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ORIGINAL ARTICLE

Group B Streptococcal Colonization among Pregnant Women and Neonates in a Tertiary Care Hospital in South India

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Abstract

Objectives To assess the prevalence of maternal and neonatal group B Streptococcal colonization, incidence of neonatal systemic illness, and antibiotic sensitivity of isolates.

Methods This prospective cohort study was conducted in a South Indian tertiary care hospital. Rectovaginal swabs from pregnant mothers at $36^{0/7}$–$37^{6/7}$ wk gestation and throat and rectal swabs from their neonates at 48 h of age were collected. Presence of group B Streptococcus (GBS) was identified by broth enrichment step, and traditional microbiologic methods and antibiotic sensitivity of isolates was noted. All mothers received intrapartum antibiotic prophylaxis (IAP). Culture-positive sepsis, clinical sepsis, pneumonia, meningitis, and urinary tract infection were defined as neonatal systemic illness. Neonates of colonized mothers were followed at 3 mo for late-onset sepsis.

Results Of the 310 mothers, 40 were GBS colonized (prevalence: 12.9%; 95% CI 9.2%, 17.6%). None of the neonates were colonized. Maternal GBS colonization was significantly associated with premature rupture of membrane (RR - 2.93, 95% CI - 1.66–5.16) and neonatal systemic illness (RR - 2.78, 95% CI - 1.39–5.54). Positive correlation was noted between duration of IAP ≤ 4 h and neonatal illness and between maternal GBS colonization and Apgar at 1 min ≤ 4. Clindamycin resistance was noted in 20%. All neonates remained well at 3 mo follow-up.

Conclusion High maternal colonization alerts the need for GBS screening in India. Clindamycin resistance among GBS isolates questions its effectiveness as alternative therapy in penicillin allergy.

Keywords Group B Streptococcus · *Streptococcus agalactiae* · Pregnant women · Neonatal/Newborn · Colonization · Risk factors · Prevalence

Introduction

Group B Streptococcus (GBS) or *Streptococcus agalactiae*, a gram-positive diplococcus in the Lancefield group B was first described in 1887 as causative agent in bovine mastitis, and in 1935 as human pathogen [1]. In the 1970s, GBS became one of the leading infectious causes for early neonatal morbidity and mortality in developed countries with case-fatality rates around 50% [2]. The emergence of prevention strategies in the 1970s, and the issuance of universal screening in 2002 resulted in a drastic decline in GBS infections [3]. Unfortunately, GBS infections still remain an under-recognized problem in developing countries like India [3].

GBS colonizes the lower genitourinary and gastrointestinal tracts in pregnant mothers. Though usually asymptomatic, it can cause infections like chorioamnionitis, postpartum endometritis, urinary tract infections, febrile illness, and rarely, endocarditis in pregnant mothers [4]. The primary risk factor for GBS infection in neonates and young infants being maternal GBS colonization [5], other risk factors include gestational age < 37 wk, very low birth weight, premature rupture of membranes (PROM), intra-amniotic infection, younger maternal age group, black race, heavy rectovaginal colonization, GBS bacteriuria, and previous sibling with GBS infection. Vertical transmission occurs usually during labor or after membrane rupture with a

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50% transmission rate. Around 1%–2% of these newborns develop early-onset GBS (EOGBS) infection in the absence of intrapartum antibiotic prophylaxis (IAP) [4]. EOGBS occurs through vertical transmission, fetal or neonatal aspiration during labor or delivery, or both and manifests clinically within the first 2 d of life as sepsis, pneumonia, and meningitis. Late-onset GBS (LOGBS) develops in infants after 7 d and up to 3 mo of age, predominantly by horizontal transmission from mother, and less commonly acquired from hospital or community [4]. Universal prenatal screening by rectovaginal culture, appropriate specimen collection and processing, IAP, and neonatal care are the vital measures to prevent EOGBS infection. Universal GBS screening is recommended by American College of Obstetricians and Gynecologists (ACOG) for pregnant women between $36^{0/7}$ and $37^{6/7}$ wk gestation. Intrapartum antibiotics are mandatory for those women who tests positive with exception of preterm cesarean birth with intact membranes [4]. There are no definitive guidelines formulated by health departments for screening and prevention of GBS in India. Available data from India project a 1.76%–16% colonization rate and a vertical transmission rate of 53%–56% [3].

The objectives of the study were to estimate the prevalence of group B Streptococcal colonization in pregnant women between $36^{0/7}$ and $37^{6/7}$ wk of gestation attending antenatal clinic in a tertiary care center, to assess GBS colonization rates in neonates, to study the incidence of neonatal systemic illness, and to describe the antibiotic-sensitivity profile of the GBS isolated from the mother–infant dyads and thereby help in antibiotic stewardship.

Material and Methods

A prospective cohort study was conducted in the obstetric and neonatology departments in a tertiary level hospital from September 2019 to July 2020 after approval from institutional scientific committee and ethics committee. All pregnant women attending the antenatal clinic were counseled at 34 wk. Rectovaginal swabs from pregnant mothers who consented for the study were taken at $36^{0/7}$–$37^{6/7}$ wk gestation before the first per vaginal examination. Throat and rectal swabs from their neonates were taken at 48 h of age and sent to the microbiology department. The swabs were cultured for GBS and identified by broth enrichment step and traditional microbiologic methods. The swabs inoculated in Todd Hewitt broth, supplemented with colistin (10 µg/mL) and nalidixic acid (15 µg/mL), were then plated onto 5% sheep-blood agar plates. Following incubation for 24 h at 37 °C under 5% CO_2 atmospheric air, the Todd Hewitt broth was then subcultured onto 5% sheep-blood agar. The primary plates and the plates subcultured from Todd Hewitt broth were also checked for growth at 24 and 48 h. Candidate isolates detected on agar media were presumptively identified as GBS if they were catalase negative and produced a positive Christie, Atkins, and Munch-Peterson (CAMP) factor reaction, and were also subjected to latex-agglutination test [6]. Plates were then classified as showing no growth of GBS. Antibiotic susceptibility test was noted for all the GBS isolates by Kirby–Bauer disk-diffusion test. The sensitivity of ampicillin, amoxicillin–clavulanic acid, ampicillin–sulbactam, cefotaxime, clindamycin, penicillin, and vancomycin were checked. Inducible clindamycin resistance was checked by disk-diffusion D zone test. Infants of colonized mothers were followed up at 3 mo of age for late-onset GBS.

In the authors' setting, all pregnant mothers receive intrapartum antibiotic prophylaxis with 2 g of ampicillin or cefuroxime, if penicillin allergic, regardless of the GBS status. Symptomatic neonates born to colonized mothers and those with risk factors for early-onset sepsis had blood cultures taken and were treated with antibiotics until CRP was negative and blood cultures were sterile. If blood culture was negative and neonate asymptomatic, antibiotics were stopped after 48 h. If the neonate remained symptomatic, antibiotics were given for 5 d if blood culture negative, or longer based on the organism if blood culture positive. Complete blood count and C-reactive protein (CRP) of symptomatic neonates were taken at 24 h of age and 48 h, respectively.

Definitions

- Leucocytosis: High WBC count $> 30 \times 10^9$/L on day 1 of life and $> 20 \times 10^9$/L subsequently.
- Leucopenia: Low WBC count $< 5 \times 10^9$/L.
- Elevated CRP: > 10 with rising trend of CRP.
- Fever: A temperature of ≥ 37.5 °C in newborns is considered as the threshold for fever.
- Early-onset disease: A diagnosis by way of isolation of bacteria from sterile sites such as blood or CSF within 7 d after birth.
- Late-onset disease: Disease develops in infants after 7 d and up to 3 mo of age.
- Clinical sepsis: Unwell neonate with clinical signs of sepsis, with negative blood culture.
- Neonatal systemic illness: Either one of culture positive sepsis, clinical sepsis, pneumonia, meningitis, or urinary tract infection was defined as neonatal systemic illness.
- The primary outcome was the prevalence rate of group B Streptococcal colonization in pregnant mothers between 36 and $37^{6/7}$ wk of gestation. The secondary outcome was to estimate the incidence of EOGBS and colonized infants who had late-onset sepsis.

The demographics, microbiological and clinical details of the participants were recorded from the hospital databases

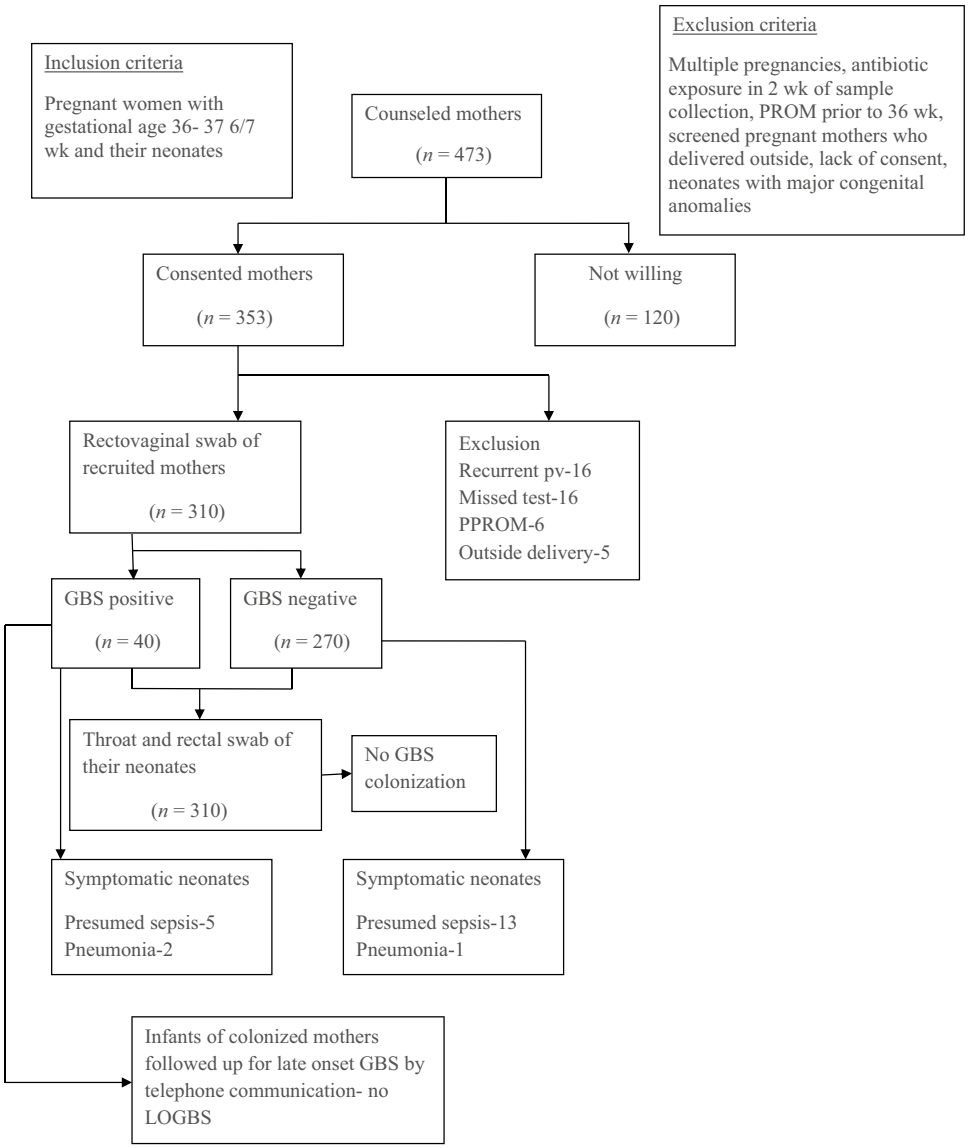

Fig. 1 Enrolment chart. *GBS* Group B streptococcus; *PROM* Premature rupture of membranes; *PPROM* Preterm premature rupture of membranes

during the follow-up of the study participants during the entire study period. Data elements included in this study were: maternal age, gestational age, parity, mode of delivery, duration of membrane rupture (in hours), previous obstetric history, antibiotic use for mother and newborn, newborn gender, birth weight, occurrence of early-onset neonatal systemic illness, neonatal intensive care unit (NICU) admission, and duration of stay.

The study conducted in Vellore by Santhanam et al. showed a prevalence rate of 7.6% (9). An estimated sample size for a prevalence of 7.6% to give a precision of 3% needed a minimum of 300 mother–baby dyads. Assuming 20% dropout rate, the screening of 360 mothers was needed. Continuous variables were analyzed using 2-sample *t*-tests. Categorical variables were analyzed and compared by chi-square or Fisher exact tests. Odds ratios (ORs) and their 95% confidence intervals (CIs) were calculated to assess the risk associated with maternal GBS colonization and neonatal outcome. A p value less than 0.05 was considered statistically significant.

Results

A total of 2278 mothers delivered during the period of the study from September 2019 to July 2020; of which, 2175 deliveries were at ≥ 36 wk gestation (Fig. 1). Figure 1 shows the enrolment of patients. Tables 1, 2, 3, and 4 show the maternal and neonatal demographics and their association with maternal GBS colonization. Of the 310 mothers screened for GBS, 40 mothers were GBS positive, and the prevalence rate was 12.9% (95% CI 9.2%, 17.6%). The

mean age of mothers was 27.4 ± 3.3 y. The mean gestation of babies born to GBS- colonized and not colonized mothers were 38.4 ± 0.9 wk and 38.5 ± 0.9 wk, respectively. The mean birth weight of babies born to GBS-colonized and not colonized mothers were 2.977 ± 0.373 kg and 3.079 ± 0.271 kg, respectively. The median (IQR) of total WBC count, absolute neutrophil count, and CRP of the neonates of GBS-colonized mothers with neonatal systemic illness were 18,640 cells/mm^3 (15,400–21,590), 12,209 cells/mm^3 (9879.2–14,200.2), and 9 mg/L (3.19–15), respectively.

There was significant association between GBS colonization and PROM (*p* - 0.0001, RR - 2.93, 95% CI - 1.66–5.16). Twenty one out of 40 pregnant mothers had PROM (52.5%), though there was no significant association in relation to hours of PROM (*p* 0.61, RR - 0.72, 95% CI - 0.22–2.39). No association was found with respect to age, occupation, gravida, gestational age, mode of delivery, and medical illness. Though statistically insignificant, there was positive association between maternal GBS and previous abortions (*p* 0.06; OR - 2.06, 95% CI - 0.96, 4.44).

None of the neonates were GBS colonized from the throat and rectal swabs or blood culture positive for GBS. There was significant association between mother's GBS status and their newborns requiring neonatal intensive care unit (NICU) admission for neonatal systemic illness (17.5%, *p* 0.009), whereas only 5.5% of newborns born to GBS negative mothers needed NICU admission for neonatal systemic illness. No positive association was found between maternal GBS colonization and gender, sex, birth weight, neonatal resuscitation, and duration of NICU stay. Though statistically not significant, there was positive association between the duration of intrapartum antibiotics within 4 h of delivery and systemic illness in neonates of GBS-colonized mothers (*p* 0.63, RR - 1.84, 95% CI - 0.98–3.45) (Table 5).

All maternal GBS isolates were ampicillin, cefotaxime, penicillin and vancomycin sensitive; however, 20% of isolates were resistant to clindamycin, which signifies the emerging resistance pattern of organism to antibiotics. None of the babies born to GBS-colonized mothers required hospitalization for late-onset GBS disease, which was confirmed

Table 1 Maternal demographics

Demographics	Criteria	Number of mothers, *N* (%)
Maternal age in years	< 20	1 (0.32)
	20–24	59 (19.0)
	25–29	176 (56.7)
	> 30	74 (23.8)
Occupation	Home maker	136 (43.8)
	Government job	33 (10.6)
	Private job	128 (41.2)
	Student	13 (4.2)
Gestational age at delivery in weeks	36−36+6/7	18 (5.8)
	37−38+6/7	148 (47.7)
	≥ 39	144 (46.4)
Gravida	Primi	207 (66.7)
	Multi	103 (33.2)
Medical illness during pregnancy	Pregnancy-induced hypertension	4 (1.3)
	Gestational diabetes	50 (16.1)
	Hypothyroidism	62 (20)
Mode of delivery	Vaginal	246
	Cesarean	52
	Instrumental	12
Indications of cesarean section	Abnormal presentation	3
	Nonprogress of labour	34
	Fetal distress	5
	Meconium-stained amniotic fluid	2
Antibiotic sensitivity of GBS isolates	All sensitive	32 (80)
	Clindamycin resistant	8 (20)

Table 2 Maternal demographics, risk factors, and maternal GBS colonization

Maternal demographics	Criteria	GBS positive *N* (%)	GBS negative *N* (%)	Relative risk (95% CI)	*p* value
Age in years	< 30	32 (80)	204 (75.5)	1.25 (0.60 – 2.60)	.54
	≥ 30	8 (20)	66 (24.4)		
Occupation	Home maker	19 (47.5)	117 (43.3)		.86
	Govt	5 (12.5)	28 (10.3)		
	Private	15 (37.5)	113 (44.8)		
	Student	1 (2.5)	12 (4.4)		
Gestational age in weeks	36–36+6/7	2 (5)	16 (5.9)		.62
	37–38+6/7	22 (55)	126 (46.6)		
	39–41	16 (40)	128 (47.4)		
Gravida	Primi	23 (57.5)	184 (68.1)	0.67 (0.38 – 1.20)	.18
	Multi	17 (42.5)	86 (31.8)		
PROM	Yes	21 (52.5)	64 (23.7)	2.93 (1.66 – 5.16)	**.0001**
	No	19 (47.5)	206 (76.2)		
PROM in hours	< 18	19 (47.5)	60 (22.2)	0.72 (0.22 – 2.39)	.61
	> 18	2 (5)	4 (1.5)		
Mode of delivery	Vaginal	34 (85)	224 (83)	1.14 (0.51 – 2.58)	.75
	Cesarean	6 (15)	46 (17)		
Medical illness	Pregnancy induced hypertension	1 (2.5)	3 (2.5)		.39
	Gestational diabetes	3 (7.5)	47 (7.5)		
	Hypothyroidism	6 (15)	56 (15)		
Previous abortions	Yes	11 (27.5)	42 (15.5)	1.84 (0.98 – 3.45)	.06
	No	29 (72.5)	228 (84.4)		

GBS Group B streptococcus, *PROM* Premature rupture of membranes

by telephonic interviews and review of electronic medical records at around 3 mo of age.

Discussion

GBS infection is one of the leading causes of mortality and morbidity in developed countries, whereas it still remains an iceberg in developing and underprivileged countries. The prevalence of GBS is influenced by various factors like socioeconomic status, geographic region, and ethnicity.

The present study had a GBS prevalence rate of 12.9% which was comparable with the study by Patil et al. (12.15%) in Maharashtra [7] and Dalal et al. (12.03%) [8]. A few studies conducted in India showed a variable prevalence rate from 2 to 16%, Vellore (7.6% in 305 samples) [9], Chandigarh (7.5% in 200) [10], Pondicherry (2.3% in 300) [11], Sikkim (4.77% in 524) [12], Delhi (15% in 300) [13], Karnataka (16% in 50 samples) [14], Uttar Pradesh (2% in 300) [15], and Maharashtra (2.52% in 317 samples) [16]. The samples taken for the studies included rectal, vaginal, throat, endocervix, and high vaginal. Rectovaginal swab was used according to the latest ACOG guidelines, similar to study conducted by Santhanam et al. in Vellore [9].

According to recent meta-analysis by Russel et al. in 2017, the global GBS prevalence rate was 18% with regional variations, with a lower prevalence rate in Southern Asia (12.5%) and Eastern Asia (11%) [17] similar to the present study estimation. The colonization rate in the present study was lower compared to that of Ethiopia (25.5%) [18], Palestine (21%) [19], South Africa (48.2%) [20] and higher than in Greece (6.6%) [21], China (8.2%) [22], Korea (8%) [23]. Sociodemographic factors like age, occupation, educational status, obstetric factors like gravidity, gestational age, parity did not show positive association with the maternal colonization like other various studies reported [21].

In the study by Sharmila et al., there was positive association between gravidity and maternal GBS, though statistically not significant [11], whereas it was statistically significant in the study by Dechen et al. [12]. Another study in India by Khatoon et al. showed significant correlation of GBS with age, parity, socioeconomic status, and that GBS is a risk factor for PROM and preterm labor [15]. Patil et al. also estimated a significant association between PROM, preterm delivery, intrapartum temperature [7].

Newborn throat and rectal samples were tested at 48 h of life to minimize the contamination. As a local policy, intrapartum antibiotics are given for all pregnant mothers during

Table 3 Neonatal demographics

Neonatal characteristics	Criteria	Number of neonates, *N* (%)
Gender	Male	167 (53.9)
	Female	143 (46.1)
Birth weight in kg	< 2.5	19 (6.1)
	2.5–4	287 (92.6)
	≥ 4	4 (1.3)
Needed PPV	Yes	19 (6.1)
	No	287 (92.6) 4 (1.3)
Apgar 1 min	≤ 4	6
	> 4	11
NICU admission	Yes	58 (18.7)
	No	252 (81.3)
Reason for NICU admission	Transient tachypnea	13
	Clinical sepsis	19
	Congenital pneumonia	3
	Neonatal jaundice	36
	Poor feeding	5
	Hypoglycemia	1
	Intraventricular hemorrhage	2
	Seizure	1
	Subgaleal bleed	1
	Erbs palsy	1
	Polycythemia	1
	Adrenal hemorrhage	1
Antibiotics required	Yes	22 (7.1)
	No	288 (92.9)
Duration of NICU Stay	≤ 3	44
	> 3	10

NICU Neonatal intensive care unit; *PPV* Positive pressure ventilation

delivery. One neonate in the present study born to GBS-positive mother who did not receive IAP was symptomatic hours after birth with tachypnea, tachycardia, lethargy, raised counts, CRP with radiograph findings, though blood, urine, and GBS swab cultures were sterile and was treated as pneumonia. Patil et al. [7] showed a significant association with low birth weight in neonates born to GBS-colonized mothers in contrast to the present study where only 10% GBS-colonized mothers had low-birth-weight (LBW) neonates.

Though none of the neonates in the present study showed GBS colonization, a statistically significant association between GBS-colonized mothers and NICU admission of their neonates was found, similar to the study by Patil et al. [7]. NICU admissions with GBS-specific systemic illness included clinical sepsis and pneumonia. In the present study, among the neonates of GBS-colonized mothers, 15 neonates were admitted to NICU for various reasons. Seven of them had neonatal systemic illness, likely due to GBS; of which, 1 mother did not receive intrapartum antibiotic prophylaxis. There was a positive association between neonatal systemic illness and whether GBS-colonized mother received intrapartum antibiotic prophylaxis within 4 h of delivery, though not statistically significant. One neonate of GBS- colonized mother had adrenal hemorrhage, which was similar as in the clinical report by Angelis et al. [24].

In the present study, GBS isolate susceptibility tests showed 20% resistance to clindamycin as in a study by Shah et al. [25]. The Center for Diseases Control (CDC) recommends GBS isolate susceptibility testing especially to clindamycin and erythromycin, since they are the drugs of choice for penicillin-allergic women at high risk for anaphylaxis [22]. The present study shows a higher resistance as compared to various studies in the world, as in Ethiopia (3.2%) [23], South Africa (17.2%) [26] and lower than with various studies as in Kenya (30.4%) [27], China (52.4%) [28]. Fortunately, the present study did not show multidrug resistance unlike other studies [9, 18, 27, 28]. Majority of the studies in India showed antibiotic resistance with gentamicin [15, 16], erythromycin [9], tetracycline [9, 16], kanamycin [15, 16].

Passive immunization of neonates through transplacental transfer of type-specific serum antibodies from their mothers may have eliminated the neonatal invasive GBS disease in the authors' center. Studies by Kohli-Linch et al. and Tann et al. present high morbidity rates of GBS meningitis and encephalopathy, respectively, which highlights the need for universal GBS screening and management of neonatal GBS infection [29, 30]. However, none of the babies in the present study had late-onset sepsis.

Although 353 mothers consented for the study out of 473 counseled mothers, rectovaginal swabs could be collected only for 310 mothers (Fig. 1). Unexpected COVID-19 pandemic and the subsequent lockdown hindered reaching the estimated sample size of 360, which allowed for the 20% dropout rate for the prevalence of 7.6% (9). There could be selection bias in view of the lack of adequate staff for counseling, lack of consent by the mothers, and preterm deliveries in the consented mothers, but they are not likely to differ from the present included study population, and hence, unlikely to vary with their GBS status. Neonatal GBS colonization could not be documented probably because of the regional policy of intrapartum antibiotic prophylaxis for all the mothers, irrespective of the colonization status. The effect of such a policy on the neonate's microbiome also needs to be considered. However, the study depicts an effect of maternal GBS on neonatal sepsis and NICU admissions.

Table 4 Neonatal demographics risk factors and association with maternal GBS colonization

Neonatal characteristics	Criteria	GBS positive	GBS negative	Relative risk (95% CI)	*p* value
Gender	Male	26 (65)	141 (52.2)	1.59 (0.86 – 2.92)	.13
	Female	14 (35)	129 (47.7)		
Birth weight in kg	< 2.5	4 (10)	15 (5.5)	1.70 (0.68 – 4.29)	.27
	≥ 2.5	36 (90)	255 (94.4)		
Needed PPV	Yes	3 (7.5)	14 (5.2)	1.48 (0.41 – 5.41)	.54
	No	37 (92.5)	256 (94.8)		
Apgar at 1 min	≤ 4	2	4	3.67 (0.41 – 32.59)	.24
	> 4	1	10		
NICU admission	Yes	15 (37.5)	43 (15.9)	2.61 (1.47 – 4.62)	**.001**
	No	25 (62.5)	227 (84.1)		
Neonatal systemic illness	Yes	7 (17.5)	15 (5.5)	2.78 (1.39 – 5.54)	**.003**
	No	33 (82.5)	255 (94.4)		
Neonatal systemic illness in relation to GBS**	Clinical sepsis	5*	14		
	Pneumonia	2	1		
Duration in NICU (days)	≤ 3	10 (25)	34 (12.6)		.06
	> 3	5 (12.5)	5 (1.8)	0.45 (0.19 – 1.04)	

*One neonate had transient perinatal depression

**None of the neonates had septicemia, meningitis, or culture−positive urinary tract infection

Table 5 Neonates born to colonized mothers

Time of first dose of antibiotic and delivery	Neonatal-specific systemic illness* *N* = 7 (%)	No neonatal-specific systemic illness *N* = 33 (%)	Odds ratio (95% CI)	*p* value
≤ 4 h	3 (42.8)	13 (39.4)	1.54 (0.27 – 8.82)	.63
> 4 h	3 (42.8)	20 (60.6)		

*One pregnant mother did not receive IAP and the neonate developed pneumonia

Conclusion

Maternal GBS colonization rate was as high as 12.9% in the present study which alarms the need for universal screening for GBS. Intrapartum antibiotics have dramatically decreased the EOGBS infection, but antibiotic resistance is an emerging disaster to deal with. This study showed clindamycin resistance, which cautions its ineffectiveness as a reliable alternative empiric therapy to penicillin-allergic pregnant mothers in future. Replacing intrapartum antibiotics with cost-effective GBS vaccines may prove beneficial to mothers and neonates by decreasing the adverse effects of antibiotics on both by promoting antibiotic stewardship and managing the LOGBS disease.

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Authors' contributions LMW contributed to conception and design, acquisition, analysis, and interpretation; SJ contributed to design, acquisition and interpretation; RRC contributed to design and acquisition; RAB contributed to conception and design, acquisition, analysis, and interpretation and was the senior author; All authors drafted manuscript, critically revised manuscript, gave final approval and agree to be accountable for all aspects of work ensuring integrity and accuracy. RAB will act as the guarantor for this paper.

Data Availability Yes.

Declarations

Ethics Approval The study was approved by the Institutional Ethics Committee, Renai Medicity, Kochi, Kerala; No-HR-RIMS/RMIEC/155–8/2019 dated 6–9-2019.

Conflict of Interest None

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