ORIGINAL ARTICLE

Social and sexual risk factors for bacterial vaginosis

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Background: A number of sexual and social risk factors for bacterial vaginosis (BV) have been identified. However, many previous studies have used small numbers of patients, or highly selected or convenience samples, or poorly defined populations. This study aims to clarify potential sexual and non-sexual risk factors for BV.

Methods: Women attending the Sydney Sexual Health Centre with BV, between March 1991 and July 1999, were included. Controls were randomly selected women without BV. Information on the demographics, clinical findings, and sexual and non-sexual risk behaviours were extracted from the clinic database and analysed using SPSS and SAS. A logistic regression model was used to establish which associations with BV persisted.

Results: 890 women with BV and 890 controls were studied. Factors that were independently associated with BV were ⩾3 male sexual partners in the past 12 months (OR = 1.60, 95% CI: 1.19 to 2.04), at least one female sexual partner in the past 12 months (OR = 2.1, p = 0.003), a past pregnancy (OR = 1.5, p<0.0006), and smoking. In contrast, women with BV were significantly less likely to have used hormonal contraception (OR = 0.60, 95% CI: 0.51 to 0.81) or to have used condoms consistently (OR = 0.5, 95% CI: 0.31 to 0.71) than controls.

Conclusion: Our findings may be important for planning a preventive strategy for BV by discouraging smoking and increasing condom use and hormonal contraception among women.

Bacterial vaginosis (BV) is a common clinical entity characterised by abnormal greyish white, homogenous, malodorous vaginal discharge. It results from an overgrowth of anaerobic bacteria in the vagina replacing or markedly reducing the normal vaginal flora.[1] Up to 50% of women harbouring these abnormal bacteria are asymptomatic.[2]

The prevalence of BV varies from 5–26% in pregnant women[3–8] to 24–37%[2 9–11] in those attending sexually transmitted infection (STI) clinics. BV is associated with pelvic inflammatory disease (PID), significant reproductive morbidity, including premature rupture of membranes and preterm delivery, and increased risk of HIV transmission and acquisition. Hence, timely therapeutic intervention and preventive strategies in selected populations may decrease BV associated morbidity and the risk of HIV transmission. Treatment with metronidazole has a reported cure rate of 78–85% compared with clindamycin (82–85%), but recurrences are common.[12 13] Hence, it may be important to explore primary preventive strategies.

Preventive strategies target the risk factors or behaviours for a disease. Previous studies have identified a number of risk factors and behaviours associated with BV, including the number of lifetime male sexual partners, recent partner change, lower age of first intercourse, having had a female sexual partner in the past 12 months, being unemployed, being unmarried, working as a sex worker, smoking, and failure to use condoms.[13–18] However, many previous studies have used small numbers of patients, or highly selected or convenience samples, or poorly defined patient populations. There is a paucity of published literature investigating risk variables for BV, particularly in Australian populations. Consequently, this study aims at investigating the potential sexual and non-sexual risk factors for BV in an urban sexual health clinic population in Australia.

METHODS

Study subjects

Subjects included all women diagnosed with BV on their first clinic visit to the Sydney Sexual Health Centre (SSHC) between March 1991 and July 1999. Controls were randomly selected from those who first attended the clinic on the same day as the study subjects but without a diagnosis of BV. Ethical approval was obtained from South Eastern Sydney Area Health Service research ethics committee. All data were de-identified and only group data were analysed. Consequently, individual consent was not obtained.

Data collection

Detailed information on the demographics, clinical findings, and sexual and non-sexual health risk behaviours were extracted from the SSHC computerised database. This information was originally recorded on standardised medical records and entered into the database after verification by trained staff. Missing information was retrieved from the clinical case notes. To avoid selection bias, no patient was excluded from the study irrespective of the completeness of data. A wide range of demographic, clinical, sexual and non-sexual risk behaviour data were evaluated. These included age; marital status (never married, married/de facto, separated/divorced/widowed); occupation; ethnic origin; country of birth; contraception; parity; number of male and female sexual partners in the past 3 months, 12 months and lifetime; condom use; cigarette smoking; alcohol intake; injecting drug use (IDU); and concurrent STI diagnoses. Age was grouped into a categorical variable (⩽19, 20–29, 30–39, ⩾40 years) for analysis. Condom use was classified as none, sometimes (<50% of times), usually (>50% of times), and always (100% of times). Cigarette smoking was classified as 0, 1–9, 10–19, ⩾20 cigarettes per day and alcohol intake as ⩾140 g per week or ⩽140 g per week. STIs included gonorrhoea, chlamydia, genital herpes, genital human papillomavirus, PID, trichomonas, syphilis, and candidiasis. The diagnosis of BV was based on Spiegel's Gram stain criteria and on the presence of clue cells on microscopic examination of the vaginal smear.

Statistical methods

Data cleaning, manipulation, and statistical analysis were undertaken using SPSS and SAS. Comparisons between

patients and controls were assessed using Pearson χ^2 test. Univariate analysis was used to calculate crude odds ratio (OR) and 95% confidence interval (CI). A backwards elimination logistic regression model for multivariate analysis was used to establish which association with BV persisted. The variables included in multivariate analysis were those significant on univariate analysis at $p<0.05$ and those considered important based on published literature. The variables significant at $p<0.01$ in the final logistic regression model were analysed individually to obtain unadjusted OR and as a group to obtain adjusted OR. A test for trend was performed on factors found significant on multivariate analysis.

RESULTS

Of the 15 567 first clinic women who attended SSHC during the study period, 890 (5.7%) were diagnosed with BV. Table 1 compares the demographic characteristics, obstetric history, and use of contraception of women with BV and controls. The demographic variables comparing women with BV and controls were similar. However, women with BV were statistically significantly more likely to be separated, divorced, or widowed ($p = 0.01$), more likely to be unemployed ($p = 0.04$), and more likely to be have been born in Asia ($p = 0.05$) than controls although the differences between the groups were small.

Women with BV were significantly less likely to be using hormonal contraception ($p<0.0001$), more likely to use IUD ($p = 0.003$) or nil/inadequate contraception ($p = 0.02$) than those who did not have the condition. Patients with BV were more likely to have a past pregnancy than controls ($p<0.001$).

Concurrent STIs and sexual risk behaviour (table 2)

Women with BV had a significantly higher rate of concurrent PID (OR = 2.50, 95% CI: 1.38 to 1.64, $p = 0.002$) and a lower rate for genital herpes (OR = 0.50, 95% CI: 0.34 to 0.72, $p = 0.002$) and candidiasis (OR = 0.70, 95% CI: 0.50 to 0.97, $p = 0.03$) than controls.

Women with two or more male partners in the past 3 months (OR = 1.50, 95% CI: 1.18 to 1.85) and three or more in the past 12 months (OR = 1.60, 95% CI: 1.27 to 2.02) were significantly more likely to have BV than those with fewer partners. Women with one or more female partners in the last 12 months (OR = 2.30, 95% CI: 1.48 to 3.54) and two or more in their lifetime (OR = 2.00, 95% CI: 1.32 to 2.95) were more likely to have BV than those with fewer or no female sexual partners. Women with BV were significantly less likely to always use condoms in comparison to women without BV (OR = 0.70, 95% CI: 0.51 to 0.85).

Social risk behaviour (table 3)

Women with BV were significantly more likely to consume higher quantities of alcohol ⩾140 g per week (13% *v* 10%, OR = 1.4, $p = 0.04$), to report injecting drug use in the past (8% *v* 4%, OR = 0.4, $p<0.0001$), and to be smokers than those without BV (51% *v* 36%, OR = 1.8, $p<0.0001$). In addition, the number of cigarettes smoked per day by women with BV was significantly higher than controls.

Multivariate analysis (table 4)

Age, occupation, marital status, hormonal contraception, IUD, nil/inadequate contraception, past pregnancy, male sexual partners (past 12 months), ⩾1 female sexual partners (past 12 months), condom use, cigarettes per day, alcohol intake, and IDU were included in multivariate analysis. Genital HSV, PID, termination of pregnancy, miscarriage, and live births, though found significant on univariate analysis, were not included independently in the logistic regression as they were not considered causal/risk variables for BV.

Table 1 Demographic characteristics, contraceptive use, and obstetric history of women with BV compared with controls (univariate analysis)

Characteristic	Women with BV n = 890 (%)	Controls n = 890 (%)	Odds ratio (95% CI)	p Value (overall)
Age (y)				(NS)
⩽19	51/889 (6)	67/890 (8)	0.80 (0.52 to 1.11)	NS
20–29	530/889 (60)	527/890 (59)	1	–
30–39	223/889 (25)	213/890 (24)	1.00 (0.83 to 1.30)	NS
⩾40	85/889 (9)	83/890 (9)	1.00 (0.74 to 1.41)	NS
Marital status				(0.005)
Never married	592/867 (68)	601/864 (69.5)	1	–
Married/de facto	139/867 (16)	169/864 (19.5)	0.80 (0.65 to 1.07)	NS
Separated/divorced/widowed	136/867 (16)	94/864 (11)	**1.50 (1.10 to 1.96)**	0.01
Occupation				(<0.05)
Employed	450/852 (53)	492/859 (57)	1	–
Unemployed	151/852(18)	124/859 (14)	**1.30 (1.02 to 1.74)**	<0.05
Benefits	34/852 (4)	23/859 (3)	1.60 (0.94 to 2.79)	NS
Home duty	45/852 (5)	34/859 (4)	1.50 (0.91 to 2.30)	NS
Student	112/852 (13)	138/859 (16)	0.90 (0.67 to 1.74)	NS
Sex work	60/852 (7)	48/859 (6)	1.40 (0.92 to 2.04)	NS
Country of birth				(NS)
Australia/NZ	452/832 (54)	504/833 (61)	1	–
N America/Europe	198/832 (24)	169/833 (20)	1.10 (0.43 to 2.74)	NS
Asia	132/832 (16)	123/833 (15)	**1.30 (1.00 to 1.60)**	0.05
Others	51/832 (6)	36/833 (4)	1.20 (0.88 to 1.52)	NS
Contraceptive used				
Hormonal	238/835 (29)	307/822 (37)	**0.70 (0.54 to 0.82)**	<0.0001
IUD	45/835 (5)	20/822 (2)	**2.30 (1.34 to 3.90)**	<0.01
Diaphragm/cervical cap	24/835 (3)	24/822 (3)	1	–
Nil/inadequate	256/835 (31)	211/822 (26)	**1.30 (1.03 to 1.59)**	<0.05
Obstetric history				
Past pregnancy	489/865 (57)	391/862 (45)	**1.60 (1.30 to 1.89)**	<0.0001
Termination of pregnancy	291/726 (40)	226/729 (31)	**1.50 (1.20 to 1.78)**	<0.001
Miscarriage	128/723 (18)	88/725 (12)	**1.60 (1.18 to 2.06)**	<0.01
Live birth	184/723 (25)	129/724 (18)	**1.50 (1.19 to 1.88)**	<0.001

NS, not significant.

Table 2 Sexual risk factors of women with BV compared with controls (univariate analysis)

Characteristic	Women with BV n = 890 (%)	Controls n = 890 (%)	Odds ratios (95% CI)	p Value (overall)
Concurrent STIs				
Gonorrhoea	5 (<1)	7 (<1)	0.70 (0.23 to 2.25)	NS
Chlamydia	91 (5)	28 (2)	1.70 (0.91 to 3.07)	NS
Genital HPV	105 (6)	115 (7)	0.90 (0.68 to 1.20)	NS
Genital HSV	45 (3)	86 (5)	**0.50 (0.34 to 0.72)**	<0.001
PID	37 (2)	15 (1)	**2.50 (1.38 to 4.64)**	<0.005
Candidiasis	65 (4)	91 (5)	**0.70 (0.50 to 0.97)**	<0.05
Male sex partners 3 months				(<0.01)
0	134 (15)	128 (14)	1.20 (0.90 to 1.54)	NS
1	519 (58)	581 (65)	1	–
⩾2	237 (27)	180 (20)	**1.50 (1.18 to 1.85)**	<0.001
Male sex partners 12 months				(<0.0001)
0	84 (10)	71 (8)	1.40 (0.99 to 2.00)	NS
1	305 (34)	363 (41)	1	–
2	198 (22)	230 (26)	1.00 (0.80 to 1.31)	NS
⩾3	303 (34)	225 (25)	**1.60 (1.27 to 2.02)**	<0.0001
Male sex partners lifetime*				(NS)
0	71 (8)	72 (8)	–	–
1	45 (5)	59 (7)	–	–
2–5	238 (27)	267 (30)	–	–
⩾6	536 (60)	491 (55)	1	–
⩾1 Female sex partner 12 months	68 (8)	31 (3)	**2.30 (1.48 to 3.54)**	<0.0001
Female sex partners (lifetime)				(<0.01)
0	777 (87)	819 (92)	1	–
1	40 (5)	31 (4)	1.40 (0.84 to 2.10)	NS
⩾2	73 (8)	39 (4)	**2.00 (1.32 to 2.95)**	<0.001
Condom use 3 months				(<0.05)
None	348/831 (42)	302/832 (36)	1	–
Sometimes (<50%)	124/831 (15)	109/832 (13)	1.00 (0.73 to 1.33)	NS
Usually (>50%)	120/831 (14)	126/832 (15)	0.80 (0.62 to 1.11)	NS
Always (100%)	160/831 (19)	212/832 (26)	**0.70 (0.51 to 0.85)**	0.001

*Odds ratios and 95% confidence intervals (CI) not calculated on individual number of lifetime partners because the variable was not significant at p<0.05. NS, not significant.

Factors that were independently associated with BV were ⩾3 male sexual partners in the past 12 months (OR = 1.6, p = 0.001), at least one female sexual partner in the past 12 months (OR = 2.1, p = 0.003), a past pregnancy (OR = 1.5, p<0.0006), and smoking. A test for trend for cigarette smoking was significant, indicating that the risk of BV increased as the number of cigarettes smoked daily increased (trend $\chi^2 = 20.03$ 1 df, p<0.0001). In contrast, women with BV were significantly less likely to have used hormonal contraception (OR = 0.6, 95% CI: 0.51 to 0.81) or to have used condoms consistently (OR = 0.5, 95% CI: 0.31 to 0.71) than controls.

DISCUSSION

This study showed that several sexual, reproductive, and social risk variables were independently associated with BV. Women with a past pregnancy, more than three male sexual partners in past 12 months, one or more female sexual partner in past 12 months, and cigarette smoking were all more likely to have BV, whereas those on hormonal contraception and those using condoms usually or always were less likely to have BV. Cigarette smoking was the strongest independent non-sexual risk factor for BV (OR = 1.9, 95% CI: 1.40 to 2.61) and the risk of BV was directly proportional to the number of cigarettes smoked. Smoking has been found to be associated with BV in previous studies.[14 19–21] Possible pathogenic mechanisms linking cigarette smoking and BV need further exploration. One possible explanation is that cigarette smoke contains various chemical constituents like nicotine, cotinine, and benzo[a]pyrene diol epoxide (BPDE). These chemicals have been demonstrated in cervical mucus of smokers and may directly alter the vaginal microflora or may act by depleting Langerhans cells in cervical epithelium leading to local immunosuppression.[23] This may be responsible for change of cervical flora causing BV.

BV was independently associated with a number of sexual factors, including multiple male sexual partners in last 12 months and one or more female sexual partner in past 12 months. Other studies have also reported an association

Table 3 Non-sexual risk behaviour of women with BV compared with controls (univariate analysis)

Characteristic	Women with BV n = 890 (%)	Controls n = 890 (%)	Odds ratio (95% CI)	p Value (overall)
Alcohol intake (⩾140 g/week)	113 (13)	85 (10)	**1.40 (1.02 to 1.85)**	<0.05
Smoker	453 (51)	323 (36)	**1.80 (1.50 to 2.20)**	<0.0001
Cigarettes per day				(<0.0001)
0	437 (49)	566 (64)	1	–
1–9	134 (15)	112 (12)	**1.60 (1.71 to 2.05)**	<0.01
10–19	148 (17)	116 (13)	**1.70 (1.26 to 2.17)**	<0.001
⩾20	171 (19)	95 (11)	**2.30 (1.76 to 3.09)**	<0.0001
Injecting drug use	69/885 (8)	34/873 (4)	**1.40 (1.02 to 1.85)**	<0.0001

Table 4 Risk variables independently associated with BV

Characteristic	Adjusted OR (95% CI)*	p Value
Hormonal contraception	**0.60 (0.51 to 0.81)**	<0.0001
Past pregnancy	**1.50 (1.18 to 1.80)**	<0.001
Male partners (past 12 months)		
0	1.20 (0.78 to 1.90)	NS
1	1	–
2	1.00 (0.78 to 1.35)	NS
⩾3	**1.60 (1.19 to 2.04)**	0.001
⩾1 female partner (past 12 months)	**2.00 (1.27 to 3.33)**	<0.01
Condom use (past 3 months)		
None	1	–
Sometimes	0.80 (0.59 to 1.14)	NS
Usually	**0.60 (0.43 to 0.83)**	<0.01
Always	**0.50 (0.3 to 0.71)**	<0.001
Cigarettes per day		
0	1	–
1–9	**1.40 (1.03 to 1.89)**	<0.05
10–19	**1.50 (1.09 to 2.01)**	<0.05
⩾20	**1.90 (1.40 to 2.61)**	<0.0001

*Adjusted for all other variables in table using logistic regression. NS, not significant.

between BV and sexual factors, including the lifetime number of sexual partners, recent partner change, and lower age of first intercourse.[14–18, 23] One possible explanation is that there is an exogenous ''factor'' in semen that may create an imbalance in vaginal microflora necessary for the development of BV or transmission of an as yet unknown STI. Exposure to this may occur more frequently in women who have more partners and in those who use condoms inconsistently. The observation that women who used condoms consistently were less likely to have bacterial vaginosis suggests that condoms may be useful both in preventing BV and also in reducing the likelihood of recurrences in women prone to the infection. A randomised controlled study to evaluate the latter suggestion should be considered.

Several studies have reported on the increased incidence of BV in women who have sex with women (WSW).[24–27] The pathogenesis of BV in WSW is not clear. McCaffrey *et al* were not able to link any specific sexual practice to BV in lesbians,[25] although receptive cunnilingus has been recently linked to BV in WSW.[28] One possible explanation is that there are many similarities between the anaerobic bacteria associated with gingivitis and those associated with BV. Detailed studies are required in this group of WSW to delineate their propensity to develop BV.

We found that gravidity was independently associated with BV. In addition, on univariate analysis, we found that termination of pregnancy, miscarriage, and live birth were each associated with increased risk of BV. We are unaware of any previous published data showing similar findings, though women who are diagnosed with BV during pregnancy are more likely to have a termination than those who are not.[29] The reasons for this finding are unclear but may be related to changes in the vaginal flora as a consequence of hormonal factors or perhaps changes in sexual behaviour during pregnancy. Little is known about sexual behaviour during pregnancy although a recent study suggested that cunnilingus was common.[30] In addition, there is some evidence to suggest that male partners commonly have unprotected vaginal sex with other women during their partner's pregnancy.[31]

Women using hormonal contraceptives were found to be at a lower risk for BV, as has been reported in many previous studies,[15 32] but interestingly have a high risk of acquiring chlamydia and vaginal candidiasis.[31] Oestrogens stimulate vaginal epithelial cells to produce more glycogen. This creates a more favourable environment for lactobacilli and thus may prevent colonisation by anaerobes, although a clear mechanism still remains undefined. Previous studies have demonstrated positive relation between IUD and BV but in our study IUD, though significant on univariate analysis, was not independently associated with development of BV.[32]

The strengths of this study include the large sample size, the diversity of the sexual reproductive and social risk variable history we were able to obtain, the study design, and the statistical analysis we were able to perform. Consequently, we believe that this study has helped to clarify the diverse and often conflicting risk variable information about BV.

Potential limitations of the study include the use of data that were derived from an existing database with the possibility of less rigorous standards of diagnosis for BV and other conditions than would be possible in a prospective study. Additionally, it was not possible to obtain information on certain factors of potential relevance, such as cunnilingus. Secondly, in common with some previous studies, this was a ''convenience'' sample. Finally, although a number of factors were found to be associated with BV, the small odds ratios suggest that the individual predictive values maybe low.

In conclusion, we found both sexual and non-sexual risk factors to be important in the possible causation of BV. We were able to confirm the link between cigarette smoking, multiple male sexual partners in past 12 months, ⩾1 female sexual partner in past 12 months, and infrequent condom use with BV in women attending sexual health clinic in Australia, along with the protective effect of hormonal contraception. In addition, we report association of BV with past pregnancies. More studies may be needed to confirm and evaluate the cause of this association. Our findings may be important for planning a preventive strategy for BV by discouraging smoking and increasing condom use and hormonal contraception among women.

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