Infectious Diseases in Obstetrics and Gynecology 6:25–29 (1998)

Intrapartum Management Relating to the Risk of Perinatal Transmission of Group B Streptococcus

E.M. Levine,[1]* C.M. Strom,[1,2] V. Ghai,[2] and J.J. Barton[1]

[1]*Department of Obstetrics and Gynecology, Illinois Masonic Medical Center, Chicago, IL*

[2]*Department of Pediatrics, Illinois Masonic Medical Center, Chicago, IL*

ABSTRACT

Objective: To review the incidence of neonatal group B streptococcal (GBS) sepsis and its associated risk factors in our obstetrical population.

Methods: A computerized perinatal database of over 17,000 births (from 1992 to 1996) was queried for the incidence of neonatal GBS sepsis. A more detailed review of 895 births (from the first quarter of 1997) was undertaken to identify the incidence of risk factors known to be associated with neonatal GBS sepsis.

Results: In our institution, 30 cases of neonatal early-onset GBS sepsis were identified in over 17,000 births (or 1.7/1,000 deliveries). Risk factors were identified in 17 of those cases (56%). There were two neonatal fatalities. Chemoprophylaxis was provided in 15% of the total deliveries.

Conclusions: In spite of the lack of a uniform policy for identifying patients suitable for GBS chemoprophylaxis, we found only a 43% incidence of neonatal GBS sepsis occurring without risk factors present. Identification of antepartum or intrapartum risk factors in our series, therefore, would have identified the majority of cases resulting in neonatal GBS sepsis, which may have benefited from intrapartum therapy. Some negative potential consequences of chemoprophylaxis are discussed, raising questions regarding the recent recommendations of the Centers for Disease Control and Prevention. Infect. Dis. Obstet. Gynecol. 6:25–29, 1998. © 1998 Wiley-Liss, Inc.

KEY WORDS

group B streptococcus; neonatal sepsis; pregnancy

The risk of perinatal genital carriage of group B streptococcus (GBS) ranges from about 10–30%, depending on the manner in which screening cultures are performed and the populations screened.[1,2] The risk of vertical transmission of GBS from such mothers yielding early-onset neonatal sepsis is about .5%, and the risk of mortality from this sepsis is currently about 15%.[3–6] The overall risk of neonatal mortality from GBS sepsis is therefore about 1–4 per 10,000 births. The Centers for Disease Control and Prevention (CDC) has recommended either one of two protocols for the prevention of this serious outcome,[7] though no randomized studies exist which show the two protocols to be equivalent in efficacy. Numerous studies have shown the efficacy of intrapartum chemoprophylaxis in preventing neonatal GBS,[4,8,9] though the question remains as to which parturients would benefit most from this chemoprophylaxis: those with documented GBS cervical carriage, those with identified risk factors, or both.

At our institution, there are private and public patients. Therefore, intrapartum management varies, and intrapartum chemoprophylaxis has been provided to those women with identified risk factors, as well as those who were found to be GBS-positive antepartum. As such, there has not been a consistent manner of management of GBS risk at

*Correspondence to: Dr. Elliot M. Levine, Illinois Masonic Medical Center, 836 W. Wellington, Chicago, IL 60657. E-mail: Infodoc@immc.org

Clinical Study

Received 28 January 1998
Accepted 25 March 1998

TABLE 1. Deliveries from January through March, 1997 (n = 895)[a]

Risk factors for GBS neonatal sepsis	No.	Prophylaxis given, no. (%)
Prematurity	169	19 (11%)
Prolonged rupture of membranes	83	8 (10%)
Intrapartum fever	11	10 (91%)
Previous child with GBS sepsis	0	0 (0%)

[a]GBS, group B streptococcus.

our institution over the past five years. The risk of neonatal GBS can, nonetheless, be determined, irrespective of antibiotic use.

We conducted a retrospective review of over 17,000 deliveries with respect to the incidence of GBS neonatal sepsis in our institution. Thirty cases of early-onset invasive GBS neonatal sepsis were found and analyzed. It is the intent of this report to 1) examine the risk of GBS neonatal sepsis in our population, 2) compare the stated risk in our population with others, and 3) consider the practicality of strategies for the management of GBS risk in populations similar to ours.

METHODS

Our own proprietary computerized perinatal database of 17,867 births dating from January 1, 1992, to December 31, 1996, was queried for the incidence of neonatal sepsis. Specific medical records were selected for review, and 30 cases were found to have involved GBS sepsis, including positive blood and/or cerebrospinal fluid cultures. The appropriate maternal medical records were then reviewed to see if any risk factors existed for the vertical transmission of GBS.

As the details were more readily available for a recent single quarter (January–March 1997), the medical records of the 895 deliveries that occurred during that time period were looked at to ascertain the percentage of patients with intrapartum risk factors who did and did not receive intrapartum chemoprophylaxis (see Table 1). In addition, these same records were reviewed to determine the length of time between the admission of the patient to the labor and delivery unit and delivery.

RESULTS

The 30 maternal cases that were delivered in our institution from 1992 to 1996 in which early-onset GBS neonatal sepsis occurred (1.7/1,000 deliveries) are listed in Table 2. At least one risk factor was identified in 17 of those cases (57%). There were two neonatal fatalities in this group. Intrapartum chemoprophylaxis with intravenous ampicillin was given in one of the cases, indicated because of the presence of identified risk factors, and initiated more than four hours prior to delivery. Neonatal GBS sepsis occurred in this case despite the chemoprophylaxis provided.

Of the 895 deliveries between January 1, 1997, and March 31, 1997, there were 234 (26%) cases in which one or more intrapartum risk factors were identified (listed in Table 1). In 34 (15%) of those cases, intrapartum antimicrobial chemoprophylaxis was given. Ampicillin was generally the drug of choice, except in cases of intrapartum fever, when a broader spectrum antibiotic was used, such as cefoxitin. In addition, the same 895 cases from the first quarter of 1997 were reviewed to identify those deliveries that occurred within one hour from the time of admission. Eighty-two (9%) such cases were identified.

DISCUSSION

Multiple studies have shown that antepartum genital cultures are imprecise in predicting GBS genital carriage at delivery, given the intermittent nature of the genital carriage of GBS.[1,10–13] It is the presence or absence of GBS at the time of delivery that determines the risk of its perinatal transmission. While the parturient identified as a GBS carrier is best treated with intrapartum antimicrobial chemoprophylaxis to minimize the risk of resultant GBS neonatal sepsis,[6,8] it is not successful every time it is used, as is suggested by our own data.

Following one of the two CDC protocols calling for the intrapartum antimicrobial treatment of women found to be GBS positive antepartum would result in at least 15% of all parturients being treated (potentially 600,000 women annually in the United States). This would be done in an attempt to reduce neonatal mortality from the current rate of 1–4 per 10,000 deliveries. Aside from considerations of cost, attention should be paid to the possibility of causing excess morbidity and mortality as a result of this screening. One such source of this morbidity and mortality is the risk of anaphylaxis. It has been estimated that one fatality can be ex-

TABLE 2. Maternal cases

ID	GBS positive[a]	Prophylaxis/Treatment given	Preterm	Prolonged rupture of membranes	Intrapartum fever	Previous baby with GBS
MR	ND[b]	N	N	N	N	N
NH	ND	N	N	N	Y	N
LG	ND	N	N	N	N	N
TK	ND	Y	N	N	Y	N
ML	ND	N	N	N	N	N
MC	ND	N	N	N	N	N
JJ	ND	N	Y	N	N	N
MN	ND	N	N	N	N	N
ME	ND	N	Y	N	N	N
CT	ND	N	N	N	N	N
SA	ND	N	N	N	Y	N
JD	ND	N	N	Y	N	N
GJ	ND	N	N	Y	N	N
PL	ND	N	Y	N	N	N
JL	ND	N	N	N	N	N
DB	ND	N	N	N	N	N
PR	ND	N	N	Y	N	N
MG	ND	Y	N	Y	Y	N
AG	ND	N	N	N	N	N
PG	ND	N	Y	N	N	N
DD	ND	Y	N	Y	Y	N
MP	ND	N	Y	N	N	N
MA	ND	N	N	N	N	N
CR[c]	ND	N	N	N	N	N
LB[c]	ND	N	Y	Y	N	N
GP	ND	N	N	N	Y	N
YA	ND	N	N	Y	N	N
MC	ND	N	N	N	N	N
CS	ND	N	Y	N	N	N
MC	ND	N	N	N	N	N

[a]GBS, group B streptococcus.
[b]ND, Not determined.
[c]Neonatal death.

pected for every 100,000 patients treated with penicillin.[14,15] Extrapolating from this estimate, it appears that six women could die annually if the CDC's recommendation were followed. It should be further emphasized that the fetus can also be adversely affected in anaphylactic reactions, as has been reported.[16,17]

Additionally, the volume of antibiotic use recommended by such a protocol will undoubtedly have an impact on the emergence of resistant bacterial strains. It is not unreasonable to assume such antimicrobial selective pressure will inevitably cause the antibiotics that we currently use for the treatment of streptococcal bacterial infections to be less effective in the future. Such changes in the patterns of bacterial resistance have been seen against ampicillin by *Escherichia coli* and against penicillin by *Neisseria gonorrhoeae* and, more recently, by *Streptococcus pneumoniae.*[18–20]

Just as the CDC protocol recommends prophylaxis for GBS carriers without risk factors (not justified by the objective studies performed by Boyer et al.[2,4,8]), the CDC recommendation will likely cause practitioners to provide prophylaxis for GBS carriers *and* those with risk factors as well. Medicolegal concerns will likely provide the impetus for this. While it is important to obtain informed consent from patients regarding methods of selection for GBS prophylaxis, one must wonder what can be explained to a patient, given that there are no data to justify using one method versus another.

As a practical matter, if a patient who is identified as a GBS carrier delivers within one hour of admission, the antibiotic given such a patient will likely be ineffective in preventing neonatal GBS sepsis.[21] This is, of course, assuming that all such patients are even properly identified when they are admitted to the labor and delivery suite. In our

study, 9% of patients would fall into this category. It can be estimated, then, that 9% of those women found to be GBS carriers would not be adequately treated during labor because of their "late" arrival to labor and delivery. This would offer a practical limitation on the effectiveness of antepartum GBS screening.

In our study, an antepartum genital culture would have had to be performed for almost 18,000 patients to identify those GBS carriers who should receive intrapartum antimicrobial prophylaxis in an attempt to prevent the additional 13 cases of GBS sepsis in babies born to women without risk factors. These numbers become important when considering GBS prevention strategies and their attendant risks. It should be noted that the incidence of GBS neonatal sepsis in our series is 1.7/1,000 deliveries, which is similar to that reported elsewhere[22,23] when intrapartum chemoprophylaxis is not given. Also, it was noted that 26% of the patients delivering in the first quarter of 1997 had risk factors for GBS, as opposed to the 18% reported by Boyer et al.[2] This may be due to the fact that our institution is a tertiary care facility drawing a population at higher risk than that seen elsewhere. Also, our figure includes 52 patients who were, by definition, preterm at delivery, yet at greater than 36 weeks of gestation, a gestational age at which many practitioners may choose not to offer prophylaxis.

Clinicians have been taught to "First, do no harm." It seems that the potential for harm from GBS screening may not have been adequately considered, or it may have been underestimated. Future studies will hopefully provide additional insights into the clinical process of GBS prevention with an eye towards its practical aspects. This would be a valuable addition to the various relevant decision analyses recently performed[24,25] and would help promote evidence-based clinical practice.

REFERENCES

1. Anthony BF, Eisenstadt R, Carter J, Kim KS, Hobel CJ: Genital and intestinal carriage of group B streptococci during pregnancy. J Infect Dis 143:761–766, 1981.
2. Boyer KM, Gadzala CA, Burd LI, et al.: Selective intrapartum chemoprophylaxis of neonatal group B streptococcal early-onset disease. I. Epidemiologic Rationale. J Infect Dis 148:795–801, 1983.
3. Anthony BF, Okada DM, Hobel CJ: Epidemiology of group B streptococcus: Longitudinal observations during pregnancy. J Infect Dis 137:524–530, 1978.
4. Boyer KM, Gadzala CA, Kelly PD, Burd LI, Gotoff SP: Selective intrapartum chemoprophylaxis of neonatal group B streptococcal early-onset disease. II. Predictive value of prenatal cultures. J Infect Dis 148:802–809, 1983.
5. Baker CJ, Edward MS: Group B streptococcal infections. In: Remington JS, Klein JO (eds): Infectious Diseases of the Fetus and Newborn Infant. 4th ed. Philadelphia: W.B. Saunders, pp 980–1054, 1995.
6. American College of Obstetricians and Gynecologists. Prevention of early-onset group B streptococcal disease in newborns. ACOG Committee Opinion no. 173. Washington, DC: ACOG, 1996.
7. Centers for Disease Control and Prevention. Prevention of perinatal group B streptococcal disease: A public health perspective. MMWR 45(RR-7):1–24, 1996.
8. Boyer KM, Gotoff SP: Prevention of early-onset neonatal group B streptococcal disease with selective intrapartum chemoprophylaxis. N Engl J Med 314:1665–1669, 1986.
9. Iams JD, O'Shaughnessy R: Antepartum versus intrapartum selective screening for maternal group B streptococcal colonization. Am J Obstet Gynecol 143:153–156, 1982.
10. Hoogkamp-Korstanje JAA, Gerards LJ, Cats BP: Maternal carriage and neonatal acquisition of group B streptococci. J Infect Dis 145:800–803, 1982.
11. Yancey MK, Schuchat A, Brown LK, et al.: The accuracy of late antenatal screening cultures in predicting genital group B streptococcal colonization at delivery. Obstet Gynecol 88:811–815, 1996.
12. Regan JA, Klebanoff MA, Nugent RP, et al.: Colonization with group B streptococci in pregnancy and adverse outcome. Am J Obstet Gynecol 174:1354–1360, 1996.
13. Levine EM: Group B streptococci and pregnancy. Am J Obstet Gynecol 175:1084, 1996.
14. Idsoe O, Guthe T, Willcox RR, et al.: The nature and extent of penicillin side reactions with particular reference to fatalities from anaphylactic shock. Bull World Health Organ 38:159–188, 1968.
15. Rudolph AH, Price EV: Penicillin reactions among patients in venereal disease clinics: A national survey. JAMA 223:499–501, 1973.
16. Gallagher JS: Anaphylaxis in pregnancy. Obstet Gynecol 71:491–493, 1988.
17. Heim K, Alge A, Marth C: Anaphylactic reaction to ampicillin and severe complication in the fetus. Lancet 337:859–860, 1991.
18. Reichart CA, Neumann T, Foreman P, Zenilman JM, Hook EW: Temporal trends in gonococcal antibiotic re-

sistance in Baltimore. Sex Transm Dis 19:213–218, 1992.

19. Armor SA, Monif GR, Harrah WD: Penicillinase-producing *Neisseria gonorrhoeae* arrives in Nebraska. Nebr Med J 75:255–257, 1990.
20. McCracken GH: Emergence of resistant *Streptococcus pneumoniae:* A problem in pediatrics. Pediatr Infect Dis J 14:424–428, 1995.
21. Boyer KM, Gadzala CA, Kelly PD, et al.: Selective intrapartum chemoprophylaxis of neonatal group B streptococcal early-onset disease. III. Interruption of mother-to-infant transmission. J Infect Dis 148:810–816, 1983.
22. American College of Obstetricians and Gynecologists. Group B Streptococcal infections in pregnancy. ACOG Technical Bulletin no. 170. Washington, DC: ACOG, 1992.
23. Greenspoon JS, Wilcox JG, Kirschbaum TH: Group B streptococcus: The effectiveness of screening and chemoprophylaxis. Obstet Gynecol Surv 46:499–508, 1991.
24. Rouse DJ, Goldenberg RL, Cliver SP, et al.: Strategies for the prevention of early-onset neonatal group B streptococcal sepsis: A decision analysis. Obstet Gynecol 83: 483–494, 1994.
25. Mohle-Boetani JC, Schuchat A, Plikaytis BD, Smith JD, Broome CV: Comparison of prevention strategies for neonatal group B streptococcal infection. JAMA 270: 1442–1448, 1993.