

## NEONATAL GROUP B STREPTOCOCCAL COLONIZATION AND MATERNAL UROGENITAL OR ANORECTAL CARRIAGE\*

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During the 1970s, group B streptococci (GBS) were clearly identified as important neonatal pathogens. There are two well-recognized forms of the disease; early onset and late onset. Early onset disease occurs within the first few days of life, develops rapidly, and has a very high mortality rate, most often associated with sepsis or pneumonia. In contrast, late onset disease often occurs insidiously, is difficult to distinguish from other bacterial infections and is commonly associated with meningitis. The highest incidence of meningitis occurs in the first month of life and GBS are the major etiologic agents. Additionally, GBS are causative agents of late intrauterine death, spontaneous abortion, premature labor, and early rupture of membranes<sup>1-9</sup>.

It is reported that neonatal group B streptococcal infections are closely related to maternal anorectal and urogenital carriage<sup>1-6</sup>. Sampling of the genital tract for GBS at any time between conception and delivery has revealed prevalence rates of 5 to 28 percent. The vertical transmission of GBS to neonates from their carrier mothers is found to be 50 to 75 percent<sup>3</sup>. In order to determine the incidence of group B streptococcal carriage, which causes early neonatal infections in pregnancy, antepartum screening has no advantage; therefore an intrapartum approach is recommended<sup>10</sup>.

Although intrauterine fetal complications and neonatal infections are widely seen in Turkey, studies concerning group B streptococcal carriage are very rare. Our

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study was designed to investigate the relation between the neonatal group B streptococcal infection and maternal carriage in our region.

### Material and Methods

One hundred and ten women delivered at Sivas Maternity Hospital and their newborn infants were included in the study without any selection. The infants and mothers were observed during the first five days after delivery. Their obstetrical histories were obtained, and physical and routine laboratory examinations were carried out.

Specimens were obtained with a sterile cotton swab from the urethra, vagina and rectum of each parturient during labor. Within a few minutes after birth and on the fourth day of life, swab specimens were also taken from the external auditory canal, throat and umbilicus of the infants. These specimens were immediately inoculated into 10 ml of Todd-Hewitt broth containing 5% defibrinated sheep-blood, 8  $\mu\text{g/ml}$  gentamicin sulphate and 15  $\mu\text{g/ml}$  nalidixic acid<sup>11-13</sup>. Then, the cultures were incubated for 16 to 24 hours at 37°C anaerobically. The growing microorganisms were inoculated onto the surface of sheep-blood agar plates and subject to anaerobic reincubation. Colony counts and identifications were determined by means of the morphological appearance, Gram's stain, biochemical peculiarities (such as the Bacitracin disc test, salt tolerance test, CAMP test, hippurate hydrolysis test) and Lancefield precipitation tests. GBS antigens were obtained according to the Rantz-Randall method. GBS were identified by a "Bacto Streptococcus Grouping Antisera Set" (Code 2974-32). Serotyping of GBS subgroups could not be performed. Antibiotic sensitivity tests were carried out by the Kirby-Bauer Disc diffusion method<sup>11-16</sup>.

### Results

Age distribution of the pregnant women, number of pregnancies and gestational age of the infants are shown in Tables I-III.

TABLE I: Age Distribution of the Mothers

| Age Group<br>(yrs) | Number of Parturients |
|--------------------|-----------------------|
| 16 - 20            | 30                    |
| 21 - 25            | 38                    |
| 26 - 30            | 30                    |
| 31 - 35            | 5                     |
| 35 +               | 7                     |
| Total              | 110                   |

TABLE II: Number of Pregnancies of the Mothers

| <u>Number of Pregnancies</u> | <u>Number of Mothers</u> |
|------------------------------|--------------------------|
| 1                            | 33                       |
| 2                            | 23                       |
| 3                            | 28                       |
| 4                            | 10                       |
| 5                            | 16                       |
| Total                        | 110                      |

TABLE III: Gestational Age of the Infants

| <u>Gestational Age</u> | <u>Number of Infants</u> |
|------------------------|--------------------------|
| 40 weeks               | 68                       |
| 39 weeks               | 26                       |
| 38 weeks               | 9                        |
| 37 weeks               | 7                        |
| Total                  | 110                      |

Twenty of the 110 women (18.18%) in our study yielded beta hemolytic streptococci from one or more sites. The same group of streptococci was isolated from both the rectum and urethra of three pregnant women; thus the total number of cultures grown was twenty-three.

Beta hemolytic GBS were isolated from nine pregnant women. Thus the overall maternal carriage rate was found to be 8.18 percent. Colonization of GBS was most frequently seen in the rectum (seven cases). Rectal colonization was followed by urethral colonization. GBS were not isolated from the vagina (Table IV).

Six of the 110 infants yielded GBS within a few minutes after birth; in four it was isolated from the umbilicus and in the other two from the throat. On the fourth day of life, it was noted that GBS were still present (Table V).

Group B streptococci were isolated from the infants of six of the nine carrier-mothers (66.66%); omphalitis being demonstrated in three of the six. Thus the frequency of transmission to the neonates was found to be 66.66 percent among maternal carriers. In the early neonatal period, the colonization rate of GBS was found to be 5.45 percent among the newborn population. Beta hemolytic streptococci, except group B, were isolated from eight newborn infants

immediately after birth and from twelve infants on the fourth day of life. No neonatal infection was detected among the other infants during the five days following birth. GBS were found to be sensitive to ampicillin, penicillin, cephalosporin, clindamycin, carbenicillin and rifampicin.

TABLE IV: Distribution of Beta Hemolytic Streptococci Isolated from Parturients According to the Lancefield Grouping Technique

| Isolation site | GROUPS |   |   |   |   |                | Total |
|----------------|--------|---|---|---|---|----------------|-------|
|                | A      | B | C | D | G | N <sub>G</sub> |       |
| Rectum         | —      | 7 | 1 | 3 | — | 1              | 12    |
| Urethra        | —      | 2 | — | 2 | — | 2              | 6     |
| Vagina         | —      | — | 1 | 2 | 1 | 1              | 5     |
| Total          | —      | 9 | 2 | 7 | 1 | 4              | 23    |

N<sub>G</sub> : Non-grouping streptococci.

TABLE V: Distribution of Beta Hemolytic Streptococci Isolated from Newborns According to the Lancefield Grouping Technique

| Isolation site                 | A Few Minutes After Birth |   |   |   |   |                |       | On the Fourth Day of Life |   |   |   |   |                |       |
|--------------------------------|---------------------------|---|---|---|---|----------------|-------|---------------------------|---|---|---|---|----------------|-------|
|                                | Groups                    |   |   |   |   |                |       | Groups                    |   |   |   |   |                |       |
|                                | A                         | B | C | D | G | N <sub>G</sub> | Total | A                         | B | C | D | G | N <sub>G</sub> | Total |
| Throat                         | —                         | 2 | 1 | — | 1 | —              | 4     | 2                         | 2 | 1 | — | 1 | —              | 6     |
| External<br>Auditory-<br>Canal | —                         | — | — | 2 | — | 1              | 3     | 1                         | — | — | 2 | 1 | 1              | 5     |
| Umbilicus                      | —                         | 4 | — | 2 | — | 1              | 7     | —                         | 4 | — | 2 | — | 1              | 7     |
| Total                          | —                         | 6 | 1 | 4 | 1 | 2              | 14    | 3                         | 6 | 1 | 4 | 2 | 2              | 18    |

N<sub>G</sub> : Non-grouping streptococci.

## Discussion

GBS are the most serious causative agents of neonatal infections leading to high morbidity and mortality. They create two different types of neonatal infections; early onset and late onset<sup>1,3,5,16</sup>:

1. Early-onset infections occur within the first few days of life. The infant obtains GBS from the birth canal. In pregnant women, the main reservoirs of GBS are the rectum and the urethra. The mother's birth canal and the newborn infant may easily contract the organism from these sites.
2. Late-onset infections often occur insidiously and are difficult to distinguish from other bacterial infections. They are commonly associated with meningitis, gastroenteritis, cellulitis and osteomyelitis. These are considered nosocomial infections<sup>1-3</sup>.

GBS are thought to adhere to the vaginal and urethral epithelium of carrier mothers, however, without creating active infections. Although the incidence of sexually transmitted and inflammatory diseases of the cervix and urethra are the same among group B streptococcal carriers and non-carriers, these incidences may increase during pregnancy. Thus the perinatal, natal and postnatal complications increase among carrier mothers and their infants. Identification of the major body reservoirs of GBS is of both theoretical and practical importance. It can provide a clearer picture of the epidemiology and pathophysiology of streptococcal carriage and neonatal infection, a better understanding of the immune responses to the carrier state, and the ability to design and evaluate rational chemoprophylaxis. Therefore, the determination of perinatal colonization of GBS is very important. The age of the mother, the number of previous pregnancies and the gestational period may affect GBS colonization. Teenagers have a higher rate of colonization than older women, in whom multiparity is associated with a low colonization rate. If the infant is preterm, the colonization rate is high<sup>2-4,17,18</sup>. The prevalence rate of GBS in maternal genital and anorectal sites has been reported to be 5 to 25 percent in several countries<sup>1-3,18-20</sup>. The varying rates of incidence among countries depend on both culture, techniques and population differences<sup>2</sup>. Ayhan and Günalp<sup>1</sup> have reported that the prevalence rate of GBS was six percent among pregnant women in Ankara, Turkey. In our study, intrapartum group B streptococcal colonization was found to be 8.18 percent. The low prevalence values both in our study and in Ayhan and Günalp's study may be explained by religious and traditional factors that require personal cleanliness, thus preventing bacterial contamination.

A persistent carrier-state, heaviness of maternal colonization, a positive culture at labor and the number of positive culture sites are maternal factors that influence neonatal acquisition. Higher colony counts in the birth canal were associated with

a higher risk of transmission to the infant<sup>18-20</sup>. In this study, the frequency of transmission to the neonates was found to be 66.66 percent among maternal carriers.

In Turkey the etiological role of GBS in neonatal infections has not yet been clearly identified. Although this study did not include a large population, we could say that the neonatal group B streptococcal colonization rate is 5.45 percent in Turkey. Fortunately, neonatal group B streptococcal infections have not reached a dangerous level in our country. If, however, the bacteriological shift seen in developed countries should occur in Turkey, in the near future, then the determination of group B streptococcal colonization in the prevention of neonatal infections is very important.

## Summary

We studied 110 women and their newborn infants to determine the relation between maternal carriage and neonatal group B streptococcal colonization. Vaginal, urethral and rectal swabs were obtained from the pregnant women during labor. Within a few minutes after birth and on the fourth day of life, swab specimens were also taken from the external auditory canal, throat and umbilicus of the infants. The overall maternal carriage rate was found to be 8.18 percent. The frequency of transmission to the neonates was recorded as 66.66 percent among maternal carriers. In the early neonatal period, the colonization rate of GBS was found to be 5.45 percent among the newborn population.

## REFERENCES

1. Ayhan Z, Günalp A. Ankara'daki gebe kadınların beta hemolitik grup B streptokok kolonizasyon prevalansı ve serotiplere. *T K1 Tıp Bil Araşt Derg* 2:151, 1984.
2. Anthony BF. Carriage of group B streptococci during pregnancy: a puzzler. *J Infect Dis* 145:789, 1982.
3. Fischer G, Horton RE, Edelman R. From the National Institute of Allergy and Infectious Diseases. Summary of the National Institutes of Health workshop on group B streptococcal infection. *J Infect Dis* 148:163, 1983.
4. Daum RS, Smith AL. Bacterial sepsis in the newborn. *Clin Obstet Gynecol* 22:385, 1979.
5. Christensen KK, Dahlander K, Ekström, et al. Colonization of newborns with group B streptococci: relation to maternal urogenital carriage. *Scand J Infect Dis* 13:23, 1981.
6. Linden V, Christensen KK, Christensen P. Type-specific serum antibodies against group B streptococci among pregnant women: relation to urogenital carriage and age. *Scand J Infect Dis* 14:189, 1982.
7. Christensen KK, Dahlander K, Ingemarsson I, et al. Relation between maternal urogenital carriage of group B streptococci and postmaturity and intrauterine asphyxia during delivery. *Scand J Infect Dis* 12:271, 1980.
8. Regan JA, Chao S, James LS. Premature rupture of membranes, preterm delivery, and group B streptococcal colonization of mothers. *Am J Obstet Gynecol* 141:184-186, 1981.

9. Peavy KJ, Chalhub EG. Occult group B streptococcal infection: an important cause of intrauterine asphyxia. *Am J Obstet Gynecol* 146:989, 1983.
10. Iams JD, O'Shaughnessy R. Antepartum versus intrapartum selective screening for maternal group B streptococcal colonization. *Am J Obstet Gynecol* 143:153, 1982.
11. Baker CJ, Clark DJ, Barrett FF. Selective broth medium for isolation of group B streptococci. *Appl Microbiol* 26:884, 1973.
12. Sonnenwirth AC. Gram positive and gram negative cocci. In Sonnenwirth AC, Jarret L. (eds). *Gradwohl's Clinical Laboratory Methods and Diagnosis*. St. Louis: CV Mosby Co, 1980, pp 1629-1665.
13. Christensen KK, Christensen P. Typing of group B streptococci from the throat and urogenital tract of females. *Scand J Infect Dis* 10:209, 1978.
14. Koneman E. *Color Atlas and Textbook of Diagnostic Microbiology*. New York: Har-Row, 1979, pp 222-237.
15. Persson K, Bjerre B, Hansson H, Forsgren A. Several Factors influencing the colonization of group B streptococci-rectum probably the main reservoir. *Scand J Infect Dis* 13:171, 1981.
16. Krugman S, Katz SL. *Infectious Diseases of Children* (7th ed). St. Louis: CV Mosby Co, 1981, pp 208-219.
17. Anthony BF, Okada DM, Hobel CJ. Epidemiology of group B Streptococcus: longitudinal observations during pregnancy. *J Infect Dis* 137:524, 1978.
18. Hoogkamp-Korstanje JA, Gerards LJ, Cats BP. Maternal carriage and neonatal acquisition of group B streptococci. *J Infect Dis* 145:800, 1982.
19. Pass MA, Gray BM, Khare S, Dillon HC. Prospective studies of group B streptococcal infections in infants. *J Pediatr* 95:437, 1979.
20. Dillon HC Jr, Gray E, Pass MA, Gray BM. Anorectal and vaginal carriage of group B streptococci during pregnancy. *J Infect Dis* 145:794, 1982.