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A Review on *Ureaplasma urealyticum*

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Abstract

Ureaplasma urealyticum and other *ureaplasma* species are bacteria that are typically sexually transmitted. *Ureaplasma* is one of the most common infections in sexually-active men and women. *Ureaplasma* belongs to the family *Mycoplasmataceae*, class *Mollicutes* order *Mycoplasmatales*. *Ureaplasma urealyticum* are some of the smallest known organisms that can self-replicate on laboratory media. *Ureaplasma* has been implicated in neonatal morbidity and mortality including congenital pneumonia, preterm delivery, low birth weight and intrauterine growth retardation. If cervical cultures for ureaplasma and mycoplasma are positive, both the patient and her sexual partner are usually treated with antibiotics such as doxycycline. *Ureaplasma urealyticum* can be transmitted in various ways, including directly by sexual transmission through direct contact between couples, vertically from mother to offspring, through hospital-acquired infections from transplanted tissues, or through unprotected sexual contact with an infected person, whether vaginal, oral or anal. *Ureaplasma* spp. genital infections have been reported with an increasing frequency in HIV-infected patients.

Keywords: *Ureaplasma urealyticum*, cervical cultures, antibiotics.

Introduction

Ureaplasma urealyticum

Ureaplasma urealyticum and other *ureaplasma* species are bacteria that are typically sexually transmitted. *Ureaplasma* is one of the most common infections in sexually-active men and women. It is often under-recognized. New York Urology Specialists is one of the few specialized practices in New York City that tests for and treats *Ureaplasma*, mycoplasma and *Trichomonas* in men (Joyce, 2016; Ochiabuto and Obeagu, 2018).

Etiology of *ureaplasma urealyticum*

Shepard (2005) described tiny *mycoplasmas* he found within the human urogenital tract. Denys (2012) confirmed these *mycoplasmas* as being self replicating entities that grew at a low pH. Growing in colonies resembling fried eggs they were put in the *Ureaplasma* genus. The cells were either spherical or coccobacillary shaped Gram-positive microbes that cannot be seen with the naked eye. The cell diameter ranges from 0.1 to 1.0 μm . Its type strain is T960. Named in 1974 by Shepard with its official name *Ureaplasma urealyticum* there were 14 identified serotypes (servars). A few of them were associated with diseases. Within these

serotypes two distinct groups were recognized. Separated depending on their phenotypic markers such as clustering of antigenic types, polypeptide patterns of whole cell preparations, differential inhibition by manganese, and polymorphism among their ureases, pyrophosphatases and diaphorases (Blanchard *et al.*, 2000). They were known as biovar 1 and biovar 2. After long debates in 2002 they were separated into two distinct species: *Ureaplasma parvum* and *Ureaplasma urealyticum* (Back *et al.*, 2003). Serovars 1,3,6 and 14 were designated as *Ureaplasma parvum* (previously *Ureaplasma urealyticum* biovar 1) due to their slightly smaller genome size. While its etiologic significance in many aspects of adverse pregnancy remains controversial, recent evidence indicates that *Ureaplasma urealyticum* in the absence of other organisms is a cause of chorioamnionitis (Jensen *et al.*, 2008). Their possible roles in certain pathologic conditions in humans. Because of their extremely fastidious nature and the lack of reliable means for cultivation on artificial media, detection of these mycoplasmal organisms rests primarily with molecular techniques (Bry *et al.*, 2007). Relatively little is known about their importance as human pathogens, with the notable exception of *M genitalium*, an organism that has been the focus of a considerable number of clinical research studies in recent years (Kafetzis *et al.*, 2004).

Taxonomy

Ureaplasma belongs to the family *Mycoplasmataceae*, class *Mollicutes* order *Mycoplasmatales*. *Ureaplasma* was first discovered in 1954 by Shepard *et al.*, as a pathogen causing non-gonococcal urethritis in men (Grenabo *et al.*, 2008). Since the organisms produced small colonies (7-15 µm diameter), they were originally called T (tiny) strains, T-strain Mycoplasmas or T-Mycoplasmas. They were deemed unique among the Mycoplasmas of human origin in that they metabolized urea and not arginine or glucose (Honma *et al.*, 2007).

It was thus proposed that there should be a new genus and species designation for these unique organisms within the order *Mycoplasmatales*. They were named *Ureaplasma urealyticum* (Shepard *et al.*, in 1974).

Structure

Ureaplasma urealyticum are some of the smallest known organisms that can self-replicate on

laboratory media. *U. urealyticum* is often associated with *Mycoplasmas* and shares a similar cell structure. *Mollicutes* and *Ureaplasma urealyticum*, are known for having the following characteristics:

1. They range from 125-250 nm in size.
2. They are highly pleomorphic, because they lack a cell wall.
3. They are bound by a triple-layered "unit membrane" that contains a sterol, causing them to stain as gram negative.
4. *Ureaplasma urealyticum* can reproduce in cell-free media, agar. (The center of the colony is usually embedded beneath the surface.) However, they require the addition of cholesterol, which is required to make their "unit membrane."
5. *Mycoplasmas* (and therefore also *U. urealyticum*) are completely resistant to penicillin because they lack a cell wall, but are inhibited by tetracycline or erythromycin.
6. Growth of *Mycoplasmas* is inhibited by specific antibodies.
7. Mollicutes generally lack TCA cycle
8. *Mycoplasmas* have an affinity for mammalian cell membranes (Waites *et al.*; 2005).

Genetic Type

Ureaplasma urealyticum is a bacterium belonging to the family Mycoplasmataceae

Epidemiology

United States

Ureaplasma species have been isolated from cervicovaginal specimens in 20-50% of men who are asymptomatic and sexually active. *Mycoplasma hominis* has been isolated from cervicovaginal specimens in 15-40% of men who are asymptomatic and sexually active (Mallard *et al.*, 2005). These rates are somewhat higher in females. Only subgroups of adults who are

colonized in the lower urogenital tract develop symptomatic illness from these organisms. Nongonococcal urethritis is the most common sexually transmitted infection. *Ureaplasma* species and *mycoplasma genitalium* may account for a significant portion of cases that are not due to chlamydiae. *Mycoplasma genitalium* is much less likely to be present in the urogenital tract of asymptomatic persons (Kong *et al.*, 2000). More than 20% of liveborn infants may be colonized by *Ureaplasma*, and infants born preterm most likely harbor the organisms. Colonization declines after

age 3 months. Less than 5% of children and 10% of adults who are not sexually active are colonized with genital *mycoplasmal* microorganisms (Smith *et al.*, 2008)

NIGERIA

Although few studies have investigated the geographic distribution of genital *ureaplasma* infections, the facts that:

1. They are present on mucosal surfaces in so many healthy persons.
2. They can be transmitted venereal suggest that variation in prevalence of these organisms in adults is more likely related to behavioural variables such as number of sexual partners and socioeconomic status rather than to geographic or climatic differences.

SEX

No obvious sex predilection is reported for infections due to genital *ureaplasma* species, except for the differences in urogenital diseases such as salpingitis and endometritis, which are gender-specific. The carriage rate of genital *Ureaplasma* species in the lower urogenital tract is somewhat greater for females than for males (Aaltone *et al.*, 2002).

AGE

Ureaplasma species are common commensal inhabitants of the lower genitourinary tract in adolescents and adult men who are sexually active. The organisms can be transmitted venereally and vertically from male to their female couple. Neonates who acquire the organisms are usually colonized in the upper and sometimes lower respiratory tracts with occasional dissemination to the bloodstream and CSF Cheesbrough (2005). Clinically significant infections may ensue in individuals who are sexually active and in neonates but are rare to nonexistent in older. *Ureaplasma* and *mycoplasma* are bacteria that can be commonly found in the reproductive tract of men. It is somewhat more problematic to label these two bacteria as reproductive tract pathogens because they are often found in fertile, healthy couples in addition to those with infertility. Although the presence of these two bacteria has been hypothesized to play a role in infertility and miscarriage, the specific mechanisms by which they impair fertility remains unclear (Ianaro *et al.*, 2000).

RACE

Differences in carriage of genital *ureaplasma* organisms and subsequent disease are more likely related to sexual behavior and socioeconomic status than to race. Colonization appears to be more common in whites than in African Americans, but it is not clear whether this is a true racial difference as opposed to a socioeconomic factor (Naing *et al.*, 2006).

UREAPLASMA IN THE NEWBORN INFANT

Ureaplasma has been implicated in neonatal morbidity and mortality including congenital pneumonia, preterm delivery, low birth weight and intrauterine growth retardation (Joste *et al.*, 2000) and it is thought to infect or colonise up to 37% of newborns. It has been implicated in the pathogenesis of neonatal pneumonia and meningitis and there are also reports of a systemic inflammatory response in babies with evidence of *Ureaplasma* in the lower respiratory tract as evidenced by an elevated white blood cell count, particularly in the first 2 days of life (Kirchner *et al.*, 2007).

There have been a number of case reports of *Ureaplasma* infection of the cerebrospinal fluid (CSF) (Waites *et al.*, 2005) suggest that *Ureaplasma* spp. may in fact be a relatively common cause of CSF infection in the neonatal population. Their study, along with other case reports, records a range of central nervous system clinical outcomes for infants in whom such an infection is diagnosed — from spontaneous resolution (Olomu *et al.*, 2009), to development of hydrocephalus or intraventricular haemorrhage, to mortality in association with erythromycin resistant *Ureaplasma* spp.

INCUBATION PERIOD

If cervical cultures for *ureaplasma* and *mycoplasma* are positive, both the patient and her sexual partner are usually treated with antibiotics such as doxycycline. As these bacteria may have been present for many years without causing any symptoms (Waites *et al.*, 2005), the finding of *ureaplasma* and *mycoplasma* on cervical cultures does not in any way indicate infidelity or sexual misconduct. Usually the symptoms improve or resolve over a period of weeks or months, and an improvement in the strength of your immune system may also contain the infection (David 2013).

MODES OF TRANSMISSION

Ureaplasma urealyticum can be transmitted in various ways, including directly by sexual transmission through direct contact between couples, vertically from mother to offspring, through hospital-acquired infections from transplanted tissues, or through unprotected sexual contact with an infected person, whether vaginal, oral or anal (Koletar 2013).

MORBIDITY AND MORTALITY RATE

Assessing morbidity and mortality for diseases specifically caused by genital *ureaplasma* infections is difficult because few studies systematically evaluate them and some conditions with which they are involved can be polymicrobial (eg, pelvic inflammatory disease, urethritis) (Peltier 2003). Difficulty in detecting the more fastidious species, such as *Mycoplasma genitalium* and *Ureaplasma urealyticum*, further complicates such assessments.

In adults with an intact and functional immune system, infections associated with genital *ureaplasma* organisms are usually localized and do not result in severe illness, attesting to their relatively low virulence and perceived status as

opportunists (Viscardi *et al.*, 2006). Persons with antibody deficiencies reportedly have developed severe pulmonary infections, destructive arthritis and osteomyelitis associated with subcutaneous abscesses, and other disseminated infections of various organ systems (Cassell *et al.*, 2014). Deaths have occurred in neonates with bloodstream invasion by *Ureaplasma* species and meningitis caused by *Mycoplasma hominis*; however, in some instances, the organisms spontaneously disappeared from CSF without treatment.

CHRONIC LUNG DISEASE OF PREMATURITY

The association between the presence of *Ureaplasma* and the development of CLD remains controversial and hotly debated. The pathogenesis of CLD is multi-factorial with prematurity, ventilator induced lung injury, oxygen therapy, patent ductus arteriosus, fluid balance and infection, both ante- and post-natal, all appearing to have a role to play (Pinna *et al.*, 2006).

It is often difficult to dissect out the effect of one particular risk factor as there is overlap between the risk factors in this multi-factorial disease.

Pulmonary *Ureaplasma* colonisation is strongly linked to preterm delivery and the question remains if this pulmonary colonisation with *Ureaplasma* is an independent risk factor for CLD (Daniel *et al.*, 2009). There have been numerous studies investigating the role of *Ureaplasma* in the development of CLD, as well as many reviews and metaanalyses published in recent years. The consistent observation in many publications is the difficulty in interpreting evidence from available studies due to small sample sizes, vastly different inclusion criteria, different methods of sampling and testing, different diagnostic criteria for various outcomes including CLD (Kwak *et al.*, 2014). Much call for definitive studies but few addresses in detail the obstacles to an adequately powered clinical trial.

Many studies at the time did not use a definition of oxygen dependency at 36 weeks to diagnose CLD thus association with this outcome was not available. Since that review, several further studies have been completed, including one by (Kotecha *et al.*, 2004) who sought *Ureaplasma* in

bronchoalveolar lavage fluid from 17 preterm neonates without clinical or laboratory evidence of infection in either the mother or infant and reported that 6 were positive for *Ureaplasma urealyticum*. Of the 6 babies with *Ureaplasma*, 5 developed CLD whereas only 4 of the 11 babies without *Ureaplasma* developed CLD. Their data strongly implicated *Ureaplasma* in the development of the pulmonary inflammatory response observed in infants who progress to develop CLD. In a cohort of 126 preterm deliveries (Kafetzis *et al.*, 2009), found a significant increase in CLD as well as mortality among *Ureaplasma* colonised infants. Waarde *et al.* (2014) found that *Ureaplasma* was significantly associated with both CLD and lower gestational age but logistic regression analysis failed.

MICROSCOPY

Lack of a rigid cell wall makes it nearly impossible to directly visualize *Ureaplasma* by light microscopy. Though Gram stain precludes visualisation, it is useful to rule out other causative bacteria. DNA fluorochrome stains like acridine orange and Hoechst 33258 may be useful in centrifuged samples like amniotic fluid (Waites *et al.*, 2005).

CLINICAL SIGNS AND SYMPTOMS

1. For most people, *Ureaplasma* remains in the genitals and has no effect or symptoms.
2. A continual dull ache or pain around the genitals or lower abdomen.
3. Burning or pain when urinating.
4. *Ureaplasma* has been associated with a number of diseases such as non-specific urethritis (NSU) and sterile pyuria.
5. Whether *Ureaplasma* can cause infertility, chorioamnionitis, stillbirth, premature birth, and, in the perinatal period, pneumonia, bronchopulmonary dysplasia and meningitis is contentious.
6. Prostatitis (inflammation of the prostate gland) causing more frequent urination or reduced urine flow.
7. Inflammation of testicles, urethra, epididymis, fallopian tubes, other areas of the body depending on where the infection was received.
8. Fatigue (Redelinghuys *et al.*, 2014).

TREATMENT OF UREAPLASMA INFECTION

Ureaplasma can be effectively treated and cured with a course of antibiotics. If one is suffering any symptoms, it is important to provide a urine sample for testing by a pathology lab. This will rule out the possibility of infection by a more dangerous bacteria /protozoa (Rodriguez *et al.*, 2014)

Conventional medicine usually treats a *U. urealyticum* infection with antibiotic doxycycline or streptomycin. Of course both partners must be treated, and outside of a strictly monogamous relationship there is a high chance of re- infection. It requires a strong course of antibiotics, and there is a possibility that your digestive and other beneficial bacteria will be devastated, with a risk of development of IBS and other problems (Shepard *et al.*, 2014). The natural home remedy approach to treating *ureaplasma urealyticum* is to leave them alone - in a healthy person they are a commensal - in other words, they should cause no problems, and most sexually active people have them. If your symptoms are serious and a test has confirmed a *u. urealyticum* infection and no other infections or causes (Vogel *et al.*, 2015), a natural antibiotic such as colloidal silver may be able to contain the bacterial overgrowth. Usually the symptoms improve or resolve over a period of weeks or months, and an improvement in the strength of your immune system may also contain the infection (Basar ,2004).

Since syndromes characteristic of genitourinary infection is not caused only by genital Mycoplasmas but also by various other organisms, antibiotic susceptibility to all of them must be taken into account while prescribing empirical therapy (Waites *et al.*, 2014). Taking this into consideration, the preferred treatment is azithromycin 1 g orally as single dose or doxycycline 100 mg orally twice a day for 7 days. However, since resistance to tetracyclines is on the rise, a patient who fails to respond to doxycycline may also be treated with erythromycin 500 mg orally for 7 days. Fluoroquinolones also demonstrate efficacy equal to doxycycline in the treatment of non gonococcal urethritis (Buve *et al.*, 2005). The efficacy and safety of a 7-day course of sparfloxacin were comparable to those of doxycycline in a double-blinded randomized multicenter study of 725 men

with clinically diagnosed NGU from chlamydial infection and 5 PMN on urethral microscopy who were tested by culture for *N. gonorrhoeae*, *C. trachomatis*, *U. urealyticum*, and *M. Hominis* (Buve *et al.*, 2005). However, *Ureaplasma sp.* is known to cause persistent infection in which case a prolonged course of antibiotics may be required.

Recently, a randomized trial from Turkey evaluated clarithromycin in 74 preterm infants and found that treatment for 10 days decreased the incidence of BPD (Deguchi *et al.*, 2004). Although no significant side-effects were reported in these studies, antibiotics must be used with caution in preterm neonates since prolonged antibiotic exposure has been associated with increased rates of necrotizing enterocolitis or late onset sepsis. For invasive mycoplasmal infections like infection of CSF, tetracyclines are the best treatment (Couldwell *et al.*, 2010).

According to a study done at the University of Kurtkukale Faculty of Medicine (Kurtkkale, Turkey) *Ureaplasma urealyticum* showed no resistance to doxycycline, 4.2% resistance to tetracycline, 12.5% resistance to erythromycin and 16.7% resistance to ofloxacin (Basar *et al.*, 2004). As a result, physicians prescribe 100mg of doxycycline twice a day. Other physicians have also found 500 mg of azithromycin twice a week to be effective. These treatments are usually long-term. Other research on using fluoroquinolones and/or clindamycin for treatment is still underway (Ondondo *et al.*, 2010).

RISK OF HAVING SYMPTOMS

1. The older you are when you get your initial *ureaplasma* infection, the more likely that you will suffer a mild pain, an NSU or some other symptoms.
2. If your immune system is weak, there is an increased chance of suffering from the above symptoms (Ondondo *et al.*, 2010).

UREAPLASMA AND HIV

Ureaplasma spp. genital infections have been reported with an increasing frequency in HIV-infected patients. In a study by (Martinelli *et al.*, 2004) involving 187 human immunodeficiency virus type 1 (HIV-1)-infected male patients with no clinical signs of urethritis, the prevalence of *U. urealyticum* was higher in AIDS patients (12.3%) than in asymptomatic, HIV-1-infected patients

(8.5%) and in healthy individuals (7%) (Maeda *et al.*, 2004) demonstrated a 6% infection rate by *Ureaplasma* in HIV infected patients compared to 2% in healthy volunteers (Shepard *et al.*, 2005). these studies indicate that infection with *Ureaplasma* sp., potentially increase the susceptibility of acquiring and transmitting HIV. Other genital *Mycoplasmas* like *M. hominis* and *M. genitalium* have been termed as candidate co factors in the pathogenesis of AIDS since they act in synergy with HIV virus and exacerbate the retroviral disease though not proven, *Ureaplasma* may have a similar role to play in HIV infected patients (Shepard *et al.*, 2005).

LABORATORY DIAGNOSIS

If you are suffering any symptoms, it is important to provide a urine sample for testing by a pathology laboratory.

METHOD

Methods for laboratory detection of *ureaplasmas* have been greatly improved over the past years because of effective molecular- based techniques. Relatively rapid bacterial growth makes the identification of most positive cultures possible within two to four days, but culture cannot differentiate between the species (Waites and Taylor-Robinson 2005).

Molecular-based methods, such as PCR, are able to detect and identify *U. Parvum* and *U. urealyticum* separately. For target sequences, 16S rRNA gene, 16S rRNA to 23S rRNA intergenic spacer regions,

the urease gene, and *mba* gene are mainly used (Waites *et al.*, 2012). In addition, a number of diverse genotyping methods have been developed for identification of *Ureaplasma* serotypes: restriction fragment length polymorphism (RFLP), pulsed-field gel electrophoresis (PFGE), a high-resolution melt (HRM) PCR assay, real-time PCR, and multilocus sequence typing (MLST) assay (Paynes *et al.*, 2014; Zhang *et al.*, 2014).

CULTURE

Culture remains the most economical and practical means of detection for laboratories with a low to moderate sample volume. Culture also has an advantage of providing antimicrobial susceptibility testing (Waites *et al.*, 2005).

Whether culture- or non-culture-based detection methods should be used for diagnostic purposes depends on the resources and facilities available in individual laboratories and the species being sought (Robertson *et al.*, 2002). Indirect, serological test methods for *ureaplasmas* include micro immunofluorescence, metabolism inhibition, and enzyme immunoassay¹³, but the interpretation of antibody titers is difficult because of their ubiquity in healthy people so it is of limited clinical use.

SEROLOGY

Serological test methods for *Ureaplasma* include microimmunofluorescence, metabolism inhibition, and enzyme immunoassay, but the ubiquity of *Ureaplasma* in healthy people makes interpretation of antibody titres against these organisms difficult. No serological assays for the genital *Mycoplasmas* have been standardized, and they are not available for diagnostic purposes (Saada *et al.*, 2015).

MOLECULAR

Ureaplasma species are common commensals in the lower urogenital tracts of healthy people. Therefore, a positive PCR assay from specimens from these sites is usually expected. An increased bacterial load determined by real-time PCR is more valuable as a clinical indication of infection (Busolo *et al.*, 2014). Positive PCR results for *Ureaplasma* species from the urethra in men with urethritis, from tracheal aspirates of neonates with respiratory distress, from the bloodstream or cerebrospinal fluid in neonates with pleocytosis, and from normally sterile extragenital sites should be considered diagnostic of clinically significant infection (Shepard *et al.*, 2014).

PATHOLOGY

The 2 *Ureaplasma* biovars, *Ureaplasma urealyticum* and *Ureaplasma parvum*, are now designated as separate species. Separation of these species is not possible except via molecular techniques such as polymerase chain reaction (PCR). Therefore, they are now considered together as *Ureaplasma* species (Shepard *et al.*, 2014). *U. parvum* is generally the most common species detected in various clinical specimens but *U. urealyticum* is apparently more pathogenic in conditions such as male urethritis. This

differential pathogenicity at the species level has not been shown consistently for other disease conditions (Taylor-Robinson *et al.*, 2000).

PATHOGENESIS

Ureaplasma species are the smallest free-living organisms and are unique among prokaryotes in that they lack a cell wall. This feature is largely responsible for their biologic properties, including lack of a Gram stain reaction and non susceptibility to many commonly prescribed antimicrobial agents, including beta-lactams. *Ureaplasma* organisms are usually associated with mucosae. They usually reside extracellularly in the respiratory and urogenital tracts and rarely penetrate the submucosa, except in the case of immunosuppression or instrumentation, when they may invade the bloodstream and disseminate to numerous organs and tissues. Some species also occur as intracellular pathogens (Xiao *et al.*, 2011). Among the 17 species isolated from humans, 4 types of organisms are of major concern. *Mycoplasma pneumoniae* is a well-established pathogen; it is rarely isolated from healthy persons. *Mycoplasma hominis* and *Ureaplasma* species, known collectively as the genital mycoplasmal organisms, are generally considered opportunists that cause invasive infections in susceptible populations (Waites *et al.*, 2012).

BIOLOGICAL

Urogenital mycoplasmosis of humans is typically associated with *Mycoplasma genitalium*(MG), *Mycoplasma hominis*(MH), *Ureaplasma urealyticum*(UU), *Ureaplasma parvum*(UP), or a complex infection of more than one of these species. *Mycoplasma penetrans* and *Mycoplasma pirumare* not commonly associated with clinical signs (Xiao *et al.*, 2010),

PHYSICAL

Rather than listing the many nonspecific historical and clinical findings of various entities that may be associated with infection with *Mycoplasma* or *Ureaplasma* species, emphasizing the need to consider these organisms as potential etiologic agents in the conditions named above is more important in order to perform the necessary diagnostic tests and to provide appropriate antimicrobial treatment that provides coverage for

them. Consider a *Mycoplasma* or *Ureaplasma* infection when persons with hypogammaglobulinemia present with septic arthritis, chronic pulmonary infection, and any other inflammatory condition or infection that does not respond to antimicrobial treatment that is not likely to be effective against these organisms (Iwasaka *et al.*, 2006).

Ureaplasma urealyticum, cross-resistance was found between erythromycin and macrolides (except josamycin) (40–80 %) and between erythromycin and ciprofloxacin (79 %). Antibiotic resistance over the test period did not vary significantly. Because of geographical differences among antibiotic resistance, local in-vitro susceptibility testing is recommended to avoid failure of therapy. *U. urealyticum* are causes of nonchlamydial nongonococcal urethritis in men (McIver *et al.*, 2009).

Conclusion

Ureaplasma urealyticum and other *ureaplasma* species are bacteria that are typically sexually transmitted. *Ureaplasma* is one of the most common infections in sexually-active men and women. *Ureaplasma* has been implicated in neonatal morbidity and mortality including congenital pneumonia, preterm delivery, low birth weight and intrauterine growth retardation. *Ureaplasma urealyticum* can be transmitted in various ways, including directly by sexual transmission through direct contact between couples, vertically from mother to offspring, through hospital-acquired infections from transplanted tissues, or through unprotected sexual contact with an infected person, whether vaginal, oral or anal. *Ureaplasma* spp. genital infections have been reported with an increasing frequency in HIV-infected patients.

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