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***Trichomonas vaginalis*: Complications and Treatment**

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Abstract

Trichomonas vaginalis is a pear-shaped organism that propels itself with four whip-like flagella that protrude from its front end. Trichomonads participate in a host-parasite relationship, causing them to adhere to epithelial cells. *Trichomonas vaginalis* has been described as a common cosmopolitan parasite of both males and females. The paper reviewed *Trichomonas vaginalis* from history, prevalence, complications, diagnosis to treatment.

Keywords: *Trichomonas vaginalis*, history, prevalence, complications, Diagnosis, treatment

Trichomonas vaginalis

Trichomonas vaginalis is a pear-shaped organism that propels itself with four whip-like flagella that protrude from its front end. A fifth flagellum, attached to an undulating membrane, extends rearward (Heine and McGregor, 1993) A barbed tail, called an axostyle, projects from the trailing end of *T. vaginalis*. It is believed that trichomonads attach themselves to tissue with their axostyles, and that this causes some of the irritation and inflammation associated with a trichomoniasis infection (Petrin *et al.*, 1998). Trichomonads vary in size from approximately 5 to 20 micrometers wide. In a wet mount preparation of vaginal secretions, the live

organism can often be recognized by its motion, which has been described as jerky, swaying, or tumbling. *T. vaginalis* is anaerobic, and it grows best in oxygen-free, low acidity environments. Maximum growth and metabolic function is achieved at a pH of 6.0 (Benchimol, 2004). Two other species of trichomonads infect humans: *T. tenax* and *Pentatrichomonas hominis*. Although there is some debate about the role these species may play in urogenital infection, a recent study found no evidence linking either to vaginal trichomoniasis in humans (Adu-Sarkodie *et al.*, 2007).

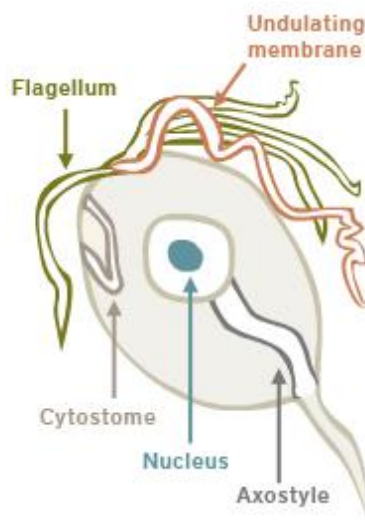


FIG 1: Trophozoite of *Trichomonas vaginalis* (Pereira-Neves *et al.*, 2003)

Trichomonads multiply via binary fission. The *Trichomonas* species exist only as trophozoites and do not take on a cyst form, but recent research has suggested that under unfavorable conditions they may assume a pseudocyst form (Pereira-Neves *et al.*, 2003). *T. vaginalis* is a primitive eukaryotic organism that is in most respects similar to other eukaryotes, but its energy metabolism bears a stronger resemblance to that of anaerobic bacteria. In January 2007, researchers funded by The Institute for Genomic Research published a draft sequence of *T. vaginalis*' genome. We now know that the organism's genome is among the largest on record—approximately the size of the human genome—and that it contains a large number of repeated or transposable genes (Carlton, 2007). The insights gained through this effort may someday lead to improved diagnostic methods and treatment protocols.

History of *Trichomonas vaginalis*

The first trichomonad species, *Trichomonas tenax*, was identified in 1773 by Miller, who found it in a culture of dental calculus (Spence, 1992). *T. tenax* has been associated with gingivitis. It is uncertain if the inflammatory process is attributed to *T. tenax* or if the organism simply inhabits the area (Thomason and Gelbart, 1989). Donné first discovered and named *Trichomonas vaginalis* in 1836 after finding the

the organism in genital secretions of both women and men. *T. vaginalis*' pathogenicity was initially thought to be non-pathogenic because a majority of infected patients were asymptomatic. The development of culture medium in the 1940s allowed more detailed study of the organism and its pathogenicity (Hesseltine, 1942).

In 1942, Hesseltine inoculated 70 pregnant volunteers with unadulterated *T. vaginalis* culture (10,000 to 120,000 trichomonads per inoculum.) Fifty-three patients were tested for trichomoniasis using vaginal smear and 17 received clinical observation. Seven of the 53 patients (13%) developed trichomoniasis shortly after inoculation. Two additional patients who tested negative for trichomoniasis prior to giving birth tested positive after giving birth (Hesseltine, 1942). In 1953, Lanceley inoculated five male volunteers with 2 ml of *T. vaginalis* culture (2 million protozoa per ml) and another five with a control sample. Three of the five volunteers inoculated with culture developed urethritis from which *T. vaginalis* organisms were removed (Lanceley and McEntegart, 1953). *Pentatrichomonas hominis* (formerly *Trichomonas hominis*) was identified by Davaine in 1854 in human stool samples. *P. hominis* is found in the large intestine and has been associated with diarrheal problems. Like *T. tenax*, the pathogenicity is unknown.

The inoculation of *T. tenax* and *P. hominis* into the vagina generally does not produce infection. Recently, however, researchers using PCR reported the discovery of *P. hominis* in the vaginas of two women (Crucitt *et al.*, 1991). The women tested negative for *T. vaginalis* and showed no symptoms. Likewise, although *T. vaginalis* in the mouth or intestines was not thought to foster infection, there have been several recent reports to the contrary (Rein and Muller, 1990). *T. tenax* is known to cause broncho-pulmonary infections in patients with pre-existing pulmonary or debilitating disease (Stratakis *et al.*, 1999). In 2003, Duboucher

reported finding numerous *T. vaginalis* organisms in the lungs of a 41-year-old man infected with HIV (Duboucher *et al.*, 2003). A number of trichomonad species infect animals. In cattle, *Tritrichomonas foetus* is a highly prevalent sexually transmitted infection that causes infertility, embryonic death, and abortion (Rae *et al.*, 2004). In cats and dogs, *T. foetus* and *Pentatrichomonas hominis* cause chronic diarrhea (Foster *et al.*, 2004; Gookin *et al.*, 2005). *Tetratrichomonas gallinarum* and *Trichomonas gallinae* are responsible for a wide range of diseases in birds.

Life cycle of *Trichomonas vaginalis*

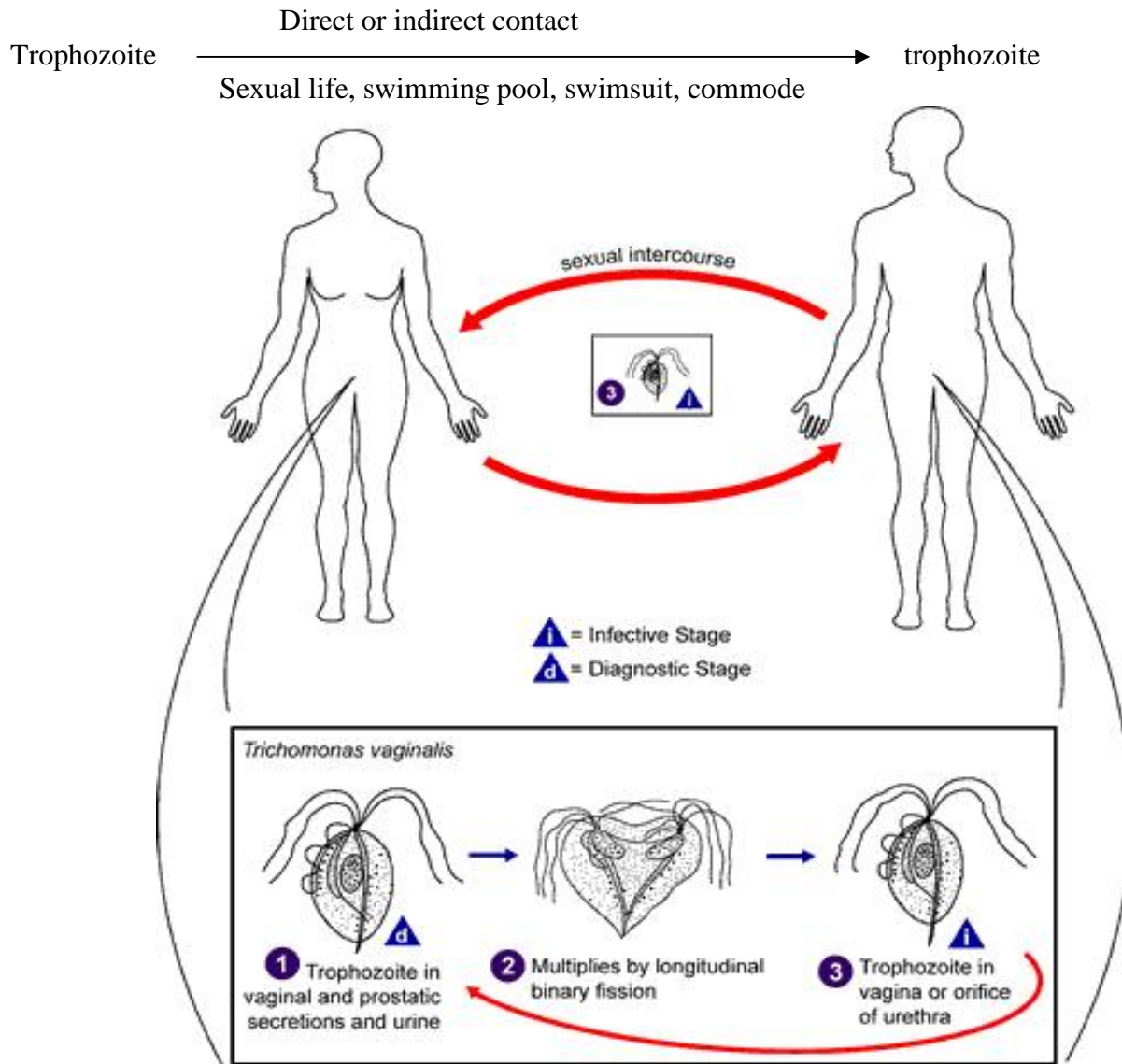


Fig 2: Life cycle of *Trichomonas vaginalis*

Source:(Pereira-Neves *et al.*, 2003).

Trichomonas vaginalis resides in the female lower genital tract and the male urethra and prostate ①, where it replicates by binary fission ②. The parasite does not appear to have a cyst form, and does not survive well in the external environment. *Trichomonas vaginalis* is transmitted among humans, its only known host, primarily by sexual intercourse ③.

Pathogenesis of *Trichomonas vaginalis*

The pathogenicity of *T. vaginalis* is not thoroughly understood. Trichomonads participate in a host-parasite relationship, causing them to adhere to epithelial cells. The ability of trichomonads to adhere is affected by time, temperature, and pH level. *T. vaginalis* grows best in an anaerobic environment with a pH > 6 (Diamond, 1986).

Binding of *T. vaginalis* to vaginal epithelial cells for colonization and infection is dependent upon specific parasite surface proteins. Parasites treated with tinidazole, metronidazole or other nitroimidazoles lose their ability to adhere, making them ineffective disease agents. Hemolysis, the destruction of red blood cells such that hemoglobin is released, is also correlated with virulence. Pathogenicity of trichomonas vaginalis has been seen in Trichomoniasis which has been seen to increase in severity during or slightly after menstruation (Graves and Gardner, 1993). The relationship between *T. vaginalis* growth and protective lactobacilli is a complex one. It is currently unknown whether *T. vaginalis* infection alters the vaginal environment by creating an anaerobic situation, or if anaerobes in the vagina precede *T. vaginalis* growth. The vagina contains glycogen, and vaginal glycogen levels are elevated in women of reproductive age. Glycogen is broken down into glucose, a nutrient *T. vaginalis* requires for growth. In women, *T. vaginalis* may be found in the vagina and the exterior cervix in over 95 per cent of infections, but is only recovered from the endocervix in 13 per cent. The urethra and Skene's glands are also very commonly infected. Dissemination beyond the lower urogenital tract is extremely rare and is

not found even in severely immunocompromised patients. In men the urethra is the most common site of infection, but the organism has also been recovered from epididymal aspirates (Krieger, 1981). Epidemiological studies have recently linked trichomoniasis in women with a modest increase in the risk of human immune deficiency infection via sexual intercourse and with adverse pregnancy outcome and have suggested that it might be the actual cause of a few per cent of cases of cervical neoplasia (Petrin *et al.*, 1998).

Epidemiology of *Trichomonas vaginalis*

Despite the difficulty often experienced in isolating the organism from male contacts of infected women, all epidemiological evidence suggests that the vast majority of infections are sexually acquired. Few studies have been made of genuinely unselected populations, but it has recently been estimated that, world-wide, 170 million new infections occur each year. Most surveys have examined either pregnant women or those attending STD clinics; there are wide national variations, but most report 10–25 per cent infected, although the full range is 0–63 per cent. In most clinical surveys female cases outnumber male 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 industrialized nations.

Prevalence of *Trichomonas vaginalis*

Trichomonas vaginalis has been described as a common cosmopolitan parasite of both males and females. Approximately 180 million people are infected worldwide yearly, while in the United States of America, 5 million women and 1 million men are infected annually. Incidence rates of 5% have been reported in asymptomatic patients attending family planning clinics, 50–75% in Commercial Sex Workers, and 0.9 to 39.6% in sexually transmitted infection clinics (Bowden, and Garnett, 2000).

Trichomonas vaginalis is shown to be the most common parasite causing curable sexually transmitted disease. It is more common than both chlamydia and gonorrhea. It is estimated that in the United States 7.4 million new cases of trichomoniasis appear annually compared with 3 million cases of chlamydia and 718,000 cases of gonorrhea (Weinstock *et al.*, 2004)

In late 2007, researchers with the CDC reported a 3.1% prevalence of *T. vaginalis* infection in a sample of 3,754 women aged 14-49 (Sutton *et al.*, 2007). The prevalence of trichomoniasis in women varies greatly depending on the population studied. A study of women reporting to a college clinic for routine reproductive care appointments found a trichomoniasis prevalence of 4.8% (Thornton *et al.*, 2003). Several studies have shown a much higher prevalence of infection (10-18.5%) among young women living in urban areas and the prevalence in inner city STD Clinics is typically close to 25% (Ohlemeyer *et al.*, 1998). Men are not diagnosed with trichomoniasis as frequently as women. Two primary reasons for this are that the symptoms of *Trichomonas* infection are less pronounced in men, and the detection of infection is more complicated. Studies of male STD clinic patient populations have reported prevalence between 11 and 17% (Hobbs *et al.*, 2006). The prevalence of trichomoniasis among male sexual partners of infected women is over 73% (Schwebke and Burgess, 1994)

The aforementioned CDC study showed a large racial disparity among women infected with *T. vaginalis*. The prevalence of trichomoniasis among non-Hispanic black women was 10.3 times higher than that of non-Hispanic white or Mexican American women (13.3% vs. 1.3% and 1.8%, respectively) (Sutton *et al.*, 2007).

Prevalence of *Trichomonas vaginalis* among adolescents

Of approximately 18.9 million new STD cases in the United States in the year 2000, it has been estimated that 9.1 million were among people between 15 and 24 years old, and that

trichomoniasis represented 1.9 million of these cases (Weinstock *et al.*, 2004).

Sutton (2007) found that on a national level the prevalence of trichomoniasis increases with age (Soper, 2004). Studies of smaller segments of the population, however, have shown a particularly high prevalence of trichomoniasis among low income, African American females living in urban areas (Sutton *et al.*, 2007).

Factors that have been associated with increased *T. vaginalis* infection rates among adolescent females include:

- Confirmed marijuana use
- Typical sex partners being at least five years older
- Sex with non-steady partners
- History of delinquency

Causes of *Trichomonas vaginalis* infection

Trichomonas vaginalis is a tiny parasite which causes an infection. In women the infection can be found in the vagina and the urethra (tube where urine comes out). In men it can be found in the urethra. The infection is easily passed from one person to another through sexual contact. Anyone who is sexually active can get it and pass it on (Wiesenfeld *et al.*, 2001). The *Trichomonas vaginalis* parasite is spread sexually by penis-to-vagina intercourse or vulva-to-vulva (the genital area outside the vagina) contact with an infected partner. Women can get the disease from infected men or women, but men usually only get it from infected women. A common misbelief is that infection can be spread by toilet seats, wet towels or hot tubs. This is not likely, since the parasite cannot live long on objects and surfaces. Sometimes antibiotics, birth control pills, hormones, and douching can cause vaginal irritation and lead to infection. Other causes may be tight-fitting clothes, intercourse without enough lubrication, childbirth, or cuts on the vagina.

Sites of *Trichomonas vaginalis* infection

T. vaginalis infection is generally confined to the urogenital tract. There have, however, been rare reports of trichomonads being found in other sites

such as the lungs and cerebrospinal fluid. These cases have usually been accompanied by a severe underlying disease. Rarely have the organisms been identified as *T. vaginalis*, but were most likely *T. tenax* or *T. hominis* (Rein and Muller, 1990).

A case study published in 2003 tells of a 41 year old HIV+ male who was hospitalized due to fever and dyspnea. A cytologic examination of his bronchoalveolar lavage fluid revealed numerous *T. vaginalis* organisms. This is the first case where *T. vaginalis* was found in the lungs of an adult. Data collected suggest that trichomonads are overlooked parasites and may be implicated in various human pathologies (Duboucher *et al.*, 2003).

Women

T. vaginalis organisms may be isolated from the cervix, vagina, Bartholins glands, bladder, urethra and occasionally the upper reproductive/urinary tract.¹ Over 95% of infections have been isolated from the vagina and only 5% from the urinary tract of adult women (Grys , 1964). The urethra and Skene's glands are infected in 90% of cases. There have also been instances where organisms were isolated from bladder urine (Thomason and Gelbart, 1989)

T. vaginalis may also act as a carrier for other pathogenic organisms. Keith conducted an in vitro study in 1986 to observe the attachment between *T. vaginalis* and other bacteria that inhabit the urogenital areas. Using scanning electron microscopy, one finding displayed a cluster of cocci attached to a trichomonad and two other demonstrated multiple cocci and *E. coli* attached to a *T. vaginalis* organism. Trichomonads have been shown to migrate to the fallopian tubes and peritoneal cavity. Thus, by carrying bacteria or viruses on their surfaces, it is possible that *T. vaginalis* organisms contribute to upper genital tract infections (Keith *et al.*, 1986)

In 2003, Rendón-Maldonado identified bacterial and protozoal STDs as important factors in the epidemiology of HIV-1. The research team incubated three subtypes of HIV-1 (A, B, and D)

with HIV-1 infected lymphocytes and observed the interactions with immunofluorescence microscopy and transmission electron microscopy. Results showed that trichomonads may internalize HIV-1 particles for a short time period. Under in vitro conditions, trichomonads ingest and digest HIV-1 infected lymphocytes (Rendón-Maldonado *et al.*, 2003).

T. vaginalis may actually transmit viruses. Pindak (1989) found virus-containing cell fragments engulfed by trichomonads and internalized in vacuoles. Viable reoviruses were recovered from the trichomonads for nine days and genital herpes simplex virus for six days, suggesting the possibility of transmission of viruses by *T. vaginalis* (Pindak *et al.*, 1989). More recent findings have further implicated *T. vaginalis* as host to multiple viruses, simultaneously (Benchimol *et al.*, 2002)

Men

In men the urethra is the most common site for *T. vaginalis* infection. Organisms can also be detected in the epididymis, semen, and urine (Krieger, 1981). *T. vaginalis* was first located in prostatic fluid in 1936 by Drummond, who examined prostatic secretions from husbands of infected women (Drummond, 1936). The organism has since been identified as a cause of prostatitis (Guenthner *et al.*, 2005; Gardner *et al.*, 1986).

Risk factors associated with Trichomonas infections

Several risk factors for the acquisition of trichomoniasis have been identified, including multiple sexual partners, black race, history of previous STD and coexistent infection with *Neisseria gonorrhoeae* (Sobel, 1997). Approximately 8% to 50% of patients with *T. vaginalis* have concomitant infections. After conducting PCR analysis on the vaginal swabs of a nationally representative sample of 3,754 women, Sutton (2007) reported that the following factors are associated with an increased likelihood of *T. vaginalis* infection:

- Being of non-Hispanic black ethnicity
- Being born in the United States

- Increased number of sex partners
- Increased age
- Lower education
- Poverty
- Douching (Sutton *et al.*, 2007)

Numerous studies have identified previous infection with an STD as a risk factor for infection with trichomoniasis (Sobel, 1997; Niccolai *et al.*, 2000). In a study of 1,236 female STD clinic patients, Peterman (2006) reports that 16.5% of women treated for trichomoniasis were reinfected within three months. Interestingly, 14.8% of patients who had gonorrhea at their first visit had trichomoniasis at their three-month follow-up, but only 3.6% had reacquired gonorrhea (Peterman *et al.*, 2006)

Transmission of *Trichomonas vaginalis*

Trichomoniasis is a sexually transmitted disease. Nonsexual transmission is extremely rare since *T. vaginalis* infection is generally confined to the urogenital tract (Martin *et al.*, 1999). Asymptomatically infected individuals are an important vector and act as a stealth factor in trichomoniasis transmission. More than 70% of male partners of infected females are also infected. The incubation period before symptoms of trichomoniasis arise is 4-28 days in approximately 50% of infected women. Many studies have shown that treatment of the male partner(s) of infected women improves both cure rates and recurrence rates. The CDC's STD treatment guidelines advise that sex partners of patients with *T. vaginalis* should be treated. Live *T. vaginalis* organisms in urine and semen samples have been found after being exposed to air for several hours. Although organisms are able to survive for hours on damp towels and clothes of infected women (CDC, 2006). There have been no well-documented cases regarding transmission through these means. The only known nonvenereal form of transmission is from a mother to her child during delivery. Approximately 5% of female babies born of infected mothers contract the infection (Schwebke and Burgess, 2004).

Health complications of *T.vaginalis* infection in women

Although infection with *Trichomonas vaginalis* parasite had long been regarded as a sexually transmitted infection of minor importance, evidence recently accumulated implicates *Trichomonas vaginalis* as a contributor to a variety of adverse outcomes in both women and men (Soper, 2004).

Risk Factor for HIV

T. vaginalis has been associated with amplifying transmission and acquisition of HIV infection (Sorvillo and Kerndt, 1998). Treatment of women infected with *T. vaginalis* resulted in a 4.2-fold reduction in the quantity of HIV-1 in vaginal secretions (Wang *et al.*, 2001)

Associated with Herpes Simplex Virus-2 Acquisition

Incident trichomoniasis was an independent predictor of herpes simplex virus-2 incidence; women with trichomoniasis were almost 4 times as likely to acquire HSV-2 infection (Gottlieb *et al.*, 2004).

Contributor to Female Infertility

Trichomonads may serve as vectors for spread of other organisms by carrying these pathogens up the fallopian tubes (Keith *et al.*, 1986). Several studies have shown *T. vaginalis* to be a risk factor for tubal infertility (Grodstein *et al.*, 1993)

Pelvic Inflammatory Disease (PID)

A significantly higher rate of pelvic inflammatory disease among women with trichomoniasis has been shown compared with uninfected women (Moodley *et al.*, 2002). A separate study demonstrated that women colonized with *Chlamydia* and *Trichomonas* were significantly more likely to have symptomatic upper tract disease (Paisarntantiwong *et al.*, 1995)

Cervical Neoplasia

Infection with *T. vaginalis* has been associated with a 2-fold increased risk of cervical neoplasia, even after controlling for human papillomavirus (HPV) infection (Gram *et al.*, 1992).

Preterm Birth

Pregnancy complications such as preterm labor and delivery, as well as low birth weight infants have been associated with *T. vaginalis* infection in several studies. Treatment of asymptomatic trichomoniasis in pregnancy is controversial (Diclemente *et al.*, 2004).

Health complications of *T.vaginalis* infection in men

Risk Factor for HIV

Among HIV positive men with symptomatic *Trichomonas* urethritis, there was a 6-fold increase in HIV concentration in semen compared to HIV positive men without *Trichomonas*. Treatment of HIV infected men with *T. vaginalis* resulted in reduced excretion of HIV in semen (Krieger *et al.*, 1993).

Contributor to Male Infertility

Among men infected with *Trichomonas*, there was a significant decrease in both sperm motility and viability versus control subjects. Treatment of *Trichomonas* showed significant improvements in sperm motility, viability, viscosity and particulate matter (Gopalkrishnan *et al.*, 1990)

Nongonococcal Urethritis (NGU)

Trichomoniasis may be an important cause of nongonococcal urethritis. A recent study found that in men with NGU, 19.9% were infected with *Trichomonas* (Schwebke and Hook, 2003). The Centers for Disease Control STD treatment guidelines recommend inclusion of *Trichomonas* therapy for men with recurrent NGU (CDC, 2006).

Chronic Prostatitis

A recently study involving men with chronic prostatitis found that in 71% of the men the cause of prostatitis was infective trichomoniasis with specific infection in 19% of the men (Skerk *et al.*, 2002)

Symptoms of *Trichomonas vaginalis* infection

Women who have trichomoniasis may notice a frothy, yellow-green vaginal discharge with a strong, foul odor. There may be soreness, itching and irritation of the genital area, urination or sexual intercourse may be uncomfortable or painful. Symptoms are not consistent among women. Symptoms usually appear in women within 5 to 28 days of exposure, but up to 50% of women may not show any symptoms (Weston and Nicol, 1963). If not treated within 6 months, however, 30% of infected asymptomatic women will develop symptoms (Rein and Muller, 1990).

Common clinical signs of infection include:

- Vulvar erythema (redness)
- Inflammation
- Excess of white blood cells seen on a wet mount preparation of vaginal discharge
- Motile trichomonads in the wet mount preparation
- Vaginal pH above 5

Most of these symptoms overlap with those of bacterial vaginosis, complicating diagnosis.

Symptoms *Trichomonas vaginalis* infection in Men

Most men with trichomoniasis do not show symptoms of infection, and those who seek treatment typically do so because of an infected partner.

Symptoms in men typically include urethral discharge, pain during urination, mild local itching, and burning after sexual intercourse. *T. vaginalis* is also a known cause of prostatitis (Guenthner *et al.*, 2005; Gardner *et al.*, 1986). Men who are unresponsive to antimicrobial therapy for nonspecific urethritis should be tested for *T. vaginalis* since 15-20% will be infected with the organism (Thomason and Gelbart, 1989, Schwebke and Hook, 2003).

Diagnosis of *Trichomonas vaginalis*

The most commonly used diagnostic test is at best 60-70% sensitive, according to the CDC (Lossick and Kent, 1991).

For both women and men, a health care provider must perform a physical examination and laboratory test to diagnose the presence of *Trichomonas vaginalis*



FIG 3: Two *T. vaginalis* trophozoites obtained from *in vitro* culture.
Source: (Soper, 2004).

CDC Division of Parasitic Diseases

To diagnose trichomoniasis in a woman, her health care provider will first look at her vagina and vaginal discharge. Normal discharge is usually clear, but in trichomoniasis it may appear yellow or greenish in color. The discharge may be tested for abnormal or foul odor using a potassium hydroxide (KOH) "whiff test," and its acidity may be checked. If trichomoniasis is suspected, further tests will be conducted. Diagnostic methods range from simple visual detection under a microscope to polymerase chain reaction (DNA analysis.) Each method has its advantages, but no single method is ideal (CDC, 2006).

Wet Mount

For a wet mount, a microscope slide is prepared by suspending a specimen in saline solution. The slide is then visually examined for trichomonads. Wet mount is the most common method used to

diagnose trichomoniasis. It is at best 60% sensitive, however, and is best used as a screening tool.

Culture/InPouch™ TV (BioMed Diagnostics)

For this method, a specimen is placed in a culture medium for 2-7 days before it is examined. If trichomonads are present in the original specimen, they will multiply while in culture and be easier to detect. Culture is considered the gold standard for the diagnosis of trichomoniasis. It is both highly sensitive and highly specific. Its disadvantages include cost and prolonged time before diagnosis. BioMed Diagnostics' InPouch TV system is a two-chambered bag that allows one to perform a wet mount using the upper chamber and a culture using the lower chamber. The wet mount's fast results allow some patients to begin treatment without having to wait, untreated, for the results of the more sensitive culture (Smith *et al.*, 2001).

Affirm™ VPIII Microbial Identification Test (BD)

BD's Affirm VPIII Test is a moderately complex DNA probe for vaginitis. Identification of *Candida* (yeast) species, *Gardnerella vaginalis*, and *Trichomonas vaginalis* is possible from a single vaginal sample. The test's sensitivity for detecting *T. vaginalis* is high and it can provide results in as little as 45 minutes.

OSOM® Trichomonas Rapid Test (Genzyme Diagnostics)

Genzyme Diagnostics' OSOM Trichomonas Rapid Test is a new point-of-care, antigen-detecting diagnostic test for trichomoniasis. By inserting a vaginal swab into a test tube with 0.5 mL of a special buffer, mixing the solution vigorously by hand, removing the swab, and then inserting a test strip, physicians and staff can read results in 10 minutes. The OSOM test is more sensitive than wet mount (Huppert *et al.*, 2007).

Polymerase Chain Reaction

In polymerase chain reaction (PCR), sample is treated with enzymes that amplify specific regions

of *T. vaginalis*' DNA. After amplification, the numbers of DNA fragments are quantified. PCR has proven to be the most accurate diagnostic method in recent studies. PCR is currently only used in research, not clinical settings (Wendel *et al.*, 2002).

Other Tests

Potassium Hydroxide (KOH) "Whiff Test"

The "whiff" test is a rudimentary technique that may be used as part of a clinical diagnosis. The test is conducted by mixing a swab of vaginal fluid with a 10% potassium hydroxide solution, then smelling it. A strong amine (fishy) smell could be an indication of trichomoniasis or bacterial vaginosis (Gelbart, 1989).

Vaginal pH Test

Trichomonads grow best in less acidic environments, and elevated vaginal pH may be an indication of trichomoniasis. A healthcare provider performs the test by touching pH paper to the vaginal wall or to a vaginal swab specimen, then comparing it to color scale to determine the pH (Lossick and Kent, 1991).

Papanicolaou test (Pap smear)

The Papanicolaou test is a microscopic examination of a stained specimen. It is mainly used as a diagnostic test for the screening of various cervical abnormalities and genital infections. While it may occasionally detect trichomonads, it has a high diagnostic error rate and is not suitable for screening unless used in conjunction with a more sensitive test (Lobo *et al.*, 2003).

Treatment of *Trichomonas vaginalis* infections

Trichomonas vaginalis infections can usually be cured in both women and men with a prescription drug given by mouth.

According to the CDC's STD Treatment Guidelines, the drugs of choice for trichomoniasis in both men and women are a 2 gram dose of

either oral metronidazole or tinidazole. An alternative regimen of metronidazole is suggested at 500 mg, twice a day, for seven days (CDC, 2006).

Intravaginal treatment with metronidazole vaginal gel has been shown to be ineffective and is not recommended.

Medicines that will cure infections with *trichomonas vaginalis* include

- Tinidazole (Tindamax[®])
- Metronidazole

Prevention of infections caused by *Trichomonas vaginalis*

Infections caused by *Trichomonas vaginalis* can be prevented by the following:

Use a latex condom properly every time you have sexual intercourse with every partner. Always use water-based lubricants (like Maxilube[®], K-Y[®] Jelly, or Astroglide[®]) with latex condoms. Do not use oil lubricants like petroleum jelly, baby oil or cooking oil because they can cause latex condoms to break.

Know your partner's sexual history. The more people that you or your partner have had sex with, the greater your risk of encountering someone who has this or other STDs.

Other than abstaining from sex altogether, the best way to avoid transmission of sexually transmitted diseases is to be in a long-term, mutually monogamous relationship (no other sex partners for you or your partner) with a partner who has been tested and you know is not infected.

Conclusion

Trichomonas vaginalis is a pear-shaped organism that propels itself with four whip-like flagella that protrude from its front end. Trichomonads participate in a host-parasite relationship, causing them to adhere to epithelial cells. *Trichomonas vaginalis* has been described as a common

cosmopolitan parasite of both males and females. Although infection with *Trichomonas vaginalis* parasite had long been regarded as a sexually transmitted infection of minor importance, evidence recently accumulated implicates *Trichomonas vaginalis* as a contributor to a variety of adverse outcomes in both women and men. It is associated Risk Factor for HIV, Associated with Herpes Simplex Virus-2 Acquisition, Contributor to Female Infertility, Pelvic Inflammatory Disease (PID), Cervical Neoplasia, Preterm Birth and also complications in men.

References

- Adu-Sarkodie, Y., Opoku, B.K., Crucitti, T., Weiss, H.A. and Mabey, D. (2007): Lack of evidence for the involvement of rectal and oral trichomonads in the aetiology of vaginal trichomoniasis in Ghana. *Sex Transm Infect.* 83(2):130-132.
- Benchimol, M. (2004): Trichomonads under microscopy. *Microsc Microanal.* 10(5):528-550.
- Benchimol, M., Chang, T.H. and Alderete, J.F. (2002): Trichomonas vaginalis: observation of coexistence of multiple viruses in the same isolate. *FEMS Microbiol Lett.* 215(2):197-201.
- Bowden, F.J. and Garnett, G.P. (2000): *Trichomonas vaginalis* epidemiology: parameterising and analyzing a model of treatment interventions. *Sex Transm Infect.*, 76: 248-256.
- Carlton, J.M. (2007): Draft genome sequence of the sexually transmitted pathogen *Trichomonas vaginalis*. *Science.* 315 (5):207-212.
- Centers for Disease Control and Prevention (2006): Sexually Transmitted Diseases Treatment Guidelines. *MMWR.* 55(11):1-94.
- Crucitti, T., Abdellati, S., Ross, D.A., Chagalucha, J., Dyck, E. and Buve, A. (1991): Detection of Pentatrachomonas hominis DNA in biological specimens by PCR. *Int J Fertil.* 42: 212-217.
- Diamond, L.S. (1986): In vitro cultivation of the trichomonadide: a state of the art review. *Acta Univ Caroline - Biol.* 30: 221-228.
- Diclemente, R.J., Wingood, G.M., Crosby, R.A., Rose, E., Lang, D., Pillay, A., Papp, J. and Faushy, C. (2004): A descriptive analysis of STD among urban pregnant African-American teens: data from a pilot study. *J Adolesc Health.* 34(5):376-83.
- Drummond, A.C. (1936): Trichomonas Infection of the prostate gland. *Am J Surgery.* 31(1):98-103.
- Duboucher, C., Noël, C., Durand-Joly, I., Gerbod, D., Delgado-Viscogliosi, P., Jouveshomme, S., Leclerc, C., Cartolano, G.L., Dei-Cas, E., Capron, M. and Viscogliosi, E. (2003): Pulmonary coinfection by *Trichomonas vaginalis* and *Pneumocystis* sp. as a novel manifestation of AIDS. *Hum Pathol.* 34(5): 508-511.
- Foster, D.M., Gookin, J.L., Poore, M.F., Stebbins, M.E. and Levy, M.G. (2004): Outcome of cats with diarrhea and Tritrichomonas foetus infection. *J Am Vet*
- Gardner, W.A. Jr., Culberson, D.E. and Bennett, B.D. (1986): *Trichomonas vaginalis* in the prostate gland. *Arch Pathol Lab Med.* 1986. 110(5):430-432.
- Gelbart, S.M. (1989): *Trichomonas vaginalis*. *Obstet Gynecol.* 74(3): 536-541.
- Gookin J.L., Birkenheuer, A.J., St John, V., Spector, M. and Levy, M.G. (2005): Molecular characterization of trichomonads from feces of dogs with diarrhea. *J Parasitol.* 91(4): 939-943.
- Gopalkrishnan, K., Hinduja, I.N. and Kumar, T.C. (1990): Semen characteristics of asymptomatic males affected by *Trichomonas vaginalis*. *J In Vitro Fert Embryo Transf.* 7(3):165-167.
- Gottlieb, S.L., Douglas, J.M. Jr., Foster, M., Schmid, D.S., Newman, D.R., Baron, A.E., Bolan, G., Iatesta, M., Malotte, C.K., Zenilman, J., Fishbein, M., Peterman, T.A. and Kamb, M.L. (2004): Incidence of herpes simplex virus type 2 infection in 5 sexually transmitted disease (STD) clinics and the effect of HIV/STD risk-reduction counseling. *J Infect Dis.* 190(6):1059-1067.
- Gram, I.T., Macaluso, M., Churchill, J. and Stalsberg, H. (1992): *Trichomonas vaginalis* (TV) and human papillomavirus (HPV) infection and the incidence of cervical

- intraepithelial neoplasia (CIN) grade III. *Cancer Causes Control.* 3(3):231-236.
- Graves, A. and Gardner, W.A. Jr. (1993): Pathogenicity of *Trichomonas vaginalis*. *Clin Obstet Gynecol.* 36(1): 145-152.
- Grodstein, F., Goldman, M.B. and Cramer, D.W. (1993): Relation of tubal infertility to history of sexually transmitted diseases. *Am J Epidemiol.* 137(5): 577-584.
- Grys, E. (1964): Topography of trichomonadosis in the reproductive organ of the woman. *Wiad Parazytol.* 122-124.
- Guenther, P.C., Secor, W.E. and Dezzutti, C.S. (2005): *Trichomonas vaginalis*-induced epithelial monolayer disruption and human immunodeficiency virus type 1 (HIV-1) replication: implications for the sexual transmission of HIV-1. *Infect Immun.* 73(7):455-460.
- Heine, P., and McGregor, J.A. (1993): *Trichomonas vaginalis*: a reemerging pathogen. *Clin Obstet Gynecol.* 36(1): 137-144.
- Hesseltine, H. (1942): Experimental human vaginal trichomoniasis. *J Infect Dis.* 71:127.
- Hobbs, M.M., Lapple, D.M., Lawingm, L.F., Schwebke, J.R., Cohen, M.S., Swygard, H., Atashili, J., Leone, P.A., Miller, W.C. and Seña, A.C. (2006): Methods for detection of *Trichomonas vaginalis* in the male partners of infected women: implications for control of trichomoniasis. *J Clin Microbiol.* 44(11): 394-399.
- Huppert, J.S., Mortensen, J.E., Reed, J.L., Kahn, J.A., Rich, K.D., Miller, W.C. and Hobbs, M.M. (2007): Rapid antigen testing compares favorably with transcription-mediated amplification assay for the detection of *Trichomonas vaginalis* in young women. *Clin Infect Dis.* 45(2):194-198.
- Keith, L.G., Friberg, J., Fullan, N., Bailey, R. and Berger, G.S. (1986): The possible role of *Trichomonas vaginalis* as a "vector" for the spread of other pathogens. *Int J Fertil.* 31(4):272-277.
- Krieger, J.N. (1981): Urologic aspects of trichomoniasis. *Invest Urol.* 18(6):411-417.
- Krieger, J.N., Jenny, C., Verdon, M., Siegel, N., Springwater, R., Critchlow, C.W. and Holmes, K.K. (1993): Clinical manifestations of trichomoniasis in men. *Ann Intern Med.* 118(11):844-849.
- Lanceley, F. and McEntegart, M.G. (1953): *Trichomonas vaginalis* in the male; the experimental infection of a few volunteers. *Lancet.* 1(14):668-671.
- Lobo, T.T., Feijó, G., Carvalho, S.E., Costa, P.L., Chagas, C., Xavier, J. and Simoes-Barbosa, A. (2003): A comparative evaluation of the Papanicolaou test for the diagnosis of trichomoniasis. *Sex Transm Dis.* 30(9): 694-699.
- Lossick, J.G. and Kent, H.L. (1991): Trichomoniasis: trends in diagnosis and management. *Am J Obstet Gynecol.* 165(4): 217-222.
- Martin, H.L., Richardson, B.A., Nyange, P.M., Lavreys, L., Hillier, S.L., Chohan, B., Mandaliya, K., Ndinya-Achola, J.O., Bwayo, J. and Kreiss, J. (1999): Vaginal lactobacilli, microbial flora, and risk of human immunodeficiency virus type 1 and sexually transmitted disease acquisition. *J Infect Dis.* 180(6):863-868.
- Moodley, P., Wilkinson, D., Connolly, C., Moodley, J. and Sturm, A.W. (2002): *Trichomonas vaginalis* is associated with pelvic inflammatory disease in women infected with human immunodeficiency virus. *Clin Infect Dis.* 34(4):519-522.
- Niccolai, L.M., Kopicko, J.J., Kassie, A., Petros, H., Clark, R.A., Kissinger, P. (2000): Incidence and predictors of reinfection with *Trichomonas vaginalis* in HIV-infected women. *Sex Transm Dis.* 27(5):284-288.
- Ohlemeyer, C.L., Hornberger, L.L., Lynch, D.A. and Swierkosz, E.M. (1998): Diagnosis of *Trichomonas vaginalis* in adolescent females: InPouch TV culture versus wet-mount microscopy. *J Adolesc Health.* 22(3):205-208.
- Paisarntantiwong, R., Brockmann, S., Clarke, L., Landesman, S., Feldman, J. and Minkoff, H. (1995): The relationship of vaginal trichomoniasis and pelvic inflammatory disease among women colonized with *Chlamydia trachomatis*. *Sex Transm Dis.* 22(6):344-347.
- Pereira-Neves, A., Ribeiro, K.C. and Benchimol, M. (2003): Pseudocysts in trichomonads- new insights. *Protist.* 154(3):313-329.

- Peterman, T.A., Tian, L.H., Metcalf, C.A., Satterwhite, C.L., Malotte, C.K., DeAugustine, N., Paul, S.M., Cross, H., Rietmeijer, C.A. and Douglas, J.M. Jr. (2006): High incidence of new sexually transmitted infections in the year following a sexually transmitted infection: a case for rescreening. *Ann Intern Med.* 145(8): 564-572..
- Petrin, D., Delgaty, K., Bhatt, R. and Garber G. (1998): Clinical and microbiological aspects of *Trichomonas vaginalis*. *Clin Microbiol Rev.* 11(2): 300-317.
- Pindak, F.F., Mora de Pindak, M., Hyde, B.M., Gardner, W.A. Jr. (1989): Acquisition and retention of viruses by *Trichomonas vaginalis*. *Genitourin Med.* 65(6):366-371.
- Rae, D.O, Crews, J.E, Greiner, E.C. and Donovan, G.A. (2004): Epidemiology of *Trichomonas foetus* in beef bull populations in Florida. *Theriogenology.* 61(4): 605-618.
- Rein, M.F. and Muller, M. (1990): *Trichomonas vaginalis* and trichomoniasis. In: Holmes, K.K. (ed). Sexually Transmitted Diseases. New York: McGraw Hill. Pp. 481-492.
- Rein, M.F. and Muller, M. (1990): *Trichomonas vaginalis* and trichomoniasis. In: Holmes, K.K. (ed.) Sexually Transmitted Diseases. McGraw Hill, New York. Pp. 481-492.
- Rendón-Maldonado, J., Espinosa-Cantellano, M., Soler, C., Torres, J.V. and Martínez-Palomo, A. (2003): *Trichomonas vaginalis*: in vitro attachment and internalization of HIV-1 and HIV-1-infected lymphocytes. *J Eukaryot Microbiol.* 50(1): 43-48.
- Schwebke, J.R. (2002): Update of trichomoniasis. *Sex Transm Infect,* 78: 378-379.
- Schwebke, J.R. and Burgess, D. (2004): Trichomoniasis. *Clin Microbiol Rev.* 17(4):794-803.
- Schwebke, J.R. and Hook, E.W. (2003): High rates of *Trichomonas vaginalis* among men attending a sexually transmitted diseases clinic: implications for screening and urethritis management. *J Infect Dis.* 188(3):465-468.
- Skerk, V., Schönwald, S., Krhen, I., Markovinovic, L., Beus, A., Kuzmanovic, N.S., Kruzic, V. and Vince, A. (2002): Aetiology of chronic prostatitis. *Int J Antimicrob Agents.* 19(6):471-474.
- Smith, K., Harrington, K., Wingood, G., Oh, M.K., Hook, E.W. and DiClemente, R.J. (2001): Self-obtained vaginal swabs for diagnosis of treatable sexually transmitted diseases in adolescent girls. *Arch Pediatr Adolesc Med.* 155(6):676-679.
- Sobel, J.D. (1997): Vaginitis. *N Engl J Med.* 337(26):1896-1903.
- Soper, D. (2004): Trichomoniasis: under control or undercontrolled? *Am J Obstet Gynecol.* 190(1): 281-290.
- Sorvillo, F. and Kerndt, P. (1998): *Trichomonas vaginalis* and amplification of HIV-1 transmission. *Lancet* 17;351(9097):213-4.
- Spence, M. (1992): Trichomoniasis. *Contemp OB/GYN.* 21: 132-141.
- Stratakis, D.F., Lang, S.M., Eichenlaub, S., Löscher, T., Stein, R. and Huber, R.M. (1999): Pulmonary trichomoniasis: diagnosis based on identification of irritation in bronchoalveolar lavage. *Pneumologie.* 53(12): 617-619.
- Sutton, M., Sternberg, M., Koumans, E.H., McQuillan, G., Berman, S. and Markowitz, L. (2007): The prevalence of *Trichomonas vaginalis* infection among reproductive-age women in the United States. *Clin Infect Dis.* 45(10): 319-326.
- Thomason, J.L. and Gelbart, S.M. (1989): *Trichomonas vaginalis*. *Obstet Gynecol.* 74(3): 536-541.
- Thornton, A.C., Dale, T., Fortenberry, D. and Logan, T.K. (2003): Occurrence of trichomoniasis in college women. *J Adolesc Gynecol.* 12(2): 34-38.
- Wang, C.C., McClelland, R.S., Reilly, M., Overbaugh, J., Emery, S.R., Mandaliya, K., Chohan, B., Ndinya-Achola, J., Bwayo, J. and Kreiss, J.K. (2001): The effect of treatment of vaginal infections on shedding of human immunodeficiency virus type 1. *J Infect Dis.* 183(7):1017-1022.
- Weinstock, H., Berman, S. and Cates, W. Jr. (2004): Sexually transmitted diseases among American youth: incidence and prevalence estimates. *Perspect Sex Reprod Health.* 36(1): 6-10.
- Wendel, K.A., Erbeling, E.J., Gaydos, C.A. and Rompalo, A.M. (2003): Use of urine polymerase chain reaction to define the prevalence and clinical presentation of

- Trichomonas vaginalis* in men attending an STD clinic. *Sex Transm Infect.* 79(2):151-153.
- Wendel, K.A., Erbeding, E.J., Gaydos, C.A. and Rompalo, A.M. (2002): *Trichomonas vaginalis* polymerase chain reaction compared with standard diagnostic and therapeutic protocols for detection and treatment of vaginal trichomoniasis. *Clin Infect Dis.* 35(5):576-580.
- Weston, T.E and Nicol, C.S. (1963): Natural history of trichomonal infection in males. *Br J Vener Dis.* 39: 251-257.
- Wiesenfeld, H.C., Lowry, D.L., Heine, R.P., Krohn, M.A, Bittner, H., Kellinger, K., Shultz, M. and Sweet, R.L. (2001): Self-collection of vaginal swabs for the detection of Chlamydia, gonorrhea, and trichomoniasis: opportunity to encourage sexually transmitted disease testing among adolescents. *Sex Transm Dis.* 28(6): 321-325.

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