Semiquantitative Bacterial Observations With Group B Streptococcal Vulvovaginitis

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Abstract

Objective: Group B streptococcal (GBS) vulvovaginitis is a poorly-delineated clinical entity. The purpose of this study is to report semiquantitative data from four cases of GBS vulvovaginitis and to comment on their significance in terms of the in vitro inhibitory capabilities of GBS.

Methodology: Four patients whose clinical presentations were consistent with GBS vulvovaginitis, from whom GBS was isolated and for whom semi-quantitative as well as qualitative microbiologic data existed, were identified.

Results: To produce vulvovaginitis, GBS must be at a high multiplicity (10⁸ CFU/g of vaginal fluid). Single coisolates were identified in three of the four cases (two cases of Escherichia coli and one case of Staphylococcus aureus). Group B streptococcus does not inhibit either of these bacteria in vitro.

Conclusion: When the growth requirements for the demonstration of in vitro inhibition for GBS or lack thereof are met in vivo, the in vivo observations are consistent with those projected from the in vitro data. Infect. Dis. Obstet. Gynecol. 7:227–229, 1999. © 1999 Wiley-Liss, Inc.

KEY WORDS
GBS vulvovaginitis; bacterial interference

Vulvovaginitis due to the group B streptococcus (GBS) is a poorly delineated clinical entity. The medical literature and most books on sexually transmitted diseases contain little or no information dealing with GBS vulvovaginitis. Characteristically, patients present with a chief symptom of vulvar burning or pain. A significant vaginal discharge is not a characteristic complaint. On physical examination, the labia minora will have a fiery-red to cherry-red appearance. The areas of erythema are tender to touch.

Most clinical vaginal specimens are screened for identifiable pathogens. Little regard is given to bacteria concomitantly present. In reviewing records from a number of clinical vaginitis studies in which more comprehensive microbiologic study was performed, four women were identified who

had clinical symptoms, findings, and microbiologic prerequisites that warranted the diagnosis of GBS vulvovaginitis. The purpose of this paper is to characterize their semiquantitative microbiologic data as it relates to qualitative microbiologic observations dealing with GBS³ and to in vitro inhibition data dealing with GBS.⁴

MATERIALS AND METHODS

Clinical Presentations

The primary symptom of all four patients was vulvar burning or pain. Only one of the patients reported significant vaginal discharge. The discharge was described as creamy white. On physical examination, all four patients had fiery-red labia minora that were edematous. In one patient, the erythema

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TABLE I. Microbiologic data derived from patients with GBS vulvovaginitis

Case number	Bacterial isolates
I	GBS, >108 CFU/g E. coli >108 CFU/g
2	GBS > 10° CFU/g
3	GBS +4 growth, E. coli +4 growth
4	GBS, >10 ⁸ CFU/g S. aureus 10 ² CFU/g

extended to approximately 2 inches from the anus and involved both labia. Vaginal examination revealed marked mucosal tenderness. The vaginal erythema was less intense than that which involved the labia. No cervical motion tenderness was identified. The remainder of the physical examination was unremarkable in all four patients.

Wet-mount examinations revealed an acid pH with varying degrees of inflammatory exudate present and the absence of lactobacillus on visual examination. When symptomatology was of long duration, a significant number of reparative cells were present. No protozoans or fungi were identified.

Microbiology

The quantitative microbiologic techniques used in the clinical studies varied; the preweighted swab technique was used in two cases, quantitative loops were used in one case, and in the remaining case, gross visualization of bacterial growth on a scale of trace to +4 was used.^{5,6} The qualitative microbiology was that which was standard for the research studies.⁷

RESULTS

The qualitative and semiquantitative microbiologic data is listed in Table 1. In one case, GBS was the sole isolate (case #2). In the remaining three cases, a single coisolate was identified. In two cases, the organism was *Escherichia coli*; in one case, the organism was *Staphylococcus aureus*.

DISCUSSION

In 1995, Chaisilwattana and Monif published a comprehensive study on the in vitro ability of GBS to inhibit gram-positive and gram-variable constituents of the bacterial flora of the genital tract.³ In

vitro, GBS was shown to inhibit other beta hemolytic streptococci, diphtheroides, lactobacillus, and Gardnerella vaginalis. Variable inhibition by GBS was observed with viridans, streptococci, nonhemolytic (not group B or D) streptococci, peptostreptococci, and enterococci.3 The GBS strains tested did not inhibit the growth of coagulase-negative staphylococci, S. aureus, or any gram-negative organisms.3 Isolates of GBS were uniformly inhibited by coagulase-negative staphylococci, but were not inhibited by S. aureus. 4 When quantitative studies were performed analyzing the interrelationships within the bacterial flora of the female genital tract, GBS was found not to be an infrequent isolate with either G. vaginalis, lactobacillus, or the coagulasenegative staphylococci.4

These types of observations put the validity of the in vitro inhibition data into question. The technique used to demonstrate in vitro inhibition required replication of GBS to the peak of its growth curve (>108 CFU/g). What the quantitative data presented in the study suggests is that when these conditions or requisites of growth are achieved and result in clinical disease, the in vitro observations appear valid. In one of the four cases, GBS was the sole isolate. In the three remaining cases, the single coisolates were either E. coli or S. aureus, bacteria not inhibited in vitro by GBS. None of the bacteria inhibited by GBS were present. Of some interest was the fact that, in contrast to E. coli, the multiplicity of S. aureus was low, suggesting there may be some residual inhibitory influence over grampositive organisms that is lacking for gram-negative bacteria, specifically, E. coli. When one reviews cases of polymicrobial septicemia in large series, it is not uncommon to find coisolation of group A or B streptococci and E. coli. In a previously cited study, Carlson et al. had suggested a possible facilitating interrelationship of S. aureus, GBS, and selective Enterobacteriaceae.4

With limited observations, one must be very cautious in the conclusions drawn from any such studies; however, the unlikelihood that a large series subjected to rigorous qualitative semiquantitative microbiology will be carried out in the near future is reason to present the hypothesis that in vitro and in vivo inhibition do correlate if one reproduces the conditions required in terms of the density of organisms per gram of vaginal fluid.

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