REITER'S DISEASE, RESPONDING TO METHOTREXATE

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SYNOPSIS

A case of Reiter's disease with particularly severe keratoderma blenorrhagica is described. This report details the favourable response to methotrexate therapy.

INTRODUCTION

Reiter's disease is not a benign condition. Permanent disability can result. It is a syndrome of unknown cause, the two most constant features of which are a nonsuppurative usually polyarticular arthritis and an inflammatory process of the urogenital tract, manifesting as nonspecific urethritis, or the lower intestine. Other features include conjunctivitis, iritis, circinate balanitis and buccal erosions. Keratoderma occurs in about 8 per cent of venereal cases (Hancock, 1965) and may be very widespread (Montgomery et al, 1959). Early attacks are usually self limiting but there is a liability to recurrences and later chronicity which may result in painful deformities of the feet, sacro-iliitis, atypical spondylitis, multiple attacks of anterior uveitis and aortic incompetence.

No specific therapy exists for Reiter's disease. Urethritis is usually treated with a course of tetracycline though the aetiological agent remains obscure. Drug treatment for arthritis consists of phenylbutazone or indomethacin; corticosteroid therapy is indicated only when the disease is progressive and this is usually associated with severe keratoderma and marked weight loss. Farber and his colleagues (1967) reported the successful use of methotrexate in patients with Reiter's disease, particularly those with crippling and incapacitating arthritis. In this report, methotrexate was exhibited primarily to control the keratoderma blenorrhagica which markedly improved.

CASE REPORT

A 34 year old male Indian developed joint pains with fever some 3 months prior to admission while he was in India on

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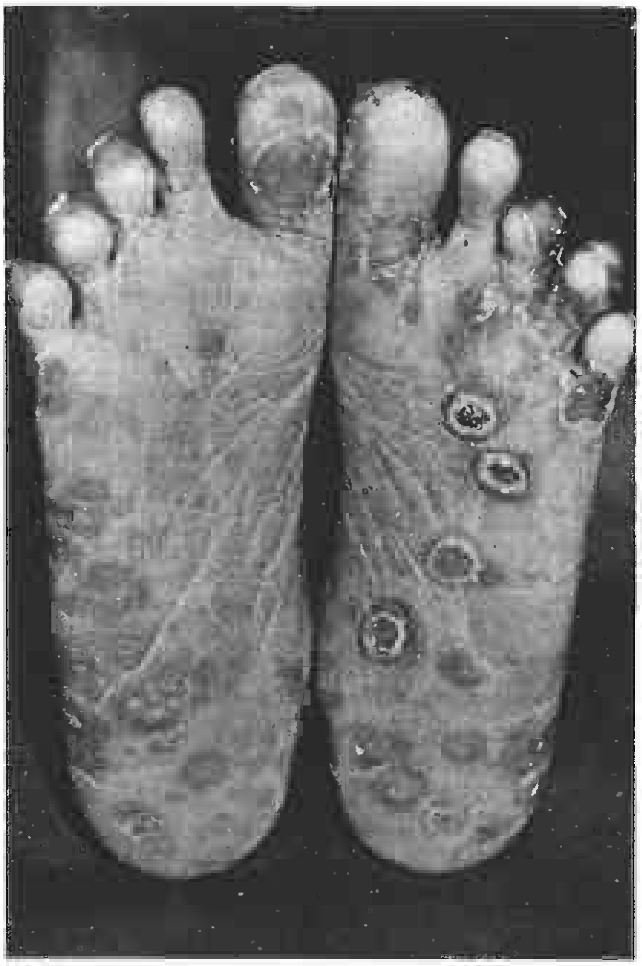

Fig. 1. SOLES—areas of keratoderma.

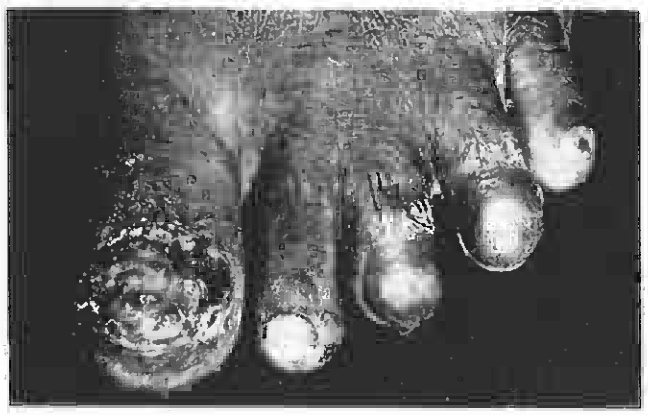

Fig. 2. LEFT FOOT—pustular lesion over big toe, loss of nails over 3rd and 4th toes.

Fig. 3. SCALP—thick crusted lesions.

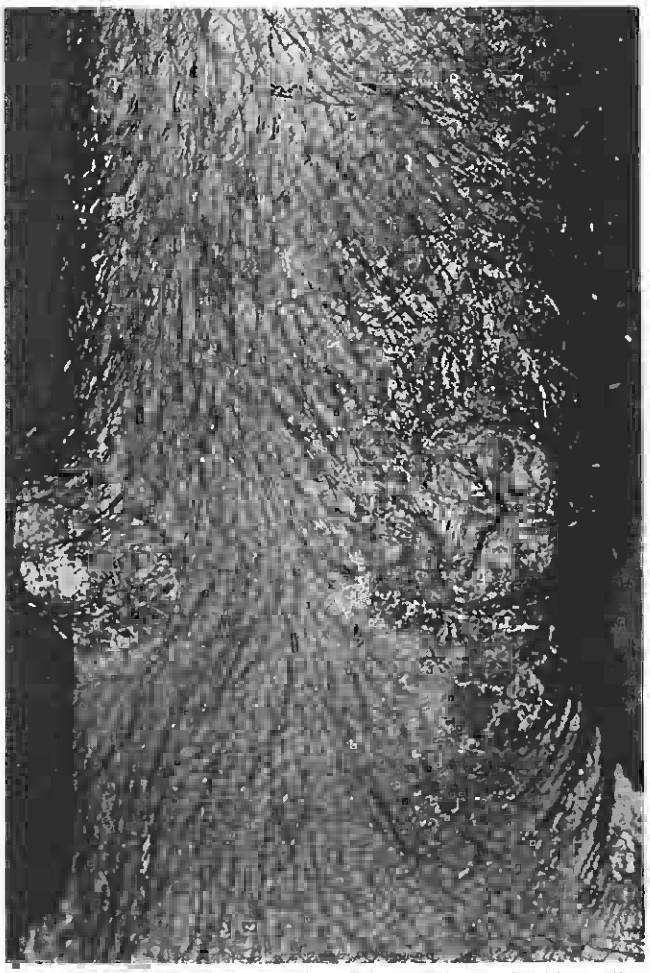

Fig. 4. LEFT WRIST—hyperkeratotic lesions, looking like barnacles.

vacation. In October 1976 he developed urethral discharge with dysuria. He denied any history of venereal exposure. This improved with treatment given by a doctor. Two weeks later the left ankle became painful and swollen. This failed to improve with further medication and 3 weeks later skin lesions started to appear. Associated with this was a low grade fever.

Joint involvement was confined to the left ankle initially but three weeks later, after the first crop of skin lesions, the right ankle was similarly involved. Later as the ankles were improving, the knees were affected. This limited movement and he was unable to walk.

The skin rash began first over both wrists, later spreading to involve the scalp and the trunk. Both soles were also affected (Fig. 1). The lesions progressed through various stages starting as erythematous papules which then became encrusted and hard. There was no itch or bleeding. They

were not painful except in the soles on attempted walking. Toe nails became involved and subsequently dropped off (Fig. 2).

He was febrile on examination. The skin showed erythematous papules on the nose and right cheek. Over the ankles, thighs, wrists, scalp and anterior chest, erythematous lesions with heaped up crusts varying from 0.5 cm to 3 cm diameter were present. Some crusts had fallen off to reveal a dry reddish base. The whole scalp was hard with a thick layer of crust (Fig. 3). The palms and soles showed keratotic lesions. A few toe nails had dropped off. Erythematous patches with an irregular edge were seen around the penile meatus. Both knee joints were swollen with effusion. The other systems including the eyes, heart, lungs and abdomen were normal.

Laboratory tests revealed a haemoglobin of 13.9 g/dl, a leucocyte count of 21,200/mm^3 with 90 per cent polymorphs. The erythrocyte sedimentation rate was 128 mm in the first hour. Urinalysis was normal. The GCFT, VDRL were negative. So were the RA and antinuclear factors. Liver function tests were normal. The X-rays of the chest, hands, sacroiliac joints, knees, feet and the calcaneum were normal. Blood uric acid was 3.9 mg/dl.

He was given a two week course of tetracycline and started on prednisolone 10 mg t. d. s. The fever gradually settled and the arthritis responded. He appeared generally better but after 2 weeks on steroids a new crop of skin lesions erupted and his arthritis relapsed. It was decided to give him methotraxate in weekly doses starting with 10 mg orally. This was increased to 15 mg orally for the next 4 weeks and then 20 mg intravenously weekly. The prednisolone was slowly tailed off. The skin lesions stopped erupting and those already erupted progressed to hyperkeratotic, raised plaques before falling off to leave intact clear skin. Those on the scalp showed similar response.

On discharge after 8 doses of methotrexate all his skin lesions were quiescent and he was able to fully weight bear on his soles. Only a few large hyperkeratotic lesions remained. The scalp hair had grown about 1 cm lifting the crusts with it to reveal normal skin beneath. All the arthritis had resolved with no sequelae.

He has remained well on follow up.

DISCUSSION

This patient was at first thought to have septic arthritis of the knee joints. Together with the early skin lesions psoriatic arthropathy could not be excluded. In view of the history of urethritis preceding these lesions Reiter's disease was thought likely. As the condition evolved and the characteristic skin lesions of keratoderma blenorrhagica developed, this diagnosis was clinched.

The severity of the skin lesions is striking in this case, being both very extensive as well as very hyperkeratotic, the keratoderma looking like barnacles clinging onto the skin (Fig. 4). However these lesions are superficial, not involving the dermis and so leave no scars when they resolve.

Some authors have suggested that Reiter's disease may be a variant of psoriasis. This similarity is well borne out by this case. Methotrexate was started when new crops of skin lesions appeared despite corticosteroid therapy. There is little doubt that methotrexate is responsible for his remission.

Chu (1976) reported a case where oral methotrexate in 25 mg weekly doses was used and by the 22nd week all signs of Reiter's disease disappeared. Podurgiel et al (1973) feel that the drug may be given safely in weekly dosage for up to 4 years without causing liver fibrosis and cirrhosis.

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