## **Letters to the Editor**

### Infection related arthritis induced by tonsillar *Chlamydia trachomatis* and *Streptococcal* infection

Sir,

Several studies on *C. trachomatis* induced tonsillitis or pharyngitis have been reported (1-4). However, reactive arthritis (ReA) or infection related arthritis (IRA) (5) in relation to *C. trachomatis* infection of tonsils has not been reported. We describe two patients with IRA in which their tonsils were concomitantly infected with *C. trachomatis* and *Streptococcus*.

A 29-year old female had sore throat, pyrexia of 39°C and arthritis from November 1991. Physical examination revealed swelling of elbows and knee joints. Laboratory examinations revealed WBC of 9,800/mm3, CRP 4.3 mg/dl, ESR 47 mm/ hour, anti-streptolysin O (ASO) 416 U (normal range < 240) and antistreptokinase (ASK) 1280 U (normal range in adult < 1280). Group C Streptococcus was isolated from throat swabs. Rheumatoid factor (RF) was negative and antinuclear antibody (ANA) was 1:160 . Antibodies against C. trachomatis were positive; IgG 3.45 (index: < 0.9) and IgA 1.83 (< 0.9). Sore throat, pyrexia and arthritis did not subsided in spite of administration of piperacillin 2 g/day for 7 days. Therefore, tonsillectomy was performed.

Microabscesses were demonstrated in the resected tonsils and *C. trachomatis* but no other bacteria was isolated from the abscess. RNA for *C. trachomatis* was demonstrated. Chlamydial DNA was not demonstrated from the cervical swab. No recurrence of tonsillitis and arthritis was noted thereafter.

A 56-year old housewife admitted to our hospital in 1998 for sore throat, pyrexia of 38.5°C and arthritis of both knees and right wrist joints. Her right wrist and right 1st and 2nd metaphalangeal joints were markedly inflamed and swollen. Tonsils were inflamed with two ulcers. Laboratory examination revealed WBC of 11,100 /mm3, CRP 23.2 mg/dl and ESR 131 mm/hour. RF and ANA were negative. ASO was revealed to be 320 U and ASK was 1:640. Antibodies against were C. trachomatis IgG 3.47 and IgA 1.43. No other bacteria except alphaand beta-Streptococcus were isolated from the tonsillar swab. DNA for C. trachomatis was demonstrated from the tonsillar swab but not from the cervical swab. Results for mycoplasm, Ebstein-Barr virus, syphilis, hepatitis B and C virus, cytomegalovirus, HIV, C. pneumoniae, mycobacterium and Legionella pneumophlila were negative. Infective endocarditis was not demonstrated with blood culture and echocardiogram.

She was treated with panipenem/betamipron 2 g/day; however, improvement of pyrexia was not noted. Therefore, minocycline of 200 mg/day was administrated. Pyrexia subsided within 5 days and arthritis subsided within two weeks after the administration of minocycline. No recurrence of tonsillitis and arthritis was noted thereafter. Several studies on pharyngeal C. trachoma tis infection have shown a recent increase in the prevalence rate in the USA of 3.7% among patients from the genitourinary medicine clinic and recent reports in Japan have revealed coinciding increase in the prevalence rate (1,6). We previously reported a case of IRA induced by Pseudomonas aeruginosa isolated from a tonsillar abscess (7, 8). Therefore, it is evident that Strepto coccus is not the sole bacteria that induces IRA following tonsillitis. Our patients did not have spondyloarthropathy nor HLA-B27. Since our patients did not consent to synovial biopsy, the role of C. trachomatis and Streptococci in IRA is obscure (9,10). We supposed that both bacteria were causative agents for IRA. The route of transmission of C. trachomatis infection is also unclear. Although oro-genital sexual contact was suspected, both patients denied this fact. The pharyngeal mucosa is not suitable for colonization by C. trachomatis (1, 3); however, once C. trachomatis colonizes in the tonsillar crypts, it is apparently more difficult to eradicate than urethral C. tra chomatis, and prolonged or repeated administrations of antibiotics are necessary (3, 4). In conclusion, much attention should be focused on the detection of C. trachomatis, especially in the tonsillar microabscess, which may result in the inducement of IRA or ReA.

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# **Reactive arthritis induced** by *Gardnerella vaginalis*

Sirs,

Reactive spondyloarthropathies are conditions in which arthritis occurs after urogenital or gastrointestinal infections. The spectrum of the arthritogenous agents is large and the list is growing continously. We describe a patient with *Gardnerella vaginalis*induced reactive arthritis and dermatitis.

A 38-year-old woman was admitted for fever, skin rash and polyarthritis of new onset. Two weeks before she has suffered from dysuria, a pussy vaginal discharge and lower abdominal pain, together with fever up to  $39^{\circ}$  and chills. One week later, acute arthritis of the wrists appeared only to be shortly followed by additive inflammation of the small joints of the hands, elbows, knees and ankles and by the appearance of an itchy skin rash on both upper and lower extremities.

Examination revealed acute synovitis of the mentioned joints and an urticarial rash over the arms and calves. The temperature was 38.6°, blood pressure was 110/70 and the pulse was 90 per minute. Gynecological examination showed vaginal inflammation and discharge. The rest of the physical examination was not contributory. The ery-throcyte sedimentation rate was 80 mm Hg