Ankylosing Spondylitis Associated with *Trichomonas vaginalis* Infection

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A patient is described who developed signs and symptoms of ankylosing spondylitis after prostatitis due to *Trichomonas vaginalis*. Chronic prostatitis of unknown cause has previously been reported as being common in patients with ankylosing spondylitis. The observations in this case raise the possibility that *T. vaginalis* might play a role in the prostatitis and pathogenesis of ankylosing spondylitis in some patients.

Mild, chronic prostatitis appears to be common in patients with ankylosing spondylitis (11–13). The cause of this prostatitis is not known. *Trichomonas vaginalis* is a common cause of infection of the female genital tract, and transmission of this organism is thought to be venereal (3, 16). Men are generally presumed to be asymptomatic carriers of the parasite (2). However, in a small percentage of men *T. vaginalis* appears to be the cause of nongonococcal urethritis or prostatitis or both (9, 10). The patient described in this report is presented to point out a possible association between *T. vaginalis* prostatic infection and ankylosing spondylitis.

**CASE REPORT**

A previously healthy 35-year-old white man first noted a clear, urethral discharge approximately 8 days after sexual contact. The discharge was examined for gram-negative intracellular diplococci and cultured. No organisms consistent with *Neisseria gonorrhoeae* were observed, and standard cultures were negative. After a 10-day course of tetracycline (250 mg four times a day), the patient’s symptoms abated although mild dysuria and watery urethral discharge persisted. A 14-day course of tetracycline (500 mg four times a day) was then given. The dysuria and discharge continued, accompanied by the gradual development of frequency and mild perineal discomfort. Physical examination revealed a boggy, mildly tender prostate. Three-glass urinalysis after prostatic examination revealed the highest number of leukocytes (8 to 10 per high-power field) to be in the first-void urine. Culture of clean-catch urine revealed no growth. A combination of trimethoprim-sulfamethoxazole was then given without resolution of symptoms.

Approximately 2 months after the onset of symptoms, the patient was evaluated for the presence of *trichomonads*. *T. vaginalis* was recovered from the urethral prostatic secretions by culture in Trichosel broth (BBL Microbiology Systems, Cockeysville, Md.). A four-fold increase in antibody titer to a *T. vaginalis* antigen by the indirect hemagglutination inhibition technique was also demonstrated (8). During this evaluation it became known that several weeks previously the regular sex partner of this patient had developed acute trichomoniass for the first time. Both the patient and his partner were treated successfully with metronidazole.

About 10 days after the development of prostatic symptoms, mild morning back stiffness and spasms of the paraspinous muscles began for the first time. The back pain and stiffness became progressively more severe and were accompanied by the development of evanescent pains in the distribution of the sciatic nerve without neurological deficit. Weight loss and migratory periarticular joint pains involving a number of large and small joints also developed. No episodes of acute joint redness or effusion occurred. The symptoms were controlled with indomethacin. The patient was followed for 4 years and continued to have symptoms, primarily related to the axial skeleton, with intermittent pain in and around variable peripheral joints. Although initial sacroiliac films showed no sacroilitis, subsequent pelvic films were consistent with early sacroilitis (sacroiliac joint erosions and sclerosis). The patient is HLA-B27 positive. There have been frequent, brief, and mild episodes of prostatitis symptoms during the follow-up period, but these have become progressively less frequent. Attempts to isolate *T. vaginalis* during some of these episodes were unsuccessful.

**DISCUSSION**

The presence of *T. vaginalis* prostatitis in this patient at the time of developing signs and symptoms of ankylosing spondylitis could have been coincidental. However, there has never been an adequate explanation for the prostatitis that is seen frequently in patients with ankylosing spondylitis (11–13). *T. vaginalis* infection is often not documented in men because the agent is infrequently considered as a cause of prostatitis, the symptoms are often mild and overlooked, and...
the organism is difficult to demonstrate in men. All of these factors are reasons why T. vaginalis might be considered in the etiology of the cryptic prostatitis observed in male ankylosing spondylitis patients.

Ankylosing spondylitis is known to be associated with the histocompatibility antigen B27 (HLA-B27) (14). What appear to be infectious triggers of arthritis are well described for other HLA-B27-related arthropathies such as Reiter’s syndrome and reactive arthritis (4). In addition to causing prostatic signs and symptoms, could T. vaginalis be an infectious trigger of ankylosing spondylitis? The isolation of certain enteric organisms from patients with active ankylosing spondylitis (5) and the possible antigenic similarities between these organisms and HLA-B27 have given rise to speculation that these similarities are somehow related to ankylosing spondylitis, possibly by a mechanism referred to as molecular mimicry (1). The molecular mimicry hypothesis suggests that immunological cross-reactivity between certain bacterial organisms and HLA-B27 is responsible for the pathogenesis of spondylitis (1). Although not specifically shown for T. vaginalis, certain parasites have been shown to acquire host antigens during infection (15). If this were to happen with T. vaginalis, the molecular mimicry hypothesis might also be applicable to infections with this parasite. The parasite could cause chronic prostatic infection and in the process acquire the host HLA-B27 antigen, making it immunologically reactive. No specific studies have been done to examine cross-reactivity between HLA-B27 and T. vaginalis. However, antibodies to prostatic tissue have been demonstrated in the sera of patients with ankylosing spondylitis (6).

T. vaginalis as a precipitating agent of ankylosing spondylitis could reasonably explain a few of the unique epidemiological features of ankylosing spondylitis. Although spondylitis does occur in prepubertal individuals, the onset of disease is usually in the second or third decade, during the years generally considered as the peak of sexual activity. Presumably, exposure to T. vaginalis would be most likely to occur during those years. The preponderance of clinical ankylosing spondylitis in men raises the possibility that prostatic infection may somehow be related to this observation and the fact that clinical sacroiliitis is relatively uncommon in women. The presence in women of ankylosing spondylitis initiated by T. vaginalis infection would have to be explained by other mechanisms. However, an association between spondylitis and salpingo-oophoritis has been reported (7).

This case report does not prove any association between ankylosing spondylitis and T. vaginalis. However, the observation raises the possibility that an association may exist and offers some theoretical justification for further study into the relationship.

LITERATURE CITED