REITER’S DISEASE IN TWO BROTHERS*

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Reiter’s disease affecting more than one member of a family has been reported only rarely. Paronen (1948) described three families in whom several members, including women and children, developed Reiter’s disease following dysentery. All other reported cases of familial aggregation of Reiter’s disease have concerned males alone and have never occurred as a sequel to dysentery. Trier (1950) and Conska (1958) both reported the condition occurring in two brothers, Morton (1958) reported Reiter’s disease in first cousins, and Gough (1962) reported the disease in a father and son.

We report a further occurrence of Reiter’s disease in two brothers; they lived 400 miles apart and a common contact was excluded. There was no family history of other joint disease or of psoriasis.

Case Reports

Case 1. A 29-year-old married man, with no previous history of articular disease, was admitted to hospital in June, 1965, with a 6-week history of urethral discharge. There had been pain and swelling in the toes of the right foot for 2 weeks and in the right ankle and left knee joint for 3 days. He denied any extra-marital exposure and had had no diarrhoea.

Examination.—He had urethritis, circinate balanitis, and mild bilateral conjunctivitis. The left knee and right ankle joints were swollen and tender with a limited range of movement. The right wrist, right sacro-iliac, and left temporo-mandibular joints were also involved. Keratoderma blennorrhagica later developed on the soles of the feet (Fig. 1).

Haemoglobin concentration 15·2 g./100 ml.
Erythrocyte sedimentation rate (ESR) was 40 mm. in one hour and subsequently rose to 86 mm. in one hour.
Total leucocyte count 11,000/cu. mm.; normal differential count.
Wassermann reaction, gonococcal complement-fixation test, and latex particle test for rheumatoid factor all negative.

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Cultures of mid-stream urine and urethral discharge sterile.
Radiographs of all involved joints and heels initially normal.

By September, 1965, a number of erosions had developed at the bases of the proximal phalanges of both feet.

Treatment.—He was given demethylchlortetracycline in a dose of 1,200 mg./day for 10 days and phenylbutazone in an initial dose of 400 mg./day.

Result.—The ESR gradually fell, and after 4 months it was 1 mm. in one hour and the patient was symptom-free. There was no recurrence of symptoms during the following 12 months.
Case 2. A 21-year-old unmarried man, with no previous history of articular disease, was admitted to hospital in September, 1967, with a 2-week history of pain and swelling of the left great toe, left knee, right ankle, right wrist, and the first and second metacarpophalangeal joints of the right hand. He had also had a greenish urethral discharge occasionally associated with dysuria for the same length of time. He had no diarrhoea or conjunctivitis. He admitted a casual sexual contact some 4 weeks previously.

Examination.—The left knee, right wrist, and first and second metacarpophalangeal joints of the right hand were swollen and tender with a limited range of movement. There was a urethral discharge and circinate balanitis. One week after admission keratoderma blennorrhagica developed on the soles of the feet (Fig. 2).

![Fig. 2.—Soles of the feet in Case 2.](image)

Reiter's Disease in Two Brothers

Haemoglobin concentration 14·7 g./100 ml.
ESR 17 mm. in one hour.
Total leucocyte count 11,900/cu. mm.; normal differential count.
Serological tests for gonorrhoea and syphilis negative.
Sensitized sheep cell test repeatedly negative.
Serum uric acid 3·4 mg./100 ml.
Cultures of mid-stream urine, urethral discharge, and prostatic fluid sterile.
Radiographs of feet, ankles, and knees showed some rarefaction, but no erosive changes. Those of the hands, wrists, and sacro-iliac joints were normal.

Treatment.—He was given aspirin (3-4 g./day) for 7 weeks, but the joint symptoms and signs were unaltered, and he continued to have a urethral discharge, circinate balanitis, and keratoderma blennorrhagica. During this time the ESR remained raised and rose as high as 46 mm. in one hour. In view of the report of the successful treatment of a case of Reiter's disease with lincomycin hydrochloride (Mowat, Chalmers, Alexander, and Duthie, 1967), the drug was given in a dose of 4 g./day for 2 weeks.

Result.—On the completion of this course the patient was symptom-free with an ESR of 4 mm. in one hour, and the only abnormality on examination was the residual keratoderma blennorrhagica which persisted for a further week. There has been no recurrence of symptoms.

Discussion

Weinberger and Bauer (1955) thought that the diagnosis of Reiter's disease should be restricted to cases in which the triad of non-specific urethritis, conjunctivitis, and acute polyarthritis described by Reiter (1916) was present. However, Montgomery, Poske, Barton, Foxworthy, and Baker (1959) stated that keratoderma blennorrhagica was an equally important diagnostic feature.

Keratoderma blennorrhagica involves chiefly the soles, palms, and nails. Circinate balanitis is essentially the same type of lesion and differs clinically by virtue of the effects of the prepuce. Similar lesions are also frequently found on the hard palate. The incidence of keratoderma blennorrhagica in Reiter's disease has been reported as 8 per cent. by Hancock (1960), 14 per cent. by Csonka (1958), and 33 per cent. by Popert, Gill, and Laird (1964). All these authors report that circinate balanitis occurs rather more frequently.

Reiter's disease, as it occurs in the United Kingdom and North America, is usually associated with sexually-transmitted non-specific urethritis (Brit. med. J., 1966), but the disease arises in only a small number of those who contract this infection; Morton (1958) suggested an incidence of 3 to 4 per cent., but Laird (1958) and Csonka (1958) reported 1·7 and 0·8 per cent. respectively. The Ministry of Health (1967) reported that 327 (1·07 per cent.) of 30,464 male cases of non-specific urethritis were complicated by arthritis in 1966.

Various authors have attempted to explain why some patients with non-specific urethritis should develop the other features of Reiter's disease. Laird (1958) postulated that the disease might arise from sensitization of the tissues following infection in the genito-urinary or alimentary tracts. Csonka (1958) suggested that there might be an hereditary predisposition to the articular manifestations of Reiter's disease on the basis of the increased incidence of rheumatoid arthritis in the relatives of
patients with Reiter’s disease. Lawrence (1965) has given preliminary results which suggest an increased incidence of psoriasis and ankylosing spondylitis in relatives of patients with Reiter’s disease; he has postulated a possible genetic basis for the disease.

Wright and Reed (1964) suggested that there was a link between Reiter’s disease and psoriatic arthropathy. This was supported by Maxwell, Greig, Boyle, Pasienczny, and Schofield (1966). Both these groups drew attention to the similarity between the lesions of keratoderma blennorrhagica and pustular and rupioid psoriasis depending on whether the lesions are on the soles and palms or body respectively. Psoriasis is a condition with a strong familial aggregation and a small number of individuals develop the distinctive arthropathy.

It is possible, therefore, that there is some genetic basis to Reiter’s disease, particularly in those who develop keratoderma blennorrhagica. The present report adds support to this concept.

It is also possible that some common genetic factor governs the occurrence of joint disease both in individuals with non-specific urethritis and in those with psoriasis.

Summary

The occurrence of Reiter’s disease in two brothers is reported. A possible genetic basis for the disease is discussed.

The response of one patient (Case 2) to lincomycin hydrochloride is described.

We wish to thank Prof. J. J. R. Duthie for permission to publish the details of Case 2.

REFERENCES


La maladie de Reiter chez deux frères

Résumé

L’apparition de la maladie de Reiter est rapportée. La possibilité d’une base génétique de cette maladie est discutée. La réaction d’un des deux malades (le cas No. 2) à la lincomycine chlorhydrique est décrite.