Chloroquine-induced Vitiligo

A Case Report and Review of the Literature

Sir.

Hyperpigmentation of the skin is reported in 10–25% of patients receiving antimalarials for more than 4 months (1). Hypopigmentation of hair and freckles is another pigmentation disturbance which has been observed (2). Vitiligo-like hypopigmentation of the skin has been described in 4 Africans with

Fig. 1. Depigmentation on the face, especially in the periorbital area. Note the darker areas on the glabella, indicating repigmentation only a few weeks after discontinuation of the chloroquine therapy.

dark skin. We here present a patient who developed hypopigmentation on the face when taking chloroquine for malariaprophylaxis.

CASE REPORT

A 6-year-old girl of Ethiopian origin, born in Norway, was presented at our department in the spring of 1994 because of depigmented



Fig. 2. Almost complete repigmentation 18 months after the first clinical examination.

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patches in the face. Since the age of 2 years she had been suffering from atopic eczema. She was otherwise healthy. There was especially no previous history of vitiligo, thyreopathy or other autoimmune diseases in her nor in her nearest family. The depigmentation had started when visiting Eritrea. As a prophylactic against malaria, she had been given 250 mg chloroquine weekly. After 3 weeks of therapy the parents noticed the first skin changes in their daughter. The girl experienced no subjective complaints. There were no signs of visual disturbances; she played as usual and got a lot of sun and fresh air. In all she received ten tablets of chloroquine before the family returned to Norway. Here the parents discontinued the medication. After an initial attempt with antimycotic ointment, she was seen in our department showing the classical picture of vitiligo (Fig. 1). Clinical controls at 1, 3, 6 and 18 months (Fig. 2) showed almost complete repigmentation of the skin. The repigmentation did not seem to start perifollicularly; instead patchy repigmentation was seen.

DISCUSSION

Hypopigmentation of the skin during chloroquine therapy has been reported in 4 patients only (3–5). All of the patients were Africans with dark skin, who had been exposed to the African sun. After discontinuation of the drug therapy, the skin rapidly repigmented. In 2 of the patients, the retinopathy which developed during the malaria-prophylaxis did not improve (3). One patient had a reduced minimal erythema dose in the UVB range during the chloroquine therapy, and the photopatch testing showed positive reactions to chloroquine and UVA (4). The 4th patient received chloroquine for rheumatoid arthritis. He also experienced repigmentation after cessation of therapy, but he received ascorbic acid 100 mg daily as placebo (5). Interestingly, factors which influence the binding of chloroquine to melanin are those typical of cation exchange. It has been shown that the free radical content of

melanin is reduced by pretreatment with ascorbic acid (6), which could explain the clinical improvement in this patient. Abnormal reactions to sunlight have previously been observed in patients on chloroquine therapy.

In conclusion, the skin depigmentation in our patient resolved almost completely upon discontinuation of the chloroquine therapy. Although, like the other 4 patients described, she was exposed to intense sunlight, one may only speculate whether the underlying mechanism for the depigmentation is a photosensitivity reaction.

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Accepted September 18, 1995.

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