Photosensitivity Induced by Piroxicam

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A woman with rheumatoid arthritis developed an erythematous–bullous eruption on light-irradiated areas following sun exposure. Treatment with piroxicam, had been initiated 14 days earlier. The clinical picture, the relationship in time to the drug administration and a positive photopatch test gave reason to suspect piroxicam-induced photosensibility. Piroxicam (Felden®) is a new non-steroid anti-inflammatory agent which was registered in Sweden in 1981. Various adverse skin reactions have been described. Thus, there has been one case of Lyell's syndrome with a fatal outcome (7), two case reports of erythema multiforme-like reactions (1, 3) and two reports of patients with erythemato-papulous and also bullous eruptions restricted to light-exposed skin areas (3). *Key words: Drug eruption; Photosensitivity; Piroxicam.* (Received November 30, 1982.)

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CASE REPORT

A 60-year-old woman with a family history of rheumatoid arthritis and diabetes but without an atopic background had suffered from disabling rheumatoid arthritis for the past 14 years. She also had a contact allergy to nickel. There was no history of any previous drug reaction. She had been treated with a low dose of prednisolone (Prednisolon®, ACO), 5 mg/day, and with indomethacin (Indomee®), 100 mg/day, for the last year, and a combination of dextropropoxyphene and paracetamol (Distalgesic®) for the last 2 years. Other medications included oxazepam (Sobril®) and nitrazepam (Mogadon®), also administered for the last 2 years. In April 1982, therapy with flupenthixol (Fluanxol®), 1 mg/day was initiated. On May 20th, treatment with piroxicam (Felden®), 20 mg/day, was started. Fourteen days later, following intensive exposure to the sun, a generalized skin reaction developed.

Physical examination revealed an erythematous eruption limited strictly to light-exposed skin areas. Bullous lesions were observed on the forearms. Apart from joint deformities due to the rheumatoid arthritis, there were no other physical abnormalities.

Routine laboratory tests on the blood and urine, including blood sugar, serum creatinine, urea nitrogen, WR and liver function tests, revealed a sideropenia, but were otherwise normal. Signs of inflammatory activity were observed at serum protein electrophoresis and there were also signs of consumption of serum complement factors (C3 and C4). There were no antinuclear or anti-DNA antibodies and no intercellular or antibasement membrane antibodies to skin. Tests for circulating immune complexes and cryoglobulins proved negative. Determination of porphyrine in erythrocytes and urine gave normal results. There were no serological signs of bacterial or virus infections. Urine samples and swabs from the throat were sterile on culture. The rheumatoid factor titre was 1/400.

Biopsy specimens from involved skin and from a positive photopatch test area showed a dermal inflammatory cellular infiltrate, composed mainly of lymphocytes. In one specimen a subepidermal bulla was observed. No deposition of immunoglobulins or complement factors was detected.

Piroxicam, flupenthixol and nitrazepam were all discontinued and the dose of prednisolone was

increased to 30 mg/day with a gradual reduction to 5 mg for a period of 2 weeks. Clinical improvement was observed and the skin eruption disappeared within 2 weeks.

A photopatch test was performed according to the Scandinavian standard procedure (4) which has been used since 1980. The photopatch tray contained 23 substances, excluding compositae mix from the standard tray but including thio-urea and the drugs piroxicam, flupenthixol, oxazepam and nitrazepam. The other agents tested were substances with antimicrobial actitivy, different therepeutic agents, e.g. promethazine, sun-screening agents and substances from plants, different varities of wood and lichen, and substances from the cosmetic industry. A positive reaction with erythema, oedema and papules was observed only for piroxicam (a capsule of Felden®, 20 mg, was suspended in 3 ml of aqueous solution), in the uncovered test series 24 and 48 hours after exposure to 5 J/cm^2 of UVA. No reaction was seen on non-irradiated control sites. The phototest evaluation of UVB sensitivity gave normal results and there was no skin reaction to 5 J/cm^2 of UVA itself. The patient's skin was of type III (5). Her IDP was 5 min. There was no reaction to piroxicam in 5 control patients tested with the photopatch technique.

DISCUSSION

Photosensitivity in association with piroxicam has recently been observed by other authors (2, 3). The present case further illustrates the possibility that piroxicam may be a photosensitizing agent. The diagnosis of a piroxicam-induced photosensitivity reaction was based on the association between skin involvement following sun exposure and the administration of this agent, the clinical picture with skin lesions only on light-exposed skin areas, and the positive photopatch test.

The other drugs administered were not believed to be incriminated. There was no association between the therapeutic introduction of these drugs and development of the clinical eruption, moreover the two drugs that were discontinued in addition to piroxicam, namely flupenthixol and nitrazepam, caused no immediate or late complications on subsequent reinstitution.

The pathogenetic mechanism is obscure. The histological reports and the positive photopatch test may indicate involvement of a photoallergic reaction, but histological and photopatch test findings are not conclusive evidence of a photoallergy (6).

In conclusion, the present case supports previous observations indicating that piroxicam may be a photosensitizing agent. The clinical significance remains to be evaluated.

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