Follicular Mycosis Fungoides – Augmentation by PUVA Therapy

Sir,

The plaque type of mycosis fungoides is frequently associated with alopecia. Its cause is mostly mucinous follicularis resulting in permanent alopecia associated with cystic follicular lesions. In rare cases involvement of hair follicles by T-cell infiltrate leads to transient alopecia. We here describe a case of alopecia due to follicular mycosis fungoides with additional unusual features.

CASE REPORT

A 22-year-old white man presented with rather inconspicuous discrete hypopigmented lesions on both legs, which had developed during the past 2 years. The lesions then generalized and transformed into erythematous infiltrated and scaly plaques. Alopecia and follicular keratoses finally occurred. Several biopsies, including immunopathology, confirmed the diagnosis of mycosis fungoides. Follicular mucinosis was, however, not found.

Histopathologic findings before PUVA therapy revealed a dense dermal infiltration of lymphocytes, with marked epidermotropism and involvement of hair follicles in the absence of mucinosis (Fig. 1).

Routine laboratory tests, a peripheral blood smear and CD4/CD8 ratio were normal. Chest X-ray and abdominal ultrasound did not demonstrate any pathologic findings. Topical corticosteroids combined with oral photochemotherapy (8-MOP plus UVA) were started. PUVA therapy induced a strong flaring-up reaction with erythema, scaling and burning. In addition, new lesions were unmasked, revealing a much more extensive skin involvement than suspected on clinical inspection before therapy (Fig. 2).

Histopathologic findings at that time included parakeratosis, slight acanthosis with necrotic keratinocytes, vacuolization of basal cells and moderate infiltration of lymphocytes and histiocytes in the stratum papillare. This picture combined the features of regressing T-cell lymphoma and a marked phototoxic effect as a consequence of PUVA therapy.

DISCUSSION

This case report should draw attention to three peculiar features: (1) alopecia not associated with mucinosis follicularis but due to follicular involvement by the specific infiltrate of mycosis fungoides; (2) the young age of our patient; and (3) an intensive intralesional inflammatory skin reaction induced by PUVA therapy.

Two cases of mycosis fungoides recently reported by Lacour et al. were characterized by marked follicular manifestations without mucinosis leading to alopecia (1). Our histopathologic findings were similar. In contrast to these patients our patient has not yet developed comedo-like lesions and epidermal cysts. Cutaneous T-cell lymphoma usually occurs in middle-aged and elderly patients. Our patient was only 22 years old when the diagnosis of mycosis fungoides was made. In the literature several cases of cutaneous T-cell lymphomas in persons younger than 40 years of age have been reported (2–4). Handfield et al. describe the case of a 25-year-old Jamaican woman with hypopigmented mycosis fungoides (65); this case is notably similar to ours.

PUVA therapy is generally accepted as an effective therapy for mycosis fungoides. Long-term side-effects include pigmentary changes, an increased risk of developing skin cancers and chronic actinic damage. Rare short-term side-effects include pruritus, hypertrichosis, nail changes, acneiform eruptions and blisters in the absence of phototoxic reactions. Wolf et al. reported an isolated case of PUVA-induced lymphomatoid papulosis in a patient with mycosis fungoides (6). When PUVA therapy was started, our patient developed an unusually strong reaction with burning, erythema and infiltration within the plaques and areas of alopecia. Furthermore, widespread new lesions were unmasked by PUVA treatment. After several weeks of PUVA therapy, the alopecia and erythematous infiltrates showed a marked improvement and almost disappeared.

It is a well-known phenomenon that plaques of mycosis fungoides react more intensively by an inflammatory response to PUVA treatment than normal skin (7). The mechanisms responsible for this inflammatory reaction are unknown but are

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Fig. 1. Before PUVA therapy: dense dermal infiltration of lymphocytes, with marked epidermotropism and involvement of hair follicles in the absence of mucinosis. Hyperkeratotic plug in the acroinfundibulum.
likely to involve the release of inflammatory mediators by T-cells infiltrating upper dermis or epidermis following phototherapy.

REFERENCES

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Fig. 2. Flare-up of lesions and unmasking of widespread new lesions during PUVA treatment.