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Prevalence and Significance of Vaginal Group B Streptococcus Colonization in Pregnant Women from Osijek, Croatia

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ABSTRACT

The aim of the study was to determine the prevalence of vaginal group B streptococcus (GBS) colonization in pregnant women from Osijek area, the possible effect of GBS colonization on pregnancy outcome and neonatal complications and the role of intrapartum prophylaxis in this context. This retrospective case-control study took place at the Department of Gynecology and Obstetrics, Osijek University Hospital Center from December 2003 to June 2006. A total of 118 pregnant women was enrolled in study and divided into two groups: 59 women in 35th–37th week of gestation, free from risk factors for infection (control group); and 59 women in 25th–41st week of gestation with risk factors for infection. Low vaginal swab for GBS isolation and identification on selective and enriched medium was obtained from each woman. GBS colonization was recorded in 29 (24.6%) women: 12 (20.3%) control and 17 (28.8%) women at risk of infection, yielding a statistically non-significant difference ($\chi^2=1.480489$; $p<0.48$). Early neonatal infection was observed in six (20.7%) neonates born to 29 mothers with GBS colonization, pointing to a correlation between vaginal GBS colonization and early neonatal infection ($r_s=0.99$). Early perinatal infection was found in 22 (18.6%) neonates, including 17 (28.8%) pregnancies with risk factors, pointing to a significant correlation between vaginal GBS colonization, risk factors and early perinatal infection ($\chi^2=88.68$; $p<0.001$); however, gestational age and pregnancy outcome were not influenced by GBS colonization. In eight (36.4%) newborns, early neonatal infection developed in spite of intrapartum administration of antibiotics; three of these children were born to GBS positive mothers, and perinatal GBS infection was demonstrated in one (0.84%) child. Study results revealed a relatively high rate of GBS colonization in the population of pregnant women in Croatia, occasionally leading to early neonatal infection. Large studies are needed to develop national strategy for the prevention of GBS infection in Croatia.

Key words: group B streptococcus (GBS), pregnancy, neonatal infection, early neonatal group B streptococcal septicemia (ENGBSS), prevention

Introduction

The prevalence of genital system colonization with beta-hemolytic serogroup B streptococcus (group B streptococcus, GBS) is estimated to 5–40% of healthy women, varying among different populations. Vaginal or rectal GBS colonization, found in 10–30% of pregnant women¹, can cause puerperal sepsis, vaginitis, urinary infections in otherwise normal individuals, skin infections and endocarditis².

In the 1990s, guidelines for the prevention of neonatal GBS infection were developed in most western European countries and the USA, which have resulted in a significant reduction in the incidence of these infections in these countries. In spite of this favorable tendency, GBS remains the major cause of neonatal morbidity and mortality of infectious etiology in the western world^{2,3}.

In newborns, the disease develops as early-onset neonatal infection within the first 7 days of birth or as the late-onset form that develops at 7 or more days of birth. Neonatal infection manifests with sepsis, pneumonia and meningitis, however, soft tissue and bone infection may also occur^{1,4,5}. The mean rate of GBS transmission to newborns is 60% (40–70%). Early-onset neonatal infection is recorded in 1–3% of children born to mothers with GBS colonization¹. The newborn's passage through the infected birth canal may result in GBS induced early neonatal septicemia, with a mortality rate of 20–30%⁶. Early-onset neonatal disease caused by GBS accounts for 80% of all GBS infections in childhood and is associated with severe neurologic sequels in 5% of cases⁷.

Late-onset neonatal disease results from horizontal transmission, with a prevalence of 1.3–1.6/1000 newborns¹. The pathogenesis of this form of GBS caused disease is less known. In some cases, it develops upon microorganism acquisition during the newborn's passage through the birth canal¹.

The risk factors that can lead to the development of early neonatal group B streptococcal septicemia (ENGBSS) are well defined. Besides maternal vaginal swab positive for GBS, the following factors can result in an increased incidence of early-onset neonatal infection: amniotic sac rupture for more than 18 hours, premature delivery (<37 weeks), amniotic sac rupture before 37th week of gestation, body temperature at delivery of 38 °C or more, GBS in urine during pregnancy, and neonate born with ENGBSS^{8,9}. Although vaccination of pregnant women that would also protect the newborns has been investigated^{1,10}, chemoprophylaxis is the only preventive measure currently available. The wide use of intrapartum antibiotic chemoprophylaxis has reduced the incidence of early-onset neonatal disease but has limited effects due to inadequate routine use of rapid tests (such as polymerase chain reaction, PCR). Thus, intrapartum antibiotic prophylaxis is generally administered *ex iuvantibus*, in premature and precipitate deliveries in particular. Such prophylaxis may prove inefficient and inappropriate according to choice and dosage, and may lead to the development of antibiotic resistance, hypersensitivity reactions, GBS substitution by other infective agents, gram-negative bacteria in particular, and inefficiency of preventive measures for the late-onset neonatal GBS infection^{7,8}.

The protocols for prevention of GBS infection have not yet been adopted in many countries, including Croatia. Two alternative approaches in the preventive strategy are available to obstetricians, i.e. the approach based on screening and another one based on risk factors. The former is based on the screening of all pregnant women at 35th–37th week of gestation to detect anogenital GBS colonization. All pregnant women identified as GBS carriers (even without the presence of risk factors) should receive antibiotic therapy (chemoprophylaxis) by venous route until delivery^{1,11,12}. The alternative approach is based on risk factors and antibiotics are administered intrapartum to pregnant women where delivery occurs

before 37th week of gestation, amniotic sac rupture for more than 18 hours, body temperature higher than 38 °C, a history of a newborn with GBS infection, and in women with GBS bacteriuria detected during pregnancy. Chemoprophylaxis is administered to pregnant women with risk factors recorded on admission to the hospital, irrespective of GBS identification^{1,13–15}.

The aim of the present study, the first of this kind in Croatia, was to determine the prevalence of vaginal GBS colonization in the population of pregnant women in the Osijek area; to determine the prevalence of early-onset neonatal infection and its association with maternal GBS colonization and intrapartum prophylaxis; to determine whether GBS colonization is more likely in women with risk pregnancy and whether maternal GBS colonization is an additional risk factor for premature delivery, untimely amniotic sac rupture, positive urinary culture and development of early-onset neonatal infection of the newborn; and to determine correlation of GBS positive urinary culture and vaginal GBS colonization.

Materials and Methods

The study included 118 pregnant women from the Osijek area, east Croatia, followed-up at the Department of Gynecology and Obstetrics, Osijek University Hospital Center, between December 2003 and June 2006. Study women were divided into two groups: control group consisting of 59 pregnant women at 35th to 37th week of gestation, free from risk factors for GBS infection, examined and followed-up at Outpatient Obstetric Clinic, Department of Gynecology and Obstetrics, Osijek University Hospital Center; vaginal swab was obtained on routine follow-up examination; and study group consisting of 59 pregnant women at 25th to 41st week of gestation, with risk factors for GBS infection (premature delivery, untimely amniotic sac rupture, body temperature >38 °C, signs of intra-amniotic infection, urinary culture positive for GBS, hospitalized at Division of Pregnancy Pathology, Department of Gynecology and Obstetrics, Osijek University Hospital Center; vaginal swab was obtained on hospital admission.

The two groups of pregnant women were analyzed according to age, number of deliveries, level of education, marital status, gestational age, newborn sex, type of delivery, number of fetal/neonatal deaths, birth weight, Apgar score, and early neonatal period of their infants. The prevalence of vaginal GBS colonization was determined in both study groups. The early neonatal period was monitored and newborns with clinical signs of perinatal infection were analyzed. The diagnosis of perinatal infection was based on clinical picture, laboratory test findings and microbiology of blood culture, urinary culture, gastric aspirate and tip of tubus. In neonates with perinatal infection, comparison with the maternal gestational vaginal swab microbiology findings, GBS positive in particular, and with/without intrapartum antibiotic prophylaxis was performed.

Urinary culture was done in all women exhibiting signs of urinary tract infection. The sample for vaginal colonization testing was obtained without the use of speculum, i.e. the swab was obtained through introitus by wiping the inferior vaginal wall and anus^{11,12}. Isolation and identification of the grown bacteria were performed at laboratory of microbiology, according to Centers for Disease Control and Prevention (CDC) recommendations^{11,12}. All samples were incubated in aerobic conditions at a temperature of 37°C in selective and enriched Todd-Hewitt buillon with the addition of gentamicin (8 mg/L) and nalidixic acid (15 mg/L). Then, sample subcultivation on solid nutritive medium (streptococcus selective agar, Biolife, Italy; Todd-Hewitt broth, Biolife, Italy) was performed. Gram-positive catalase-negative cocci with a narrow β hemolysis zone were tested by the method of latex agglutination, a standard microbiology method for identification of group B streptococci (Prolex TM, latex agglutination system, Streptococcal Grouping Latex kit for identification of groups A, B, C, D, F or G streptococci; Pro-lab Diagnostics, Canada).

Prior to enrolment in the study, each woman was thoroughly informed on the objectives and purpose of the study, and gave her informed consent for inclusion. The study was approved by the Osijek School of Medicine Ethics Committee, and all procedures used in the study were performed in line with ethical standards, recommendations of the Osijek School of Medicine Ethics Committee and Helsinki Declaration provisions.

Statistical analysis of the data obtained was performed by use of the specialized Statistica 6.0, StatSoft Inc. software, processed by the methods of descriptive statistics and compared by use of χ^2 -test and Spearman correlation coefficient.

Results

The group of pregnant women free from risk factors after 35th week of gestation (control group) and the group of women with the presence of risk factors (premature delivery with or without untimely amniotic sac rupture, premature amniotic sac rupture for >18 hours, elevated body temperature, signs of intra-amniotic infection, positive urinary culture, and delivery of a newborn with ENGBSS in history) were statistically significantly comparable according to all epidemiological variables (age, sequence of deliveries, level of education, marital status, type of delivery, and newborn age and Apgar score).

Analysis according to gestational age revealed a high statistically significant between-group difference in gestational age and newborn birth weight. The rate of premature delivery (below 37th week of gestation) was 5.1% in the control group *versus* 61.0% in the group at risk ($\chi^2=30.26$; $p<0.001$). The newborn birth weight of ≤ 2500 g was recorded in 5.1% and 42.4% of control group and risk group women, respectively ($\chi^2=19.37$; $p<0.001$).

Microbiology of low vaginal swab revealed GBS colonization in a total of 29 (24.6%) women, including 12

(20.3%) control group women and 17 (28.8%) risk group women ($\chi^2=1.48$; $p<0.48$), yielding no statistically significant between-group difference (Table 1).

Early neonatal period in children born to mothers with GBS colonization is illustrated in Table 2. Signs of early-onset neonatal infection were observed in six (20.7%) children of 29 GBS colonized mothers. Spearman correlation coefficient ($r_s=0.99$) indicated very high mathematical correlation between vaginal GBS colonization and early neonatal infection irrespective of the causative agent identified.

Clinical and laboratory signs of early-onset perinatal infection were found in 22 (18.6%) newborns (Table 1), i.e. five (8.5%) newborns from the control group and 17 (28.8%) newborns from the risk group ($\chi^2=88.68$; $p<0.001$). These results pointed to a high, statistically significant between-group difference in the presence of risk factors, while at the same time suggesting the presence of risk factors to be independent of vaginal colonization. This in turn implied that bacterial colonization had no effect on gestational age and pregnancy outcome.

Eight (36.4%) of 22 newborns with the signs of early-onset neonatal infection including four children born to GBS positive mothers had received intrapartum antibiotic prophylaxis. The causative agent was isolated in three (75.0%) and GBS infection was demonstrated in one (0.84%) of these four newborns (Table 3).

Positive urinary culture was found in 26 (50.0%) of 52 women exhibiting symptoms of urinary tract infection (Table 4). GBS was isolated in 12 (23.1%) women and was by far the most common causative agent. The correlation of GBS positive urinary culture and vaginal GBS colonization is shown in Table 5. Vaginal GBS coloniza-

TABLE 1
PREVALENCE OF VAGINAL GROUP B STREPTOCOCCUS (GBS) COLONIZATION IN PREGNANT WOMEN FROM OSIJEK AREA ACCORDING TO STUDY GROUPS

Vaginal GBS colonization	Control group (%)	Risk group (%)	Total (%)
(+) positive	12 (20.3)	17 (28.8)	29 (24.6)
(-) negative	47 (79.7)	42 (71.2)	89 (75.4)
Total	59 (100.0)	59 (100.0)	118 (100.0)

TABLE 2
EARLY NEONATAL PERIOD IN NEWBORNS TO MOTHERS WITH GROUP B STREPTOCOCCUS (GBS) COLONIZATION FROM OSIJEK AREA ACCORDING TO STUDY GROUPS

Diagnosis	Control group (%)	Risk group (%)	Total (%)
Infection	1 (8.3)	5 (29.4)	6 (20.7)
Asphyxia	0 (0.0)	1 (5.9)	1 (3.5)
Intracranial hemorrhage	0 (0.0)	1 (5.9)	1 (3.5)
Normal finding	11 (91.7)	10 (58.8)	21 (72.3)
Total	12 (100.0)	17 (100.0)	29 (100.0)

TABLE 3
EARLY PERINATAL INFECTION IN NEWBORNS TO MOTHERS FROM OSIJEK AREA ACCORDING TO THE USE OF INTRAPARTUM PROPHYLAXIS

Microbiology (isolate)	Control group n=5 (8.5%)		Risk group n=17 (28.8%)		Total n=22 (18.6%)	
	Intrapartum prophylaxis		Intrapartum prophylaxis		Intrapartum prophylaxis	
	Yes	No	Yes	No	Yes	No
Total	2	3	6	11	8	14
Blood culture and urinary culture sterile	1	0	4 (1*)	6 (1*)	5 (1*)	6 (1*)
Gastric aspirate <i>Escherichia coli</i>	0	0	0	1	0	1
Gastric aspirate <i>Klebsiella</i>	0	0	0	1	0	1
MRSE tip of tubus	0	2	0	2	0	4
Blood culture <i>Staphylococcus epidermidis</i>	0	1	1*	0	1 ¹	1
Urinary culture <i>Klebsiella</i>	1*	0	0	1*	1*	1*
Blood culture GBS	0	0	1*	0	1*	0

*GBS (+) positive mother; GBS – group B streptococcus

TABLE 4
ANALYSIS OF URINARY CULTURE IN PREGNANT WOMEN FROM OSIJEK AREA TESTED FOR GROUP B STREPTOCOCCUS (GBS) COLONIZATION

Microbiology (isolate)	Control group n=29 (49.2 %)	Risk group n=23 (39.0%)	Total n=52 (44.1%)
GBS	2 (6.9)	10 (43.5)	12 (23.1)
<i>Escherichia coli</i>	6 (20.7)	1 (4.3)	7 (13.5)
<i>Enterococcus faecalis</i>	2 (6.9)	2 (8.7)	4 (7.7)
<i>Proteus mirabilis</i>	1 (3.5)	0 (0.0)	1 (1.9)
<i>Staphylococcus aureus</i>	1 (3.5)	0 (0.0)	1 (1.9)
<i>Staphylococcus epidermidis</i>	0 (0.0)	1 (4.3)	1 (1.9)
Sterile finding	17 (58.6)	9 (39.1)	26 (50.0)

tion was present in 12 women with GBS positive urinary culture, pointing to their high correlation ($r_s=0.99$) whereby the likelihood of vaginal colonization considerably increased with positive urinary culture.

Discussion and Conclusion

Although being normal commensal and part of the complex microflora of the gastrointestinal and genitourinary tract, *Streptococcus agalactiae* (GBS) is a major cause of severe neonatal infections^{1,4–7,16}. Some 10% to 35% of pregnant and non-pregnant women are (mostly

asymptomatic) GBS carriers^{8,17}. The increasing utilization of antibiotics as the only prophylaxis currently available, especially in cases of penicillin hypersensitivity, is a matter of growing concern about the increasing bacterial resistance, thus questioning the efficacy of intrapartum prophylaxis¹⁷. In addition, it remains obscure why GBS causes colonization in some pregnant women and leads to infection in others; colonization density, virulence variability among GBS clones and infection sensitivity of the host play a role in the development of infection^{17,18}.

In Croatia, there are no data or systematic monitoring of the prevalence of neonatal disease irrespective of the causative agent, including the prevalence of neonatal infection caused by GBS. There is no consensus on the monitoring of pregnant and non-pregnant women with GBS colonization either.

On comparison of the group of pregnant women with risk factors with control group, the rate of women with GBS colonization was not statistically significantly higher in the former. However, the risk of early-onset neonatal infection was statistically significantly increased by the presence of GBS colonization in the group of women with risk factors, but had no impact on the term (presence of risk factors in the antenatal period) and outcome

TABLE 5
CORRELATION OF GROUP B STREPTOCOCCUS (GBS) POSITIVE (+) URINARY CULTURE AND GBS POSITIVE (+) VAGINAL SWAB IN PREGNANT WOMEN FROM OSIJEK AREA

Positive (+) vaginal swab	GBS positive (+) urinary culture		
	Control group (%)	Risk group (%)	Total (%)
Total	2 (3.4)	10 (16.9)	12 (10.2)
GBS	1 (50.0)	6 (60.0)	7 (58.3)
Other bacteria	1 (50.0)	4 (40.0)	5 (41.7)

of delivery. Our study results suggested that vaginal GBS colonization significantly increased the likelihood of early neonatal disease in the newborn irrespective of the causative agent, since more than 20% of children born to mothers with GBS colonization developed this neonatal disease.

Furthermore, the use of intrapartum antibiotic prophylaxis (in women with or without bacterial colonization) was found to fail to prevent the onset of early neonatal disease with certainty, since over one-third (36.4%) of children developed the disease in spite of prophylaxis. Despite prophylaxis, at least one causative agent, including GBS in one child, was demonstrated in three of four (75.0%) children born to GBS positive mothers. These findings have opened an array of other issues in the field. Namely, due to the lack of rapid tests such as PCR, including antibiotic sensitivity of the causative agents, in daily routine, intrapartum antibiotic prophylaxis is generally administered on the basis of individual assessment and experience, *ex iuvantibus*. In our study, results of swab testing were not known during labor, in the group at risk in particular, because rapid tests were not available and intrapartum prophylaxis was not used, not even in women with verified bacterial colonization. On the other hand, the prophylaxis administered without the antibiotic sensitivity report (of GBS and other agents) was, conceivably, only partially efficient. In addition, the possible development of resistance, hypersensitivity reaction to antibiotic therapy and GBS substitution by another causative agent(s), gram-negative in particular, should always be considered.

Correlation was also found between GBS positive urinary culture and vaginal GBS colonization, whereby the former increased the likelihood of the latter; accordingly, GBS positive urinary culture should be considered as an indication for prompt therapy introduction, as suggested by most literature reports¹⁹. A meta-analysis of the prevalence of vaginal GBS colonization in 24,093 women from 13 European countries, reported in 2008, found it to range from 6.5 to 36.0%, but exceeding 20.0% in one-third of the studies included³. The same report describes regional variations in the prevalence of vaginal GBS colonization among particular regions of Europe, i.e. 19.7–29.3% in eastern Europe, 11.0–21.0% in western Europe, 24.3–36.0% in northern Europe, Scandinavia in particular, and 6.5–32.0% in southern Europe³. Data obtained in our study on vaginal GBS colonization recorded in 24.6% of study women are comparable with these data, yet indicating the rate of GBS colonization in Croatian women to exceed the rate recorded in western European countries.

Our study as the first of the kind in Croatia suffered from some limitations. Verified additional and independent risk factors such as diabetes²⁰, increased body mass index²¹, intensity of sexual activities and number of partners^{22,23}, increased number of examinations and of intravaginal and intrauterine manipulations in general²⁴ were not taken in consideration and should be involved in future studies.

CDC has issued recommendations for the prophylaxis of GBS infection^{11,12,25}. Based on these recommendations and epidemiological studies, national strategies for the prevention of GBS infections, aimed at identification of pregnant women at a high risk of neonatal GBS infection in their newborns, have been developed in a number of countries, e.g., Canada, UK, Ireland, Australia, etc.^{23,26,27}. In Croatia, data on the prevalence of GBS colonization and early neonatal infection, GBS induced in particular, should first be collected at the national and regional level to start considering development of the procedure algorithm for GBS colonization. This would certainly require large-scale and comprehensive studies. In addition, in the era characterized by the need of the most cost-effective and at the same time highly efficient and efficacious health care, the question arises whether it is reasonable to perform any preventive actions if the incidence of ENGBSS is lower than 0.6 *per* 1000 live births^{9,11,12}.

Anyhow, respective studies should be continued and extended in Croatia. The announced introduction of a GBS vaccine to be administered to pregnant women^{1,10} while also protecting the newborns is perceived as a definitive solution, although the issues of indications, route and timing of administration remain open. Maternal immunization against GBS appears to settle at long term the problem of prevention of neonatal sepsis, premature delivery and growth retardation in children^{28,29}. GBS type Ia, Ib and III capsular polysaccharides appear to be efficient in the induction of type-specific antibodies in healthy vaccinated pregnant women²⁸. Antibodies to GBS surface protein also contribute to the protection from neonatal infection³⁰. The GBS type Ia, II, III and V vaccine is expected to provide protection from more than 90% of infections, suggesting that the program of GBS vaccination should take in consideration geographical variations as well as monitoring of the prevalence GBS serotypes as major guidelines to identify the components of a polyvalent GBS vaccine⁷. Along with the development of polyvalent GBS vaccine, the question arises of vaccination timing, since vaccination in pregnancy is certainly controversial. Therefore, vaccination could potentially be performed in women of reproductive age before pregnancy, could be offered to adolescents, or even included in regular pediatric immunization²⁸.

In conclusion, GBS colonization of pregnant women in Croatia is a complex problem because of its potential severe sequels for both the mother and the child on the one hand, and for the need of savings in health care in general while ensuring the most efficient health care for this vulnerable population group on the other hand. Therefore, it is necessary for both clinicians and public health professionals to be involved in solving the issue because definitive conclusions on the development of the national program of prevention of GBS infections and on the type of health care measures to be included in such a program under current conditions can only be made upon large-scale and comprehensive epidemiological studies.

REFERENCES

1. SIMPSON JE, GRAVETT MG, Other infectious conditions in pregnancy. In: JAMES DK, STEER PJ, WEINER CP, GONIK B (Eds) High Risk Pregnancy; Management Options. 2nd Edition (W.B. Saunders, London, 2002).
2. MULLER AE, OOSTVOGEL PM, STEEGERS EAP, DÖRR PJ, Acta Obstet Gynecol Scand, 85 (2006) 1027.
3. BARCAITE E, BARTUSEVICIUS A, TAMELIENE R, KLIUCINSKAS M, MALECKIENE L, NADISAUSKIENE R, Acta Obstet Gynecol Scand, 87 (2008) 260.
4. BENITZ WE, GOULD JB, DRUZIN ML, Pediatrics, 103 (1999) e78.
5. ALSOUB H, NAJMA F, ROBIDA A, Pediatr Infect Dis J, 16 (1997) 418.
6. ADRIAANSE AH, LAGENDIJK I, MUYTJENS HL, NIJHUIS JG, KOLLÉE LA, J Perinat Med, 24 (1996) 531.
7. LIN FY, PHILIPS JB 3RD, AZIMI PH, WEISMAN LE, CLARK P, RHOADS GG, REGAN J, CONCEPCION NF, FRASCH CE, TROENDLE J, BRENNER RA, GRAY BM, BHUSHAN R, FITZGERALD G, MOYER P, CLEMENS JD, J Infect Dis, 184 (2001) 1022.
8. KUBOTA T, NOJIMA M, ITOH S, J Infect Chemother, 8 (2002) 326.
9. LYYTIKÄINEN O, NUORTI JP, HALMESMÄKI E, CARLSON P, UOTILA J, VUENTO R, RANTA T, SARKKINEN H, AMMÄLÄ M, KOSTIALA A, JÄRVENPÄÄ AL, Emerg Infect Dis, 9 (2003) 469.
10. NSAGHA DS, BELLO CS, KANDAKAI-OLUKEMI YT, East Afr Med J, 77 (2000) 34.
11. CENTERS FOR DISEASE CONTROL AND PREVENTION, Morb Mortal Wkly Rep, 49 (2000) 793.
12. SCHRAG S, GORWITZ R, FULTZ-BUTTS K, SCHUCHAT A, Morb Mortal Wkly Rep, 51 (2002) 1.
13. BERGERON MG, KE D, MÉNARD C, PICARD FJ, GAGNON M, BERNIER M, OUELLETTE M, ROY PH, MARCOUX S, FRASER WD, N Engl J Med, 343 (2000) 175.
14. STAN CM, BOULVAIN M, BOVIER PA, AUCKENTHALER R, BERNER M, IRION O, BJOG, 108 (2001) 840.
15. REISNER DP, HAAS MJ, ZINGHEIM RW, WILLIAMS MA, LUTHY DA, Am J Obstet Gynecol, 182 (2000) 1335.
16. BHUSHAN R, ANTHONY BF, FRASCH CE, Infect Immun, 66 (1998) 5848.
17. HANSEN SM, ULDBJERG N, KILIAN M, SØRENSEN UB, J Clin Microbiol, 42 (2004) 83.
18. WHITNEY CG, DALY S, LIMPONGSANURAK S, FESTIN MR, THINN KK, CHIPATO T, LUMBIGANON P, SAUVARIN J, ANDREWS W, TOLOSA JE; GLOBAL NETWORK FOR PERINATAL AND REPRODUCTIVE HEALTH, J Matern Fetal Neonatal Med, 15 (2004) 267.
19. DAIMARU-ENOKI LC, MORGAN M, NICHOLS WS, SILVERMAN NS, J Reprod Med, 50 (2005) 496.
20. RAMOS E, GAUDIER FL, HEARING LR, DEL VALLE GO, JENKINS S, BRIONES D, Obstet Gynecol, 89 (1997) 257.
21. STAPLETON RD, KAHN JM, EVANS LE, CRITCHLOW CW, GARDELLA CM, Obstet Gynecol, 106 (2005) 1246.
22. MEYN LA, MOORE DM, HILLIER SL, KROHN MA, Am J Epidemiol, 155 (2002) 949.
23. GILBERT R, Int J Epidemiol, 33 (2004) 2.
24. ADAIR CE, KOWALSKY L, QUON H, MA D, STOFFMAN J, MCGEER A, ROBERTSON S, MUCENSKI M, DAVIES HD, CMAJ, 169 (2003) 198.
25. CENTERS FOR DISEASE CONTROL AND PREVENTION, Morb Mortal Wkly Rep, 56 (2007) 701.
26. MONEY DM, DOBSON S, CANADIAN PAEDIATRIC SOCIETY, INFECTIOUS DISEASES COMMITTEE, J Obstet Gynaecol Can, 26 (2004) 826.
27. HEATH PT, BALFOUR G, WEISNER AM, EFSTRATIOU A, LAMAGNI TL, TIGHE H, O'CONNELL LA, CAFFERKEY M, VERLANDER NQ, NICOLL A, MCCARTNEY AC; PHLS GROUP B STREPTOCOCCUS WORKING GROUP, Lancet, 363 (2004) 292.
28. SHET A, FERRIERI P, Indian J Med Res, 120 (2004) 141.
29. COLBOURN T, ASSEBURG C, BOJKE L, PHILIPS Z, CLAXTON K, ADES AE, GILBERT RE, Health Technol Assess, 11 (2007) 1.
30. LARSSON C, LINDROTH M, NORDIN P, STÅLHAMMAR-CARLEMALM M, LINDAHL G, KRANTZ I, Arch Dis Child Fetal Neonatal Ed, 91 (2006) 403.

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UČESTALOST I ZNAČAJ KOLONIZACIJE RODNICE STREPTOKOKOM GRUPE B TRUDNICA S PODRUČJA GRADA OSIJEKA, HRVATSKA

SAŽETAK

Cilj istraživanja bio je utvrditi učestalost kolonizacije rodnice streptokokom grupe B (GBS) trudnica grada Osijeka. Istražiti ima li utvrđena kolonizacija i kakav utjecaj na ishod trudnoće te na nastanak komplikacija kod rođene djece, te kakva je kod toga uloga i značaj intrapartalne profilakse. Među trudnicama s područja grada Osijeka praćenim na Odjelu za ginekologiju i opstetrijiju Kliničkog bolničkog centra Osijek, u razdoblju od prosinca 2003. godine do lipnja 2006. godine, provedeno je poprečno-presječno istraživanje. Obrađeno je 118 trudnica podijeljenih u dvije skupine. U probirnoj (kontrolnoj) skupini bilo je 59 trudnica u dobi od 35 do 37 tjedana trudnoće, bez rizičnih čimbenika za infekciju, a u drugoj 59 trudnica s rizičnim čimbenicima za infekciju, trajanja trudnoće od 25 do 41 tjedna. Svakoj trudnici uzet je donji vaginalni obrisak radi izolacije i identifikacije GBS na selektivnoj i obogaćenoj podlozi. Kolonizacija GBS-om utvrđena je kod 29 (24.6%) žena. U probirnoj skupini kod 12 (20.3%), a u rizičnoj skupini 17 (28.8%), bez statistički uočene razlike ($\chi^2=1.480489$, $p<0.48$). Kod 6 (20.7%) novorođenčadi od 29 koloniziranih žena GBS-om utvrđena je rana neonatalna infekcija, što govori o korelaciji kolonizacije rodnice GBS-om i rane neonatalne infekcije ($r_s=0.99$). Rana perinatalna infekcija javila se je kod 22 (18.6%) novorođenčadi, pri čemu 17 (28.8%) iz trudnoća žena s rizičnim faktorima, što ukazuje da postoji značajna povezanost kolonizacije rodnice GBS-om, rizičnih faktora i rane infekcije djeteta ($\chi^2=88.68$, $p<0.001$), no kolonizacija ne utječe na trajanje i ishod trudnoće. Kod 8 (36.4%) djece se je rana neonatalna infekcija javila usprkos intrapartalnoj primjeni antibiotika, 3 od njih bila su djeca GBS pozitivnih majki, a kod jednog (0.84%) od njih dokazana je perinatalna infekcija GBS-om. Istraživanje je pokazalo kako je i u Hrvatskoj kolonizacija GBS-om značajna pojava u populaciji trudnica, koja u određenog broja trudnica može rezultirati razvojem rane neonatalne infekcije u njihove novorođenčadi. Nužno je provesti šira i sveobuhvatnija istraživanja temeljem kojih će se donijeti nacionalna strategija za prevenciju neonatalnih GBS infekcija u Hrvatskoj.