Our results suggest that universal vitiligo represents the high end of the spectrum in general vitiligo. This is represented by an extreme clinical presentation with (sub)total depigmentation, a strong familial expression of the disease, and high prevalence of comorbidity, such as thyroid dysfunction, rheumatoid arthritis, and alopecia areata. Health-related QOL is comparable to what is found in general vitiligo except for poorer functioning in daily life and physical HRQOL, which probably reflects comorbidity.

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Scalp Psoriasis: A Paradigm of “Switch-On” Mechanism to Anagen Hair Growth?

Psoriasis is characterized by an increase of epidermal proliferation. The consequence of psoriasis on hair growth is not well known. It was previously suggested that both psoriatic epidermal lesions and anagen hair growth shared the same “switch-on” mechanism based on the comparison of the Koebner phenomenon and wound-induced hair growth.1 We provide new data to support this hypothesis.

Methods. We studied 7 consecutive patients (including the patient in the index case) with sharply demarcated, chronic, untreated psoriatic plaques on their scalps (Table). The index case involved a 41-year-old man with androgenetic alopecia and a history of flares of cutaneous psoriasis who decided to shave his scalp completely. After shaving, he developed psoriatic plaques on his scalp, and when his hair started to grow back, he noticed that the hair appeared to be denser in the areas affected by the psoriatic plaques than in other areas of the scalp. He had 1 plaque in the occipital region (Figure 1) and 2 plaques in the vertex region (Figure 2).

Results. The 6 cases other than the index case were studied with videomicroscopic analysis. The psoriatic areas of the scalp (Figure 3, A) were compared with adjacent nonaffected areas (Figure 3, B). Videomicroscopic analysis demonstrated that the denser appearance of the plaques was attributable to a higher hair count, not to the length of the hair, which was always the same in both the normal areas and the areas with the psoriatic plaques.
Comment. We report an increased density of hair in psoriatic plaques. Psoriasis- and wound-induced hair growth may share the same switch-on mechanism. In 1956, Argyris reported that wounding of the skin induced hair growth. Recently, Osaka et al demonstrated the critical involvement of macrophages in wound-induced hair growth in a model of mice. Apoptosis signal-regulating kinase 1 (ASK1) is a member of the mitogen-activated protein kinase family that is required for the recruitment and activation of macrophages. Wound-induced hair regrowth is impaired in ASK1-deficient mice. It activates both c-Jun NH2-terminal kinase and p38 mitogen-activated protein kinase pathways in response to inflammatory cytokine and physical stress. Transplantation of cytokine-activated bone marrow–derived macrophages also strongly induces hair growth in both ASK1-deficient and wild-type mice. Interestingly, recent studies provide support for a major contribution of monocytes and macrophages in psoriasis initiation. Marble et al demonstrated an increase of macrophages and dermal dendritic cells in prepsoriatic skin.

The role of psoriasis in hair growth induction is further supported by the recent finding of an increased level of nuclear β-catenin in the suprabasal psoriatic epidermis compared with uninvolved or normal skin. β-Catenin is known to control the hair cycle, and its expression is correlated with the induction of the anagen phase and the differentiation of stem cells. The ability to induce growth of new hair follicles by transiently activating β-catenin signaling in adult mouse epidermis is additional corroborating evidence.

In conclusion, we suggest that scalp psoriasis may be caused by an increase in the recruitment of stem cells, resulting in a switch-on entry in the anagen phase. As compared with wound-induced hair growth, monocytes and macrophages could have a key role in this cascade of events in psoriasis by upregulation of the Wnt/β-catenin pathway.

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We read with interest the report by Creamer et al in the September issue of the Archives, which described 10 patients with eczematoid dermatitis that occurred after allogeneic hematopoietic stem cell transplantation. The authors considered this eruption to represent a novel form of chronic cutaneous graft-vs-host disease (GVHD). We have seen several bone marrow transplant recipients who presented with a similar eruption and considered them to have one of the papulosquamous variants of GVHD and not a novel form. The decision to restrict or separate various presentations of a disease may be regarded as “lumping” or “splitting” and is the rationale for classification schemes. Creamer and colleagues suggest that a distinction of this clinical presentation of GVHD is important because all 10 patients developed erythroderma and had substantial associated morbidity and mortality. However, the increased morbidity and mortality might be attributable to the extent of disease (erythroderma) rather than to the presence of eczematous findings that were observed clinically and histologically in these patients. Moreover, the clinical and pathologic findings in this series of patients are more consistent with those designated as acute GVHD, even though Creamer and coauthors’ patients had a protracted and “chronic” skin course.

Traditionally, chronic GVHD is defined by manifestations that present after 100 days posttransplantation. This concept has since been refuted, and most recognize that the clinical manifestations of acute GVHD may occur before or after 100 days posttransplantation. Acute GVHD reactions may be precipitated by donor T-lymphocyte infusions for treating relapses or after tapering of immunosuppressive therapy. Of note, 8 of the 10 patients described developed the eruption after the aforementioned precipitating factors (5 of 10 after immunosuppression tapering and 3 of 10 within weeks of donor lymphocyte infusions). We therefore conclude that the eczematoid presentation may be more accurately classified as a variant of acute GVHD.

The histopathologic findings also support this argument. The criteria for histopathologic diagnoses of chronic GVHD include changes that are typical of other chronic conditions, ie, hypergranulosis, acanthosis, dermal thickening, dermal sclerosis, and/or fibrous thickening of the fascial septa. The 10 patients described by Creamer and colleagues had features of acute GVHD, but the features required for the histopathologic diagnosis of chronic GVHD were absent.

In conclusion, it is our opinion that eczematoid GVHD represents acute GVHD and should be treated as such. Standard treatment involves increasing the immunosuppressive therapy. Second-line or salvage therapy should be started if disease progression is noted after 3 days, if no response is seen after 7 days, or if an incomplete response is noted after 14 days. Psoralen–UV-A (PUVA) is often considered a second- or third-line treatment in corticosteroid-resistant cases. The article of Creamer et al is not clear regarding when PUVA was added to the regimen, or if the initial treatment had failed. It must be remembered that PUVA is not standard first-line therapy and that patients with extracutaneous disease tend to have poorer responses. We agree that in corticosteroid-resistant patients PUVA is a modality that can be helpful and allow reduction in corticosteroid doses.

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In reply

We were interested to read Cook-Norris and Weenig’s comments on our description of 10 patients who, after allogeneic hematopoietic stem cell transplantation, developed a chronic eczema that was histopathologically characterized by features of both GVHD and dermatitis. We have described this eruption as an eczematoid form of chronic cutaneous GVHD. However, Cook-Norris and Weenig suggest that the clinical pathologic features point away from a chronic GVH reaction and argue that the dermatosis represents a papulosquamous variant of acute GVHD.

In our experience, acute cutaneous GVHD presents and behaves differently from the eruption that Cook-Norris and Weenig described. Acute GVHD is an exanthem of macular, usually morbilliform, erythema with acral and mucosal involvement. In hyperacute GVHD, extensive keratinocyte necrosis may lead to blistering and a toxic epidermal necrolysis–like picture. Most cases of acute GVHD can be controlled rapidly with systemic immunosuppressive therapy. By contrast, the eruption that we have called eczematoid GVHD is characterized by widespread, usually erythematous, dermatitis accompanied by intense pruritus, scaling, desquamation, and a susceptibility to staphylococcal infec-