DISCUSSION

We report two cases of scleromyxedema with cerebral disease, treated with alkeran and dermabrasion. These add support to previous data (6, 7) indicating an increased risk of central nervous system disease in scleromyxedema. The first case presents a firm diagnosis of herpes encephalitis, which makes suspicion of cerebral involvement caused by scleromyxedematous plaques less likely. Immunoglobulin disturbance, with paraprotein formation might, however, have helped to lower the patient's resistance to the herpes infection. In the second case, we must so far assume that we are dealing with a coincidence of two diseases, but it is noteworthy that the histologic appearance of a meningeoma is dominated by large stellate fibroblasts, just as found at the skin microscopy.

Available literature concerning effective treatment of scleromyxedema is sparse. Feldmann & Shapiro (2) reported a case in 1969 with a dramatic response to melphalan (alkeran), and Wright & Franco (8) treated a case successfully with alkeran and dermabrasion, while Hill & Crawford (4) used radiation therapy.

In our first patient we were able to start treatment with melphalan (alkeran) and later dermabrasion, before the skin had turned completely sclerotic around the natural openings of the face. At present it seems that progression of the disease has been arrested. The existing sclerotic areas in the glabella and on the cheeks have been and will be further treated with dermabrasion. In the second patient it is hard to say if there has been any effect of the treatment with alkeran. Treatment has only proceeded systematically for a very short time. Dermabrasion has had some beneficial effect.

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Absence of Cataract Ten Years after Treatment with 8-Methoxypsoralen

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Abstract. Thirteen patients treated with 4–41 g of 8-methoxypsoralen (8-MOP) for vitiligo were examined with regard to ocular damage 2–12 years after start of the treatment. No signs of eye damage were registered. This result indicates the need for more comprehensive retrospective examinations of 8-MOP-treated patients in order to gain information on the practical risk of cataract formation.

Key words: Cataract; 8-methoxypsoralen

The risk of cataract formation during photochemotherapy (PUVA) is a source of concern to an increasing number of dermatologists. However, cataracts after PUVA treatment have been observed only in experimental animals treated with PUVA for prolonged periods with very high doses of psoralens (2, 3). The UVA light penetrates both the cornea and the aqueous humor, i.e. it reaches the lens, also in the human eye (1). 8-methoxypsoralen (8-MOP) has been found both in aqueous humor and lens after oral administration (6). The physical prerequisites for a photochemical reaction thus are available also in the lens of the human eye. Despite this fact, long-term use of psoralens for the treatment of vitiligo in many patients does not seem to have caused any cataracts (4). However, no adequate follow-up studies with ophthalmologic examination of treated patients seem to have appeared as yet.

PATIENTS AND METHODS

Mainly during the 1960s, patients with vitiligo were treated orally with 8-MOP, Meladenine®, 10 mg *t.i.d.* in

Table I. *Patients treated with 8-methoxypsoralen for vitiligo*

Patient	Age	Period of treatment	Total dosis (g)	Ophthalmologic status in 1977–78
S. B.	73	1965–67	4	Normal
K. J.	30	1965–68	5	Normal
D. L.	48	1967–68	5	Normal
I. H.	53	1966–69	8	Normal
L.-E. F.	25	1968–69	9	Normal
M. G.	46	1968–69	9	Normal
S. A.	69	1965–69	10	Normal
R. J.	62	1967–68	10	Normal
E. G.	33	1966–68	11	Normal
E. T.	45	1973–76	12	Normal
L. K.	45	1965–67	15	Normal
B. L.	31	1975–77	21	Normal
B. S.	38	1965–77	41	Normal

combination with natural sunlight by one of the authors (E. H.). Sunglasses were not recommended during the therapy, which was given mainly in the summer-time. We have managed to trace 13 of these patients and had them examined by ophthalmologists in 1977–78. Determination of visual acuity, slit lamp examination and ophthalmoscopy in mydriasis were performed. The patient data are shown in Table I.

RESULTS

The patients had received between 4 000 and 41 000 mg of 8-MOP (Table I). Ophthalmologic investigation 2 to 10 years after cessation of treatment did not reveal any pathologic features in cornea, anterior eye chamber, or lens. No opacities in the media were found except such of normal occurrence. The fundus examination was quite normal in all patients. No subjective symptoms related to the treatment were registered.

DISCUSSION

The amounts of 8-MOP administered to the patients are equivalent to 100 to 1 000 doses of 40 mg, a standard single dose of 8-MOP in PUVA treatment. Despite the fact that no warnings against sunlight were given to the patients, no subjective or objective signs of eye damage were observed.

This finding cannot be expected to have general validity. In the first place the photoenergy of the sunlight at the latitude in question (about 60–65°) is not of the same magnitude as in areas closer to the Equator. Secondly, the number of patients examined is small and the peak serum level lower than in the PUVA situation. However, we think that the results may justify a less rigorous sunglass regimen than that currently recommended by certain authors (5).

In this context a study of the uptake of 8-MOP in the retina may have relevance. 8-MOP was found in the retina of rats 1–4 hours after administration. The kinetics of this uptake was not studied and so nothing is known of its persistance (7). The risk of damage to the retina must be substantially smaller than the risk for the lens, as most of the UVA is absorbed in the cornea, lens, aqueous humor and vitreous humor, less than 4% reaching the retina.

Corresponding investigations among psoralen-treated vitiligo patients in other parts of the world ought to elicit valuable information on the real risk of eye damage run by the patients treated with PUVA.

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