

pearance of multinucleate cells (4, 10) but we did not see these effects in human skin fibroblasts (8). Many actions of anti-inflammatory drugs are mediated through inhibition of prostaglandin synthesis or availability and griseofulvin can block the effects of prostaglandin E_2 (9). Dose-response curves for prostaglandins tend to follow a characteristic bell-shape with opposite effects at high and low concentrations (5). Part of such a curve might be apparent in Fig. 1 if prostaglandin was responsible for the effects on cell proliferation. In fact only one cell strain showed stimulated proliferation at low griseofulvin concentration. Other drugs with anti-inflammatory properties have given similarly inconsistent low-dose stimulation in our previous work (7), so the evidence for prostaglandin involvement is not convincing.

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Exanthema fixum Due to Ultraviolet Radiation

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Abstract. A case of exanthema fixum caused by sun exposure and reproducible by UV-A and UV-B exposure tests is described.

Key words. Exanthema fixum; Photosensitivity; Ultraviolet radiation

The concept of exanthema fixum was introduced by Brocq, who in 1894 described a rash consisting of sharply demarcated round or ovoid edematous plaque caused by phenazone (1). The number of drugs known to be capable of producing this characteristic lesion is now large and still increasing (2, 5, 6). Various foods as possible causes have also been mentioned (3). However, as far as is known, there is no report among existing literature of fixed eruptions having been caused by electromagnetic waves.

CASE REPORT

A 47-year-old man was referred to our clinic with a rash which had occurred after sun exposure of 30 min at about 60°N latitude (Oslo). The man was not taking medicaments of any kind. He had experienced an identical eruption each time he had stayed in the sun for a certain time over the last four summers. The lesion appeared during or after the exposure, itched and disappeared in a couple of weeks, leaving slightly pigmented areas. There were partly detached, partly confluent ovoid plaques 2-6 cm in diameter with erythema, pigmentation and a sharp demarcation from normal skin on the patient's legs (Fig. 1). Histological examination showed superficial and deep subacute inflammation, not specific for, but consistent with the diagnosis of exanthema fixum (4).

MATERIALS AND METHODS

After the rash had disappeared, light tests were performed. For the UV-B test a Xenon Arc Lamp with a Schott WG 295 cut-off filter was used. This gives a continuous spectrum of electromagnetic waves above 295 nm. For the UV-A test a Waldmann UV 800 with Sylvania



Fig. 1. The patient after sun exposure.

F 20 WIPUVA tubes which gives a clean UV-A spectrum with maximum at 365 nm was used.

UV-B test. First the MED (24 h) was determined on the healthy skin of the patient's back. Then 50% of this energy amount was given where one of the characteristic lesions was known to appear. Previously uninvolved skin in the same area was also exposed to the same dose. The patient was seen and a biopsy taken after 24 h.

UV-A test. The patient's left lower leg was exposed to 12.5 J/cm². (The chosen dose is less than half of that which usually gives UV-A erythema in non-sensitive persons.) The patient was seen after 24 h.

Photopatch test according to the Nordic standard was performed.

RESULTS

The UV-B test produced a distinct reaction with the appearance of a red, pigmented plaque. Histological examination showed deep perivascular and superficial inflammation consistent with the diagnosis of a fixed eruption (4). There was no reaction at the test spot in the normal skin.



Fig. 2. The patient after UVA exposure of his left lower leg. Notice that the exanthema is localized at exactly the same areas as in Fig. 1, but restricted to the left lower leg.

The UV-A test produced the characteristic lesion in the exposed area (Fig. 2) with a distribution identical with that seen after sun exposure (Fig. 1). There was no skin reaction between the plaques.

The photopatch test was negative.

DISCUSSION

There are numerous reports on exanthema fixum medicamentosum. Reactivation without the suspected medicament has also been described, but no reports on photosensitivity seem to have appeared. However, Welsh supposed that photoactivation may be of significance (7). Our patient has a fixed exanthema which is activated by small doses of sunlight and by UV-A and UV-B doses of below MED in tests.

The fact that these low doses are sufficient to elicit the particular response, may speak in favour of a photoallergic reaction. On the other hand, in

the case of photoallergy, one would rather have expected a narrower action spectrum. And the photopatch test does not confirm the supposition of photoallergy. It should be mentioned that the Xenon testing, in addition to UV-B, also included UV-A radiation, but this was assumed to be negligible.

The conclusion is that the patient has a photosensitivity with unknown mechanism but with clinical signs identical with a fixed drug eruption.

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Propylthio-uracil-induced Cutaneous Vasculitis

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Abstract. Three cases of cutaneous vasculitis, leukocytopenia and arthralgia, presumably caused by the antithyroid drug Propylthio-uracil, are presented. Acute vasculitis of the superficial and deep dermal blood vessels accompanied by vascular thrombus formation were found in biopsy specimens. Direct immunofluorescence studies demonstrated deposits of C₃ or IgM and C₃ in the walls of vessels in affected and unaffected skin, suggesting immune complex deposition. The skin lesions and leukocytopenia rapidly disappeared on discontinuation of

the drug, while the arthralgia continued for weeks or months.

Key words: Propylthio-uracil; Cutaneous vasculitis; Immune complex disease

Propylthio-uracil (PTU) is regularly used in the treatment of hyperthyroidism. Adverse reactions may arise at any time during therapy but are most commonly noted during the first 3 weeks (7). The most serious complication, agranulocytosis, occurs in 0.2-0.3% of patients, while skin rashes occur in 3-5% of adults and up to 18% of children (7). PTU is recognized as a frequent cause of allergic vasculitis and even lupus-like (1) and polyarteritis nodosa-like syndromes (6) have been reported. Purpura and vasculitis due to PTU have been described in several case reports showing clinical features varying from one patient to another. They may arise at any time during treatment, and may vary from a mild purpuric rash to severe vasculitis with multisystemic involvement (8).

We report here 3 cases of vasculitis due to PTU seen within one year.

CASE 1

A 13-year-old girl was admitted suffering from malaise and a rash on the face and the arms. She had been treated with PTU 250 mg a day and levothyroxin (Eltroxin*) 0.1 mg per day for 2 years for thyrotoxicosis. Two weeks before admission to the clinic, she initially noted reddening of the right cheek and one week later, she subsequently developed localized tender erythema of the cheeks, the right ear lobe and left arm. On admission the lesions were bluish red, thickened and painful, especially on the ears. Other lesions developed during the following days, while the original lesions subsided. Systemic symptoms including fatigue and migratory polyarthralgia accompanied the skin symptoms.

PTU and Eltroxin was discontinued. The skin lesions disappeared without ulceration or scarring and the arthralgia subsided within 2 weeks.

The thyrotoxicosis recurred, and treatment was resumed with karbimazol, without side effects.

Laboratory findings

Initially a white blood cell count of 2.0 (normal 3.0-9.0) billions/l, rising after withdrawal of PTU to 6.0 billions/l. ESR and platelet counts were normal, and there were no coagulation defects.

Histopathology

The main changes were found in the reticular dermis where the vessel walls and their surroundings were infiltrated with red blood cells. In addition, perivascular