



SCIREA Journal of Clinical Medicine

<http://www.scirea.org/journal/CM>

ISSN: 2706-8870

October 28, 2019

Volume 4, Issue 6, December 2019

Group B Streptococcal colonisation among Sri Lankan mothers

Authors

Dr. H.D.W.S Kudagammana

Medical Microbiologist, Department of Medical Laboratory Science, Faculty of Allied Health Sciences, University of Peradeniya, Sri Lanka

Dr. R.M.C.J Rathnayaka

Consultant Obstetrician & Gynecologist Dept. of Gynecology & Obstetrics Faculty of Medicine, University of Peradeniya, Sri Lanka

Mr. B.W.M.S.B Weerasooriya

Technical Officer, Department of Medical Laboratory Science, Faculty of Allied Health Sciences, University of Peradeniya, Sri Lanka

Dr. K.M.A.G Karunathilaka

Medical Officer, Department of Medical Laboratory Science, Faculty of Allied Health Sciences, University of Peradeniya, Sri Lanka

Mr. J.A.C.N Kumara

Medical Laboratory Scientist, Department of Medical Laboratory Science, Faculty of Allied Health Sciences, University of Peradeniya, Sri Lanka

Abstract

Knowledge on Group B Streptococcus (GBS) carriage among Sri Lankans is very low compared to other countries of South Asian region. A descriptive cross sectional study was

carried out to determine GBS colonization among consecutive 50 pregnant mothers at or beyond 35 weeks of gestation at T H Peradeniya attending obstetric clinics. Low vaginal/perianal swabs obtained were enriched and sub-cultured on standard media followed by identification and confirmation of GBS using validated microbiology protocols and standard antisera. Of all fifty subjects screened, 10% showed GBS colonization. Our study showed maternal colonization with GBS rate similar to recent data of other South Asian countries.

Key words: Group B streptococcus, Colonisation, Pregnancy

Introduction

Streptococcus agalactiae is a Gram-positive-coccus in chains, which gives a narrow zone of β hemolysis on blood agar. According immunologic reactivity of major cell wall polysaccharides, *S. agalactiae* is classified as Group B Streptococcus (GBS) and further classified into nine serotypes (Ia, Ib, and II-VIII) according to capsular polysaccharides. GBS produces virulent factors such as pilli, backbone proteins for para-cellular translocation. It shows growth rate dependent invasion, α C protein mediated translocation across epithelial barriers, lipoteicoic acid, pore forming β -haemolysins, fibrinogen binding proteins and many different surface proteins which help and support in overall process of host cell entry¹.

This organism shows intermittent colonization on urogenital region or lower gastro-intestinal tract of pregnant or non-pregnant women and also men and less commonly in oropharynx¹. Maternal to infant transmission occurs in utero or during birth of a baby, in vaginally colonized women. Transmission can also occur through direct and indirect contact in health-care settings².

GBS has been recognized as a common cause of neonatal sepsis in two different entities; “Early-onset Group B Streptococcal disease” (EOGBS) and “Late-onset Group B Streptococcal disease” (LOGBS).

Most EOGBS infections are observed around 12 hours to 6 days of birth. This occurs due to ascending spread of GBS from the vagina to amniotic fluid, which is then aspirated by the infant. Pathogens may enter the respiratory tract and cause pneumonia and septicemia, which may progress to shock, or meningitis and even death if not treated promptly².

LOGBS occurs from 7 days to 3 months after birth. Bacteraemia and pyogenic meningitis are common manifestations and many other clinical syndromes including septic arthritis, osteomyelitis can occur, after getting infected which transferred vertically or horizontally from health care workers¹. Among survivors of group B streptococcal meningitis, early- or late-onset, approximately 50% showed permanent neurologic sequelae³.

In 2015-16, a pilot study has found that 14% of pregnant mothers attending maternal clinics at T H Peradeniya were colonized with GBS⁴.

An international estimate in 2017 for maternal GBS colonization worldwide revealed relatively lower prevalence of 12.5% in Southern Asia and 11% in Eastern Asia⁵. Data discovered bacterial serotypes of I–V accounting for 98% of identified colonising GBS isolates worldwide. Serotype III, showed higher association with invasive disease, mounting to 25% of all, but was less frequent in some South American and Asian countries. Serotypes VI–IX were more commonly seen among Asian population⁵.

According to studies from Germany in 2006 and also in 2017, serotype III was the most prevalent and it was about 28% of their isolates. Contribution of serotypes II, Ia, V, Ib and serotype IV were about 21%, 17%, 16%, 15% and 3% respectively^{5,6}.

To prevent EOGBS and to prevent maternal complications, Centers for Disease Control and Prevention (CDC) suggests to screen all pregnant mothers for GBS in between 35-37 weeks of gestation, and to give prophylactic antibiotics during child birth if indicated². Penicillin is the drug of choice during labor and it prevents 90% of early onset of GBS infection².

Many countries like USA, France, Spain, Belgium, Canada, and Australia show falling incidences of EOGBS and maternal complications through screening their pregnant mothers at 35 to 37 weeks of gestation to prevent these perinatal and early neonatal infections^{7,8}.

The aim of this study was to assess GBS colonization rates during pregnancy in order to minimize morbidity and mortality in neonates and perinatal mothers in Sri Lanka and to evaluate antibiotics sensitivity pattern of isolated GBS,

Method

This descriptive cross sectional study was carried out at maternity clinic at T H Peradeniya over a period of four months from June to September 2016 as a pilot study. The samples size of 44 was calculated to detect a 13% of colonization rate -according to latest available data for

Asia⁹ with 95% level of confidence and 10% confidence interval. Consecutive 50 samples from mothers with POA of 35 or more who attended the maternity clinic from June – September 2016 and willing to participate after obtaining written consent were included to the study. Low vaginal and/or perianal swab were collected by the medical officer of the clinic and were enriched and processed at the Microbiology Laboratory at Faculty of Allied Health Science, University of Peradeniya.

Ethical approval obtained from Ethics Review Committee of Faculty of Medicine, University of Peradeniya (2016/EC/34)

Results

A total of five subjects (10%) were colonized with GBS at low vagina and/or rectum (Figure 1) out of 50. Of those, 3 were in 20-29 year age group and 2 in 30-40 year age group (Figure 2). Distribution of parity and GBS carriage is shown in Figure 3.

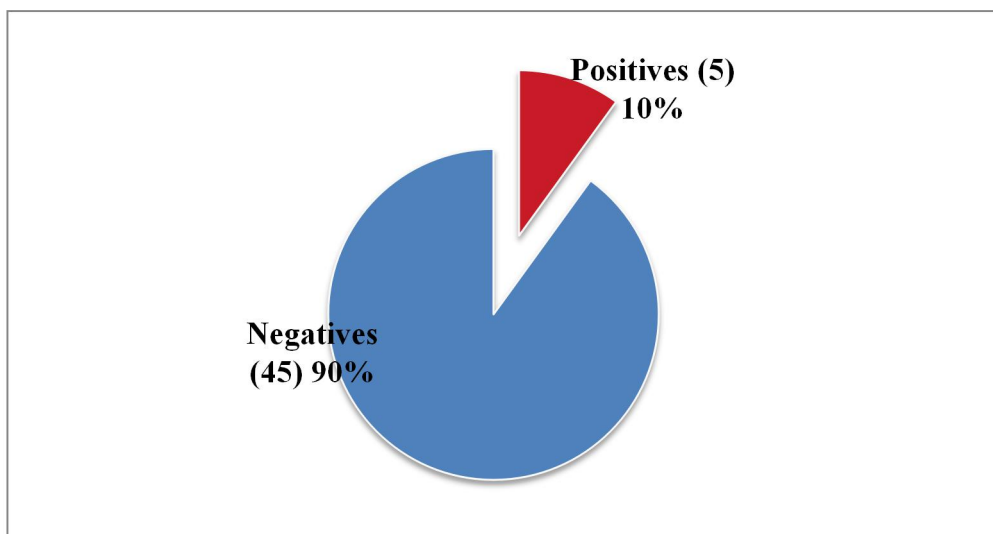


Figure 1: GBS colonization rate

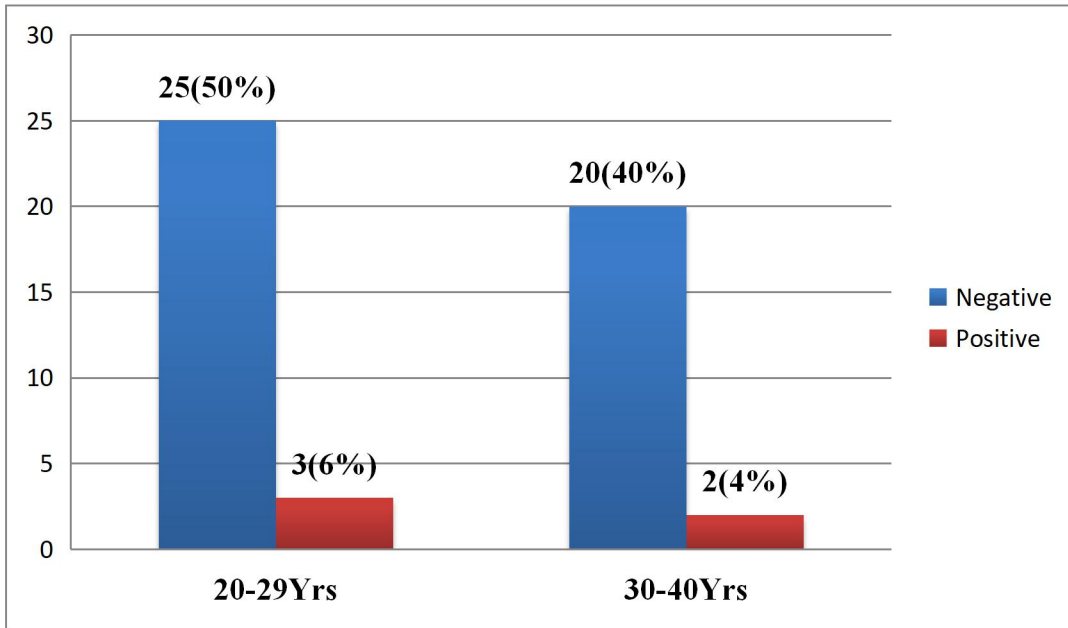


Figure 2: Distribution of age and GBS carriage

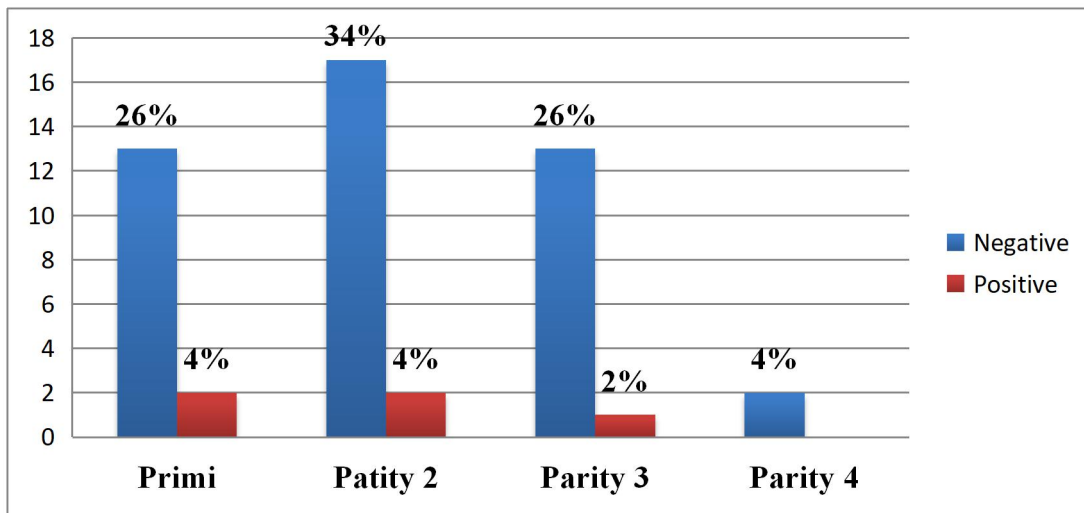


Figure 3: Distribution of parity and GBS carriage

There were no documented past history of GBS colonization, UTI, neonatal sepsis or GBS related sepsis among the study population.

All isolates were 100% sensitive to Penicillin, ampicillin, Cefotaxime, Clindamycin and erythromycin according to CLSI-2015-M100-A24.

Discussion

The overall prevalence for GBS colonization in different countries show figures ranging from 5% to 30% depending on the different regions of the world with a rate of 13% in South Asian region^{4,5,9}. This study revealed rate of maternal colonization with GBS as 10%, which is comparable to latest data of South Asian countries. These mothers need early administration of intravenous antibiotics during labor to prevent early neonatal invasive disease in the first week of life^{2,9}.

Penicillin is the antibiotic of choice used as a prophylactic agent and for the treatment of GBS. Our study showed 100% sensitivity for Penicillin which is comparative to most of the studies conducted over the world^{2,7}. Clindamycin is the alternative for Penicillin/Ampicillin allergy. All our isolates were 100% sensitive to Clindamycin in contrast to different studies^{2,7}.

Screening of pregnant women at >35 weeks of gestation is recommended as a useful tool to obtain early information, for timely action and to minimize possible early onset neonatal sepsis and perinatal maternal septic events.

The work was funded by the annual allocations for the Microbiology Laboratory of the Department of MLS, Faculty of Allied Health Science, University of Peradeniya.

References

- [1] Morven SF, and Carol JB. Streptococcus agalactiae (Group B Streptococcus) in Mandell, Douglas, and Bennett's Principles of Infectious Diseases. 8th ed. Philadelphia: Elsevier; 2015. p. 3140-48d
- [2] Jennifer RV, Lesley McGee, Stephanie JS. Prevention of Perinatal Group B Streptococcal Disease Revised Guidelines from CDC, 2010 Morbidity and Mortality Weekly Report www.cdc.gov/mmwr November 19, 2010 / Vol. 59 / No. RR-10
- [3] Libster R, Edwards KM, Levent F, Morven S. E, Marcia AR, Castagnini RNDL, Cooper T, Sparks RC, Baker CJ and Shah PE. Long-term outcomes of group B streptococcal meningitis. Pediatrics. 2012;130: p. e8-e15.
- [4] Dulmini Nanayakkara, Veranja Liyanapathirana, Chaminda Kandauda, Champika Gihan, Asela Ekanayake and Dinuka Adasooriya, 2018: Maternal vaginal colonization with selected potential pathogens of neonatal sepsis in the era of antimicrobial resistance, a

single center experience from Sri Lanka BMC Infectious Diseases 18:35 <https://doi.org/10.1186/s12879-018-3262-y>

- [5] Brimil N, Barthell E, Heindrichs V, Kuhn M, Lutticken R and Spellerberg A. 2006: Epidemiology of Streptococcus agalactiae colonization in Germany. The International Journal of Medical Microbiology. 296(1): p. 39 – 44
- [6] Russell NJ, Seale AC, O'Driscoll M, O'Sullivan C, Bianchi-Jassir F, Gonzalez-Guarin J, Lawn JE, Baker CJ, Bartlett L, Cutland C, Gravett MG, Heath PT, Le Doare K, Madhi SA, Rubens CE, Schrag S, Sobanjo-TMA, Vekemans J, Saha, SKM. 2017 GBS Maternal Colonization Investigator Group. Maternal Colonization With Group B Streptococcus and Serotype Distribution Worldwide: Systematic Review and Meta-analyses. Clinical infectious diseases: an official publication of the Infectious Diseases Society of America, 65(suppl_2), S100-S111.
- [7] Phares CR, Lynfield R, Farley MM, Mohle-Boetani J, Harrison LH, Petit S, Craig AS, Schaffner W, Zansky SM, Gershman K, Stefonek KR, Albanese BA, Zell ER, Schuchat A, Schrag SJ. 2008: Active Bacterial Core surveillance/Emerging Infections Program Network. *JAMA* 299(17):p. 2056-65. doi:10.1001/jama.299.17.2056
- [8] Rodriguez-Grangert J, Alvargonzalez JC, Beradi A, Berner R, Kunze M, Hafnagel M, Melin P, Decheve A, Orefici G, Poyert C, Telford J, Efstration A, Killian M, Krizova P, Baldassari L, Spellerberg B, Puertas A and Fraile RM. 2012: Prevention of group B streptococcal neonatal disease revisited. The DEVANI European project. European Journal of Clinical Microbiology & Infectious Diseases, 31(9): p. 2097–104
- [9] http://www.who.int/immunization/newsroom/press/news_group_b_strep_stillbirths_infant_deaths_2017/en/#.W_gIzom_PFc.email last viewed 2018 Nov