



Prevalence and Determinants of Anogenital Colonization by Group B Streptococcus Infection Among HIV Positive and Negative Women in Calabar, Nigeria

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Abstract

Objectives: Despite significant global decline in neonatal mortality, the rates are still unacceptably high in sub-Saharan African countries. One of the common and preventable causes of neonatal mortality is neonatal infection with group B streptococcus (GBS) microorganism. This study aims to determine the prevalence of anogenital colonization by GBS bacteria among HIV positive women, factors influencing colonization and the antimicrobial sensitivity in women attending antenatal clinic in our center.

Materials and Methods: This was a prospective study conducted at the antenatal clinics of University of Calabar Teaching Hospital (UCTH), Calabar. A total of 84 eligible and consented HIV positive women and 84 HIV negative women that were within 35 to 37 weeks of gestational age matched for age and parity were studied.

Results: Eighteen subjects tested positive to GBS infection with overall prevalence of 10.7%. However, 13 (15.5%) subjects with HIV infection tested positive to GBS infection and that was significantly higher compared with 5 (6.0%) among women without HIV infection. The prevalence of GBS infection was significantly higher among subjects with primary education (54.5%). Among HIV positive subject, there was significantly higher prevalence of GBS infection among concordant couples compared to discordant couples ($P=0.04$). Most of the subjects were sensitive to ceftriaxone (88.9%) and erythromycin (72.2%), and drug sensitivity was least with ampicillin (16.7%).

Conclusion: Anogenital colonization with GBS is high among pregnant women in our center and significantly higher among HIV infected subjects. Preventive approach to GBS colonization is a worthy measure and there is need to institute GBS screening among high risk pregnancies such as HIV infected women.

Keywords: Group B streptococcus, HIV, Neonatal mortality, Antimicrobial sensitivity, Calabar

Introduction

Sub-Saharan African region still contributes the highest proportion of the global burden of neonatal mortality due to infections (1). Many neonates are still dying of preventable causes of deaths including vertical transmission of Group B streptococcus (GBS) (2). Consequently, GBS infection remains an important cause of perinatal morbidity and mortality including neonatal meningitis, pneumonia and sepsis (3). It is also responsible for significant maternal peripartum diseases such as chorioamnionitis, endometritis and urinary tract infections (4). Cervicitis, preterm premature ruptures of membranes, preterm labor and stillbirth have also been reported among GBS colonized mothers. It is also a known cause of several infectious diseases in older children, women, immune-compromised patients, and the elderly (5). Hence, maternal infection and subsequent vertical transmission to their infants is a significant cause of maternal and neonatal morbidity and mortality.

GBS is a gram-positive bacterium which is widely distributed in nature and a normal flora of the gastrointestinal and female genital tracts. The neonate

is at greater risk of GBS infection upon delivery and premature babies are at greatest risk of death and disease (6). Most frequently the neonate becomes infected with GBS during labor through vertical transmission from the GBS colonized mother. Vertical transmission of GBS from colonized mother can result in 50% to 75% of their neonates becoming colonized with GBS. About 1% to 2% of these infants, who acquire GBS from their mothers, will develop invasive disease, with case fatality rate of 4% to 10% (7,8).

Neonatal septicemia due to GBS infection could either occur within 7 days postpartum which is termed early onset disease (EOD) which accounts for about 80% of GBS neonatal infections or between 7 days and 90 days postpartum which is termed late onset disease (LOD) which accounts for the remaining 20%. During labor, a healthy mother colonized with GBS does not always show signs or symptoms of colonization and the disease (GBS) is only occasionally associated with urinary tract infection; therefore vertical transmission from mother to neonate during delivery may occur unnoticed and result in neonatal disease (9). The occasional asymptomatic nature

Received 10 January 2017, Accepted 15 April 2017, Available online 25 April 2017

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of GBS infection and consequent transmission to the neonates emphasizes the importance of screening of these women between 35 to 37 weeks of gestation (9). Maternal detection of GBS during pregnancy and administration of treatment to the mother during labor will lead to decreased incidence of neonatal GBS colonization and subsequently a reduction in the incidence of neonatal GBS disease. The Centers for Disease Control and Prevention (CDC) revised guideline recommended universal screening for GBS for all pregnant women from 35 to 37 weeks of gestation (7). Despite these measures, GBS is a main cause of infectious mortality and morbidity among newborns (10).

In a study done in Brazil, 19.8% of HIV infected pregnant women between 35 to 37 weeks gestation were found to be GBS colonized, which is higher than the prevalence of 14.1% in the control group without HIV. A similar pattern of the disease was shown in a study in Southern Africa (11). A cross-sectional study of GBS colonization on 509 pregnant women in the DRC found 50% prevalence among HIV positive women which is significantly higher than 23.7% among HIV negative subjects (12). However studies conducted in Uganda and Malawi showed that there was no significant association with HIV status (3,13).

It is obvious that no consensus has been reached concerning association of HIV infection with GBS colonization. The introduction of highly active antiretroviral drugs has markedly increased the survival rate of HIV infected women and it is important to determine whether this group of women has more predispositions to acquisition of GBS infection. Therefore, this work was designed to evaluate the association between GBS and HIV infection in pregnancy. The findings may suggest preventive measures such as antenatal screening, intrapartum antibiotic prophylaxis and possible GBS vaccination in condition of high prevalence in the region under study. Despite high clinical significance of GBS infection, there is a paucity of literature on the epidemiology of GBS infection among pregnant women in our locality.

Materials and Methods

This was a prospective comparative study conducted at the antenatal care clinics of University of Calabar Teaching Hospital (UCTH) over a period of 16 weeks from March 1, 2016 to June 31, 2016. UCTH is a tertiary healthcare facility which serves as the main referral center for Cross River state with estimated population of 2 888 966 people (14).

Inclusion Criteria

The inclusion criteria for cases were consented pregnant women who were confirmed HIV positive and were within 35 to 37 weeks gestational age while the control group subjects were consented HIV negative pregnant women within 35 to 37 weeks of gestation.

Exclusion Criteria

Non-consenting pregnant women were excluded from the

study and any pregnant women that received any form of antibiotic therapy within 2 weeks prior to commencement of the study were also excluded.

Sample collection

For each HIV positive subjects that was recruited, a socio-demographically similar HIV negative pregnant woman was recruited by simple random sampling. Socio-demographic similarity by age, marital status, and parity were chosen. Eligible pregnant women that consented to participate were counseled on the objectives and benefits of the study, and available treatment options. Recruitment was followed by data collection of socio-demographic characteristics, obstetric history and collection of laboratory samples for assessment of GBS colonization and sensitivity testing. Patients were kept in dorsal position with both knees flexed, observing routine aseptic procedure, with gloved hands, patients' anogenital samples were collected as follows: the labia minora were parted, a sterile swab stick was inserted up to 2 to 3 cm into the vagina and vaginal wall was swabbed circumferentially. A separate swab was used to swab the anus at the level of the anal sphincter. Swabs specimen collected were transported to the hospital Microbiology Research laboratory using Amies transport medium after proper labeling of specimen for analysis. After collecting the swabs from the vagina and anus, culture of the specimen was done within one hour of collection. The analysis of the specimen was done by a microbiologist and a laboratory scientist who was designated for the study. Isolates which showed characteristic morphology (small (0.5 to 1 mm), round, domed, smooth surfaced, translucent, mildly β -hemolytic or γ -hemolytic, entire edged colonies) on sheep blood agar were presumptively identified as *Streptococcus agalactiae*. Colonies with or without narrow zone of hemolysis were gram stained. To distinguish between streptococci and staphylococci, enzyme catalase test was done. GBS was identified as non-motile, gram positive and catalase-negative cocci. The absence of effervescence was recorded as catalase negative which is a characteristic of GBSs.

Analysis of Data

Data was entered and analyzed using SPSS version 20.0. Frequency distributions of socio-demographic and obstetric characteristics of subjects were presented on frequency tables and charts. The prevalence or proportion of subjects with positive GBS test results was presented as total prevalence. Chi-square test and Fisher exact *P* value were used to compare categorical variable while independent *t* tests was used to compare means of the variables in the assessment of risk factors for GBS colonization. The results were presented in tables and graph. *P* values less than 0.05 were considered statistically significant at 95% CI.

Results

A total of 168 subjects were surveyed, from equal

proportion of two groups of pregnant women with and without HIV infection. From this overall number, 18 subjects tested positive to GBS infection yielding an overall prevalence of 10.7%. The prevalence of GBS infection among HIV infected women was 15.5% while it was 6.0% among HIV negative women.

The prevalence of GBS infection was significantly higher among the subjects with primary (54.5%), compared with secondary (8.6%) and tertiary (6.6%) levels of education ($P=0.00$) as shown in Table 1. Other socio-demographic factors, including age groups, occupation, tribe, residential location and alcohol consumption did not show significant differences in prevalence of GBS infection ($P>0.05$).

There was no significant difference in mean values of various anthropometric and obstetric factors assessed comparing the subjects with and without GBS infection

(Table 2).

Subjects with HIV infection had significantly higher prevalence of GBS infection, compared with those without HIV infection (15.5% vs. 6.0, $P=0.04$) as shown in Table 3. Also, among HIV positive subjects, there was significantly higher prevalence of GBS infection among concordant couples compared with discordant couples (29.6 vs. 8.3%, $P=0.04$). Other obstetric factors including parity, use of contraception and last period of unprotected sex, did not show significant differences in the prevalence of GBS infection.

Figure 1 shows a bar chart of Drug sensitivity to GBS. Among the 18 pregnant women who tested positive to GBS infection, most subjects were sensitive to ceftriaxone 16 (88.9%) and erythromycin 13 (72.2%), and drug sensitivity was least with ampicillin 3 (16.7%) and

Table 1. Socio-Demographic Characteristics of the Women and Associated With GBS Infection (N = 168)

Variable	GBS Present No. (%)	GBS Absent No. (%)	Total No. (100)	P Value
Age group (y)				
<20	0 (0)	8 (100)	8 (100)	0.73
21-25	2 (5.6)	34 (94.4)	36 (100)	
26-30	8 (13.3)	52 (86.7)	60 (100)	
31-35	6 (12.2)	43 (87.8)	49 (100)	
36-40	2 (14.3)	12 (85.7)	14 (100)	
>40	0 (0)	1 (100)	1 (100)	
Highest educational level				
Primary	6 (54.5)	5 (45.5)	11 (100)	0.00
Secondary	7 (8.6)	74 (91.4)	81 (100)	
Tertiary	5 (6.6)	71 (93.4)	76 (100)	
Occupation				
Trader	5 (9.8)	46 (90.2)	51 (100)	0.92
Civil servant	4 (15.4)	22 (84.6)	26 (100)	
Farmer	4 (12.1)	29 (87.9)	33 (100)	
Student	1 (6.7)	14 (93.3)	15 (100)	
House wife	0 (0)	8 (100)	8 (100)	
Artisan	3 (12.5)	21 (87.5)	24 (100)	
Unemployed	1 (9.1)	10 (90.9)	11 (100)	
Tribe				
Ibibio/Annang	6 (15.4)	33 (84.6)	39 (100)	0.75
Efik	4 (14.8)	23 (85.2)	27 (100)	
Ibo	2 (8.0)	23 (92.0)	25 (100)	
Ekoi	1 (4.3)	22 (95.7)	23 (100)	
Ejagham	2 (9.1)	20 (90.9)	22 (100)	
Others	3 (9.4)	29 (90.6)	32 (100)	
Location of subject				
Urban	7 (8.3)	77 (91.7)	84 (100)	0.32
Rural	11 (13.1)	73 (86.9)	84 (100)	
Alcohol consumption				
Yes	4 (10.8)	33 (89.2)	37 (100)	0.98
No	14 (10.7)	117 (89.3)	131 (100)	

Abbreviation: GBS, group B streptococcus.

Table 2. Anthropometric and Obstetric Factors Associated With GBS Infection (N = 168)

Variable	GBS Present, No. (%)	GBS Absent, No. (%)	T test	P Value
Age				
Mean (SD)	30.4 (3.9)	28.8 (4.7)	1.4	0.15
Range	22-38	18-41		
BMI				
Mean (SD)	29.0 (5.0)	28.8 (4.9)	0.14	0.89
Range	23.4-43.6	20.1-44.4		
ART duration (y) (n = 84)				
Mean (SD)	3.4 (2.1)	3.7 (2.9)	0.23	0.82
Range	2-7	1-16		
CD4 count (cells/mL) (n = 84)				
Mean (SD)	434 (198)	530 (252)	1.16	0.25
Range	118-877	180-1227		
GA at booking (wk)				
Mean (SD)	22.0 (7.7)	21.3 (7.2)	0.39	0.70
Range	8-34	8-36		
GA at delivery (wk)				
Mean (SD)	37.6 (1.6)	37.9 (1.5)	0.87	0.39
Range	35-40	35-41		
Parity				
Mean (SD)	1.5 (1.3)	1.5 (1.2)	0.02	0.98
Range	0-5	0-6		
Gravidity				
Mean (SD)	2.6 (1.4)	2.7 (1.3)	0.25	0.80
Range	1-6	1-7		
Number of children				
Mean (SD)	1.5 (1.4)	1.5 (1.2)	0.04	0.97
Range	0-5	0-5		

Abbreviations: GBS, group B streptococcus; GA, gestational age; ART, anti-retroviral therapy; SD, standard deviation.

Table 3. Retroviral and Obstetric Factors Associated With GBS Infection (N = 168)

Variable	GBS Present, No. (%)	GBS Absent, No. (%)	Total No. (100)	Fisher Exact P Value
Retroviral status				
Positive	13 (15.5)	71 (84.5)	84 (100)	0.04
Negative	5 (6.0)	79 (94.0)	84 (100)	
Partner HIV status (n = 84)				
Discordant	2 (8.3)	22 (91.7)	24 (100)	0.04
Concordant	16 (29.6)	38 (70.4)	54 (100)	
Unknown	3 (50.0)	3 (50.0)	6 (100)	
Time of HIV diagnosis (n = 84)				
Before pregnancy	8 (15.7)	43 (84.3)	51 (100)	0.94
During index pregnancy	5 (15.2)	28 (84.8)	33 (100)	
Receiving ART (n = 84)				
Yes	9 (12.5)	63 (87.5)	72 (100)	0.07
No	4 (33.3)	8 (66.7)	12 (100)	
Parity category				
Nulliparous	5 (13.9)	31 (86.1)	36 (100)	0.49
Multiparous	13 (9.8)	119 (90.2)	132 (100)	
Previous contraceptive use				
Yes	3 (20.0)	12 (80.0)	15 (100)	0.22
No	15 (9.8)	138 (90.2)	153 (100)	
Last period unprotected sex				
Within last 3 days	2 (4.4)	43 (95.6)	45 (100)	0.47
4-7 days	5 (13.9)	31 (86.1)	36 (100)	
8-28 days	7 (15.9)	37 (84.1)	44 (100)	
28 days to 3 months	2 (9.5)	19 (90.5)	21 (100)	
Beyond 3 months	2 (9.1)	20 (90.9)	22 (100)	

Abbreviations: GBS, group B streptococcus; ART, anti-retroviral therapy

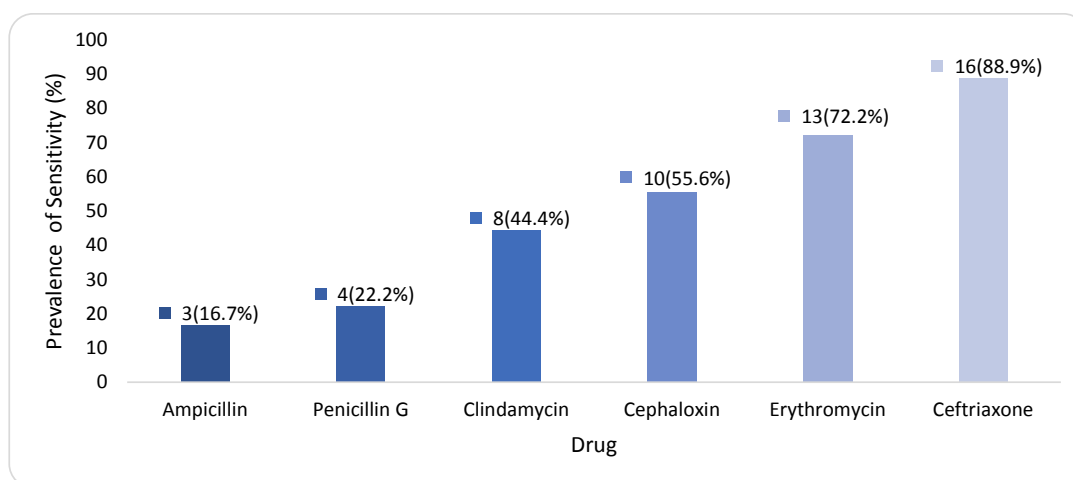


Figure 1. The Prevalence of Drug Sensitivity to GBS.

penicillin G 4 (22.2%).

Discussion

Overall prevalence of anogenital GBS colonization in this study was 10.7%. This is similar to 9% prevalence previously reported in Calabar, the same study setting (15), and 11.3% reported in Ile-Ife, Nigeria (16). Comparable prevalence rates of 10% and 9.8% were also reported in similar studies in Ibadan (17) and Maiduguri (18), Nigeria, respectively. However, much lower prevalence rate of 1.33% was reported in Uyo (19) and 6.6% was reported in Jos, Nigeria (20). On the contrary, higher prevalence rates of 14%, 28.8% and 21.61% have been reported in earlier studies in Zaria, Nigeria (21), Uganda (22) and South Africa (23), respectively. A study in the DRC reported overall colonization rate of 20% amongst pregnant women (12). Outside Africa, low colonization rates were reported in places such as Greece (6.6%) (24) and India (9.66%) (25). The disparity in prevalence reported in the various studies agrees with possible roles played by geographical, environmental, and genetic factors and sampling technique in GBS prevalence in the various study settings (26). Disparity in prevalence may also be due to differences in GBS identification techniques used, with possible higher sensitivity of detection using Lancefield antigen following culture enrichment rather than direct culture (27). In this study, anal and vaginal swabs were separately collected, cultured and analyzed which is expected to have increased sensitivity to GBS. Further studies are required to identify specific aspects of lifestyle, behavior or cultural practices that may be responsible for increase in prevalence of GBS in different geographical locations.

In this study, prevalence of anogenital colonization with GBS among HIV infected subjects was found to be higher compared with non-infected subjects, and this was statistically significant (15% vs. 6.0%, $P=0.04$). Our result agrees with a recent study in DRC where the prevalence of GBS in pregnant women was significantly higher among HIV positive women compared with HIV

negative women. However, such an association was not found in other previous studies (28,29). A similar study in Malawi showed no significant difference in prevalence of GBS infection among HIV infected and non-infected subjects (21.7% vs. 19.4%, $P=0.32$) (28). In addition, in California there was no significant association between GBS colonization and HIV status (29). The reasons for these discrepancies remain unclear. Implication of HIV infection as possible enabling factor for GBS infection may be based on immunosuppressive effect of retroviral disease, which increases susceptibility to infections. This may however be difficult to substantiate in this study where the mean CD4 count was not found to be significantly different comparing those with and without GBS infection. Similar study in Malawi found no significant difference in prevalence of GBS colonization among HIV positive women that had higher CD4 count levels (28).

In this study, level of education was significantly associated with GBS infection and was significantly higher among the subjects with primary (54.5%), compared with secondary (8.6%) and tertiary (6.6%) levels of education. Level of education may be associated with lower socioeconomic status, poor personal and environmental hygiene and poor access to preventive health services. This probably may not be unconnected to the difference in the level of hygiene and readiness to access health care demonstrable by the subjects with different levels of education. Other socio-demographic factors such as age, occupation and alcohol consumption were not significantly associated with GBS infection, suggesting that they are not risk factors that determine infectivity with GBS infection in the study setting.

None of the anthropometric and obstetric factors were found to be significantly associated with GBS infection. This includes mean CD4 count, which suggest that CD4 count may not be the sole determinant of the level of immunity, hence may not be a significant determinant of GBS infection. This is especially so when other factors

may determine level of immunity, including nutrition, stress and genetics. Similarly, duration on ART was not found to be significantly different comparing subjects with and without GBS infection.

Most infections with GBS in the study area were sensitive to ceftriaxone (88.9%) and erythromycin (72.2%), and drug sensitivity was least with ampicillin (16.7%) and penicillin G (22.2%). There was no difference in drug sensitivity comparing the subject with and without HIV infection. A previous study in the same setting (Calabar), found sensitivity to ampicillin (100%) and penicillin G (100%) (15), while in different setting found resistance to ampicillin (100%) and penicillin (100%) (16). It is not surprising to find this low sensitivity with penicillin and ampicillin. This may be a reflection of pattern of antibiotic use and abuse in the study setting, especially with the common non-prescription use of beta-lactam antibiotics for treatment of many clinical syndromes which encourages resistance. High sensitivity to ceftriaxone and erythromycin may be due to limited exposure of subjects to the prescription antibiotics which are relatively more expensive. Other environmental and socio-economic factors including poor hygiene and purchase of cheap but poor quality of drugs as well as genetic factors may also play key roles in determining pattern of antibiotic sensitivity found in this study (30,31).

Conclusion

This study revealed that there was a significantly higher prevalence of GBS anogenital colonization among HIV infected subjects. With the high prevalence of HIV especially in the study setting, preventive approach to GBS colonization is a worthy measure to cut down on the complication in the neonates. Level of education has a reverse relationship with rate of colonization, higher in pregnant women with low educational status. There was a marked sensitivity to ceftriaxone and erythromycin but resistance to penicillin G and ampicillin.

Recommendations

This high prevalence suggests the need to institute antenatal GBS screening protocol among high risk pregnancies especially among low-literate and HIV positive women. Those that test positive should receive follow-up treatment to prevent fetomaternal adverse effects. There is also, need for improvement in health education and counseling with emphasis on appropriate antibiotic use, personal and environmental hygiene, especially through the antenatal care clinics. There should be concerted effort towards discouraging self-medication and indiscriminate use of antibiotics which may promote drug resistance. This may require strengthening legal and health institutions responsible for pharmacovigilance and surveillance.

Conflict of Interests

None.

Ethical Issues

Ethical clearance for the study was obtained from the University of Calabar Teaching Hospital Research and Ethics Committee with approval number UCTH/HREC/1/2015-016 before the commencement of the study.

Financial Support

None.

References

1. Elikwu CJ, Oduyebo O, Ogunsola FT, Anorlu RI, Okoromah CN, Konig B. High group B streptococcus carriage rates in pregnant women in a tertiary institution in Nigeria. *Pan Afr Med J.* 2016;25:249. doi:10.11604/pamj.2016.25.249.9433
2. Clifford V, Garland SM, Grimwood K. Prevention of neonatal group B streptococcus disease in the 21st century. *J Paediatr Child Health.* 2012;48(9):808-815. doi:10.1111/j.1440-1754.2011.02203.x
3. Joachim A, Matee MI, Massawe FA, Lyamuya EF. Maternal and neonatal colonisation of group B streptococcus at Muhimbili National Hospital in Dar es Salaam, Tanzania: prevalence, risk factors and antimicrobial resistance. *BMC Public Health.* 2009;9:437. doi:10.1186/1471-2458-9-437
4. Monyama MC, Bolukaoto JY, Chukwu MO, et al. Group B streptococcus colonisation in pregnant women at Dr. George Mukhari Hospital, South Africa. *Southern African Journal of Infectious Diseases.* 2016;31(3):74-78. doi:10.1080/023120053.2016.1156308
5. Paoletti LC, Rench MA, Kasper DL, Molrine D, Ambrosino D, Baker CJ. Effects of alum adjuvant or a booster dose on immunogenicity during clinical trials of group B streptococcal type III conjugate vaccines. *Infect Immun.* 2001;69(11):6696-6701. doi:10.1128/iai.69.11.6696-6701.2001
6. Mullaney DM. Group B Streptococcal Infections in Newborns. *Journal of Obstetric Gynecologic Neonatal Nursing.* 2001;30(6):649-658. doi:10.1111/j.1552-6909.2001.tb00012.x
7. Verani JR, McGee L, Schrag SJ. Division of Bacterial Disease, National centre for Immunization and Respiratory Disease Control and Prevention (CDC). Prevention of Perinatal group B streptococcal disease-revised guidelines from CDC, 2010. *MMWR Recomm Rep.* 2010;59(RR-10):1-36.
8. Heath PT, Balfour G, Weisner AM, et al. Group B streptococcal disease in UK and Irish infants younger than 90 days. *Lancet.* 2004;363(9405):292-294. doi:10.1016/s0140-6736(03)15389-5
9. Hassan IA, Onon TS, Weston D, et al. A quantitative descriptive study of the prevalence of carriage (colonisation) of haemolytic streptococci groups A, B, C and G in pregnancy. *J Obstet Gynaecol.* 2011;31(3):207-209. doi:10.3109/01443615.2010.541570
10. Saleem S, Rouse DJ, McClure EM, et al. Chlorhexidine vaginal and infant wipes to reduce perinatal mortality and morbidity: a randomized controlled trial. *Obstet Gynecol.* 2010;115(6):1225-1232. doi:10.1097/AOG.0b013e3181e00ff0
11. Madhi SA, Radebe K, Crewe-Brown H, et al. High burden of invasive *Streptococcus agalactiae* disease in South African infants. *Ann Trop Paediatr.* 2003;23(1):15-23.

- doi:10.1179/000349803125002814
12. Mitima KT, Ntamako S, Birindwa AM, et al. Prevalence of colonization by *Streptococcus agalactiae* among pregnant women in Bukavu, Democratic Republic of the Congo. *J Infect Dev Ctries.* 2014;8(9):1195-1200. doi:10.3855/jidc.5030
 13. Milledge J, Calis JC, Graham SM, et al. Aetiology of neonatal sepsis in Blantyre, Malawi: 1996-2001. *Ann Trop Paediatr.* 2005;25(2):101-110. doi:10.1179/146532805x45692
 14. National Population Commission of Nigeria. Report of the 2006 census and provisional results. <http://www.population.gov.ng/index.php.htm>. Accessed January 2015.
 15. Nwachukwu N, Utsalo S, Kanu I, Anyanwu E. Genital Colonization of Group B *Streptococcus* at term pregnancy in Calabar, Nigeria. *Internet J Pediatr Neonatol.* 2006;7(2):1-4.
 16. Onipede A, Adefusi O, Adeyemi A, Adejuyigbe E, Oyelese A, Ogunniyi T. Group B *Streptococcus* carriage during late pregnancy in Ile-ife, Nigeria. *Afr J Clin Exp Microbiol.* 2012; 13(3):135-143. doi: 10.4314/ajcem.v13i3.2
 17. Donbraye-Emmanuel OB, Okonko ID, Donbraye E, et al. Isolation and characterization of Group B *Streptococci* and other pathogens among pregnant women in Ibadan Southwest Nigeria. *J Appl Biosci.* 2010;5902(29):1781-1792.
 18. Okon KO, Usman H, Umar Z, Balogun S. Prevalence of Group B *Streptococcus*(GBS) colonization among pregnant women attending antenatal clinic of Tertiary Hospital in Northeasten Nigeria. *Ame J Res Commun.* 2013;1(6):54-61
 19. Onwuezobe IA, Effiom RA. Prevalence and associated risk factors of Group B *streptococcus* in pregnant women attending antenatal care in a Nigerian urban hospital. *Ibom Med Jo.* 2016;9(1):1-7.
 20. Nsagha DS, Bello CS, Kahdakai-Olukemi YT. Maternal Carriage in Pregnancy of Group B *Streptococcs* in Jos: Relation of Endocervical Anorectal Colonization. *Nig Qt J Hosp Med.* 1997;7(1):53-56.
 21. Uhiara JE. Group B *streptococcal* carriage among parturients and their neonates in Zaria, Nigeria. *Afr J Med Med Sci.* 1993;22(3):79-83.
 22. Namugongo A, Bazira J, Fajardot Y, Joseph N. Group B *Streptococcus* Colonization among Pregnant Women Attending Antenatal Care at Tertiary Hospital in Rural Southwestern Uganda. *Int J Microbiol.* 2016;2016:3816184. doi:10.1155/2016/3816184
 23. Cutland CL, Madhi SA, Zell ER, et al. Chlorhexidine maternal-vaginal and neonate body wipes in sepsis and vertical transmission of pathogenic bacteria in South Africa: a randomised, controlled trial. *Lancet.* 2009;374(9705):1909-1916. doi:10.1016/s0140-6736(09)61339-8
 24. Tsolia M, Psoma M, Gavrioli S, et al. Group B *streptococcus* colonization of Greek pregnant women and neonates: prevalence, risk factors and serotypes. *Clin Microbiol Infect.* 2003;9(8):832-838.
 25. Narava S, Rajaram G, Ramadevi A, Prakash GV, Mackenzie S. Prevention of perinatal group B *streptococcal* infections: a review with an Indian perspective. *Indian J Med Microbiol.* 2014;32(1):6-12. doi:10.4103/0255-0857.124286
 26. Dauby N, Chamekh M, Melin P, Slogrove AL, Goetghebuer T. Increased risk of group B *streptococcus* invasive infection in HIV-exposed but uninfected infants: a review of the evidence and possible mechanisms. *Front Immunol.* 2016;7:505. doi:10.3389/fimmu.2016.00505
 27. El Aila NA, Tency I, Claeys G, et al. Comparison of different sampling techniques and of different culture methods for detection of group B *streptococcus* carriage in pregnant women. *BMC Infect Dis.* 2010;10:285. doi:10.1186/1471-2334-10-285
 28. Gray KJ, Kafulafula G, Matemba M, Kamdoloji M, Membe G, French N. Group B *Streptococcus* and HIV infection in pregnant women, Malawi, 2008-2010. *Emerg Infect Dis.* 2011;17(10):1932-1935. doi:10.3201/eid1710.102008
 29. Shah M, Aziz N, Leva N, Cohan D. Group B *Streptococcus* colonization by HIV status in pregnant women: prevalence and risk factors. *J Womens Health (Larchmt).* 2011;20(11):1737-1741. doi:10.1089/jwh.2011.2888
 30. Sahoo KC, Tamhankar AJ, Johansson E, Lundborg CS. Antibiotic use, resistance development and environmental factors: a qualitative study among healthcare professionals in Orissa, India. *BMC Public Health.* 2010;10:629. doi:10.1186/1471-2458-10-629
 31. Alekshun MN, Levy SB. Molecular mechanisms of antibacterial multidrug resistance. *Cell.* 2007;128(6):1037-1050. doi:10.1016/j.cell.2007.03.004

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