UROGENITAL MYCOPLASMAS (SEE ALSO CHAP. 163)

EPIDEMIOLOGY

M. hominis, *M. genitalium*, *U. urealyticum*, and *U. parvum* can cause urogenital tract disease. The significance of isolation of these organisms in a variety of other syndromes is unknown and in some cases is being investigated. *M. fermentans* has not been shown convincingly to cause human disease.

While urogenital mycoplasmas may be transmitted to a fetus during passage through a colonized birth canal, sexual contact is the major mode of transmission, and the risk of colonization increases dramatically with increasing numbers of sexual partners. In asymptomatic women, these mycoplasmas may be found throughout the lower urogenital tract. The vagina yields the largest number of organisms; next most densely colonized are the periurethral area and the cervix. Ureaplasmas are isolated less often from urine than from the cervix, but *M. hominis* is found with approximately the same frequency at these two sites. Ureaplasmas are isolated from the vagina of 40–80% of sexually active, asymptomatic women and *M. hominis* from 21–70%. The two microorganisms are found concurrently in 31–60% of women. In men, colonization with each organism is less prevalent. Mycoplasmas have been isolated from urine, semen, and the distal urethra of asymptomatic men.

CLINICAL MANIFESTATIONS

Urethritis, Pyelonephritis, and Urinary Calculi In many episodes of *Chlamydia*-negative nongonococcal urethritis, ureaplasmas may be the causative agent. These organisms may also cause chronic voiding symptoms in women. The common presence of ureaplasmas in the urethra of asymptomatic men suggests either that only certain serovars are pathogenic or that predisposing factors, such as lack of immunity, must exist in persons who develop symptomatic infection. Alternatively, disease may develop only upon initial exposure to ureaplasmas. Ureaplasmas have been implicated in epididymitis. *M. genitalium* also appears to cause urethritis. *M. genitalium* and ureaplasmas do not have a known role in prostatitis. *M. hominis* does not appear to play a primary etiologic role in urethritis, epididymitis, or prostatitis.

Evidence suggests that *M. hominis* causes up to 5% of cases of acute pyelonephritis. Ureaplasmas have not been associated with this disease.

Ureaplasmas play a limited role in the production of urinary calculi. The frequency with which ureaplasmas reach the kidney, the predisposing factors that allow them to do so, and the relative frequency of urinary tract calculi induced by this organism (compared with other organisms) are not known.

Pelvic Inflammatory Disease *M. hominis* can cause pelvic inflammatory disease. In most episodes, *M. hominis* occurs as part of a polymicrobial infection, but the organism may play an independent role in a limited number of cases. Some data also support an association of *M. genitalium* with pelvic inflammatory disease. Ureaplasmas are not thought to cause pelvic inflammatory disease.

Postpartum and Postabortal Infection Studies implicate *M. hominis* as the primary pathogen in ~5–10% of women who have postpartum or postabortal fever; ureaplasmas have been implicated to a lesser degree. These infections are generally self-limited; however, if symptoms persist, specific antimicrobial therapy should be given. Ureaplasmas also appear to play a role in occasional postcesarean wound infections.

Non-urogenital Infection In rare instances, *M. hominis* causes non-urogenital infections, such as brain abscess, wound infection, poststernotomy mediastinitis, endocarditis, and neonatal meningitis. These infections are most common among immunocompromised and hypogammaglobulinemic patients. Ureaplasmas and *M. hominis* can cause septic arthritis in immunodeficient patients. Ureaplasmas probably cause neonatal pneumonitis; their significant role in the development of bronchopulmonary dysplasia—the chronic lung disease of premature infants—has been documented in a number of studies. It is unclear whether ureaplasmas and *M. hominis* cause infertility, spontaneous abortion, premature labor, low birth weight, or chorioamnionitis.

DIAGNOSIS

Culture and PCR are both appropriate methods for the isolation of urogenital mycoplasmas. Culture of these organisms, however, requires special techniques and media that generally are available only at larger medical centers and reference laboratories. Serologic testing is not recommended for the clinical diagnosis of urogenital *Mycoplasma* infections.

TREATMENT UROGENITAL *MYCOPLASMA* INFECTIONS

Because colonization with urogenital mycoplasmas is common, it appears at present that their isolation from the urogenital tract in the absence of disease generally does not warrant treatment. Macrolides and doxycycline are considered the antimicrobial agents of choice for *Ureaplasma* infections (Table 212-2). *Ureaplasma* resistance to macrolides, doxycycline, quinolones, and chloramphenicol has been reported. *M. hominis* is resistant to macrolides. Doxycycline is generally the drug of choice for *M. hominis* infections, although resistance has been reported. Clindamycin is generally active against *M. hominis*. Quinolones are active in vitro against *M. hominis*. For *M. genitalium,* the agent of choice appears to be azithromycin; treatment failures have been reported with other macrolides as well as with quinolones.

213 Chlamydial Infections

Charlotte A. Gaydos, Thomas C. Quinn

Chlamydiae are obligate intracellular bacteria that cause a wide variety of diseases in humans and animals.

ETIOLOGIC AGENTS

The chlamydiae were originally classified as four species in the genus *Chlamydia: C. trachomatis*, *C. pneumoniae, C. psittaci*, and *C. pecorum* (the last species being found in ruminants). The *C. psittaci* group has been separated into three species: *C. psittaci, C. felis*, and *C. abortus.* The mouse pneumonitis strain (MoPn) is now classified as *C. muridarum*, and the guinea pig inclusion conjunctivitis strain (GPIC) is now designated *C. caviae*.

C. trachomatis is divided into two biovars: trachoma and LGV (lymphogranuloma venereum). The trachoma biovar causes two major types of disease in humans: ocular trachoma, the leading infectious cause of preventable blindness in the developing world; and urogenital infections, which are sexually or neonatally transmitted. The 18 serovars of *C. trachomatis* fall into three groups: the trachoma serovars A, B, Ba, and C; the oculogenital serovars D–K; and the LGV serovars L_1–L_3. Serovars can be distinguished by serologic typing with monoclonal antibodies or by molecular gene typing. However, serovar identification usually is not important clinically since the antibiotic susceptibility pattern is the same for all three groups. The one exception applies when LGV is suspected on clinical grounds; in this situation, serovar determination is important because a longer treatment duration is required for LGV strains.

BIOLOGY, GROWTH CYCLE, AND PATHOGENESIS

BIOLOGY

During their intracellular growth, chlamydiae produce characteristic intracytoplasmic inclusions that can be visualized by direct fluorescent antibody (DFA) or Giemsa staining of infected clinical material, such as conjunctival scrapings or cervical or urethral epithelial