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Effect of trichomoniasis therapy on genital HIV viral burden among African women

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Abstract

Background—Our objective was to test the hypothesis that treatment for trichomoniasis among HIV-infected women not taking antiretrovirals in South Africa would be associated with decreased HIV genital shedding.

Methods—HIV-infected women presenting for routine HIV care were screened for trichomoniasis using self-collected vaginal swabs with a rapid point-of-care immunochromatographic antigen test. Women testing positive were offered enrollment into a prospective cohort study if they had documented HIV infection, were ages 18–50, and not receiving antiretroviral therapy. Recent use of post-exposure prophylaxis or antibiotic therapy, active genital ulcers, or systemic illness were exclusion criteria. Cervical swabs were collected for gonococcal and chlamydial testing and those testing positive were excluded. Women were treated with directly-observed oral therapy with 2 grams of oral metronidazole. A follow-up visit was scheduled one month after therapy and partner letters were provided. Paired cervical wicks and plasma were collected for viral load measurement.

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Results—557 women were screened. 60 tested positive for trichomoniasis, 10 subsequently met exclusion criteria, 4 were lost to follow-up. Of 46 women evaluated at follow-up, 37 were cured, 80.4%. Plasma viral load was not significantly different after therapy, $p=0.93$. Genital tract viral load decreased by 0.5 $\log_{10}$, $p<0.01$. The mean genital tract viral load ($\log_{10}$) decreased from 4.66 ($<3.52 - 6.46$) to 4.18 ($<3.52 - 6.48$), $p<0.01$ following therapy.

Conclusions—Screening and treatment of vaginal trichomoniasis decreases genital shedding of HIV among South African women not receiving antiretrovirals at one month following therapy.

Keywords

HIV; trichomoniasis; women; genital shedding; metronidazole

Introduction

Trichomonas vaginalis is the most common nonviral sexually transmitted infection worldwide.^{1–3} In general, reports of *Trichomonas vaginalis* prevalence in sub-Saharan Africa range from 11–34%,^{4–6} and in selected populations are as high as 74%.^{7,8} At least 16% of Zimbabwean women with trichomoniasis were asymptomatic upon direct questioning and some reports suggest that nearly half of women with trichomoniasis are asymptomatic.^{9,10} Despite the high prevalence of the infection and the frequency of asymptomatic infection, trichomoniasis is not routinely screened for in most settings. *T. vaginalis* infection is believed to enhance HIV transmission.⁹ An estimate of the annual number of new HIV infections in the United States attributable to trichomoniasis assumed a co-factor effect of 2–5 fold increased risk.^{11,12} Both HIV and trichomoniasis are associated with alterations in the vaginal inflammatory cytokine milieu.^{13–17} In a study conducted in Malawi, men with urethritis had higher HIV viral RNA concentrations in semen if infected with *T. vaginalis* than did controls with urethritis of unidentified cause.¹⁸

In a study of Kenyan women performed by Wang et. al., there was a significant decrease in HIV-1 RNA genital shedding after a 10-day course of metronidazole.¹⁹ Were screening for trichomoniasis and treatment with simple, inexpensive therapy to decrease genital HIV viral load, it could potentially translate into decreased transmission and acquisition of HIV. This is particularly true in resource-limited settings in which anti-retroviral therapy (ART) use for HIV is not widespread. Work performed by the Rakai Study Group has clearly shown that there is an increase in HIV transmission risk with increased viral load. The odds of HIV transmission increases by 2.45 for each $\log_{10}$ increase in serum HIV viral load.²⁰ It is also well-established that there is good correlation between plasma and genital shedding, with a 2.6 odds increase in the^{21,22} likelihood of detecting genital shedding with each $\log_{10}$ increase in plasma viral load. And recent work shows that higher genital tract shedding does, in fact, increase sexual transmission risk²³. The objective of this study was to estimate whether single-dose metronidazole therapy for trichomoniasis given to HIV-infected women not taking ART would decrease genital HIV shedding.

Materials and Methods

Study population

This was a prospective cohort study performed at Thembu Lethu clinic in Johannesburg, South Africa. The site holds a wellness clinic for HIV-infected women recently engaged in care. The study protocol was approved by the University of Witwatersrand Ethics Committee in South Africa as well as at the Institutional Review Board at Women & Infants Hospital of RI. Eligible women were ages 18–50, non-pregnant with CD4 cell counts greater than 200/mm³, not receiving anti-retroviral therapy, willing to avoid use of intravaginal

products during the approximately one month study period, and subject and partner willing to use condoms with sexual intercourse during the study period. Women were offered education about sexually transmitted infections and this study. Full, informed consent was signed if the woman was interested in participating in the study. They were given a brochure and instructed on method of self-collection of in the clinic of a vaginal swab. The research assistant collected the swabs from the subject and performed a rapid immunochromatographic test for trichomoniasis (Genzyme Diagnostics, Cambridge, MA) while the subject waited. Those testing positive for trichomoniasis were offered enrollment into the cohort.

Study procedures

At enrollment, the subject was interviewed and underwent a pelvic/speculum examination to collect study specimens. A blood sample was collected for determination of plasma viral load. The speculum examination involved a routine method of specimen collection and collection of samples for PCR testing for *N. gonorrhoea* and *C. trachomatis*. Women with positive tests for gonococcal or chlamydial infection were excluded. Cervical viral load samples were collected with a wick (TearFlo strips, Sigma Pharmaceuticals, Monticello, IA) as was a dry slide for gram staining for the diagnosis of bacterial vaginosis via the method of Nugent²⁴. Nugent gram stain score result accuracy was confirmed with over 90% concordance with the reference laboratory of Sharon Hillier, PhD. (Pittsburgh, PA).

Infected women were treated at enrollment on-site with 2 grams of oral metronidazole directly observed therapy. The subjects were also given condoms and asked to use them for intercourse during the study period. Subjects were given partner letters to seek medical care but uptake of partner therapy was not measured. The follow-up examination was performed one month later with specimen collection performed in a fashion identical to the first examination, as well as a follow-up rapid trichomoniasis test for confirmation of cure.

The cervical wick specimens were prepared for shipment by placing the strips in 9.0 mL of NASBA Lysis Buffer and frozen at -20° until the assays were run. The samples were shipped to Emory University where the genital viral load measurements were performed using bioMérieux Nuclisens EasyQ HIV-1 v1.1 (Durham, NC). The lower limit of detection of the assay was 3,300 copies/mL. The plasma viral loads were drawn at the same visit and were conducted in the local South African reference laboratory using the Roche linear assay (Pretoria, South Africa), with a lower limit of detection of 400 copies/mL.

Analysis

The cohort of study participants was characterized by summarizing the baseline demographic covariates using mean/median and range for continuous variables and count and percent for categorical and count variables. Changes following trichomoniasis therapy from the baseline were compared using McNemar's tests for categorical outcome variables such as detectable viral load and contraception use, and paired-t tests for continuous outcome variables such as log₁₀ transformed plasma/genital tract viral load. Two versions of plasma/genital tract viral loads were compared: 1) a dichotomized version as 'detectable' and 'undetectable', and 2) a log₁₀ transformed version to achieve normal approximation (undetectable viral loads were imputed by mid-values of the respective lower limits of detection before the log₁₀ transformation). To adjust for the effect of potential confounders, we also conducted a regression analysis. Age, trichomoniasis cure, contraceptive use, and BV were included in the regression as independent covariates to account for their potential confounding effects. Detectable viral load changes from baseline to follow up were examined using conditional logistic regression model; log₁₀ transformed viral loads were examined using linear mixed-effect model²⁵. In these two models, participant ID was used

as a stratifying factor to account for repeated measures. In this paper, we present the findings from the mixed-effect model, which are consistent with the finding from the conditional logistic regression model (in both the direction of association and significance). Two-sided p-values of <0.05 were regarded as statistically significant. All statistical analyses were conducted using R (version 2.12, Vienna, Austria).

The planned study sample size (N=57) was calculated based on the hypothesis that: 1) trichomoniasis treatment by metronidazole would result in one log₁₀ decrease in the genital shedding of HIV viral load at follow-up compared with the baseline; 2) genital viral load would have a standard deviation of 2 log₁₀; 3) within-person correlation is 0.15; and 4) 5% loss-to-follow-up. This sample size would lend us a power of 0.80 for a two-sided significance (alpha) of 0.05 based on a paired t-test.

Results

A total of 557 women were screened for vaginal trichomoniasis at the clinic. 60 of these, 10.7% (95% Confidence interval (CI), 7.5–12.6) tested positive. All of these women agreed to enrollment and were provided with directly observed therapy with 2 grams of oral metronidazole. 10 women were subsequently excluded due to meeting the pre-specified exclusion criteria, while an additional 4 were lost to follow-up (Figure 1). The majority of the study participants reported being single and only 21/46 (46%) reported condom use (Table 1).

In this cohort of women not taking anti-retroviral therapy, mean log₁₀ plasma viral load was unchanged among the participants following metronidazole administration (p=0.93) (Table 2), and there were comparable proportions of women with detectable virus at baseline and following therapy (p=0.99). Genital tract viral load decreased significantly (p<0.01) and was less likely to be detectable following therapy (p=0.04) (Figure 2). The frequency of BV, defined as a Nugent score ≥ 7, was not statistically different following therapy.

In order to assess the independent effect of therapy on genital tract viral load, and to assess for the effect of potential confounding variables, we conducted a regression analysis. The outcome variable was log-transformed to approximate a normal distribution and the regression model included random effects to account for the effects of repeated measures. Plasma viral load was the most significant predictor of genital tract viral load. However, the only other significant predictor of genital tract viral load in the model was trichomoniasis therapy. It remained significant even after controlling for the potential confounding variables age, contraceptive use, and BV (Table 3). And, while follow-up after therapy was associated with a decrease in genital shedding of HIV, cure of trichomoniasis was not in this regression model.

Discussion

We found that single dose directly observed therapy with oral metronidazole for vaginal trichomoniasis at point-of-care decreases genital shedding of HIV-1 among South African women not taking anti-retroviral therapy. Plasma viral load was not affected by therapy, as anticipated since metronidazole has no known activity against HIV-1. This finding supports the theory that trichomoniasis causes an inflammatory state, predisposing to the increased genital shedding of HIV-1. The trichomoniasis cure rate at one month was over 80% even in the absence of partner therapy or known reliable condom use. The reason that cure of trichomoniasis was not associated with decrease in HIV shedding is unknown. This may be related to small cell sizes in the regression model but could also indicate that metronidazole decreases genital shedding by some other mechanism than eradication of trichomonads. The

anti-inflammatory properties of metronidazole could play a role. The implications for the results of this study are that there is a potential for a screening and treatment program to decrease HIV transmission. It will be important for trials of screening and treatment to not only document change in shedding of virus but also how that translates into transmission risk.

There have been recent disappointing findings from large trials aimed at decreasing risk of HIV transmission by treating genital herpes after much promise from early studies. Nagot et al. reported a randomized controlled trial aimed at reducing both plasma and genital HIV-1 viral load²⁶. The study showed a promising decrease in both genital and plasma viral load and thus trials aimed at decreasing HIV acquisition and transmission were undertaken. Unfortunately, the subsequent high quality randomized trial of HSV suppression therapy by infected partners did not reduce the risk of transmission to un-infected partners²⁷. Genital herpes and its therapy may not be comparable with trichomoniasis for a number of reasons. Of most concern for therapy, herpes is not something that can be cured. It requires twice daily therapy in a patient that is likely to be asymptomatic. Trichomoniasis can be treated with a single dose of a medication that is inexpensive. Further, the genital tract viral load decrease that is required to decrease transmission is not clearly known. Female to male transmission increases with a hazard ratio of 2.38 (95% CI, 1.13–4.78) with each log₁₀ increase of endocervical HIV-1 viral load²³. It may be that the 0.29 log₁₀ decrease seen with herpes suppression²⁶ reported in the Nagot study was not adequate to decrease transmission risk.

Those trials that have included therapy for trichomoniasis have been predominantly syndromic therapy or aimed at multiple sexually transmitted infections²⁸. Similar to the herpes trials, these may have resulted in inadequate improvement in genital viral shedding. Alternatively, they may have included sexually transmitted infections that do not respond to therapy by diminished shedding.

Our study has several limitations. It was a single arm treatment trial in which each subject served as her own control without a separate control group. Because of the relatively small sample size, we used genital tract shedding as a surrogate marker for transmission risk. We cannot draw any causal relationship between therapy for vaginal trichomoniasis and risk of HIV transmission. Because of the local social environment, we were not permitted to provide therapy for partners but only partner letters or measure partner uptake of therapy. Therefore, we had to rely upon the subjects to both give the letters to their partners as well as abstain from intercourse or use condoms until their partner was treated. This may have lead to a number of reinfections.

Strengths of the study include its prospective nature and the use of directly observed therapy in an HIV treatment government clinic, eradicating any concern about compliance with medication that is problematic in many studies. Because there is no medication to take home and because the therapy was taken at the time of the study visit, compliance was 100%. Thus, we can exclude non-compliance as a reason for not seeing a larger decrease in genital shedding. As has been seen in other studies examining HIV-infected women, the prevalence of BV was high at 43%. Another strength of the study was our ability to control for BV, which has been previously shown to increase shedding of HIV^{29–33}.

Point of care testing for vaginal trichomoniasis is currently somewhat expensive but uptake of therapy was very good in this general HIV clinic, with a prevalence that was about what was expected even with the use of self-collected samples. Directly observed therapy with metronidazole is both convenient and inexpensive. If future studies are able to demonstrate

efficacy in decreasing HIV transmission risk with therapy for trichomoniasis, an approach using point of care testing and directly observed therapy should be considered.

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Summary

Among antiretroviral naive African women, point-of-care treatment of vaginal trichomoniasis with a single dose of directly observed metronidazole significantly decreased HIV genital shedding, but did not alter plasma viral load.

Figure 1.
Flow diagram of participants included in the study.

Figure 2. Median log-transformed genital tract viral loads at enrollment (Visit 1) and follow-up (Visit 2). GTVL: genital tract viral load

TABLE 1

Baseline Characteristics of Study Cohort

Baseline Characteristics (N = 46)	Mean or Count	Range or Percentage
Age (yr)	35	(18–48)
Marital status		
Single	29	63%
Prior deliveries		
Have at least 1 child	37	80%
No. children	1.9	(0–6)
Occupation		
Employed	22	50%
Unemployed	22	50%
Tobacco use	5	11%
Presumed mode of HIV acquisition		
Sexual	46	100%
Any contraception		
Yes	24	52%
Form of contraception *		
Condoms	21	88%
Sterilization	5	21%
Injectable	5	21%
IUD	0	0%
Bacterial vaginosis †	20	43%

* Not mutually exclusive.

† Nugent score [mtequ] 7.

IUD indicates intrauterine device.

TABLE 2

Pre- and Post-Treatment Plasma and Genital Viral Load (N = 46)

	Pretreatment		Posttreatment		P
	Count or Mean	Percentage or Range	Count or Mean	Percentage or Range	
Trichomoniasis					
Yes	46	100%	8	17%	[It]0.01 *
Contraception					
Yes	24	52%	24	52%	1 *
Nugent Score					
[ltequ]3	7	15%	10	22%	0.17 *
4-6	19	41%	11	24%	
[intequ]7	20	43%	25	54%	
PVL (log ₁₀)	4.22	([It]2.60-5.87)	4.21	([It]2.60-5.87)	0.93 †
Detectable ([mt]400 copies/mL)	45	98%	44	96%	0.99 *
GT VL (log ₁₀)	4.66	([It]3.52-6.46)	4.18	([It]3.52-6.48)	[It]0.01 †
Detectable ([mt]3300 copies/mL)	38	82.61%	28	62%	0.04 *

* Based on McNemar test for comparing paired categorical outcomes.

† Based on paired *t* test; viral load was log₁₀ transformed with undetectable values imputed by mid values.

PVL indicates plasma viral load; GTV, genital tract viral load.

TABLE 3

Impact of Therapy and Other Factors on Genital Tract Viral Load by Random Effects Model Adjusted for Covariates

	Change in log ₁₀ GTVL (95%CI)	P
Postmetronidazole therapy	[minus]0.44 ([minus]0.76, [minus]0.12)	[lt]0.01
PVL (log ₁₀) (per each 1 log increase)	0.55 (0.35, 0.75)	[lt]0.001
Age (per 10 y)	0.00 ([minus]0.02, 0.3)	0.86
Trichomoniasis cure	0.22 ([minus]0.40, 0.83)	0.49
Contraception use	[minus]0.02 ([minus]0.42, 0.37)	0.90
BV		
NS: 4–6	0.07 ([minus]0.44, 0.59)	0.78
NS: [mtequ]7	0.07 ([minus]0.42, 0.55)	0.79

GTVL indicates HIV-1 RNA genital tract viral load; PVL, HIV-1 RNA plasma viral load; CI, confidence interval; BV, bacterial vaginosis; NS, Nugent Gram stain score.