluoresceinated secondary antimouse IgG monoclonal antibody with Evans blue counterstain (original magnification ×100). B, Immunocytochemical localization of chlamydial inclusions was performed using a blend of primary monoclonal antibodies against* C pneumoniae *lipopolysaccharide and major outer membrane protein antigens (monoclonal blend 807; Chemicon Int, Temecula, Calif). Brown inclusion aggregates of these* C pneumoniae *antigens (arrowheads) were visualized using a peroxidase-labeled antimouse secondary monoclonal antibody and counterstained with hematoxylin (original magnification ×100).*

and in vascular tissues.[2] Whether *C pneumoniae* is an innocent bystander or an opportunistic pathogen in chronic wounds and idiopathic diseases is an unresolved clinically relevant issue.[1,6]

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Hyper-IgE, Eosinophilia, and Immediate Cutaneous Hypersensitivity to Insect Antigens in the Pruritic Papular Eruption of Human Immunodeficiency Virus

In Brazil, pruritic papular eruption (PPE) has been found in 11.7% of patients with human immunodeficiency virus (HIV) and/or acquired immunodeficiency syndrome (AIDS) (hereinafter HIV/AIDS) and has served as a dermatological marker of HIV infection.[1] Cutaneous lesions are similar to prurigo or strophulus and have been likened to insect bites by the patients. Eosinophilia in HIV-positive patients may be associated with an immunodepressed status and possibly with increased serum IgE levels attributed to PPE or to the base disease.[2] We investigated whether PPE could be considered a prurigo or strophulus caused by insect bites.

Patients, Materials, and Methods. After approval by the ethics comittee at our institution, this study was conducted on 43 patients with HIV/AIDS who had not been using antihistamines or corticosteroids, 24 with PPE and 19 without. There was no significant difference between the 2 groups caused by the effects of opportunistic infections. Eosinophil, $CD4^+$, and $CD8^+$ cell counts

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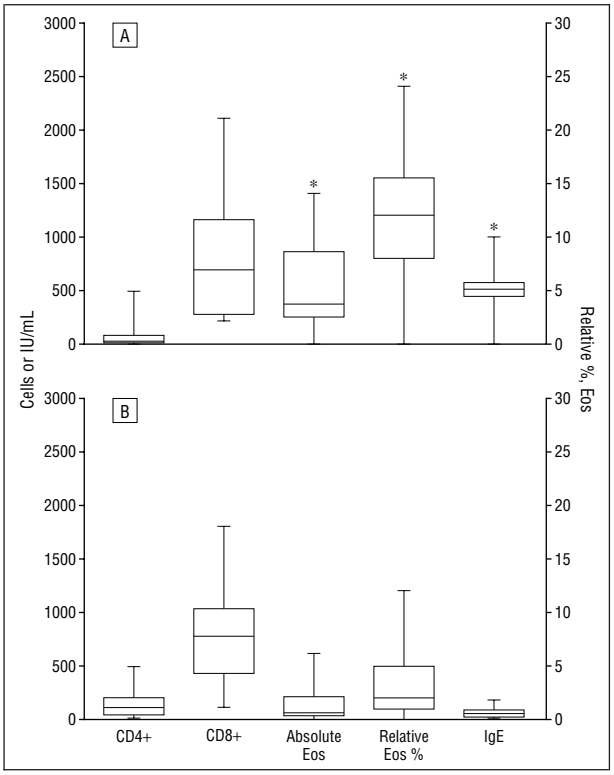

Determination of the CD4⁺, CD8⁺, cell, and eosinophil (Eos) counts (cells per milliliter) and IgE levels (IU/mL) in the peripheral blood in patients with human immunodeficiency virus/acquired immunodeficiency syndrome (median [horizontal line across each box] and 25%-75% range [top and bottom lines of each box]). A, Group with pruritic papular eruption; B, control group without pruritic papular eruption. The asterisk indicates $P<.01$.

were determined by flow cytometry in peripheral blood. Total IgE levels were measured in the peripheral blood of 18 patients (11 with PPE) by enzyme-linked immunosorbent assay (normal value, ≤100 IU/mL). The prick test was performed on 25 patients (17 with PPE) using the following antigens: household dust (Acari), mites, fungi, epithelium (mix), feathers (Alergomed, RJ; Rio de Janeiro, Brazil), and an insect body (Alerbrás, RJ; Rio de Janeiro). A 1:1000 titer of histamine was used as the positive control, and the vehicle was used as the negative control. Data were analyzed by the Mann-Whitney and Fisher exact tests.

Results. The CD4⁺/CD8⁺ ratio was reduced in the PPE group compared with the control group (mean ratios, 0.06 and 0.15, respectively; $P<.01$), and the absolute and relative eosinophilia were observed in 69.6% and 67% of the patients in the PPE group, and in 10.5% and 36.8% of the control subjects, respectively (**Figure**). Total IgE levels were higher in the PPE group than in the controls (mean levels, 510.2 and 54 IU/mL, respectively). The possibility of an association of eosinophilia and increased IgE levels with intestinal parasitosis in these patients was considered, but the patients with and without PPE were from the same region and socioeconomic level, leading us to accept the association of hyper-IgE with PPE and to consider the presence of eosinophilia an auxiliary finding in the diagnosis of PPE.

A positive response to 3 or more antigens was detected in 7 (41.2%) of the patients in the PPE group compared with 2 (25%) of the control subjects. Among these responses, only the one for insect body was significant ($P<.05$; **Table**).

Positive Response to the Immediate Hypersensitivity Skin Test in Patients With HIV/AIDS, With and Without PPE*

Antigen	With PPE (n = 17)	Without PPE (n = 8)	Fisher Test
House dust	8	1	NS
Mites	4	3	NS
Fungi	6	4	NS
Epithelium (mix)	2	2	NS
Feathers	5	1	NS
Insect body	14	3	$P<.05$

**HIV indicates human immunodeficiency virus; AIDS, acquired immunodeficiency syndrome; PPE, pruritic papular eruption; and NS, not significant.*

Comment. We found no reports of the use of prick test in PPE. Penneys et al[3] detected high titers of circulating antibodies against mosquito saliva in patients with PPE. Pradinaud et al[4] mentioned that HIV-associated prurigo could be considered an arthropod bite reaction.

Because the immune response is altered in the base disease, PPE may be compared with hypersensitivity to medications, which is common in patients with HIV. On this basis, the positive response to the prick test and/or the presence of eosinophilia and hyper-IgE with PPE may result from the reaction to insect bites occurring while the patient is in the immunodepressed state. In addition to these arguments, the increase in levels of serum interleukin 5 in patients with HIV/AIDS[5] may explain the maintained hyperproduction of IgE and the chronic nature of the lesions.

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"Easy Dressing": An Economical, Transparent Nonporous Film for Wound Care After Laser Resurfacing

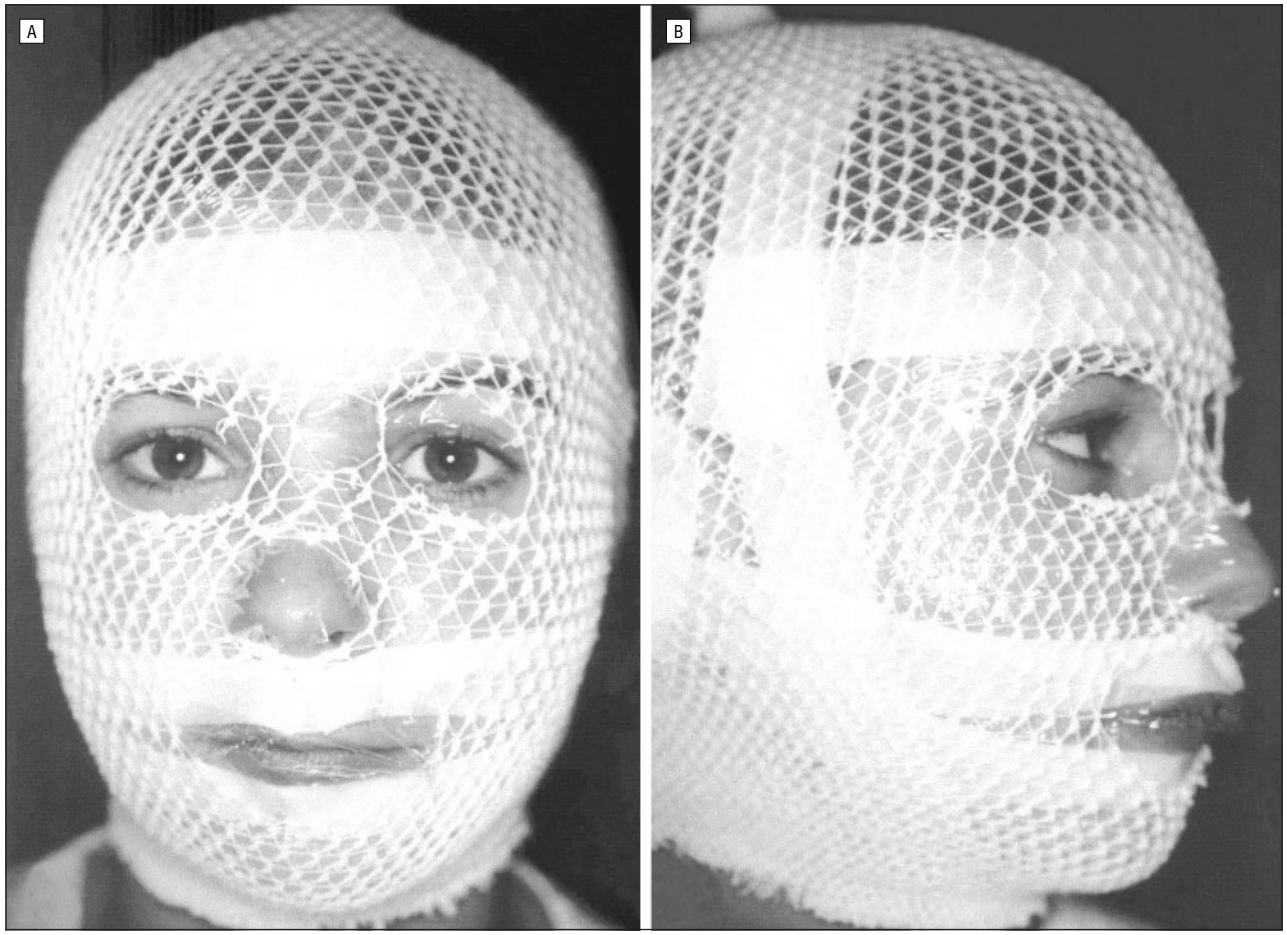
Laser resurfacing for the rejuvenation of photoaged skin has become a popular procedure, but good postoperative care is essential. One treatment option is to use commercially available occlusive or semiocclusive[1,2] dressings manufactured from a variety of compounds.[3,4] These dressings offer the advantages of reduction in pain and discomfort but at a high cost to the patient in addition to the resurfacing costs.

We present "easy dressing" as an economical alternative, which consists of readily available transparent polyethylene film known as "cling film," paper tape, and elastic tubular mesh. One 30-m roll of cling film can accommodate 20 or more dressing procedures, and the 3 ingredients together cost only a fraction of what the commercially available dressings cost.

A full-face resurfacing requires approximately 80 cm of film. The paper tape is then placed along the cut edges on both sides of the film for ease of handling. The film is then carefully rolled onto a tubular cardboard or metal former and bagged to await sterilization, which is done in the steam autoclave for 20 minutes at 120°C followed by 15 minutes of cooling. Packs of easy dressing should be prepared as required soon before or during the resurfacing procedure, as we have noticed that the sterilized film tends to become difficult to handle after long storage. Independent and extremely stringent double-controlled tests that we have done of random samples of sterilized easy dressing have produced negative results for bacterial colonies.

To use easy dressing, the resurfaced area is first cleaned off with a damp gauze and flupametsone-gentamicin cream (Flutenal gentamicin; Recordati Elmu SL, Madrid, Spain) is applied. After 30 minutes, the excess cream is gently patted off with a dry gauze. Observing sterility protocol, an assistant opens the cling film pack and holds the roll by inserting a finger in each end. The surgeon unrolls and cuts enough to cover the top half of the face completely with a good overlap, placing the film with the long edge to the top of the forehead and the bottom edge just covering the end of the patient's nose. The film is pressed carefully onto the face, following the contours, and adheres well. A second sheet of film is prepared and applied with the top long edge placed right up to the patient's nose, leaving the nostrils clear, then gently stretched to overlap with the first sheet, covering the bottom third of the patient's face.

In the center of a length of adhesive paper tape equal to the width of the lower sheet of film a small cut is made,



Patient with "easy dressing" in place: A, frontal view; B, lateral view. Note the paper tape above and below the mouth, and the tape from beneath the chin extending over the top of the head and down the contralateral side. The elastic tubular mesh holds everything in place.

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and the edges of the tape are folded back to form a shallow "V," which is placed with the center over the filtrum facing the upper lip and firmly fixed to the film. A second piece of tape of the same length is prepared and placed with the center of the V facing the middle of the lower lip, and fixed across the film below the lower lip. A third length of tape is run from under the chin, across both sheets of film, over the top of the patient's head, and down the other side. Openings are carefully cut with sterile scissors in the film over the eyes and at the mouth. Finally, a length of elastic tubular mesh bandage of an appropriate width holds the easy dressing in place. Openings are carefully cut over the eyes and the mouth (**Figure**).

The patient returns on the day after the resurfacing, and the easy dressing is carefully removed. The exudate, which is still in liquid form because the nonporous nature of the film prevents oxidation, is carefully and gently patted off with dry gauze. Flupametasone gentamicin cream is applied over the treated area and left on for half an hour. The resulting mixture of excess cream and spotty exudate is then carefully patted off with a dry gauze, and the easy dressing is applied again as above. The patient then returns 48 hours later, and the easy dressing is removed. Following this, our usual open healing regimen is indicated. Despite the nonporous nature of the dressing, no serous, bacterial, or fungal complications have occurred in more than 80 patients treated with our easy dressing technique; erythema is possibly better controlled; and healing has been at least comparable with other dressing types we've used in the past.

Cling film is easy to find, inexpensive, and can be steam sterilized. It is comparatively easy to use, fitting different treated areas and contours, and it adheres very well to the cream-covered tissue. Because the film is transparent, the tissue can be easily observed through the dressing, and because it is impermeable, the serous exudate remains liquid and is easily cleaned off. Finally, in their recent review article, Newman and coworkers[4] highly rated a polymer film dressing in the resolution of erythema and very highly in epithelization, pain relief, and absence of pruritis.

The most important advantages of easy dressing remain its low cost and excellent availability. We have found no real disadvantages.

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Carbamazepine-Induced, CD30+, Primary, Cutaneous, Anaplastic Large-Cell Lymphoma

Anticonvulsant agents may cause nodal and extranodal lymphoproliferative disorders, including dermatopathic lymphadenitis, atypical lymphoid proliferation, and cutaneous pseudolymphoma.[1] In most reported cases, regression follows the discontinuation of treatment with the causative drug, but in rare cases a true lymphoma may develop.[2,3] Herein, we describe the clinicopathologic features of the first case of a CD30+, primary, cutaneous, anaplastic large-cell lymphoma (ALCL) possibly correlated to carbamazepine administration.

Report of a Case. A 13-year-old girl experienced lipothymic episodes during her first menses. Electroencephalography findings revealed anomalous waves, and she was administered carbamazepine (Tegretol; Novartis Farma, Origgio ([Va], Italy) in increasing doses until the dose reached 600 mg daily. After 8 months of treatment, the patient was admitted to our dermatology department for an erythematous macular eruption clinically diagnosed as pityriasis rosea. The macular eruption regressed, but 1 month later she suddenly developed multiple painless reddish skin nodules that grew and ulcerated quickly (**Figure 1**); the nodules, located on the neck, trunk, and arms, varied in size from 0.5 to 6 cm, and only a few partially self-regressed. The patient was healthy; neither lymphadenopathy nor splenomegaly was detected; findings of the routine blood workup were unremarkable; cultures of swabs from cutaneous lesions yielded no bacterial pathogens. Paul-Bunnell test results were negative, and viral titers showed no clinically relevant rise.

Results of a histologic examination of a skin nodule revealed an area of epidermic pseudoepitheliomatous hyperplasia overlying a dermohypodermic diffuse lymphoid infiltrate of large anaplastic cells, scattered and in large cohesive clusters, the clusters more prominent in the deeper part of the lesion. The cellular infiltrate effaced the dermal architecture; the anaplastic cells showed a moderate degree of epidermotropism and, in some areas, a perivascular array. Almost all the anaplastic cells expressed the CD30/Ki-1 antigen (**Figure 2**), the TIA-1

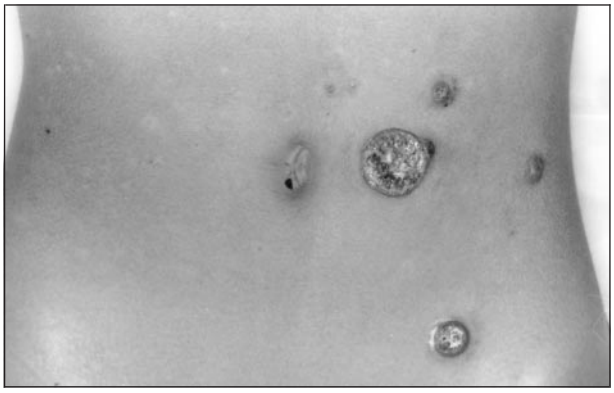

Figure 1. *Multiple ulcerated nodules of varying sizes on the abdomen.*

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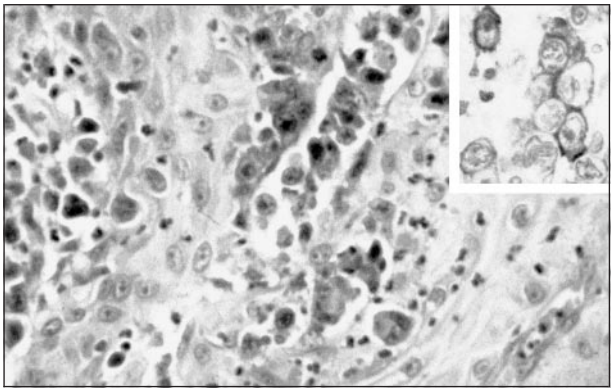

Figure 2. *The neoplastic lymphoid infiltrate depicted in the main photograph consists of anaplastic lymphoma cells (hematoxylin-eosin stain; original magnification, ×40) that positively stain (inset) with the anti–CD30/Ki-1 monoclonal antibody Ber-H2 (membrane and paranuclear "dotlike" immunostaining) (alkaline phosphatase–anti-alkaline phosphatase method; original magnification, ×100).*

antigen (a molecule associated with cytotoxic granules), and various T-cell antigens (CD3, CD43, and CD45RO), but they were negative for the monoclonal antibody ALK-1 (provided by Prof Brunangelo Falini, MD) specific for the p80 NPM-ALK. The $CD30^+$ cells were intermingled with a reactive population of lymphocytes.

Results of molecular analysis corroborated the T-cell lineage of the cellular infiltrate, showing a monoclonal rearrangement of the γ subunit of the T-cell receptor. Findings from chest radiography, abdomen ultrasonography, and total-body computed tomography scan excluded lymphoma extracutaneous localizations. Combining these clinicopathologic findings we diagnosed, $CD30^+$, primary cutaneous ALCL in accordance with the World Health Organization classification of hematological malignancies and the European Organization for Research and Treatment of Cancer classification for primary cutaneous lymphomas. Subsequently, new nodules similar to the preexisting lesions appeared on the abdomen. Carbamazepine treatment was tapered and then stopped; the patient received 8 sessions of radiotherapy with regression of 5 cutaneous lesions. Other untreated lesions self-regressed after 4 months, with considerable scarring. Presently the patient, 3 years after the lymphoma diagnosis, is healthy and her lymphoma is in complete remission.

Comment. About one third of carbamazepine adverse reactions affect the skin, mainly causing dermatitis, but also pseudolymphomas; recently a carbamazepine-induced pseudolymphoma with $CD30^+$ cells has been described.[4] However, carbamazepine-related lymphomas are unusual, and detailed information on the clinical features and outcome of such malignancies has been provided in 2 cases only. The first[2] was a nodal, large-cell, non-Hodgkin lymphoma developed after 4 months of carbamazepine administration; the other[3] was a nodal, immunoblastic, T-cell, non-Hodgkin lymphoma diagnosed after 1 month of carbamazepine therapy. Notably, in both cases the lymphadenopathies spontaneously resolved after treatment with carbamazepine was discontinued, suggesting an atypical lymphoid proliferation instead of a true malignancy.[3]

However, a clinical comparison between ours and the above-mentioned cases might be partially inappropriate because of the exclusively extranodal locations of the disease in our patient and the different therapeutic approaches. In our case the possibility of a nonneoplastic, reactive $CD30^+$ infiltrate has therefore been considered, including mainly other subtypes of $CD30^+$, primary, cutaneous lymphoproliferative disorders[5] with specific reference to lymphomatoid papulosis and lesions with borderline histologic characteristics between lymphomatoid papulosis and ALCL. In our patient, the clinical features of the skin lymphoma lesions (multiple, large nodules only partially regressing) and histomorphological characteristics (large, often cohesive sheets of $CD30^+$ anaplastic cells) excluded a diagnosis of lymphomatoid papulosis, whereas findings of histomorphological analysis and chiefly molecular biology findings (monoclonal rearrangement for T-cell receptor γ) pointed more to ALCL than to borderline lesions.

Even if no conclusive relationship between carbamazepine and $CD30^+$, primary, cutaneous ALCL may be drawn from a single case study, we deem it important for clinicians to be aware of the possible serious adverse effects of carbamazepine treatment, as the discontinuation of the drug treatment can play a crucial role in the regression of the clinical picture, sparing aggressive and harmful treatments.

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