Reversal of canities

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young adults. It is unclear whether MP is distinct from PG.2 The reported MP cases have similar clinical features, such as facial and preauricular ulceration, while, in contrast to PG, they lack undermining or surrounding erythema. Defects in cell-mediated and humoral immunity as well as lymphocyte, neutrophil, and monocyte function have been reported, but none of these findings have been demonstrated consistently.3

Malignant pyoderma remains a therapeutic challenge.3,4 Although high-dose systemic corticosteroids and dapsone may induce clinical remission in many patients, refractory cases have been described.2 Treatments with systemic corticosteroids, cyclosporine, dapsone, azathioprine, tacrolimus (FK506), chlorambucil, mycophenolate mofetil, methotrexate, and infliximab have all been reported to improve the skin manifestations to various degrees.2,4,5 In the present case, rapid disease progression, significant tissue destruction, and refractoriness to steroids prompted increased immunosuppression with a combination of agents including tacrolimus, dapsone, and corticosteroids. The clinical response was excellent. In conclusion, it appears that this combination is effective and safe in patients with MP, and it can be considered first-line treatment in aggressive disease refractory to corticosteroids.

Reversal of Canities

Report of a Case. A 67-year-old, otherwise healthy man presented with spontaneous repigmentation of his gray hair (Figure).

Comment. The reports on this phenomenon are scant in the literature,5,6 though it may not be as rare as assumed. In fact, it is not too uncommon to see spontaneous repigmentation along the same individual hair shaft in early canities (loss of hair pigment resulting in graying of the hair). Moreover, melanocytes taken from white hair follicles can be induced to pigment in vitro.3 The most dramatic cases of return of normal hair color from white

Figure. Clinical appearance of malignant pyoderma. A, Patient with extensive papulopustules and skin ulcers prior to treatment. B, Patient with cribriform scars 6 months after treatment.

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are probably examples of pigmented hair re-growth following alopecia areata or the Marie Antoinette syndrome. Repigmentation of hair observed in Addison disease has been attributed to a mechanism similar to that seen in alopecia areata, in view of the known association between these 2 presumably autoimmune diseases. On the other hand, it may also be explained through the effect of elevated levels of melanocyte-stimulating hormone, which applies to pigmentation of skin and hair in Nelson syndrome and ectopic adrenocorticotropic hormone syndrome as well. Since the stimulation of pigment formation may also affect the hair, a conspicuous darkening of the hair should suggest the possibility of these disorders. In our patient, however, careful evaluation for underlying endocrinologic disorders or neoplastic disease did not reveal any abnormalities.

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Figure. Clinical images of spontaneous hair repigmentation. A, Gray scalp hair of patient before onset of spontaneous repigmentation. B, Universal reversal of canities. C, Close-up view of hair strands: while the distal hair shafts are white, the proximal hair shafts show dark pigmentation.

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