A STUDY OF GROUP B STREPTOCOCCUS COLONIZATION IN PREGNANT WOMEN

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BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled "**A STUDY OF GROUP B STREPTOCOCCUS COLONIZATION IN PREGNANT WOMEN"** is a bonafide record work done by **DR. FARGANA N** under my direct supervision and guidance, submitted to the Tamil Nadu Dr. M.G.R. Medical University in partial fulfillment of University regulation for M.S Branch II – Obstetrics & Gynaecology.

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DECLARATION

I, **Dr. FARGANA N** solemnly declare that the dissertation titled "**A STUDY OF GROUP B STREPTOCOCCUS COLONIZATION IN PREGNANT WOMEN"** has been prepared by me. I also declare that this bonafide work or a part of this work was not submitted by me or any other for any award, degree, diploma to any other University board either in India or abroad.

This is submitted to The Tamilnadu Dr. M. G. R. Medical University, Chennai in partial fulfillment of the rules and regulation for the award of M.S degree Branch – II (Obstetrics & Gynecology) to be held in May 2022.

Place : Madurai **Dr. FARGANA N**

Date :

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INTRODUCTION

Streptococci have an important position in clinical medicine as human pathogens. Among them Group A *Streptococci* have had a special place as a causative agent of important clinical disease recently. Other members of this family are now coming to light as human pathogens and Group B *Streptococci* (GBS) have emerged as important pathogens within the last few decades.

The GBS are known to cause a wide variety of infections in adults, but clinical interest in these bacteria mainly relates to their ability to cause serious neonatal illnesses especially meningitis and sepsis. In developed countries these organisms are the leading cause of neonatal sepsis and meningitis with a case fatality rate of 40 -80 %. The reason for this is not clear.

Streptococcus agalactiae is the only species that carries the Group B antigen. This organism was initially recognized as a cause of puerperal sepsis. Although this disease is now relatively uncommon, *S. agalactiae* has become better known as an important cause of septicaemia, pneumonia and meningitis in newborn children as well as a cause of serious disease in adults.

Group B streptococcus (GBS) also known as Streptococcus agallactiae is an important cause of maternal and neonatal morbidity and mortality in many parts of the world. It is also implicated in adverse pregnancy outcome. Maternal colonization has been found to be a major risk factor for invasive neonatal GBS disease within 6 days of birth.

In more than 80% of these cases, neonatal GBS infection is acquired during pregnancy and delivery by direct mother to child transmission of the pathogen.

Epidemiologic studies showing the full extent of GBS colonization effect on pregnancy in the country are needed. There is no protocol for screening for GBS prophylaxis in pregnancy in India.

Estimation of GBS colonization rates amongst women during pregnancy and determining its effect on pregnancy can contribute to prevention of negative pregnancy outcomes.

Group B *Streptococci* are Gram-positive cocci (0.6 to 1.2 micrometre size) that form short chains in clinical specimens and longer chains in culture; features that make them indistinguishable on Gram stain from *Streptococcus pyogenes*. They grow well on nutritionally enriched media and in contrast with

the colonies of *Streptococcus pyogenes,* the colonies of *Streptococcusagalactiae* are buttery with a narrow zone of beta haemolysis. Some GBSstrains (1 - 2%) are non-haemolytic, although their prevalence may be underestimated because non-haemolytic strains are not commonly screened for the group B antigen.

Strains of *Streptococcus agalactiae* can be characterised on the basis of three serologic markers.

1. The B antigen or Group-specific Cell Wall Polysaccharide antigen.
2. Type-specific Capsular Polysaccharide Antigen (Ia, Ia/c, Ib/c, II, IIc, II to Viii)
3. The Surface Protein or c protein.

If a pregnant women carries the GBS bacterium in her vagina or rectum at the time of labor and delivery, there is a 1 in 100 chance that her baby will become sick from GBS infection. The risk rises to 4% if a pregnant woman carries the bacterium and also has certain risk factors. These risk factors

include-

- Pre-term delivery before 37 weeks gestation
- Prolonged rupture of membranes (longer than 18 hrs without delivering the baby)
- Fever (100.4^0F or higher) during labour

Other risk factors include having a previous pregnancy resulting in a GBS-infected baby or having a urinary tract infection caused by GBS.

Babies who become sick with GBS infection take the bacterium into their bodies by ingesting GBS-containing amniotic or vaginal fluids during labor and delivery.

There are two forms of GBS infection in infants - Early-onset and Late-onset. Babies with Early-onset infection develop symptoms within seven days of birth; most commonly with in the first day of life. Babies with a late-onset infection develop symptoms between seven days and 3 months of age.

About 80% of all GBS infections in newborns are Early-onset infections. Early-onset infections are almost always transmitted from mother to baby around the time of delivery. Late-onset infections can be contracted at delivery or acquired after birth from contact with the mother or other people who are GBS carriers. Babies with an Early-onset infection suffer from one or more conditions like Pneumonia, Sepsis (Blood infection) and less commonly

Meningitis. Babies with Late-onset infections usually have Sepsis or Meningitis.

In spite of treatment with antibiotics, about 5% of babies with GBS die. Preterm babies are more likely to die from the illness than are full term babies. Most babies who survive GBS go on to develop normally. However among these who develop meningitis, up to 5 % suffer lasting nuerologic damage that can include cerebral palsy, sight and hearing loss, mental retardation, learning disabilities and seizures.

Since 2002, the US guidelines**4** have advised that all pregnant women should be offered screening for GBS carriage at 35–37 weeks of gestation and those found to be colonised with GBS (or labouring before this time) should be offered intrapartum antibiotic prophylaxis (IAP), usually in the form of intravenous benzylpenicillin or ampicillin. IAP has been shown to significantly reduce the risk of culture-positive early-onset but not late-onset disease (occurring 7 or more days after birth). There is also indirect evidence of an impact on neonatal deaths. A longitudinal analysis of disease-related neonatal mortality in the USA showed a decline in mortality in the first week after birth, coinciding with the introduction of IAP.**5** A 2016 report from the USA shows a continuing fall in the incidence of GBS infection without any increase in deaths from other causes of neonatal disease.**6** A Cochrane review of

three trials (all at high risk of bias) including 500 women concluded that IAP for colonised mothers reduced the incidence of EOGBS disease (relative risk 0.14; 95% CI 0.04–0.74) although the numbers of deaths were too small to assess the impact of the intervention on mortality.**7**

There have been no randomised studies addressing whether routine screening has had any impact on all-cause mortality. A positive antenatal screen will result in the recommendation of IAP which carries some risks for the mother and baby. These include anaphylaxis,**8** increased medicalisation of labour and the neonatal period, and possibly, infection with antibiotic-resistant organisms when broad-spectrum antibiotics, such as amoxicillin, are used for prophylaxis.**9**, **10** In the UK, most guidelines recommend that the first-line drug for GBS-specific IAP should be benzylpenicillin, also known as penicillin G. The UK National Screening Committee examined the issue of strategies for the prevention of EOGBS disease in 2016–17 and in March 2017 recommended that routine screening using bacteriological culture or near-patient testing techniques should not be introduced into UK practice.**11**

Role of vaccination to prevent EOGBS disease

An effective vaccine given to pregnant women would be expected to induce high levels of GBS-specific immunoglobulin G in the woman and, via transplacental transfer, in her baby, resulting in protection against neonatal GBS disease (both

EOGBS and late-onset GBS). Phase II trials of a trivalent GBS conjugate vaccine in pregnant women in South Africa and Malawi have demonstrated safety as well as efficient transplacental transfer of vaccine-specific antibodies.**11**, **12** Vaccine manufacturers are now developing pentavalent formulations (i.e. covering 5 of the 10 possible GBS serotypes) which would cover an estimated 96% of EOGBS cases in the UK. Another, or additional, potential mechanism of vaccine protection may be through reduction of maternal GBS colonisation and transmission to the baby. However, no clear effect of vaccination on colonisation was observed in the 2016 pregnancy trial with the trivalent conjugate vaccine.**11** Studies in the UK suggest that vaccination against GBS would be acceptable to pregnant women.**13**, **14**

Lancefield Group B Streptococcus or Streptococcus agalactiae is a type of bacteria that cause illness in newborn babies, pregnant women and was attributed to infant mortality in many parts of the world.Little is known about the burden of perinatal GBS colonisation and related infections in India. GBS has re-emerged as a major pathogen during the last few decades. The vagina and perianal region,rectum are considered major reservoirs of GBS and colonisation of these regions is a risk factor for subsequent infection in pregnant women and newborns.

Worldwide GBS colonization varies between 12% and 27%, however this prevalence varies from place to place, meaning we cannot rely on prevalence of a neighbouring country or continent to estimate the prevalence in our setting. Knowing the prevalence will help determine whether there is a need for Screening of pregnant women attending antenatal clinic for anogenital GBS colonization. While identifying factors associated with its colonization will lead to targeted screening of High risk pregnant women using minimal resources available. To reduce the incidence of neonatal disease caused by GBS, the Centers for Disease Control and Prevention recommends the use of intrapartum antibiotic prophylaxis in pregnant women who are vaginorectal carriers of GBS. Penicillin is recommended as first line agent for prophylaxis, while Ampicillin is considered as an acceptable alternative.In pregnant women allergic to penicillin, Clindamycin or Erythromycin is recommended.Hence, regional knowledge about resistance profile to GBS will guide for appropriate antibiotic prophylaxis

AIMS OF THE STUDY

1. To find out the prevalence of Group B Streptococci colonization in pregnant women.

2. To find out the association of maternal colonization with Group B Streptococci in pregnancy and maternal complications like Premature Rupture of Membranes (PROM), Preterm labor, Maternal fever, Puerperal sepsis.

3. To gain knowledge about antibiotic susceptibility and resistance pattern of the isolates of GBS

REVIEW OF LITERATURE

Group B *Streptococci* is an important cause of neonatal infection in the Western Hemisphere. The recognition that the maternal colonizationwith the organism is a key factor in the occurrence of GBS associatedneonatal morbidity and mortality was a milestone in the history of perinatal health[1]. A nation-wide change in health practices helped diminish mortality and morbidity associated with the disease. In India, however, the spectrumof GBS disease remains a largely under-recognized problem.

Puerperal sepsis has been described for centuries and ancient Indian texts in 1500 BC have recorded that good hygiene leads to a reduction in perinatal disease[2]. In 1879 Louis Pasteur identified *Streptococcus* as the causative organism for puerperal sepsis [3]. Since the early 1930's when Rebecca Lancefield proposed a grouping system for hemolytic *Streptococci*, Group A *Streptococcus* – *Streptococcus pyogenes* was widely acknowledged as the major pathogen associated with puerperal sepsis[4]. GBS was initially thought to be a commensal until 1937, when Fry reported 7 cases of GBS-associated puerperal fever with 3 deaths [5].

There is a spectrum of maternal and fetal GBS infections ranging from asymptomatic colonization to sepsis. *Streptococcus agalactiae* has been implicated in adverse pregnancy outcomes, including preterm labor, prematurely ruptured membranes, clinical and sub-clinical chorioamnionitis, and fetal and neonatal infections. The bacterium can also cause bactriuria, pyelonephritis, and postpartum metritis. Barbosa-Cesnik *et al.* 2003 have reported postpartum maternal osteomyelitis [6] and Berkowitz and McCaffrey, 1990 have reported mastitis by GBS [7].

A higher prevalence has been found in younger women. . GBS is not considered a sexually transmitted infection and treatment of partners does not prevent re-colonisation of treated women. Throughout pregnancy colonization can be transient, intermittent or chronic [8].

GBS SEROTYPES AND PATHOGENESIS

There are 9 antigenically distinct serotypes based on their capsular polysaccharide structure (types Ia, Ib, II-VII) identified to date. In the United States and Western Europe, types Ia, II and III accounted for 85 per cent of the isolates from infants [9,10]. Recent studies in the US have demonstrated that Serotypes Ia, III and V (in descending frequency) accounted for 78 – 87 per cent of early-onset (less than seven days after birth) invasive disease in

newborn infants and parturient women [11,12]. Late-onset GBS disease in infants 7 -90 days of age is dominated by serotype III, followed by serotypesIa and V [12].

Studies from India show a variable distribution of serotypes. But the most common isolates belong to types III, II and Ib [13,14, 15].

The most important risk factor for early-onset GBS infection in the neonate in the presence of the organism in the maternal genitourinary tract at the time of delivery. Ascending bacteria from the maternal genital tractreach the amniotic fluid, usually after rupture of the amniotic membranes [1, 16]. Alternatively the newborn can come into contact with GBS during passage through the birth canal. When the foetus aspirates contaminated amniotic fluid, GBS reach the lower respiratory tract and damage pulmonaryepithelial cells, resulting in pneumonia and respiratory distress usually within the first few hours after birth. Severe GBS sepsis occurs with intravascular invasion of bacteria and failure of the host to eliminate the pathogen [17, 18]. Ascending infection can also occur through intact chorioamniotic membranes, with subsequent events occurring in utero, resulting in still births or death within hours after birth [16]. The pathogenesis in late-onset disease is less clear. Horizontal transmission plays a major role,such as by close contact with the mother, breast-feeding and nosocomial

transmission.

The polysaccharide capsule is the most important virulence factor [18]. However, the role of surface localized GBS proteins in pathogenesis and protection is under intensive investigation [18]. The presence of maternal serum antibodies to specific capsular polysaccharides of GBS serotypesappears to be protective against acquisition of neonatal GBS disease ascolonized pregnant women with high levels of serum antibodies were less likely to have neonates with invasive GBS infection [19, 20]. Moreover, infected infants had low levels of specific antibody to the infective serotype [21].

PREVALENCE OF GROUP B *STREPTOCOCCI*

During the 1970's and 1980's GBS emerged as a major pathogen in the United States and Western Europe with reported mortality rate of 15% - 50 % [22, 23]. In US 10-30% pregnant women are asymptomatic carriers ofGBS in the genital and gastrointestinal tract at the time of delivery. The prevalence of GBS colonization in pregnancy is variable [24] .

Boyer KM, Gadzala et al (1982) reported that women who had positive GBS cultures between 26 and 28 weeks gestation only 65% remained colonized at term while 8% of those with negative prenatal cultures were positive for GBS at term [25]. Treatment of these colonized

mothers succeeded in temporarily eradicating the organism, but most of the women were recolonised within 6 weeks. At birth 50 – 65% of infants who are born to colonized mothers have positive GBS cultures from mucus membranes and skin[26]. Approximately 98% of colonized newborn remain healthy. But 1–2% had invasive GBS infection. The overall incidence of neonatal GBS infection was approximately 2/1000 live births in UnitedStates prior to the introduction of intrapartum prophylaxis [23]

Scrag and associates (2002 – 2003) reported a colonization rate of 20 – 30% in a nation wide cohort sampled at a mean of 35 weeks [27] .

El-Kersh TA, Al-Nuaim LA et al (2002) studied the carrier state of GBS in Saudi females during 3rd trimester of pregnancy . This study included 217 pregnant women and documented a GBS colonization rate of 27.6%. Additionally, 50% of GBS colonized mothers vertically transmitted the organism to their newborns [28].

Orrett FA. *et al*. analyzed 201 third trimester pregnant women of Trinidad and Tobago, West Indies. The prevalence of vaginal and rectal GBS colonization was 32.9%. GBS were isolated more frequently from women aged more than 24 years (36.6%) than those younger than 24 years (26.9%). Colonization rates were significantly greater among multigravid women than primigravid women.

Dillon HC Jr, Gray E, *et al* (1982) did a longitudinal prospective study of carriage of GBS during pregnancy in 2,500 women over a three-year period. Carriage was documented in 18% of the women by anorectal culture, in 4 % by vaginal culture, and in 13 % by simultaneously obtained anorectal and vaginal cultures (Overall carriage rate, 35%). The intestinal tract appeared to be a primary reservoir for colonization in pregnant women [29].

Terry RR Kelly FW. *et al*, (1999) did a study on 608 pregnant women between 1995 and 1997 to consider a number of possible risk factors for GBS. 14.0% of the study subjects were found to be colonized with GBS. White non-Hispanic women had a GBS colonization prevalence of 13.6%; for all others, prevalence was 18.7%. No statistically significant differences were found in regard to age, weight, number of prenatal visits income level, marital status, history of drug use, or parity. This study identified smoking asa possible risk factor for GBS infection with the GBS colonization rate for smokers was 33.1% versus 16.4% for nonsmokers. The authors concluded that routine screening for GBS infection during pregnancy may be beneficialbecause no strong risk factors for colonization exist [30].

Eren A, Kucukercan M, *et al*. (2005) studied 500 Turkish pregnant women and their newborn infants by collecting vaginal and rectal swabs from mothers, and umbilical and throat swabs from their infants. Maternal

and infant colonization rates were found to be 9.2 % and 1.6 %, respectively. Vertical transmission rate was 15.2 %. Although invasive serotypes were predominant, the rarity of GBS disease in their study was thought to be due to low rates of maternal carriage or to their possessing protective levels of GBS-specific IgG antibody in their sera [31].

Gerard P, Verghote–D'Hulst M, *et al* did an epidemiological study and controlled trial of prophylactic treatment of the newborn. Colonization with GBS of the genital tract was studied in 1115 women during the last trimester of pregnancy. 6.82 % of women were found to harbour this bacterium. It was also more frequent in primigravidae. Rupture of the amniotic membranes for more than 24 hours was more often associated with GBS carriage by the mother. 42.6% of the infants born to GBS positive mothers were colonized at birth. The study also indicated that immediate therapy with penicillin of infants of GBS positive mothers has no definite advantage upon delayed treatment [32].

Schrag SJ, Zell ER *et al* in their multistate retrospective cohort study they compared the effectiveness of the screening and risk-based approaches in preventing early-onset GBS disease in a sample of 629, 912 live births in 1998 and 1999. Antenatal screening was documented for 52 percent of the mothers. The risk of early-onset disease was significantly lower among the

infants of screened women than among those in the risk-based group. So routine screening for GBS prevents more cases of early-onset disease than the risk-based approach [33, 34, 35, 36, 37].

Al-Sweih N, Maiyegun S *et al* (2004) Kuwait University, studied the prevalence of GBS in Kuwait population. Anal, vaginal and combined anal and vaginal specimens were obtained from pregnant women at 35-37 weeks of gestation. The combined vaginal and anal specimens were positive for GBS in 6.4 % of women. [38].

PREVALENCE OF GROUP B *STREPTOCOCCI*

Year of Study	**Population studied**	**Sample Size**	**Maternal Prevalence**
El-Kersh TA, Al-Nuaim LA et al (2002)	Saudi Arabia	217	27.6 %
Orrett FA. *et al.* (2004)	West Indies	201	32.9 %
Dillon HC Jr, Gray E, *et al* (1982)	USA	2500	35 %
Terry RR, Kelly FW. *et al*, (1999)	USA	608	14 %
Eren A, Kucukercan M, *et al.* (2005)	Turkey	500	9.20%
Gerard P, Verghote–D'Hulst M, *et a*l (1979)	Belgium	1115	6.82 %
Al-Sweih N, Maiyegun S	Kuwait	110	6.4 %

et al (2004)			

STUDIES IN INDIA

Epidemiological studies in India have shown lower colonization and infection rates in general [13, 39, 40, 41].The reason is not known or the problem has not been adequately studied. There are only a few reports available however on closer analysis.

Stoll BJ, Schuchat A. (1998) reported that with the use of adequate culture techniques and microbiological media some of the GBS colonization rates reported from India and other developing countries are similar to those reported in United States [39].

Kulkarni AA, Pawar SG, *et al*, Maharashtra studied the prevalence of GBS colonization and its risk factors in 317 pregnant women at the time of labour and in their neonates in 1998-1999. The GBS colonization rate in pregnant women at labour and in neonates was 2.52% and 1.26% respectively with a frequency of transmission of 50%. Only one risk factor has been seen in two GBS colonized mothers (i.e. in one case premature delivery and in the other case premature rupture of membranes) to be

associated with GBS transmission . All isolates were sensitive to Ampicillin, Erythromycin and Penicillin followed by Chloramphenicol 66.6% (12/18). All isolates were resistant to Gentamicin, followed by Tetracycline (17/18) 94.4%, and Kanamycin (16/18) 88.8% [42].

Mhaskarrita, Sathyan Sharad *et al* (2005) St. John's Medical College Hospital Bangalore had done a Selective risk factor based screening of pregnant women for GBS colonization. A retrospective analysis was done for the occurrence of GBS colonization among 741 pregnant women who were at risk i.e. they had at least one of the following risk factors namely- prolonged rupture of membranes (>18 hours), preterm labor (<37 weeks), intrapartum fever, vaginal discharge, and previous baby with GBS infection, Vaginal swab and urine cultures indicated GBS. The occurrence of neonatal GBS infection was also studied.

The occurrence of GBS colonization was 1.62% and neonatal GBS infection was 0.53 per 1000 live births. [43,44,45, 46] .

Goyal R, Singh NP *et al*, GBS was examined in 304 pregnant women in Delhi, [2004]. Vaginal specimens were collected and examined for GBS. GBS was isolated from only 4 women (1.3%). It is suggested that GBS infection is not a problem in this population, and mass screening for GBS

during pregnancy is not needed [47].

Dalal BS, Lahiri A *et al* (1998) did a study on 507 pregnant Indian women; 12 per cent were reported to have GBS isolated from the throat and vagina, and 10 per cent had positive vaginal cultures alone [48].

Chaudhary U, Sabherwal U *et al* have reported colonization rates of 5 to 6 per cent, but no selective broth media were used in these cases [13].

PREVALENCE OF GROUP B STREPTOCOCCI IN INDIA

Year of Study	**Population studied**	**Sample Size**	**Maternal Prevalence**
Kulkarni AA, Pawar SG, *et al* (2001)	Govt.Medical College Maharashtra Miraj	317	2.52 %
Mhaskarrita et al (2005)	St. John's Medical College, Bangalore	741	1.62 %
Goyal R, Singh NP (2004)	Guru Tegh Bahadur Hospital, Delhi	304	1.3 %

Jamie WE, Edwards RK *et al* (2004) performed a prospective cohort study to determine whether the rates of recovery of Group B *Streptococci* from vaginal and perianal cultures and combined vaginal and rectal cultures are equivalent. 36% had a positive culture from at least 1 site. A significant

finding was that the detection rate of Group B *Streptococci* from combined vaginal-perianal specimens is not significantly different from the detection rate from vaginal-rectal specimens. The conclusion was that pregnant women need not be subjected to the discomfort of collection of a rectal specimen [49,50,51].

GROUP B *STREPTOCOCCAL* INFECTION IN NEONATES

It is estimated that about half of all babies born to women carrying GBS will themselves be colonized with GBS. However, the vast majority of these infants will not develop symptomatic GBS infection. A nation-wide study involving active surveillance of infants younger than 90 days showed 377 cases of confirmed early-onset GBS neonatal sepsis out of a total population of 794, 037 live births. It was calculated that the risk of early- onset neonatal sepsis developing as a result of being colonized is about 5per 1000 [52.]

Recent evidence has suggested that the incidence of culture-proven GBS neonatal sepsis is likely to underestimate the true incidence of this infection in neonates. Data collected prospectively in one centre in the UK for over a year for 413 neonates who underwent a septic screen in the first 72h after birth have suggested that the true incidence of early-onset neonatal

GBS sepsis may be as high as 3.6 per 1000 live births [53]. This was based on the presence of GBS colonization and symptomatic neonatal sepsis without isolation of any organism from a usually sterile site such as blood orCSF.

In the US, the incidence of culture-confirmed, early-onset GBS neonatal sepsis was 2-3 cases per 1000 live births in the early 1980 [54]. Following the introduction of a national screening and treatment programme advocated by the centers for disease control and prevention, the incidence of confirmed GBS neonatal sepsis has fallen to 0.32 per 1000 live births which is the lowest ever recorded (Fig.1) [55, 56]. In contrast, the rate of late-onset GBS sepsis has remained fairly constant at 3.35 per 1000 live births overthis period, leading to the suggestion that nosocomial infection plays a large part in the etiology of late-onset disease [56].

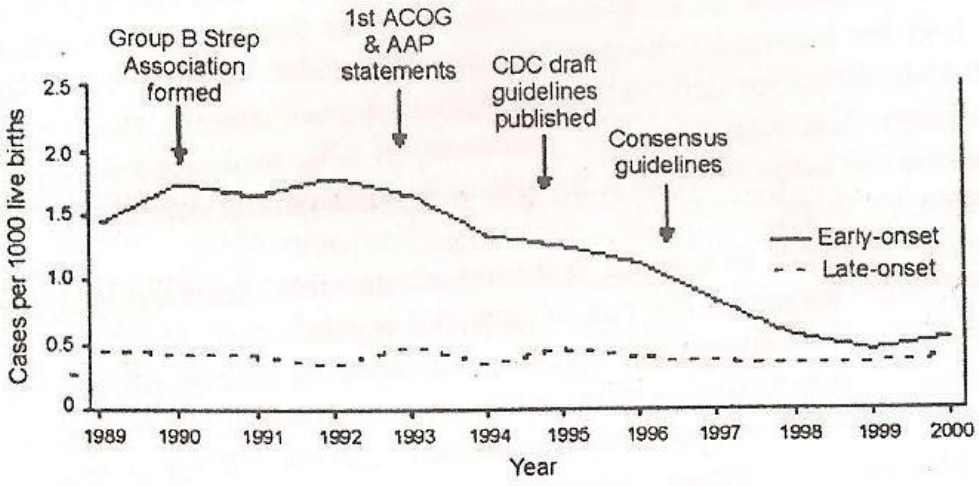


Fig. 1 Incidence of early- and late-onset invasive GBS disease – selected Active Bacterial Core surveillance areas, 1989–2000, and activities for prevention of GBS disease. ACOG, American College of Obstetricians and Gynecologists; AAP, American Academy of Pediatrics. Adapted from CDC (Early onset group B streptococcal disease, United States, 1998–1999. *MMWR* 2000; 49: 793–796) and Schrag *et al*.

NEONATAL MORBIDITY

Two distinct clinical syndromes are recognized, early-and late onset disease. Early onset GBS disease occurs within the first 7 days of life, although most cases are evident in the first 24 h after birth. As can be demonstrated by serotyping GBS isolates from colonized mothers and infants, transmission of early-onset disease is vertical [25]. Infection may be acquired by the intraammiotic route, or directly during passage through the birth canal. The initial presentation is respiratory distress in more than 80 percent of neonates [57]. Pneumonia and septicaemia are the most common manifestations and 5 to 10 percent neonates will also have meningitis. The

incidence of early-onset disease is about 10 times higher in premature than in term neonates. Late-onset disease develops in infants after 7 days and up to 3 months of age, the median age of onset being 1 month. Transmissioncan be either horizontal (from other infected infants or health care workers) or vertical (from the mother due to close proximity). These infants almost always have an unremarkable early neonatal history, and later present with meningitis or sepsis Osteoarticular infections and cellutitis can also occur [58]. The case fatality rate in the US has dramatically decreased over the last 3 decades, from up to 50 per cent in the 1970s to 6 per cent in the early 1990s [59].

Currently most centers and countries have experienced a decline in early –onset neonatal sepsis to rates of less than 1 to 2 per 1000 live births. Most reports, however, indicate no change or variable increases in rates of non-GBS organisms, such as *E. coli* and other Enterobacteriaceae [60, 61, 62, 63.]

There is evidence that the major association with current intrapartum antimicrobial prophylaxis has been an increase in non-GBS early–onsetsepsis in preterm, low-birth weight neonates, and especially very-low-birth weight neonates [63, 64]. Stoll and associates (2002) observed a marked reduction in Group B streptococcal sepsis in these preterm neonates; thiswas offset by an increase in E coli sepsis.

Late-onset neonatal GBS sepsis is less well understood. Cited rates vary from 0.5 to 2 cases per 1000 live births and account for about 50 percent of GBS disease in newborns [65,66]. The incidence of late-onset disease has remained stable despite widespread use of intrapartum antimicrobials, suggesting that GBS screening and chemoprophylaxis intervention may not affect late-onset disease. Lin and co-workers (2003) reported that preterm birth before 34 weeks was the major risk factor [66]. Stoll BJ, Hansen N (2002) found that the late-onset sepsis was identified in a fourth of very-low-birth weight newborns; there was a preponderance of gram-positive organisms, mainly coagulase-negative staphylococci.

The most recent report from the CDC 2002 reports mortality rate of per 100,000 population [67]. Case series from India also report high mortality. In a 1975 study where 8 cases of neonatal GBS infection were followed over a period of 18 months, 6 infants died within the first week of diagnosis [68]. A more recent study published in 1999 reported 10 infants with GBS infection, of which 1 died on the second day of life. However, there is insufficient data presented to calculate the actual mortality rate [69].

Kuruvilla KA, Thomas N *et al* (1999) The incidence of GBS bacteraemia was 0.17 per 1000 live births. Lethargy, respiratory distress and poor perfusion were the presenting features in eight symptomatic babies.

Two babies had meningitis, three required ventilatory support and one died. There were no cases of late onset disease. The low incidence could be due to the low rate of colonisation and high prevalence of protective antibody in the mothers.

The estimated incidence of neonatal GBS infection in India can be calculated from Indian epidemiological data reporting maternal and infant GBS colonization rates as 10 and 5 per cent respectively. Since about 2 per cent of colonized neonates develop true infection, the attack rate of neonatal GBS infection in India may be calculated as approximately 1 per 1000 live births. Bearing in mind the above estimated attack rate and current Indian demographic data (midyear population count in the year 2001 was approximately 1027 million, and birth rate was 26 births per 1000 population per year), the projected total number of GBS infection in newborn infants in India may be a s high as 26,700 cases per year [70].

Strakova ,Motlova *et al* (2003) isolated GBS from 239 full-term and 46 preterm newborns. They reported the incidence of Early onset diseasedue to GBS in Czech Republic is 0.7-1.0 per 1000 live births [71].

NEONATAL PREVALENCE

Year of Study	Population studied	Sample Size	Maternal Prevalence
Kuruvilla et al 1999	CMC Vellore	60119	0.17/1000 live birth
Kulkarni & pawar etal 2001	Govt.Medical College. Miraj	317	1.26 %
Mahskarrita etal 2005	St.John's Medical College Bangalore	741	0.53 / 1000 live birth
National Surveillance Study	UK	568	0.72 / 1000 live birth
Strakova & Motlova etal 2003	Czech Republic	285	0.96 / 1000 live birth

Klebanoff MA, Regan JA, and associates (1995) did a double-blind study to determine whether Erythromycin treatment of pregnant women colonized with GBS would reduce the occurrence of low birth weight. Erythromycin or placebo was given to pregnant women beginning during the third trimester and before 30 weeks and continuing for 10 weeks or until 35 weeks 6 days of pregnancy. Study concluded that treating pregnant women colonized with GBS with Erythromycin was not effective at prolonging gestation or reducing low birth weight [72].

Kurien Anil Kuruvilla, Swati Pillai etal studied on babies born in the Christian Medical College Hospital (CMCH), Vellore,(1995-1996) .Infants with clinical signs of sepsis or those who were born to mothers with potential risk factors for infection were screened for sepsis. Risk factors in the mother for EOS included prolonged rupture of membranes (PROM >24 hours), maternal pyrexia, untreated urinary tract infection (UTI), chorioamnionitis, or multiple vaginal examinations.The incidence of neonatal bacterial sepsis is 9.8 per 1000 livebirths. *E. Coli* and *Klebsiella* were the most common organisms causing EOS and LOS. *Enterococcus fecalis* was also a major pathogen, both in EOS and LOS [73].

OTHER ORGANISMS ASSOCIATED WITH GENITAL TRACT INFECTION

Any infection can lead to preterm labour and premature rupture of membranes .Recently lower genital tract infections has been associated with preyerm labour .one of the important issue being highlighted is bacterial vaginosis .Bacterial vaginosis describes a polymicrobial alteration of vaginal flora resulting from overgrouth of the anaerobic bacteria and gardenella vaginalis than lactobacillus .The major bacterial vaginosis associated organisms are *Gardenella vaginalis*, anaerobic gram negative rods, *Bacteroides*, *Peptostreptococcus*, *Mycoplasma hominis* and *Ureaplasma urealyticum*.

PREVENTION STRATEGIES

Lacking randomized trials, consensus opinions and guidelines on prevention strategies have been promulgated by the American College of Obstetricians and Gynecologists (2002) and the Centers for Disease Control and Prevention (2002). These guidelines advocate a culture –based screening approach to identify women who should receive intrapartum prophylaxis. This recommendation was derived from a multistate, retrospective cohort study of live births in 1998 and 1999 from the Active Bacterial Surveillance/Emerging Infections program network, which suggested that the culture-based approach was superior to a risk-based approach.

With the culture-based approach, women are screened for GBS colonization at 35 to 37 weeks, and intrapartum antimicrobials are given to rectovaginal carriers. Previous siblings with GBS invasive disease and prior identification of GBS bacteruria are also considered indications for prophylaxis. A risk – based approach is recommended for women with un-known GBS culture results at the time of labor.

The choice of antimicrobials may be important in terms of allergic reaction; selection of resistant GBS strains; and emergence of other pathogens, including antimicrobial-resistant strains, as agents of neonatal

sepsis. The centers for disease control and prevention recommendations specify penicillin as a first-line agent [66]. Ampicillin is an acceptable alternative [73]. For women with penicillin allergy, if the risk of anaphylaxis is low, cefazolin is recommended [74]. If the risk of anaphylaxis is high, selection of a prophylactic agent is dependent on GBS susceptibility testing. Patients with isolates susceptible to clindamycin or erythromycin may be given either drug. Antimicrobial resistant strains require vancomycin prophylaxis. This treatment scheme is dependent on laboratory capability to perform susceptibility testing.

Importantly, there have been no randomized controlled trials comparing antenatal screening. In addition, there have been no randomized trials comparing the different screening strategies and whether prenatal GBS screening has a significant impact on overall neonatal sepsis. For these reasons, clinicians in other countries state that there is in sufficient evidence to recommended screening for GBS carriage [75,76,77].

Alternative prevention strategies have been described with limited evidence to recommend them. These include intramuscular benzathine penicillin G and chlorhexidine vaginal lavage [78]. In another study, Haberland colleagues (2002) reported that intrapartum rapid PCR screening for GBS may be superior to current strategies; however, this must be proven effective

in clinical trails [79]. In GBS- Positive women, appropriate vaginal examinations or indicated intrauterine fetal monitoring should not be avoided as their avoidance could actually prolong labor and thus increase therisk of infection[80].

Implementation of several protocols is associated with diminishedGBS. There are, however, still major concerns about antimicrobial resistance, particularly among very-low birth weight neonates. Ongoing surveillance is necessary to monitor protocol efficacy for prevention of early-onset GBS sepsis as well as for any effects on maternal morbidity, overall neonatal sepsis, and resistant infections.

VACCINATION

Some protection against serious neonatal infection is conferred by maternal antibodies. Indeed, Lin and colleagues (2001) have confirmed that the susceptibility to invasive GBS disease correlates with deficiency in maternal type-specific antibody levels [81]. Baker and co-workers reported that maternal immunization to type III antigen produces antibody in about 60 percent of women Monovalent tetanus toxoid conjugate vaccines are immunogenic for common GBS disease –associated serotypes [82,83,84]. Paoletti and Modoff (2002) have reviewed the progress toward development of a multivalent vaccine [85].

It is recommended that 3 g intravenous benzylpenicillin be given as soon as possible after the onset of labour and 1.5 g 4 hourly until delivery. To optimise the efficacy of IAP, the first dose should be given at least 4 hours prior to delivery. There is evidence that benzylpenicillin levels in cord blood exceed the minimum inhibitory concentration for GBS as early as 1 hour after maternal administration[51] but it is not known how this relates to neonatal colonisation or disease. There is also evidence that giving penicillin for 2 hours before delivery reduces neonatal colonisation[52, 53] but evidence from 2013[54] suggests that 4 hours of penicillin is more effective than 2 hours at reducing the risk of EOGBS disease. Amoxicillin is an alternative but the Cochrane review[7] found no difference between amoxicillin and benzylpenicillin and thus, the narrower spectrum antibiotic is preferred.

MATERIALS AND METHODS

Women admitted to Govt Rajaji Hospital, Madurai with labour pain, preterm labour, premature rupture of membranes were included in the study irrespective of gestational age, parity, or socioeconomic status. 150 women were included in the study from Jan 2020 to July 2021.

Source of data

All women admitted in labour room in Department of Obstetrics and Gynaecology at Madurai Medical College, Madurai.

Methods of collection of data

- Study design : A prospective study
- Study Period : 6 month
- Sample design : simple random sampling
- Sample size : 150 women (300 swabs)

A prospective study of Group B Streptococcus in pregnant women admitted in labour room was carried out in the department of OG, Government Rajaji Hospital , Madurai.

A total of 150 women under inclusion criteria have been studied prospectively. Two swabs were taken as one from the lower one-third of vagina and another from the anorectal region with a sterile cotton swab, before pelvic examination. These were transported to Microbiology Department In Stuart's transportation medium.

Samples were introduced into enrichment broth[Todd Hewitt(TH)Broth with Gentamicin and Nalidixic acid. They were incubated overnight aerobically at 35 degree Celsius with 5 to 10% CO2.After 24 to 36 hours, 10ul loopful of the TH broth culture was then subcultured on5% she blood agar and the plates were incubated overnight at 37 degree celsius in 10%CO2. Then the enrichment broth subcultures were examined for presence GBS colonies.Beta hemolytic colonies on sheep blood agar plates,suggestive of GBS were identified by using standard microbiological techniques(smear microscopy, catalase test, bacitracin susceptibility).These colonies were further confirmed by serogrouping using a latex agglutination antigen detection kit.

Detailed information on maternal characteristics like age, parity, gestational age, obstetrical history was noted.Risk status like preterm delivery, premature rupture of membrane, duration of rupture of membrane,intrapartum temperature was noted.All evidence of presence of early signs of sepsis like poor cry, lethargy, poor feeding, respiratory distress,temperature were noted till the stay of mother in the hospital.Reports were entered and analysis was done to know to significance.

Inclusion criteria:

➢ Antenatal mother with any age

➢ Antenatal mother with any parity

➢ Gestational age more than 35week

Exclusion criteria:3

➢ Increased risk of PPH

➢ Already receiving antibiotic

➢ Who have undergone pelvic examination prior to vaginal swab

➢ Cessarean deliveries

Source of data

All women admitted in labour room in Department of Obstetrics and Gynaecology at Madurai Medical College, Madurai.

METHODS

Sample Collection

At the time of admission to the labour Room, two swabs were collected from all pregnant women who were admitted in labour ward.

Maternal Sample Collection

After obtaining the informed written consent, two swabs were collected from each mother-one from the vagina and the other from the rectum.

Vaginal Sample Collection

One swab was collected from the lower vagina taken prior to first pelvic examination. No antiseptic preparation of the perineum or vulva was carried out before swabbing. Speculum was not used for culture collection.

Rectal Swab Collection

A different swab was inserted through the anal sphincter and a sample was taken.

Both the vaginal swab and the rectal swab were placed in selective broth medium and transported to the laboratory.

Procedure for Processing the Clinical Specimen for culture of GBS

1. The swab is inoculated into the Todd-Hewitt broth supplemented with Gentamycin-8 micrograms/ml and Nalidixic Acid-15 micrograms/ml.
2. Inoculated Todd-Hewitt broth was incubated for 24 hours at $37^{O}C$ in ambient air
3. The samples were then transferred to 5% Sheep's Blood Agar and

incubated for a further 24 hours at 37^{O}C.

4. Plates were then examined for growth of Group B Streptococci. If no growth was found, the plates were incubated for an additional day and re-examined for growth of the organism. If no growth was found on the second examination, the plates were declared as negative.

Demonstration of Group B *Streptococci*.

GBS – appearance in 5% sheep blood agar

The colony is usually gray, soft, shiny, convex, moist, regular and about 1 mm in diameter and surrounded by a small hazy zone of beta haemolysis.

Gram stain

Gram stain of pinpoint colonies is done to demonstrate the presence of Gram positive cocci arranged in short chains.

Confirmation of GBS

GBS Growth was confirmed by CAMP Test.

Antibiotic sensitivity

The isolates were tested for antibiotic sensitivity by disc diffusion method of Kirby-Bauer. Antibiotics tested were Pencillin, Ampicillin,

Erythromycin. Tetracycline, Chloramphenicol, and Vancomycin.

Post-Natal Follow-up

All mothers in the study were followed up during the period of admission, and by outpatient clinic visits after discharge, for a period of 45 days for fever, urinary tract infection, and vaginal discharge.

Neonates were followed up during the period of admission, and by outpatient clinic visits after discharge, for a period of 90 days after delivery for late onset infections such as meningitis, sepsis and pneumonia.

RESULTS AND ANALYSIS

In our study samples were taken from One hundred fifty women who were admitted to the labour room in Govt. Rajaji Hospital from July 2020 to June 2021. Two swabs were taken as one from the lower one-third of vagina and another from the anorectal region with a sterile cotton swab, before pelvic examination.

TABLE – 1

AGE VS SWAB POSITIVITY

Age	Swab Positive	Swab Negative	Total
< 20	3	15	18
21 - 25	11	83	94
26 - 30	3	18	21
> 30	1	16	17
Total	18	132	150

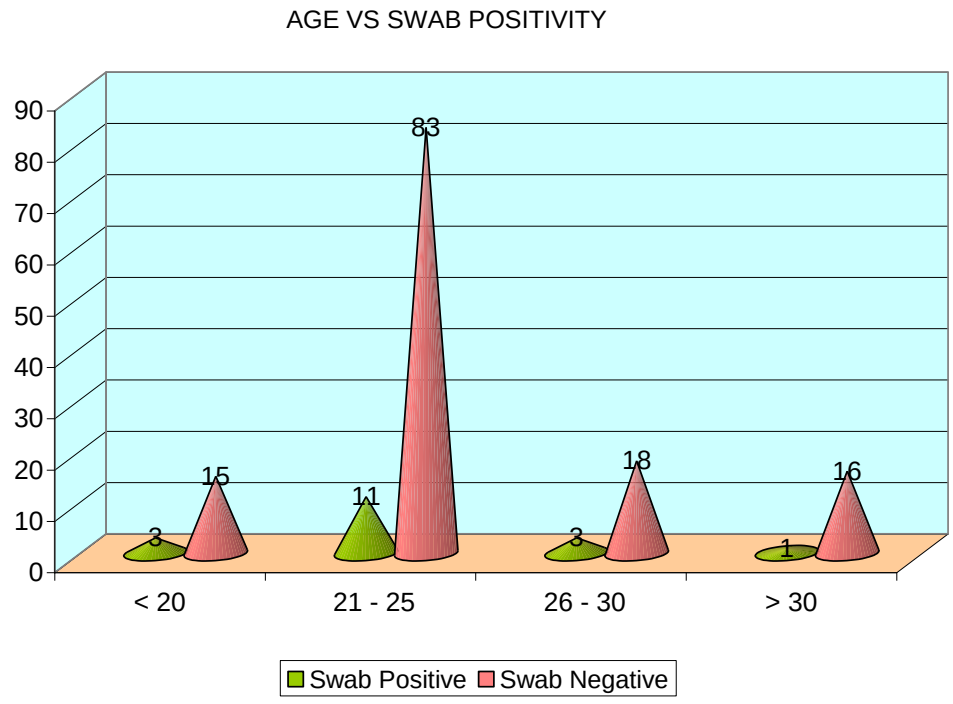


TABLE – 2

OBSTETRIC CODE VS SWAB POSITIVITY

obstetrical code	Swab Positive	Swab Negative	Total
Primi	12	63	75
Multi	5	66	71
Grand multi	1	3	4
Total	18	132	150

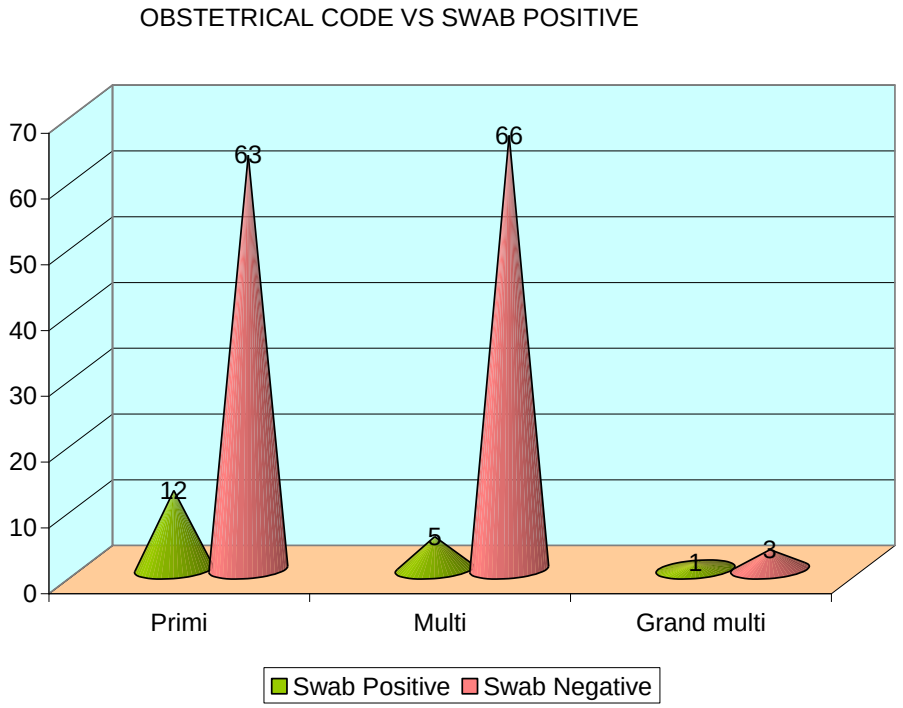


TABLE – 3

SOCIO ECONOMIC CLASS VS SWAB POSITIVITY

Socio economic class	Swab Positive	Swab Negative	Total
I	0	2	2
II	2	17	19
III	3	24	27
IV	7	33	40
V	6	56	62
Total	18	132	150

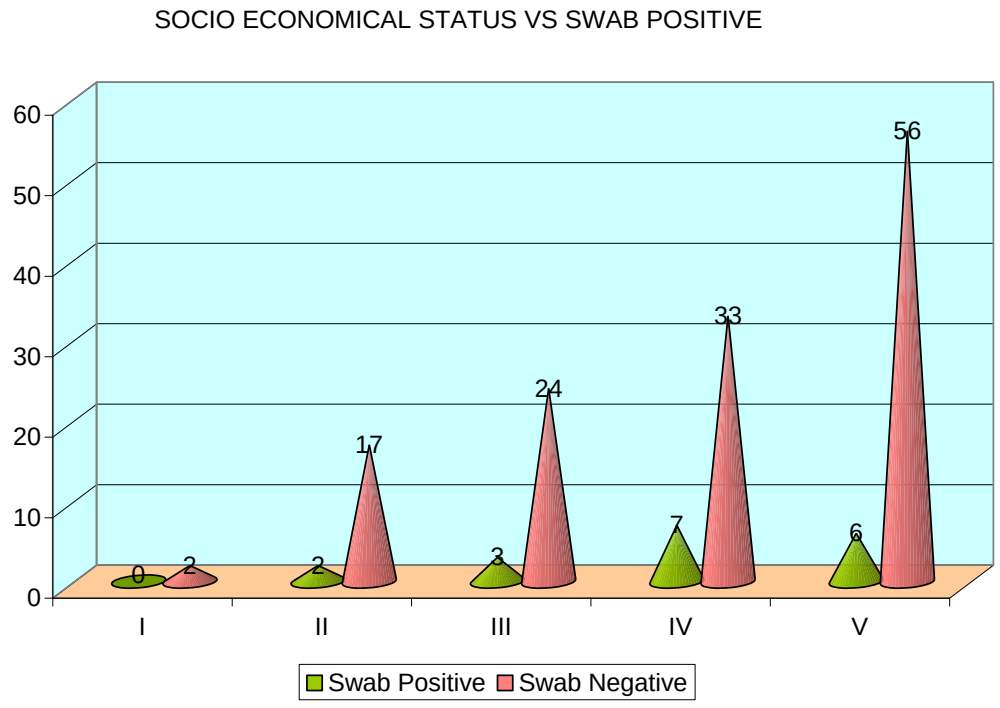


TABLE – 4

BMI VS SWAB POSITIVITY

BMI	Positive	Negative	Total
< 18.5	2	5	7
18.6 - 24.9	5	65	70
25.0 - 29.9	6	54	60
> 30	5	8	13
Total	18	132	150

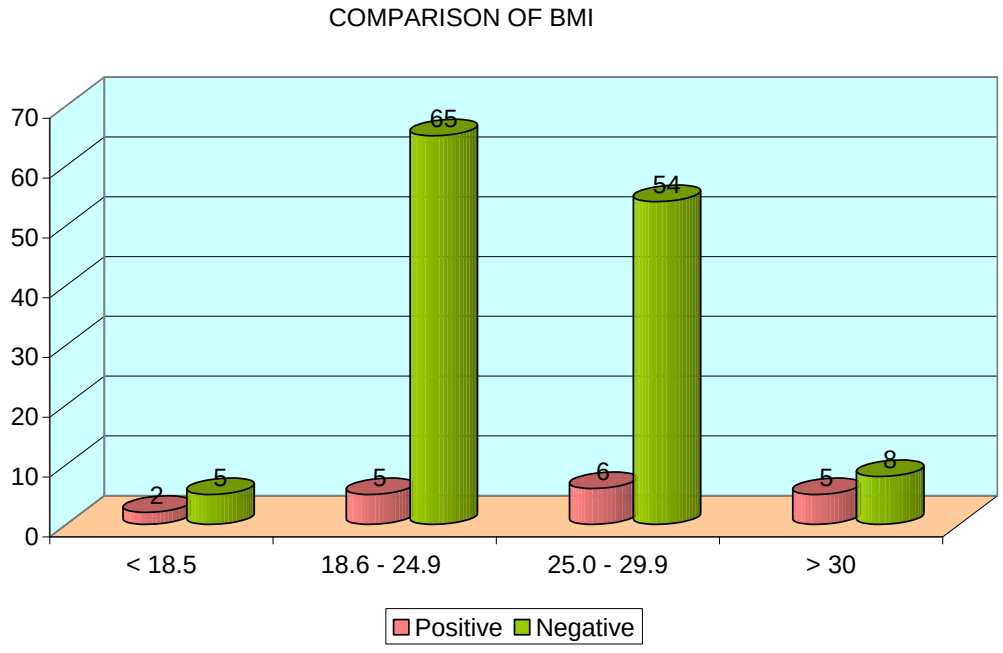


TABLE – 5

COMORBIDITIES VS SWAB POSITIVITY

Comorbidities	Positive	Negative	Total
HTN	2	30	32
DM	6	15	21
Heart disease	1	14	15
Obesity	11	64	75
Seizure disorder	1	7	8
Hypothyroidism	5	32	37

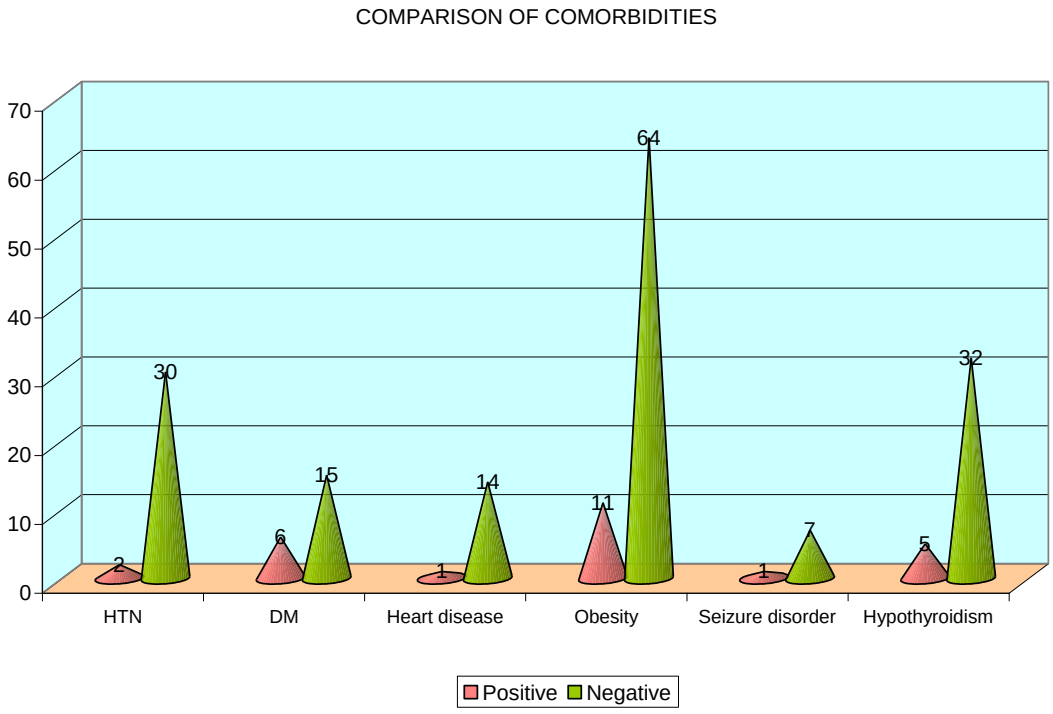


TABLE – 6

GESTATIONAL AGE VS SWAB POSITIVITY

GA at onset of labour	Swab Positive	Swab Negative	Total
35	7	5	12
36	5	13	18
Term	4	104	108
Post term	2	10	12
Total	18	132	150

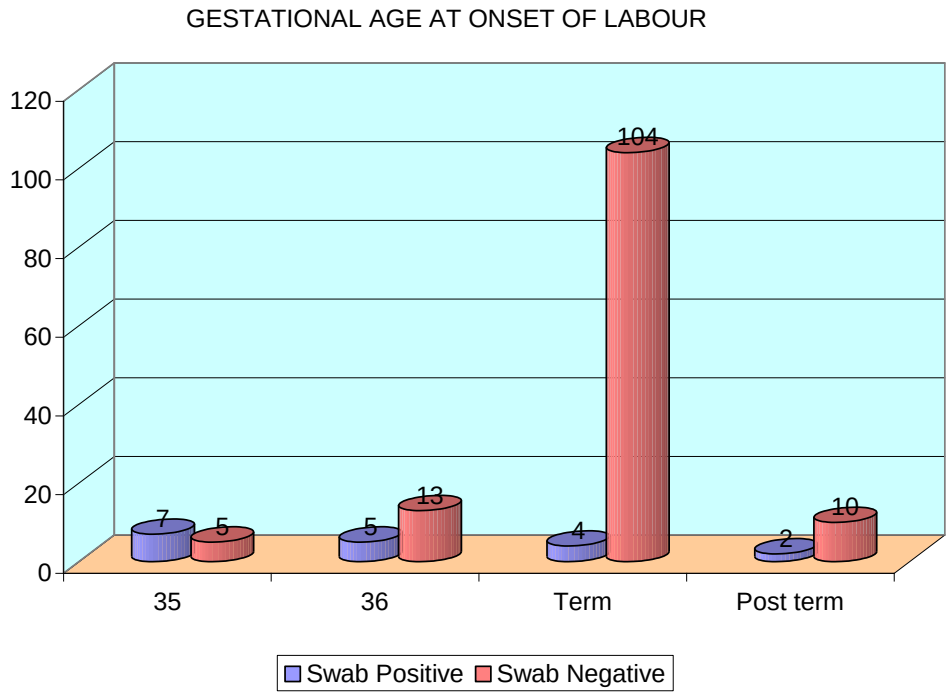


TABLE – 7

PROM VS SWAB POSITIVITY

PROM	Swab Positive	Swab Negative	Total
Yes	14	24	38
No	4	108	112
Total	18	132	150

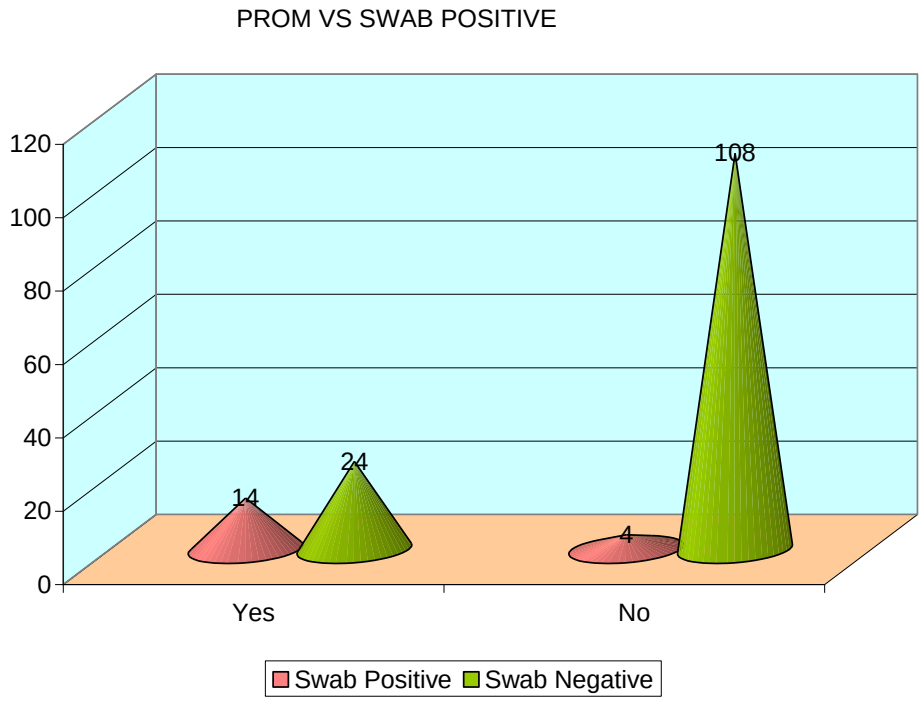


TABLE – 8

GENDER OF BABY VS SWAB POSITIVITY

Gender of Baby	Swab Positive	Swab Negative	Total
Male	13	74	87
Female	5	58	63
Total	18	132	150

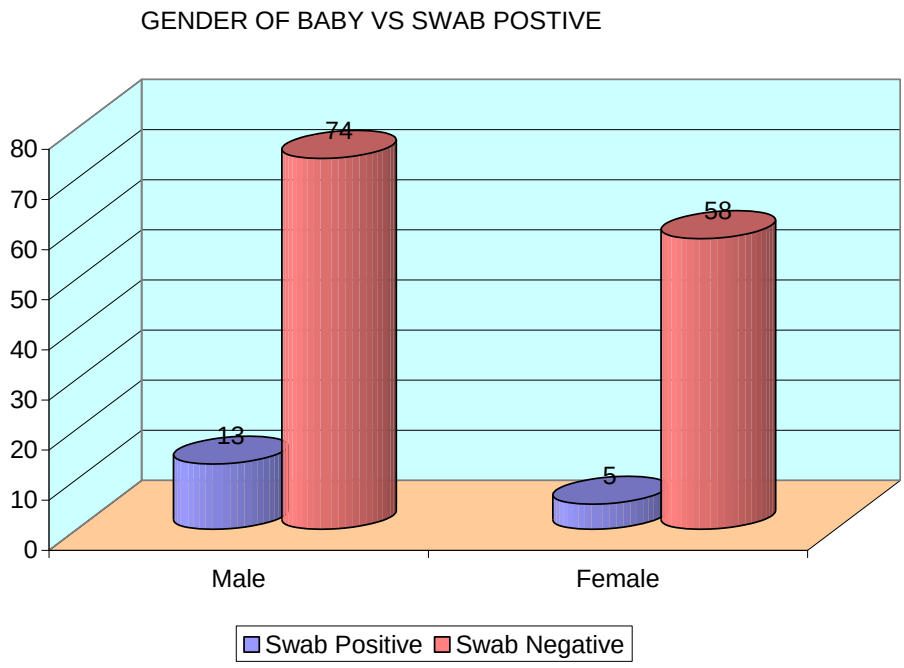


TABLE – 9

WEIGHT OF BABY VS SWAB POSITIVITY

Weight of Baby	Swab Positive	Swab Negative	Total
1.5 - 2.0	8	2	10
2.0 - 2.50	7	16	23
2.51 - 3.00	2	73	75
> 3.00	1	41	42
Total	18	132	150

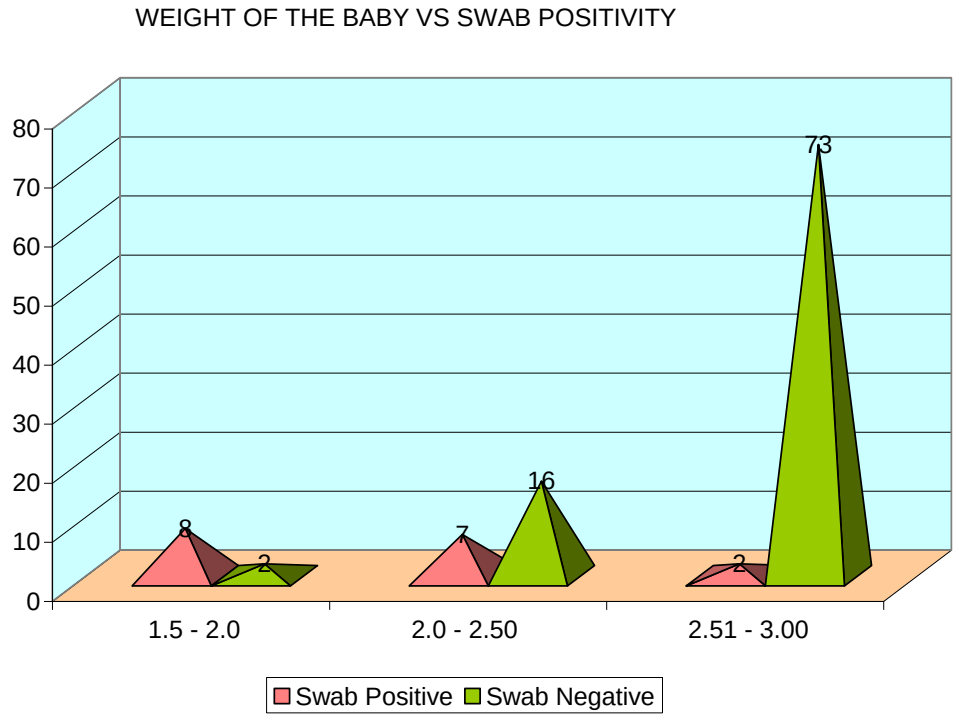


TABLE – 10

REPORT SWAB FOR GBS

Report swab for GBS	Positive	Negative	Total
Vaginal	14	135	149
Rectum	2	147	149
Both (V & R)	2	0	2
Total	18	282	300

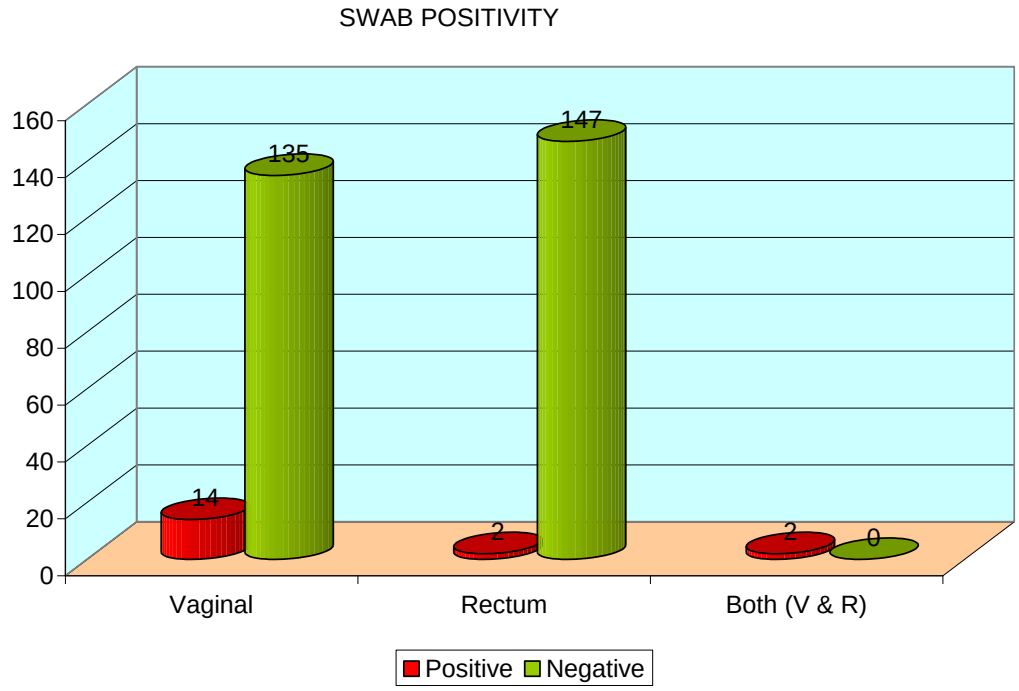


TABLE – 11

POST PARTUM COMPLICATIONS VS SWAB POSITIVITY

Post Partum Complications	Positive	Negative	Total
Fever	8	16	24
UTI	4	20	24
Lochia foul smelling	1	0	1
Uterine subinvolution	2	3	5
Nil	3	93	96
Total	18	132	150

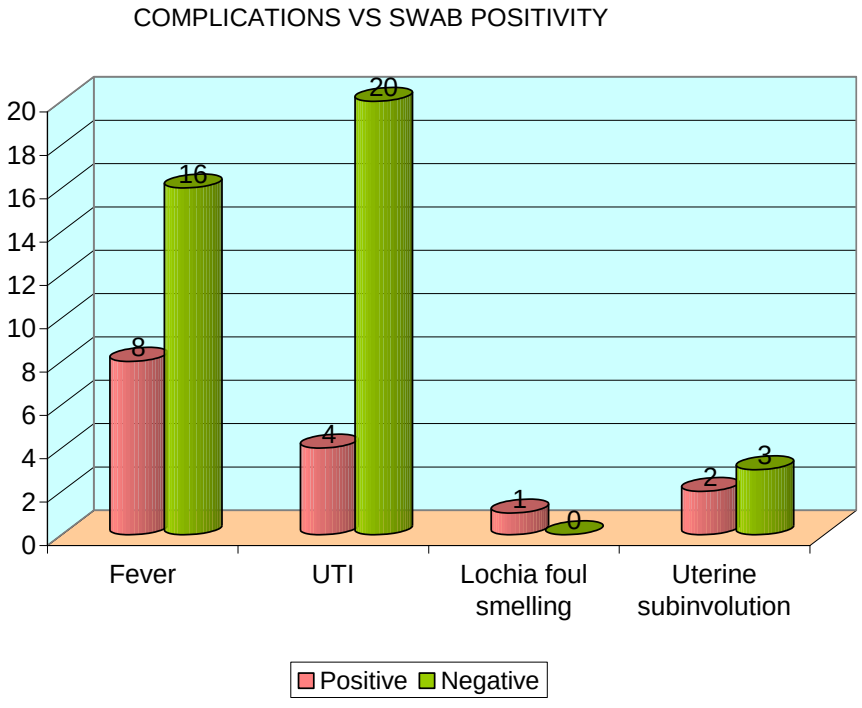


TABLE – 12

ADVERSE PREGNANCY OUTCOME VS SWAB POSITIVITY

Adverse pregnance outcome	Positive	Negative	Total
Prev. h/o preterm labour	8	13	21
Instrumental delivery	4	8	12
PROM	14	24	38
Preterm labour	12	18	30
Prolonged labour	2	2	4
Intrapartum sepsis	1	0	1

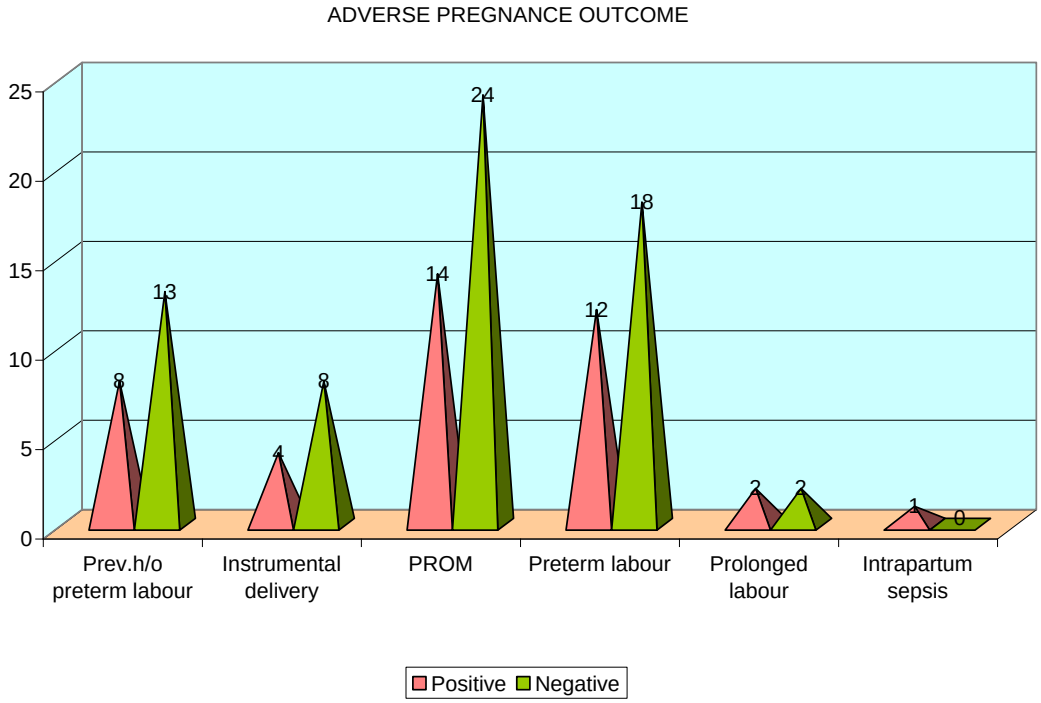


TABLE – 13

COMPARISON OF APGAR

Apgar	1 min		5 min	
	Positive	Negative	Positive	Negative
2 to 4	3	3	2	3
5 to 6	4	5	3	6
> 7	11	124	13	123
Total	18	132	18	132

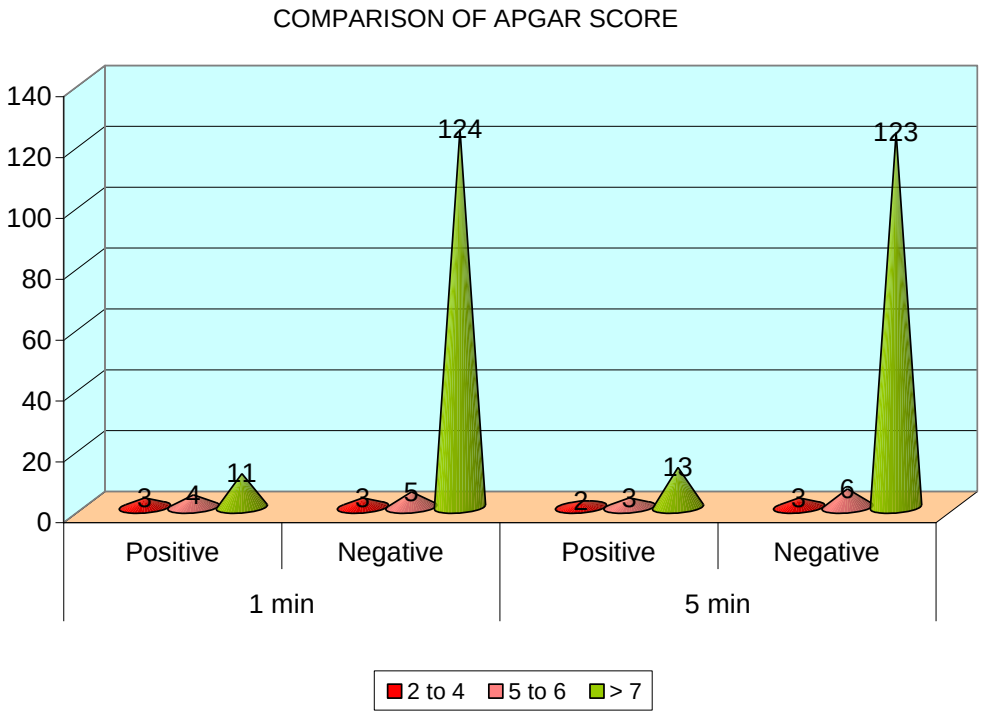


TABLE – 14

NICU ADMISSION

NICU admission	Positive	Negative	Total
Yes	15	23	38
No	3	109	112
Total	18	132	150
P VALUE	< 0.001 Significant		

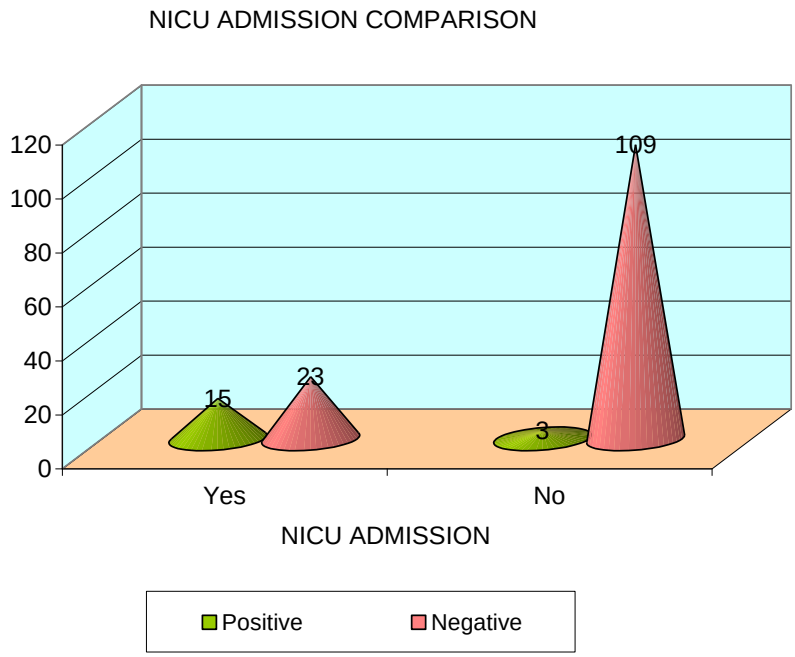
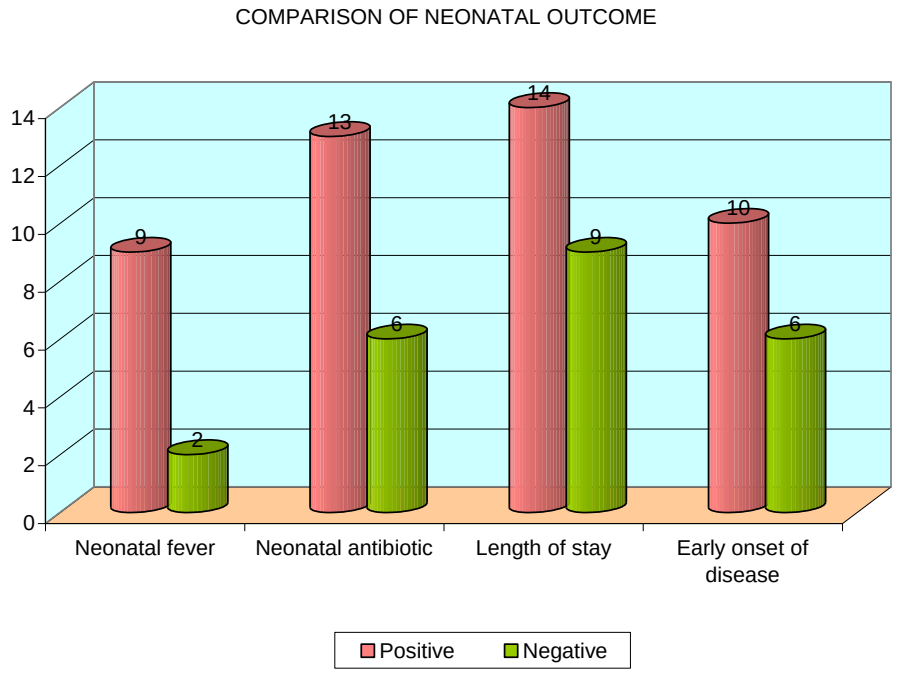


TABLE – 15

NICU ADMISSION VS NEONATAL OUTCOME

Neonatal outcome	Positive	Negative	Total	p value
Neonatal fever	9	2	11	0.001 Sig
Neonatal antibiotic	13	6	19	<0.001 Sig
Length of stay	14	9	23	0.003 sig
Early onset of disease	10	6	16	0.032 sig



DISCUSSION

The Group B *Streptococci* (GBS) are known to cause a wide variety of infections in adults, but clinical interest in these bacteria mainly relates to their ability to cause serious neonatal illness, especially meningitis and sepsis. In developed countries these organisms are the leading cause of neonatal sepsis and meningitis with a case fatality rate of 40 to 80%. [85]. However, in developing countries like India, the problem has not beenadequately studied and there are only a few reports available [86,87,88,89]. In the present study most of the females were from 21 to 25 years of age group.

In our study of 150 women, we have found a prevalence of GBS colonization in mothers to be 1.2%. This is comparable with other studies conducted in India, such as those by Kulkarni et al, Mhaskarrita et al, and Singh et al, which have found a prevalence ranging from 1.1% to 2.2%. [42, 43, 44, 45, 46, 47]

Studies conducted in other countries, however have prevalance rates to be higher, ranging from 6.4% to 35.0% [28, 29, 30]. The reason for thisdifference between studies conducted other countries is not clear.

The National Screening Committee does not recommend universal bacteriological screening for GBS.

Their view is that there is no clear evidence to show that testing for GBS routinely would do more good than harm. The reasons quoted are:

- Many women carry the bacteria and, in the majority of cases, their babies are born safely and without developing an infection.
- Screening women late in pregnancy cannot accurately predict which babies will develop GBS infection.
- No screening test is entirely accurate. Between 17% and 25% of women who have a positive swab at 35–37 weeks of gestation will be GBS negative at delivery. Between 5% and 7% of women who are GBS negative at 35–37 weeks of gestation will be GBS positive at delivery.
- In addition, many of the babies who are severely affected from GBS infection are born prematurely, before the suggested time for screening.
- Giving all carriers of GBS IAP would mean that a very large number of women would receive treatment they do not need; this may increase adverse outcomes to mother and baby.

If GBS was detected in a previous pregnancy, irrespective of carrier status this pregnancy, explain to women that the likelihood of maternal GBS carriage in this pregnancy is 50%. Discuss the options of IAP, or bacteriological testing in late pregnancy and then offer of IAP if still positive.

If performed, bacteriological testing should ideally be carried out at 35–37 weeks of gestation or 3–5 weeks prior to the anticipated delivery date, e.g. 32–34 weeks of gestation for women with twins.

IAP should be offered to women with a previous baby with early- or late-onset GBS disease.

Women with GBS urinary tract infection (growth of greater than 10^5 cfu/ml) during pregnancy should receive appropriate treatment at the time of diagnosis as well as IAP

Antibiotic prophylaxis specific for GBS is not required for women undergoing planned caesarean section in the absence of labour and with intact membranes.

Women who are known GBS carriers should be offered immediate IAP and induction of labour as soon as reasonably possible.

In women where the carrier status is negative or unknown, offer induction of labour immediately or expectant management up to 24 hours. Beyond 24 hours, induction of labour is appropriate.

Women who are pyrexial (38°C or greater) in labour should be offered a broad-spectrum antibiotic regimen which should cover GBS in line with local microbiology sensitivities.

Bacteriological testing for GBS carriage is not recommended for women with preterm rupture of membranes. IAP should be given once labour is confirmed or induced irrespective of GBS status.

For those with evidence of colonisation in the current pregnancy or in previous pregnancies, the perinatal risks associated with preterm delivery at less than 34 $^{+0}$ weeks of gestation are likely to outweigh the risk of perinatal infection. For those at more than 34 $^{+0}$ weeks of gestation it may be beneficial to expedite delivery if a woman is a known GBS carrier.

Women with known GBS colonisation who decline IAP should be advised that the baby should be very closely monitored for 12 hours after birth, and discouraged from seeking very early discharge from the maternity hospital

There is no evidence that intrapartum vaginal cleansing will reduce the risk of neonatal GBS disease.

In adult women, GBS carriage in the genital tract, perineal skin and gastrointestinal tract is of great importance in view of its significance in GBS neonatal infection, whether asymptomatic mucous membrane colonization or symptomatic invasive infection (early onset septicemia, meningitis etc.)

There is no evidence that treating GBS colonisation before labour is beneficial. Therefore, a prelabour-positive GBS culture does not change management in pregnancies with a gestation of less than 34^{+0} weeks because the high morbidity associated with early preterm birth means that early delivery is not indicated unless there are overt signs of infection. The risk of GBS infection is higher with preterm delivery and the mortality rate from infection is increased (20–30% versus 2–3% at term)37, 38 and this therefore justifies IAP in all cases of preterm labour.

The NICE guideline *Preterm labour and birth*47 recommends that all women with preterm prelabour rupture of the membranes should be offered oral erythromycin 250 mg, 4 times a day for a maximum of 10 days or until the woman is in established labour (whichever is sooner). Oral penicillin should be considered for the same duration in women who cannot tolerate erythromycin or in whom erythromycin is contraindicated.

A large multicentre randomised controlled trial (RCT) of elective delivery at 34–36 weeks of gestation for preterm spontaneous rupture of membranes versus conservative management48 has demonstrated no significant differences in neonatal disease, morbidity or mortality. As a result, there is no indication to

prefer one form of management over the other at this gestational age although IAP should be given once labour starts. There may be disadvantages with conservative management beyond 34^{+0} weeks of gestation in the presence of known GBS colonisation and in this group, early intervention may be preferable.49

Considerable work has been done to determine the complications associated with GBS carriage. A number of factors are involved, like maternal urinary tract infection, premature or prolonged rupture of membranes,premature delivery, and peak intrapartum fever greater than $37.5^{O}C$.

Some workers give weightage to lack of type specific antibodies by neonates due to failure of transplacental transfer of these antibodies. This is responsible for GBS colonization, or in turn GBS disease in the newborn.

For better isolation of GBS from clinical material the use of transport medium has revealed the critical consideration. Several works tried different methods of transportation of clinical specimens from patients to the laboratory. In the present study we used Todd-Hewitt's broth as selective broth, and the samples were then cultured in Sheep Blood Agar.

The evidence suggests that water birth is not contraindicated for GBS-positive women who have been offered the appropriate IAP.41-43

The evidence does not suggest that using polymerase chain reaction technology for near-patient testing is feasible in UK maternity labour ward settings.40 The technology for near-patient testing continues to improve and it is possible that this may confer benefits in the future. An ongoing cluster randomised trial is testing whether the use of near-patient testing in labour can reduce the use of IAP in women who present with clinical risk factors who would be eligible for IAP.

SEROTYPES AND FACTORS AFFECTING VIRULENCE

The Group B β-hemolytic *Streptococcus* (*S. agalactiae*) contains a Lancefield-grouping antigen, a type-specific cell-surface polysaccharide and protein antigens.

The prevalence of the various Group B capsular serotypes varies over time and may differ from place to place. Prior to the 1990s, most Group B *Streptococcal* disease was caused by serotypes Ia, Ib, II, III, and V; serotypes IV and VI through VIII were relatively uncommon. During the early to mid-1990s, serotype V stains began to emerge, with the percentage of isolates in this group increasing from 2.6% in 1992 to 20% in 1994 [90,91]. Studies conducted in the U.S. and abroad indicate that serotypes Ia, Ib, II, III and V now predominate among vaginal isolates and clinical isolates from patients [92,94,94]. Recently, serotypes VI and VII have appeared as the predominant serotypes in Japan [95].

Neonatal early-onset disease due to serotype VIII has also been reported in Japan [96]. Type II strains of GBS account for 60% of isolates from cases of neonates sepsis and over 80% of isolates from infants with meningitis, suggesting that this GBS serotype possesses enhanced virulence [97]. The type IIIcapsular polysaccharide is composed of a repeating structural backbone consisting of galactose, glucose, and N-acetyl-neuraminic acid (sialic acid). Thepresence of this molecule on the surface of the organism inhibits activation of

the alternative complement cascade and prevents phagocytosis. Removal of sialic acid residues with neuraminidase leads to complement activation phagocytosis, and intracellular killing of the organisms and diminished virulence on intravenous challenge in a rat model [98,99].

GBS also produce a variety of other potential virulence determinants. Like the Group A *Streptococci*,GBS also produce C5a peptidase. C5a is a complement component cleavage product that is produced by alveolar epithelial cells, acts as an attractant for inflammatory cells, and is involved in the process of pulmonary inflammation [100]. The C5a peptidase produced by the *Streptococci* cleaves C5a at the C-terminus, thereby interfering with C5-mediated neutrophil chemotaxis [101]. New information indicates that this peptidase also binds to fibronectin [102].

Group B β-hemolytic *Streptococci* are a major cause of disease in the neonatal and perinatal periods. Women become colonize with the organism in the vagina and the rectum, and vaginal colonization is found in 10-35% of pregnant women; up to 60% of the colonized women will carry the organism intermittently [103,104]. Colonization of the vagina may actively reflect contamination from the rectum, with the gastrointestinal tract being the principal reservoir of the organism.

COMPLICATIONS

GBS is associated with a spectrum of maternal and fetal infections ranging from asymptomatic colonization to sepsis. *Streptococcus* agalactiae has been implicated in adverse pregnancy out comes, including preterm labor, prematurely ruptured membranes, clinical and subclinical chorioamnionitis, and fetal and neonatal infections. It is associated with about 20% of postpartum endometritis, 25% of bactremias following caesarean section, and 25-30% of cases of asymptomatic bacteriuria during and after pregnancy, they are also associated with a variety of infections in nonpregnant adults [105].

In view of this increased risk of EOGBS, IAP should be offered in the presence of maternal pyrexia. Since a raised temperature can indicate chorioamnionitis, a broad-spectrum antibiotic, rather than penicillin G, is recommended in this situation. The antibiotic regimen of choice will depend on local microbiology guidance; intravenous amoxicillin 2 g every 6 hours (or intravenous cefuroxime 1.5 g every 6 hours in women with a nonanaphylactic reaction to penicillin) is acceptable in this context.35

If known to be colonised with GBS, women should be offered immediate IAP because of the increased risk of EOGBS disease with prolonged rupture of membranes.32

As recommended in NICE clinical guideline 7033 women should be offered induction of labour immediately or up to 24 hours after spontaneous rupture of membranes with unknown carrier status.32

Women who are known GBS carriers who are to be delivered by caesarean section after spontaneous rupture of membranes should be offered IAP and delivered by category 2 or 3 caesarean depending on other clinical findings.31

There is evidence that membrane sweeping does not increase the risk of EOGBS disease

Mothers who have had a previous baby affected by early- or late-onset GBS are at increased chance of another affected baby compared with women of similar carrier status who have not had an affected baby. The reasons for this increased risk are not clear but may indicate persistence of carriage of a virulent strain of GBS or a deficient immune response.24-26 In view of this potentially increased risk, and the possibility of false-negative antenatal testing, we recommend giving IAP in such cases and maternal bacteriological tests are not recommended.

Assuming that approximately 50% of women will be recurrent carriers, the risk of EOGBS disease should be approximately 2 to 2.5 times that quoted for the total population.17-21 The risk of EOGBS disease in the baby in this circumstance is likely to be around 1 in 700 to 1 in 800.3 At this risk level, some women would choose IAP and others would not. Bacteriological testing in this circumstance would help to refine the risk. A positive bacteriological test in this circumstance would indicate a risk of 1 in 400, but the risk would be 1 in 5000 if the mother is GBS negative. A significant number of mothers may therefore choose to avoid IAP if they test negative.

If bacteriological tests for GBS are to be performed in pregnancy they should ideally be performed at 35–37 weeks of gestation22 in order to determine carriage status close to delivery. There is no evidence to support the practice of varying the timing of screening. However, in women where preterm delivery is anticipated, earlier testing is justified.

In our study, Pregnant mothers who were obese were more likely to colonized with GBS in their anogenital areas compared to mothers who were nonobese. The underlying aetiology of the association between GBS colonization and obesity that we identified is not clear; little is known about the biologic mechanism of colonization, which consequently limits its interpretation. However, it may be related to changes in the gastrointestinal microbial ecology with obesity. Animal and human studies demonstrate a shift towards increased Firmicutes (the phylum to which GBS belongs) and decreased *Bacteroides* with obesity as quoted by Ley et al. [14]. These shifts reflect increased energy-reabsorbing potential of different ratios of Firmicutes and *Bacteroides*, especially in the digestion of fatty acids and dietary polysaccharides. In addition, probably poor perineal hygiene may also contribute to GBS colonization, whereby the participant's size may prevent them from thorough anogenital cleaning as suggested by Steenwinkel et al., 2008 [6].

This finding of GBS being associated to obesity was similar to Shah et al., 2011 [15], who conducted a retrospective double cohort study at San Francisco General Hospital,

California, between 2007 and 1997 and found out that obesity was one of the factors that are significantly associated with GBS rectovaginal colonization.

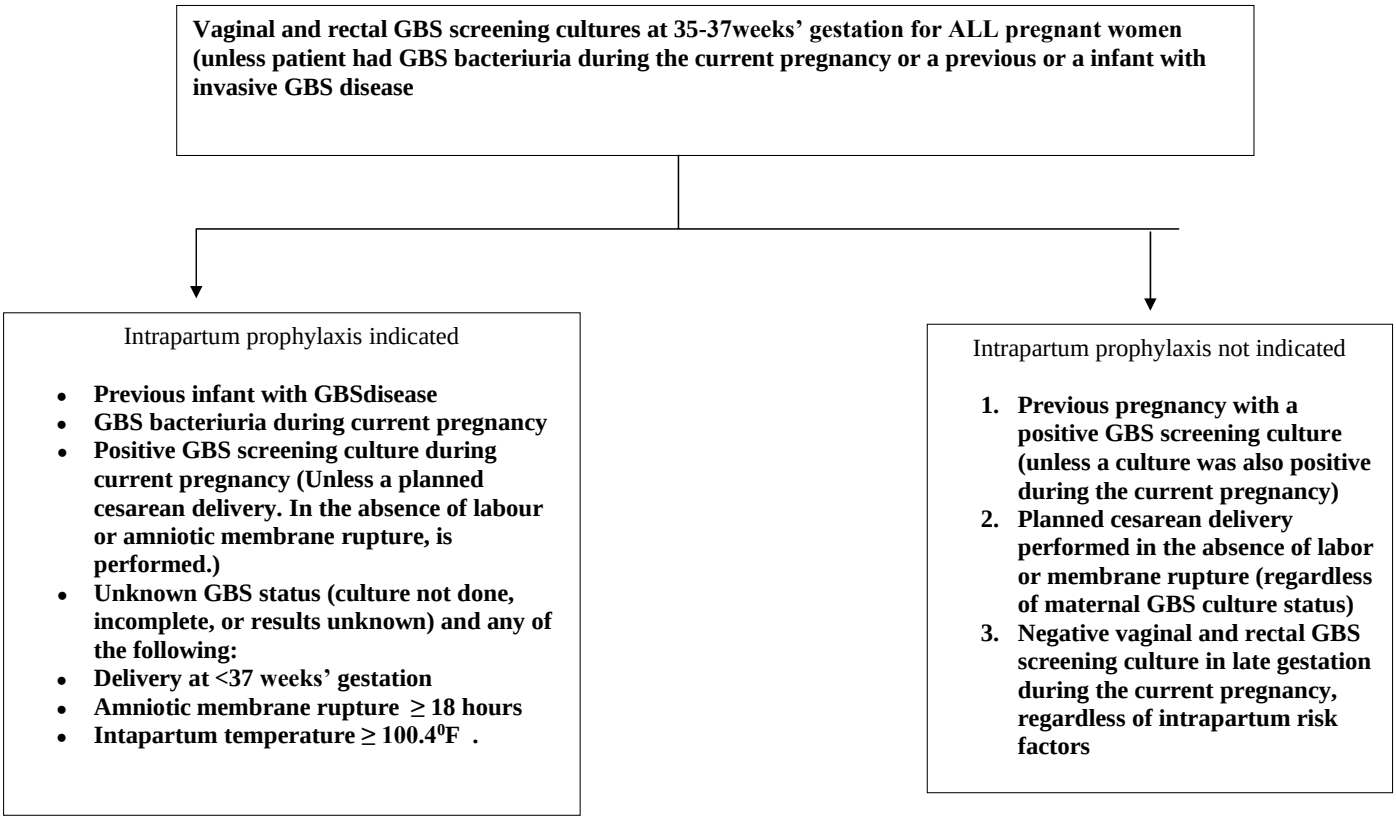
NEONATAL INFECTIONS

Early-Onset disease occurs with an incidence of 0.7 in 1.000 to 3.7 in 1,000 live births and is associated with in utero or perinatal organism acquisition 106. The organism is acquired either by ascending infection in uterobefore delivery, through ruptured fetal membranes, or during passage through a birth canal that is colonized with GBS. Although a substantial proportion of these infants (approximately 50%) will be colonized with GBS, only 1-2% of them become infected [107]. Onset of disease occurs during the first 5 days of life;in more than half the cases, infants become ill within the first 12 to 20 hours after birth [108]. The disease spectrum includes bacteremia, pneumonia,

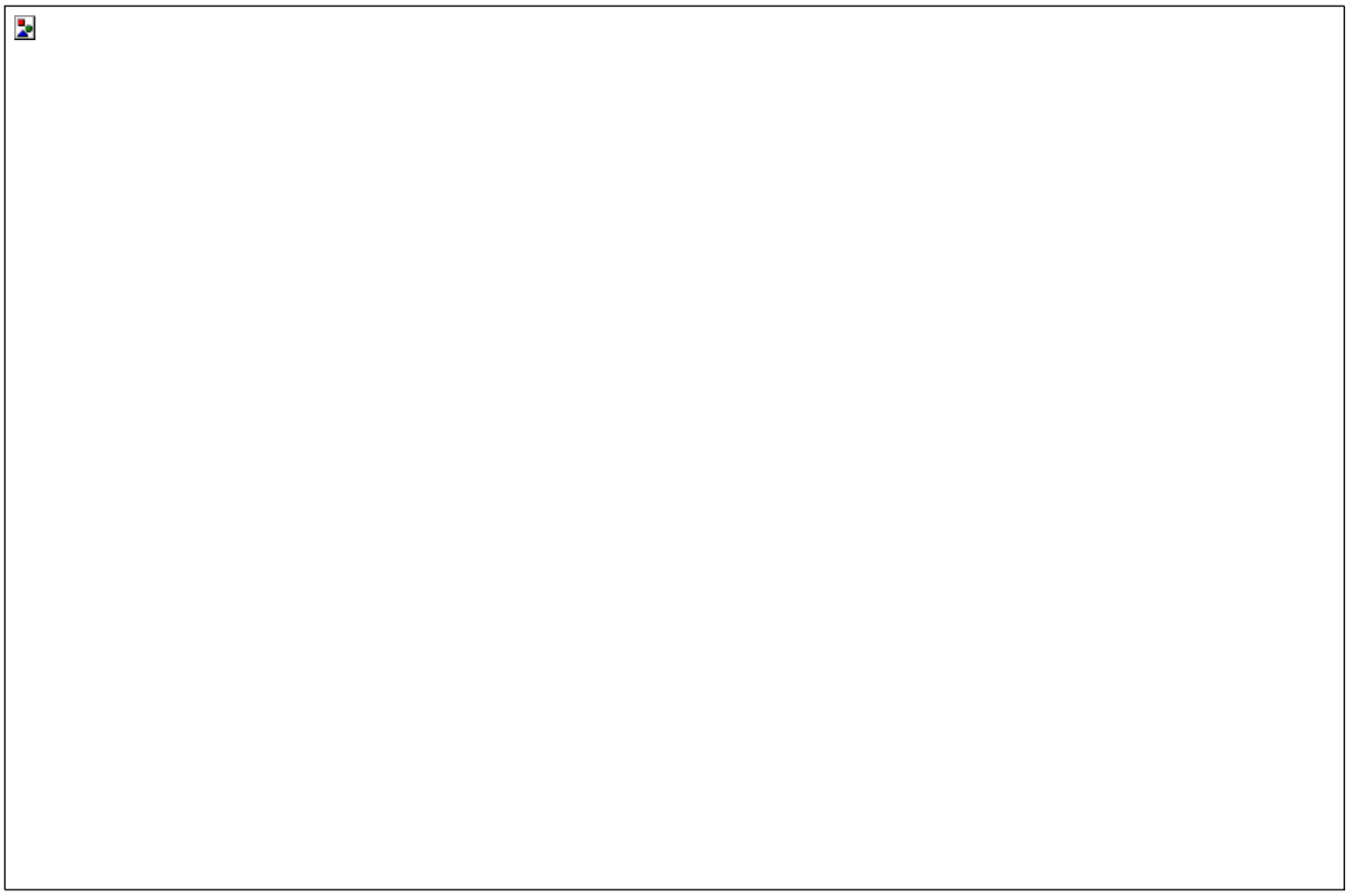
meningitias, septic shock, and neutropenia. Although more than 50% of cases occur in full-term infants, a higher attack rate and greater morbidity are associated with preterm infants. Mortality owing to early – onset disease in full-term infants ranges from 2% to 8%; higher mortality rates are seen in premature infants and are inversely proportional to the birth weight of the neonate [109]. Maternal factors that increase the risk for early –onset infection of the neonate include premature labour, prolonged rupture of the fetal membranes, postpartum bacteremia, maternal amnionitis, heavy vaginal colonization with Group B *Streptococci*, and Group B *Streptococci* bacteriuria [110,111].

Late –onset disease occurs with an incidence of 0.5 in 1,000 to 1.8 in 1,000 live births [98]. Disease becomes clinically evident 7 days to 3 months (average, 3 to 4 weeks) after birth. Whereas about half of the late –onsetinfections are acquired from the birth canal of colonized mothers, the remainingcases result from postnatal organism acquisition from the mother or other caregivers or nosocomially [112]. Bacteremia with accompanying meningitis is thepredominant clinical presentation [113]. Mortality associated with late-onset disease is about 10-15%. Up to 50% of children with late onset meningitis will have permanent neurologic complications and sequelae [114]. The distribution of Group B *Streptococci* serotypes also varies according to whether it is early- onset disease without meningitis, the serotype distribution is equally divided

among types Ic, II, and III. Among similarly infected neonates with meningitis, serotype III strains account for over 90% of the isolates. On the other hand, group B streptococcal meningitis in adults is associated primarily with serotype II organisms.



CDC GUIDELINES FOR GROUP B STREPTOCOCCUS PROPHYLAXIS



CONCLUSION

Our study revealed that GBS prevalence in AN mother attaining Madurai Rajaji medical college was 1.2%. The isolation rate of GBS was improved by selective enrichment media. The mean age in our study was found to be 24.48 and the median gestational age was 37wks.

In our study primi gravida women were more often associated with GBS colonization, though it was not statistically significant. In a study involving South Indian perception, multi gravida women were significantly more colonized than primi gravida. In another study parity was found to be unrelated to GBS carriage .Therefore further studies are needed to confirm the correlation between parity and colonization by GBS .

Women reporting from rural living area were significantly more often colonized 77.8% with GBS compared with those living in urban area 22.3% in our study.

Strong association was observed between GBS colonization and previous history of still birth and preterm labour. p value < 0.05.

Many women in the current study were recorded as having comorbidities 90%. Out of which Diabetes mellitus 21 cases and increased BMI 68 cases has

been reported to be significantly associated with GBS colonization. This has been attributed to immuno suppression.

Majority of isolates in our study were sensitive to Penicillin 80% Ampicillin 56%, Erythromycin 71% and clindamycin 33%. Most are resistant to Tetracyclin and Gentamycin. Two isolates were resistant to Erythromycin. 7 isolates were resistant to tetracycline. None of the isolates were resistant to Penicillin.

It was also found that the incidence of Preterm Labour in those mothers who were positive for GBS was 25.0%; compared to 3.5% in those mothers who were negative for GBS.

Similarly, the incidence of Premature Rupture of Membranes in those mothers who were positive for GBS was 25.0%; compared to 10.2% in those mothers who were negative for GBS.

These results indicate that there is a definitive increased risk of Pre-term Labour, Premature rupture of Membranes and Neonatal Sepsis in pregnant women who are colonized with GBS.

In our study, the prevalence of Group B *Streptococci* in the Maternal samples was found to be 1.2%. The incidence of Preterm

Labour in those mothers who were positive for Group B *Streptococcus* was 25.0%; compared to 3.5% in those mothers who were negative for Group B *Streptococcus.*

Similarly, the incidence of Premature Rupture of Membranes in those mothers who were positive for Group B *Streptococcus* was 25.0%; compared to 10.2% in those mothers who were negative for Group B *Streptococcus.*

Of 38 NICU admissions 15 (39.5%)were found to be babies of GBS positive mother, 23 (60.5%) were found to be babies of GBS negative mother. Admissions were mainly due to prematurity ,low birth weight ,suspected septicemia and respiratory distress.

Infant of mother colonized with GBS (9) (50%) were more likely to have temperature more than 38°c compared to infant of GBS negative mother(2) 8.7%.

13 babies of GBS positive mother received antibiotics while only 6 babies of GBS negative mother received antibiotics. Babies of GBS positive mother admitted at NICU had a longer hospital stay compared

with babies of GBS negative mother.

The prevalence of maternal colonization with Group B *Streptococci* in our study population is low at only 1.2 %, which is comparable with other studies conducted in India (1.2 – 2.5 %). But the reported prevalence in Western countries is higher ranging from 6.4 –35 %.

The low occurrence of Group B Streptococci maternal colonization (1.2%) shows that selective risk factor based screening and anti-biotic prophylaxis should be considered as an effective protocol for preventing neonatalmorbidity and mortality due to Group B Streptococci than a mass screening for Group B Streptococci during pregnancy.

Recommendations for future research

- Cluster randomised trial of screening for GBS carriage with the offer of IAP for carriers to investigate the benefits and harms of a bacteriological screening programme.

- Studies of the virulence of specific strains identified using genetic markers and of serological correlates of protection.
- What is the long-term prognosis and associated costs for infants who survive EOGBS disease?
- What is the safety, immunogenicity and efficacy of a GBS vaccine in pregnant women?
- Can serocorrelates of protection against GBS be defined and used to facilitate the licensure of a GBS vaccine without the need for large-scale prelicensure efficacy trials in pregnant women?

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PROFORMA

Patient particulars

S.No:

Name: Age:

Ip No: Unit:

Education:

Address:

Socioeconomic status:

D.O.Admission: D.O.Discharge:

D.O.Delivery:

Mode of delivery:

Past history:

Family history

Examinations:

General examination:

icterus: pedal edema: pallor:

Physical examination:

height : weight: BMI: Vitals :BP: PR: SP02:

Systemic examination: CVS: RS:

Per abdomen examination:

Per vaginal examination

Diagnosis:

UTI IN CURRENT PREGNANCY :

SWAB :

VAGINAL INTROITUS TO ANUS:

TIME OF RUPTURE OF MEMBRANES :

DURATION OF LABOUR :

MODE OF DELIVERY :

SEX : WEIGHT OF BABY :

REPORT OF VAGINAL SWAB CULTURE :

REPORT OF RECTAL SWAB CULTURE:

POST PARTTUM COMPLICATIONS:

1) FEVER 2) UTI 3) FOUR SMELLING LOCHI