# Group B Streptococcal Colonisation among Pregnant Women Attending a Tertiary Care Hospital of Northeast India

AMIT KUMAR SINGH<sup>1</sup>, TASO BEYONG<sup>2</sup>, LOVELEENA AGARWAL<sup>3</sup>, CHUING LUNDUP<sup>4</sup>, VERBENA BEZBARUAH<sup>5</sup>

(CC) BY-NC-ND

# **ABSTRACT**

Microbiology Section

**Introduction:** Group B *Streptococcus* (GBS) is an important cause of maternal as well as neonatal morbidity and mortality worldwide. Early identification of colonisation of GBS among pregnant women plays an important role in preventing neonatal disease by taking measures such as antibiotic prophylaxis. In India, the spectrum of disease caused due to GBS is largely unrecognised due to lack of screening practices and also there is no specific guideline to prevent the disease.

**Aim:** To determine the group B streptococcal colonisation and their antibiotic susceptibility profile among pregnant women of North-east India.

**Materials and Methods:** The study was a hospital based crosssectional survey conducted from April to June 2019. A total of 295 pregnant women with gestational age more than 35 weeks attending the Outpatient and Inpatient Departments were included in the study. Two vaginal swabs and two rectal swabs were collected from each participant and were processed according to standard laboratory protocol. Identification of GBS was done on the basis of Christie-Atkins-Munch-Peterson (CAMP) test and Lancefield grouping by latex agglutination test. Antibiotic susceptibility profile was also obtained for these isolates for certain antibiotics. Chi-square test was applied to determine the association of isolation among different types of cases.

**Results:** Out of 295 pregnant women, 15(5.1%) showed GBS colonisation. There was no significant association found with age or socioeconomic status. However, GBS colonisation was found to be significantly associated with increasing gravidity (p-value=0.03). GBS colonisation of vaginal flora is significantly associated with rectal colonisation (p-value <0.01). Although certain isolates were found to be resistant to macrolide antibiotics (66.7%), all strains were uniformly sensitive (100%) to penicillin, levofloxacin, quinupristin-dalfopristin, vancomycin and linezolid.

**Conclusion:** A low rate of colonisation was determined among the pregnant women and it is not associated with age and socioeconomic status. However, it is suggested that routine screening of pregnant women especially multigravida women should be done to prevent the transmission to the newborn.

Keywords: Group B streptococcus carriers, Meningitis, Neonatal mortality, Sepsis

# **INTRODUCTION**

Maternal sepsis is an important cause of maternal mortality worldwide [1]. The prevalence of maternal sepsis is highest in South Asia and sub-Saharan Africa leading to 14% and 10% of maternal deaths respectively [2]. Although the maternal deaths due to sepsis declined due to knowledge of hygienic childbirth practice and antibiotics, still up to 10% of pregnant women experience febrile morbidity [3]. Maternal infection have both short and long-term effects not only on maternal health but also lead to preterm labour, stillbirth, neonatal sepsis and have long term effect on health and growth of child [4,5]. Data on aetiology of maternal sepsis leading to maternal, perinatal and neonatal mortality and morbidity are limited. GBS; *Streptococcus agalactiae* which is a part of commensal flora in vagina, intestinal tract, is an important cause of maternal as well as neonatal morbidity and

Is an important cause of maternal as well as neonatal morbidity and mortality worldwide [6]. Risk factors associated with GBS colonisation among pregnant women includes age, socioeconomic status, literacy, delivery at <37 weeks, intrapartum temperature >38°C, premature rupture of membrane [7]. It is an important pathogen causing maternal sepsis because it is colonised in 1 in 5 pregnant women, and there is an increased risk of invasive disease in pregnancy [8]. Although due to advances in antenatal and neonatal care, the fatality rates has been decreased but, it still remains an important cause of perinatal morbidity and mortality [9]. In pregnant women, it is an important cause of chorioamnionitis, postpartum endometritis, urinary tract infections, postcaesarean febrile illness and endocarditis [10]. Among newborns, approximately 50-60% of the cases of GBS happen in first week (early-onset) causing sepsis, pneumonia, and stillbirth whereas late onset disease primarily manifests as meningitis [11,12].

Streptococcus agalactiae is an encapsulated gram-positive cocci and part of flora of lower gastrointestinal and genital tracts of pregnant women [13]. The rate of colonisation of GBS in vagina and rectum among pregnant women varies with geographical locations, ethnic group and age [14]. It varies from as low as 3% to as high as 60% with a vertical transmission rate of approximately 50-60% [13,15]. In developed countries, GBS carriage rates is found to be between 20-40%, whereas studies from the developing countries showed a comparatively lower prevalence rates [16,17]. In India, data is very limited, few studies showed a lower prevalence ranges from 2.3% to 5.8% [17,18]. Early identification of colonisation of GBS among pregnant women plays an important role in preventing neonatal disease by taking measures such as antibiotic prophylaxis [19].

In order to prevent the neonatal diseases due to GBS, Centres for Disease Control and Prevention (CDC) recommends the use of intrapartum antibiotic prophylaxis among vaginorectal carriers of GBS [20]. The antibiotic agents used for prophylaxis are penicillin, and ampicillin as an alternative. Among penicillin allergic patient, erythromycin or clindamycin is recommended. Although GBS are generally susceptible to these antibiotics; but may show varying resistance to them [21].

In India, the spectrum of disease caused due to GBS is largely unrecognised due to lack of screening practices and also there is no specific guideline to prevent the disease. It also has a wide geographic variation, so knowledge of colonisation rate and its antibiotic susceptibility is important in preventing neonatal infection among GBS colonisers. This will also help to decide the role of routine screening of GBS among pregnant women to prevent maternal and neonatal morbidity.

# MATERIALS AND METHODS

The present study was a hospital based, observational cross-sectional survey conducted in the Department of Microbiology of Tomo Riba Institute of Health and Medical Sciences, Naharlagun, Arunachal Pradesh after obtaining ethical clearance from the Institutional Ethical Committee for the study (No. TRIHMS/ETHICS/01/2019-20/5) from April to June 2019. Informed consent form was duly filled and signed by the participants. A sample size of 295 antenatal women attending the Obstetrics Outpatient and Inpatient Departments was included in the study.

Inclusion criteria: Pregnant women with gestational age >35 weeks, without any medical complications, single or multiple pregnancy, intact or ruptured membranes, unknown GBS status and also those without any history of previously affected children with GBS were included in the study.

**Exclusion criteria:** Women already underwent pelvic examination prior to vaginal swab, known GBS status, with history of children previously affected with GBS or history of antibiotic uptake during past 2 weeks, those with pre-existing medical conditions complicating pregnancy.

### **Data Collection**

After enrolment of the pregnant women for the study on the basis of inclusion and exclusion criteria, information was recorded on preformed questionnaire which includes identification, demographic variables, maternal age, gravidity, parity, gestational age, and maternal complications. Socioeconomic status was determined on the basis of Kuppuswamy Socioeconomic scale which includes occupation, education of head of family and monthly income of the family. Kuppuswamy's socioeconomic status was categorised into upper, upper-middle, lower middle, upper lower, lower class [22].

Sample collection: Four swabs were taken from each patient after obtaining written informed consent from them. Cotton-tip sterile swab in a transport tube was used for sample collection. Two swabs were taken from lower- third of vagina and two swabs were taken from the rectum. Swabs were taken with aseptic precautions before the pelvic examination of the patients by the attending physician as per standard laboratory protocol. Swabs were transported immediately to the microbiology laboratory for further processing.

#### Sample Processing

The swabs sent to the laboratory were processed as mentioned below:

**Inoculation of specimens and direct smear examination:** The swabs were inoculated in the culture media and direct smear examination. One vaginal swab and one rectal swab were inoculated on 5% sheep Blood Agar medium (BA) and a chromogenic selective agar medium (HiCrome Strep B Selective Agar) and incubated aerobically at 37°C temperature overnight. Other set of vaginal and rectal swab were inoculated in Todd-Hewitt broth, an enrichment medium for GBS for 24 hours at 37°C and were subcultured later on 5% sheep Blood Agar (BA) plate and chromogenic selective agar medium. After incubation, the media was observed for growth and identified. Direct smear examination was also done by preparing the smear from the swab from which culture plates were inoculated.

Identification of Group B Streptococcus (GBS): After the growth of colonies on the incubated culture plates, the GBS was identified on the basis of colonial morphology, gram staining, catalase test, Christie, Atkins, Munch-Peterson (CAMP) test and Streptococcal Lancefield grouping.

Antimicrobial susceptibility testing: Antimicrobial susceptibility testing was performed by Kirby-Bauer disc diffusion method. Antibiotic discs used were Penicillin (10 units), Ampicillin (10 µg), Clindamycin (2 µg), Erythromycin (15 µg), Vancomycin (30 µg), Chloramphenicol (30 µg), Levofloxacin (5 µg), Linezolid (30 µg), Quinupristin-dalfopristin (15 µg) applied as four discs on a 100 mm plate. Zone diameter interpretation was done according to Clinical

and Laboratory Standards Institute (CLSI) 2018 criteria [23]. Quality control used was *Streptococcus pneumoniae* American Type Culture Collection (ATCC) 49619.

# STATISTICAL ANALYSIS

All data was collected and recorded in Microsoft (MS) Office Excel Sheet. Chi-square test was applied to determine the association of isolation among different types of cases. The p-value <0.05 was considered as statistically significant.

## RESULTS

Out of 295 pregnant women, included in the study, 15 (5.1%) were found to be positive for GBS colonisation. [Table/Fig-1] showed age wise distribution of pregnant women. Pregnant women of age group 26-30 years are highest in number followed by 15-25 years age group. GBS colonisation was positive in 2 (11%) of women of age group of more than 35 years followed by 5 (7.6%) women of age group 31-35 years. Although pregnant women of age group 26-30 years are highest in number but the rate of GBS colonisation among them is about 4.8%, which is almost equal to overall rate of colonisation of 5.1%. Statistical analysis of the data showed that the GBS colonisation is not significantly associated with any of the age group.

Age (years)	GBS positive cases (n)	GBS negative cases (n)	Total (N=295)	Prevalence	p-value
15-25	2	85	87	2.3%	0.15
26-30	6	118	124	4.8%	0.86
31-35	5	61	66	7.6%	0.29
>35	2	16	18	11.0%	0.23
Total	15	280	295	5.1%	
[Table/Fig-1]: Age wise distribution of cases.					

[Table/Fig-2] showed the distribution of cases on the basis of their socioeconomic status. Pregnant women from lower class constitute the highest number of cases. However, pregnant women of upper middle class found to have a higher prevalence of 10.7%. Statistical analysis of the data showed that the GBS colonisation is not significantly associated with any of the socio-economic group.

Socioeconomic status	GBS positive cases	GBS negative cases	Total	Prevalence	p-value
Upper middle class	3	25	28	10.7%	0.15
Lower middle class	7	116	123	5.7%	0.25
Lower class	5	139	144	3.5%	0.22
[Table/Fig-2]: Distribution of cases on the basis of socioeconomic status. GBS: Group B Streptococcus					

[Table/Fig-3] showed distribution of cases on the basis of gravida of pregnant women. It showed that GBS colonisation was highest among women with gravida more than 3, i.e., 8.9%, while women with primigravida had a lesser rate of colonisation. Pregnant women with higher gravida (G>3) has significant rate of colonisation of GBS (p=0.03).

Gravida	GBS positive cases	GBS negative cases	Total	Prevalence of positive cases	p- value
G-1	4	103	107	3.7%	0.42
G-2	2	85	87	2.3%	0.15
>G-3	9	92	101	8.9%	0.03*
[Table/Fig-3]: Distribution of cases on the basis of gravida. *p-value <0.05 is considered as significant; Chi-square used to calculated p-value					

Rectal swab obtained from pregnant women showed that only 12 (4.1%) pregnant were positive for GBS colonisation [Table/Fig-4]. Out of 15 GBS positive cases from the vaginal swab, 11 were

positive in the rectal swab. GBS colonisation of vaginal microflora is significantly associated with rectal colonisation ( $\chi^2$ =194.297, p-value <0.01).

	No. of vaginal swabs				
Rectal swab results	GBS positive cases	GBS negative cases	Total (%)		
GBS positive cases	11	1	12 (4.1%)		
GBS negative cases	4	279	283 (95.9%)		
Total	15	280	295		
[Table/Fig-4]: Comparison of GBS colonisation rate among pregnant women.					

Antibiotic susceptibility pattern of all the 15 GBS isolates is documented in [Table/Fig-5]. All the strains were uniformly sensitive to penicillin, levofloxacin, quinupristin-dalfopristin, vancomycin and linezolid. Isolates were found resistant to clindamycin (40%), erythromycin (33.3%), chloramphenicol (20%) and ampicillin (20%).

Antibiotics	No. of isolates sensitive (n)	% of sensitive isolates		
Penicillin	15	100		
Ampicillin	12	80		
Erythromycin	10	66.7		
Clindamycin	9	60		
Chloramphenicol	12	80		
Levofloxacin	15	100		
Quinupristin-dalfopristin	15	100		
Vancomycin	15	100		
Linezolid	15	100		
[Table/Fig-5]: Antibiotic susceptibility pattern of GBS isolates (n=15)				

# DISCUSSION

The GBS was found to be an important cause of infection during perinatal period both to mother and newborns. The clinical importance of GBS lies in the fact that despite it can cause serious neonatal infections such meningitis and wide range of invasive and noninvasive infections to the mother, intrapartum antibiotic prophylaxis to the colonised mothers can help in reduction of burden of early onset disease in the new born. To achieve this, CDC has also provided guidelines to screen the pregnant women not more than five weeks before delivery to determine the colonisation of GBS [24].

It has suggested that, there is widespread variation in the colonisation of GBS with geographic location, sociodemographic status, sexual activity and gravidity [18]. Studies showed that developed nations had a higher rate of colonisation as compared to developing countries. Among developed countries, USA has wide range of variation in rate of colonisation from 15-40%, whereas Sweden, UK and Canada has 25.3%, 21.3% and 19.5% rate of colonisation, respectively. Among developing countries, Lebanon and Brazil has got 17.7% and 17.9% colonisation rate, whereas in India, it was found to be very low in Vellore (5.8%) and Pondicherry (2.3%) [16].

In this study, the rate of GBS colonisation among pregnant women was found to be 5.1%, which is low in comparison to a study conducted by Santhanam S et al., Nancy A and Deepak M, Chaudhary M et al., Saha SK, et al., Clouse K et al., Strus M et al., Namugongo A et al., which showed colonisation of 7.6%, 7.7%, 15%, 15%, 19.5%, 20% and 28.8%, respectively [16,25-30]. Whereas, Sharmila V et al., and Khatoon F et al., found a very low rate of GBS colonisation of 2.3% and 2%, respectively [18,31]. Dechen TC et al., showed rate of colonisation of 4.77% which is similar to the present study [32].

The present study hasn't found any significant association of GBS colonisation with any age group which is consistent with the findings of study conducted by Sharmila V et al., Saha SK et al., Namugongo A et al., [18,27,30]. However, Khatoon F et al., and Rick AM et al., found a significant association of colonisation with increasing age [31,33]. Whereas, Nancy A and Deepak M, has found a significance

rate of colonisation among younger women (18-25 years) compared to older women (30-35 years) [25].

Khatoon F et al., found that the pregnant women of upper middle class had significant rate of colonisation [31], whereas in this study there is no significant association of socioeconomic status with rate of colonisation which is consistent with the findings of study conducted by Zusman AS et al., [34]. However, Sefty H et al., and Kim EJ et al., found that GBS colonisation was higher among those with lower socioeconomic status [35,36].

In this study, we found a significant association of colonisation with multigravida which is consistent with the findings of the study conducted by Sharmila V et al., Khatoon F et al., Kim EJ et al., Orrett FA [18,31,36,37]. On the contrary, Assefa S et al., Mohammed M et al., Onipede A et al., and Simoes JA et al., found a significant GBS colonisation among primigravida women [38-41]. However, Dechen TC et al., and Arain FR et al., has found no significant association of gravidity with the rate of colonisation [32,42].

Antibiotic susceptibility profile of the isolates obtained in this study are uniform sensitivity to penicillin, levofloxacin, quinupristin-dalfopristin, vancomycin and linezolid. Similar findings were observed in the study conducted by Sharmila V et al., Khatoon F et al., Arain FR et al., Tsolia M et al., Barcaite E et al., [18,31,42-44]. However, resistance was found for erythromycin and clindamycin which are an important alternative to penicillin in the patients allergic to penicillin. Similar findings were showed by Sharmila V et al., Strus M et al., Assefa S et al., Arain FR et al., and Tsolia M et al., [18,29,38,42,43]. Intrapartum antibiotic prophylaxis being an important tool to prevent the transmission of infection to neonate from the mother. Development of drug resistance may affect the outcome of treatment and thus, morbidity and mortality associated with the disease.

#### Limitation(s)

The present study was conducted in limited duration and the sample size was also less. Along with that follow-up of cases was not done due to the nature of study due to which resistance of certain drugs and their response in the patients were not monitored.

# CONCLUSION(S)

The present study has determined a lower rate of colonisation of GBS among pregnant women and all isolates were sensitive to penicillin. However, it showed resistance to macrolide antibiotics which are best alternatives to penicillin allergic individuals. Therefore, it can be suggested that a routine screening for all pregnant women at least five weeks prior to delivery, to detect the colonisation with GBS. Also, a significant association of colonisation was found with higher gravidity. Multigravida women need to be screened routinely few weeks prior to delivery to prevent the transmission.

#### Acknowledgement

The study was conducted as Indian Council of Medical Research shortterm studentship project for the year 2019 (Reference id: 2019-00738).

#### REFERENCES

- [1] Filippi V, Chou D, Ronsmans C, Graham V, Say L. Levels and Causes of Maternal Mortality and Morbidity. In: Black RE, Laxminarayan R, Temmerman M, et al., editors. Reproductive, Maternal, Newborn, and Child Health: Disease Control Priorities, Third Edition (Volume 2). Washington (DC): The International Bank for Reconstruction and Development/The World Bank; 2016 Apr 5. Chapter 3.
- [2] Say L, Chou D, Gemmill A, Tunçalp Ö, Moller AB, Daniels J, et al. Global causes of maternal death: A WHO systematic analysis. Lancet Glob Health. 2014;2(6):e323-33.
- [3] Maharaj D. Puerperal pyrexia: A review. Part I. Obstet Gynecol Surv. 2007;62(6):393-99.
- [4] Krohn MA, Hillier SL, Baker CJ. Maternal peripartum complications associated with vaginal group B streptococci colonisation. J Infect Dis. 1999;179(6):1410-15.
- [5] Bellizzi S, Bassat Q, Ali MM, Sobel HL, Temmerman M. Effect of puerperal infections on early neonatal mortality: A secondary analysis of six demographic and health surveys. PLoS One. 2017;12:e0170856.
- [6] Shabayek S, Spellerberg B. Group B streptococcal colonisation, molecular characteristics, and epidemiology. Front Microbiol. 2018;9:437. PMID: 29593684.

- [7] Goel N, Wattal C, Gujral K, Dhaduk N, Mansukhani C, Garg P. Group B Streptococcus in Indian pregnant women: Its prevalence and risk factors. Indian J Med Microbiol. 2020;38(3):357-61.
- [8] Hall J, Adams NH, Bartlett L, Seale AC, Lamagni T, Bianchi-Jassir F, et al. Maternal disease with Group B streptococcus and serotype distribution worldwide: Systematic review and meta-analyses. Clin Infect Dis. 2017;65(suppl\_2):S112-24.
- [9] Melin P. Neonatal group B streptococcal disease: From pathogenesis to preventive strategies. Clin Microbiol Infect. 2011;17(9):1294-303.
- [10] Schuchat A. Epidemiology of group B streptococcal disease in the United States: Shifting paradigms. Clin Microbiol Rev. 1998;11(3):497-513.
- [11] Simonsen KA, Anderson-Berry AL, Delair SF, Davies HD. Early-onset neonatal sepsis. Clin Microbiol Rev. 2014;27(1):21-47.
- [12] Bundy LM, Noor A. Neonatal Meningitis. [Updated 2019 Feb 28]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2019 Jan. Available from: https://www.ncbi.nlm.nih.gov/books/NBK532264/.
- [13] Musleh J, Al Qahtani N. Group B Streptococcus colonisation among Saudi women during labor. Saudi J Med Med Sci. 2018;6(1):18-22.
- [14] Woldu ZL, Teklehaimanot TG, Waji ST, Gebremariam MY. The prevalence of Group B Streptococus recto-vaginal colonisation and antimicrobial susceptibility pattern in pregnant mothers at two hospitals of Addis Ababa, Ethiopia. Reprod Health. 2014;11:80. PMID: 25476269.
- [15] Patil KP, Singla SS, Nagmoti MB, Swamy MK. Group B Streptococci colonisation in pregnant women: Is screening necessary? J South Asian Feder Obst Gynae. 2013;5(2):64-67.
- [16] Santhanam S, Jose R, Sahni RD, Thomas N, Beck MM. Prevalence of group B Streptococcal colonisation among pregnant women and neonates in a tertiary hospital in India. J Turk Ger Gynecol Assoc. 2017;18(4):181-84.
- [17] Mani V, Jadhav M, Sivadasan K, Thangavelu CP, Rachel M, Prabha J. Maternal and neonatal colonisation with group B Streptococcus and neonatal outcome. Indian Pediatr. 1984;21(5):357-63.
- [18] Sharmila V, Joseph NM, Babu TA, Chaturvedula L, Sistla S. Genital tract group B streptococcal colonisation in pregnant women: A South Indian perspective. J Infect Dev Ctries. 2011;5(8):592-95.
- [19] Patras KA, Nizet V. Group B Streptococcal maternal colonisation and neonatal disease: Molecular mechanisms and preventative approaches. Front Pediatr. 2018;6:27.
- [20] Schrag S, Gorwitz R, Fultz-Butts K, Schuchat A. Prevention of perinatal group B streptococcal disease. Revised guidelines from CDC. MMWR Recomm Rep. 2002;51(RR-11):01-22.
- [21] Motlova J, Strakova L, Urbaskova P, Sak P, Sever T. Vaginal & rectal carriage of Streptococcus agalactiae in the Czech Republic: Incidence, serotypes distribution & susceptibility to antibiotics. Indian J Med Res. 2004;119(suppl):84-87.
- [22] Ramesh Masthi NR, Gangaboraiah, Kulkarni P. An exploratory study on socio economic status scales in a rural and urban setting. J Family Med Prim Care. 2013;2(1):69-73. Doi: 10.4103/2249-4863.109952.
- [23] CLSI. Performance Standards for Antimicrobial Susceptibility Testing; 29<sup>th</sup> ed. CLSI Supplement M100. Wayne, PA. Clinical and Laboratory Standards Institute; 2019; 88-91.
- [24] Verani JR, McGee L, Schrag SJ. Prevention of perinatal group B streptococcal disease-revised guidelines from CDC, 2010. MMWR Recomm Rep. 2010;59(RR10):01-32.
- [25] Nancy A, Deepak M. A Meta-analysis for association of Maternal Group B streptococcus colonisation and preterm birth in Indian population (October 19, 2018). Available at SSRN: https://ssrn.com/abstract=3271425.
- [26] Chaudhary M, Rench MA, Baker CJ, Singh P, Hans C, Edwards MS. Group B streptococcal colonisation among pregnant women in Delhi, India. Pediatr Infect Dis J. 2017;36(7):665-69.

- [27] Saha SK, Ahmed ZB, Modak JK, Naziat H, Saha S, Uddin MA, et al. Group B Streptococcus among pregnant women and newborns in Mirzapur, Bangladesh: colonisation, vertical transmission, and serotype distribution. J Clin Microbiol. 2017;55(8):2406-12.
- [28] Clouse K, Shehabi A, Suleimat AM, Faouri S, Khuri-Bulos N, Al Jammal A, et al. High prevalence of Group B Streptococcus colonisation among pregnant women in Amman, Jordan. BMC Pregnancy Childbirth. 2019;19(1):177.
- [29] Strus M, Pawlik D, Brzychczy-Wloch M, Gosiewski T, Rytlewski K, Lauterbach R, et al. Group B streptococcus colonisation of pregnant women and their children observed on obstetric and neonatal wards of the University Hospital in Krakow, Poland. J Med Microbiol. 2009;58(2):228-33.
- [30] Namugongo A, Bazira J, Fajardot Y, Joseph N. Group B Streptococcus colonisation among pregnant women attending antenatal care at tertiary hospital in rural southwestern Uganda. Int J Microbiol. 2016;2016:3816184.
- [31] Khatoon F, Nigam A, Sharma NR, Srivastava R, Sangal R, Malik N. Prevalence and risk factors for group B streptococcal colonisation in pregnant women in northern India. Int J Reprod Contracept Obstet Gynecol. 2016;5(12):4361-64.
- [32] Dechen TC, Sumit K, Ranabir P. Correlates of vaginal colonisation with Group B streptococci among pregnant women. J Glob Infect Dis. 2010;2(3):236-41.
- [33] Rick AM, Aguilar A, Cortes R, Gordillo R, Melgar M, Samayoa-Reyes G, et al. Group B Streptococci colonisation in pregnant guatemalan women: Prevalence, risk factors, and vaginal microbiome. Open Forum Infect Dis. 2017;4(1):ofx020.
- [34] Zusman AS, Baltimore RS, Fonseca NS. Prevalence of maternal group B streptococcal colonisation and related risk factors in a Brazilian population. Braz J Infect Dis. 2006;10(4):242-46.
- [35] Sefty H, Klivitsky A, Bromberg M, Dichtiar R, Ben Ami M, Shohat T, et al. Factors associated with choice of approach for Group B streptococcus screening. Isr J Health Policy Res. 2016;5:42. Published 2016 Nov 15. Doi: 10.1186/s13584-016-0103-6.
- [36] Kim EJ, Oh KY, Kim MY, Seo YS, Shin JH, Song YR et al. Risk factors for group B streptococcus colonisation among pregnant women in Korea. Epidemiol Health. 2011;33:e2011010. Doi: 10.4178/epih/e2011010.
- [37] Orrett FA. Colonisation with group B streptococci in pregnancy and outcome of infected neonates in Trinidad. Pediatr Int. 2003;45(3):319-23.
- [38] Assefa S, Desta K, Lema T. Group B streptococci vaginal colonisation and drug susceptibility pattern among pregnant women attending in selected public antenatal care centers in Addis Ababa, Ethiopia. BMC Pregnancy Childbirth. 2018;18(1):135.
- [39] Mohammed M, Asrat D, Woldeamanuel Y, Demissie A. Prevalence of group B Streptococcus colonisation among pregnant women attending antenatal clinic of Hawassa health center, Hawassa, Ethiopia. Ethiop J Health Dev. 2012;26(1):36-42.
- [40] 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):135-43.
- [41] Simoes JA, Alves VM, Fracalanzza SE, de Camargo RP, Mathias L, Milanez HM, et al. Phenotypical characteristics of group B streptococcus in parturients. Braz J Infect Dis. 2007;11(2):261-66.
- [42] Arain FR, Al-Bezrah NA, Al-Aali KY. Prevalence of maternal genital tract colonisation by Group B Streptococcus from western province Taif, Saudi Arabia. J Clin Gyn Obs. 2015;4(3):258-64.
- [43] Tsolia M, Psoma M, Gavrili S, Petrochilou V, Michalas S, Legakis N, et al. Group B streptococcus colonisation of Greek pregnant women and neonates: Prevalence, risk factors and serotypes. Clin Microbiol Infect. 2003;9(8):832-38.
- [44] Barcaite E, Bartusevicius A, Tameliene R, Kliucinskas M, Maleckiene L, Nadisauskiene R. Prevalence of maternal group B streptococcal colonisation in European countries. Acta Obstet Gynecol Scand. 2008;87(3):260-71.

#### PARTICULARS OF CONTRIBUTORS:

- 1. Associate Professor, Department of Microbiology, Tomo Riba Institute of Health and Medical Sciences, Naharlagun, Arunachal Pradesh, India.
- 2. Assistant Professor, Department of General Medicine, Tomo Riba Institute of Health and Medical Sciences, Naharlagun, Arunachal Pradesh, India.
- 3. Professor, Department of Microbiology, Prasad Institute of Medical Sciences, Lucknow, Uttar Pradesh, India.
- 4. Tutor, Department of Microbiology, Tomo Riba Institute of Health and Medical Sciences, Naharlagun, Arunachal Pradesh, India.
- 5. MBBS Student (2<sup>nd</sup> year Proff.), Tomo Riba Institute of Health and Medical Sciences, Naharlagun, Arunachal Pradesh, India.

# NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR: Taso Beyong,

Department of Medicine, TRIHMS, Naharlagun, Arunachal Pradesh, India. E-mail: tasobeyong3@gmail.com

#### AUTHOR DECLARATION:

- Financial or Other Competing Interests: Yes
- Was Ethics Committee Approval obtained for this study? Yes
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. NA

PLAGIARISM CHECKING METHODS: [Jan H et al.]
ETYMOLOGY: Author Origin
Plagiarism X-checker: Dec 28, 2020

- Manual Googling: Mar 11, 2021
- iThenticate Software: Apr 01, 2021 (14%)

Date of Submission: Dec 27, 2020 Date of Peer Review: Feb 03, 2021 Date of Acceptance: Mar 11, 2021 Date of Publishing: Jul 01, 2021