Reiter’s Syndrome

EPIDEMIOLOGY

Of NGU cases, 1–3% are complicated by reactive arthritis (Keat et al., 1978; Kousa et al., 1978). Asymmetrical polyarthritis (especially in the lower extremities, sometimes occurring as sacroilitis) and tenosynovitis (in the achilles and other tendons of the feet) (Kousa, 1978; Keat et al., 1983) are the most common manifestations. Arthritis can occur in association with uveitis and urethritis, a triad that has been named Reiter’s syndrome. However, all three manifestations are seldom present simultaneously. Reiter’s syndrome can occur after intestinal infections (enteroarthritis) or after urogenital infections (uroarthritis). Uroarthritis is also called sexually acquired reactive arthritis (SARA) (Keat et al., 1983). Patients with HLA B27 antigen are a special risk group for reactive arthritis.

Sexually acquired reactive arthritis most often affects young, sexually active males. Thus it is more common in males than in females (20 : 1) (Leirisalo et al., 1982). The male preponderance might be partly due to difficulties in diagnosing SARA and Reiter’s syndrome in women. The often asymptomatic character of the genital manifestation in women can result in a high degree of nondiagnosis or misdiagnosis of Reiter’s syndrome among females.

A disease called rheumatic salpingitis has been proposed as a manifestation of reactive arthritis in females (Yli-Kerttula et al., 1984).

CHLAMYDIAL INFECTION

Since chlamydial infection has long been known as a comparatively common cause of polyarthritis in a variety of animals (Storz, 1961), there was early interest in a possible association of chlamydial infection with reactive arthritis in human beings.

The first report of the isolation of C. trachomatis (from the urethra) in a case
of Reiter's syndrome appeared in 1962 (Siboulet and Galestin, 1962). Since then there has been a continuous attempt to establish a firm etiological relationship between this organism and SARA and Reiter's syndrome. However, a great number of partly controversial and/or contradictory data have been presented over the years (cf. Kousa, 1982; cf. Keat, 1983). The precise role of *C. trachomatis* in the pathogenesis of SARA and Reiter's syndrome is still unknown, although a large proportion of patients have recently been or still are infected by the organism.

A Scandinavian study (Bengtsson et al., 1983) of 25 cases of complete and incomplete Reiter's syndrome indicated that infections with *C. trachomatis*, *Yersinia enterocolitica*, and *Campylobacter jejuni*, serotype 3, were associated with the syndrome in 17, 4, and 1 case, respectively.

*Chlamydia trachomatis* has been recovered from the urethra of a large portion of acute Reiter or SARA cases with signs of genital infection (Siboulet and Galestin, 1962; Kousa et al., 1978; Keat et al., 1980; Martin et al., 1984). Culture studies (including isolation on embryonated hen's egg) of synovial fluid and synovial biopsies have given contradictory results. Most chlamydial isolates have been made in eggs and very few in tissue cell cultures (Schachter et al., 1966; Dunlop et al., 1968; Gordon et al., 1973).

The geometric mean titer (GMT) of antichlamydial micro-IF antibodies is higher in SARA than in nongonococcal urethritis (NGU), systemic lupus erythematosus (SLE), rheumatoid arthritis (RA), and ankylosing spondylitis (AS) (Keat et al., 1980). The latter authors also found the GMT to be greater in culture-positive than culture-negative cases. Kousa and colleagues (1978) found 79 (87%) of 91 men with Reiter's syndrome to have micro-IF IgG antibodies to *C. trachomatis* as compared to 24% of controls. The GMT of antichlamydial IgG antibodies was 47.5 (Keat et al., 1980) and 69 (Kousa et al., 1978) in the two study groups, respectively. The GMT was greater in those with a less-than-3-month history of disease than those with disease of longer duration. The GMT was 188 in those with positive chlamydial urethral culture as compared to 15.2 in those with positive culture but no signs of Reiter's syndrome (Keat et al., 1980). In men with ankylosing spondylitis, no humoral antichlamydial antibody response was detected (Keat et al., 1980).

Cellular immunity to *C. trachomatis* has been found in up to 75% of Reiter cases (Schachter and Dawson, 1978). When uncomplicated chlamydial urethritis cases were compared with Reiter's syndrome cases, no difference in cellular immune reactivity was found (Amor et al., 1972).

**CLINICAL FINDINGS**

Synovitis may develop up to 1 month prior to other symptoms. The Reiter patient may also present concomitantly with genitourinary or bowel symptoms.