Maternal carriage and neonatal colonisation of group B streptococcus in eastern Turkey: prevalence, risk factors and antimicrobial resistance

A. KADANALI,¹ Ü. ALTOPARLAK,² S. KADANALI³

Department of Infectious Diseases and Clinical Microbiology,¹ Department of Microbiology and Clinical Microbiology,² Department of Obstetrics and Gynecology,³ Ataturk University, School Of Medicine, Erzurum, Turkey

SUMMARY

Our object is to determine the prevalence of group B streptococcus (GBS) carriage among pregnant women, the neonatal colonisation rate and the antimicrobial susceptibility to formulate a policy for treatment and prevention regarding perinatal GBS diseases in eastern Turkey. A total of 150 pregnant women were screened for GBS colonisation. Samples were collected from the vagina and the rectum of pregnant women, and the ear canal, throat and umbilicus of the neonates of colonised mothers. Antimicrobial susceptibility of the isolates was also investigated. GBS was isolated in at least one specimen from the 150 women in 48 cases; it was estimated that, overall, about 32% of the pregnant women and 17.3% of overall newborns were colonised with GBS. The overall rate of GBS vertical transmission was 54.2% in this study. Maternal colonisation rate was significantly higher in younger ages (p < 0.01) when maternal age of 20 years was taken as a cut-off point. All isolates were found to be sensitive to penicillin, ampicillin, cefazolin and vancomycin. Resistance to erythromycin and clindamycin were found to be 13.5 and 2.7%, respectively.

Keywords: group B streptococcus; maternal; neonatal; colonisation; resistance

INTRODUCTION

Group B streptococcus (GBS) is usually a commensal bacterium that asymptomatically colonises the vaginal and rectal areas of 10–30% of pregnant women (1). In these women, GBS can cause preterm labour, chorioamnionitis, postpartum endometritis, postpartum wound infection and sepsis. At birth, infants who are born to colonised mothers will also become colonised on their mucosal surface and skin. Neonatal GBS infection, a major cause of morbidity and mortality, occurs in two distinct syndromes of early- and late-onset diseases (2). Newborns acquire the organism intrapartum from their mother, who are colonised in the genital tract, or from the ascending spread of the organism into the amniotic fluid. Perinatal transmission can occur across intact membranes.

Maternal prenatal screening for GBS followed by offering of intrapartum chemoprophylaxis to carriers is one of the strategies used to reduce the incidence of neonatal early-onset GBS infections. Risk-based or screening-based strategies are recommended by the Centres for Disease Control and Prevention to prevent GBS disease (3). There is significant geographic variation in the proportion of women colonised with GBS; the range of colonisation reported from developing countries is somewhat different from industrialised countries (1,4).

This prospective study was aimed to determine the maternal GBS prevalence of carriage and the neonatal colonisation rate and antimicrobial susceptibility to formulate a policy for treatment and prevention regarding perinatal GBS diseases in eastern Turkey.

MATERIALS AND METHODS

This prospective study included consecutive 150 pregnant women (our unit is seeing all pregnant women) who were admitted to the University hospital Erzurum, Turkey, for routine prenatal and obstetrical care, during the period from January 2002 to February 2003. Women with diabetes mellitus, those using corticosteroids and those with a history of any kind of antibiotic usage in the last 2 weeks prior to study, were excluded from the study. Culture samples were collected with a sterile cotton swab without using an antiseptic solution and speculum from the distal third of the vagina and rectum of women. The cultures of neonates born to GBS colonised mothers were also sampled from the ear canal,
throat and umbilicus within 30 min of their lives. The pregnant women were divided into three groups according to their age (Group I: 17–26 years; Group II: 27–35 years; Group III: 36–45 years).

The swabs were placed in modified Stuart medium and sent to laboratory. Culture samples were incubated at 37 °C with the addition of 5% CO₂ for 24 h in Todd Hewitt broth media containing nalidixic acid (15 µg/ml) and gentamicin (8 µg/ml) and subcultured to a 5% sheep blood plate at 37 °C (5,6). Gram-positive and catalase-negative organisms forming β-haemolytic or non-haemolytic colonies were examined for positive CAMP (Christie, Atkins and Munch–Peterson) test, resistance to bacitracin and trimethoprim-sulphamethoxazole, positive sodium hippurate hydrolysis and negative bile esculin reaction for presumptive GBS identification (6).

Antimicrobial susceptibility of the isolates was investigated by disc-diffusion method as described by National Committee for Clinical Laboratory Standards (NCCLS) (7). Antimicrobial discs with penicillin G (10 U), ampicillin (10 µg), clindamycin (2 µg), erythromycin (15 µg), cefazolin (30 µg) and vancomycin (30 µg) were placed on the plate. Statistical analysis was carried out with the software product SAS (SAS Institute Inc., Cary, NC, USA), version 8. Statistical differences between groups were determined by χ² and student’s t-test where appropriate. A p-value of less than 0.05 was considered to be statistically significant.

RESULTS

A total 150 women at weeks 22–40 of gestation were screened for GBS colonisation. The mean age of participants was 26.1 ± 6.7 (range 17–45) years. Twenty-nine (19.3%) of women were nulliparous. Median gravidity was three. The mean gestational age at examination was 31.4 ± 5.3 weeks (range 22–40). The characteristics of GBS-positive and-negative mothers are shown in Table 1.

Overall 48 women (32%) were colonised with GBS. The vaginal and rectal colonisation rates are shown in Table 2. GBS was isolated in at least one specimen from the 150 women in 48 cases. Of these 48 cases, GBS was isolated from both vaginal and rectal swabs in 25 (16.7%) cases and only from vaginal swabs in 16 (10.7%) and rectal swabs in 7 (4.7%).

Tables 3, 4 and 5 summarise colonisation rates according to the age, gestational ages and gravidity, respectively. Differences in the colonisation rate with respect to age groups and gestational age were statistically insignificant (p > 0.05). Colonisation rates were lower in the women with four or more pregnancies compared to patients with three or less (p < 0.05).

Mean infant birth weight was insignificantly (p > 0.05) changed depending upon whether the pregnant women were GBS-positive or -negative (Table 1).

In 26 babies, GBS were isolated from the ear canal in one baby, umbilicus in one baby, throat in two babies, umbilicus and ear canal in four babies, throat and ear canal in five babies, umbilicus and throat in four babies, and umbilicus, throat and ear canal in nine babies. Overall, 17.3% of newborns have GBS carriage in our study population. Vertical transmission rate of GBS, from GBS positive mothers to their newborns, was detected as 54.2% (26/48). The swabs were placed in modified Stuart medium and sent to laboratory. Culture samples were incubated at 37 °C with the addition of 5% CO₂ for 24 h in Todd Hewitt broth media containing nalidixic acid (15 µg/ml) and gentamicin (8 µg/ml) and subcultured to a 5% sheep blood plate at 37 °C (5,6). Gram-positive and catalase-negative organisms forming β-haemolytic or non-haemolytic colonies were examined for positive CAMP (Christie, Atkins and Munch–Peterson) test, resistance to bacitracin and trimethoprim-sulphamethoxazole, positive sodium hippurate hydrolysis and negative bile esculin reaction for presumptive GBS identification (6).

Antimicrobial susceptibility of the isolates was investigated by disc-diffusion method as described by National Committee for Clinical Laboratory Standards (NCCLS) (7). Antimicrobial discs with penicillin G (10 U), ampicillin (10 µg), clindamycin (2 µg), erythromycin (15 µg), cefazolin (30 µg) and vancomycin (30 µg) were placed on the plate. Statistical analysis was carried out with the software product SAS (SAS Institute Inc., Cary, NC, USA), version 8. Statistical differences between groups were determined by χ² and student’s t-test where appropriate. A p-value of less than 0.05 was considered to be statistically significant.

RESULTS

A total 150 women at weeks 22–40 of gestation were screened for GBS colonisation. The mean age of participants was 26.1 ± 6.7 (range 17–45) years. Twenty-nine (19.3%) of women were nulliparous. Median gravidity was three. The mean gestational age at examination was 31.4 ± 5.3 weeks (range 22–40). The characteristics of GBS-positive and-negative mothers are shown in Table 1.

Overall 48 women (32%) were colonised with GBS. The vaginal and rectal colonisation rates are shown in Table 2. GBS was isolated in at least one specimen from the 150 women in 48 cases. Of these 48 cases, GBS was isolated from both vaginal and rectal swabs in 25 (16.7%) cases and only from vaginal swabs in 16 (10.7%) and rectal swabs in 7 (4.7%).

Tables 3, 4 and 5 summarise colonisation rates according to the age, gestational ages and gravidity, respectively. Differences in the colonisation rate with respect to age groups and gestational age were statistically insignificant (p > 0.05). Colonisation rates were lower in the women with four or more pregnancies compared to patients with three or less (p < 0.05).

Mean infant birth weight was insignificantly (p > 0.05) changed depending upon whether the pregnant women were GBS-positive or -negative (Table 1).

In 26 babies, GBS were isolated from the ear canal in one baby, umbilicus in one baby, throat in two babies, umbilicus and ear canal in four babies, throat and ear canal in five babies, umbilicus and throat in four babies, and umbilicus, throat and ear canal in nine babies. Overall, 17.3% of newborns have GBS carriage in our study population. Vertical transmission rate of GBS, from GBS positive mothers to their newborns, was detected as 54.2% (26/48).

Disc-diffusion resistance profiles of GBS isolates are shown in Table 6. All isolates were found to be sensitive to Penicillin, ampicillin, cefazolin and vancomycin. Resistance to erythromycin and clindamycin were found to be 13.5 and 2.7%, respectively.

DISCUSSION

GBS have been isolated from genital or lower gastrointestinal tract cultures of pregnant and non-pregnant women at rates

Table 1 | Characteristics of pregnant women and their neonates

| Characteristics              | Women with negative cultures (n = 102) | GBS carriers (n = 48) | p-value |
|------------------------------|----------------------------------------|----------------------|---------|
| Mothers age < 20             | 8/102 (7.8%)                           | 14/48 (29.2%)        | <0.01*  |
| Mean infant’s birth weight (g) ± SD | 3120 ± 403                             | 3020 ± 502           | >0.05†  |
| Multiparity (≥III)           | 50/102 (49.0%)                         | 20/48 (41.7%)        | >0.05*  |
| Gestational age at delivery  | 39.1 ± 1.2                             | 38.8 ± 1.4           | >0.05†  |
| Caesarean section            | 12/102 (11.8%)                         | 6/48 (12.5%)         | >0.05*  |

GBS, group B streptococcus. *χ² test, †Student’s t-test.

Table 2 | Vaginal and rectal colonisation rates

| Specimen         | GBS colonisation number | Percentage |
|------------------|-------------------------|------------|
| Vaginal          | 16                      | 10.7       |
| Rectal           | 7                       | 4.7        |
| Both vaginal and rectal | 25                      | 16.7       |

GBS, group B streptococcus.

Overall 48 women (32%) were colonised with GBS. The vaginal and rectal colonisation rates are shown in Table 2. GBS was isolated in at least one specimen from the 150 women in 48 cases. Of these 48 cases, GBS was isolated from both vaginal and rectal swabs in 25 (16.7%) cases and only from vaginal swabs in 16 (10.7%) and rectal swabs in 7 (4.7%).

Tables 3, 4 and 5 summarise colonisation rates according to the age, gestational ages and gravidity, respectively. Differences in the colonisation rate with respect to age groups and gestational age were statistically insignificant (p > 0.05). Colonisation rates were lower in the women with four or more pregnancies compared to patients with three or less (p < 0.05).

Mean infant birth weight was insignificantly (p > 0.05) changed depending upon whether the pregnant women were GBS-positive or -negative (Table 1).

In 26 babies, GBS were isolated from the ear canal in one baby, umbilicus in one baby, throat in two babies, umbilicus and ear canal in four babies, throat and ear canal in five babies, umbilicus and throat in four babies, and umbilicus, throat and ear canal in nine babies. Overall, 17.3% of newborns have GBS carriage in our study population. Vertical transmission rate of GBS, from GBS positive mothers to their newborns, was detected as 54.2% (26/48).

Disc-diffusion resistance profiles of GBS isolates are shown in Table 6. All isolates were found to be sensitive to Penicillin, ampicillin, cefazolin and vancomycin. Resistance to erythromycin and clindamycin were found to be 13.5 and 2.7%, respectively.

DISCUSSION

GBS have been isolated from genital or lower gastrointestinal tract cultures of pregnant and non-pregnant women at rates
Colonisation rates to the gravidity

| Gravidity | Number of carriers/number of women in group (%) |
|-----------|-----------------------------------------------|
| 1         | 9/29 (31.0)                                   |
| 2         | 19/51 (37.2)                                  |
| 3         | 13/39 (33.3)                                  |
| 4         | 5/18 (27.7)                                   |
| ≥5        | 2/13 (15.4)                                   |

Colonisation rates were lower in gravidity ≥ 4 (χ² test, p < 0.05)

Table 6 Disc-diffusion resistance profile of group B streptococcus isolates

| Antimicrobial | Number (%) |
|---------------|------------|
| Penicillin G  | 0          |
| Ampicillin    | 0          |
| Cefazolin     | 0          |
| Clindamycin   | 2 (2.7)    |
| Erythromycin  | 10 (13.5)  |
| Vancomycin    | 0          |

Differences in colonisation rate between groups were statistically insignificant (χ² test, p > 0.05)

ranging from 5 to 40% (8,9). These variations in the reported prevalence of asymptomatic colonisation relate not only to differences in the sites sampled and bacteriologic method for detection of organism but also to demographic differences in the populations studied (1). In this study, it was estimated that, overall, about 32% of the pregnant women and 17.3% of overall newborns were colonised with GBS. The overall colonisation rate was high in this study. It is possible that genetic, social and cultural factors may play a role in a community. In this study, maternal GBS carriage rates are higher than the developing world rates (10), this data interferes with the suggestion that GBS carriage was significantly higher in industrialised countries than developing world (9,11).

Maternal colonisation rate was significantly higher in younger age (p < 0.01) when maternal age of 20 years was taken as a cut-off point (Table 1). In previous studies, it was reported that maternal carriage was associated with younger age. But cut-off values for younger age were changed among different reports, some studies used 20 years of age as a cut-off point (12,13) and others accepted it as a 30 years of age (14).

Another interesting issue in GBS carriage is the relationship between parity and GBS carriage rate. Multiparity seems to be the rate-lowering factor against GBS carriage (15), this was also true for our study. Explanation of this data is difficult, as little is known about the critical defence mechanisms that may explain the apparent acquisition of increasing resistance to genital colonisation with increasing age and multiple pregnancies (16).

The overall rate of GBS vertical transmission was 54.2% in our study. Transmission rates range from 29 to 85% in most studies; rates as low as 12% have been reported from countries where colonisation rates have also been found despite the use of proper microbiological methods (1,17).

All isolates examined were susceptible to penicillin, ampicillin, cefazolin and vancomycin, but a considerable proportion was resistant to erythromycin (13.5%) and clindamycin (2.7%). The rate of resistance against erythromycin is higher than industrialised countries' rates (9.6 vs. 13.5%) (18), but lower than the study done in Turkey (21.2 vs. 13.5%) (12). The rate of clindamycin resistance was lower than the other studies (13,18,19). These findings support the use of penicillin for the prevention and treatment of GBS infections and raise concern about the use of erythromycin and clindamycin in penicillin-allergic patients.

In the majority of pregnant women with GBS carriage, GBS was frequently isolated from both vagina and rectum followed by vagina and rectum. These findings were consistent with the previous reports (20,21). It is mandatory to sample both vagina and rectum for developing effective preventive strategies against GBS carriage in pregnant women.

In conclusion, maternal and neonatal GBS colonisation rates were found to be similar with those reported from other industrialised countries. This finding is somewhat different than the suggestion that proposed high maternal GBS carriage in developed countries than developing countries. Continuing surveillance programs for perinatal GBS carriage are mandatory in communities. An accurate evaluation of the colonisation rate and, more importantly, the vertical transmission rate that could lead to neonatal invasive disease are required for the developing of the effective preventive and treatment programs in different communities.

REFERENCES

1 Edwards MS, Baker CJ. Streptococcus agalactiae (Group B Streptococcus). In: Mandell GL, Bennett JE, Dolin R, eds. Principles and Practice of Infectious Disease, 5th edn. Philadelphia: Churchill Livingstone, 2000: 2156–67.
2 Di Renzo GC, Sensini A, Mignoza MM et al. Who, when and which screening approach should be used for GBS screening and intrapartum therapy? In: Rafael ZB, Mettler L, Diedrich K, Schneider HPG, Dudenhhausen J-W, Shoham Z, eds. Controversies in Obstetrics Gynecology and Infertility, 1st edn. Israel: E. Oren Ltd, 2003: 416–24.
3 Centers for Disease Control and Prevention. Prevention of perinatal group B streptococcal disease: a public health perspective. Morb Mortal Wkly Rep 1996; 45: 1–24.
4 Schuchat A. Group B Streptococcus. Lancet 1999; 353: 51–6.
5. American Academy of Pediatrics and Committee on Infectious Diseases and Committee on Fetus and Newborn. Revised guidelines for prevention of early-onset group B streptococcal (GBS) infection. *Pediatrics* 1997; 99: 489–96.

6. Levinson W, Jawetz E, eds. *Medical Microbiology and Immunology*, 6th edn. Singapore: McGraw-Hill 2000, 85–95.

7. National Committee for Clinical Laboratory Standards. Performance standards for antimicrobial susceptibility testing. Ninth informational Suppl, M100-S9, Wayne, Pennsylvania: NCCLS, 1999; 19:1.

8. Gordon JS, Sharra AJ. Incidence, technique of isolation and treatment of group B streptococci. *Am J Obstet Gynecol* 1976; 126: 1023–6.

9. Anthony BF, Eisenstadt R, Carter J et al. Genital and intestinal carriage of group B streptococci during pregnancy. *J Infect Dis* 1981; 143: 761–6.

10. Stoll BJ, Schuchat A. Maternal carriage of group B streptococci in developing countries. *Pediatr Infect Dis J* 1998; 17: 499–503.

11. Campell JR, Hillier SL, Krohn MA, Fririer P, Zaleznik DF, Baker CJ. Group B streptococcal colonization and serotype-specific immunity in pregnant delivery. *Obstet Gynecol* 2000; 96: 498–503.

12. Arisoy AS, Altimisik B, Tunger O, Kurutepe S, Ispahi C. Maternal carriage and antimicrobial resistance profile of group B streptococcus. *Infection* 2003; 31 (4): 244–6.

13. Tsolia M, Psoma M, Gavrili S et al. Group B streptococcus colonization of Greek pregnant women and neonates: prevalence, risk factors and serotypes. *Clin Microbiol Infect* 2003; 9: 832–8.

14. Grimwood K, Stone PR, Gosling IA et al. Late antenatal carriage of group B streptococcus by New Zealand women. *Aust N Z J Obstet Gynaecol* 2002; 42: 182–6.

15. Grimwood K, Darlow BA, Gosling IA et al. Early-onset neonatal group B streptococcal infections in New Zealand 1998–99. *J Paediatr Child Health* 2002; 38: 272–7.

16. McKenna DS, Jams JD. Group B streptococcal infections. *Semin Perinatol* 1998; 22: 267–76.

17. Uh Y, Jang IH, Yoon KJ, Lee CH, Kwoon JY, Kim MC. Colonization rates and serotypes of group B streptococci isolated from pregnant women in a Korean tertiary hospital. *Eur J Clin Microbiol Infect Dis* 1997; 16: 753–6.

18. Silverman NS, Morgan M, Nichols WS. Antibiotic resistance patterns of group B streptococcus in antenatal genital cultures. *J Reprod Med* 2000; 45: 979–82.

19. Bland ML, Vermillion ST, Soper DE, Austin M. Antibiotic resistance patterns of group B streptococci in late third-trimester rectovaginal cultures. *Am J Obstet Gynecol* 2001; 184: 1125–6.

20. Moyo SR, Mudzori J, Tswana SA, Maeland JA. Prevalence capsular type distribution, anthropometric and obstetric factors of group B streptococcus (Streptococcus agalactiae) *Colonization Pregnancy Cent Afr J Med* 2000; 46: 115–20.

21. El-Kersh TA, Al-Nuaim LA, Kharfy TA, Al-Shammary FJ, Al-Saleh SS, Al-Zamel FA. Detection of genital colonization of group B streptococci during late pregnancy. *Saudi Med J* 2002; 23: 56–61.

*Paper received May 2004, accepted August 2004*