Original Article

Risk Score for Antenatal Bacterial Vaginosis: BV PIN Points

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OBJECTIVE:
Develop a clinical risk score to screen for antenatal bacterial vaginosis (BV), irrespective of symptoms.

STUDY DESIGN:
Cohort study of 913 pregnant women with last menstrual periods between January 30, 1995 and February 22, 1997. BV was evaluated by Nugent-scored vaginal smears (scores of 7 to 10 considered positive) between 24 and 29 weeks' gestation. Forty-four potential risk factors were assessed.

RESULTS:
17.8% of women had BV, of whom 22% were screened for BV by the usual care provider. Logistic regression–adjusted analyses found six predictors: vaginal pH > 4.5 (OR = 11.6, 95% confidence interval [CI] [7.8, 17.2]); black race (OR = 1.9, 95% CI [1.3, 2.8]); condom use during pregnancy (OR = 1.6, 95% CI [1.0, 2.5]); antenatal BV (OR = 1.7, 95% CI [1.0, 2.8]); absence of sperm on smear (OR = 1.7, 95% CI [1.0, 2.9]); and no history of sexually transmitted diseases (OR = 1.6, 95% CI [1.0, 2.5]). Risk score weights were 5 for an elevated vaginal pH and 1 otherwise. The sensitivity and specificity of screening women with scores ≥ 4 were both 77%; this would involve screening 33% of patients.

CONCLUSION:
Approximately 80% of our BV cases were asymptomatic, emphasizing the need for objective risk assessment. Using six factors, clinicians can identify pregnant women at risk for BV.
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INTRODUCTION

Bacterial vaginosis (BV), an infection caused by an overgrowth of *Gardnerella vaginalis*, mixed anaerobes, and/or genital mycoplasms,[1] is the most common vaginal syndrome in reproductive-age women. In the typical clinic setting, both during and outside of pregnancy, women are only tested for BV if symptomatic. However, 35% to 75% of BV cases in nonpregnant women are asymptomatic.[2–6]

BV is associated with 1.5- to 4.0-fold increased risk of spontaneous preterm delivery.[7–12] BV is also associated with approximately twice the likelihood of preterm labor,[9,12–14] three times the risk of premature rupture of the membranes,[13–15] and twice the likelihood of chorioamnionitis/amniotic fluid infection.[14,16,17]

Given the association with preterm delivery, it could be argued that all women in prenatal care should be screened for BV. In fact, some obstetricians have proposed universal screening and systemic treatment of BV during pregnancy,[18] although others[19] argue against such policy. Proposals for screening raise several issues: when in pregnancy should women be screened, when in pregnancy should treatment occur, are screening and treatment only aimed at reducing preterm delivery, and are screening and treatment worthwhile for maternal health among asymptomatic women? Screening and treating all pregnant women may not be practical or desired, yet, however, the current policy of testing only symptomatic women bypasses many true BV cases. Alternative protocols between these extremes are worth exploration, but how should a clinic decide whom to screen?

This study was conducted to develop a risk score with practical, clinical value for identifying subgroups of pregnant women at increased risk for BV so that those women can be screened regardless of symptomatology. We also calculated the proportion of true BV cases that were clinically tested (clinicians were blind to true BV status) as a surrogate measure of the proportion of pregnant women with symptoms.

MATERIALS AND METHODS

The Pregnancy, Infection, and Nutrition Study is a cohort study of pregnant women receiving prenatal care at three NC, USA sites. This residential area is typically suburban, and study details have been previously published.[20,21] Women were eligible if they had a singleton pregnancy, were at least 16 years old, spoke English, entered prenatal care before 24 weeks' gestation, planned to deliver at one of the participating facilities, and were willing to participate in the full study protocol, including pelvic examination, blood sample

Table 1 Population Description

	n	Percentage*
Total	1257	
Age		
≤20 yrs	206	17
20–30 yrs	756	61
>30 yrs	280	23
Married	508	40
≤12 yrs education	739	59
Race		
White, non-Hispanic	585	47
African American	596	47
All other	75	6
Nulliparous†	547	48
Smoker at specimen collection	265	22
Public clinic	1104	88

*Percentage excludes missings.
†Livebirths and/or stillbirths.

collection, and telephone interview. The study was approved by all affiliated institutional review boards, and informed consent was obtained at the time of recruitment.

Among the prenatal care recipients whose last menstrual period was between January 30, 1995 and February 22, 1997 (based on either ultrasound dating or reported last menstrual period), 2527 were eligible for the study, and 1456 (57.6%) were successfully recruited. Women had similar patient attributes and risk of preterm delivery regardless of recruitment status.[20] Our population size was 1257 after dropping 4 women who had taken antibiotics in the 14 days before specimen collection according to the medical chart (no women had taken antibiotics 15 to 21 days before specimen collection), 191 without BV results due to logistical delays, and 4 subsequent pregnancies to women whose previous pregnancy was in the cohort.

BV was assessed at the time of enrollment between 24 and 29 weeks' gestation. The mean gestational age at enrollment was 27.2 weeks and did not differ by BV status. BV was diagnosed with vaginal smears, which were Gram stained on an automated processor. Each vaginal smear was evaluated and scored according to Nugent et al.[22] blinded to any other information about the patient except vaginal pH (4.0 to 7.0). This scoring system counts the individual frequency of lactobacilli, *G. vaginalis*/Gram-negative rods, and *Mobiluncus* species. Weights are assigned per species, and summed to give one final score ranging from 0 to 10. Gram stain scores of 7 to 10 were considered positive for BV, and Gram stain scores of 0 to 3 (''normal'') and 4 to 6 (''intermediate'') were the referent. Scoring was performed by one of us (T. J.) and Janice French, CNMW (University of Colorado Health Science Center). Quality assurance strategies included rereading a sample of slides stratified by Nugent score; our criterion of at least 80% agreement by BV level (normal, intermediate, and definite) was met.

An extensive telephone interview was conducted, which provided data on medical history, antenatal medical events, demographics, behaviors, life events, diet, sexual activity, employment history, and physical activity during pregnancy. Laboratory results of antenatal STD cultures, urine cultures, and sickle cell/beta thalassemia hemoglobinapathies were also available. Within our cohort, a nested case (preterm deliveries)/control subset of 327 women had their medical charts abstracted, which provided additional detail on medical history, antenatal medical events, and prescribed medications. Blank fields from the chart review were presumed to be negative diagnoses. For all antenatal risk factors, a positive diagnosis was presumed if indicated on the medical chart, laboratory results, interview, and/or if there was a medication prescribed that was specific to only one medical condition, all subject to having occurred on or before the day of the genital tract specimen collection. For all medical history factors, any medical chart notation and/or positive self-report were coded as positive.

Forty-four potential risk factors were identified. The literature is limited in its assessment of potential risk factors for BV and, thus, we wanted to explore a range of factors that might influence vaginal ecology. All variables were dichotomized for analysis. For parity, gravidity, and maternal age, the distribution of varying exposure levels by BV status was examined. Five parity/gravidity schemes were explored in the unadjusted analyses, and only one met the criteria for inclusion in the adjusted models. For maternal age, the prevalence of BV in general decreased as maternal age increased, but there were insufficient numbers at many ages to fully characterise this relationship. We chose to use two variables to represent three age strata. Any variables resulting in inverse associations were recoded before modeling to ensure beta coefficients greater than zero.

We calculated bivariate associations of each potential risk factor with BV. Two risk scores were developed using logistic regression modeling (SAS Institute, Cary, NC). A ''full model'' was developed using (1) all factors with unadjusted associations with BV≥1.4 or ≤0.8 regardless of statistical significance; (2) factors thought to be important from the literature (vaginal pH,[4] douching,[23–25] race,[10,11,26,27] and multiple sexual partners[27,28]); and (3) subject to a minimum $n=8$ exposed cases. A reduced model was developed from the full model with backwards selection using -2 log likelihood ratio tests after each iteration. Effect modification with race was evaluated through stratified analysis. Although the impact of vaginal pH on the risk of BV was stronger among blacks than

Table 2 Gram Stain Results Compared to Chart Notation of Positive or Negative Clue Cell and KOH Tests

Nugent scored Gram stain	Notation of clue cells and/or KOH tests		
	Yes	No	Total
Positive (7–10)	8	28	36
Negative (0–6)	11	156	167
Total	19	184	203

Table 3 Crude ORs for each Potential Predictor and Gram-Stained BV at Gestational Weeks 24–29

	Number exposed among		Total *N*	OR* (95% CI)
	BV+	BV−		
Antenatal events†				
BV test/dx‡,§	43	109	1257	2.01 (1.37–2.97)
high vaginal pH§,‖	172	228	1248	12.04 (8.51–17.05)
gestational diabetes§	0	8	327	zero cell
vaginal bleeding‡,§	50	222	1257	1.06 (0.75–1.50)
yeast infection‡,§	57	296	1257	0.85 (0.61–1.19)
STDs‡,§,¶	36	135	1257	1.27 (0.85–1.90)
incompetent cvx§	1	5	327	0.87 (0.10–7.58)
cvx treatment§	0	0	327	zero cell
abnormal Pap§	0	6	327	zero cell
cvx cerclage§	1	5	327	0.87 (0.10–7.58)
UTI‡,§,‖,¶	39	177	1257	1.02 (0.70–1.49)
abdominal/pelvic pain‡,§	1	8	1257	0.57 (0.01–4.33)
sickle cell/thalassemia§,¶	9	33	1173	1.27 (0.60–2.69)
protein in urine§	1	10	327	0.43 (0.05–3.40)
edema§	1	0	327	zero cell
Medical history				
BV‡,§	14	50	1257	1.31 (0.71–2.42)
hypertension§	2	9	327	0.97 (0.10–4.85)
diabetes (not gestational)§	2	15	327	0.57 (0.13–2.55)
yeast infection‡	99	525	1257	0.80 (0.60–1.00)
STDs‡,§	60	316	1257	0.83 (0.60–1.15)
UTI§	30	130	327	1.01 (0.58–1.77)
DES§	1	0	327	zero cell
abnormal Pap‡,§	90	359	1257	1.27 (0.94–1.70)
cvx treatment‡,§	27	133	1257	0.93 (0.60–1.44)
nulliparous‡	92	456	1133	0.83 (0.62–1.13)
parity 3+ vs 0–2	21	67	1133	1.50 (0.90–2.40)
parity 4+ vs 0–3	7	16	1133	2.00 (0.80–4.90)
nulligravidity‡	152	660	1131	1.12 (0.80–1.58)
gravidity 7+ vs 0–6	6	10	1131	2.70 (1.00–7.60)
Demographics/behaviors				
douching‡	12	40	1131	1.37 (0.71–2.66)
teenage versus 20–30 yrs old‡	49	157	963	1.43 (0.98–2.06)
age >30 vs 20–30 yrs old‡	38	242	1037	0.71 (0.49–1.06)
single versus married‡	104	407	1019	1.68 (1.20–2.35)
living with partner versus married‡	53	184	746	1.90 (1.27–2.83)
black versus white/other‡	144	452	1257	2.32 (1.72–3.14)
education ≤12 vs >12 yrs‡	150	589	1252	1.56 (1.15–2.12)
smoking at conception‡	73	306	1133	1.11 (0.81–1.53)
smoking at specimen collection‡	55	210	1216	1.27 (0.90–1.78)
>1 sex partner since conception‡	11	38	1129	1.31 (0.66–2.62)
≥3 intercourse in last month‡	45	188	1122	1.08 (0.75–1.56)
"high" frequency of sex in first trimester‡	60	238	1050	1.18 (0.84–1.66)
sperm in vaginal smear‖	22	152	1089	0.64 (0.40–1.38)
condom during pregnancy‡	56	150	1132	1.93 (1.36–2.75)
spermicide use during pregnancy‡	4	3	1084	6.10 (1.02–41.67)
prior use of oral contraceptives‡	174	761	1132	1.17 (0.77–1.77)

(continued on next page)

Table 3 (*Continued*)

	Number exposed among		Total *N*	OR* (95% CI)
	BV+	BV−		
prior use of norplant/depoprovera‡	56	217	1132	1.22 (0.87–1.72)
High BMI versus low BMI#	57	260	1058	1.11 (0.78–1.57)
Prenatal care began in first trimester‡	120	537	820	1.28 (0.80–2.07)
private versus public care§	10	142	1257	0.29 (0.15–0.57)

*OR calculated using Fisher Exact tests when any non–zero cell had five or fewer women.
†Diagnosed on or before specimen collection day.
‡Self-reported through questionnaire.
§From medical charts.
||From specimen collection.
¶From prenatal laboratory records.
#From screener questionnaire supplemented with medical records.

whites, the impact on the risk score was negligible because vaginal pH was the strongest predictor within both racial groups. Hence, for simplicity, this effect modifier is not included in the risk scores.

Risk scores were developed using beta coefficients of the logistic models (as opposed to risk estimates), because the coefficients have an additive relationship and, thus, can be summed for clinical applications. The reduced model risk score was further simplified for clinic use by dividing by 5 so that the weights for each factor ranged from 1 to 5 rather than from 5 to 25. Receiver operating characteristic curves were developed and interpreted according to Hanley and McNeil[29] to evaluate the discriminating power of the two risk scores. These curves illustrate the ability of the risk score to distinguish women truly at increased risk for BV from those not at increased risk. The area between each curved line and the diagonal, as measured by Wilcoxon statistics, is the probability of correctly identifying patients at increased risk for BV. Sensitivity, specificity, and efficiency (true positives plus true negatives divided by the grand total) were calculated for each score threshold. Positive and negative predictive values were not calculated; because they are dependent on the prevalence of each risk factor, these figures would not be generalizable to other populations. Lastly, the reduced model was validated with bootstrap techniques using 50 samples of 200 subjects

Table 4 Logistic Regression–Adjusted ORs (AOR) for Association with BV at Gestational Weeks 24–29

	Full model		Reduced model	
	AOR (95% CI)	Risk score	AOR (95% CI)	Risk score
	$n = 958$		$n = 930$	
	model $p = 0.0001$		model $p = 0.0001$	
Antenatal BV test/dx	1.63 (0.98–2.72)	5	1.70 (1.03–2.80)	1
High vaginal pH	11.82 (7.83–17.83)	25	11.55 (7.76–17.19)	5
No history of yeast infection	1.19 (0.81–1.76)	2	–	
No history of STD	1.67 (1.08–2.59)	5	1.62 (1.06–2.48)	1
parity ≥3 vs 0–2	1.19 (0.61–2.32)	2	–	
Black versus white/other	1.86 (1.20–2.89)	6	1.91 (1.29–2.81)	1
Education ≤12 vs >12 yrs	1.46 (0.94–2.27)	4	–	
Maternal age 20–29 vs 20	1.27 (0.74–2.20)	2	–	
Maternal age ≥30 vs <20	1.38 (0.68–2.80)	3	–	
Married versus single	1.05 (0.62–1.78)	1	–	
Living with partner versus single	1.43 (0.84–2.45)	4	–	
Douching during pregnancy	1.29 (0.53–3.10)	3	–	
Condom during pregnancy	1.52 (0.95–2.44)	4	1.58 (1.01–2.47)	1
No sperm on the vaginal smear	1.64 (0.92–2.91)	5	1.67 (0.95–2.91)	1
>1 sex partner since conception	2.04 (0.85–4.88)	7	–	
Private versus private clinic	1.01 (0.40–2.55)	0	–	

AOR = adjusted odds ratio; CI = Confidence interval.

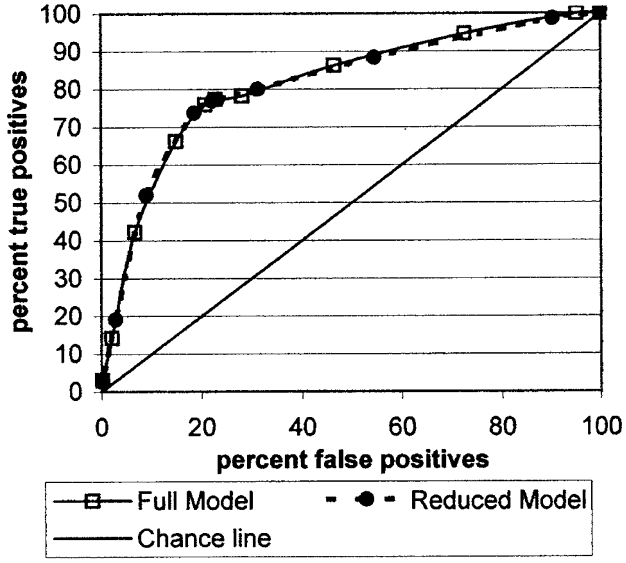

Figure 1. Receiver operating characteristic curves for full and reduced models.

chosen randomly with replacement from the cohort. From these 50 samples, the mean and median Wilcoxon statistic, sensitivity, and specificity were calculated.

RESULTS

Women ranged in age from 16 to 44 years with a mean of 25.7 years. Half were African American, and 22% were smokers at the time of specimen collection (Table 1). Characteristics of those with abstracted medical charts were quite similar to the full cohort (data not shown). Among African Americans in our cohort, 24% had 11 or fewer years of education and 36% had some college education (data not shown). The corresponding percentages for whites were 23% and 32%, respectively.

At 24 to 29 weeks' gestation, 17.8% of women had positive BV Gram stain results and another 11.3% had intermediate Nugent scores of 4 to 6. We evaluated the frequency of a chart notation of positive or negative clue cell and/or KOH whiff tests as a surrogate for symptoms that would warrant clinical testing. (In the clinics that participated in this study, it is routine practice to clinically assess BV[4] if the patient is symptomatic, either as self-reported or observed clinically.) Within the subset of 203 women with medical chart data from the same day as the vaginal smear, 9% ($n = 19$ of 203) were clinically assessed for BV as evidenced by a notation of a clue cell and/or KOH test (Table 2). (None of the chart abstracts indicated vaginal pH.) By Gram stain result, notations of tests were recorded on 22% ($n = 8$ of 36) of BV case charts: 1 had positive results for both tests, 4 had positive clue cells and negative KOH, 1 had negative clue cells and positive KOH, and 2 were negative for both tests. Hence, 78% of the true BV cases were likely to be asymptomatic, based on this subsample. Among noncases, 7% ($n = 11$ of 167) had notations of being clinically tested for BV, of which 3 had positive clue cells and negative KOH, and 8 were negative for both.

Unadjusted odds ratios (ORs) (Table 3) indicated that the strongest single risk factor was a vaginal pH > 4.5 (OR = 12.0, 95% confidence interval [CI] 8.5 to 17.1), followed by use of spermicide during the index pregnancy (OR = 6.1, 95% CI 1.0 to 41.7). Other factors with unadjusted ORs of at least 2.0 were: antenatal BV before specimen collection, parity of 4 or more, gravidity of 7 or more, and black race. Factors with OR $\leq$ 0.5 were protein in urine and receiving care at a private clinic.

The adjusted full model contained 16 predictors, of which 6 remained in the reduced model (Table 4). A high vaginal pH increased the risk of having BV 11.6- to 11.8-fold across both models (range of 95% CI 7.8 to 17.8), and was the single most important predictor. The final risk scores in the full model ranged from 0 to 25 and from 1 to 5 in the reduced model.

The receiver operating characteristic curves for each model are nearly identical (Figure 1). The Wilcoxon statistic corresponding to the area between each curved line and the diagonal is nearly identical for each model: 0.817 (standard error [SE] = 1.6%) for the full model, and 0.811 (SE = 1.7%) for the reduced model. The bootstrap samples yielded mean = 0.808 and median = 0.809 Wilcoxon statistics (data not shown).

The graphs of sensitivity, specificity, and efficiency for the full and reduced models are quite similar (reduced model displayed in Figure 2). Sensitivity and specificity are both maximized where the lines cross, which is at score thresholds of 4 and 25 for the reduced and full models, respectively. Using the reduced model and a score threshold $\geq$ 4 would prompt a clinic to screen one third of their pregnant patients for BV (wet preparations and vaginal pH per Amsel et al.[4]). Seventy-seven percent of the true BV cases would be identified if the clinic used Gram-stained vaginal smears, as was the basis for this analysis. The bootstrap samples yielded mean = 78%

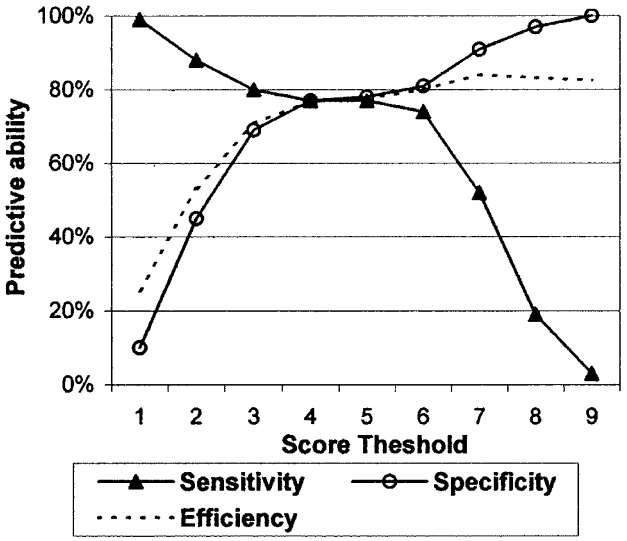

Figure 2. Sensitivity, specificity, and efficiency of reduced model.

and median = 79% sensitivities and mean = 73% and median = 72% specificities, all at a score threshold of 4 (data not shown).

DISCUSSION

Our study explored the ability of 44 potential risk factors to predict antenatal BV. Six factors were found to be most strongly predictive: an elevated vaginal pH, black race, antenatal BV earlier in the index pregnancy, condom use during pregnancy, the absence of sperm on the vaginal smear, and no STD history. A practical, clinical risk score can be determined for individual patients using a weight of 5 for an elevated vaginal pH and a weight of 1 for the remaining five factors. Note that a patient query about intercourse in the prior 24 hours would substitute for ''sperm on the vaginal slide'' in practice.

To our knowledge, no BV risk score now exists for pregnant or nonpregnant women, although a nomogram for determining the value of universal prenatal screening has been published.[30] The need for an objective clinical risk score for BV is underscored by our estimate of no symptoms in almost 80% of our BV cases, although this estimate is based on only a small subset of our population. If our estimate is true among pregnant women in general, this implies that the current clinical policy of only testing symptomatic women for BV ignores approximately 80% of the true antenatal BV cases.

Prior research supports several of the predictors. The association between vaginal pH and BV is well established, as evidenced by the inclusion of vaginal pH in the Amsel et al. clinical criteria.[4] Note that vaginal pH > 4.5 has a sensitivity of 89.3% and a specificity of 73.3% compared with Nugent-scored Gram stains,[31] and 22% of our BV cases had a normal vaginal pH. Hence, an elevated vaginal pH is not a necessary condition for BV despite its high score in our proposed system. Pregnant African Americans have increased prevalence of BV compared to other race/ethnicity groups.[10,11,26,27] Antenatal BV can recur and/or persist despite treatment,[9,32,33] thus lending credibility to the inclusion of previous BV diagnoses in the index pregnancy.

The condom, STD history, and sperm presence predictors warrant examination. Theoretically, these three factors could be related through sexual behavior. In our data, the presence of sperm on the vaginal smear, which indicates unprotected intercourse in the prior 24 hours, was as likely in women reporting condom use during the pregnancy as those denying condom use (13% vs 16%, respectively, $p = 0.35$). However, a history of STD was more likely in the condom users than in women who did not report condom use (39% vs 30%, respectively, $p = 0.02$). The impact on the reduced model from dropping the STD history variable was a significant change in the -2 log likelihood statistic, but the receiver operating characteristic Wilcoxon statistic was within 0.001 of the reduced model. Aside from the statistics, the association between BV and use of a condom during pregnancy may indicate an effect of latex or may be related to first trimester vaginal bleeding. The inverse association between sperm (recent intercourse) and BV may indicate that intercourse has a positive influence on vaginal ecology, or it may be a marker for a lack of vaginal symptoms, or it may be a marker for better overall health. The protective influence of an STD history might be due to the prescription of antibiotics in the past, which also eradicated BV.

Study limitations include potential bias from only having 52% of the eligible population recruited into the study. Analyses of demographic factors, reproductive history, and pregnancy outcomes suggest that refusals were more likely in those with lower education and health department clinic care.[20] The prevalence of BV in these data (17.8%) is similar to prior research,[34] thus suggesting little bias in the outcome of interest in our population.

The scoring system is not necessarily appropriate for BV outside of the 24 to 29 gestational week window. Little is known about the natural history of BV during pregnancy, because few studies have conducted serial assessments during gestation. Further complicating the situation is the fact that 12% to 50% of untreated antenatal BV cases resolve spontaneously,[11,35,36] and 9% to 20% of antenatal BV can recur and/or persist 4 to 12 gestational weeks later despite treatment.[9,32,33]

Self-reported and clinician-identified vaginal symptoms at the time of enrollment would have helped to better assess the interrelationships between symptomatology, clinician-initiated BV testing, and Gram stain BV results. This information would enhance our assessment of the value of our scoring system and possibly add to our understanding of typical BV symptoms during pregnancy.

We were unable to fully exclude women who had taken antibiotics in the 14 days before BV assessment, because this information was only available on the 327 women with medical record abstracts. Assuming (1) all women with antibiotic prescriptions actually take the medication, (2) the proportion of antibiotic use in our subsample with medical chart data can be extended to the full cohort, and (3) antibiotics in general reduce the incidence of BV even though metronidazole and clindamycin are the only oral antibiotics prescribed for BV, then approximately 1.2% of the cohort without medical charts, or 11 women, are erroneously included in our analysis. If these 11 women are less likely to have BV due to recent/current antibiotic use, then their inclusion biases our results toward the null.

It is possible that recording vaginal pH on the vaginal smear slide biased the Nugent score. In reviewing the data, we found that 22% of our cohort with BV had a normal vaginal pH, and 57% of women with a high pH did not have BV.

It is also possible that there are other important risk factors not included in our model, either due to noninclusion in our analysis or nonascertainment by the PIN study instruments, which have not yet been identified by the medical community. The impact of such omissions on the risk score is unknown.

Study strengths include the fact that BV was objectively measured with the gold standard Gram stain test for all subjects. All variables in the final reduced model were available from over 900 women, so the restriction of medical chart data to only a nested case–control subset did not influence the statistical power of the final results. Our data represent an unselected cohort of both public and private obstetric

patients with diversity in race in both clinic settings, and was not limited to women with either symptoms of vaginal infection or symptoms of preterm labor/delivery.

CONCLUSION

Development of this risk score will help all pregnant women with BV, especially those without symptoms, to be screened and treated. As previously reviewed,[34] some treatment trials have implied that treating women for BV during pregnancy could reduce the chance of preterm delivery, whereas others have not found this benefit. In current clinic practice, women are only screened for BV if symptomatic (both during and outside of pregnancy). Because many BV cases are asymptomatic, there is a need for an objective tool to improve the screening process. Vaginal pH alone is an insufficient clinical screening tool. Not all women with a high vaginal pH have BV, and not all women with BV have a high pH. A high vaginal pH is not a necessary requirement for a positive BV diagnosis per Amsel et al.[4] and is not part of the BV Gram stain assessment per Nugent et al.[22] The optimum balance of screening sensitivity and expense management can be determined by individual clinics using information in this manuscript. Clinics who want to screen pregnant women for BV should also consider the additional effort required by selective screening relative to universal screening. Prospective examination of this scoring system is underway in two North Carolina public health department prenatal clinics.

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References

1. Hill GB. The microbiology of bacterial vaginosis. Am J Obstet Gynecol 1993;169:450–4.
2. Bump RC, Buesching WJ. Bacterial vaginosis in virginal and sexually active adolescent females: evidence against sexual transmission. Am J Obstet Gynecol 1988;158:935–9.
3. Spiegel CA, Amsel R, Eschenbach D, Schoenknecht F, Homes KK. Anaerobic bacteria in nonspecific vaginitis. N Engl J Med 1980;303:601–7.
4. Amsel R, Toeen PA, Spiegel CA, Chen KCS, Eschenbach D, Holmes KK. Nonspecific vaginitis. Diagnostic criteria and microbial and epidemiologic associations. Am J Med 1983;74:14–22.
5. Embree J, Caliando JJ, McCormack WM. Nonspecific vaginitis among women attending a sexually transmitted diseases clinic. Sex Transm Dis 1984;11:81–4.
6. Eschenbach DA, Hillier S, Critchlow C, Stevens C, DeRouen T, Holmes KK. Diagnosis and clinical manifestations of bacterial vaginosis. Am J Obstet Gynecol 1988;158:819–28.
7. Martius J, Krohn MA, Hillier SL, Stamm WE, Holmes KK, Eschenbach DA. Relationships of vaginal *Lactobacillus* species, cervical *Chlamydia trachomatis*, and bacterial vaginosis to preterm birth. Obstet Gynecol 1988;71:89–95.
8. Riduan JM, Hillier SL, Utomo B, Wiknjosastro G, Linnan M, Kandun N. Bacterial vaginosis and prematurity in Indonesia: association in early and late pregnancy. Am J Obstet Gynecol 1993;169:175–8.
9. McGregor JA, French JI, Jones W, et al. Bacterial vaginosis is associated with prematurity and vaginal fluid mucinase and sialidase: results of a controlled trial of topical clindamycin cream. Am J Obstet Gynecol 1994;170:1048–60.
10. Meis PJ, Goldenberg RL, Mercer B, et al. The preterm prediction study: significance of vaginal infections. Am J Obstet Gynecol 1995;173: 1231–5.
11. Hay PE, Lamont RF, Taylor-Robinson D, Morgan DJ, Ison C, Pearson J. Abnormal bacterial colonisation of the genital tract and subsequent preterm delivery and late miscarriage. BMJ 1994;308:295–8.
12. Holst E, Goffeng AR, Andersch B. Bacterial vaginosis and vaginal microorganisms in idiopathic premature labor and association with pregnancy outcome. J Clin Microbiol 1994;32:176–86.
13. McGregor JA, French JI, Parker R, et al. Prevention of premature birth by screening and treatment for common genital tract infections: results of a prospective controlled evaluation. Am J Obstet Gynecol 1995;173:157–67.
14. Gravett MG, Nelson HP, DeRouen T, et al. Independent association of bacterial vaginosis and *Chlamydia trachomatis* infection with adverse pregnancy outcome. JAMA 1986;256:1899–1903.
15. McDonald HM, O'Loughlin JA, Jolley P, Vigneswaran R, McDonald PJ. Vaginal infection and preterm labor. Br J Obstet Gynaecol 1991;98:427–35.
16. Krohn MA, Hillier SL, Nugent RP, et al. The genital flora of women with intraamniotic infection. J Infect Dis 1995;171:1475–80.
17. Hillier SL, Martius J, Krohn M, Kiviat N, Holmes KK, Eschenbach DA. A case–control study of chorioamnionic infection and histologic chorioamnionitis in prematurity. N Engl J Med 1988;319:972–8.
18. McGregor JA, French JI. Prevention of preterm birth. NEJM 1998;339:1858–9 (Letter).
19. Goldenberg RL, Rouse DJ. Prevention of preterm birth. NEJM 1998; 339:1860 (Letter).
20. Savitz DA, Dole N, Williams J, et al. Determinants of participation in an epidemiological study of preterm delivery. Paediatr Perinat Epidemiol 1999;13:114–25.
21. Pastore LM, Royce RA, Jackson TP, Thorp Jr JM, Savitz DA, Kreaden US. Association between bacterial vaginosis and fetal fibronectin at 24–29 weeks' gestation. Obstet Gynecol 1999;93:117–23.
22. Nugent RP, Krohn MA, Hillier SL. Reliability of diagnosing bacterial vaginosis is improved by standard method of Gram stain interpretation. J Clin Microbiol 1991;29:297–301.
23. Foxman B, Aral SO, Holmes KK. Interrelationships among douching practices, risky sexual practices, and history of self-reported sexually transmitted diseases in an urban population. Sex Transm Dis 1998;25:90–9.
24. Zhang J, Thomas AG, Leybovich E. Vaginal douching and adverse health effects: a meta-analysis. Am J Public Health 1997;87:1207–11.
25. Rosenberg MJ, Phillips RS, Holmes MD. Vaginal douching: who and why? J Reprod Med 1991;36:753–8.
26. Goldenberg RL, Klebanoff MA, Nugent R, Krohn MA, Hillier S, Andrews WW. Bacterial colonization of the vagina during pregnancy in four ethnic groups. Am J Obstet Gynecol 1996;174:1618–21.

27. Royce RA, Jackson TP, Thorp Jr JM, et al. Race/ethnicity, vaginal flora patterns, and pH during pregnancy. Sex Transm Dis 1999;26: 96–102.
28. Nilsson U, Hellberg D, Shoubnikova M, Nilsson S, Mardh PA. Sexual behavior risk factors associated with bacterial vaginosis and *Chlamydia trachomatis* infection. Sex Transm Dis 1997;24:241–6.
29. Hanley JA, McNeil BJ. The meaning and use of the area under a receiver operating characteristic (ROC) curve. Radiology 1982;143: 29–36.
30. Glantz JC. Screening and treatment of bacterial vaginosis during pregnancy: a model for determining benefit. Am J Perinat 1997;14:487–90.
31. Schwebke JR, Hillier SL, Sobel JD, McGregor JA, Sweet RL. Validity of the vaginal Gram stain for the diagnosis of bacterial vaginosis. Obstet Gynecol 1996;88:573–6.
32. Klebanoff M, Carey JC. Metronidazole did not prevent preterm birth in asymptomatic women with bacterial vaginosis. Am J Obstet Gynecol 1999;180:S2 (SMFM Abstract).
33. Livengood CH, McGregor JA, Soper DE, Newton E, Thomason JL. Bacterial vaginosis: efficacy and safety of intravaginal metronidazole treatment. Am J Obstet Gynecol 1994;170:759–64.
34. French JI, McGregor JA. Bacterial vaginosis: history, epidemiology, microbiology, sequelae, diagnosis, and treatment. In: Borchadt KA, Noble MA, editors. Sexually Transmitted Diseases: Epidemiology, Pathology, Diagnosis, and Treatment. Boca Raton, FL: CRC Press; 1997. p. 3–39.
35. Platz-Christensen JJ, Pernevi P, Hagmar B, Andersson E, Brandberg A, Wiqvist N. A longitudinal follow-up of bacterial vaginosis during pregnancy. Acta Obstet Gynecol Scand 1993;72:99–102.
36. Gratacos E, Figueras F, Barranco M, et al. Spontaneous recovery of bacterial vaginosis during pregnancy is not associated with an improved perinatal outcome. Acta Obstet Gynecol Scand 1998;77:37–40.