Table I. *Relevant data of the patients*

Pat. no.	Sex	Age (y.)	Initial body-weight (kg)	Diagnosis	Dosage (initial treat-ment)	Duration (initial treatment)	Dosage (main-tenance treatment)	Duration (main-tenance treat-ment)	Thera-peutic results	Side effects
1	F	8	23	Psoriasis vulgaris	25 (mg/day)	6 weeks	12.5 (mg/day)	15.5 (months)	Excellent	Moderate cheilitis
2	F	8	24	Psoriasis vulgaris	25	4 weeks	12.5	12	Excellent	None
3	F	12	38.5	E.K.V.[b]	25	12 months[a]	–	–	Excellent	None
4	F	10	38	E.K.V.	30	6 weeks	15	9.5	Excellent	Moderate cheilitis
5	M	10	33	E.K.V.	30	6 weeks	15	9.5	Excellent	Dry lips

[a] Reduction of the dosage led to a slight exacerbation.
[b] Erythrokeratodermia variabilis.

Table II. *Growth rate of the patients*

Patient no.	Duration of therapy (months)	Growth rate (cm/y.)
1	17	5.6
2	13	4.6
3	12	6.0
4	11	4.6
5	11	5.5

dosage, we consider its use acceptable in children —provided that regular monitoring of the height is carried out.

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Azathioprin in the Treatment of Airborne Contact Dermatitis from Compositae Oleoresins and Sensitivity to UVA

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Seven patients with contact dermatitis from compositae oleoresins were described by Hjorth et al. 1976. The dermatitis was localized on light-exposed areas and had previously been diagnosed as photodermatitis or actinic reticuloid.

The treatment of these patients has been beset by difficulties. Topical steroids do not control the disease—and avoidance of the allergens is impossible since they are airborne.

The management of these patients, therefore, presents us with serious problems.

CASE REPORTS

Case 1. A 57-year-old male farmer with dermatitis on light-exposed areas from May to December since 1950. The skin on his face and hands was extremely lichenified and erythematous—clinically resembling actinic reticuloid. He felt the condition to be extremely disabling. He had many positive patch tests to compositive oleoresins and also proved to be sensitive to UVA. Steroid

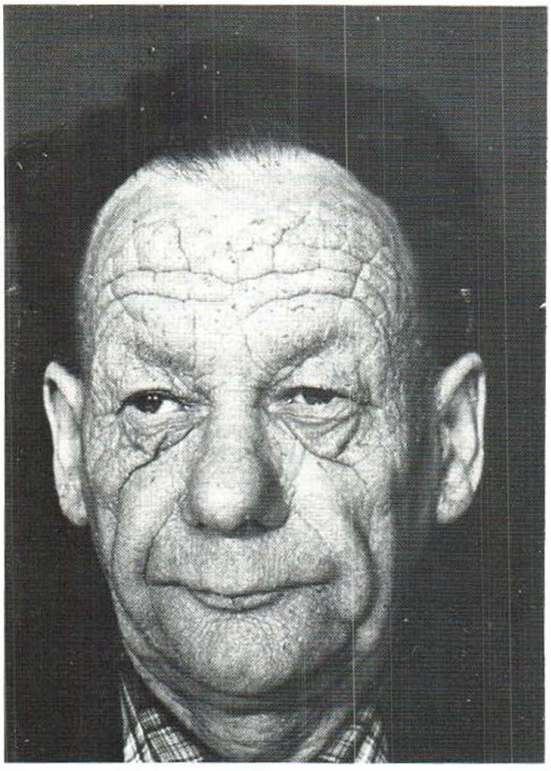

Fig. 1. December 1978, before treatment with azathioprin.

Fig. 2. October 1979, after 9 months of treatment with azathioprin.

creams had little effect. Sun protection with oral betacarotene and Piz Buin (R) cream had no effect. Systemic treatment with prednisone gave some relief.

A personal communication from D. S. Wilkinson led to treatment with azathioprin, 50 milligrams b.i.d. The treatment started in January 1979, since when the patient has been symptom-free, and the skin is of practically normal appearance (Figs. 1 and 2), although after 9 months' treatment slightly thickened.

Case 2. A 62-year-old working man with dermatitis on the face, neck and arms since 1952. The eruption is worse from August to December, though sun exposure never aggravates the condition.

The affected skin is lichenoid, lichenified and excoriated and the skin disorder has gradually worsened over the years, with increasing pruritus.

Positive patch tests to chromate, colophony and perfume have been found as well as many positive reactions to compositae. There was a normal sensitivity to UVA and UVB. Prednisone 30 mg daily was able to control the dermatitis, but tapering off to 15 mg daily resulted in a flare-up.

In July 1979 a regime of azathioprin 150 mg daily was instituted and after one month the skin lesions were considerably improved. Continued treatment has prevented the usual aggravation during the autumn, even if the dermatitis has not cleared completely after 5 months of treatment.

COMMENTS

The therapeutic results in these patients will of course lead to treatment of further patients with recalcitrant dermatitis from compositae oleoresins—with and without sensitivity to UVA.

Frain-Bell & Johnson (1) also believe that there is a definite association between sensitivity to compositae oeleoresins and actinic reticuloid and it is obvious that the effect of azathioprin should be investigated in patients with actinic reticuloid, both with and without contact allergic sensitivity.

Previous reports about treatment of patients with actinic reticuloid mention sunscreens, oral betacarotene, steroids oral and topical, chemotherapy with methotrexate, cyclophosphamide and chlorambucil—yet all have failed to bring about objective or subjective improvement (3, 4, 5).

Azathioprin is an antimetabolite which blocks the synthesis of DNA or RNA and thus affects only proliferating cells. As for other cytostatic drugs used in dermatology, the indications for the use of azathioprin are empirical, being based upon clinical investigations.

Although the mechanism of the effect of azathioprin is not known, the clinical effect has been so striking that a report is warranted.

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Cefuroxime Treatment of Urethritis Caused by a β-lactamase-producing Strain of *Neisseria Gonorrhoeae*

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Abstract. A patient who contracted urethritis from a β-lactamase-producing strain of *Neisseria gonorrhoeae* was successfully treated with the cephalosporin derivative cefuroxime. As expected, neither cefuroxime nor cefamandole was hydrolysed by plasmid-coded gonococcal β-lactamase. Cefuroxime ought to be a valuable and efficacious substitute for penicillins in the treatment of gonhorrhoea due to β-lactamase-producing gonococcal strains.

Key words: Neisseria gonorrhoeae; β-Lactamase; Plasmids; Cefuroxime

Ever since its discovery, penicillin has been of the utmost importance in the treatment of gonorrhoea. Until 1976, the resistance of *Neisseria gonorrhoeae* to penicillins has rarely created serious therapeutical problems. However, β-lactamase-producing gonococcal strains were isolated for the first time in 1976 (9, 10). The β-lactamase was found to be a TEM-1 enzyme and is thus identical with the β-lactamase found in *Haemophilus influenzae* and many Gram-negative enterobacteria. It has been established that the β-lactamase of *Neisseria gonorrhoeae* is plasmid mediated (3). Two plasmids have been found, 3.3 Mdal and 4.4 Mdal in size, respectively.

It is likely that the 3.3 Mdal is derived from the 4.4 Mdal plasmid. The latter plasmid has probably been transmitted from a β-lactamase producing strain of *Haemophilus para-influenzae* (Sparling, unpublished information). *Neisseria gonorrhoeae* strains carrying the 4.4 Mdal resistance plasmid can transfer the plasmid to sensitive strains by conjugation (cell to cell contact) due to the presence of a large conjugative 24.5 Mdal plasmid (2, 12).

The fact that all β-lactamase-producing gonococci so far examined contain either of the above-mentioned resistant plasmids, makes it possible to predict the rate of β-lactam hydrolysis for a large number of different clinically relevant β-lactams (penicillins, cephalosporins and cefamycins). Thus the TEM-1 enzyme, a typical ampicillinase, is not very effective against most cephalosporins including the new β-lactamase-resistant derivatives, cefuroxime and cefamandole. It was therefore of interest to clinically evaluate one of these drugs, cefuroxime, in the treatment of a patient with urethritis caused by a β-lactamase-producing strain of *Neisseria gonorrhoeae*.

MATERIALS AND METHODS

Strain 1773 was a gonococcal isolate from the urethal discharge of the patient, whose case is described in the text. The β-lactamase-producing strain CDC67 was obtained from Stanley Falkow and is known to carry the 2.6, 4.4 and 24.5 Mdal plasmids. The solid medium used was GC medium base supplemented with Iso-Vitalex (BBC). Plates were incubated in 6% CO_2 in a CO_2 incubator. The complete liquid medium was identical with the solid medium except that agar was omitted. β-lactamase activity when using penicillins as substrates was assayed by the automatic iodometric method of Lindström & Nordström, (1). When cephalosporins were used as substrates the spectrophotometric method of Fu & Neu (5) was used. Specific activity in nanokatal is defined as