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GROUP B STREPTOCOCCUS: PREVALENCE IN A NON-OBSTETRIC POPULATION

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Abstract

Objective: To establish and compare the prevalence of Group B streptococcus (GBS) colonization in the vaginas of non-obstetric women with and without vaginitis.

Materials and Methods: Cross-sectional analysis Group B streptococcus vaginal culture status of non-pregnant, estrogen-replete women ≥ 18 years presenting for annual gynecological exams or vaginal infection. Subjects were classified into 3 groups: no vaginitis (NV) if symptoms were absent and exam was normal, common vaginitis (CV) if microscopic exam revealed yeast, bacterial vaginosis or trichomonads, or inflammatory vaginitis (IV) if exam revealed inflammation and immature squamous cells, but no pathogens.

Results: Of the 215 women recruited: 147 (68.4%) showed no evidence of vaginitis, 41 (19.1%) had CV, and 27 (12.6%) showed evidence of IV. The overall prevalence rate of GBS was 22.8%. Vaginitis was associated with a significantly increased risk of GBS colonization [Adjusted OR: CV 2.7 (1.1-6.2); IV 2.9 (1.1-8.0)]. Logistic regression revealed pH > 4.5 , presence of abnormal discharge on exam and a women's complaint of current symptoms to be significant predictors of the presence of GBS.

Conclusion: GBS colonization occurs more commonly in women with vaginitis. This suggests that disruption of the normal vaginal bacterial environment is an important predictor for GBS colonization.

Keywords

Group B streptococcus; vaginitis; vagina; *Streptococcus agalactiae*; prevalence

Introduction

Streptococcus agalactiae or Group B streptococcus (GBS) is a known vaginal bacterial inhabitant. Previous cross-sectional studies of obstetrical patients have documented that at least 10-30% of women are regularly colonized with this organism in the vagina and rectum¹⁻⁴. Although this organism is a well-defined pathogen in the obstetrical population causing intra-

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amniotic infection, endomyometritis and neonatal infections⁵⁻⁶, its role as a potential causative agent in vaginitis in non-pregnant women is not well understood. Prior studies examining the role of GBS in gynecologic populations have mostly focused on college-aged, sexually active young adults and risk factors associated with colonization of this organism⁷⁻¹².

Common infectious vaginitis (CV) represents a disruption of normal flora followed by colonization with pathogens leading to well-described conditions like vaginal candidiasis, bacterial vaginosis and trichomoniasis. Yet, some women presenting with vaginal complaints do not show evidence of a defined pathogen and remain undiagnosed, even after empiric treatment with anti-infective agents¹³. Little if anything is known about what role GBS plays in the setting of vaginitis. The concept of an aerobic inflammatory vaginitis (IV) due to non-traditional vaginal pathogens such as Group B strep, *Escherichia coli* or other causes has emerged along with recommendations for empiric treatment¹⁴⁻¹⁵.

The current literature does not provide guidance as to whether detection of GBS colonization in women is important for the treatment of difficult vaginitis. As an initial step towards exploring the relationship of GBS in the gynecologic population, we designed a cross-sectional study to establish and compare the prevalence of GBS among non-pregnant estrogen-replete women without vaginitis, with common vaginitis, and with inflammatory vaginitis.

Materials and Methods

The protocol was approved by the Institutional Review Board at Oregon Health & Sciences University (OHSU). Non-pregnant, estrogen-replete women over the age of 18 years presenting for annual gynecological exams or for evaluation for the complaint of vaginal infection at OHSU or Planned Parenthood of the Columbia/Willamette were eligible for this cross-sectional study. Women were excluded if they were currently menstruating or bleeding, were estrogen deficient, had taken DepoProvera in the last 12 months, were currently being treated for vaginal inflammation or infection, had used antibiotics within the last 2 weeks, were lactating without return of regular menses, or had been amenorrheic for longer than 3 months unless taking hormonal contraceptives. Estrogen replete was defined as naturally menstruating, on combined hormonal contraceptives or postmenopausal estrogen or combined hormone replacement. Premenopausal women not using a reliable form of contraception underwent a urine based hCG pregnancy test. Patients with chlamydia or gonorrhea cervicitis were excluded.

All participants underwent a speculum examination to assess for odor, discharge, and vaginal tissue changes including erythema and ecchymoses. A cervical sample for a PCR-based chlamydia/gonorrhea test was obtained if this had not previously been done. The vaginal fornix or vaginal sidewall was sampled using a swab (Amies Starswab II, Etobicoke, Ontario) to collect a specimen for Group B strep culture, and small cotton applicators were used to collect discharge for measurement of pH (Hydriion Papers, Micro Essential Laboratory, Brooklyn, NY) and preparation of samples on glass slides with drops of saline or 10% potassium hydroxide for microscopic examination. All light microscopy was performed by an author. Slides were scanned at 100x and 400x. Pathogens (budding hyphae, spores, motile trichomonads, clue cells) were rated as either present or absent^{13, 16-17}. The PMN score was determined by taking an average of five high powered fields (HPFs) (0 = <1/HPF, 1+ = 1-30/HPF, 2+ = 31-60/HPF, 3+ > 60/HPF). A similar rating system based on five HPFs was used for parabasal cells (0 = 0/HPF, 1+ = 1/HPF, 2+ = 2-3/HPF, and 3+ = > 3/HPF). If clue cells were present, Amsel's criteria were followed¹⁷. The presence, absence or reduction of mature squamous cells and lactobacillus was also recorded. Information collected from this examination was used to classify subjects into the categories of no vaginitis (NV), common vaginitis (CV), or inflammatory vaginitis (IV).

Women were classified as NV if no symptoms were documented, the microscopy was negative for yeast, clue cells, and motile trichomonads, and the PMN and parabasal cell scores were < 1 . Symptomatic or asymptomatic subjects with microscopy positive for yeast, motile trichomonads, or meeting Amsel's criteria for bacterial vaginosis (BV)¹⁷ were classified as CV, while those presenting with microscopic evidence of inflammation (PMN score $\geq 1+$ and increased epithelial cell exfoliation with parabasal cells score $\geq 1+$) in the absence of evidence of a common pathogen were classified as IV. Most of these women had symptoms, typically discharge with or without itching, dyspareunia or odor.

The GBS culture was performed by collecting the specimen from the middle one third of the vagina or vaginal fornix on an Amies swab. This was incubated in s limbroth solution for a minimum of 16 hours at 35° Celsius at the Kaiser Permanente clinical laboratory, and then streaked on a trypticase soy agar plate with 5% sheep's blood and incubated for an additional 24 hours at 35° Celsius before assessing for growth.

Each participant completed a 4-page questionnaire with 28 questions. Participants answered questions about their diet, exercise, smoking, alcohol use, gynecological health history, sexual history, current symptoms, douching, antibiotic use in the last year, description of menses, history of pregnancies, history of contraceptive use, age, race, ethnicity, marital status, education level, and household income.

Data were double-entered into a computer statistical program (SPSS version 15.0 for Windows (Chicago, Ill) for analysis. Frequencies and 95% confidence intervals for GBS colonization were calculated. A chi-square analysis and logistic regression were performed to compare the prevalence of GBS between the groups. Logistic regression was also used to assess predictors of GBS positivity from the clinical findings, reproductive health history and demographic characteristics. A p value of < 0.05 was considered significant.

A sample size calculation was performed assuming that there would be a 24% difference in the prevalence of GBS between women with no vaginitis (20%) and inflammatory vaginitis (44%)¹⁸ (Power and Precision v3.2, Biostat, Englewood, NJ). Assuming a 3:1 enrollment of control: inflammatory vaginitis subjects in order to detect this level of colonization difference at a significance level of 0.05 with 80% power, we planned to enroll 120 healthy women with no vaginitis, and 40 women with inflammatory vaginitis.

Results

During the study period (January 2007-July 2008), 215 women completed enrollment: 147 (68.4%) women in the no vaginitis group, 41 (19.1%) in the common vaginitis group, and 27 (12.6%) in the inflammatory vaginitis group. All 27 women in the inflammatory vaginitis group had PMNs and parabasal cells on saline microscopy. This group was further subdivided based upon the 5 Sobel criteria¹⁸ for DIV: diffuse purulent vaginal discharge, microscopic evidence of PMNs, increased parabasal cells, elevated vaginal pH and reduced or absent lactobacilli. Twenty-one of the 27 (77.8%) IV subjects met at least three of these criteria and were classified as having DIV. Of these, 19/21 (90.5%) participants demonstrated all 5 criteria.

Demographic analysis revealed that most recruits were Caucasian, educated beyond high school, and received care within the faculty practice at OHSU. Women in the no vaginitis and common vaginitis groups tended to be younger while almost one-fifth of the women in the inflammatory vaginitis group were 50 years or older. The majority of women in the common vaginitis group were not married (n=25, 62.5%) while the majority of women in the no and inflammatory vaginitis groups were married (n= 94, 65.3%; n=17, 65.4%, respectively) (Table 1).

Positive GBS cultures were found in 26 of 147 (17.7%) women in the no vaginitis group, 14 of 41 (34.1%) in the common vaginitis group, and 9 of 27 (33.3%) in the total inflammatory group. In the subset of inflammatory vaginitis called DIV, 9 of 21 (42.9%) were GBS positive. Compared to women with no vaginitis, the risk of GBS colonization was significantly increased in women with common vaginitis (adjusted OR= 2.7, 95% CI 1.1-6.2) and inflammatory vaginitis (OR=2.9, 95% CI 1.1-8.0). Women with DIV had a 5-fold increased risk of being GBS positive (OR= 5.1, 95% CI 1.6-16.0) (Tables 2a and 2b).

Univariate analysis of historical characteristics demonstrated that a self-reported history of frequent urinary tract infections (OR=2.1, 95% CI 1.0-4.3), antibiotic use in the last month (OR=4.5, 95% CI 1.3-15.6), complaint of symptoms at time of exam (OR=3.1, 95% CI 1.6-6.1), and complaint of burning and itching at time of exam (OR=3.5, 95% CI 1.6-7.6; OR=2.7, 95% CI 1.2-5.9, respectively) were all predictors of positive GBS status. However, after adjustment for baseline characteristics, only the history of antibiotic use in last 30 days (OR=7.3, 95% CI 1.1-48.2) was a significant predictor of GBS colonization. Notably, a history of chronic vaginal infection with yeast and BV also did not significantly predict GBS positivity.

Multivariate analysis by logistic regression revealed that after adjusting for clinical and demographic characteristics, the presence of elevated vaginal pH and abnormal discharge on physical exam were the only significant predictors of positive GBS status. In particular, pH > 4.5 demonstrated a 4-fold increased risk of GBS positivity (OR=4.3, 95% CI 1.6-11.7) while abnormal discharge demonstrated a 3-fold increased risk (OR=2.9, 95% CI 1.2-7.0) (Table 3).

Clinical factors that did not significantly predict positive GBS status included presence of white blood cells (PMNs), presence of parabasal cells, clue cells, motile trichomonads and hyphae on saline and KOH wet mount.

Discussion

Our results demonstrate that the prevalence of GBS colonization is significantly increased in women with both common and inflammatory vaginitis. Although we expected to see an increased colonization rate of GBS among women with inflammatory vaginitis^{14, 18}, we were surprised to also discover an almost identical three-fold increased prevalence of GBS in common vaginitis (BV, yeast, trichomoniasis). When only those subjects meeting the criteria for desquamative inflammatory vaginitis (DIV) as defined by Sobel were considered, the risk of GBS colonization was increased five-fold with the prevalence estimate (43%) similar to the 44% reported by Sobel¹⁸.

In our study, vaginal ecosystem changes were associated with higher rates of GBS prevalence. Specifically, we found that an elevated pH, presence of abnormal discharge, and women's complaints of vaginitis symptoms significantly predicted GBS status. These factors are surrogates for a disruption in the normal lactobacillus colonization of the vagina¹⁹⁻²¹.

Whether GBS causes or contributes to a clinically symptomatic bacterial vaginitis is controversial. Although 44% of the women with DIV in Sobel's study were colonized with GBS, more than half were not colonized so a causal relationship is suspect. Furthermore, many women with inflammatory vaginitis will respond to anti-inflammatory treatment alone without antibiotics¹⁸. Therefore, the available evidence is insufficient to conclude that inflammatory vaginitis is always of immune or infectious etiology. Our results support the hypothesis that GBS colonization occurs as a result of a series of events that lead to loss of the protective acidic lactobacillus-dominant vaginal environment.

Strengths of our study include the prospective design and careful validation of diagnosis of vaginitis by direct microscopy using standard tools available to clinicians. The cross-sectional

design of our study has some inherent weaknesses. Ascertainment bias is possible, as subjects were assigned to vaginitis groups solely by clinical criteria. Asymptomatic women can commonly be positive for *Candida* on culture²²⁻²⁴. Furthermore, microscopy is insensitive in detection of trichomonads²⁵⁻²⁶. Although fungal cultures and molecular detection techniques for trichomonads would have increased the accuracy of diagnosis, these are not diagnostic tests of great utility to most clinicians. Therefore, our results are representative of the standard office evaluation of the patient. Since the result of misclassification would have been to dilute the main effect, it is unlikely that this would have influenced the main outcome of an increase in GBS colonization in women with vaginitis.

We enrolled fewer vaginitis subjects than expected. Routine use of over-the-counter anti-fungal medications and telephone triage prescriptions in our practice population curtailed our ability to enroll women in the CV group. We also found inflammatory vaginitis distinct from the diagnosis of erosive lichen planus (exclusion) hard to identify and therefore enroll²⁷⁻²⁹. Women who manifested vulvar (introital) and vaginal signs of erosive lichen planus or who were biopsy proven to have the disease were not included in the study. Although we failed to enroll our expected numbers in each of the groups, we did demonstrate a significant difference in our primary outcome of GBS prevalence when we compared subjects with no evidence of vaginitis to subjects with either common or inflammatory vaginitis. Power calculations depend on specification of hypothesized proportions and do not indicate what actual difference in the observed data would result in significance. We demonstrated a significant difference in the prevalence of GBS in women with no vaginitis (17.7%) and common vaginitis (34.1%), and in subjects with DIV (42.9%). While further enrollment would not have contributed to these outcomes, we may have observed a statistically significant difference for the inflammatory vaginitis group as a whole. Our low numbers of subjects with vaginitis also reduced our ability to detect a difference in some of our secondary outcomes (e.g. predictors of GBS positivity).

In summary, the goal of this study was to establish the prevalence of GBS in a gynecologic population. Our data reveal that GBS colonization is increased in women with common vaginal infections (e.g. yeast, BV, trichomoniasis) and in women with inflammatory vaginitis. Thus, GBS colonization likely represents a marker of a disrupted vaginal ecosystem in the gynecologic patient, and not a causal factor in vaginitis.

Precis

Non-pregnant women with vaginitis are more likely to be colonized with group B streptococcus.

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Table 1

Demographics

Demographics [‡]	Total n (%) (n = 215)	No Vaginitis n (%) (n = 147)	Common Vaginitis n (%) (n = 41)	Inflammatory Vaginitis n (%) (n = 27)	P value [*]
Age					0.020 [‡]
18-29	88 (41.7)	57 (39.3)	20 (50.0)	11 (42.3)	
30-39	75 (35.5)	58 (40.0)	11 (27.5)	6 (23.1)	
40-49	35 (16.6)	26 (17.9)	5 (12.5)	4 (15.4)	
≥ 50	13 (6.2)	4 (2.8)	4 (10.0)	5 (19.2)	
Race					0.248
White	192 (91.4)	130 (90.3)	36 (90.0)	26 (100.0)	
Non-white	18 (8.6)	14 (9.7)	4 (10.0)	0 (0.0)	
Marital Status					0.005 [‡]
Married/Partnered	126 (60.0)	94 (65.3)	15 (37.5)	17 (65.4)	
Not married	84 (40.0)	50 (34.7)	25 (62.5)	9 (34.6)	
Income					0.388
≤ \$40,000	76 (37.1)	47 (33.3)	18 (46.2)	11 (44.0)	
\$41,000-100,000	89 (43.4)	62 (44.0)	16 (41.0)	11 (44.0)	
> \$101,000	40 (19.5)	32 (22.7)	5 (12.8)	3 (12.0)	
Education					0.032 [‡]
High school or less	48 (23.0)	26 (18.2)	15 (37.5)	7 (26.9)	
> High school	161 (77.0)	117 (81.8)	25 (62.5)	19 (73.1)	

^{*} Pearson χ^2 [‡] Statistically significant $P < 0.05$ [‡] Data for all categories less than for total due to incomplete reporting

Table 2a

Prevalence of GBS among Women with No Vaginitis, Common Vaginitis, and Inflammatory Vaginitis

Group	GBS Negative	GBS Positive	Crude OR (95% CI)	Adjusted OR [†] (95% CI)
No Vaginitis	121	26 (17.7)	1.0	1.0
Common Vaginitis	27	14 (34.1)	2.4 (1.1-5.2)	2.7 (1.1-6.2)
Inflammatory Vaginitis [*]	18	9 (33.3)	2.3 (0.9-5.8)	2.9 (1.1-8.0)

^{*} Inflammatory vaginitis = presence of PMNs and parabasal cells without microscopic evidence of pathogens.

[†] Adjusted for age, marital status, education, and study group.

Table 2b

Prevalence of GBS among Women with No Vaginitis, Common Vaginitis, and Desquamative Inflammatory Vaginitis (DIV)

	GBS Negative	GBS Positive	Crude OR (95% CI)	Adjusted OR [†] (95% CI)
Group				
No Vaginitis	121	26 (17.7)	1.0	1.0
Common Vaginitis	27	14 (34.1)	2.4 (1.1-5.2)	2.7 (1.2-6.3)
DIV [*]	12	9 (42.9)	3.5 (1.3-9.1)	5.1 (1.6-16.0)

^{*} DIV Sobel Criteria: PMNs, pH > 4.5, parabasal cells, reduced lactobacillus, discharge (17)

[†] Adjusted for age, marital status, education, and study group

Table 3

Predictors of GBS Positivity by Clinical Characteristics of Vaginitis

Characteristic	GBS positive n (%) (n=49)	Crude OR (95% CI)	Adjusted OR* (95% CI)
Clinical			
pH > 4.5	25 (51.0)	3.8 (1.9-7.4)	4.3 (1.6-11.7)
Abnormal discharge	28 (57.1)	2.9 (1.5-5.6)	2.9 (1.2-7.0)
Absence of lactobacilli	13 (26.5)	2.0 (0.9-4.3)	0.8 (0.2-2.6)
Presence of WBC	32 (65.3)	1.3 (0.6-2.4)	0.5 (0.2-1.3)
Presence of parabasal cells	13 (26.5)	1.8 (0.8-3.8)	1.5 (0.5-4.8)
Presence of hyphae	8 (16.3)	1.8 (0.7-4.5)	1.7 (0.5-5.5)
Presence of clue cells	12 (24.5)	3.000 (1.3-6.9)	2.8 (0.9-8.5)

* Adjusted for age, marital status, education, and clinical characteristics