

Table 1. The UV-protective effects of six of the twenty textiles examined

Material	Weight (g/m ²)	Transmission (average of the 313, 365, 436 nm bands) (%)	Protection factors
Womens' tights, beige	31	75	1.3
Patient's shirt, white cotton	154	14	7
Thin cotton, green	87	10	10
Knitted wool, white	700	8	12
Velvet, dark green	196	2	50
Denim, dark blue	319	0.06	1 700
Patient's shirt, white cotton, two layers		5	19

as light colour. Small holes from stitches diminished the protective effect considerably. Two layers of cloth gave an additive effect. A cotton material of medium thickness transmitted 4% of the UV radiation.

DISCUSSION

We have been able to show a wide variation in the light-protective effects of twenty commonly used textiles. The range of protection factors was from 1.3 to 1 700, showing that photo-sensitive patients' choice of clothes is very important. A thin cotton shirt gives a protection factor of 10, which is more than the commonly used sun screens (2). In contrast to most sun screens, clothes protect well at various wavelengths, including long-wave UV radiation and visible light. A tightly woven material gives a greater light absorption than a loosely woven one. Furthermore, dark colour and two layers of cloth add to the protective effect. We suggest that, for the light-sensitive and the psoralen-sensitized patient, careful counselling concerning clothes is at least as important as the prescription of proper sun screens.

Of the materials tested, we found that blue denim gave the best UV-protective effect. A dark-coloured tightly woven cotton material of medium thickness also gave good protection. We therefore suggest to light-sensitive patients that they dress in the materials mentioned above and wear trousers/slacks instead of stockings.

REFERENCES

- Alsins, J., Claesson, S., Fischer, T. & Juhlin, L.: Development of high intensity narrow-band lamps and

studies of the irradiation effect on human skin. *Acta Dermatovener (Stockholm)* 55: 261, 1975.

- Cripps, D. & Hegedus, S.: Protection factor of sun-screens to monochromatic radiation. *Arch Dermatol* 109: 202, 1974.

Treatment of *Pityriasis rubra pilaris* with an Oral Aromatic Retinoid (RO 10-9359)

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Abstract. A patient with widespread PRP was first treated with an oral aromatic retinoid (RO 10-9359) and PUVA in successive periods. Retinoid treatment resulted in marked improvement but during PUVA treatment the disease became re-activated. The patient was finally treated with aromatic retinoid alone, which brought the disease to remission in 6 weeks.

Key words: Pityriasis rubra pilaris; Aromatic retinoid (RO 10-9359); Hyperproliferation; Follicular hyperkeratosis

Pityriasis rubra pilaris (PRP) is a rare, idiopathic dermatosis showing varying degrees of follicular hyperkeratosis, palmoplantar keratoderma and erythroderma.

PRP can be very distressing to the patient on account of widespread skin involvement and limitation of movement caused by thickening and fissur-



Fig. 1. The hands and forearms of the patient before treatment. The skin of the forearms is scaly and erythematous. There is marked hyperkeratosis on the dorsum of the hands.

ing of the palmoplantar skin. Many methods of therapy have therefore been tried, including topical or systemic corticosteroids, vitamin A, antimetabolites, topical retinoic acid and, lately, PUVA therapy (1, 7).

During the last few years oral retinoids, analogs of vitamin A, have been found to exert a beneficial therapeutic effect on dermatoses showing hyperproliferation and disturbed keratinization (8). Both these features appear to be present in PRP (7). To study the effect of retinoid on PRP, a patient with widespread PRP was treated with aromatic retinoid (RO 10-9359) and PUVA.

CASE REPORT

A farmer aged 55 years was hospitalized in March 1979. One month earlier small, discrete, reddish follicular papules had appeared on his face. Later the papules coalesced and spread over the body as a scaly rash, excluding some areas, as is typical in PRP. The skin on the palms and soles was thickened and yellowish. Histologic examination revealed typical features of PRP, i.e. hyperkeratosis, distended follicular openings with parakeratosis, irregular acanthosis, a distinct granular layer, and mild perivascular lymphocytic infiltration in the upper dermis.

Initial treatment consisted of aromatic retinoid with a

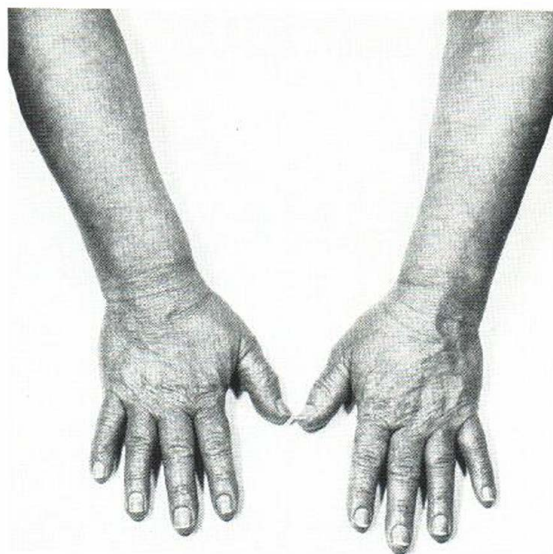


Fig. 2. Same patient after 2 weeks of treatment with an oral aromatic retinoid (RO 10-9359).

daily dose of 60 mg (0.8 mg/kg). After 2 weeks the erythema was less pronounced and the affected areas were markedly thinned. Palmoplantar hyperkeratosis had desquamated as large sheets. The following 2 weeks gave further improvement. The dose was then reduced to 50 mg and a concomitant PUVA therapy was started. After 2 weeks of combined treatment the retinoid dose was reduced to 30 mg. This treatment was maintained for the next 4 weeks, after which only the PUVA therapy was continued. Shortly after this, the disease became reactivated; new, hyperkeratotic, follicular papules and erythematous plaques appeared. PUVA treatment was finally stopped after a further 2 weeks because of a rise in sified the erythema.

In August 1979 transaminases were normalized and PUVA therapy was tried again. The first treatment, however, caused immediate exacerbation of the erythema, with fever. Later UVA alone was tried, but this too intensified the erythema.

Treatment with aromatic retinoid was resumed in October with an initial daily dose of 60 mg. This was reduced to 40 mg after 2 weeks and to 30 mg after a further 2 weeks. By this time, in 4 weeks, marked improvement had been achieved with full remission in 6 weeks. There remained only some terminal subungual hyperkeratosis, which had been very pronounced at the beginning of this treatment period. At present, after 14 weeks of treatment with retinoid, remission is still maintained on a dosage of 20 mg.

DISCUSSION

It is often difficult to evaluate the efficacy of a treatment of PRP owing to the tendency to spontaneous remission (4). However, in the case pre-

sented above the improvement could definitely be attributed to retinoid therapy.

During the first period of treatment, marked improvement was achieved with retinoid alone in 4 weeks. The improvement continued when PUVA therapy was added to retinoid. After discontinuation of retinoid the lesions appeared again in spite of continued PUVA therapy. During the second treatment period retinoid was given alone, leading to full remission in 6 weeks.

Retinoids have been found to affect both proliferation and differentiation of epithelial cells (8). In hyperproliferative epidermis as in psoriasis, they have a stabilizing effect: proliferation is slowed down and the keratinizing disorder is normalized (8, 9).

Proliferation is accelerated in PRP too, but is not as rapid as in psoriasis (7). It is probable that the action of retinoid on proliferation involves the same mechanism in both of these diseases. In our study the patients with PRP were in remission after 4 to 6 weeks of retinoid therapy; the maximum effect in psoriasis is usually seen after 6 weeks (3, 6), as might be expected on the basis of the more accelerated lesional proliferation.

Psoriasis and PRP exhibit differences in the keratinizing disorder, one of the characteristic features of PRP being follicular plugging. A similar disorder is seen in vitamin A deficiency, and is cleared with vitamin A substitution. In the light of recent investigations, however, vitamin A does not seem to play any part in the pathogenesis of PRP (7). It is evident that in cases of PRP responding to large doses of vitamin A, it acts as an active pharmacologic agent rather than as replacement therapy (5). Aromatic retinoid is an analog of vitamin A with a more specific action in the epithelial cells (8). Consequently it can more effectively combat this kind of disorder.

The only side effect that was seen during the treatment periods with retinoid was dryness of the lips, which subsided when the dose was reduced. A transient rise in transaminases was noted shortly after discontinuation of the combination treatment, when PUVA treatment alone was continued. It is obvious that simultaneous treatment with retinoid and PUVA must be given with caution, although it is shown to be well tolerated by most patients (2).

There was one additional feature in our case. Laboratory investigations revealed positive Rose-Waaler and latex fixation tests, findings sometimes

seen in association with PRP (7). The Rose-Waaler test showed a quantitative decrease from 1:1000 to 1:250 during treatment. No other signs of autoimmune aberrations were noted; nor had the patient any muscle or joint involvement.

In our study, PUVA did not bring PRP under control, in contradiction to the observations of Brenner et al. (1). On the contrary, it caused rapid exacerbation of the erythema when given as initial therapy in the active stage of the disease. The patient also exhibited sensitivity to UVA radiation given alone. Other authors have reported that even sunlight can occasionally exacerbate the eruption (4). Aromatic retinoid, on the other hand, had an excellent therapeutic effect on PRP, agreeing with preliminary observations made with retinoids (10, 11). An initial dose of even less than 1 mg/kg seems to be effective in bringing the disease to remission.

REFERENCES

1. Brenner, W., Gschnait, F., Hönigsmann, H. & Fritsch, P.: Erprobung von PUVA bei verschiedenen Dermatosen. *Hautarzt* 29:541, 1978.
2. Fritsch, P., Hönigsmann, H., Jaschke, E. & Wolff, K.: Augmentation of oral methoxalen-photochemotherapy with an oral retinoic acid derivative. *J Invest Dermatol* 70: 178, 1978.
3. Goerz, G. & Orfanos, C.: Systemic treatment of psoriasis with a new aromatic retinoid. *Dermatologica* 157 (Suppl. 1):38, 1978.
4. Griffiths, W.: Pityriasis rubra pilaris—an histological approach. *Clin Exp Dermatol* 1:37, 1976.
5. Lamar, L. & Gaethe, G.: Pityriasis rubra pilaris. *Arch Dermatol* 89:515, 1964.
6. Lassus, A.: Systemic treatment of psoriasis with an oral retinoic acid derivative (RO 10-9359). *Br J Dermatol* 102: 195, 1980.
7. Niemi, K.-M., Kousa, M., Storgårds, K. & Karvonen, J.: Pityriasis rubra pilaris. A clinico-pathological study with a special reference to autoradiography and histocompatibility antigens. *Dermatologica* 152: 109, 1976.
8. Orfanos, C., Pullman, M., Runne, U., Kurka, M., Strunk, V., Künzig, M. & Dierlich, E.: Behandlung der Psoriasis mit Vitamin A. Vitamin-A-Säure und oralen Retinoiden. *Hautarzt* 30: 124, 1979.
9. Orfanos, C. & Runne, U.: Tissue changes in psoriatic plaques after oral administration of retinoid. *Dermatologica* 157 (Suppl. 1):19, 1978.
10. Peck, G., Yoder, F., Olsen, T., Pandya, M. & Butkus, D.: Treatment of Darier's disease, lamellar ichthyosis, pityriasis rubra pilaris, cystic acne, and basal cell carcinoma with oral 13-*cis*-retinoic acid. *Dermatologica* 157 (Suppl. 1):11, 1978.
11. Schimpf, A.: Zur systemischen Anwendung eines aromatischen Vitamin-A-Säure-Derivates (RO 10-9359) bei Psoriasis und Keratosen. *Z Hautkr* 51: 265, 1976.