Original Article

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Microbiology Section

Group B Streptococcal Colonisation among Antenatal Women from a Tertiary Care Centre, Northern Kerala: A Cross-sectional Study

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ABSTRACT

Introduction: Group B *Streptococcus* (GBS) is the leading cause of neonatal sepsis all over the world. Maternal colonisation of GBS in vaginal and anorectal area poses risk for subsequent invasive disease. The prevalence of maternal colonisation varies with geographical, biological and socio-economical factors. Current recommendations consider maternal screening and antibiotic therapy to prevent GBS neonatal disease with a potential of alteration of infant gut flora.

Aim: To find out the prevalence, outcome and antimicrobial susceptibility pattern of the GBS colonisation in antenatal women.

Materials and Methods: A hospital-based descriptive cross-sectional study was conducted in Government Medical College, Kozhikode, Kerala, India, during December 2017 to May 2019 including 300 antenatal women of 35-37 weeks gestational age. Vaginal and rectal swabs were taken and were subjected to microbiological examination and culture. Data analysis was done by Statistical Package for the Social Sciences (SPSS) version 16.0. The Chi-square test and Fisher's exact test were used wherever applicable and p-value <0.05 is considered significant.

Results: Among the 300 antenatal women, 8 (2.7%) were colonised with GBS. Higher rate of colonisation was observed in women of 21-25 years, higher parity, rural dwelling and in women with poor glycaemic control. All the colonised women received antibiotic prophylaxis with intravenous ampicillin. None of the colonised women or the neonates born to them developed any invasive GBS infection. Antibiotic susceptibility testing showed that all the isolates were sensitive to penicillin, ampicillin and vancomycin but only 62.5% of the isolates were susceptible to clindamycin and 37.5% of the isolates were susceptible to erythromycin.

Conclusion: The prevalence of GBS colonisation is low in Asian countries, compared to the data from western countries. Evidence based usage of narrow spectrum antibiotics should be considered. Further studies regarding prevalence, antibiotic susceptibility pattern, cost benefit analysis of the antibiotic usage and its effect on neonatal gut flora etc, including a wider population, is a need of the hour in the setting of emergence of resistance.

Keywords: Group B *Streptococcus*, Neonatal meningitis, Vaginal colonisation

INTRODUCTION

GBS, also known as *Streptococcus agalactiae* has emerged as a leading pathogen in the last few decades in the West. *Streptococcus agalactiae* is catalase negative gram positive cocci with large (>0.5mm after 24 hours), grey to white, translucent colonies with a narrow zone of beta-haemolysis or non haemolytic in 5% Sheep Blood agar (SBA). It gives a positive Christie, Atkins, and Munch-Peterson (CAMP) factor reaction. Detecting Group B antigens in isolates by latex agglutination methods or detection by molecular methods is considered confirmative for identification [1].

The vaginal and perianal region are the major reservoirs of GBS and the colonisation of these regions is a risk factor for subsequent infection in pregnant women and newborn. Although, GBS comprise normal microbiota of human genitourinary tract, it could cause bacteraemia and urinary tract infections in the colonised adults and can cause sepsis, meningitis and lower respiratory tract infections in neonates. The transmission rate from colonised women is considered to be 1.74 /1000 women [2].

Identification of pregnant women who are asymptomatically colonised by GBS in vagina and treatment with prepartum antibiotics could reduce the complications arising postpartum to both mother and child. Centre for Disease Control and Prevention recommends culture based screening at 35 to 37 weeks of gestation [3]. Currently, ACOG recommends that all pregnant women should undergo antepartum screening for GBS at 36 0/7-37 6/7 weeks of gestation, regardless of planned mode of birth. Only those women for whom intrapartum antibiotic prophylaxis for GBS is indicated because of GBS bacteriuria during the pregnancy or because of a history of a previous GBS-infected newborn is exempted. All women whose cultures are positive for GBS should receive appropriate intrapartum antibiotic prophylaxis unless a prelabour caesarean birth is performed in the setting of intact membranes. Intravenous penicillin (5 million unit iv load and then 2.5 -3 million unit iv every 4 hours till delivery) remains the agent of choice for intrapartum prophylaxis, with intravenous ampicillin (2 gm iv load and then 1gm iv every 4 hours till delivery) as an acceptable alternative [4]. In case of high risk for penicillin allergy, clindamycin (if susceptible) or Vancomycin is the recommended agent.

Maternal colonisation by GBS was observed to range from 2.3-35% with an overall global prevalence of 18% [5,6]. The prevalence of GBS colonisation in pregnant Indian women was estimated to be 7.8% with wide heterogeneity across studies [7]. Only fewer reports are available in the literature regarding the status of people of Kerala, India [8,9]. This study was conducted to find out the prevalence of GBS in asymptomatic antenatal women admitted in a tertiary care centre in northern part of Kerala, India.

MATERIALS AND METHODS

This was a hospital-based descriptive cross-sectional study conducted at Institute of Maternal and Child Health (IMCH), Kozhikode, Kerla, India, during December 2017 to May 2019. The Institutional Ethics Committee (IEC) approval was obtained for the study (Reference number: GMCKKD/RP2017/IEC/260).

Inclusion criteria: Women who were not in active labour and willing to provide informed consent were included in the study.

Exclusion criteria: Women with antepartum haemorrhage, rupture of membranes, indications of previous caesarean sections, women who have undergone pelvic examination less than two hour prior to vaginal swab and those receiving antibiotics or has received antibiotic in third trimester were excluded from the study.

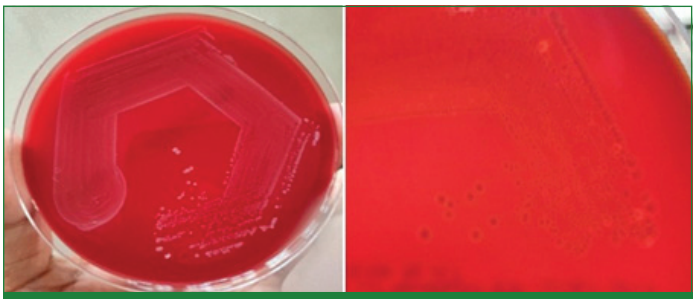
Sample size calculation: The required sample size was calculated as 300 using the formula 4pq/d2, considering average estimated global prevalence of 18% and Confidence interval: 95% [6].

Single vaginal swab and rectal swab, taken prior to pelvic examination, were inoculated in modified Todd Hewitt broth and incubated at 37°C for 24 hours. Broths were observed for turbidity after 24 hours of incubation and subcultured on to 5% SBA and Chromogenic agar (CHROM) media. Plates were examined after 24 hours and 48 hours of incubation at 37°C. The growth of GBS was identified as follows [Table/Fig-1-5] [10].

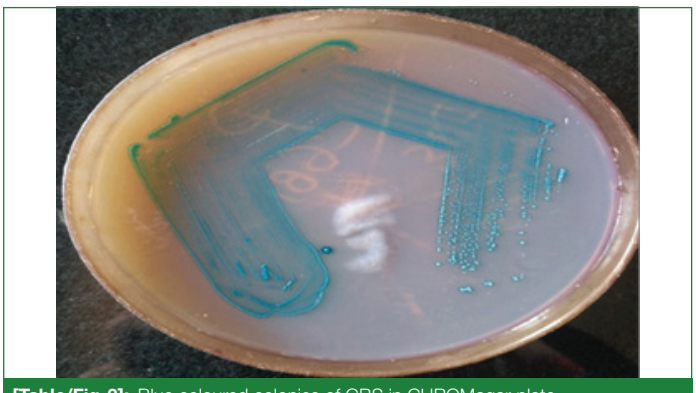
Sl. no.	Test done	Feature
1.	Sheep blood agar	Beta/gamma haemolytic, grey, mucoid colonies [Table/Fig-2]
2.	Mac Conkey's agar	Pink pin point colonies
3.	CHROM AGAR (HiChromStrepB selective agar)	Blue coloured colonies [Table/Fig-3]
4.	Gram stain	Gram positive cocci in pairs and chains [Table/Fig-4]
5.	Catalase	Negative
6.	Aesculin hydrolysis	Not hydrolysed
7.	CAMP Test	Positive [Table/Fig-5]
8.	Arginine dihydrolase	Hydrolysed
9.	Voges-proskauer test	Negative
10.	Co-trimoxazole susceptibility (1.23/23.75 µg)	Resistant
11.	Bacitracin susceptibility (0.04 U)	Resistant
12.	Latex agglutination (HiStrep Latex test kit)	Agglutination with group B specific antisera [Table/Fig-6]

[Table/Fig-1]: Phenotypic characterization of GBS [10].

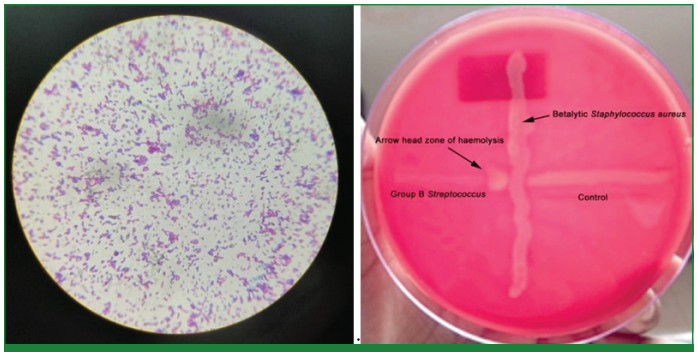
The isolates were confirmed as GBS by Lancefield grouping (agglutination using Group B antigen [HiStrep Latex test kit] [Table/Fig-6].



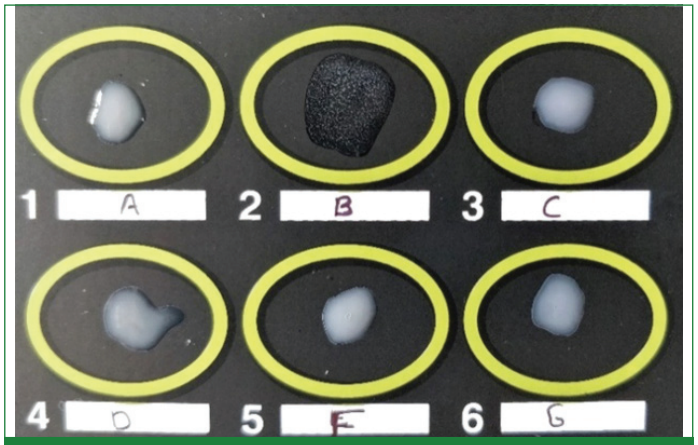
[Table/Fig-2]: Beta/gamma haemolytic, grey, mucoid colonies on sheep Blood Agar.



[Table/Fig-3]: Blue coloured colonies of GBS in CHROMagar plate.



[Table/Fig-4]: Gram positive cocci in pairs and chains (100X). **[Table/Fig-5]:** CAMP test. (Images from left to right)

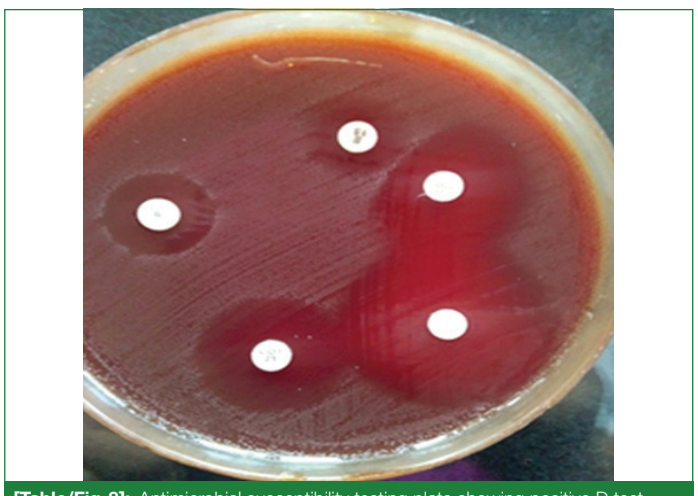


[Table/Fig-6]: Agglutination with group B specific antisera.

Antibiotic susceptibility testing of confirmed isolates was done using Kirby Bauer technique on Muller Hinton agar (using commercially available discs from Himedia) according to Clinical Laboratory Standard Institute (CLSI) guidelines [11] [Table/Fig-7]. D-Test for clindamycin was done by placing erythromycin (15 µg) and clindamycin (2 µg) discs 12 mm apart. Flattening of zone of inhibition of clindamycin near to erythromycin disc (referred to as D zone) indicates inducible clindamycin resistance [Table/Fig-8] [11].

Test group	Antimicrobial agent	Disc content	Interpretive categories and zone diameter breakpoints (in mm)		
			S	I	R
A	Penicilin	10 U	≥24	-	-
A	Ampicillin	10 µg	≥24	-	-
A	Erythromycin	15 µg	≥21	16-20	15
A	Clindamycin	2 µg	≥19	16-18	15
B	Vancomycin	30 µg	≥17	-	-
C	Linezolid	30 µg	≥21	-	-

[Table/Fig-7]: Zone diameter breakpoints for GBS (CLSI M100).



[Table/Fig-8]: Antimicrobial susceptibility testing plate showing positive D test.

Mothers confirmed with GBS carriage and their new born were followed-up till day seven by monitoring their medical records and the details were recorded.

STATISTICAL ANALYSIS

Data entry and analysis were done using statistical software SPSS for Windows Version 16.0 (SPSS Inc, Chicago, IL, USA). Percentages were calculated for categorical variables. Means and Standard Deviations (SD) were calculated as required for numerical variables. The Chi-square test and Fisher's-exact test were used. The relative risk, 95% confidence interval and p-values of outcome parameters were calculated. The p-value <0.05 is considered significant.

RESULTS

Among 300 antenatal women, eight were found to be colonised with GBS (2.7%). The study population comprised of antenatal women at 35-37 weeks of gestation with mean age of 22.93 yrs. About 167/300 (55.7%) hailed from urban areas while 133/300 (44.3%) were from rural area, 153/300 (51%) were primigravida. The demographic factors and co-morbid conditions of the study population and the association of the factors with GBS colonisation is depicted in [Table/Fig-9]. 2/8 (25%) colonised women harboured both vaginal and rectal colonisation [Table/Fig-10].

Sl. no	Factors considered	Distribution of study population (n=300)	Distribution of GBS colonisation (n=8)	Association with GBS colonisation (p-value)*
1.	**Age distribution: 18-34 yrs**			
	<20	78 (26%)	2 (25%)	0.609
	21-25	158 (52.7%)	3 (37.5%)	
	26-30	59 (19.6%)	3 (37.5%)	
	>31	5 (1.7%)	0	
2.	**Residence**			
	Rural	133 (44.3%)	4 (50%)	0.744
	Urban	167 (55.7%)	4 (50%)	
3.	**Occupation**			
	Home maker	271 (90.3%)	6 (75%)	0.252
	Student	5 (1.7%)	1 (12.5%)	
	Daily wage worker	12 (4%)	1 (12.5%)	
	Business	1 (0.3%)	0	
	Teacher	7 (2.3%)	0	
	Others	4 (1.3%)	0	
4.	**Gravida**			
	Primigravida	153 (51%)	2 (25%)	0.281
	2nd gravida	134 (44.7%)	5 (62.5%)	
	3rd gravida	10 (3.3%)	1 (12.5%)	
	4th gravida	3 (1%)	0	
5.	**Diabates mellitus**			
	Present	59 (19.7%)	4 (50%)	0.029
	Absent	241 (80.3%)	4 (50%)	
6.	**Hypertension**			
	Present	29 (9.7%)	2 (25%)	0.137
	Absent	271 (91.3%)	6 (75%)	

[Table/Fig-9]: Demographic factors and co morbid conditions of the study population.
*Fisher's exact test

Site of colonisation	Number of cases
Vaginal	3 (37.5%)
Rectal	3 (37.5%)
Recto-vaginal	2 (25%)

[Table/Fig-10]: Anatomical distribution of colonisation.

In the study population, 19.7% had diabetes and of these, 6.8% were colonised with GBS while only 1.7% of the non diabetic population had GBS colonisation. Diabetes mellitus was found to have statistically significant correlation with GBS colonisation in antenatal women (p-value=0.029) [Table/Fig-9].

All eight isolates tested were penicillin, ampicillin and vancomycin susceptible. Antibiotic susceptibility pattern of GBS isolates is shown in [Table/Fig-11]. Erythromycin resistance was observed in 5/8 (62.5%) isolates and 3/8 (37.5%) of the isolates were resistant to clindamycin [Table/Fig-12].

Antibiotics	Sensitive	Resistant
Penicillin	8 (100%)	0
Ampicillin	8 (100%)	0
Erythromycin	3 (37.5%)	5 (62.5%)
Clindamycin	5 (62.5%)	3 (37.5%) (D test positive-1 isolate)
Vancomycin	8 (100%)	0

[Table/Fig-11]: Antibiotic susceptibility pattern of GBS isolates.

Erythromycin	Clindamycin	No of isolates	Phenotype
S	S	2	
R	R	1	cMLSB
S	R	1	L-phenotype
R	S	3	M phenotype
D test positive		1	iMLSB

[Table/Fig-12]: Resistance phenotypes of GBS (n=8).
cMLSB: constitutive macrolide-lincosamide-streptogramin B; i:indusible

Outcome: Intrapartum history and outcome of babies born to colonised women is shown in [Table/Fig-13,14] respectively. All the colonised women received intravenous ampicillin therapy .None of the women developed invasive GBS disease. The infants born to colonised women were followed-up to seven days and none of them developed GBS infection.

Intrapartum history			
1	Temperature	Normal-7 (87.5%)	Elevated-1 (12.5%)
2	Liquor	Normal-5 (62.5%)	Meconium stained-2 (25%)
			Foul smelling-1 (12.5%)
3	Rupture of membranes	Spontaneous-5 (62.5%)	Artificial-3 (37.5%)
4	Antibiotic prophylaxis (Inj. Ampicillin iv)	Received-8 (100%)	Not Received-0
5	Mode of delivery	NVD-7 (87.5%)	LSCS-1 (12.5%)

[Table/Fig-13]: Intrapartum history of women colonised with GBS (n=8).
NVD: Normal vaginal delivery; LSCS: Lower segment caesarean section

1.	**Birth weight**	
	Normal	6 (75%)
	Low	2 (25%)
	Very low	0
2.	Early onset sepsis	0
3.	Meningitis	0
4.	Pneumonia	0

[Table/Fig-14]: Outcome of neonates born to GBS colonised women (n=8).

DISCUSSION

GBS is a leading pathogen causing neonatal sepsis worldwide. The prevalence of GBS varies with different population. Maternal GBS colonisation is the most important predictor of perinatal GBS infections to both mother and baby. Studies have shown that the transmission rate can be as high as 45.02% [12]. Thus maternal carriage of GBS is a major concern for the obstetrician and neonatologist.

The present study was designed to find out the prevalence of colonisation of GBS in lower vagina and rectum in asymptomatic

antenatal women of 35-37 weeks of gestation showed a prevalence of 2.7%. Various studies reported from India shows that the prevalence rate of GBS in antenatal women varies between 2-15% [Table/Fig-15] [9,12,13,15-17]. A meta-analysis by Ashary N et al., including 36 Indian studies (1981-2019) comprising of 9778 samples, estimated that the prevalence of GBS in pregnant Indian women as 7.8% [7]. None of the study had prevalence equivalent to the estimated average global prevalence of 18% [6]. Published systematic literature reviews also shows a lower prevalence of GBS in the southern and eastern Asian countries [6]. A study conducted in racially diverse group of pregnant women also demonstrated lowest colonisation in women of South Asian origin (23.3%; OR=0.8) although they all were residing at UK [15].

Study	Year of publication	Place	Study population	GBS colonisation rate
Sharmila V et al., [12]	2011	Puducherry	300	2.30%
Khatoon F et al., [13]	2016	Uttar Pradesh	300	2%
Santhanam S et al., [15]	2017	South India	305	7.60%
Goel N et al., [16]	2020	Delhi	469	3.3%
Gogoi M et al., [17]	2021	Assam	345	15.1
Warrier LM et al., [9]	2022	Kerala	315	12.9%
Present study	2022	Northern Kerala	300	2.7%

[Table/Fig-15]: GBS colonisation rate in various Indian studies [9,12,13,15-17].

In present study 37.5% of the colonised women had either vaginal or rectal carriage only while 25% had both rectal and vaginal carriage. Various studies showing colonisation in different sites are shown in [Table/Fig-16] [12,16]. Rectal carriage was low compared to the vaginal carriage and the low isolation rate may also be due to the profound growth of other normal rectal flora in culture based studies. Wider use of non culture based techniques like nucleic acid amplification tests can provide better insights in this regard, but is in limited use because of financial constraints.

Study	Year of publication	Vaginal only	Rectal only	Combined rectovaginal
Sharmila V et al., [12]	2011	28.6%	28.6%	42.9%
Goel N et al., [16]	2020	53.3%	26.6%	20%
Present study	2022	37.5%	37.5%	25%

[Table/Fig-16]: Prevalence of GBS colonisation in vagina, rectum and both [12,16].

In current study, about 51% of the study population were primiparous women and 44.7% were second gravida women and GBS colonisation was found to be highest in second gravida (62.5%) followed by primi gravida (25%). Although a recently published study conducted in a large population with prevalence of colonisation 29.1%, showed that higher parity (≥2) is associated with higher colonisation (35.3%), with the odds of colonisation over 40% higher than for nulliparous women [18], present study failed to show any statistically significant correlation with parity. This could be due to higher proportion of primigravida (51%) in the study population. The rural or urban dwelling and the about 19.7% of the study population had pregnancy complicated by diabetes mellitus, among which four were colonised with GBS. Diabetes mellitus was found to be a statistically significant risk factor for GBS colonisation (p-value=0.029). [Table/Fig-17] [19-22] shows other studies suggesting diabetes mellitus as a risk factor for GBS colonisation and invasive disease.

Risk factor	Studies	Year of publication, place	Relative risk
Diabetes mellitus	Jackson LA et al., [19]	1995, Atlanta	3.0 (95% CI, 1.9-4.7)
	Farley MM et al., [20]	1993, Atlanta	30 (95% CI, 11-79)
	Edward JM et al., [21]	2019, USA	1.12 (95% CI, 1.01-1.23)
	Ramos E et al [22]	1997, USA	3.1 (95% CI, 1.6, 4.1)
	Present study	2022, Northern Kerala	4.08 (95% CI, 1.05-15.85)

[Table/Fig-17]: Correlation of GBS colonisation with diabetes mellitus.

Penicillin is considered to be the drug of choice for GBS prophylaxis in antenatal women. All the isolates (8/8) were sensitive to penicillin and ampicillin. Although GBS are considered penicillin susceptible universally, resistance are being reported from GBS isolated from clinical samples like sputum, joint aspirate etc. Penicillin resistant Group B *Streptococcus* (PRGBS) was first reported from Japan, (14 isolates from sputum samples) with isolation rate of 2.3% during the period of 2005-2006 [23]. Molecular characterisation of those strains showed that altered PBP 2X due to Q557E and V405A amino acid substitutions are the basis of the reduced susceptibility [15]. Later on PRGBS was reported from USA and Canada [24-26]. The four isolates reported from USA were from invasive GBS disease and had PBP 2X mutation (Q557E) corresponding to a resistance-conferring pneumococcal mutation. The PRGBS reported from Canada were isolated from prosthetic joint.

Among the eight isolates, 62.5% (5/8) were erythromycin resistant and 37.5% (3/8) were resistant to clindamycin. M phenotype (macrolide—Streptogramin B resistance and Lincosamide susceptibility) was observed in 3 (37.5%) isolates, cMLSB (constitutive macrolide—lincosamide—Streptogramin B resistance) phenotype (both erythromycin and clindamycin resistant) was observed in 1 isolate (12.5%), iMLSB (inducible resistance) was observed in one (12.5%) isolate (D-Test Positive) and L-phenotype in one (12.5%) isolate (Erythromycin sensitive but clindamycin resistance). Resistance of erythromycin varies from 4-58.3% and clindamycin from 2.3-57.9% for in published literatures [27-30]. Active Bacterial Core surveillance (ABCs, CDC 2010) reported 28% resistance to clindamycin and 49% resistance to erythromycin [31]. [Table/Fig-18] gives a comparison of the different resistance rates reported from across the world [12,32-36]. The increased rate of erythromycin resistance prompted CDC to exclude it from the alternative drug options for GBS prophylaxis in case of penicillin anaphylaxis.

On the verge of the reports of increasing antimicrobial resistance, although low prevalence of GBS is identified, it is imperative to conduct more studies in this regard, particularly focusing upon the antimicrobial susceptibility pattern.

Study	Year of study	Place of study	Total number of GBS isolates considered	Percentage of resistance reported					
				Penicillin	Ampicillin	Erythromycin	Clindamycin	Vancomycin	Linezolid
Castor ML et al., [32]	1996-2003	USA	2937	0	0	25.6	12.7	0	0
Quiroga M et al., [33]	2004-06	Brazil	62	0	0	9.7	3.3	0	0
Sharmila et al., [12]	2011	Pondicherry	7	0	-	14.2	0	-	-
Berg BR et al., [34]	2014	USA	387	0	-	45.2	28.7	0	-
Present study	2017-19	Northern Kerala	8	0	0	62.5	37.5	0	0
Safari D et al., [35]	2021	Indonesia	53	0	0	19	21	0	0
Van Du V et al., [36]	2021	Vietnam	272	0	0	76.23	58.21	0	-

[Table/Fig-18]: Percentage of resistance of GBS reported from various studies [12,32-36].

Limitation(s)

The recommended screening interval has changed from 35-37 weeks (per CDC 2010 guidelines) to 36 0/7 to 37 6/7 weeks (ACOG 2019 recommendations) [37]. Due to lack of resources serotyping of the isolates and genomic analysis for virulence factors were not done. Only those women who were admitted in a tertiary care centre were included in the study. This might have resulted in an unequal distribution of cases with respect to various variables under consideration.

CONCLUSION(S)

The prevalence of GBS colonisation is low in our community compared to the western studies. Intrapartum antibiotic therapy proved to be 100% effective in preventing maternal and neonatal infection in perinatal period. Being a low prevalence area, it might be more cost effective to give intrapartum antibiotics to mothers in labour with identified risk factors that place their new born at higher risk of early-onset infection. The increasing rate of antimicrobial resistance also mandates for an evidence based usage of antibiotics. Screening should be considered mandatory atleast in women with penicillin allergy, since the alternative drugs shows high rate of resistance.

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