TRICHOMONIASIS

Urogenital trichomoniasis is caused by infection with the protozoan *Trichomonas vaginalis*; about 170 million new cases each year may well make it the world's commonest non-viral sexually transmitted infection.

In clinical specimens or culture *T.vaginalis* is a motile, round or oval flagellate 10 - 13 :m long and 8 - 10 :m wide; fixed and stained it is about 25% smaller. Diagnostic features include the jerky motility, undulating membrane and microtubular rod (the axostyle), which runs through the body and projects as a thin spine from the posterior end. In contact with vaginal epithelial cells *in vitro* the organism becomes extremely flattened and adherent; probably this is the normal morphology in women. The life cycle is simple; no resistant cysts are formed and there are no intermediate or reservoir hosts.

Two other trichomonads - *T.tenax* and *Pentatrichomonas hominis* - are uncommon and probably harmless human parasites of the mouth and large bowel respectively. All three species are site specific and urogenital trichomoniasis is not due to contamination from other sites.

Epidemiology

Although it is often difficult to isolate the organism from male contacts of infected women, all epidemiological evidence suggests that the vast majority of infections are sexually acquired. *T.vaginalis* has been shown to survive for many hours at room temperature if kept damp so the theoretical possibility of non-venereal transmission exists. It is also known that a very small proportion of female babies of infected mothers will become infected during birth, but the infection is transient and trichomoniasis discovered in a child should immediately raise the suspicion of sexual abuse.

Very few studies have been made of genuinely unselected populations; the majority have examined either pregnant women or those attending STI clinics. There are wide variations but most report 10 - 25% infected although the full range is 0 - 63%. Usually female cases outnumber males by 5 or 10 to 1. In several developed countries there has been a steady decline in the incidence of trichomoniasis in the past two decades, but this has not occurred in less-developed countries nor in deprived inner-city areas in industrialised nations. Human trichomoniasis is becoming a disease of the underprivileged.

Pathogenesis

In vitro T.vaginalis has a well defined, contact-mediated, cytotoxic effect, but the relationship of this to pathogenesis *in vivo* is not known. The organism activates complement and attracts neutrophils; several together can kill the parasite, but their presence in large numbers may be responsible for much of the pathology observed. It seems likely that differences in clinical severity are due to both host and parasite factors.

Symptoms

In women trichomoniasis may present as anything from an asymptomatic infection (10-50% of cases) to an acute inflammatory diseases with a copious and malodorous discharge; vulvovaginal soreness and irritation; dysuria and dyspareunia are also frequently mentioned. The discharge may vary over time and, untreated, the infection may be spontaneously lost or persist for months or years. A recent study showed trichomoniasis significantly associated with symptoms of yellow vaginal discharge and vulvar itching and signs of colpitis macularis (strawberry cervix), purulent vaginal discharge and vulval and vaginal erythema. Colpitis was seen frequently if colposcopy was undertaken, but hardly ever by naked-eye examination. Vaginal pH is usually elevated.

The majority of men with trichomoniasis are asymptomatic, but the parasite is responsible for a small but increasing proportion of cases of non-gonococcal urethritis.

Pathology

In women, *T.vaginalis* may be found in the vagina and the exterior cervix in over 95% of infections, but is only recovered from the endocervix in 13%. The urethra and Skene's glands are also commonly infected. In men the urethra is the most common site of infection, but the organism has also been recovered from epididymal aspirates. Dissemination beyond the lower urogenital tract is extremely rare even in severely immunocompromised patients.

Previously regarded as unpleasant but harmless, epidemiological studies have recently linked trichomoniasis in women with a modest increases in the risk of heterosexual HIV transmission and with adverse pregnancy outcome and have suggested that it might be the actual cause of a few percent of cervical neoplasias.

Laboratory diagnosis

The symptoms and signs are not sufficient to establish the diagnosis, which must be made by detecting the parasite. This is most frequently achieved by wet-film microscopic examination of vaginal (not endocervical) secretions, urethral scrapings, centrifuged urine sediment or prostate fluid. The specimen should be examined as soon as possible - a motile trichomonad is unmistakable. Sensitivity is moderate, 50-70% in women but only 10 - 20% in men

Culture provides significantly greater sensitivity; media vary in efficiency but Diamond's TYM and the very convenient if rather expensive InPouch® system are amongst the best.

Antigen detection, DNA probe, and PCR-based tests have been developed; none have yet found widespread acceptance but the sensitivity of the PCR-based methods offers exciting new possibilities for making an accurate diagnosis on specimens obtained in less invasive ways including self-administered tampons.

Treatment

The 5-nitroimidazole drugs provided the first and so far only group of effective chemotherapeutic agents. Doses given here are for metronidazole and should be adjusted to give the equivalent amount of other compounds. Two regimens are used - the original one of 250 mg three times a day for 7 days, or a single 1.6 or 2-g dose. Cure rates in women are similar (about 95%) with both regimens if male sexual partners are also treated, but appear to be lower with the single-dose regimen if they are not. Only the 7-day regimen has been extensively evaluated in males, where it is equally effective. Treatment failures with any of the 5-nitroimidazole drugs are rare, but a proportion is due to resistant parasites.

References

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