Trichomonas vaginalis: Old Parasite, New Understanding and a Silent Epidemic

To the everyday physician who may not have a microbiological diagnosis, empirical treatment in syndromic approach to vaginal discharge or suspected trichomoniasis consists of oral metronidazole 200 mg given 3 times a day for 7 days or a single 2 gm dose. Success rates range from 82 to 88%, and reach almost 95% when the sexual partner is also treated simultaneously. Centre for Disease Control had quoted that 5% of all *T. vaginalis* patients isolates in the States in 1989 had nitroimidazole resistance. When resistance does occur, experimentation must start with different varieties of existing treatments and protocols including furazolidone, mebendazole, butaconazole and benzoizothiazolinon. On the other hand, only one vaccine against *T. vaginalis* had been produced from inactive lactobacilli and was thought to work by inducing antibodies to abnormal lactobacilli and *T. vaginalis* without adversely affecting growth of normal lactobacilli. Clinical trials have, however, yielded inconclusive results.

Ever since first described by Donné in early nineteenth century, trichomoniasis has been relegated to a mere nuisance disease of women, causing unpleasant, uncomfortable or sometimes slightly irritating vaginal discharge. In the last 50 years, research had gone from developing axenic culture and defining nutritional requirements to finding an effective treatment. By now, it is clear that *T. vaginalis* survives in the ever-changing vaginal microflora environment. During menstruation, for example, dramatic changes occur with influx of erythrocytes, host macromolecules, serum constituents and large changes in pH. These provide nutrients as well as a supply of iron which upregulates adhesions, immunogens, and C3-degrading proteinases which are essential for stress response of the parasite.

Heat shock proteins, of sizes 35-165 kDa, act as chaperones and stabilize changes in stress-related protein synthesis. A P-glycoprotein, which is usually associated with multidrug resistance (although this has not been seen in *T. vaginalis*), has also been associated with the stress response. Animal models with mouse have been most popular, but intravaginal infection has been successful only after pre-estrogenization, partly because *T. vaginalis* is not a naturally occurring organism in mice. *Trichomonas foetus* which are found in cattle can probably provide us more information.

To the epidemiologist, previous arguments on routes of transmission have now given way to the realization of the magnitude of disease caused by this commonest non-viral sexually transmitted disease in the world. The burden of disease is estimated to be 107 million cases worldwide, with the majority (92%) occurring in women. Prevalence rates of as high as 46% in women of childbearing age have been reported in sub-Saharan Africa. A study using a combination of wet-mount and polymerase chain reaction noted a prevalence of 20.8% and 12.2% in symptomatic and asymptomatic Malawian men respectively. Our local data showed a prevalence of 2.8% amongst commercial sex workers.

A more disturbing scenario, however, has been the correlation between trichomoniasis and a 5-fold increased risk of acquiring human immunodeficiency virus, although apparently this increase appears to be present for the receptive partner only. It appeared that trichomonads not only disrupt the epithelial barrier but also appear to recruit CD4 T-lymphocytes to the genital area. Significantly increased viral loads have also been demonstrated in semen of men with trichomonal urethritis. For the present, where resources permit, these findings strongly suggest a case for aggressive screening and treatment for *T. vaginalis* infection in risk groups to reduce transmission of disease in the community.

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