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HIV Infection and Disturbances of Vaginal Flora During Pregnancy

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Summary: Disturbances of vaginal flora are common among women of reproductive age. In areas of sub-Saharan Africa where the prevalence of HIV is high, the frequency of bacterial vaginosis (BV) is also high. In this study, we assessed the association of BV and other disturbances of vaginal flora with prevalent HIV infection in two cross-sectional studies among pregnant women in urban Malawi. The prevalence of HIV-1 was 23% in 1990 and 30% in 1993. Overall, 30% of the women had BV, 59% had mild or moderate disturbance of vaginal flora, and only 11% had normal vaginal flora. Increasing prevalence of HIV was significantly associated with increasing severity of disturbance of vaginal flora ($p < .00001$, χ^2 trend test). This trend of increased prevalence persisted after controlling for concurrent sexually transmitted diseases (STDs), sexual activity, and socioeconomic factors. After multivariate adjustment for potential confounders, the odds ratio for the association of BV with prevalent HIV infection was 3.0 (95% confidence interval [CI], 2.4–3.8), that of moderate vaginal disturbance with HIV infection was 2.2 (95% CI, 1.7–2.8), and that of mild vaginal disturbance with HIV infection was 1.6 (95% CI, 1.3–2.1). Among women with BV, HIV infection was higher among younger women than older, implying more recent infection. Although these studies were cross-sectional, our data suggest that BV could be associated with increased susceptibility to HIV infection. **Key Words:** Bacterial vaginosis—HIV infection—Malawi—Women—Sexually transmitted diseases.

The Joint United Nations Programme on HIV/AIDS (UNAIDS) estimates that approximately 21 million individuals infected with HIV are in sub-Saharan Africa. This constitutes 68% of all HIV infections and 80% of all HIV infections in females in the world (1). Therefore, identification of risk factors amenable to prevention is highly desirable to control spread of heterosexual transmission of HIV and to reduce consequences of HIV/AIDS on women, children, and families.

Diseases that cause inflammation of the genital tract have been of particular concern. For example, ulcerative and nonulcerative sexually transmitted diseases (STDs) were shown to be associated with HIV transmission (2,3), and in a community trial, treatment of STDs was associated with a 40% decreased incidence of HIV (4). However, the potential relation with HIV transmission of more frequent genital conditions which cause no inflammation of the vaginal or cervical mucosa (e.g., bacterial vaginosis [BV]), have not been adequately examined. BV is characterized by disturbances in the vaginal flora resulting in loss of lactobacilli, an increase in other predominantly anaerobic flora, and an increased vaginal pH

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(5). If a causal relation between BV and HIV infection is established, the implications of rectifying abnormal vaginal flora to control HIV transmission could be substantial because prevalence of BV is high among women of reproductive age in developing and developed countries (6). Two cross-sectional studies suggest that BV may be associated with prevalent HIV infections in nonpregnant women (7,8). In this study, we assess the association of disturbances of vaginal flora, especially BV, with prevalent HIV infection among pregnant women in urban Blantyre, Malawi.

METHODS

Two large surveys of HIV and STDs were conducted in 1989 to 1990 and 1993 at the Queen Elizabeth Central Hospital (QECH), a tertiary care facility in Blantyre, southern Malawi, a country in southeastern Africa. Consecutive, first visit, antenatal women were recruited to determine prevalence of HIV and STDs. After pretest counseling and obtaining informed consent, pregnant women were screened for HIV and syphilis. A routine antenatal physical assessment was followed by a speculum-aided pelvic examination and collection of vaginal and cervical specimens. Sociodemographic and behavioral data were collected by trained interviewers using structured questionnaires. All women were counseled after the testing; treatment of STDs (i.e., gonorrhea, syphilis, trichomoniasis, and yeast infections) and condoms were provided in the clinic at no cost. Referrals to other specialized clinics in the hospital were also available.

HIV testing was carried out using enzyme immunoassay (EIA) and Western blot. In the 1990 survey, all women with a positive result on a repeat EIA (Wellcozyme, Wellcome Diagnostics, Dartford, Kent, U.K.) were confirmed by a Western blot test (Bio-Rad, Hercules, CA, U.S.A.). In the 1993 survey, women were screened using two independent EIA tests (Wellcozyme and Behring, Marburg, Germany) and only borderline specimens were confirmed by a Western blot test. Syphilis testing was performed using the rapid plasma reagin (RPR) test (Macro-Vue, Becton-Dickinson, Cockeysville, MD, U.S.A.) for screening and fluorescent treponemal antibody (FTA) or *Treponema pallidum* hemagglutination assay (TPHA; Sera-Tek, Miles Inc., Elkhart, IN, U.S.A.) tests for confirmation. Vaginal wet mounts were examined for clue cells, motile trichomonads, and yeast cells. Cervical swabs to isolate *Neisseria gonorrhoeae* were smeared on modified Thayer-Martin media and positive cultures were determined by colonial morphology and Gram stain.

Disturbances of vaginal flora and BV were based on four clinical criteria: a vaginal pH >4.5, measured by pH paper on a vaginal swab obtained from lateral and posterior fornices; an increased homogeneous vaginal discharge; presence of clue cells in >20% of vaginal epithelial cells, detected by mixing vaginal fluid with a drop of normal saline on a slide and examining under high-power magnification; and a positive result to an amine or whiff test, performed by mixing a few drops of 10% potassium hydroxide with vaginal fluid. Women with none of the four clinical criteria were classified as having normal vaginal flora. Disturbance of vaginal flora was classified as ''mild'' if only one criterion was present, ''moderate'' if two criteria were present, and ''severe'' if three or more criteria were present. We identified women with three or more clinical criteria (i.e., severe disturbance of vaginal flora) as having BV. The clinical definition of BV proposed by Amsel et al. (9) was based on three of four clinical criteria described previously.

Prevalence rates of HIV, STDs, and disturbances of vaginal flora were estimated. Associations of BV and other risk factors with HIV prevalence were calculated and compared using frequency tables and exact or trend tests. Adjustment for potential confounding was carried out using multiple logistic regression. Selection of variables for inclusion in the multivariate model was based on biologic and epidemiologic importance (e.g., variables with biologic plausibility and/or risk factors that have been reported in other studies to be associated with HIV infection). Confidence intervals were calculated for point estimates.

RESULTS

Of 9890 antenatal women who had been requested to participate in the 1990 and 1993 surveys at QECH in Blantyre, 9148 women (6684 in 1990 and 2464 in 1993) agreed to be tested for HIV, and 742 (7.5%) women refused. Most of these women (~75%) were in their second trimester of pregnancy. A statistically significant increase was found in the prevalence of HIV between 1990 and 1993 among women who had been screened (prevalence of 23.0% in 1990 and 30.1% in 1993; $p < .0001$). Among 9126 women who had both HIV and disturbance of vaginal flora results available, the prevalence of BV was higher in 1990 (31.1% of 6677) than in 1993 (27.0% of 2449; $p < .001$). Most women had either a mild or a moderate disturbance of vaginal flora (60.0% in 1990 and 57.5% in 1993). The prevalence of mild vaginal disturbance was 36.4% in 1990 and 43.6% in 1993; the prevalence of moderate disturbance of vaginal flora was 23.6% in 1990 and 13.9% in 1993. Only 8.9% in 1990 and 15.6% in 1993 of the women screened had normal vaginal flora.

Table 1 shows the prevalence of disturbed vaginal flora associated with sociodemographic and behavioral characteristics for women studied in 1990 and 1993. The prevalence of BV was higher among younger (<30 years) than among older women (29.5% versus 27.5%). Among women with higher socioeconomic status, as indicated by availability of electricity in the house, the prevalence of BV was lower compared with women with low socioeconomic status (26.5% versus 29.5%). The prevalence of BV did not vary by maternal education (Table 1), husband's education, parity, or marital status (data not shown). Women who reported having multiple sexual partners during the last year compared with monogamous women, had higher prevalence of BV (31.4% versus 28.5%). However, another indicator of sexual activity, use of traditional vaginal agents (compounds), did not influence the prevalence of disturbances of vaginal flora. Women with trichomoniasis, gonorrhea, or syphilis compared with women without these STDs, had a statistically significant increase in prevalence of BV and dis-

TABLE 1. *Prevalence of disturbance of vaginal flora among pregnant women with selected risk factors, 1990 and 1993 surveys combined, Blantyre, Malawi*

Risk factor	No. of women (N = 9890)[c]	Disturbance of vaginal flora				p Value[b]
		Normal (%)	Mild (%)	Moderate (%)	Severe[a] (%)	
Sociodemographic						
Age:						
<30 years	6789	11.0	39.0	20.6	29.5	<.004
≥30 years	2675	12.4	40.8	19.4	27.5	
Education						
Illiterate	1708	11.1	38.4	20.0	30.5	.15
Literate	7775	11.4	39.8	20.2	28.6	
Have electricity[d]						
Yes	1717	12.2	41.1	20.2	26.5	<.01
No	7752	11.2	39.1	20.2	29.5	
Sexual activity						
Number of partners						
≥2	1185	10.9	36.1	21.6	31.4	<.01
1	8331	11.5	40.1	20.0	28.5	
Used vaginal agents[e]						
Yes	3221	10.5	39.9	20.5	29.1	.27
No	6262	11.4	39.8	20.2	28.6	
Concurrent STDs						
Gonorrhea						
Yes	376	3.7	26.1	26.6	43.6	<.001
No	8728	10.0	39.8	20.8	29.5	
Trichomoniasis						
Yes	2838	3.2	22.6	29.5	44.7	<.001
No	6299	12.7	46.7	17.2	23.4	
Syphilis						
Yes	1001	6.7	37.6	23.0	32.6	<.001
No	8308	10.9	39.7	20.3	29.1	
Candidiasis						
Yes	1729	12.9	42.9	20.0	24.2	<.001
No	7410	9.0	38.4	21.2	31.4	

[a] Bacterial vaginosis: 3 or more clinical criteria present (see Methods).
[b] χ^2 test for trend.
[c] Differences between the overall total and subtotals are due to missing data including laboratory test results for disturbance of vaginal flora or STDs (e.g., women refusing pelvic examination or HIV testing).
[d] Index of higher socioeconomic status.
[e] Traditional vaginal agents for treatment of STDs or tightening of vaginal walls.
STDs, sexually transmitted diseases.

turbed vaginal flora (Table 1). Unlike other concurrent STDs, women who had candidiasis showed an inverse and a statistically significant association. The prevalence of severe disturbance of vaginal flora was lower among women with candidiasis compared with women with no candidiasis (24.2% versus 31.4%).

An increase in prevalence of HIV occurred with increasing severity of the disturbance of vaginal flora; the prevalence of HIV was highest among women with BV (i.e., women with three or more clinical criteria; Table 2). For example, in 1990, 10.1% of women with normal flora versus 30.8% of women with BV were infected with HIV; in 1993, 18.1% of women with normal flora versus 43.9% of women with BV were infected with HIV. This increasing trend of HIV prevalence was highly significant and consistent among women surveyed in 1990 and 1993 (Table 2). Among women with disturbed vaginal flora, HIV prevalence was higher in young women (<30 years) compared with older women (≥30 years), whereas among women with normal flora, the influence of age on HIV prevalence was minimal (Fig. 1). The trend of increased HIV prevalence with increas-

TABLE 2. *Association of disturbances of vaginal flora and prevalent HIV infection among pregnant women surveyed in 1990 and 1993*

Disturbance of vaginal flora	1990 HIV prevalence	1993 HIV prevalence
Overall[a]	1505/6677 (22.5%)	733/2449 (29.9%)
Normal	60/596 (10.1%)	69/381 (18.1%)
Mild	413/2429 (17.0%)	260/1067 (24.4%)
Moderate	392/1575 (24.9%)	114/340 (33.5%)
Severe[b]	640/2077 (30.8%)	290/661 (43.9%)
χ^2 trend	181.5 ($p < .00001$)	103.2 ($p < .00001$)

[a] Overall HIV prevalence among women with both HIV and disturbance of vaginal flora results available.
[b] Bacterial vaginosis based on three of four clinical criteria (see Methods).

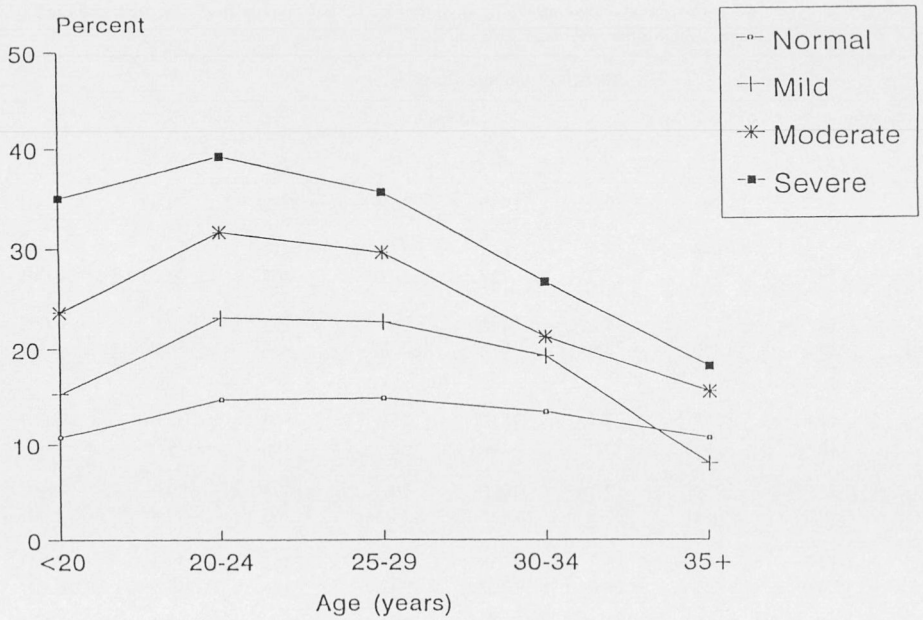

FIG. 1. Age-specific HIV prevalence among pregnant women screened in 1990 and 1993 stratified by severity of disturbance of vaginal flora.

ing severity of disturbance of vaginal flora was consistent in each age group, with the exception of women 35 years or older. For example, in each age group, the prevalence of HIV was highest among women with BV, intermediate in women with moderate and mild disturbance of vaginal flora, and lowest in women with normal flora. Because HIV transmission is influenced by sexual activity and other STDs, we performed stratified analyses to examine the association of prevalent HIV and BV after controlling for sexual activity and concurrent laboratory confirmed STDs (Table 3). Two variables, number of sexual partners and use of traditional vaginal agents (either for treatment of STDs or for tightening of vaginal walls) (10), were used as surrogate measures of sexual activity. A significant trend was shown of increasing HIV prevalence with increasing severity of vaginal flora disturbance independent of sexual activity. Likewise, a significant trend existed of increased prevalence of HIV with increased severity of disturbed vaginal flora among women with or without concurrent STDs. In all these analyses, women with normal flora had the lowest HIV prevalence, women with mild or moderate disturbance of vaginal flora had an intermediate HIV prevalence, and women with BV had the highest HIV prevalence. As expected, women with multiple partners or STDs (i.e., gonorrhea, trichomoniasis, or syphilis) had higher HIV prevalence compared with women with only one partner or with no STDs (Table 3).

Interestingly, the prevalence of HIV was higher among women with BV who had a single partner or no STD compared with women with normal vaginal flora and have multiple partners or concurrent STDs (Table 3). For example, the prevalence of HIV was 32.3% among women with BV who had a single partner compared with 17.3% among women with normal flora and multiple partners. Similarly, the prevalence of HIV was 32.6% among women with BV who had no gonorrhea compared with 28.6% among women with normal flora and gonorrhea.

The prevalence of HIV was similar among women with BV who had or did not have concurrent vulvovaginal candidiasis (34.1% versus 33.0%; Table 3). Given that candidiasis could suggest immunosuppression following HIV infection, we examined the prevalence of candidiasis among HIV-infected and uninfected women with concurrent BV. No difference was found in rates of candidiasis among HIV-infected and uninfected women with BV; the prevalence of candidiasis was 14.8% among 930 HIV-infected women with BV compared with 15.5% among 1811 uninfected women with BV. We also assessed prevalence of BV on a smaller sample of HIV-infected women in whom $CD4^+$ counts (using FACScan flowcytometry, Becton-Dickinson) were available. The prevalence of BV was 48.3% among 234 women with $CD4^+$ counts >500 cells/mm^3 compared with 41.6% among 214 women with $CD4^+$ counts ≤500 cells/mm^3 ($p = .18$).

The association between prevalent HIV and BV persisted after adjusting for potential confounders in multivariate logistic regression analysis (Table 4). A strong, statistically significant association was found between HIV infection and BV and a clear trend of increased odds

TABLE 3. *Prevalence of HIV among pregnant women with disturbance of vaginal flora stratified by sexual activity and concurrent sexually transmitted diseases (1990 and 1993 surveys combined)*

	Disturbance of vaginal flora								
	Normal		Mild		Moderate		Severe (BV[a])		
Risk factor	*N*	%	*N*	%	*N*	%	*N*	%	*p* Value[b]
Overall	977	13.2	3496	19.3	1915	26.4	2738	34.0	<.001
Sexual activity									
1 partner	896	12.8	3125	18.3	1658	24.8	2331	32.3	<.001
⩾2 partners	81	17.3	371	27.2	257	37.0	407	43.2	<.001
Used traditional vaginal agents[c]									
No	695	14.2	2271	17.6	1235	24.1	1797	32.9	<.001
Yes	280	13.6	1206	22.4	675	30.8	938	36.0	<.001
Concurrent STDs[d]									
Gonorrhea									
No	870	11.6	3282	18.3	1810	25.4	2564	32.6	<.001
Yes	14	28.6	98	48.0	100	43.0	164	54.5	<.06
Trichomoniasis									
No	800	11.3	2759	17.3	1080	20.4	1473	29.0	<.001
Yes	91	19.8	644	27.2	835	34.3	1265	39.8	<.001
Syphilis									
No	912	12.7	3164	18.3	1658	24.4	2395	32.3	<.001
Yes	65	20.0	332	28.0	257	39.3	343	45.5	<.001
Candidiasis									
No	668	13.0	2670	20.3	1566	28.0	2319	34.1	<.001
Yes	224	9.4	739	14.9	345	19.6	418	33.0	<.001

[a] Bacterial vaginosis (BV) based on three of four clinical criteria (see Methods).
[b] χ^2 test for trend.
[c] For treatment of STDs or tightening of vaginal wall; women with missing data are excluded.
[d] Few STD results not available.
STD, sexually transmitted disease.

of HIV infection with increased disturbance of vaginal flora. These findings were consistent among women surveyed in 1990 and 1993. Overall, concurrent STDs (with the exception of yeast infection), sexual activity (as demonstrated by number of sexual partners and use of traditional vaginal agents), young maternal age, parity, and higher socioeconomic status were significantly associated with prevalent HIV.

TABLE 4. *Association of disturbances of vaginal flora with prevalent HIV infection controlling for other risk factors: pregnant women surveyed in 1990 and 1993*

	Adjusted odds ratio (95% confidence interval)		
Risk factor	1990	1993	Overall[a]
Disturbance of vaginal flora			
Mild	1.67 (1.25–2.26)	1.60 (1.13–2.31)	1.65 (1.32–2.07)
Moderate	2.26 (1.68–3.08)	2.09 (1.40–3.17)	2.21 (1.74–2.81)
Severe[b]	2.98 (2.24–4.03)	3.37 (2.34–4.92)	3.04 (2.43–3.84)
Syphilis	1.57 (1.31–1.87)	1.98 (1.49–2.65)	1.67 (1.44–1.94)
Gonorrhea	2.72 (2.14–3.45)	1.25 (0.69–2.25)	2.43 (1.95–3.04)
Trichomoniasis	1.71 (1.50–1.94)	1.46 (1.18–1.80)	1.64 (1.47–1.83)
Candidiasis	0.91 (0.78–1.05)	0.76 (0.51–1.10)	0.88 (0.76–1.01)
Used traditional vaginal agents	1.31 (1.15–1.50)	1.59 (1.29–1.97)	1.38 (1.24–1.54)
Sexual partners ⩾2	1.89 (1.60–2.24)	1.94 (1.39–2.70)	1.87 (1.61–2.17)
Age <30 years	1.57 (1.31–1.88)	1.39 (1.03–1.89)	1.52 (1.30–1.78)
Parity			
Primiparous	1.43 (1.19–1.71)	1.14 (0.86–1.52)	1.34 (1.15–1.56)
1–2 children	1.34 (1.14–1.57)	1.19 (0.93–1.54)	1.30 (1.13–1.48)
Have electricity[c]	1.65 (1.42–1.91)	1.08 (0.84–1.39)	1.47 (1.29–1.67)

[a] 1990 and 1993 data combined.
[b] Bacterial vaginosis based on 3 of 4 clinical criteria (see Methods).
[c] Indicates higher socioeconomic status.

DISCUSSION

Investigation of these two large and independent populations of pregnant women show that disturbances of vaginal flora, and especially its severe form (i.e., BV), are strongly associated with prevalent HIV infection. Although these studies were cross-sectional and cannot show that BV preceded HIV infection, a possible interpretation is that BV could increase acquisition of HIV among women of reproductive age. The evidence for this causal argument is as follows. First, a strong trend of association exists between HIV infection and disturbance of vaginal flora, suggesting that severity of the disturbance of vaginal flora or increased certainty of diagnosis is associated with concurrent HIV during pregnancy. Second, among women with BV, HIV infection was higher among younger women than older women, implying that the HIV infection is most likely to be recent. Third, the association of HIV infection with BV was not explained by likely confounding factors such as concurrent STDs or sexual activity (Tables 3 and 4). Fourth, it is unlikely that HIV infection preceded severe disturbance of vaginal flora because BV was not more common in women with immunosuppression due to HIV. BV was similar among women with low or high $CD4^+$ counts and women with or without candidiasis, albeit vaginal candidiasis was less predictive of clinical AIDS (11).

The results of the two surveys in 1990 and 1993 were consistent although they were conducted 3 years apart, on two different cohorts of pregnant women, and during a period when HIV prevalence was rising. Our findings are in agreement with a population-based study in the Rakai district of Uganda where a similar association between prevalent HIV and BV was recently reported (7). Among women in Uganda, the association of prevalent HIV and abnormalities of vaginal flora were not related to differences in sexual activity or STDs, and as in the present study in Malawi, HIV prevalence was higher among younger than among older women who had BV. Another cross-sectional study in Thailand among 144 commercial sex workers also reported positive associations between BV and HIV infection (8). Therefore, these investigations among pregnant and nonpregnant women show remarkably similar associations between BV and HIV.

It is biologically plausible that susceptibility to HIV infection is increased in women with BV as a result of changes in the vaginal ecosystem as a result of an increase in vaginal pH and loss of lactobacilli. Low vaginal pH has been demonstrated to inhibit CD4 lymphocyte activation and to decrease in recruitment of HIV target cells, such as CD4 lymphocytes and macrophages, in the vagina (12). Thus, a less acidic vaginal pH might also support HIV survival and multiplication (13). In addition, depletion of lactobacilli could lead to loss of hydrogen peroxide and other antibacterial factors, which are protective against pathogenic agents possibly including HIV (14,15).

Our data are consistent with findings from other studies that reported BV to be associated with risk factors such as low socioeconomic status, sexual activity, and concurrent STDs (9,16). The prevalence of severe BV and moderate disturbance of vaginal flora were particularly high among women who had trichomoniasis or gonorrhea. It is not known whether these pathogenic organisms modify vaginal flora or if disturbed vaginal flora facilitates acquisition of these STDs (17). In contrast, the prevalence of severe and moderate vaginal flora disturbances were lower in women with candidiasis compared with women without candidiasis (Table 1). This is reassuring inasmuch as vaginal *Candida* infections favor a lower vaginal pH ($\leq$4.5). Other studies that used Gram stain to characterize vaginal flora also reported similar results in which vaginal candidiasis was associated with normal and intermediate flora, and *Chlamydia trachomatis* and *N. gonorrhoeae* infections were more frequent among women with BV or intermediate flora (17). We did not test for Chlamydia infections because its prevalence has been reported to be low in Malawi even among STD clinic attendees (18). We also did not consider condom use, contraceptive use, or the role of menstrual cycle in our analysis because these women were pregnant on enrollment, and condom use is inconsistent and low (19,20).

A small but statistically significant decline in BV prevalence occurred between the two surveys in 1990 and 1993. A similar trend of decline has also been reported for STDs such as gonorrhea, syphilis, and trichomoniasis in these cohorts during the same period (20). HIV prevalence, however, significantly increased. Our data suggested a strong association between disturbances of vaginal flora other than BV and prevalent HIV infection. Therefore, the increase in HIV prevalence could be attributed to vaginal disturbances that actually increased; for example, mild vaginal disturbance was higher among women surveyed in 1993 (43.6%) compared with women surveyed in 1990 (36.4%). It is also likely that other risk factors, especially factors which we were not able to estimate or identify, or the interaction of several factors, could have added to the rising HIV prevalence. Inadequate measurement of some risk factors included in the analysis is another potential limitation (e.g., socioeconomic status).

The prevalence of moderate BV among Ugandan

women was 44.5% and that of severe BV was 6.4% (7) based on results of Gram staining and morphologic classification (21). A BV prevalence of 52% (based on Gram staining) was also reported among pregnant women attending a large tertiary care hospital serving mainly black women in KwaZulu/Natal, South Africa (22). The estimate of BV prevalence we reported in this study among pregnant women in 1990 and 1993 is lower than the estimates from Uganda and S. Africa. It is likely that our estimates of BV are conservative, because a high correlation has been found between clinical criteria and use of Gram stain for diagnosis of BV (23,24). Most women in our study had an intermediate state, either mild or moderate disturbance of vaginal flora; and women with intermediate flora are more likely to progress to BV than women with normal flora (25).

Data from the Rakai STD control for HIV prevention suggest that oral 2 g metronidazole can reduce, but not cure, BV. In pregnant women given one dose of metronidazole at any time during pregnancy the postpartum prevalence of BV was 39.1%, compared with 52.8% in control mothers (relative risk = 0.74; 95% confidence interval [CI], 0.67–0.81). In the fourth round of the main trial, metronidazole at 10-month intervals reduced significantly the prevalence of BV (43.6% in intervention and 53.8% in control; relative risk = 0.82; 95% CI, 0.68–0.97; Gray RH, personal communication, August 1998).

The high prevalence of BV among African women together with the rapid heterosexual spread of HIV in this population underscores the importance of investigating the potential role of disturbance of vaginal flora on HIV acquisition or transmission. An explanation of a causal relation between BV and HIV in the cross-sectional studies conducted in Malawi and Uganda is supported by preliminary longitudinal data from both sites showing that BV is associated with incident HIV (26,27). Increased acquisition of HIV because of BV during pregnancy could increase vertical transmission of HIV, in addition to the known serious obstetric and gynecologic sequelae, such as preterm delivery, premature rupture of membranes, chorioamnionitis, and pelvic inflammatory disease (28). Therefore, screening and treating pregnant women with BV could reduce several adverse reproductive outcomes.

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