REITER'S DISEASE - CLINICAL PROFILE OF EPIDEMIC FORM

H. S. GIRGLA * S. K. BHATTACHARYA † M. L. MEHRO RA ‡
G. M. SINGH ‖ P. N. SOMANI § A. SHARMA ¶

Summary

Absence of urethritis need not exclude the possibility of Reiter's disease in young males where conjunctivitis and polyarthritis are cardinal features. Appearance of cutaneous lesions early in the course of the disease heralds a poor prognosis specially in the rare epidemic form of the disease.

2 cases of Reiter's disease are reported. Both belonged to the dysentric type of the disease; sometimes referred to as the epidemic form. Relatively high dose of steroids was necessary to control symptoms.

Reiter's disease is characterised by the triad of non-gonococcal urethritis, conjunctivitis, and a subacute or chronic polyarthritis. It is frequently associated with characteristic muco-cutaneous lesions. The epidemiology of the condition is sometimes suggestive of a contagious disease. In many cases the disease follows venereal exposure, while in some it has been reported in striking association with shigella dysentery[1,2]. In some others no antecedent sexual exposure or evidence of enteric infection have been demonstrated. Several early workers considered a Mycoplasma organism as a possible causative agent[3]. More recently a Bedsoniae strain has been implicated by Schachter and associates[4]. Coryne bacteriae[5] and chlamydiae[6,7] were also one time suspects. Of late the findings of raised levels of betaglobulins[8] and the presence of HL-A antigen B-27[9] in a majority of these cases have lead to speculation of a possible role of auto-immune mechanism in the causation of this disease.

Though the manifestations of the disease may vary from case to case, the diagnosis usually presents no problem. The following two cases illustrate this.

Case 1

A 17 year old unmarried Hindu male was hospitalised in August 1974 when he presented with erythematous maculo-papular lesions over hands and feet of 10 days[1] duration. Earlier he had been seen elsewhere because of episodes of fever, painful swollen joints and watering from eyes, which had been occurring for 6 years. Fever used to be high grade and was each time preceded by a bout of dysentery. Skin lesions were absent during the earlier episodes.

* Reader in Dermatology and Venereology
† Reader in Medicine
‡ Lecturer in Pathology, M. L. N. Medical College, Allahabad
‖ Professor of Dermatology and Venereology
§ Professor of Medicine
¶ Senior Resident in Medicine
Institute of Medical Sciences
Banaras Hindu University
Varanasi-221005
Received for publication on 29—5—1979

Patient denied history of urethritis or sexual contact.

At the time of hospitalisation, the patient was restless and febrile. Tender lymph nodes were palpable in the axillae and groin. Conjunctivae showed marked congestion with purulent discharge. Interphalangeal, wrist, knee and elbow joints were swollen, and tender, their movements restricted. There was proximal muscular wasting. Skin lesions were present all over involving also dorsa of hands and feet. These were mostly discrete, dry and painless papules of 2 to 4 mm size. There were few pustules and few hyperkeratotic lesions. Nail beds were swollen and nails were dry, opaque and on the verge of falling off.

Haematological investigations did not reveal any abnormality. Stool and urinalysis were normal. Prostatic fluid obtained after massage revealed plenty of epithelial cells and few pus cells. The fluid was sterile on culture. Latex fixation test, blood VDRL test and LE cell tests were all negative. Sigmoidoscopy revealed no abnormality. X-ray of knee and ankle joints revealed increase in joint spaces and bilateral calcaneal spur. Biopsy from a relatively fresh skin lesion revealed marked degree of hyper-keratosis and parakeratosis. Stratum corneum showed collections of neutrophils. Spongiform pustules were seen in the upper Malphigian layer. Superficial dermis showed large number of dilated capillaries containing and surrounded by neutrophils.

Patient was initially put on oral phenylbutazone (300 mg) and tetracycline (1g) in divided doses along with other supportive measures. These failed to produce any change in the condition even after two weeks. With the addition of prednisolone (60 mg per day) orally, patient started to show improvement. He was discharged after 4 weeks with advise to continue prednisolone (30mg per day) for two weeks. During subsequent follow-ups attempts to reduce the dose of prednisolone further was met with reappearance of skin lesions.

Case 2

A 17 year old unmarried male student was admitted to hospital on 24th June, 1975 with high fever associated with multiple painful swollen joints of 2 week's duration. His illness started with a two days' history of blood stained loose stools four to six times a day. Fever persisted and was soon associated with burning pain and discharge from eyes. After another 24 hours pain and swelling appeared in left knee joint which was soon followed by similar involvement of all the other large joints of the body. Subsequent to this the patient developed skin lesions which appeared in crops first over the back and then on the abdominal wall, axillae and glans penis (Fig. 1). Skin lesions were asymptomatic. Patient denied any history of

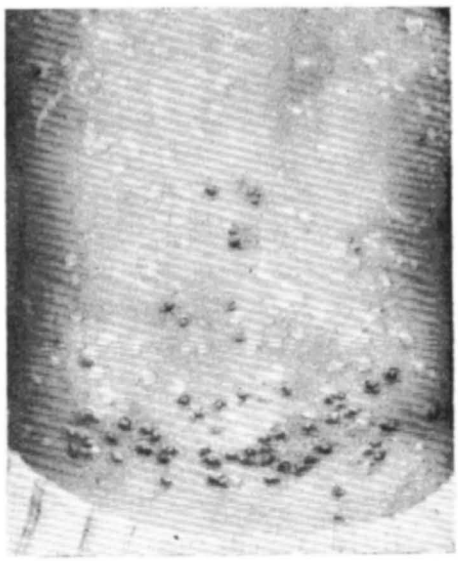
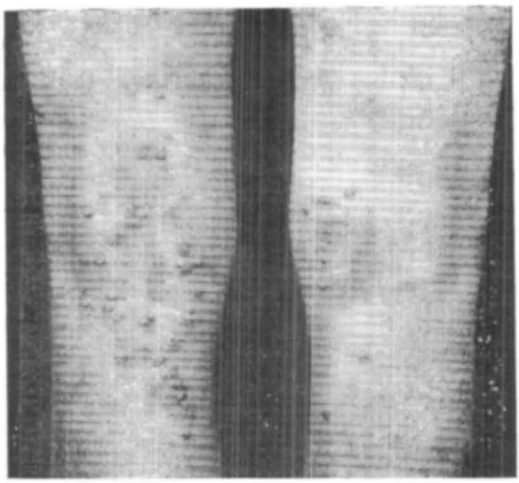


Fig. Mucocutaneous lesions in Reiter's disease (case-2)
(a) Cutaneous lesions over the trunk



(b) Cutaneous lesions over the popleteal fossa

urethral discharge or sexual exposure. Past and family histories were non contributory.

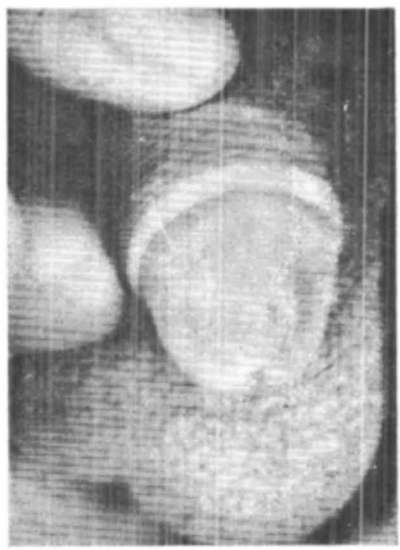
General physical examination revealed a young patient in moderate distress. His temperature was 102° F, and pulse rate 120 per minute. All large joints and joints of feet were swollen and tender. There was flexion deformity of interphalangeal joints. Movements at the joints were restricted and painful. Skin lesions were mostly generalised reddish brown, discrete flat-topped papules. Few pustules were present and other lesions were hyperkeratotic. Systemic examination revealed no abnormality.

Investigation reports were as follows : Total leucocyte count 16,000 per cu. mm., polymorphs, 75%, lymphocytes, 21%, eosinophils, 4%, haemoglobin, 12.5 gms. per cent, and erythrocyte sedimentation rate 5mm in first hour. Blood VDRL, latex fixation test and LE cell tests were negative. Blood culture yielded growth of staphylococcus aureus. Serum uric acid was 5.2 mg per cent. Radiological examination of joints and sigmoidoscopy revealed no abnormality. Biopsy of a skin lesion showed features similar to those of case 1. Electrocardiogram was normal.

While in hospital, the patient initially received phenylbutazone 300 mgs per day with tetracycline 1 gm per day in divided doses. The arthralgia and skin lesion showed no signs of regression even after 2 weeks of therapy. Prednisolone was then added. Within two weeks patient became asymptomatic and skin lesions crusted and fell off. However, each time as the dose of prednisolone was reduced skin lesions reappeared usually in the axillae, cubital fossae and behind the knee joints. There was no joint symptoms.

Discussion

Although Hans Reiter[10], a German physician, is credited with the description of this disease which bears his name, the entity was in fact first described by Benzamin Brodie in 1818. The epidemiology of this disease suggests existence of two forms. The classic triad of urethritis, conjunctivitis and polyarthritis in association with mucocutaneous lesions constitutes what is



(c) Mucocutaneous lesions over the glans and prepuce.

known as the endemic form where the portal of entry of the causative organism is believed to be the genitourinary system. The epidemic form is rarely seen[11]. The portal of entry here is the gastrointestinal tract. This is suggested by presence of mild evanescent diarrhoea which may occur 1 to 3 weeks prior to the onset of the triad in about a third of the patients[12]. Diarrhoea did herald the onset of symptoms in both the cases under discussion thus these can be considered as the rare epidemic forms of the disease.

Acute arthritis of Reiter's syndrome occur usually within two weeks after the onset of urethritis. It is usually polyarticular, migratory and characterized by signs of severe inflammation[12]. The distal joints do not present the marked overgrowth of synovia seen in rheumatoid arthritis. Asymmetric involvement of large joints is frequent. Patients occasionally exhibit severe tenderness over certain periosteal surfaces such as the heels, iliac crests and spinous processes. In the cases presented here arthritis involved the large joints which showed signs of acute inflammation and in turn was responsible for disability which was absolute. Interphalangeal joint involvement and deformity occurred only in one case. Periosteal tendernes was singularly absent. The X-ray evidence of unilateral inflammatory sacro-iliac joint disease which has been reported in several series has been correlated with the frequency of recurrences of Reiter's syndrome. Often no symptoms were associated with this finding initially[13-16]. Such an involvement was not noted in either of the cases reported here.

The skin lesions of Reiter's syndrome have attracted much attention in recent years. The muco-cutaneous reaction can be considered a pathognomonic feature of this syndrome at the bed side. The skin lesions are identical to those described as keratosis blennorrhagica and occur in about 30 per cent of the patients.[11] They are most often seen on the weight bearing areas of soles of feet where they first appear as slightly elevated, reddish yellow waxy cones of 4 to 5 mm size. In course of time they enlarge, spread and become confluent[11]. In both the cases reported here painless, non-pruritic hyperkeratotic cutaneous lesions appeared first on the trunk and then the axillae and cubital fossae. Dorsa of hands and feet were involved in only one patient (case-1) where toe and finger nails which had separated from the nail beds and become opaque and denuded. Histopathological changes and spongiotic abscesses like hyperkeratosis, parakeratosis, presence of Munro's abscesses and elongation and fenestration of rete pegs are similar to those seen in pustular psoriasis, with which the disease is often confused[12,17]. At times skin lesions may take the form of erythema nodosum[16].

About 80 per cent of the cases have been reported to show painless, dusk red, slightly elevated lesions of 2 to 10 mm size on the mucous membranes of the palate, alveolar processes and cheek and also on the glans penis and foreskin.[11]. Only the last two sites mentioned were involved in the two cases reported here.

Laboratory aids are of little value in diagnosis. Though both our patients were seronegative, one patient (case 2) yielded staphylococcus aureus in blood culture. This is an unusual feature in Reiter's disease. Cardiac involvement in the course of the disease is known but was not observed in our cases. Although the majority of patients eventually recover completely[18], recurrent episodes are characteristic of the disease in about 75 percent of cases. In both our cases reappearance of skin lesions alone was noted on reduction of prednisolone dosage. This observation does not support the contention of Faxworthy[19], who opined

that steroids are of value in controlling pain but do not affect the course of the disease. Painful heel is a common complaint during the acute phase of the disease. Our second case who had this symptom showed bilateral calcaneal spur on radiological examination.

References

1. Noer HR : An experimental epidemic of Reiter's syndrome. J Am Med Assn, 198 : 693, 1966.
2. Sharp JT : Reiter's Syndrome - a review of current status and hypothesis regarding its pathogenesis. Current Problems in Dermatology (Basel). 5 : 157, 1973.
3. Dienes L, Ropes MW, Smith WE, et al : The role of pleuropneumonia like organisms in genitourinary and joint disease. New Eng J Med, 238 : 509, 1948.
4. Schachter J, Barnes MG, Jones JP, et al : Isolation of bedsoniae from joints of patients with Reiter's syndrome. Proceed Soc Experiment Biol Med, 122 : 283, 1968.
5. Furness G, Kamat MH, Kaminsky Z, et al : Isolation of Coryne bacteria from non-specific urethritis. J Urol (Baltimore). 106 : 557, 1971.
6. Ford DK : Reiter's syndrome. Bull Rheum Dis, 20 : 588, 1970.
7. Dunlop FM, Vaughan - Jackson JD, Darouger S: Chlamydial infection incidence in non-specific urethritis, Brit J Ven Dis, 48 : 424, 1972.
8. Csonka GW, Basset EW, Furness G : Raised levels of an unknown betaglobulin in the serum of patients with non-specific urethritis and Reiter's Disease. Brit J Ven Dis, 50 : 17, 1974.
9. Morris R : HLA B-27-a clue to the diagnosis and pathogenesis of Reiter's syndrome. New Eng J Med, 290 : 554, 1974.
10. Reiter H : Uber ein bisher unerk annte Spirochaet infection (Spirochaetosis arthritica). Deutsehe Medicale Wehnchr. 42 : 1535, 1916.
11. Montogomery MM and Poske RM : Dermal disorders and rheumatic diseases. Med Clin North Am, 49 : 94, 1965.
12. Ferguson RH and Polly HF : Variants of rheumatoid arthritis-Reiter's syndrome. Med Clin North Am, 52 : 504, 1968.
13. Ford DK : Arthritis and venereal urethritis. Brit J Ven Dis 29 : 123, 1953.
14. Oates JK and Young AC : Sacro-ilitis in Reiter's disease. Brit Med J, 1 : 1013, 1959.
15. Reynolds DF and Csonka GW : Radiological aspects of Reiter's syndrome (Venereal arthritis). J Fac Radiol (London). 9 : 44, 1958.
16. McMillan A : Reiter's disease in a female presenting as erythema nodosum. Brit J Ven Dis, 51 : 343, 1975.
17. Kulka JP : The lesions of Reiter's Syndrome. Arthritis and rheumatism. 5 : 195, 1962.
18. Weinberger HW, Ropes MW, Kulka JP, et al : Reiter's syndrome, clinical and pathological observations, Medicine, 41 : 35, 1962.
19. Foxworthy DT, Poske RM, Barton EM, et al : Adreno - corticotropin and cortisone in the treatment of severe Reiter's syndrome. Annals Int Med, 44 : 52, 1956.