Case report

Acute psoriatic arthropathy precipitated by oxprenolol

D. G. Macfarlane and L. Settas

From the University Department of Medicine, Bristol Royal Infirmary, Marlborough Street, Bristol

SUMMARY A previously normal 58-year-old woman developed a widespread psoriatic rash and asymmetrical peripheral polyarthritis a week after beginning treatment with oxprenolol for hypertension. Skin and joint disease resolved simultaneously after drug withdrawal and have not recurred.

In 1976 a 58-year-old woman was found by her family doctor to have raised blood pressure, and she began treatment with 5 mg of bendrofluazide and 250 mg of methyldopa daily. In July 1981 the blood pressure was still high, so oxprenolol 40 mg daily was added. One week later she noticed that the entire left ring finger was swollen, stiff, and moderately painful. Suspecting infection, her doctor prescribed cotrimoxazole. She took 960 mg (2 tablets) on the first day and 480 mg on the second. Shortly after taking the third tablet she noticed a nonirritant erythematous rash on the backs of the hands and, thinking the antibiotic responsible, took no more. However, the rash became more extensive over the next 2 weeks, spreading to the scalp, behind the ears, distal arms, and legs. During these 2 weeks she began to feel generally unwell, and the right knee became painful and swollen. She was prescribed naproxen 500 mg twice a day, but within a few days the left knee, wrists, and interphalangeal joints of the hands were also painfull inflamed. She was admitted urgently to hospital. There was no past or family history of arthritis or psoriasis. On examination (Fig. 1) she had discrete erythematous papules covered with silvery scales on the arms, legs, round the elbows, under the breasts, behind the ears, in the pubic area, and on the scalp. Several nails on the hands and feet showed marked onycholysis. There was asymmetrical swelling of the interphalangeal joints of the hands, and the area overlying the involved joints was coloured dusky-red. The wrists were tender and swollen, with limitation of movement, and small effusions were present in both knees.

Investigations: haemoglobin 10.4 g/dl; white cell count 6.9 x 10⁹/l; 83% neutrophils, 13% lymphocytes; 3% monocytes; 1% eosinophils; platelets 573 x 10⁹/l; plasma viscosity 2.1 cp; blood film showed

Accepted for publication 17 February 1983.
Correspondence to D. G. Macfarlane, Lewisham Hospital, Lewisham High Street, London SE13 6LH.

Fig. 1 Hands showing rash and joint swelling.
Acute psoriatic arthropathy precipitated by oxprenolol

Microcytic, hypochromic red cells. Serum iron was 3.3 μmol/l and total iron binding capacity 46 μmol/l. Urea, electrolytes, and liver enzymes were normal; albumin 31 g/l; globulin 29 g/l. Tests for rheumatoid factor and antinuclear factor were negative. Biopsy of a skin lesion showed parakeratosis and elongation of the rete pegs with clubbing of their lower parts. The dermis was oedematous and contained a moderate chronic inflammatory cellular infiltrate. Radiology of the hands (Fig. 2) showed soft tissue swelling of the affected joints and sclerosis of the terminal phalanges. The knees, feet, and chest films were normal. The tissue type was: A3, W24, B18, W60.

The oxprenolol was stopped one week after admission and the bendrofluazide and methyldopa continued. The skin lesions were treated with daily polytar baths and shampoos, daily ultraviolet light, twice daily dithranol 0.2% to all lesions, and 1% hydrocortisone cream to the ears and hairline. The arthropathy was treated with bed rest, splints and naproxen 500 mg t.d.s., benorylate 1.5 g t.d.s. and slow-release indomethacin 75 mg at night. The skin and joints responded to this regimen, and after 7 weeks' inpatient treatment she was fully mobile and discharged home. When seen as an outpatient one month later she was free of joint symptoms, but there was residual 'rain drop' pigmentation of the skin (Fig. 3). One year later she was completely well, and there has been no recurrence of rash or arthritis.

Discussion

The acute arthritis had features of psoriatic arthropathy. The initial manifestation was a dactyilitis or 'sausage-digit' followed by a polyarthritis which was asymmetrically distributed in the hands. The rash,
which developed synchronously, had the clinical and histological features of psoriasis.

The evidence that oxprenolol was the precipitating agent is circumstantial but strong. It was the only new drug the patient had taken prior to the onset of symptoms. The cotrimoxazole was prescribed to treat the dactylitis and she took only 3 tablets. She had been on her other medication for several years without side effects and had no antecedent history of streptococcal infection or emotional stress, factors known to precipitate or exacerbate psoriasis. The skin and joint lesions resolved simultaneously and in association with oxprenolol withdrawal, and there has been no recurrence.

Practolol, oxprenolol, and other betablockers have been reported to cause psoriasiform eruptions, but to our knowledge this is the first report of synchronous onset of arthritis and psoriasis caused by oxprenolol. Conclusive proof would have necessitated challenging the patient with the drug, but in view of the rapidity of onset and severity of the illness this was considered to be unjustifiable.

The possible link between a drug and psoriatic arthropathy is of considerable theoretical as well as practical interest, since it suggests that disturbance of a single mechanism, possibly a cellular enzyme, can trigger both the joint and skin disease in psoriasis.

We thank Dr P. A. Dieppe and Dr R. M. Harman for allowing us to report this case.

References