Chlamydia is the most common STD in North America infecting men and women. The majority of these cases occur in the adolescent population (McCance & Huether, 2006). The infection named after the bacterium that causes it, Chlamydia trachomatis is transmitted through vaginal, anal, or oral sex and is susceptible to inexpensive, readily accessible antibiotics. However, this infection often goes untreated because 75% of women and over 25% of men are asymptomatic (MediResource Inc., n.d.). Untreated Chlamydia in women is the leading cause for etopic pregnancies and infertility (McCance & Huether).

Chlamydia alternates between two distinct stages: (1) elementary bodies (EB) and (2) reticulate bodies (RB). Elementary bodies have a rigid outer membrane and are resistant to harsh environmental conditions when outside their eukaryotic host cells. The elementary bodies bind to receptors on host cells and are able to enter by endocytosis.

RB is the second stage whereby Chlamydia develops into a metabolically active parasite within the cell and is replicated by binary fusion until the cell death. As a result up to 1000 new EB are disseminated. The strains of C.trachomatis that create urogenital infections require squamous-columnar and columnar epithelial cells as host. Chlamydia in women typically begins at the cervix or as lower genital tract infections (MediResource Inc., n.d.). In the cervix, Chlamydia can contribute to infertility as a result of alterations in the mucosa and increased presence of inflammatory cells (Pellati et al., 2008).

 In 50% of the cases of lower genital tract infections, the micro-organism will clear spontaneously while cervical infections may persist for years. 10% of cervical infections will ascend up to the upper genital tract and induce pelvic inflammatory disease (PID) (Land, 2004). When infection ascends to the uterus, fallopian tubes and peritoneum, the PID can damage the interior of the reproductive organs and cause scar tissue to form, thus leading to infertility and ectopic pregnancies (MediResource Inc., n.d.). Basically, this inflammation and scar tissue cause tubal obstructions/adhesions which disrupt the passing of the oocyte through the tubes (Pellati et. al, 2008).

However, not all women with asymptomatic Chlamydia of the upper genital tract infection will develop PID. One current hypothesis is that only hosts repeatedly exposed to the microorganism by either persistent infection or re-infection will develop extensive tissue damage in the upper genital tract. As cited by Lang, this tissue injury appears to be induced by cell-mediated immunologic reactions, including a delayed hypersensitivity response, which leads to fibrosis and scarring and ultimately tubal factor subfertility (2004).

Studies have established a relationship between tubal factor subfertility with women with Chlamydia IgG antibodies in their serum (Land, 2004). Therefore, in Ms. T situation, concern that her past hx of Chlamydia may be impeding her ability to be pregnant can be addressed with an inexpensive antibody test.

Another part of the equation in infertility is the male. Men can also be affected by Chlamydia infections. The primary site of infection is the penile urethra. It can also lead to epididymitis and orchitis. So, there is also the possibility that inflammation could lead to a degree of male infertility. For example, inflammation in the epididymus could lead to tubal blockage and epithelial damage that may affect spermatogenesis. This aspect is still under investigation but is something to think about in terms of screening and intervention (Cunningham & Beagley, 2008).

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