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Prevalence of group B Streptococcus colonization in antenatal women at the Queen Elizabeth Central Hospital, Blantyre - a preliminary study

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Abstract

97 antenatal clinic attendees were recruited in a study aimed to determine the prevalence of group B streptococcus *(Streptococcus agalactiae)* among pregnant women at the Queen Elizabeth Central Hospital, Blantyre. Participants were interviewed using a standard questionnaire to gather demographic and other relevant information such as history of current pregnancy, antibiotic use within the last two weeks, previous miscarriages and stillbirths. Two specimens (low vaginal and rectal swabs) were taken per participant and processed using standard bacteriological methods. Age range of the participants was 19 to 37 years with a mean of 27.3 (SD 5.68) while parity ranged from 0 to 6 (mean of 3.1). All but 2 of the women were married; 95% had some form of education and 36.1% had previously had bad pregnancy outcomes. Specimen analysis showed that sixteen (16.5%) of the participants were GBS positive yielding a total of 27 isolates all of which were sensitive to penicillin G and erythromycin. Of those with GBS, 7 (44%) reported being HIV positive, 5 (31%) negative, while 4 refused to disclose their HIV serostatus.14 (87.5%) of the 16 GBS-positive women had had bad pregnancy outcomes prior to the present study and while colonization appeared to decrease with age, it increased with the number of previous bad pregnancy outcomes (p=<0.05)

Introduction

Group B streptococcus (GBS or *Streptococcus agalactiae*) is the leading cause of perinatal morbidity and mortality being responsible for meningitis, pneumonia and sepsis in neonates[1]. It is also responsible for significant maternal peripartal disease including bacteraemia, chorioamnionitis, endometritis, urinary tract infections and for serious bacterial illness in non-pregnant adults[2]. GBS can also pass through the cervix without causing cervicitis and cross-intact amniotic membrane into the amniotic fluid causing amnionitis thereby infecting the foetus *in utero*[3,4]. GBS colonization, even when it is asymptomatic, has been associated with adverse pregnancy outcomes such as low birth weight, pre-term delivery, premature rupture of the membranes[5,6] The prevalence of GBS among black pregnant women both in South Africa and the United States has been shown[7,8,9] to be higher than in women of other racial groups and in general terms GBS prevalence among pregnant women worldwide ranges between 10 and 30%[10]. Most data on GBS epidemiology over the years has come from Europe and North America and to date only Zimbabwe in Africa has an active research programme on GBS colonization and burden of disease[10,11].

The aims of the present preliminary study were to determine the prevalence of GBS among antenatal women at the QECH, Blantyre, Malawi and also to stimulate research interest in this area which may indirectly impact the high maternal mortality rate observed in this hospital.

Materials and methods

Patient recruitment:

All women attending the antenatal clinic at the QECH with gestational age >34 weeks were eligible to participate in the study while those who had had antibiotic treatment within the last two weeks prior to recruitment were excluded from the study which was carried out between June 28th and July 23rd, 2004.

All those who met the study criteria were asked to participate in the study and were enrolled upon signing the consent form. All consenting participants were interviewed using a standard questionnaire prepared to gather demographic and other relevant information such as history of current pregnancy, previous miscarriages and still births. HIV testing was routinely done by the nursing staff as part of antenatal services and study participants were free to disclose or withhold their HIV status.

Specimen collection

Two swab specimens were taken per participant and using a speculum, a low vaginal swab (LVS) was taken while cotton-wool swabs were taken from the rectum. The swabs were immediately transferred to the Microbiology laboratory, College of Medicine and processed within one hour of collection.

Specimen culture and microscopy

The two swabs were each inoculated into Blood in Tryptone blood agar (BTBA) with 10µg of colistin sulphate/ml. The inoculated media were incubated anaerobically at 37^{0C} overnight following which they were screened for typical GBS b-haemolytic colonies. All the colonies obtained were processed for Gram staining and microscopy.

Colonies that showed β-haemolysis were picked and re-streaked onto new blood agar plates containing 10mg/ml of nalidixic acid in 5% sheep blood in blood agar (BA) base. The plates were again incubated overnight at 37^{0C}. Final inspection before discarding as negative was done after 48 hours incubation.

All isolates were identified by the use of CAMP (Christie, Artkins and Munch-Petersen) test[2]. The majority of *β*-haemolytic or nonhaemolytic GBS produce a diffusible extra-cellular protein (CAMP factor) that acts synergistically with staphylococcus *β-lysin* to cause enhanced or synergistic lysis of erythrocytes[2].

A pregnant woman was deemed to have been positive for GBS colonization when either or both of the swabs (vaginal or rectal) grew GBS. All GBS positive isolates were tested for antibiotic sensitivity by the disk diffusion method as earlier described[12].

Ethical clearance

Permission to carry out this study was granted by the College of Medicine Research and Ethics Committee (COMREC)

Results

Participants demography

97 pregnant women participated in the study. Age range was 19 to 37 years with a mean of 27.3 years and parity among the women was 0 to six (mean 3.1). The average age of GBS pos-

itive women was 22.3 years and GBS colonization appeared to decrease with age (fig.1) while GBS negative women on the other hand had an average age of 28.7 years (P=0.007) All but two of the participants were married. Thirty-five (36.1%) of the patients admitted having had bad pregnancy outcomes previously and of the 16 patients positive for GBS, 14 (87.5%) of them had a history of bad pregnancy outcomes which was strongly associated with the number of previous bad pregnancy outcomes (fig II). Ninety-two (95%) of our study population had some form of education that ranged from primary to tertiary level (fig III) but this proved not to be statistically significant with respect to GBS colonization. (p=>0.05)

Fig I. Age distribution and GBS colonization

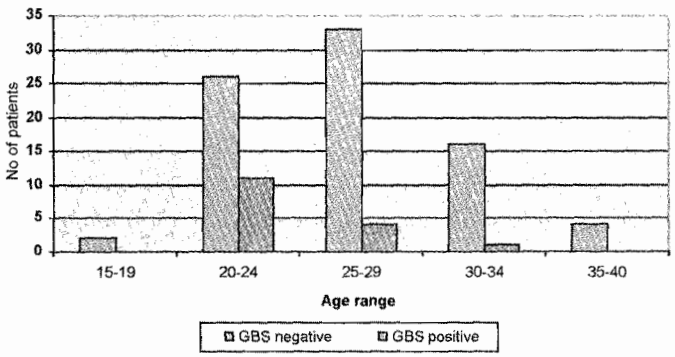
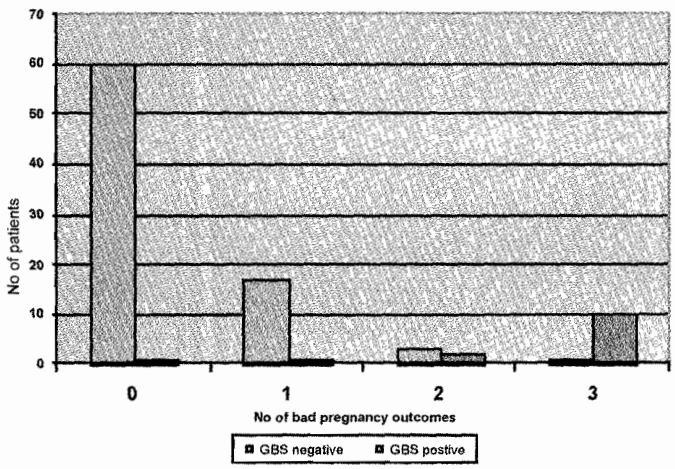


Fig II: Relationship between the number of previous bad pregnancy outcomes and GBS colonization



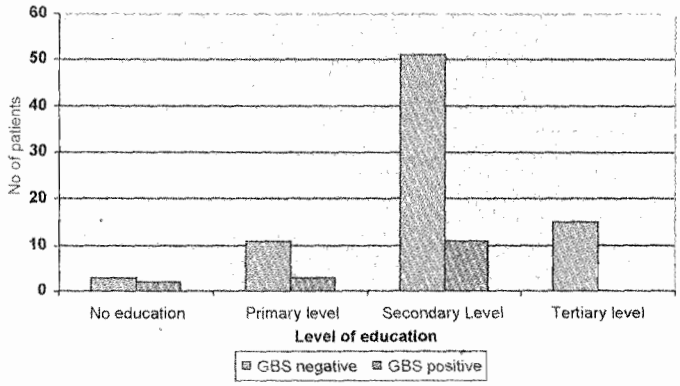
GBS bacteriology

Swab specimens from 92 (95%) of the 97 participants grew a wide-range of bacterial organisms but only sixteen (16.5%) were GBS positive yielding a total of 27 isolates. GBS isolates were recovered from both vaginal and rectal swabs in eleven patients, three were from vaginal swabs only while two isolates were obtained solely from the rectal swabs. All the GBS isolates were sensitive to penicillin and erythromycin.

GBS and HIV serostatus

Of participants with GBS colonization, 7 (44%) and 5 (31%) were HIV-positive and negative respectively while 4 (25%) refused to disclose their HIV sero-status.

Fig III: Relationship between level of education and GBS colonization



Discussion

This is a preliminary study to determine the GBS colonization rate among a limited number of pregnant women attending antenatal clinic at the QECH. The overall colonization rate obtained in this study was 16.5% compared to 20%, 31% and 32% obtained in three separate studies in Zimbabwe[13,14]. Stoll and Schuchat[14] in their study on maternal carriage of group B *streptococci* in selected developing countries reported the following colonization rates; Nigeria (20%), Ivory Coast (19%) Togo (4%) Gambia (22%) and Mozambique (1%). The above rates mostly relate to vaginal colonization only, although the Zimbabwe, Togo and Gambia studies, like in our present study, included the rectum. In our study most of the GBS isolates recovered from the two sites (vagina and rectum) of the same pregnant women were concordant (11/16 or 69%) with only 31%(5/16) showing discordant results and this is in agreement with the result of an earlier study carried out in Zimbabwe[1].

Previous studies[15,16] on the relationship between rectal and vaginal colonization in pregnant women suggested that the gastrointestinal tract may be the primary site of colonization by group B *streptococci* and that vaginal colonization may represent contamination from the rectum. In both studies the ratio of rectal colonization to that of vaginal colonization of GBS was 2:1. In a study by Moyo et al[1] in Zimbabwe, they found a ratio of 1:2 in favour of vaginal colonization. In the present study we have found a ratio of 1:1 having isolated GBS from the rectum in 13 specimens against 14 from the vagina. From this result and the observation of Moyo et al[1], it is difficult to assume that the gastro-intestinal tract is the primary source of GBS and that vaginal expression is secondary. However, irrespective of the source, GBS colonization rate of the vagina appears to decrease with age as was found in this study (fig 1)

The fact that 14 (87.5%) of the 16 patients who were GBS positive had a history of bad pregnancy outcomes indicating that such episodes could predispose to GBS colonization in subsequent pregnancies (fig II). Although 7(44%) of the 16 GBS positive patients were also HIV-positive, we can not conclude in this study whether or not GBS colonization correlates with HIV seropositivity. On the other hand, poor socio-economic status of women is usually implicated[10,17] as one of the risk factors for GBS colonization but in this study one marker for socio-economic status i.e the level of education (fig III) proved not to be statistically significant. (p=>0.05)

In this study all GBS isolates were susceptible to the recommended antibiotics viz penicillin G and erythromycin as

observed in previous studies[1,13,18] even though in other settings there have been reports of resistance to both antibiotics of up to 35%[19,20]. It is believed that differences in antimicrobial use, prophylaxis practice and serotype frequency may result in regional differences in the susceptibility of GBS to antibiotics[20].

Although the sample size in this study was limited and we did not have the means to serotype the GBS isolates recovered from participants, the study still raises awareness of the possibility of GBS posing serious health hazards to both pregnant women and their neonates in Malawi. A larger study of GBS colonization rate, disease burden and treatment protocols is therefore indicated in Malawi.

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References

1. Moyo SR, Modzori J, Tswana SA, Maeland JA. Prevalence, capsular type distribution, authropometric and obstetric factors of group B *streptococcus (Streptococcus agalactiae)* colonization in pregnancy. Central Africa Journal Medicine (2000) 46 (5) 115-120.
2. Perovic O, Group B Streptococcal infections. South Africa Journal of Epidemiology and Infection (1998) 13(1): 26-33.
3. Moyo SR, Tswana SA, Nystrom L, Bergstrom S, Blomberg A, Ljungh A. Intrauterine death and infections during pregnancy. International Journal of Obstetrics and Gynaecology (1995) 51: 211-218
4. Moyo SR, Hagerstrand I, Nystrom, Tswana SA, Bloomberg J, Bergstrom S, Ljungh A. Stillbirth and intra-uterine infection, histological choriamnionitis and microbiological findings. International Journal of Obstetrics and Gynaecology (1996) 54: 115-123
5. WHO publications. Maternal and infant mortality-socially unjustifiable tragedies (1998).
6. Baker CJ, Edwards MS. Group B streptococcal infections. In Remington J, Klein JO, eds. Infectious diseases of the fetus and newborn infants. 4th ed. Philadelphia WB Saunders (1995) 980-1054.
7. CDC publication. Perinatal Group B Streptococcal Disease. Background, Epidemiology and Overview of Revised CDC Prevention Guidelines (2002).
8. Blanckaert H, Frans J, Bosteels J, Hansen M, Verhaegen J. Optimisation of prenatal group B streptococcal screening. European Journal of Clinical Microbiology and Infectious Disease (2003): 22(10) 619-621.
9. Schuchat A. Epidemiology of Group B Streptococcal disease in the United States. Shifting paradigms. Clinical Microbiology Review (1998) 11:3; 497-513.
10. Moyo RS. Studies on *Streptococcus agalactiae* surface anchored markers with emphasis on strains and human sera from Zimbabwe. PhD thesis. Norwegian University of Science and Technology (2002) 45-48
11. Moyo SR, Boyer M, Charlebois I, Hamel J. Urogenital occurrence and persistence of group B streptococcus colonization in pregnancy in Zimbabwe. Infection and Immunity (2000) 68(10).
12. Bauer AW, Kirby WMM, Sherris JC, Turick M. Antibiotic susceptibility testing by a standardized disk method. American Journal of Clinical Pathology (1966) 45: 493-6.
13. Mason PR, Katzenstein DA, Chimbira T, Ntimavalye L. The Pueperal Sepsis Study Group: Microbial flora of the lower genital tract of women in labour at Harare Maternity Hospital. Central African Journal of Medicine (1989) 35(3) 337-44.
14. Stoll BJ, Schuchat AMD. Maternal carriage of group B streplococci in developing countries. International Paediatrics Disease Journal (1998) 17: 6: 499-503.
15. Badri MS, Zawanch S, Cruz AC Mantilla G, Baer H, Spellacy WN. Rectal colonization with group B streptococcus relation to vaginal colonization of pregnant women. Journal of Infection and Disease. (1977) 135 (2) 308 – 312.
16. Dillon HC Jr, Gray E, Pass MA, Gray BM. Anorectal and vaginal carriage of group B streptococci during pregnancy. Journal of Infection and Disease (1982) 145 (6) 794 – 99.
17. Schuchat A. Group B Streptococcus. Lancet (1999) 353: 51-56.
18. Baker CJ, Webb BJ, Barrett FF. Antimicrobial susceptibility of group B Streptococci isolated from a variety of clinical sources. Antimicrobial Agents and Chemotherapy (1976) 10: 128-131.
19. Lin F-YC, Azimi PH, Weisman LE, Philips JB, Reagan J, Clark P. Antibiotic susceptibility profiles for group B streptococci isolated from neonates. Clinical Infection and Disease. 1995-1998, (2000) 31: 76 – 9.
20. Uh Y, Jang IH, Hwang GY, Yoon KJ, Song W. Emerging erythromycin resistance among group B streptococci in Korea. European Journal of Clinical Microbiology and Infectious Disease (2001) 20: 52-54.