# TO STUDY THE OUTCOME OF NEONATES BORN TO MOTHERS WITH PROM WITH SPECIAL REFERENCE TO EARLY ONEST SEPTICAEMIA AND ITS CORRELATION WITH VAGINAL PATHOGENS WITH PROM

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# TITLE OF THE TOPIC

## "To study the outcome of neonates born to mothers with PROM with special reference to Early onset Septicaemia and its correlation with vaginal pathogens in mothers with PROM "

(MD PEDIATRICS)

# LIST OF ABBREVIATIONS

| | | |
|---|---|---|
| PROM | : | Premature rupture of membranes |
| PPROM | : | Preterm premature rupture of membranes |
| EOS | : | Early onset sepsis |
| PMN | : | Polymorphonuclear neutrophil |
| RDS | : | Respiratory distress syndrome |
| NNPD | : | National neonatal perinatal data |
| DIC | : | Disseminated intravascular coagulation |
| CRP | : | C-reactive protein |
| CSF | : | Cerebro spinal fluid |
| NBT | : | Nitroblue tetrzolium |
| LAP | : | Leucocyte alkaline phosphatase |
| µESR | : | Micro-erythrocyte sedimentation rate |
| BCS | : | Buffy coat smear |
| NICU | : | Neonatal intensive care unit |
| UTI | : | Urinary tract infection |
| CFT | : | Capillary filling time |
| IVIG | : | Intravenous immunoglobulin |
| G-CSF | : | Granulocyte colony stimulating factors |
| GM-CSF | : | Granulocyte monocyte-colony stimulating factor |
| LSCS | : | Lower segment caesarean section |
| WBC | : | White blood count |

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# INTRODUCTION:

Premature rupture of membranes (PROM) refers to the rupture of fetal membranes prior to the onset of labor regardless of gestation. It is subdivided into the preterm premature rupture of membranes (PPROM) which is rupture d before 37 weeks of gestation and term premature rupture of membranes (TPROM), which is ruptured after 37 weeks of gestation. Prolonged rupture of membranes is any rupture of membranes that persists for more than 24 hours and prior to the onset of labor[1].

One of the most frequent issues in obstetrics, PROM complicates 8% to Approximately 10% of term pregnancies and 1% of all preterm pregnancies1. Neonatal sepsis is a clinical syndrome characterized by signs and symptoms of infection with or without accompanying bacteremia in the first month of life[(2,3).] It encompasses various systemic infections of the newborn such as septicaemia, meningitis, pneumonia, arthritis, osteomyelitis, and urinary tract infection. Neonatal sepsis is diagnosed when generalized systemic features of sepsis are associated with the pure growth of bacteria from one or more sites. According to pooled hospital data based on neonatal perinatal data (NNPD), the incidence of neonatal sepsis is around 30 per 1000 live births[4]. Neonatal sepsis can be divided into two main subtypes depending on whether the onset is during the first 72 hours of life or later. Early onset sepsis and Late-onset sepsis.

Neonatal sepsis contributing to neonatal mortality rate is 13.6%[3]. The risks of new born morbidity and death are greatly influenced by Latent period and gestation lengths in PROM.

Globally, 3 million newborns die every year within the first week of life[4,5]. 99% of these deaths occur in low- and middle-income countries with sepsis and pneumonia accounting for a quarter of neonatal death. Although the incidence of sepsis in developing countries is thought to be as high as 170 per 1000 live births, the diagnosis of sepsis is often missed due to non-specific or absent signs and symptoms. Frequently, new-born's are discharged from the within 24 hours after delivery, possibly leading to a late diagnosis of an early A significant cause of infant fatalities is early-onset neonatal sepsis. There's a Demand in order to decrease infant morbidity and mortality by early identification of sepsis. The danger of infection and pregnancy complications are the mother's main PROM side effects. This risk rises as the membrane rupture lasts longer. Due complications for new-born's include fetal discomfort, sepsis, premature birth, and altered pulmonary development4. For patients with PPROM, the most likely outcome is preterm delivery within one week, with its associated morbidity and mortality risks such as respiratory distress, necrotizing enterocolitis (NEC), intra-ventricular haemorrhage, and sepsis5. In the absence of early specific and sensitive diagnostic tools for neonatal sepsis, management of infants born to mothers with PROM proves to be a dilemma, especially for asymptomatic neonates at birth[7]. Early onset Usually manifesting within the first 72 hours of life, sepsis (EOS), It results from Organisms common in the genital tract or the labour and delivery room. Most commonly caused due to gram-negative organisms especially E. coli, Klebsiella, and Enterobacter species.

The majority of neonates with early onset sepsis manifest as respiratory distress, due to intrauterine pneumonia. There is an early-onset bacterial infection either as a result of ascending infection following membrane rupture or throughout when the infant passes through an infected birth canal or during recovery in the delivery room.

The new born is deemed to be infected when at least three of the following risk indicators are present.:

1. VLBW or premature child.
2. Mother having fever during labour or two weeks after delivery.
3. A prolonged membrane rupture that lasts more than 18 hours.
4. Liquor amnii that smell foul or are soiled with meconium.
5. Difficult labour and a complicated instrumental delivery.
6. Asphyxia at birth and difficult resuscitation.
7. A single filthy examination or more than three while in labor[4].

EOS in new-borns born to PROM mothers caused by organisms prevalent in the genital tract of mothers with PROM or in the labor room and maternity operation theatre[2].

Nearly 90% of neonates with early-onset group B streptococcus have signs of infection within 12 hours of delivery[(11,12)]. Early stages of infection may be subtle. A baby may not be feeding well or excessively sleepy. Tachypnoea, Apnea or other respiratory distress is the most common presenting sign[(13,14)] . Recognition of risk factors for the acquisition of bacteria led to changes in antiseptic practices in the delivery room and in newborn nurseries

and implementation of screening for maternal bacteriuria[15]. Colonization of infants in NICUs after 2 weeks of life tends to originate from NICU and may involve species different from those that are maternally acquired[16,17]

Early onset neonatal sepsis is the main cause of mortality and morbidity in neonates born to mothers with the prom. Hence this study is taken up to identify different outcomes and institute proper treatment for early-onset neonatal sepsis at the earliest.

## AIMS AND OBJECTIVES

1. To study the outcome of neonates born to mothers with PROM.

2. To study the incidence of early onset septicemia and its correlation with vaginal pathogens in mothers with PROM.

# REVIEW OF LITERATURE

Early-onset bacterial sepsis continues to be a leading factor in newborn fatalities and morbidity. However, the rate of sepsis-related deaths later fell by 21% from 1974 to 1994. The fundamental approach to treating neonates with sepsis has not altered significantly over the ages. There will probably be more improvisation in the conclusion as a result. Excellent comprehension of the prenatal risk factors that cause sepsis and better methods to identify infected people and strategies that address these issues in infected newborns18.

**Definition**

- prom was defined by Ekwall in 1961 as the breaking of membranes before the beginning of labour at any stage of pregnancy[19].
- Hughes in 1972 defined prom as an amniotic sac before the onset of uterine contractions.

Premature rupture of membranes (PROM) is a condition that is defined by the breaking of the foetal membranes before labour. Preterm Premature rupture of membranes (PPROM) refers to PROM before 37 weeks of gestation[20].

**INCIDENCE:**

Spontaneous PROM occurs relatively frequently. Its incidence varies from 2.7 to 17%. Following are a few-

Kilbride H.W and Thibeault DW- 10%[21].

Aktar and sharma – 3.3%[22].

Cart and greffray- 5.7%[23].

However, 1% of the patients with PROM are preterm.

**Etiology :**

Numerous factors can cause membrane rupture. At term, the membranes may weaken due to physiological changes and shearing forces produced by uterine contractions[25,26]. An intrauterine infection has been demonstrated to be a a significant factor in PPROM, particularly at earlier gestational ages[27]. Low socioeconomic status, STDs, prior preterm births (particularly those caused by PROM), vaginal haemorrhage, cervical conization, and smoking while pregnant are all factors that have been linked to an increase in PROM[28,29,30,31] may also be linked to uterine distension (hydramnios, twins), emergency cervical surgery, past antepartum antibiotic use, and preterm labor[32,33,34].

The maternal-fetal medicine (MFM) network discovered that prior PPROM, positive fetal fibronectin at 23 weeks, and a short cervix (25mm) at 23 weeks were risk factors for PPROM. Some genital bacteria produce collagenases, proteases, and phospholipases, which may work to weaken membranes.

In cases of PPROM, positive cultures of the amniotic fluid are obtained in about 30% of cases[35].

Increased incidences of PPROM have been linked to deficiencies in vitamin C, copper, zinc, and general nutritional health as measured by body mass index. It has been claimed that pre-existing medical disorders including maternal hypertension or diabetes may have some influence on PPROM[36].

**Maternal outcome :**

Acute chorioamnionitis, subclinical chorioamnionitis, preterm placental separation, and postpartum endometritis are the maternal problems most frequently linked to PROM.

**Acute chorioamnionitis :**

One of the most dangerous conditions is chorioamnionitis, a type of intrauterine infection. Obstetrician in practise issues that were later discovered by the Paediatrician.

Maternal and neonatal death and morbidity have been linked in large part to bacterial contamination of the amniotic cavity.

Acute chorioamnionitis causes complications between 0.5 percent and 10 percent of pregnancies, but in complicated pregnancies that last longer than 24 hours, the incidence may reach 3 to 25 percent[37]. When a patient is delivered via cesarean section, maternal morbidity rises by a factor of five, increasing maternal mortality to 0.18 percent. The length of PROM and the number of vaginal checks affect the mother's incidence.

The risk of chorioamnionitis in various studies is variable.

Some of the studies had the following incidence :

Clark – 10.7%[38]

Gunn – 5.2%[39]

Aktar and Sharma – 24%[40]

A histologic abnormality known as acute chorioamnionitis is an inflammation of the fetal membrane. It is challenging to diagnose acute Chorio amnionitis since the signs and symptoms are not specific. An accurate link between physical findings and test results aids in the formation of a preliminary diagnosis that may later be supported by bacteriological and histological investigations. classical signs consist of:

1. Pyrexia before delivery of 100.4F or more.
2. Maternal or fetal tachycardia
3. Uterine tenderness
4. Leucocytosis
5. Foul-smelling amniotic fluid.

When the patient is febrile and one of the other two conditions listed above is true, a clinical diagnosis is made[41]. The measurement of CRP, a molecule that significantly rises in individuals with infection and inflammation, is a helpful blood test. With no fluctuation owing to gestational age, the maximum acceptable CRP levels during pregnancy are 0.9 mg/dl32. CRP levels in pregnant women with acute chorioamnionitis are typically higher than 3.0 or 4.0mg/dl[42].

If the diagnosis is unclear, the amniotic fluid obtained during trans-abdominal amniocentesis may be examined, and polymorphonuclear leukocytes found there is a sign of clinical amnionitis[43]. Gram staining of the deposit of a sample of centrifuged amniotic fluid can be quite helpful in determining the presence of chorioamnionitis[44].

Cervical samples, which typically support a high number of aerobic and anaerobic organisms, it is challenging to isolate a single organism[45].

E. coli and Klebsiella were the two most frequent pathogens isolated from the vaginal tract of mothers. The most frequent bacterium linked to the colonization of the maternal tract in western nations was Group B.Streptoccoci[46]. According to a study from Delhi, E. coli, Staphylococcus, and Klebsiella was the most typical bacteria found in the maternal vaginal tract[47]. The diversity of bacteria found in the vaginal canal may reflect regional differences in genital flora. In women with PPROM and acute chorioamnionitis, Ureaplasma urealyticum, Mycoplasma, Bacteroides bivies, Gardanella vaginalis, Pepto streptococci, Fusobacterium, and Enterococci are other bacteria that are frequently isolated in the amniotic fluid of the placenta[48].

If vaginal birth is not prohibited and labor has not already begun, the patient with acute or overt chorioamnionitis should start receiving antibiotic treatment as soon as the diagnosis is determined.

When antibiotics are administered prior to birth rather than afterward, there is evidence from several trials that the rate of maternal and new-born problems

is lower[49].

**Sub Clinical Chrioamnionitis :**

The appearance of uterine contractions is frequently the only sign of chrioamniotic infection. A change from a reactive to a non-reactive pattern in the Non-Stress Test (NST) and the absence of respiratory movements in the biophysical profile (BPP) are additional indicators of subclinical illness[50].
Desai BR from Belgaum, India, revealed that in the instance of PROM, an estimate of C-reactive protein was more effective at diagnosing subclinical infection than cervical swab culture, placental culture, and histology[51].

**Placental Separation :**

Abruption typically takes place during PROM and is not severe enough to result in fetal death or DIC. A gradual reduction in intrauterine surface area that results in placenta detachment is the cause of abruption in patients with PROM[52].

**Fetal and Neonatal Outcome :**

According to surveys, the rupture of the membranes occurs just prior to the start of labor in around one-third of low births and one-tenth of term deliveries[53].
The risks for morbidity and mortality in the fetus and infant have changed significantly as a result of the extensive changes in obstetric therapy of PROM over the past ten years. Major categories of prenatal and neonatal outcome significance include:

a. Fetal Growth.

b. Fetal and neonatal infections

c. Perinatal asphyxia

d. Neonatal RDS

e. Congenital anomalies

f. Perinatal mortality

**a. Fetal Growth**

A thorough analysis of fetal growth in relation to PROM revealed that, based on menstruation dates, 22.5 percent of babies were tiny for gestational age (birth weight less than the 10th percentile for gestational age). It was found that dates occur most frequently than predicted[54].

Preterm labor and PROM complicate the sonographic measurements of fetal growth during preterm gestation. Separate considerations were given to other risk factors that contribute to IUGR[55].

**b. Fetal and Neonatal Infections :**

Amnion, chorion, and amniotic fluid serve as a fetus defense against bacterial invasion. Fetal and newborn infections become the obstetrician's top priority once the membranes have ruptured.

Neonatal sepsis is described as having systemic clinical indications of infection together with the presence of positive bacterial cultures in the blood, CSF, or urine[56]. For infants born to women with PROM, the rate of neonatal infection ranges from 1 to 2.6 percent[57].

1.3 percent of births after PROM had infection determined by clinical, bacterial criteria and 8.1 percent of those births had clinical amnionitis[58].

Numerous variables affect the incidence of fetal infection. At the time of membrane rupture, vertical transmission of all pathogens, including bacteria, has been observed in the maternal genital canal. Preterm PROM has a higher infection rate than term PROM, according to research. The amount of time between the rupture of the membranes and delivery directly affects infection. The interval and gestational age are connected. The wider this interval, the younger the fetus is by gestational age.

The last factors determining the incidence are examination and other changes. It has been found that the interval between the initial inspection and delivery lengthens the risk of infection in both the mother and the fetus.

Infant infection rates rose from 5% in cases where the delivery took place within 24 hours of the first vaginal inspection to 44% in cases where the delivery took place after 46 hours of the first vaginal examination[59]. Maternal and fetal infections are considerably less common in mothers with PROM who receive earlier and longer antibiotic treatment. Positive intraamniotic fluid culture for the diagnosis of intra amniotic infection is necessary rather than clinical criteria. Quantitative culture result of 102 colony-forming units/ml of amniotic fluid of high virulence organisms is essential for labeling the case as infective[60].

**c. Perinatal Asphyxia:**

Following PROM, several pathways increase the risk of perinatal asphyxia. They consist of:

1. Compression or prolapse of the umbilical cord.
2. Malpresentations
3. Fetal compromise following maternal fever and chorioamnionitis.

Anoxia was the main cause, accounting for 44.9 percent of deaths[51], according to Diakoku's research on a 6.4 percent rate of perinatal asphyxia mortality in patients with preterm gestation[61].

A strong correlation between maternal fever and stillbirths has been established, whether there are symptoms of chorioamnionitis or not. By lowering placental blood flow as a result of vasculitis, fever causes fetal hypoxia. After extended PROM[62], the Apgar values were low, ranging from 10.8% in the non-infected group to 30.3 percent in the infected group. Meconium staining occurs in 3 percent 42 of PROM samples[63].

Malpresentations such breech and transverse lies are frequently encountered in PROM and increase the risk of hypoxia due to labor's mechanical difficulties.

**d. Neonatal RDS:**

It is a well-known fact that babies weighing less than 2000 grams tend to develop RDS more readily. However, Aldens pointed out that prolonged PROM actually reduces the incidence of RDS in infants weighing less than 2000 grams[64].

Curet described an incidence of RDS of 28.1% in preterm babies delivered with intact membranes as compared to 9.2% in preterm babies delivered following prolonged PROM[65].

The incidence of RDS is less in infants of 1000 to 1500gms after a delay of 48 hours between rupture of membranes and delivery[66].

Sell and Harris found significantly fewer cases of RDS in patients of all gestational ages when the latent period was longer than 24 hours[67].

It is common knowledge that babies weighing fewer than 2000 grams are more likely to suffer RDS. Nevertheless, Aldens made a point to mention that prolonged PROM really lowers the incidence of RDS in children weighed under 2000gms[68].

Curet described an incidence of RDS of 28.1% in preterm babies delivered with intact membranes as compared to 9.2% of preterm babies delivered following prolonged PROM[69].

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Sell and Harris found significantly fewer cases of RDS in patients of all gestational ages when the latent period was longer than 24 hours[71]

- PROM lasting more than 24 hours was linked to an amniotic fluid L:S ratio that was higher than usual for that trimester.
- The amniotic fluid included phospholipid glycerol (PG) at an earlier time after PROM in the gestational phase.
- Neonates born preterm with aspirates from the pharynx or trachea, A mature phospholipids pattern was visible in PROM lasting longer than 24 hours at gestations between 29 and 33 weeks.

The aforementioned could be the outcome of an increase in glucocorticoids in the blood of the mother and fetus during the course of the hours following PROM[72].

Jones examined 1600 newborns and came to the conclusion that there was no the connection between a rise in RDS incidence and a lengthening of the latent the period following PROM[73].

**e. Congenital Anomalies :**

Oligohydramnios is the outcome of liquor drainage. Due to internal mechanical forces, this results in deformation. The implications worsen if oligohydramnios persists for a longer period of time.

Prolonged PROM causes two main congenital anomalies:

1. Pulmonary hypoplasia.
2. Skeletal irregularities

A significant rate of pulmonary hypoplasia has been observed after protracted PROM. Although this has clinical traits similar to RDS, the fatality rate is Higher[74]. The consequence is typically fatal, and survivors frequently develop persistent bronchopulmonary dysplasia.

Skeletal anomalies include, but are not limited to, talipes equinovarus, aberrant facial characteristics, flat hands or arthrogryposis, joint swelling, deformities, etc. Numerous problems, including intrauterine death in extreme cases, have been connected to adhesive bands[75].

**f. Perinatal mortality :**

According to a collaborative perinatal study, there are 10.8% perinatal deaths. After PROM at term, perinatal death is connected to infection and in premature because of RDS

**g. Cerebral palsy :**

It is a long-term consequence of PROM, especially in situations when there is subclinical or acute chorioamnionitis, severe intraventricular hemorrhage, or intrapartum fetal acidosis[76].

## NEONATAL SEPSIS

**Introduction:**

The most likely cause of newborn mortality is sepsis. In underdeveloped nations, it is to blame for between 30 and 50 percent of all newborn deaths. Up to 20% of newborns are thought to have sepsis, while only 1% of them die from sepsis-related causes[(77,78)].

**Definition :**

A bacterial infection that is confirmed by positive blood culture in the first 30days of life is referred as septicemia in newborns.

**Incidence :**

According to information from the National Neonatal Perinatal Data Base (NNPD 2002-03), there are 30 cases of neonatal sepsis for every 1000 live births. Staphylococcus aureus (32.5%) and Klebsiella pneumonia (13.6%) were the most frequently isolated pathogens in intramural deliveries (13.6 percent ). Klebsiella (27%) was the most frequent pathogen in extramural newborns.

Pseudomonas (15%) and Staphylococcus (15)% followed by Streptococcus (15%) and Pseudomonas (13%)80.

Classification of neonatal sepsis :

Neonatal sepsis can be classified into two major categories depending on the onset of symptoms[81].

1. Early onset sepsis.
2. Late-onset sepsis.

**Early onset sepsis (EOS) :**

Within the first 72 hours of life, it manifests. In serious circumstances, the newborn may already exhibit symptoms when born. The most common symptoms of EOS in infants are pneumonia and respiratory distress.

The maternal genital tract is typically the source of infection. An elevated risk of EOS has been linked to perinatal disorders.

Understanding these possible risk factors could aid in the early detection of sepsis. Because there is a possibility of infection rising, prolonged leakage and early membrane rupture are regarded as important sepsis risk factors.

In research by Shah G.S. that included 100 cases of newborn sepsis and 100 control neonates, 46% of the sepsis-affected infants developed PROM[84].

**Late-onset sepsis (LOS) :**

It typically shows about 72 hours after birth. Neonates typically arrive with septicemia, pneumonia, or meningitis when they have LOS, and the origin of Either a nosocomial (hospital-acquired) or community-acquired illness exists[85,86]. Low birth weight, premature hospitalization in the NICU, mechanical ventilation, invasive procedure, administration of parenteral fluids, and use of stock solutions are only a few of the factors that raise the risk of nosocomial sepsis.

Poor hygiene, inadequate cord care, and bottle feedings are among the factors that could raise the risk of community-acquired LOS.

**Organisms responsible for EOS :**

According to the newborn perinatal database (NNF 1999), E. coli and Pseudomonas were the most prevalent bacteria, with Staphylococcus aureus coming in second.

52 cases of EOS were found to have maternal genital canal colonization in research by Basavaraj M. Kerur et al. which included 102 patients. The most typical isolate was E. coli, followed by a Klebsiella species. Only one case of GBS was observed.

Klebsiella and E.coli are the most typical combination. The most frequent bacterium discovered in babies' blood cultures was Klebsiella. E. coli, Acinetobacter, and coagulase-negative staphylococcus were followed by Klebsiella. Only one case involved the isolation of Group B streptococcus. Baby's body surface bacteria and the bacteria in the mother's vaginal tract were significantly correlated. A significant association was also found between

the organism recovered from the baby's blood and the baby's surface colonization[87].

22 percent of EOS were culture-positive. The most frequently isolated microorganisms were Klebsiella pneumonia and Staphylococcus aureus[88]. Pseudomonas and Klebsiella were listed as prevalent isolates in EOS in other earlier studies[89,90].

**Incidence :**

According to Anne Schuchat et al., there were 3.5 EOS cases for every 1000 live people. 90 deliveries were from mothers whose PROM was greater than or equivalent to 18 hours.

Group B streptococci caused 13.5 percent of EOS, whereas other species were responsible with 26.7 percent of cases[91].

The prevalence of neonatal sepsis in mothers' premature newborns was 18.4 percent with PROM[92].

In patients with a positive maternal history of antibiotic intake, the prevalence of new-born infection was around 4.4%, while it was 11% in patients with a negative maternal history of antibiotic intake[93].

**Clinical features of EOS :**

Asymptomatic bacteremia, widespread sepsis, pneumonia, and/or meningitis are possible manifestations of EOS. 90% of newborns show symptoms at 24 hours of age and the clinical signs of EOS are typically seen in the first hour of life. The primary presenting symptom is respiratory distress[94].

Neonates with sepsis may exhibit any one of the following symptoms and signs:

a. Fever or hypothermia

b. Weak cry, lethargic and an unwillingness to sucking

c. Poor cardiac output

d. Hypotonia

e. New born reflexes not evident

f. Either a brady- or tachycardia

g. Apnea, gasping respiration, and respiratory distress

h. Low or high blood sugar

i. Metabolic acidosis

**Certain clinical characteristics relating to different systems**[95].

**Central nervous system :**

- Bulged AF
- Vacant stare
- cry with a high pitch
- excessive irritability
- Stupor or coma
- Convulsions
- Neck stiffness

These characteristics ought to cause a clinical suspicion of meningitis.

**Cardiovascular system :**

• Low BP

• CFT >3sec

• Shock

**GI system :**

• Intolerance to feed

• Diarrhea

• Vomiting

• Distension in the abdomen

• Ileus paralyticus and NEC

**Hepatic:**

- liver enlargement
- Increased direct bilurubin

**Renal :**

• Acute kidney failure

**Haematological :**

• petechiae, purpura and bleed

• Disseminated intravascular coagulation.

**SKIN :**

- sclerema

**Direct Indicators**

**1. Buffy Coat Smear (BCS) :**

Gram staining of the buffy blood layer that results during centrifugation and the buffy coat smear testing includes plasma separation. In 57.70% of instances of newborn sepsis, if done thoroughly, microorganisms can be found[96].

In 100 cases of suspected septicemia, the buffy coat smear examination was investigated by Gupta S et al. Forty percent of septicemia cases with positive blood cultures had negative BCS, compared to 19 out of 32 infants whose blood cultures were positive. As a result, they came to the conclusion that BCS cannot be employed as a septicemia screening test[97].

**2. Blood culture :**

Despite the expanded accessibility of cutting-edge molecular technology for identifying and disclosing microbial infections. The most significant test results produced by the clinical microbiology laboratory, in the opinion of the majority of clinicians, are still the antibiotic susceptibility report and the isolation of bacteria.

It is required in every case of septicemia because it is the gold standard for diagnosis, before giving medication. A positive blood culture with isolated organism is an indication that you require antibiotic treatment.

As a result, it is crucial to obtain a blood culture in accordance with the correct protocol. Prior to the procedure, the resident physician or staff should put on sterile gloves and prepare a patch of skin with a diameter of about 5 cm above the intended venepuncture site.

Alcohol should be used to disinfect this area completely, followed by povidone-iodine and alcohol once again. Before taking a sample, the skin needs to be given at least one minute to dry. For blood culture, one ml of blood should be adequate for 5-10ml of culture

Only samples from a clean venipuncture site should be taken because samples taken from indwelling lines and catheters are likely to be contaminated. Before a blood culture is declared sterile, it must be kept under observation for at least 72 hours.

BACTEC and BACT/ALERT culture systems, may now identify bacterial growth within 12–24 hours. 1-2 colony forming units (CFU) per ml is the minimum concentration of bacteria that these cutting-edge techniques can detect. These new machines contain a continuous monitoring system to detect the CO2 level. The BACT/ALERT system uses calorimetry to detect changes in pH, while the BACTEC101 and BACTEC102 use fluorescence sensors[98,99].

The main cause of gram-positive septicemia is staphylococci. Gram-negative septicaemia causes the vast majority of fatalities[100].

43 blood cultures from 250 ill newborns studied by Chugh K et al in 1987 were positive. The most prevalent organisms were Klebsiella species (10%), followed by Pseudomonas and Staphylococcus aureus, whereas E. coli accounted for just 7 percent of all organisms[101].

Following the implementation of the strengthened asepsis procedure, septicemia incidence, morbidity, and length of hospital stay all decreased.

**3. Lumbar Puncture (LP) :**

It is quite common for the clinical signs of septicemia and meningitis to Coincide without any particular cause, it's conceivable to have both meningitis and septicaemia symptomatology. This justifies taking extra security measures and running LP in new-borns who may develop sepsis. In EOS , LP is indicated if a septicaemia-compatible clinical presentation or a positive blood culture is present. It is not recommended if antibiotics have only been administered because of the presence of danger factors The lumbar puncture may be delayed in critically ill newborns. It should be carried out as soon as the clinical state stabilises. The percentage of meningitis in newborn sepsis ranges from 0.3% to 3%.

**Normal CSF values in Neonates:**

Cerebrospinal Fluid Components Normal Range

Cells/ mm3 0-30

PMN cells - 60%

Proteins (mg/dL) 20-170

Sugar (mg/dL) 34-119

Blood glucose- 51 (44-248)

The CSF cytology and biochemistry of neonates are different from that of older neonates. Also, there is an overlap of values in normal neonates and those with meningitis.

**4. Urine culture :**

Urine culture has a low yield in EOS and is not recommended. Cultures of urine acquired from bladder catheterization or suprapubic puncture have advised in all situations of late-onset sepsis.
Mishra et al observed that in 1.7% of cases UTI was present in association with septicemia[102].

**5. Radiology :**

Chest X-ray should be considered in the presence of respiratory distress or apnea. An abdominal x-ray is indicated in the presence of abdominal signs suggestive of NEC. Transcranial ultrasound and computed tomography (CT scan) should be performed on all patients diagnosed to have meningitis.

**6. Tracheal cultures:**

Tracheal cultures should be acquired in neonates who are intubated and have a clinical picture that suggests pneumonia, if the mother experienced chorioamnionitis and the baby had an excessive amount of EOS, or if the quality and volume of Tracheal secretions undergo significant alteration. After several days of intubation, tracheal aspirates have little usefulness.

**Indirect indicators**

**1. Blood leucocyte changes :**

**a. Leucopenia:**

It has been thought that newborn babies' total leucocyte counts are such
It is so unexpected and changeable that it is of little use in clinical diagnosis. Between 8000 to 20,000 leucocytes per millilitre throughout the first 28 days of existence free of visible sickness. Usually, neonatal sepsis is accompanied by leucopenia of less than 5000 cu/cumm. This discovery was used in conjunction with other criteria to aid in the early diagnosis of neonatal sepsis with appropriate sensitivity and specificity [103].

**b. Band count to the total neutrophil ratio :**

A band neutrophil has a sausage or band from the nucleus. The chromatin is coarse resembling that of mature cells with abundant cytoplasm.
A 1:2 nucleo-cytoplasmic ratio is expected. Total neutrophil count and band cell count ratio of 0.2 or higher indicates septicemia.[104].
At birth, the B/N ratio (band to total neutrophils) is /= 0.16 after 72 hours, it peaks at 0.12 and then starts to drop.
Monroe et al. believed in 1979 that a light or early infection led to a substantial rise in neutrophil absolute value. The metrics were really high as 17,500 [105,106]. In 1979, Edward Squire et al. noted that sepsis-related neonatal deaths were common. Typically show leucopenia, neutropenia, and

thrombocytopenia connected to typical bone marrow cell synthesis107.
In 1980, Alistair G.S. Philip conducted research on the subject of early neonatal sepsis. Leucopenia 5000/cmm and band to neutrophil together
ratio >0.2 was especially prognostic of newborn sepsis[108].

Leucopenia was discovered by Lokeshwar in 1988 to occur in 20% of patients, and band cell count in 80 percent of patients had a band to neutrophil ratio. In addition, he discovered two or more abnormal values in 94 percent of instances[109].
Robert Boyle et al(1978) also observed the usefulness of absolute neutropenia and band neutrophil ratio in identifying septic from non-septic infants with respiratory distress syndrome[110]
Low platelet counts and morphological changes in neutrophils were often severe and late signs of infection.

**Sepsis Screen :**

1) WBC count <5000/mm3
2) ANC count as per *Monroe Chart for
term neonates
3) I/T ratio>0.2
4) Micro ESR >15 mm in 1st hour
5) CRP >10mg/L

**c. Morphological changes :**

The neutrophils of septicemic neonates have been shown to have abnormal morphology with the appearance of Dohle bodies, toxic granulations, and vacuolization.

Toxic granulations are coarse deeply eosinophilic or darkly 27-staining granules irregularly distributed throughout the cytoplasm of the neutrophils. Toxic granulations equal to or more than 40% of cells are suggestive of septicemia[111].

- In order to better understand leucocyte blood images in healthy and During the first three days of life, the number of neutrophils in healthy newborns typically decreases, therefore their value indicates infection. This was observed in unwell neonates. The total white cell count has little utility in identifying illness when it drops to extremely low levels. Additionally, he discovered that beyond the first three days of life, the absolute values of polymorphonuclear cells (PMN) in healthy term and preterm infants will never rise above 7000/cumm. After 72 hours of life, there is a rise in the total number of mature neutrophils in sick newborns.[112].
- Akeunzuea et al (1974) concluded that an absolute number of neutrophils proved to be an unsatisfactory index of infection in the first 5 days of life. The early and dramatic increase in band cells occurs in infection[113].
- Zipursky et al (1976) in his study observed a significant increase in immature polymorphs and the appearance of toxic granulation. Qualitative changes in neutrophils (Dohle bodies, toxic granulation, and vacuolization) were more frequent in bacterial sepsis[114].
- Alistair G.S. Philip et al (1980) carried out a study of various hematological indices for early diagnosis of neonatal sepsis. The five most useful tests he included, were band / total neutrophil ratio (0.2 or more), leucopenia (less than 5000 cells /cumm), micro ESR (more than 15mm per the first hour), C-reactive protein (more than 0.8mg/dl) and haptoglobin (more than 25mg/dl). The sensitivity and specificity of each test varied but the combination of leucopenia and elevated band cells to total neutrophil ratio was more predictive of sepsis. In 99% of cases without sepsis, less than two tests were positive[115].

Micro ESR alone has 30% and 97% sensitivity and specificity respectively.

**2. Acute Phase Proteins**

**C-reactive protein (CRP) :**

A number of acute-phase proteins can be used to detect infections in infants. Reactive protein C is the one that has been examined the most. Two Pre-albumin and transferrin are two proteins that are negative reactants, their levels decrease during inflammation and rise during healing.

**a. Those increasing with inflammation :**

1. C-reactive protein
2. α-1 acid glycoprotein (orsomucoid)
3. Hepatoglobin
4. α-1 antitrypsin
5. Fibrinogen

**b. Those decreasing with inflammation :**

1. Prealbumin
2. Transferrin

The efficiency of CRP was evaluated in diagnosing early septicemia. The test become positive when the concentration of CRP was approximately 0.8mg%. The test was 86% specific. The sequential decrease in CRP levels during treatment is suggestive of a full recovery and an adequate response to the medications. An increase in CRP level could come before the virus recurs. Tillett et al. made the discovery of C-reactive protein in 1930. They noticed That patients' sera with acute febrile fever brought on by an extract of c-polysaccharide, a pneumococcus. The chemical that causes reactivity was a protein known as c-precipitin. Later, it was referred to as as a C-reactive

substance. Enhanced CRP quantification techniques have resulted in more widespread use in clinical medicine[116,117].

CRP is tested by capillary precipitation of the patient's sera with antisera prepared in rabbits against purified C-reactive protein or by passive agglutination using latex particles coated with anti-CRP antibody[118].

Mishra PK et al (1987) showed that CRP was positive in 69.7% of cases of proven bacterial sepsis and hence can be used as an important investigation in suspected sepsis. All the cases of pyogenic meningitis had positive CRP (serum and CSF). The sensitivity and predictive value of this test in meningitis are close to 100% making for more accurate than the classical parameters of CSF leukocytosis and low CSF glucose values[119].

**3. Thrombocytopenia :**

Cohen P et al (1966) studied platelet and granulocyte response in cases of septicaemia. He concluded that the response of these cells was parallel at the onset of the disease, but in fulminating infection granulocyte remained normal or reduced but platelet count was always decreased[120].

**5. Nitro blue tetrzolium (NBT) test :**

This technique involves the reduction of nitroblue tetrazolium to formazan by leucocytes which are actively phagocytosing and killing bacteria. The normally high reduction in neonatal leucocytes may give false positive results[121].

**6. Leucocyte alkaline phosphatase (LAP) activity :**

Sharma DK et al (1985) estimated LAP activity by the cytochemical methods and found significantly low LAP scores in both term and preterm neonates with proved internal infection[122].

**7. Gastric aspirate for polymorphs :**

This test was considered positive if polymorphs were more than 75%. The sensitivity of this test was observed to be very low (3.8%) in diagnosing septicemia[123].

**8. Micro erythrocyte sedimentation rate (Micro ESR) :**

Erythrocyte sedimentation rate is a nonspecific indication of tissue injury and is known to increase, and the rate of increase is known to be based on how severe the morbid process is. Its usefulness in Pediatrics was first described in 1933. The Launder modification of the Linzenmeier Raunet method is useful in Pediatrics since it does not require venipuncture. It requires only a few drops of capillary blood.

Clay Adam micro sedimentation rate pipette or heparinized capillary tubes are used for the estimation of micro ESR[124].

Normal values in the neonates vary from 6mm/hr in the first 3 days of life to 11mm/hr at the end of the first month (95th percentile). Micro ESR, values correlate closely with Westergren's levels at 75mm/hr and below. Its ESR values over 15mm/hr correlate progressively with higher Westergren levels. Eg: µ ESR levels of 25 and 30mm/hr are equivalent to values of 40 and 70mm/hr of Westergren.

A value of more than 10mm/hr is seen as symptomatic of infection throughout

the newborn era.[125].

Parida SN et al (1980) concluded that normal ESR value in neonates was 8mm/ hr at the 95th percentile. ESR was raised in 71.4% of infected babies while 24% of probably infected babies[126].

## 9. PROCALCITONIN

There have been reports of serum procalcitonin as a quantifiable laboratory indicator of infection-induced inflammation. liver's macrophages and monocytes during sepsis causes an increase in procalcitonin secretion. The degree of Procalcitonin rises quickly in 6 to 8 hours, plateauing between 12 and 48 hours. As a result, it offers promise as a novel marker for the early detection of a sick patient. Another advantage of procalcitonin is the rise in infections with bacteria and fungi, but no alterations with viral infections and other inflammatory condition[127].

- An high immature to total neutrophil ratio, the most sensitive leucocyte count, may be used to identify 75% of patients who acquired septicaemia within the first three days of life and 60% of all neonates with septicaemia or meningitis.
- Only 33 percent of the patients had thrombocytopenia. Additional fibrinogen analysis is neither useful for diagnosing new born infections nor for follow-up.
- In contrast, 8.8% of all new-borns diagnosed with neonatal septicaemia and meningitis have elevated C-reactive protein levels. Neutrophil indices and CRP, when combined, even increased the sensitivity of a single laboratory screening test[97].

None of the tests employed individually are trustworthy, but when combined,

these tests may aid in the rapid diagnosis of sepsis. Moreover, if the tests reveal a high Negative predictive value, the newborns may be released from the hospital earlier halting the antibiotics at the hospital, lowering the cost of care, and concern for the family[128].

**Management**

**Supportive care :**

Neonatal sepsis is a disease that involves all the organ systems of the body, through its response at the cell level. As a consequence of sepsis, a sick baby suffers from hypoxia, hypothermia, poor perfusion, hypoglycemia, coagulation disturbances manifested as generalized bleeding, and multisystem organ failure.

Monitoring can be divided into clinical and laboratory. General activity, the status of anterior fontanelle, feeding behavior, color, temperature, CFT, pulse rate, heart rate, peripheral pulsation, respiratory rate, blood pressure, oxygen saturation, abdominal girth, and urine output should be frequently monitored. Frequency of monitoring of these clinical features would depend upon the severity of the baby's illness, hence it may vary from one hour to 4-6 hours.

**Laboratory monitoring includes:**

1) blood glucose estimation
2) urine specific gravity and urine osmolality
3) urine examination
4) serum electrolytes
5) blood urea and serum creatinine
6) coagulation profile

7) blood gas analysis for pH and partial pressure of oxygen and carbon dioxide
8) x-ray chest.

**Oxygenation :**

Neonatal sepsis produces a stressful condition. Which requires the increased need for oxygen for the maintenance of normal cellular function, Oxygen should be administered to achieve a paO2 60-80% and SaO2 87-95%. Oxygen can be administered through a head box or by assisted ventilation.

**Perfusion :**

Appropriate management of fluid and electrolyte balance is a critical supportive measure in the treatment of a sick baby particularly when there is an impending or definite shock. Volume needs to be replaced. The aim of fluid therapy is to achieve good perfusion, adequate hydration, and urinary output of 1-2ml/kg/hr with normal specific gravity. There is considerable controversy regarding the use of crystalloid or colloid as the rehydration solution in septic shock. In general, crystalloids may be used initially to increase the blood volume and cardiac output, and if it fails colloids may be used.

**Vasopressor Agent :**

Fluid administration during the early stages of sepsis and septic syndrome
The use of antibiotics and other forms of supportive care could be enough to begin the treatment for the patient. However, in many patients whose therapy initiation was postponed, It may be necessary to use inotropic or vasoactive medications. Due to its - adrenergic effects on the body, dopamine is the favoured initial option. The myocardial and its impact on the peripheral

vasculature through -adrenergic signalling the renal and splanchnic dopaminergic actions that are exclusive to it. Dobutamine addition is indicated, When there is reduced myocardial function and elevated systemic vascular resistance when neonates experience myocardial infarction and severe hypotension, depression not reacting to dopamine and dobutamine in sufficient amounts, It is necessary to administer epinephrine or norepinephrine.

**Glucose Homeostasis :**

Sick babies require more glucose because of increased demands coupled with inefficient metabolism. The non-availability of adequate glucose concentration in the blood would elicit glycogenolysis thereby further aggravating acidosis. Supporting sick babies with augmented glucose infusion at a rate of 4mg/kg/min may prevent further damage.

**Thermoregulation :**

Temperature maintenance is best achieved by adequately covering babies as much as possible, keeping their environmental temperature in the Thermo neutral range, and nursing them preferably in double-walled incubators with at least 50-70% humidity. Babies with sepsis have discordance in their care and peripheral body temperature hence it is desirable to measure the core temperature and not to act on peripheral temperature.

**Nutrition :**

Adequate nutrition management is crucial for newborn babies especially small preterm and growth retarded babies. These babies hardly have any reserve even in normal circumstances and in a stressful situation with increased demands they quickly lose body weight. We must aim at providing adequate calories and protein (100-110k cal/kg and 2-4 gm/kg of protein) through the enteral or parenteral route[129].

**Antimicrobial therapy :**

The decision to start antibiotics is based on clinical features and or a positive septic screen.

**SIGNS THAT ANTIBIOTICS SHOULD BE STARTED:**

The indications for starting antibiotics in neonates at risk of EOS include any one of the following :

a. presence of at least three EOS risk factors

b. the presence of foul smelling liquor

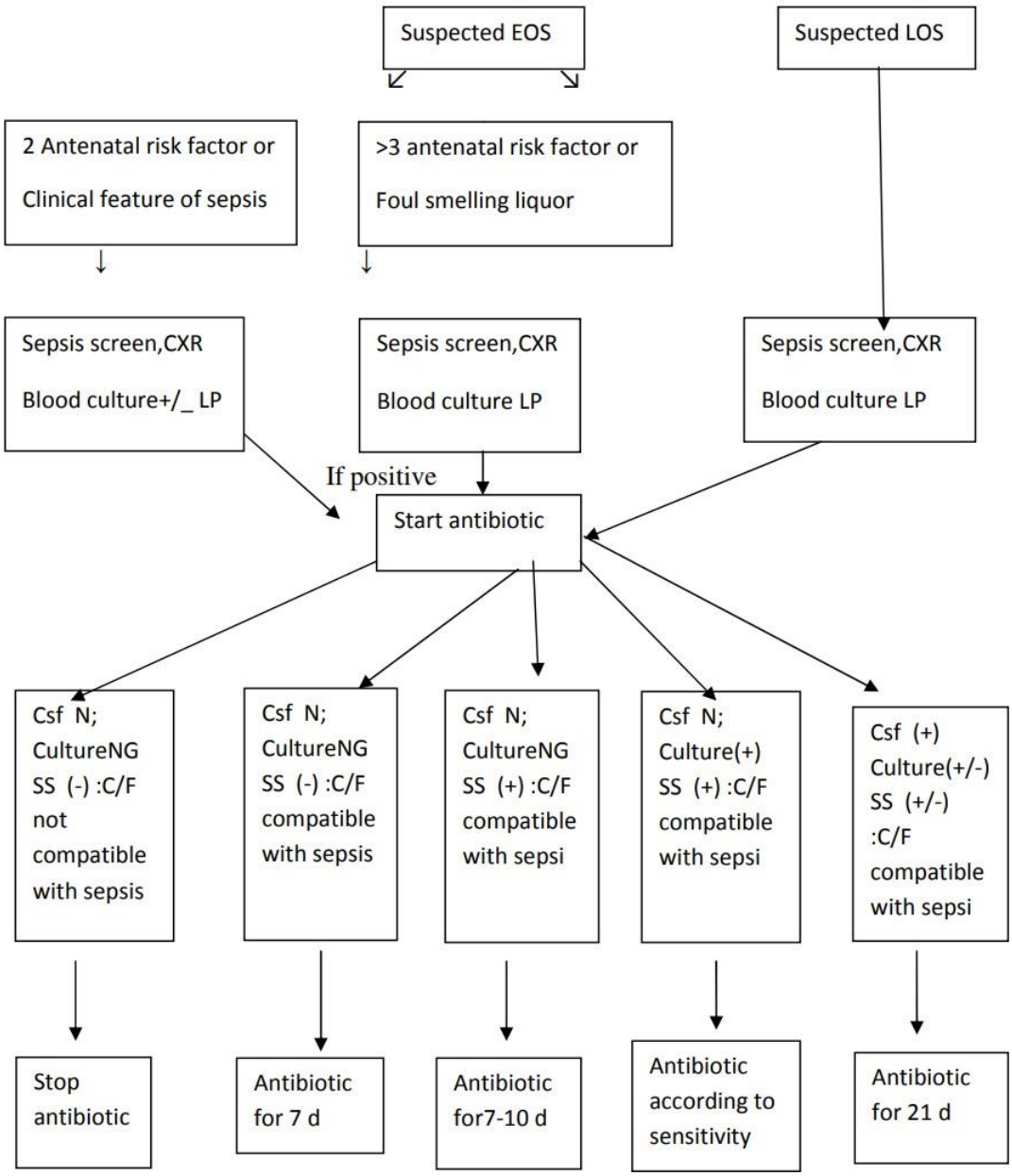
c. two prenatal risk factors present and a positive septic screen

d. clinical suspicion of sepsis is very strong

But the length of antibiotic treatment depends on the presence of blood cultures that are positive and meningitis.

**TABLE-1**

## PROTOCOL FOR SEPSIS[53]



**3. Sensitivity pattern :**

When clinical suspicion is made, antibiotics are started keeping in mind the sensitivity pattern of the prevailing organism. After obtaining sensitivity pattern therapy should be changed to the sensitive antibiotic.

**1. Hierarchy and Combination :**

Guidelines for initial combination therapy

| | First line | Second line |
|---|---|---|
| Community-acquired | Ampicillin and gentamicin / amikacin, Cefotaxime and other | cefotaxime and other aminoglycosides ciprofloxacin and other aminoglycoside |
| Nosocomial | | |

**TABLE-2**

Add Cloxacillin if the prevalence of Staphylococcus is high. If Methicillin-resistant Staphylococcus aureus (MRSA) is the predominant organism, add Vancomycin

Reserve Drugs: Imipenem / Aztreonam for multidrug resistant organism[130].

**Empirical choice and duration of antibiotic therapy in neonatal sepsis and meningitis:**

| Organism | Antibiotic | Bacteraemia | Meningitis |
|---|---|---|---|
| Group B streptococci | Ampicillin or penicillin G | 10-14 days | 21days |
| E. Coli | Cefotaxime or ampicillin and gentamycin | 14days | 21days |
| CONS | Vancomycin | 7days | 14days |
| Klebsiella | Cefotaxime or meropenem and gentamycin | 14days | 21days |
| Enterococci | Ampicillin or vancomycin and gentamycin | 10days | 21days |
| Listeria | Ampicillin and gentamycin | 10-14days | 21days |
| Pseudomonas | Ceftazidime or piperacillin/tazobactam and gentamycin or tobramycin | 14days | 21days |
| Staph. Aureus | Nafcillin | 10-14days | 21days |
| MRSA | Vancomycin | 10-14days | 21days |

**TABLE-3**

**Intravenous (IVIgG) Therapy :**

About 20 years ago, a new preparation of intravenous immunoglobulin became available, these can be administered intravenously in high doses and contain intact antibody molecules that maintain normal biological activities of the Fc fragment, such as complement activation, binding to cell surface receptors, catabolization, and opsonic activity, although with significant variability among available preparation.

Furthermore, the IgG subclass distribution is similar to that found in normal serum and includes antibodies to most of the pathogens found in neonatal infection.

Several studies have been carried out to evaluate the efficiency of IVIgG for the prophylaxis of infection in high-risk infants, with variable results, because of different methods used for patient selection, the different methods of care, the different study designs and the different IVIgG preparation and dosages used (in most of the trails 0.5g/kg weekly while in other studies IV IgG dosages have been individually regulated to maintain IgG serum level >400 or >700mg/dl).

The use of IV IgG may be a valuable tool for the prevention of infection in selected low birth weight infants with a low level of serum IgG and recurrent infection or for treatment of septicemia in sick neonates with a life-threatening disease.

**Neutrophil transfusion :**

The frequent observation of neutropenia and diminished neutrophil function in new-borns with severe infections may provide justification for the use of granulocyte transfusion. Studies have demonstrated that the survival of treated infants is better than that of controls, with particularly good outcomes for neutropenic new-borns (PMN 3.0 x 109/L during the first week of life or 1.0 X109/L there 40 after). Since hematopoietic growth factors are now widely available, the need to administer granulocytes has been significantly diminished.

**Colony Stimulating Factors therapy :**

Colony-stimulating factors are a class of glycoproteins that play a physiological role in the proliferation of mature peripheral blood cells as well as early stem cell precursors and late progenitor cells.

From newborn infants' mononuclear cells, it was discovered that G- CSF, and GM- CSF synthesis and gene expression were both reduced.

For severe congenital neutropenia, G-CSF and GM-CSF are regarded as first-line treatments. (Kastmann’s syndrome). Neutropenia in children of preeclamptic mothers and neonatal iso-immune (alloimmune) neutropenia are two other recent indications for the use of G-CSF in newborns.

In small, exploratory trials conducted on septic neonates, pentoxifylline, vitamin B12, and ethylene blue showed encouraging outcomes. Although there hasn't been much of an improvement in patients with septic shock, steroids may lower the frequency of long-term complications in children with meningitis[131].

**Exchange blood transfusion :**

The rationale for performing an exchange transfusion in neonates with sepsis includes a number of reasons that could be the possible mechanism resulting in improved survival of neonates with sepsis. These were:-

1. Improved oxygen-carrying capacity of the blood
2. Increased opsonic and granulocyte activity
3. Removal of bacteria, endotoxins, and inflammatory mediators.

At present exchange transfusion in neonatal sepsis can only be considered experimentally. The situation where one could consider exchange transfusion is in septic neonates with sclerema or unresponsive DIC, as an adjunct to antibiotic and other supportive care[132].

Risk Score Risk score uses the risk factors to predict the likelihood of developing neonatal sepsis in the newborn. It helps the physician to categorize the babies and to plan their management. One of the well-studied risk scores developed by Parmar is as follows

| Risk factor | | significant score |
|---|---|---|
| PROM >24 hours | 1 | |
| Unclean per vaginal examination | 2 | |
| Birth asphyxia | 2 | 5 or more in normal weight babies |
| Foul smelling liquor | 2 | |
| maternal intrapartum fever | 2 | 2 or more in low birth weight babies |
| prolonged labor | 1 | |
| Low birth weight | 1 | |

TABLE-4

The recently modified risk score is as follows

| risk factor | score | |
|---|---|---|
| >3 intrapartum vaginal examination | 6 | |
| clinical features of chorioamnionitis | 6 | score 0-6, careful monitoring for 72 hours for clinical features of sepsis |
| Birth weight <1500 g | 3 | |
| Male gender | 3 | score >7, septic screen followed by empirical antibiotic therapy |
| No intrapartum antibiotic | 2 | |
| Gestational age <30 weeks | 2 | |

**TABLE-5**

## MATERIALS AND METHODS

**Source of data:**

MOTHERS ADMITTED TO THE DEPARTMENT OF OBGY AND NEWBORNS ADMITTED IN DEPARTMENT OF PAEDIATRICS of Shri B. M. Patil Medical College, Hospital and Research Centre, Vijayapura.

**Type of study:**

It is a prospective observational study involving mothers with PROM and babies born to

mothers with PROM will be studied over a span of 1.5 years.

**Method of collection of Data (including sampling procedures if any)**

After taking written informed consent from the mothers with prom and fulfilling inclusion and exclusion criteria, mothers and their new-borns will be included in the study.

**MATERNAL:**

The vaginal swab will be collected in a sterile bottle and sent for culture sensitivity in mothers with prom before the start of antibiotics.

**Neonatal sepsis screening is done.**

**Screening includes:**

1) complete blood counts
2) CRP
3) Blood culture
4) I/T ratio
5) ANC

6) chest x-ray

7) Procalcitonin.

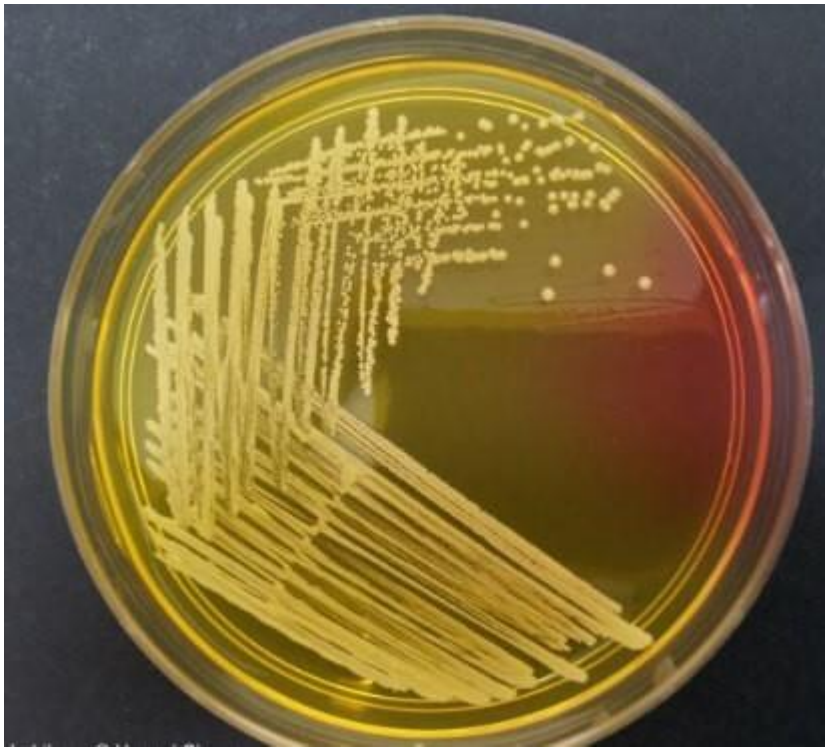


FIG.1 GROWTH OF STAPHYLOCOCCUS AUREUS

FIG.2 GROWTH OF STAPH.HOMINIS

**FIG.3 GROWTH OF KLEBSIELLA**

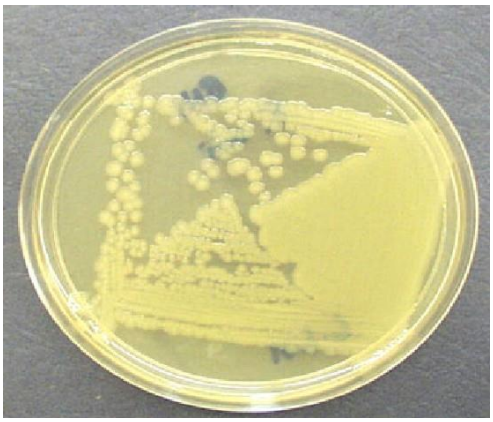


**FIG.4 GROWTH OF E.COLI**


**FIG.5 VAGINAL SWAB**

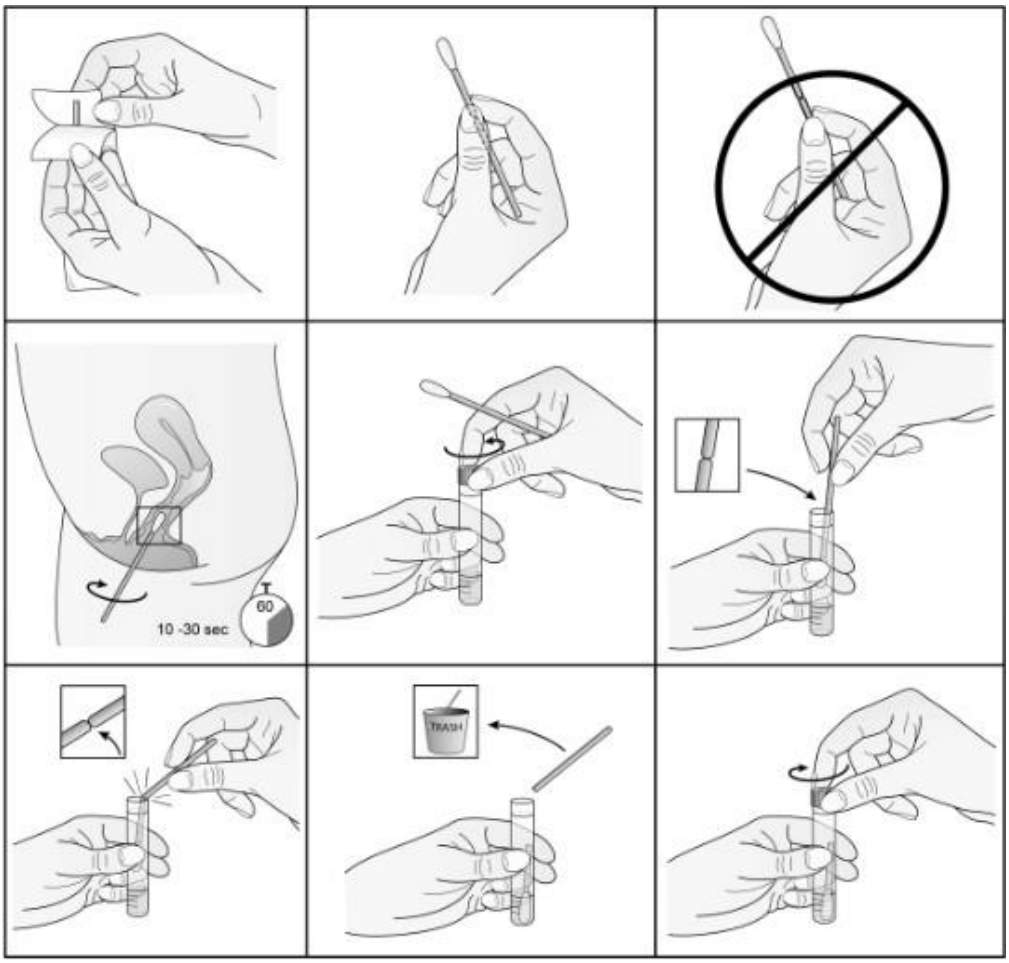



**FIG.6 STEPS OF COLLECTING VAGINAL SWAB**

**Method of study:**

A prospective observational study involving mothers with prom and new-borns born to mothers with prom admitted in the department of OBGY and NICU of BLDE hospital, Vijayapura.

Mothers with PROM and new-borns born to mothers with PROM will be considered as subjects.

Period of study – 1.5 years

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# RESULTS

The total number of neonates included in the study was 84.

**TABLE – 6**

**Sex-wise distribution of neonates**

| Sex | Number of cases | Percentage |
|---|---|---|
| Female | 28 | 37.33% |
| Male | 47 | 62.67% |
| **Total** | 75 | **100** |

The analysis of the study shows that out of 75 neonates, 28 (37.33%) were females and 47 (62.67%) were males.

**Sex wise distribution of neonates**

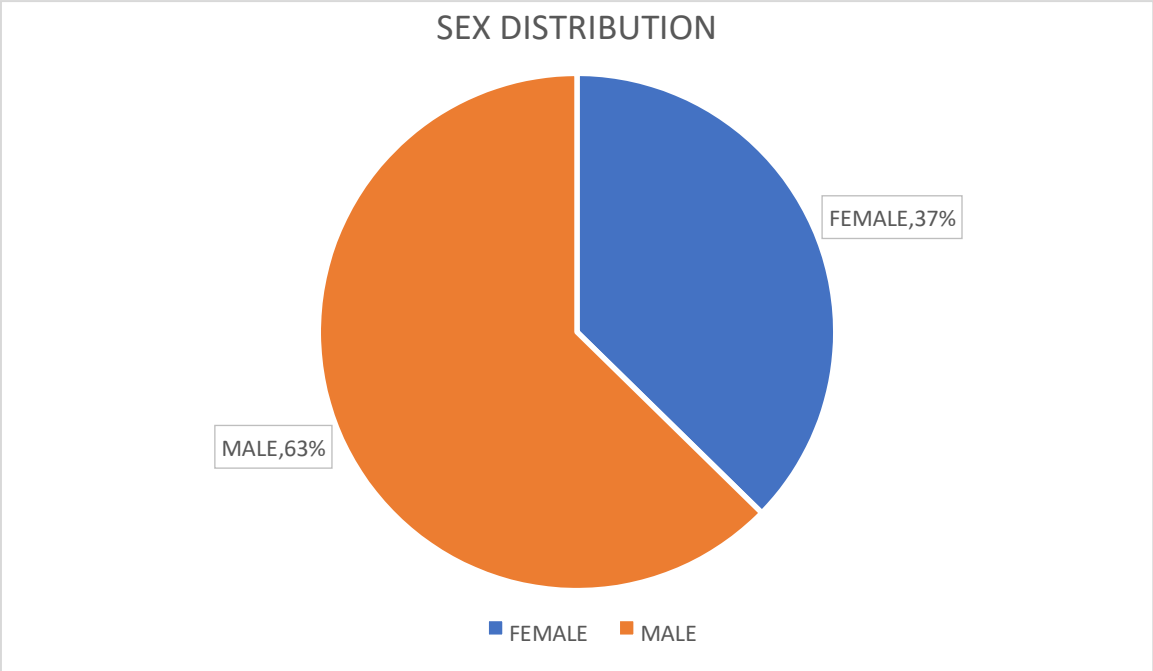



**FIGURE-7**

**TABLE- 7**

**Distribution of cases according to birth weight**

| Weight in grams | Number of cases | percentage |
|---|---|---|
| <1500 | 8 | 10.67% |
| 1500-2500 | **34** | 45.33% |

| | | |
|---|---|---|
| >2500 | 33 | 44% |
| **Total** | **75** | **100** |

The analysis shows that out of 75 cases, 8 (10.67%) cases weighing < 1500 grams, 34 (45.33%) cases were weighing between 1500-2500 grams and 33 (44%) weighing >2500 grams.

**Distribution of cases according to birth weight**

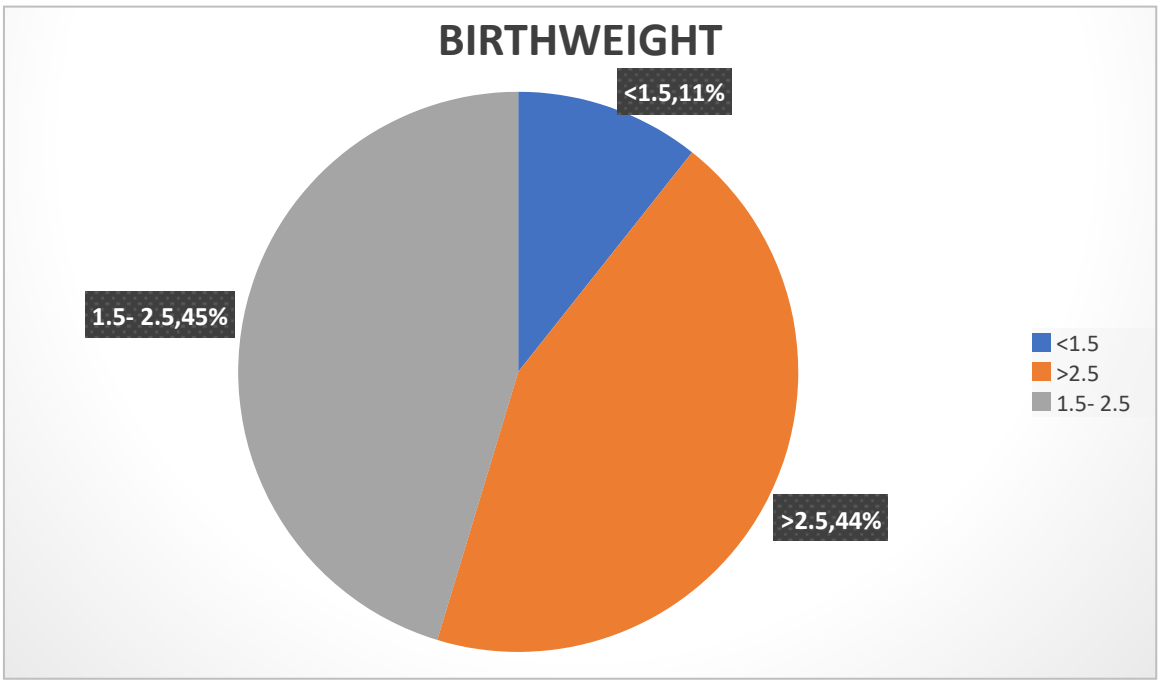



**FIGURE-8**

**TABLE- 8**

**DISTRIBUTION OF CASES ACCORDING TO GESTATIONAL AGE**

| Gestational age | Number of cases | percentage |
|---|---|---|
| **<37weeks** | **29** | 38.66% |

| | | |
|---|---|---|
| **>37weeks** | **46** | 61.33% |
| **Total** | **75** | **100** |

The analysis shows that out of 84 cases, 29 (38.66%) were of < 37 weeks and 46 (61.33%) were of gestational age >37 weeks.

**Distribution of cases according to gestational age**

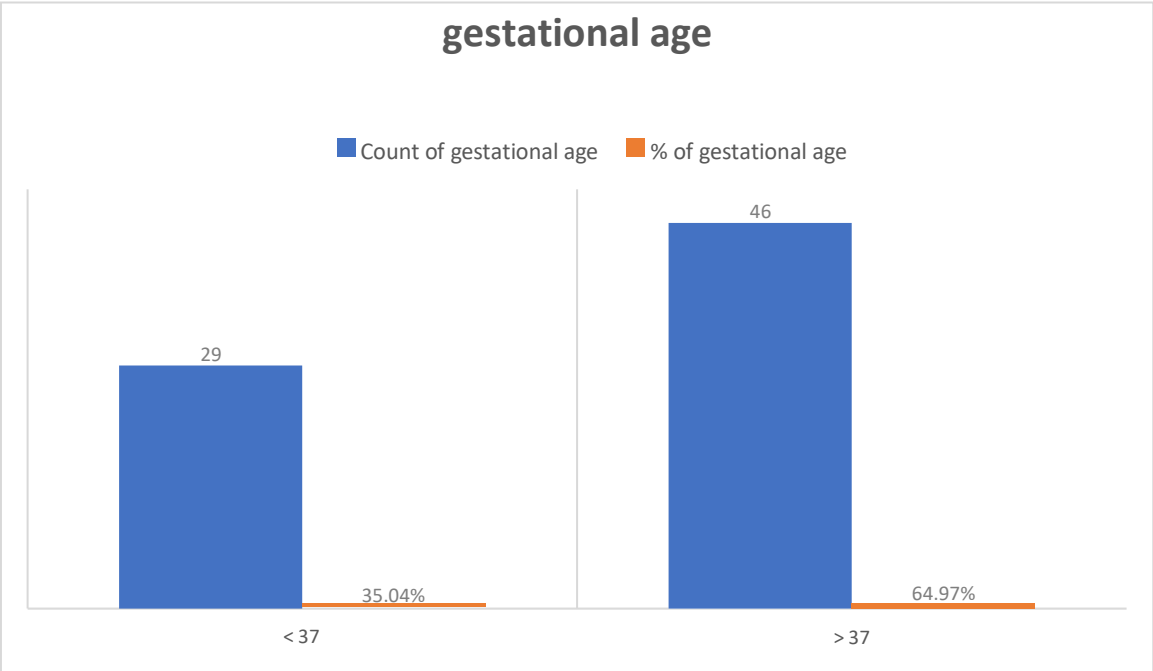



**FIGURE-9**

**TABLE- 9**

**Distribution of cases according to the mode of delivery**

| Mode of delivery | Number of cases | percentage |
|---|---|---|
| **NVD** | 15 | 20% |
| **LSCS** | 60 | 80% |
| **TOTAL** | **75** | **100** |

This table shows that 15 (20%) neonates are delivered by normal vaginal delivery and 60 (80%) were delivered by 78aesarean section.

**Distribution of cases according to the mode of delivery**

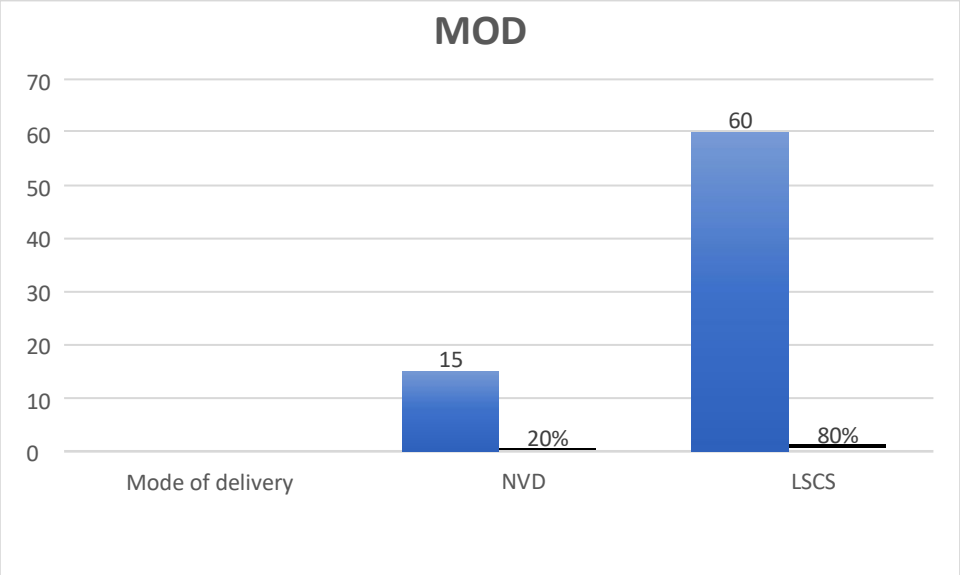



**FIGURE-10**

**TABLE- 10**

## DISTRIBUTION OF CASES ACCORDING TO TOTAL WBC COUNTS

| TOTAL WBC COUNTS | NUMBER OF CASES | PERCENTAGE |
|---|---|---|
| <5000 | 1 | 1.33% |
| 5000-20000 | 61 | 81.33% |
| >20000 | 13 | 17.33% |
| TOTAL | 75 | 100% |

This table shows that 1 (1.33%) case has a WBC<5000/cumm, 61 (81.33%) cases have a WBC count b/w 5000- 20000/cumm and 13 (17.33%) have a WBC count of >20000/cumm.

## DISTRIBUTION OF CASES ACCORDING TO TOTAL WBC COUNTS

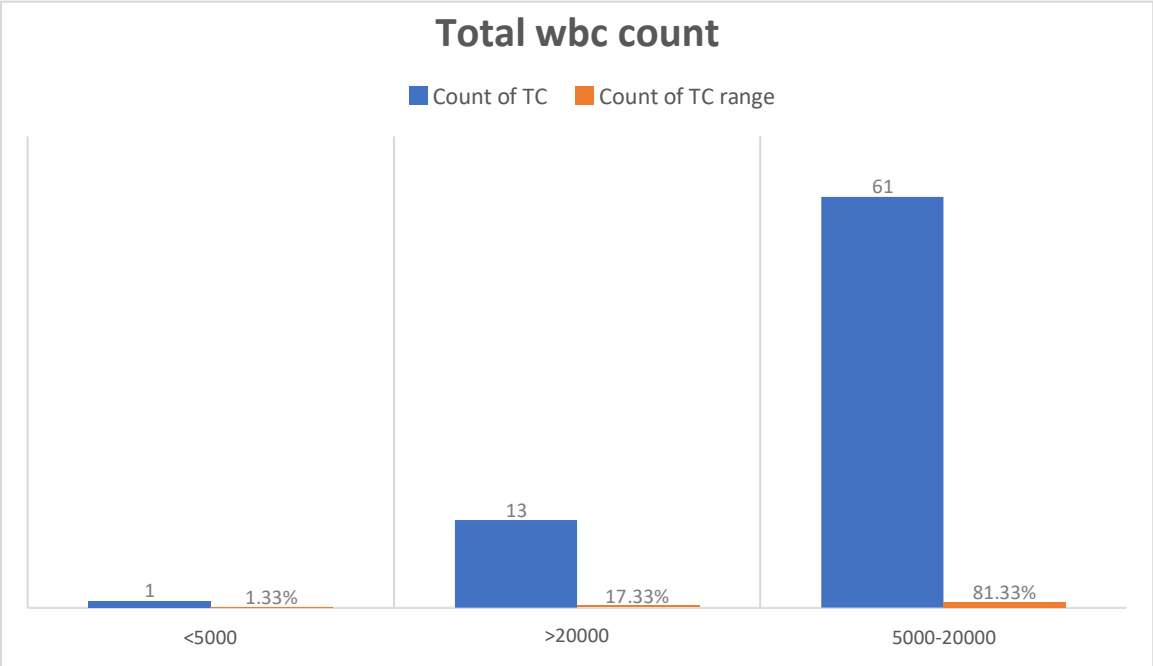



**FIGURE-11**

## TABLE- 11

## DISTRIBUTION OF CASES ACCORDING TO THE I/T RATIO

| I/T RATIO | NUMBER OF CASES | PERCENTAGE |
|---|---|---|
| <0.2 | **41** | **54.66%** |
| >0.2 | **34** | **45.335%** |
| **TOTAL** | **75** | **100%** |

This table shows that 41( 54.66%) cases have I/T ratio of <0.2 and 34(45.33%) cases have I/T ratio of >0.2.

**DISTRIBUTION OF CASES ACCORDING TO I/T RATIO**

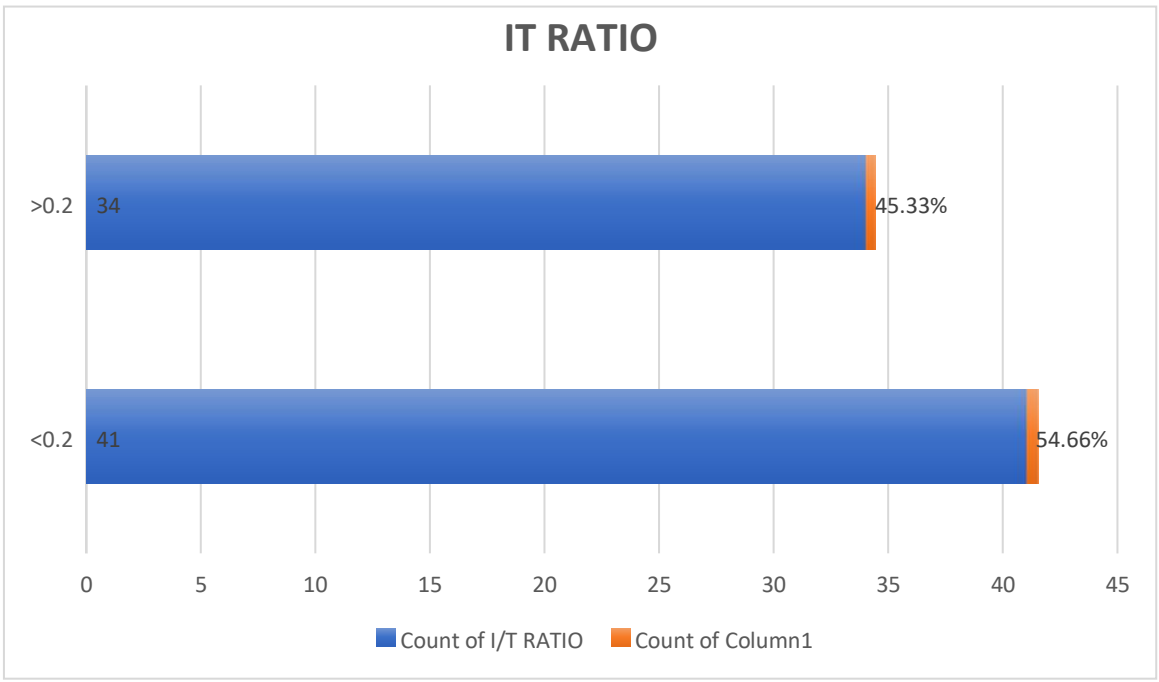



**FIGURE-12**

**TABLE- 12**

**DISTRIBUTION OF CASES ACCORDING TO ANC**

| ANC COUNT | NUMBER OF CASES | PERCENTAGE |
|---|---|---|

| | | |
|---|---|---|
| **<3500** | **13** | **17%** |
| **>3500** | **62** | **82%** |
| **TOTAL** | **75** | **100%** |

This table shows that 13(17%) cases have ANC count>3500 and 62(82%) cases have ANC count>3500.

**DISTRIBUTION OF CASES ACCORDING TO ANC**

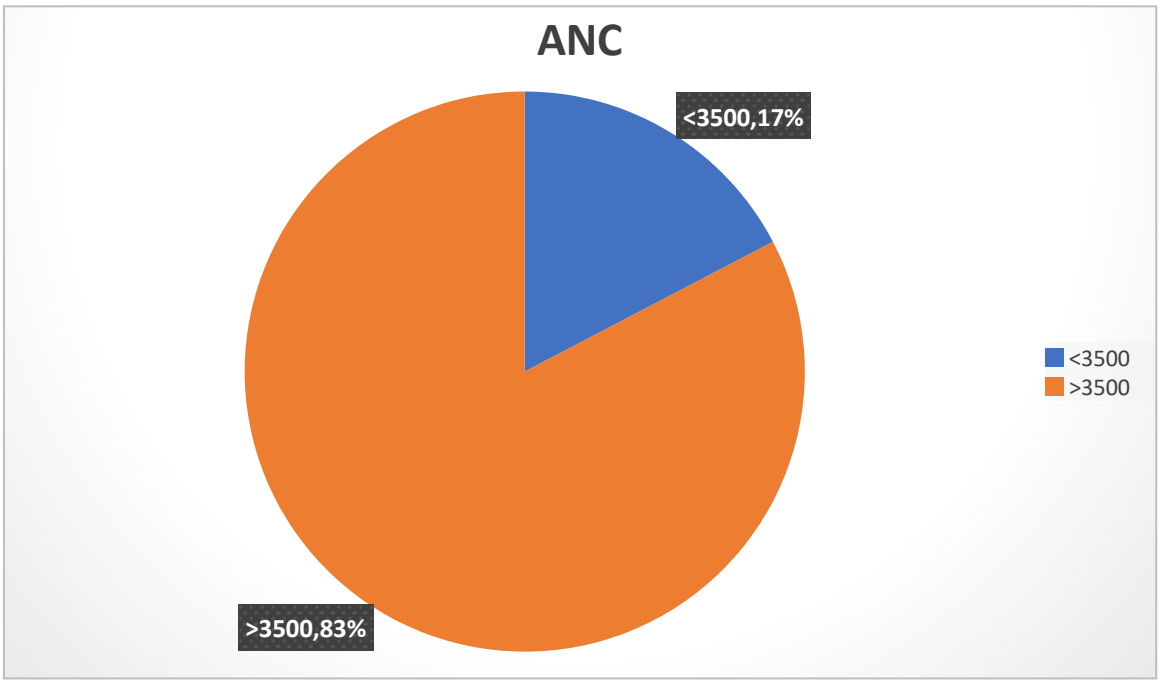



**FIGURE-13**

**TABLE- 13**

**DISTRIBUTION OF CASES ACCORDING TO PROCALCITONIN**

| PROCALCITONIN | NUMBER OF CASES | PERCENTAGE |
|---|---|---|

| | | |
|---|---|---|
| **<2.0** | **37** | **49%** |
| **>2.0** | **38** | **51%** |
| **TOTAL** | **75** | **100%** |

This table shows that 37(49%) cases have procalcitonin value less than 2.0 and 38(51%) cases have procalcitonin value more than 2.0

**DISTRIBUTION OF CASES ACCORDING TO PROCALCITONIN**

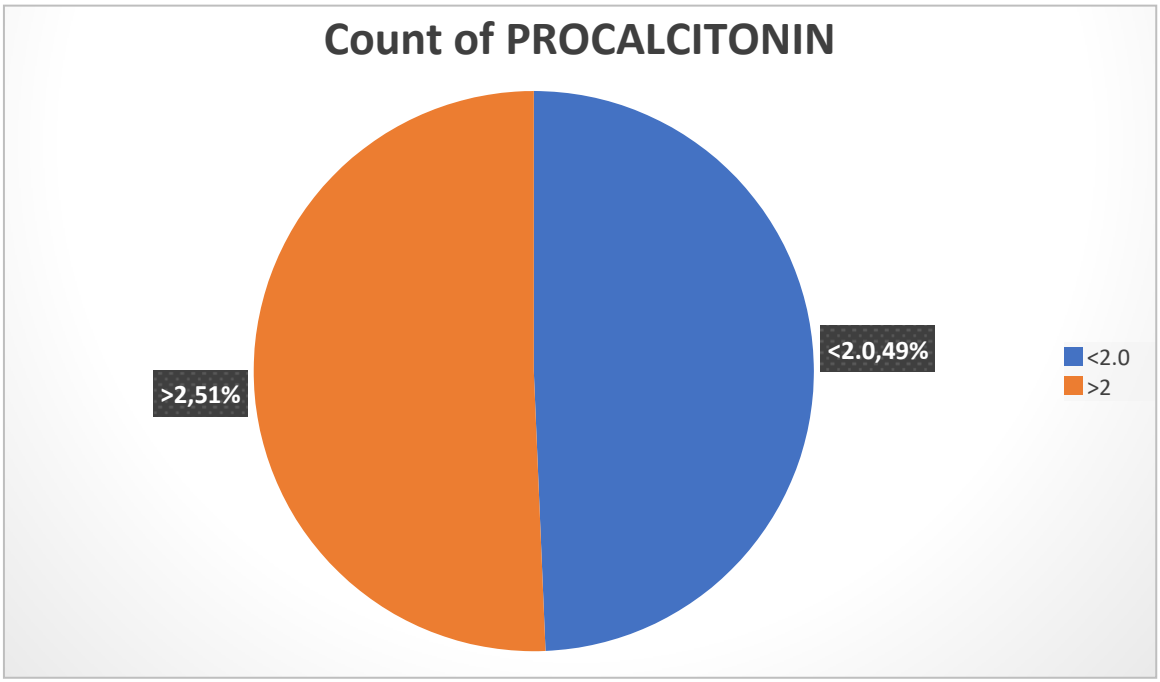



**FIGURE-14**

**TABLE-14**

**DISTRIBUTION OF CASES ACCORDING TO XRAY FINDINGS**

| X-RAY FINDINGS | NUMBER OF CASES | PERCENTAGE |
|---|---|---|

| | | |
|---|---|---|
| **NORMAL** | **34** | **45.33%** |
| **PNEUMONIA** | **9** | **12.00%** |
| **RDS** | **28** | **37.33%** |
| **TTNB** | **4** | **5.33%** |
| **TOTAL** | **75** | **100%** |

This table shows that 34(45.33%) cases have normal x-ray findings, 9(12.00%) cases have congenital pneumonia, 28(37.33%) cases have RDS and 4(5.33%) cases have TTNB.

**DISTRIBUTION OF CASES ACCORDING TO XRAY FINDINGS**

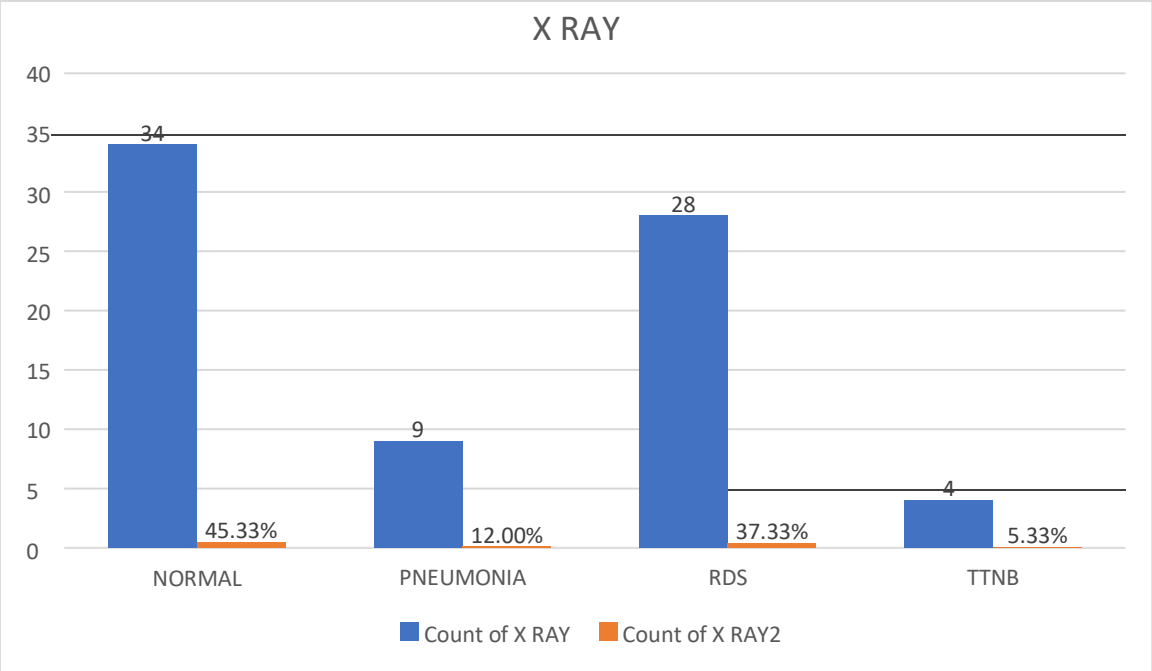



**FIGURE-15**

**TABLE- 15**

**DISTRIBUTION OF CASES ACCORDING TO VAGINAL SWAB**

| ORGANISM | NUMBER OF CASES | PERCENTAGE |
|---|---|---|
| **E.COLI** | **4** | **5.33%** |

| | | |
|---|---|---|
| **STAPH.AUREUS** | **4** | **5.33%** |
| **KLEBSIELLA** | **2** | **2.67%** |
| **STAPH.HOMINIS** | **2** | **2.67%** |
| **CANDIDA ALBICANS** | **2** | **2.67%** |
| **STAPH.EPIDERMIDIS** | **1** | **1.33%** |
| **STERILE** | **60** | **80%** |
| **TOTAL** | **75** | **100%** |

Vaginal swab culture and sensitivity was done in all cases with PROM. Out of 75 cases, 15 (22.67%) cases had growth on vaginal swab culture. Out of 75 cases E.coli was grown in 4(5.33%) cases, staphylococcus aureus growth was seen in 4(5.33%) cases, klebsiella was grown on 2(2.67%) cases, staphylococcus hominis growth was seen in 2(2.67%) cases, candida albicans growth was seen in 2(2.67%) cases, staphylococcus epidermidis growth was seen in 1(1.33%) cases.

**DISTRIBUTION OF CASES ACCORDING TO VAGINAL SWAB**

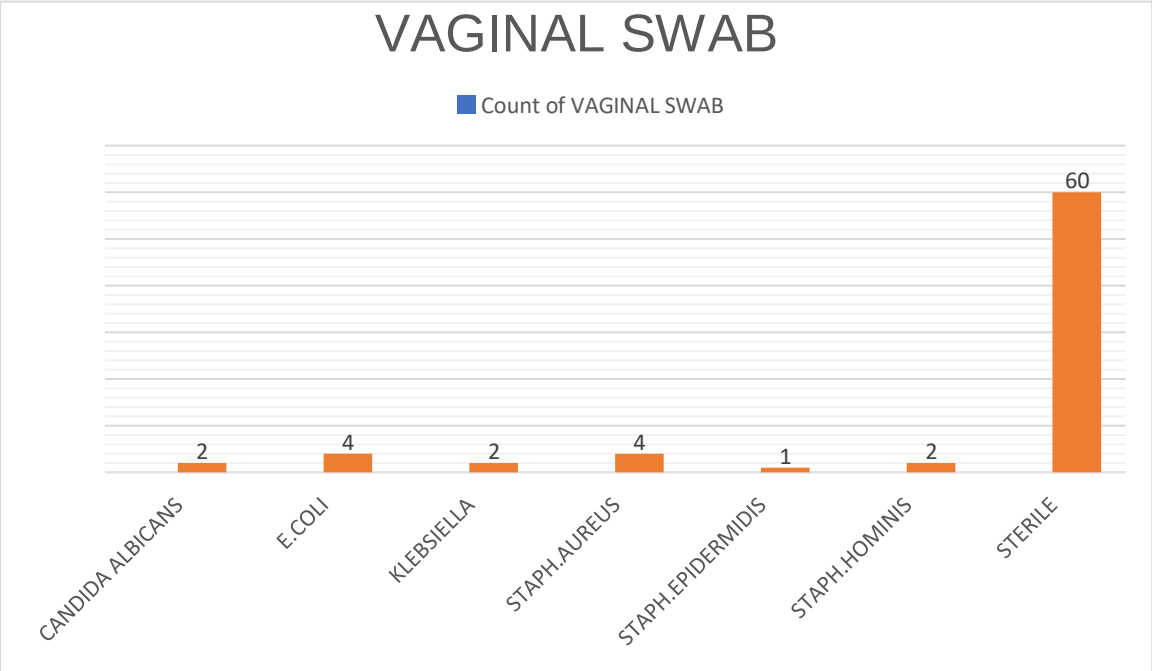



**FIGURE-16**

**TABLE-16**

**DISTRIBUTION OF CASES ACCORDING TO BLOOD CULTURE**

| BLOOD CULTURE | NUMBER OF CASES | PERCENTAGE |
|---|---|---|
| **KLEBSIELLA** | **6** | **8.00%** |
| **STAPH.AUREUS** | **3** | **4.00%** |

| | | |
|---|---|---|
| **E.COLI** | **1** | **1.33%** |
| **ENTEROCOCCUS** | **2** | **2.67%** |
| **STAPH.HOMINIS** | **1** | **1.33%** |
| **ACINETOBACTER** | **1** | **1.33%** |
| **STERILE** | **61** | **81.33%** |
| **TOTAL** | **75** | **100%** |

Blood culture and sensitivity were done in all neonates with a maternal history of PROM. Out of 75 cases, 14 (18.65%) cases had a growth on blood culture. Out of 75 cases, klebsiella was grown in 6(8.00%) cases, staphylococcus aureus growth was seen in 3(4.00%) cases, E.coli was grown in 1(1.33%) cases, enterococcus growth was seen in 2(2.67%) cases growth was seen in 1(1.33%) cases, staphylococcus hominis growth was seen in 1(1.33%) cases and Acinetobacter growth was seen in 1(1.33%) cases.

**DISTRIBUTION OF CASES ACCORDING TO BLOOD CULTURE**

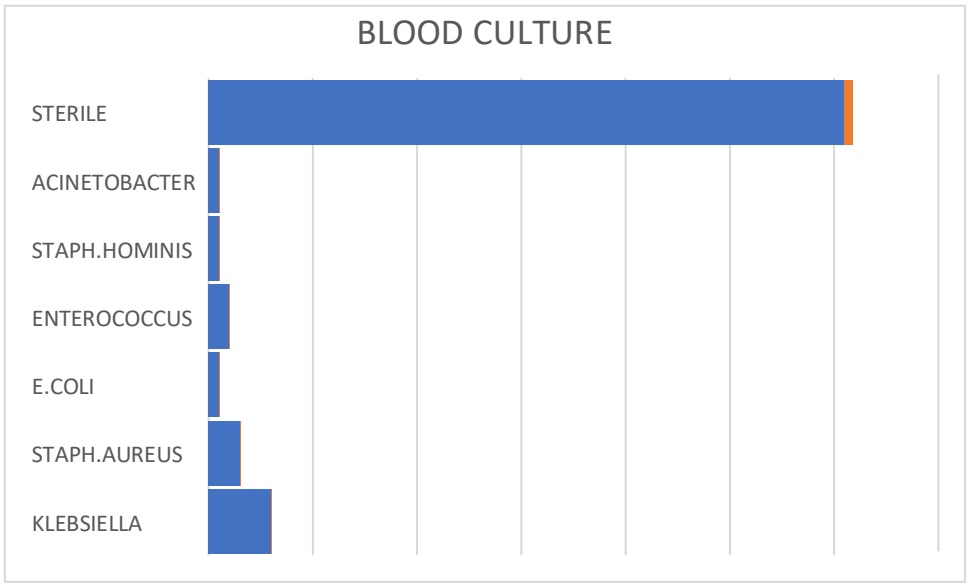



**FIGURE-17**

**TABLE-17**

**DISTRIBUTION OF CASES ACCORDING TO DURATION OF PROM**

| DURATION OF PROM | NUMBER OF CASES | PERCENTAGE |
|---|---|---|
| 12-18HOURS | 69 | 92.0% |
| >18HOURS | 6 | 8.00% |
| TOTAL | 75 | 100% |

This table shows that 69(92.0%) cases have a PROM duration of 12-18 hours and 6(8.00%) cases had PROM for more than 18 hours.

**DISTRIBUTION OF CASES ACCORDING TO DURATION OF PROM**

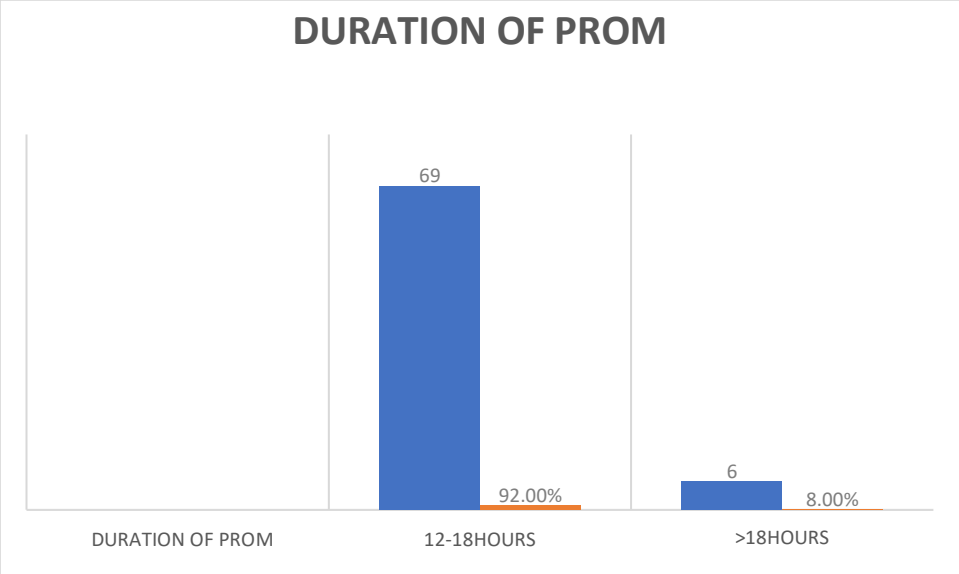



**FIGURE-18**

**TABLE- 18**

**DISTRIBUTION OF CASES ACCORDING TO CRP**

| CRP | NUMBER OF CASES | PERCENTAGE |
|---|---|---|
| <10.0 | 34 | 45% |
| >10.0 | 41 | 55% |
| TOTAL | 75 | 100% |

This table shows that 34(45%) cases had CRP value less than 10 and 41(55%) cases had CRP value more than 10.0

**DISTRIBUTION OF CASES ACCORDING TO CRP**

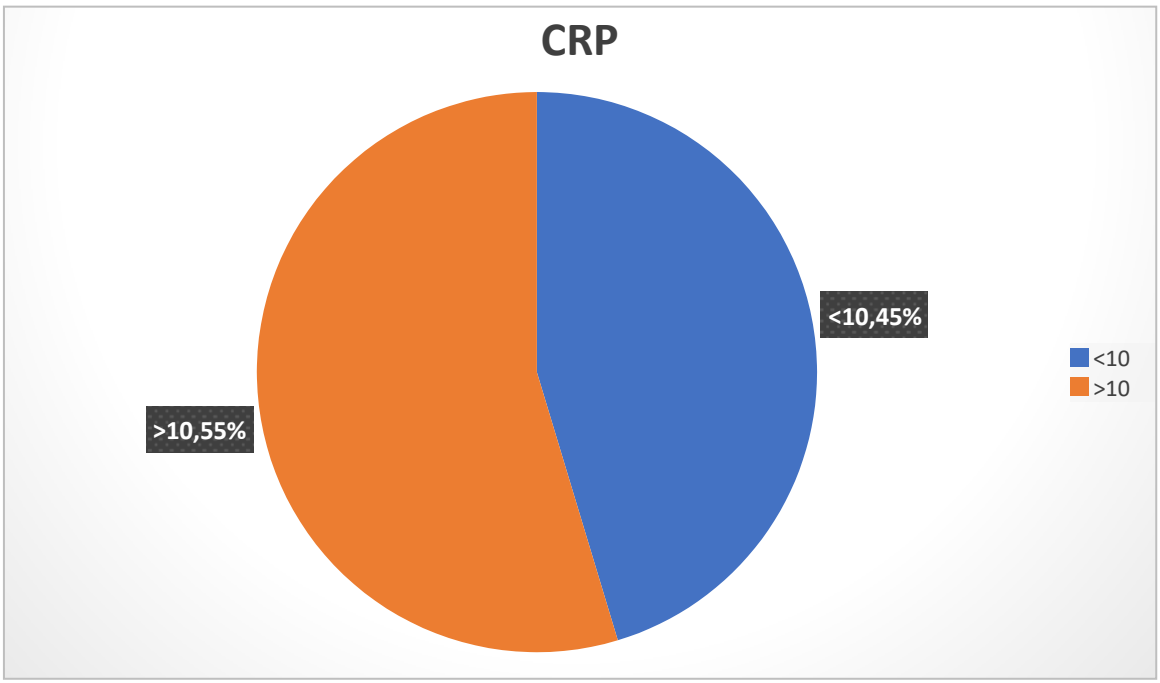



**FIGURE-19**

**TABLE-19**

## PROCALCITONIN AND SEPSIS CORRELATION

| | | | |
|---|---|---|---|
| | | | |
| | CLINICAL | CULTURE | NO |

| | | | | | |
|---|---|---|---|---|---|
| | | | SEPSIS | POSITIVE | SEPSIS |
| PROCALCITONIN | <2.0 | Count | 4 | 3 | 30 |
| | | % Within SEPSIS | 15.4% | 21.4% | 85.7% |
| | >2 | Count | 22 | 11 | 5 |
| | | % Within SEPSIS | 84.6% | 78.6% | 14.3% |
| Total | | Count | 26 | 14 | 35 |
| | | | | | |

## TABLE-20

| Chi-Square Tests | | | |
|---|---|---|---|
| | Value | df | Asymp. Sig. (2-sided) |
| Pearson Chi-Square | 34.883[a] | 2 | .000 |
| Likelihood Ratio | 38.378 | 2 | .000 |
| No of Cases | 75 | | |

## PROCALCITONIN AND SEPSIS CORRELATION

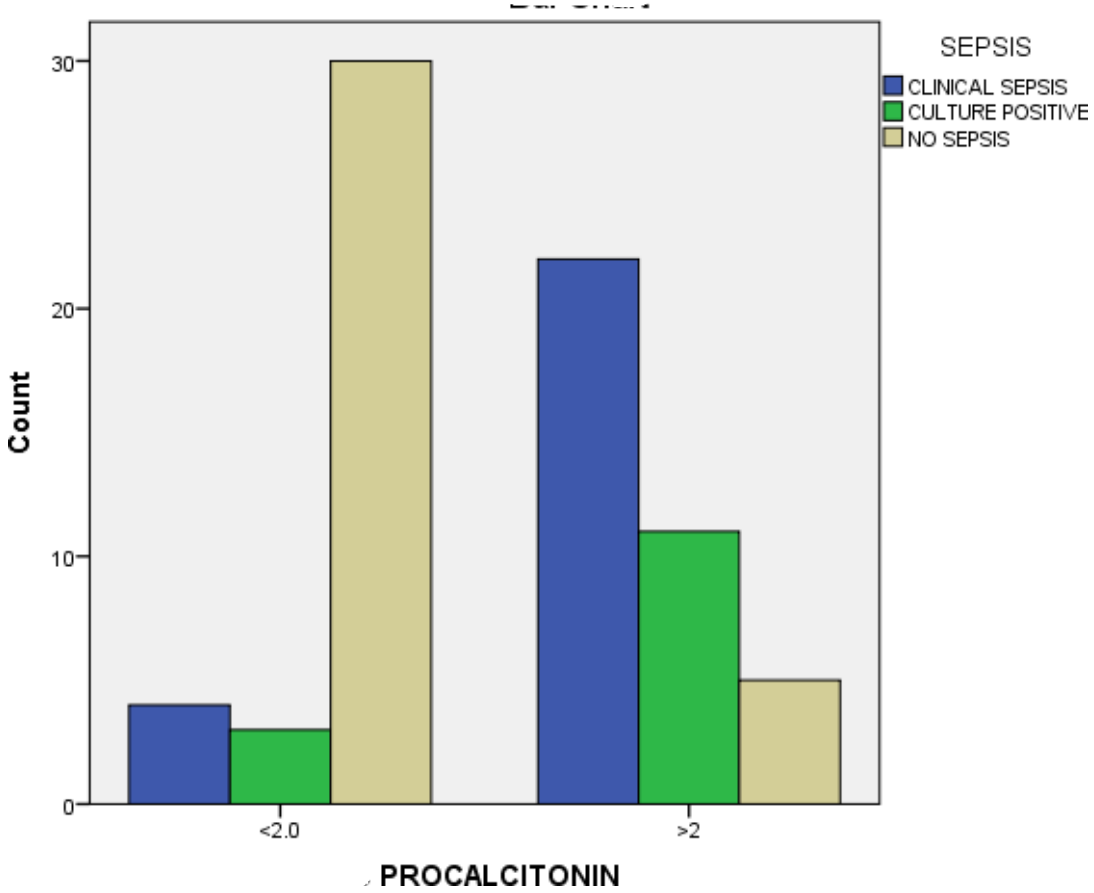



**FIGURE-20**

**TABLE-21**

## BLOOD CULTURE AND SEPSIS CORRELATION

| Chi-Square Tests | | | |
|---|---|---|---|
| | Value | df | Asymp. Sig. (2-sided) |
| Pearson Chi-Square | 75.000[a] | 14 | .000 |
| Likelihood Ratio | 72.203 | 14 | .000 |
| N of Cases | 75 | | |

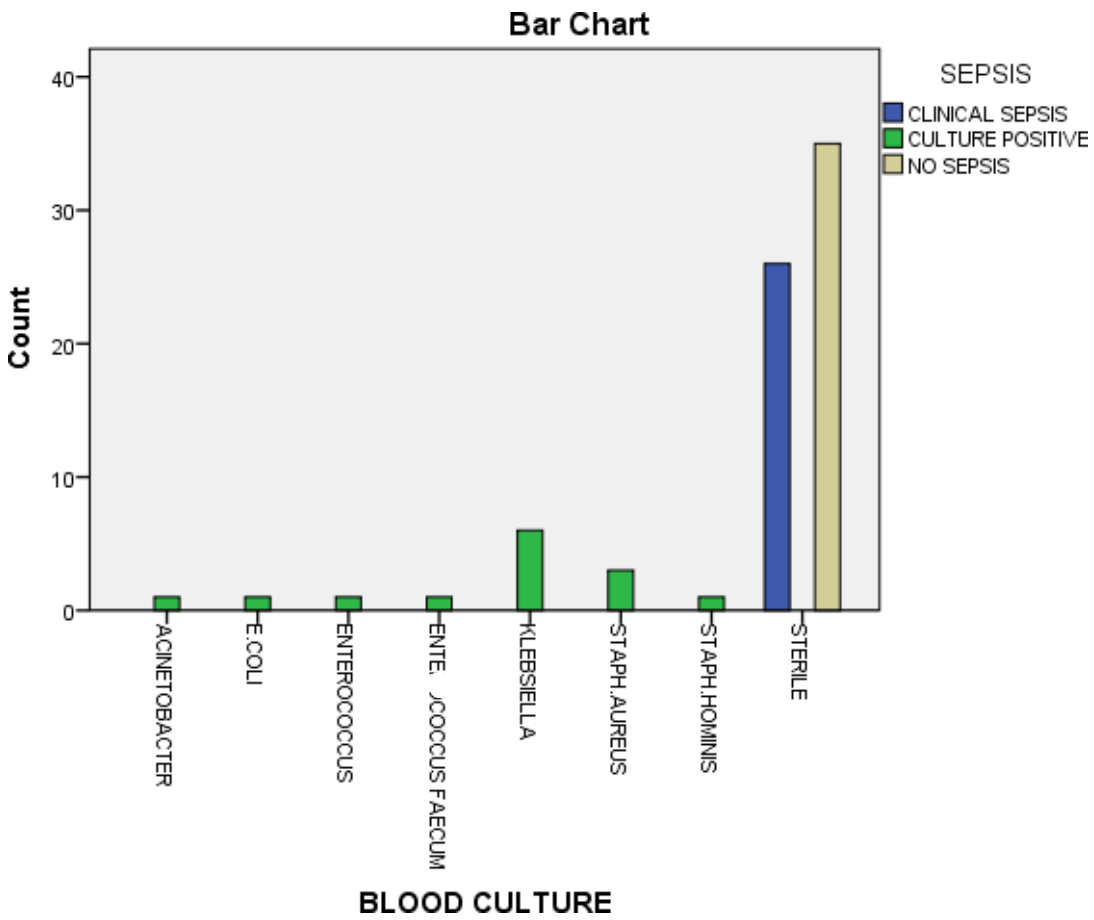


**FIGURE-21**

# TABLE-22

# CRP AND SEPSIS CORRELATION

| | | | SEPSIS | | |
|---|---|---|---|---|---|
| | | | CLINICAL SEPSIS | CULTURE POSITIVE | NO SEPSIS |
| CRP | <10 | COUNT | 0 | 3 | 31 |
| | | PERCENTAGE | 0.0% | 21.4% | 88.6% |
| | >10 | COUNT | 26 | 11 | 4 |
| | | PERCENTAGE | 100.0% | 78.6% | 11.4% |

| | | | | |
|---|---|---|---|---|
| Total | COUNT | 26 | 14 | 35 |
| | PERCENTAGE | 100.0% | 100.0% | 100.0% |

**TABLE-23**

| Chi-Square Tests | | | |
|---|---|---|---|
| | Value | df | Asymp. Sig. (2-sided) |
| Pearson Chi-Square | 51.193[a] | 2 | .000 |
| Likelihood Ratio | 63.893 | 2 | .000 |
| N of Cases | 75 | | |

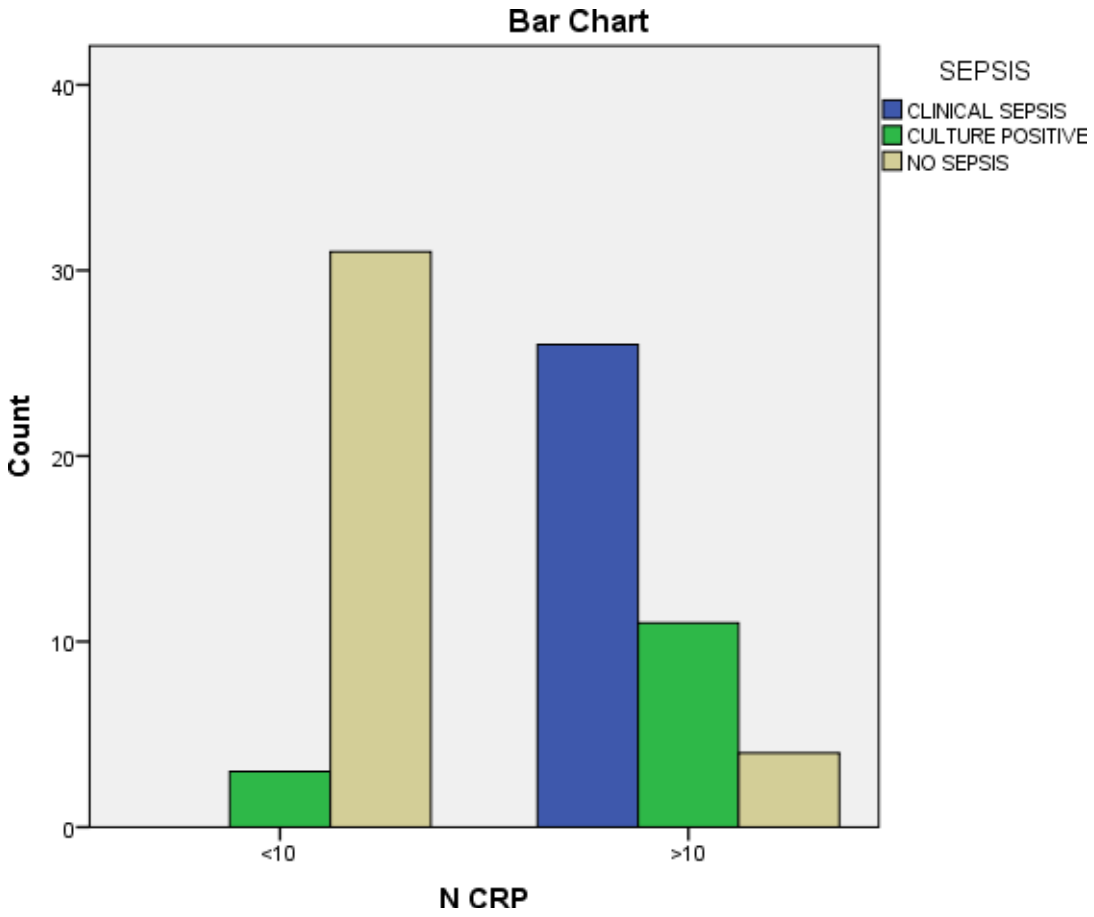



**FIGURE-22**

## TABLE-24

## IT RATIO AND SEPSIS CORRELATION

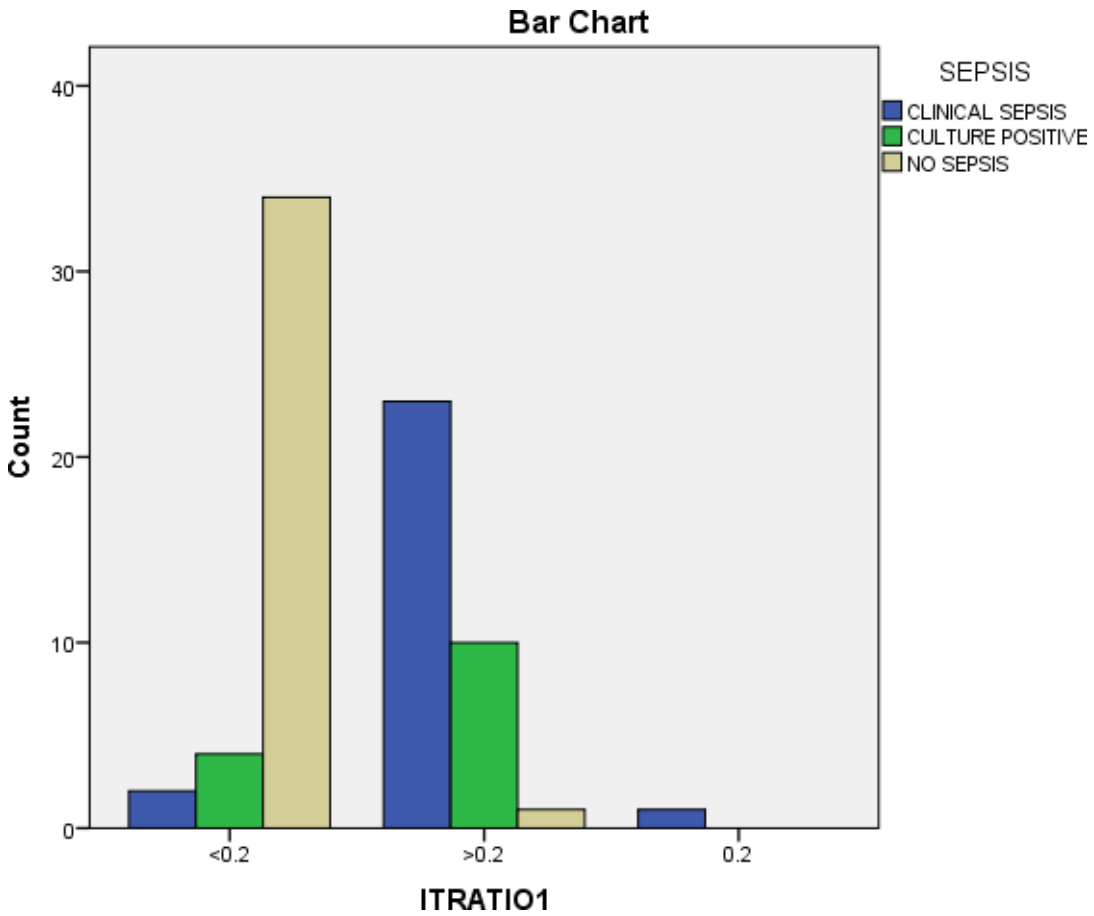
| | | | SEPSIS | | | Total |
|---|---|---|---|---|---|---|
| | | | CLINICAL SEPSIS | CULTURE POSITIVE | NO SEPSIS | |
| ITRATIO1 | <0.2 | Count | 3 | 4 | 34 | 41 |
| | | % | 11.5% | 28.6% | 97.1% | 54.6% |
| | >0.2 | Count | 23 | 10 | 1 | 34 |
| | | % | 88.5% | 71.4% | 2.9% | 45.3% |
| Total | | Count | 26 | 14 | 35 | 75 |
| | | Percentage | 100.0% | 100.0% | 100.0% | 100.0% |

**Chi-Square Tests**

| | Value | df | Asymp. Sig. (2-sided) |
|---|---|---|---|
| Pearson Chi-Square | 52.945[a] | 4 | .000 |
| Likelihood Ratio | 64.471 | 4 | .000 |
| N of Cases | 75 | | |

## FIGURE-23

## I/T RATIO AND SEPSIS




## DISCUSSION

Early onset neonatal sepsis is the main cause of mortality and morbidity

in neonates born to mothers with a prom. This study is taken up to identify different outcomes and institute proper treatment for early-onset neonatal sepsis at the earliest. It is prospective observational study involving mothers with PROM and babies born to mothers with PROM will be studied in a span of one and half year.

In this study out of 75 neonates born to PROM mothers, 29 (38.66%) were preterm and 46 (61.33%) were term. Similar findings were seen in a study by Ramesh TV et al where term neonates were 62%, and in a study by Alam et al where term neonates were 72%.[133,134]

In a study by Al- Qaqa et al14 where 38% were term and 62% were preterm, and in a study by Ocviyanti et al15 where 54.1% were preterm and 45.9% were term neonates.[135,136]

The incidence of PROM >18 hrs duration in mothers in our hospital was 8.00%. The incidence rate of 1.3% in a study by Idrisia et al, but lower than the 2.7% incidence rate in the study by Alam et al17, and the 4.2% incidence rate in the study by Sharma et al.[137]

In this study 37.33% of neonates had respiratory distress, 12.00% had pneumonia, 18.65% had culture-proven sepsis and 5.33% had TTNB. In a study by Thayi et al18 where 16.5% of neonates had respiratory distress and 11.1% had sepsis, in a study by Patil et al where 26% of neonates had respiratory distress, 14% had sepsis, 4% had an interventricular hemorrhage, and contrary finding were seen in a study by Chaangte et al12 where 2% had sepsis, 2% had

birth asphyxia and 1% had respiratory distress and this could be attributed to only term neonates being included in their study.[138]

Blood culture positivity is somewhat lower in studies compared to 18.65 percent according to this study. Positive blood culture results from the kayange et al research [26] was observed in 57 of the 121 cases of clinical sepsis, and in Eman M. Rabie et al. research [27], over 40.7% of patients with clinical sepsis had positive blood cultures. The current study's low blood culture positive could be caused by the comparatively lower sample size to the other two investigations. Similar to other things, researches conducted in Uganda and Bangladesh[139].

Klebsiella was the most prevalent isolate in this study (8.00 percent), then Staphylococcus aureus (4.00 percent ). Similar findings appeared in NNPD-2002-2003, where staph aureus and klebsiella were the two most prevalent isolates. In Shashikala conducted an another study in 242 neonates where klebsiella was the most prevalent isolate.[140]

Ramesh TV et alstudy .'s (15) found that 38% of newborns born to with PROM mothers had RDS (37%) and were preterm. Riyami NA et al.(16), according to their research, RDS (79%) is the most frequent newborn complication then sepsis (50% ). These variations may result from several management procedures and the interval between the beginning of the rupture of the delivery membranes.[141]

The most prevalent cervical pathogens were staphylococcus aureus and

Escherichia coli growth seen in our study is comparable to that seen in investigations by Ramesh et al. and Surayapalem et al., isolated E. coli (19 %), STAPH.AUREUS(11 percent), Klebsiella pneumoniae and CONS Contributing 7 percent each, Citrobacter and group B streptococcus 2% each.12 Ramesh et al., isolated E. coli(22%), Staphylococcus accounts for 20%, Klebsiella for 12%, and Pseudomonas for 8% of cases. In our study, E.coli (5.33%) Staphylococcus aureus (5.33%),candida(2.67percent), klebsiella(2.67%)and staphylococcus hominis (2.67%) were accounting 20% of the subjects. Genital tract culture positive was found in 20% of cases. Out of which E.Coli and staphylococcus aureus was the most common organism. Habeebullah and Baswaraj also in their study found E.Coli as the most common organism isolated from genital tract.[142]

In this study, 34.7% had clinical sepsis and 18.7% had culture-positive sepsis, similar results were found in a study conducted by Thayi S et al18 where clinical sepsis was observed in 30% of cases and culture-positive sepsis was observed in 11% of neonates. In a study by Nili et al4 clinical sepsis was observed in 20.2% of cases and culture-positive sepsis was observed in 5.5% of cases. These lower numbers could be attributed to the selection criteria of cases, they have included the duration of PROM from 1 hour.[143]

# SUMMARY

The prospective study includes 75 cases of neonates born to mothers with

PROM delivered in BLDE medical college, hospital, and research center, Vijayapura from January 2021 to July 2022.

- In this study, the sensitivity and specificity of CRP are 92.11% and 83.78% respectively.

- Procalcitonin has a significant association as early marker of sepsis in this study with p value<0.0001.

• 62.67% were males and 37.33% were females.

• 21% of the total neonates were born by normal vaginal delivery and 79% were delivered by cesarean section.

•26.54% of the cases had Premature rupture of membranes of <12 hrs duration, 54.88% of cases had Premature rupture of membranes of 12 to 24 hrs and 18.58 % of cases had Premature rupture of membranes of more than 24hrs.

• Most common organism isolated in maternal genitalia by cervical swab culture was E. coli (5.33%) and Staphylococcus aureus (5.33%), candida Albicans (2.67%), Klebsiella (2.67%), staphylococcus hominis (2.67%) and staphylococcus epidermidis (1.33%).

• RDS was the most common clinical manifestation (37.33%) followed by pneumonia 12.00%.

• The incidence of septicaemia was more in Premature rupture of membranes of longer duration.

• The incidence of neonatal sepsis in neonates born to mothers with PROM was clinical sepsis(28.0%) and culture-positive sepsis(17.33%).

• CRP was positive in 55% of cases.

- CRP had a positive correlation with sepsis in the present study (p-value - 0.001).

• Out of 75 cases 1.33% had leukopenia and 17.33% had leucocytosis.

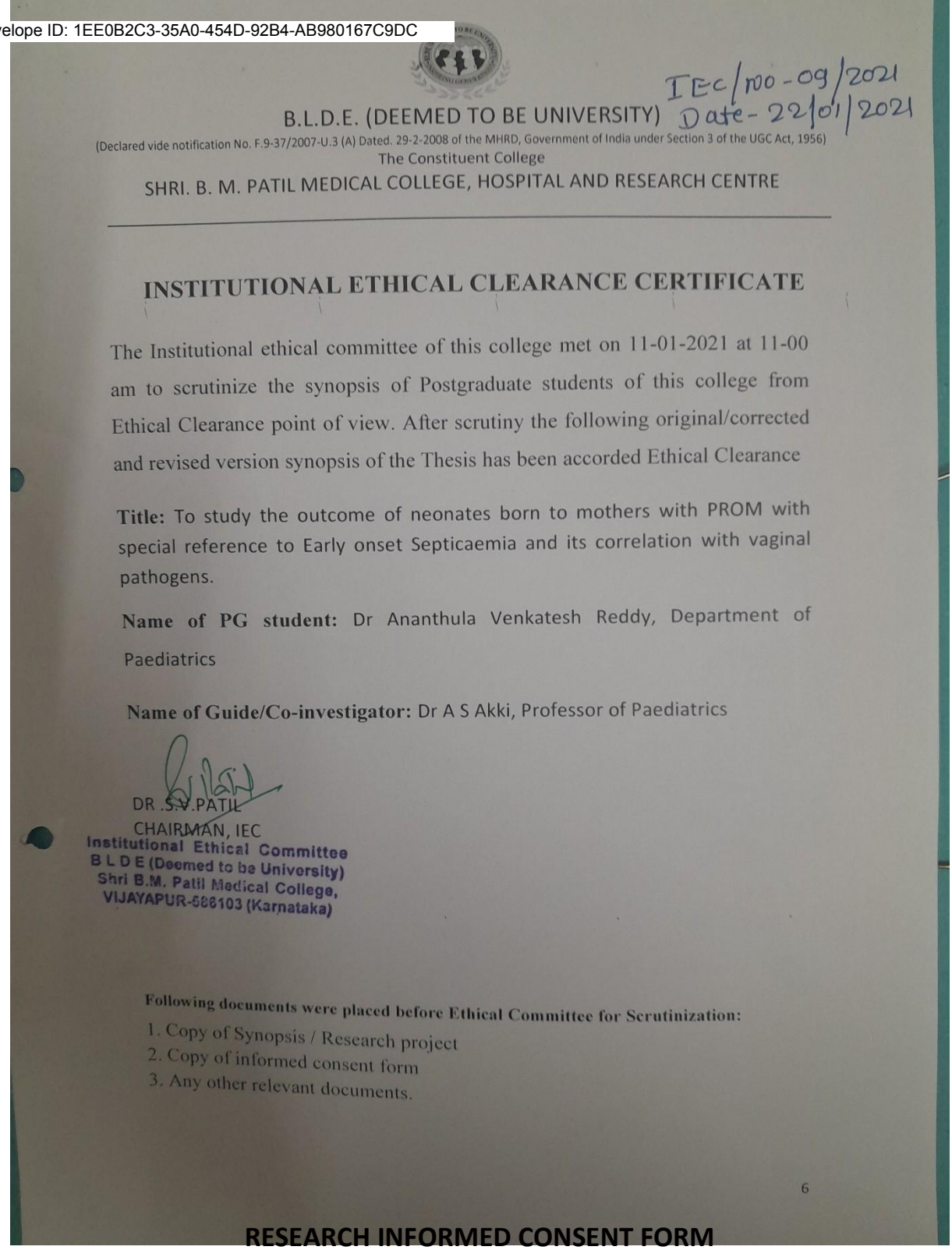
- Most common organisms isolated in blood culture were klebsiella followed by staphylococcus aureus, E.coli, Acinetobacter, and Enterococcus.

 In this study, 22(84.69%) cases had clinical sepsis, 11(76.8%) cases had culture positive sepsis and 5(14.3%) cases had no sepsis with a procalcitonin value >0.2ng/ml.

# CONCLUSION

- There is a positive correlation between procalcitonin and sepsis in neonates(p-value – 0.0001).
- Procalcitonin can be used an early marker of neonatal sepsis.
- In this study I/T ratio had a positive correlation with sepsis (p-value - 0.0011).
- I/T Ratio can be used an early marker for neonatal sepsis.
- In the present study, there is a positive correlation between blood culture and sepsis in neonates (p-value- 0.0001).
- There was a correlation between organisms isolated from the maternal genital tract and the baby's blood.
- Antibiotics can be started early in neonates by observing the sensitivity pattern in maternal vaginal swabs to prevent early onset sepsis in neonates.

**ETHICAL CLEARANCE CERTIFICATE**



B.L.D.E. (DEEMED TO BE UNIVERSITY)

IEC/no-09/2021
Date-22/01/2021

(Declared vide notification No. F.9-37/2007-U.3 (A) Dated. 29-2-2008 of the MHRD, Government of India under Section 3 of the UGC Act, 1956)

The Constituent College

SHRI. B. M. PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE

## INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Institutional ethical committee of this college met on 11-01-2021 at 11-00 am to scrutinize the synopsis of Postgraduate students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected and revised version synopsis of the Thesis has been accorded Ethical Clearance

**Title:** To study the outcome of neonates born to mothers with PROM with special reference to Early onset Septicaemia and its correlation with vaginal pathogens.

**Name of PG student:** Dr Ananthula Venkatesh Reddy, Department of Paediatrics

**Name of Guide/Co-investigator:** Dr A S Akki, Professor of Paediatrics

DR .S.V.PATIL
CHAIRMAN, IEC
Institutional Ethical Committee
B L D E (Deemed to be University)
Shri B.M. Patil Medical College,
VIJAYAPUR-586103 (Karnataka)

**Following documents were placed before Ethical Committee for Scrutinization:**

1. Copy of Synopsis / Research project
2. Copy of informed consent form
3. Any other relevant documents.

6

## RESEARCH INFORMED CONSENT FORM

**BLDE (DEEMED TO BE UNIVERSITY)**

**Shri B.M. PATIL Medical College, Hospital & Research Centre,**

**Vijayapur-586103.**

**TITLE OF THE PROJECT: "To study the outcome of neonates born to**

**mothers with PROM with special reference to early onset septicemia and its correlation with vaginal pathogens in mothers with PROM."**

**GUIDE** : A.S.AKKI MD

PROFESSOR

DEPARTMENT OF PEDIATRICS.

PG STUDENT : ANANTHULA VENKATESH REDDY

**PURPOSE OF RESEARCH:**

I have been informed that the present study will help in assessing the outcome and incidence of early-onset sepsis following PROM more 12 hours of duration. The relation of vaginal pathogens in special reference to early onset sepsis in new-borns born to Mothers with PROM more than 12 hours.

**PROCEDURE:**

I understand that after having obtained a detailed clinical history, thorough clinical examination and vaginal swab and relevant investigations, a final follow up of the new-borns born to mothers with PROM and its outcome is planned.

RISK AND DISCOMFORTS: none

**BENEFITS**:

I understand that my participation in the study will have no direct benefit to me other than the potential benefit of the research and education.

**CONFIDENTIALITY:**

I understand that the medical information produced by this study will become

a part of hospital records and will be subject to confidentiality. Information of sensitive personal nature will not be part of the medical record, but will be stored in the investigations research file.

If the data are used for publication in the medical literature or for teaching purpose, no name will be used and other identifiers such as photographs will be used only with special written permission. I understand that I may see the photograph before giving the permission.

**REQUEST FOR MORE INFORMATION:**

I understand that I may ask more questions about the study at any time; DR ANANTHULA VENKATESH REDDY at the department of Paediatrics is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of the study, which might influence my continued participation. A copy of this consent form will be given to me to keep for careful reading.

**REFUSAL FOR WITHDRAWAL OF PARTICIPATION:**

I understand that my participation is voluntary and that I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice. I also understand that DR.A.S.AKKI may terminate my participation in the study after he has explained the reasons for doing so.

**INJURY STATEMENT:**

I understand that in the unlikely event of injury to my baby resulting directly from mother and baby's participation in this study, if such injury were reported promptly, the appropriate treatment would be available to the baby. But, no further compensation would be provided by the hospital. I understand that by

my agreements to participate in this study and not waiving any of my legal rights.

I have explained to_____the purpose of the research, the procedures required and the possible risks to the best of my ability.

_____ _____

DR.ANANTHULA VENKATESH REDDY Date

(Investigator)

**PARENTS / GUARDIAN CONSENT STATEMENT:**

We confirm that DR.ANANTHULA VENKATESH REDDY is doing a study on "TO STUDY THE OUTCOME OF NEONATES BORN TO MOTHERS WITH PROM WITH SPECIAL REFERENCE TO EARLY ONSET SEPTICAEMIA AND ITS CORRELATION WITH VAGINAL PATHOGENS IN MOTHERS WITH PROM." DR.ANANTHULA VENKATESH REDDY has explained to us the purpose of research and the study procedure. We are willing to give as much as information required for the study and consent for investigations and the possible discomforts as well as benefits. We have been explained all the above in detail in our own language and we understand the same. Therefore we agree to give consent for baby's participate as a subject in this research project.

_____ _____

( Parents / Guardian) Date

_____ _____

(Witness to signature) Date

## PROFORMA

### 1. MATERNAL HISTORY

**Name: B/o IP No:**

**Age DOA**

**Parity DOD**

**2. ANTENATAL HISTORY :**

**DOC of the vaginal swab. :**

**Duration of PROM :**

**Date and time of delivery:**

**H/O multiple pregnancies. :**

**Previous history of PROM if any:**

**H/O any drug intake :**

**H/O any maternal UTI :**

**H/O previous PROM :**

**H/O prolonged rupture( >18hours) :**

**H/O maternal GBS colonisation :**

**3. BIRTH HISTORY :**

**M.O.D**

**If LSCS : emergency /elective Indication**

**APGAR 1min 5min**

**Mode of resuscitation**

**Risk factors**

**3. NEONATE :**

**Gestational age :**

**Sex :**

**Age in hours :**

**Birth weight :**

**Length :**

**Head circumference :**

**Cry - Colour - Activity - Tone -**

**Feeding-**

**4. VITALS :**

**HR- RR- CFT- SPO2-**

**SHOCK: present /absent**

**6. HEAD-TO-TOE EXAMINATION :**

**Head - AF- Face-**

**Ears -**

**Nose - oral cavity - neck-**

**Spine -**

**Orifice count -**

**Any obvious congenital anomalies or dysmorphic features :**

**7. SYSTEMIC EXAMINATION :**

**CVS :**

**RS :**

**P/A :**

**CNS :**

**10. DIAGNOSIS -**

**11. INVESTIGATIONS :**

**Maternal: vaginal swab and culture sensitivity**

**Neonate: CBC**

**CRP**

**BLOOD CULTURE**

**I/T RATIO**

**ANC**

**CHEST X RAY**

**Procalcitonin**

| SL. NO: | SEX | CBIRTH | BIRTH W | MATERN | CPRO | PROM(H | MOD | cg a | cgest | TC ra | TC | Colu | ANC | Column | I/T | N CRI | CRI | BLO | VAGINAL SWA | X RAY | C PROCAL | PR |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | FEMALE | >2.5 | 2.6 | 24 | <12 | 12 | NVD | > 37 | 38 | 5000-2 | 13040 | >3500 | 9388 | <0.2 | 0.1 | <10 | 5 | STERIL | STAPH.EPIDERMI | NORMAL | >2 | 2 |
| 2 | FEMALE | >2.5 | 2.9 | 20 | 12-18 | 14 | NVD | > 37 | 41 | >20000 | 26430 | >3500 | 20927 | >0.2 | 0.23 | >10 | 10.3 | STERIL | STAPH.AUREUS | NORMAL | >2 | 5 |
| 3 | MALE | 1.5- 2.5 | 2.3 | 24 | 12-18 | 13 | LSCS | < 37 | 36 | 5000-2 | 16880 | >3500 | 12322 | >0.2 | 0.3 | >10 | 12.2 | STERIL | STERILE | NORMAL | >2 | 2 |
| 4 | MALE | 1.5- 2.5 | 2.5 | 19 | <12 | 12 | NVD | > 37 | 40 | 5000-2 | 18750 | >3500 | 11625 | <0.2 | 0.12 | >10 | 20.5 | KLEBSI | STERILE | PNEUMONIA | >2 | |
| 5 | FEMALE | 1.5- 2.5 | 1.6 | 21 | 12-18 | 14 | LSCS | < 37 | 33 | 5000-2 | 18280 | >3500 | 13892 | <0.2 | 0.11 | <10 | 5 | STERIL | STAPH.HOMINIS | RDS | <2.0 | 0 |
| 6 | MALE | >2.5 | 2.8 | 33 | 12-18 | 16 | NVD | > 37 | 38 | 5000-2 | 13210 | >3500 | 7793 | >0.2 | 0.24 | >10 | 15.3 | STERIL | STERILE | TTNB | >2 | 2 |
| 7 | MALE | 1.5- 2.5 | 2 | 26 | <12 | 12 | NVD | < 37 | 36 | 5000-2 | 6070 | <3500 | 3459 | >0.2 | 0.29 | >10 | 25 | STERIL | STERILE | NORMAL | >2 | 4 |
| 8 | FEMALE | 1.5- 2.5 | 2.2 | 28 | <12 | 12 | NVD | > 37 | 40 | 5000-2 | 10240 | >3500 | 5222 | <0.2 | 0.14 | <10 | 5.2 | ACINE | STERILE | NORMAL | <2.0 | 0 |
| 9 | MALE | 1.5- 2.5 | 1.6 | 30 | <12 | 12 | LSCS | > 37 | 38 | 5000-2 | 9710 | <3500 | 3034 | <0.2 | 0.11 | >10 | 11.4 | STERIL | STERILE | RDS | >2 | 2 |
| 10 | MALE | >2.5 | 2.8 | 26 | 12-18 | 14 | LSCS | > 37 | 38 | 5000-2 | 15980 | >3500 | 8789 | <0.2 | 0.13 | <10 | 6.1 | STERIL | STERILE | RDS | <2.0 | 0 |
| 11 | FEMALE | 1.5- 2.5 | 2.27 | 24 | <12 | 12 | LSCS | > 37 | 39 | >20000 | 23280 | >3500 | 18158 | <0.2 | 0.1 | <10 | 5.8 | STERIL | STERILE | NORMAL | <2.0 | 1 |
| 12 | FEMALE | >2.5 | 2.8 | 25 | <12 | 12 | LSCS | > 37 | 39 | 5000-2 | 11640 | >3500 | 6751 | <0.2 | 0.13 | <10 | 6.3 | STERIL | STERILE | NORMAL | <2.0 | 0 |
| 13 | MALE | >2.5 | 2.8 | 22 | 12-18 | 14 | LSCS | > 37 | 38 | 5000-2 | 6990 | >3500 | 6962 | <0.2 | 0.16 | <10 | 5 | STERIL | STAPH.HOMINIS | PNEUMONIA | <2.0 | 0 |
| 14 | MALE | 1.5- 2.5 | 2.1 | 20 | <12 | 12 | LSCS | > 37 | 39 | >20000 | 29640 | >3500 | 18233 | <0.2 | 0.12 | <10 | 5 | STERIL | STERILE | NORMAL | <2.0 | 1 |
| 15 | MALE | >2.5 | 3.2 | 25 | 12-18 | 14 | LSCS | > 37 | 40 | >20000 | 20420 | >3500 | 14240 | >0.2 | 0.31 | >10 | 14.7 | STERIL | STERILE | TTNB | >2 | 3 |
| 16 | MALE | >2.5 | 3 | 28 | 12-18 | 15 | LSCS | > 37 | 38 | >20000 | 20170 | >3500 | 15665 | <0.2 | 0.18 | <10 | 6 | STAPH | STERILE | NORMAL | <2.0 | 0 |
| 17 | MALE | >2.5 | 2.9 | 25 | 12-18 | 18 | LSCS | > 37 | 39 | 5000-2 | 17310 | >3500 | 11251 | >0.2 | 0.21 | >10 | 11.9 | E.COLI | STERILE | NORMAL | <2.0 | 1 |
| 18 | MALE | >2.5 | 2.8 | 25 | <12 | 10 | LSCS | > 37 | 38 | 5000-2 | 11948 | >3500 | 9790 | <0.2 | 0.15 | <10 | 7.3 | STERIL | STERILE | RDS | <2.0 | 0 |
| 19 | MALE | 1.5- 2.5 | 1.8 | 22 | <12 | 12 | LSCS | < 37 | 30 | 5000-2 | 10820 | >3500 | 6275 | >0.2 | 0.27 | >10 | 14.6 | STERIL | STERILE | RDS | >2 | 2 |
| 20 | FEMALE | >2.5 | 2.6 | 30 | 12-18 | 16 | LSCS | < 37 | 30 | 5000-2 | 9876 | >3500 | 5678 | <0.2 | 0.13 | >10 | 14.6 | STERIL | STERILE | NORMAL | >2 | 2 |
| 21 | MALE | >2.5 | 3.6 | 24 | <12 | 12 | LSCS | > 37 | 39 | 5000-2 | 10470 | >3500 | 4502 | <0.2 | 0.15 | <10 | 8.2 | STERIL | STERILE | NORMAL | <2.0 | 0 |
| 22 | MALE | <1.5 | 1.3 | 25 | 12-18 | 14 | LSCS | > 37 | 37 | 5000-2 | 8190 | >3500 | 5569 | >0.2 | 0.22 | >10 | 44.2 | STERIL | E.COLI | RDS | >2 | |
| 23 | FEMALE | >2.5 | 2.7 | 20 | 12-18 | 14 | LSCS | > 37 | 38 | 5000-2 | 13490 | >3500 | 8228 | <0.2 | 0.18 | <10 | 9 | STERIL | STERILE | NORMAL | <2.0 | 0 |
| 24 | FEMALE | >2.5 | 2.6 | 26 | 12-18 | 16 | LSCS | > 37 | 37 | 5000-2 | 12440 | >3500 | 9578 | <0.2 | 0.16 | >10 | 14.4 | STERIL | STERILE | NORMAL | >2 | 2 |
| 25 | FEMALE | 1.5- 2.5 | 2 | 27 | 12-18 | 17 | LSCS | > 37 | 38 | 5000-2 | 10800 | >3500 | 66636 | >0.2 | 0.23 | >10 | 20.4 | STAPH. | STERILE | RDS | >2 | 2 |
| 26 | FEMALE | <1.5 | 1.5 | 30 | 12-18 | 18 | LSCS | < 37 | 30 | 5000-2 | 7760 | >3500 | 5199 | <0.2 | 0.11 | <10 | 9.8 | STERIL | STERILE | RDS | <2.0 | 0 |
| 27 | FEMALE | 1.5- 2.5 | 2.2 | 39 | 12-18 | 16 | LSCS | > 37 | 39 | 5000-2 | 18941 | >3500 | 14461 | >0.2 | 0.37 | >10 | 297 | KLEBSI | STERILE | PNEUMONIA | >2 | |
| 28 | FEMALE | 1.5- 2.5 | 2.1 | 38 | 12-18 | 14 | LSCS | > 37 | 38 | 5000-2 | 16787 | >3500 | 6567 | >0.2 | 0.29 | >10 | 15.9 | STERIL | STERILE | TTNB | <2.0 | 1 |
| 29 | MALE | >2.5 | 3.8 | 39 | <12 | 12 | LSCS | > 37 | 39 | 5000-2 | 16060 | >3500 | 11884 | >0.2 | 0.3 | >10 | 31.9 | STERIL | STAPH.AUREUS | NORMAL | >2 | 2 |
| 30 | MALE | >2.5 | 3.04 | 41 | 12-18 | 14 | LSCS | > 37 | 38 | 5000-2 | 17220 | >3500 | 10332 | >0.2 | 0.27 | >10 | 25 | STERIL | STERILE | NORMAL | >2 | 2 |
| 31 | MALE | >2.5 | 2.6 | 33 | 12-18 | 13 | LSCS | < 37 | 33 | 5000-2 | 10400 | >3500 | 6552 | >0.2 | 0.24 | >10 | 14.6 | STERIL | CANDIDA ALBICA | RDS | >2 | 3 |
| 32 | MALE | >2.5 | 3.6 | 25 | <12 | 12 | LSCS | > 37 | 40 | 5000-2 | 14690 | >3500 | 10429 | >0.2 | 0.26 | >10 | 81.8 | STERIL | STERILE | PNEUMONIA | >2 | |
| 33 | FEMALE | 1.5- 2.5 | 2 | 30 | 12-18 | 14 | LSCS | < 37 | 36 | 5000-2 | 10660 | >3500 | 7995 | >0.2 | 0.21 | >10 | 36.4 | STERIL | STERILE | PNEUMONIA | >2 | |
| 34 | FEMALE | >2.5 | 2.54 | 21 | <12 | 12 | LSCS | > 37 | 39 | >20000 | 51560 | >3500 | 11047 | >0.2 | 0.6 | >10 | 27.6 | KLEBSI | STERILE | TTNB | >2 | 2 |
| 35 | MALE | <1.5 | 1.2 | 20 | 12-18 | 13 | LSCS | < 37 | 29 | 5000-2 | 12350 | >3500 | 8151 | >0.2 | 0.21 | >10 | 12 | STERIL | STERILE | RDS | <2.0 | 1 |
| 36 | FEMALE | 1.5- 2.5 | 2 | 18 | <12 | 12 | LSCS | < 37 | 30 | 5000-2 | 15410 | >3500 | 9554 | <0.2 | 0.12 | <10 | 9.6 | STERIL | STERILE | RDS | <2.0 | 0 |
| 37 | FEMALE | 1.5- 2.5 | 1.7 | 26 | <12 | 12 | LSCS | > 37 | 38 | 5000-2 | 12430 | >3500 | 8203 | <0.2 | 0.14 | <10 | 9.3 | STERIL | STERILE | NORMAL | <2.0 | 0 |
| 38 | MALE | >2.5 | 2.8 | 37 | 12-18 | 14 | LSCS | > 37 | 37 | 5000-2 | 13820 | <3500 | 2487 | <0.2 | 0.1 | <10 | 5 | STERIL | STERILE | NORMAL | <2.0 | 0 |
| 39 | MALE | >2.5 | 2.8 | 37 | <12 | 12 | LSCS | < 37 | 36 | >20000 | 76800 | >3500 | 4531 | <0.2 | 0.15 | <10 | 7.6 | STERIL | STERILE | NORMAL | <2.0 | 1 |
| 40 | MALE | 1.5- 2.5 | 2.1 | 37 | 12-18 | 14 | LSCS | > 37 | 38 | 5000-2 | 19330 | >3500 | 15744 | <0.2 | 0.18 | <10 | 5.8 | STERIL | STERILE | NORMAL | <2.0 | 1 |
| 41 | FEMALE | >2.5 | 2.9 | 27 | <12 | 12 | NVD | > 37 | 38 | 5000-2 | 12440 | >3500 | 9578 | <0.2 | 0.13 | <10 | 5 | STERIL | STERILE | NORMAL | <2.0 | 0 |
| 42 | MALE | <1.5 | 1.4 | 24 | 12-18 | 13 | NVD | > 37 | 39 | >20000 | 30300 | <3500 | 1333 | <0.2 | 0.17 | <10 | 5 | KLEBSI | E.COLI | RDS | >2 | |
| 43 | FEMALE | 1.5- 2.5 | 1.9 | 25 | <12 | 12 | LSCS | > 37 | 38 | >20000 | 72500 | >3500 | 4643 | <0.2 | 0.16 | <10 | 5.7 | STERIL | STERILE | RDS | <2.0 | 0 |
| 44 | FEMALE | <1.5 | 1.3 | 22 | 12-18 | 17 | LSCS | < 37 | 34 | 5000-2 | 8190 | >3500 | 5569 | >0.2 | 0.25 | >10 | 44 | STAPH | STERILE | RDS | >2 | |
| 45 | MALE | 1.5- 2.5 | 1.6 | 24 | 12-18 | 13 | NVD | < 37 | 34 | 5000-2 | 7270 | <3500 | 3136 | >0.2 | 0.22 | >10 | 19 | STERIL | E.COLI | RDS | >2 | |
| 46 | MALE | 1.5- 2.5 | 2.1 | 35 | 12-18 | 16 | LSCS | < 37 | 34 | >20000 | 71400 | <3500 | 2856 | >0.2 | 0.24 | >10 | 11.2 | STERIL | STERILE | RDS | >2 | 2 |
| 47 | FEMALE | 1.5- 2.5 | 2.4 | 28 | <12 | 12 | NVD | < 37 | 28 | 5000-2 | 8910 | <3500 | 2494 | 0.2 | 0.2 | >10 | 21 | STERIL | STERILE | RDS | >2 | |
| 48 | MALE | 1.5- 2.5 | 2.2 | 38 | 12-18 | 13 | LSCS | < 37 | 33 | 5000-2 | 17070 | >3500 | 10412 | >0.2 | 0.33 | >10 | 45 | STERIL | STERILE | RDS | <2.0 | 1. |
| 49 | MALE | <1.5 | 1.2 | 28 | >18 | 48 | LSCS | < 37 | 31 | 5000-2 | 9686 | <3500 | 2904 | >0.2 | 0.23 | >10 | 13.3 | STERIL | STERILE | PNEUMONIA | >2 | 2 |
| 50 | FEMALE | 1.5- 2.5 | 2 | 18 | 12-18 | 18 | LSCS | < 37 | 30 | >20000 | 51410 | >3500 | 9554 | <0.2 | 0.11 | <10 | 9.6 | STERIL | STAPH.AUREUS | RDS | <2.0 | 1 |
| 51 | FEMALE | >2.5 | 2.7 | 28 | 12-18 | 15 | LSCS | > 37 | 40 | 5000-2 | 13490 | >3500 | 8228 | <0.2 | 0.14 | <10 | 9 | STERIL | STERILE | NORMAL | <2.0 | 0 |
| 52 | MALE | >2.5 | 3.1 | 25 | <12 | 12 | LSCS | > 37 | 40 | 5000-2 | 15560 | >3500 | 11047 | >0.2 | 0.27 | >10 | 27.6 | STERIL | KLEBSIELLA | NORMAL | >2 | |
| 53 | MALE | >2.5 | 2.6 | 23 | 12-18 | 14 | LSCS | > 37 | 37 | 5000-2 | 14600 | >3500 | 11242 | >0.2 | 0.24 | >10 | 32.5 | STERIL | STERILE | NORMAL | >2 | |
| 54 | MALE | <1.5 | 1.5 | 23 | <12 | 12 | LSCS | < 37 | 35 | 5000-2 | 10460 | >3500 | 6694 | <0.2 | 0.19 | <10 | 5 | STERIL | STERILE | NORMAL | <2.0 | 0 |
| 55 | MALE | >2.5 | 3.4 | 26 | 12-18 | 16 | LSCS | > 37 | 38 | 5000-2 | 12890 | >3500 | 10183 | >0.2 | 0.22 | >10 | 14.3 | STERIL | STERILE | NORMAL | >2 | 4 |
| 56 | FEMALE | 1.5- 2.5 | 2 | 20 | 12-18 | 13 | NVD | > 37 | 39 | 5000-2 | 18450 | >3500 | 14461 | <0.2 | 0.18 | <10 | 5 | STERIL | STERILE | PNEUMONIA | >2 | |
| 57 | MALE | >2.5 | 3 | 30 | <12 | 12 | LSCS | > 37 | 39 | 5000-2 | 9680 | >3500 | 7840 | >0.2 | 0.21 | >10 | 12.2 | STERIL | STERILE | NORMAL | >2 | 2 |
| 58 | MALE | 1.5- 2.5 | 2.4 | 22 | 12-18 | 15 | NVD | < 37 | 35 | 5000-2 | 10610 | >3500 | 7427 | <0.2 | 0.1 | <10 | 5 | STERIL | STERILE | NORMAL | <2.0 | 0 |
| 59 | MALE | >2.5 | 2.8 | 24 | 12-18 | 18 | LSCS | > 37 | 40 | 5000-2 | 14050 | >3500 | 10116 | >0.2 | 0.26 | >10 | 29.6 | STAPH. | STERILE | PNEUMONIA | >2 | 2 |
| 60 | MALE | <1.5 | 1.5 | 30 | 12-18 | 14 | LSCS | < 37 | 30 | 5000-2 | 8570 | >3500 | 79500 | <0.2 | 0.16 | <10 | 9.8 | STERIL | STERILE | RDS | <2.0 | 0 |
| 61 | MALE | 1.5- 2.5 | 2.1 | 23 | >18 | 48 | LSCS | < 37 | 35 | 5000-2 | 13680 | >3500 | 8755 | <0.2 | 0.09 | >10 | 10.6 | STERIL | STERILE | RDS | <2.0 | 0 |
| 62 | MALE | >2.5 | 3.35 | 19 | 12-18 | 15 | LSCS | > 37 | 39 | 5000-2 | 8230 | >3500 | 6666 | >0.2 | 0.28 | >10 | 28.4 | KLEBSI | STERILE | NORMAL | >2 | 3 |
| 63 | FEMALE | 1.5- 2.5 | 1.7 | 28 | 12-18 | 16 | LSCS | > 37 | 37 | >20000 | 20650 | >3500 | 17759 | <0.2 | 0.19 | <10 | 5 | STERIL | STERILE | RDS | <2.0 | 1 |
| 64 | FEMALE | 1.5- 2.5 | 1.9 | 25 | <12 | 12 | LSCS | < 37 | 32 | 5000-2 | 7870 | >3500 | 4643 | <0.2 | 0.12 | <10 | 5.7 | STERIL | STAPH.AUREUS | RDS | <2.0 | 1 |
| 65 | FEMALE | 1.5- 2.5 | 1.7 | 28 | 12-18 | 14 | LSCS | > 37 | 37 | >20000 | 20650 | >3500 | 17759 | <0.2 | 0.18 | <10 | 5 | STERIL | STERILE | RDS | <2.0 | 0 |
| 66 | MALE | 1.5- 2.5 | 1.9 | 29 | >18 | 19 | LSCS | < 37 | 33 | 5000-2 | 8050 | <3500 | 2093 | <0.2 | 0.11 | <10 | 5 | STERIL | STERILE | RDS | <2.0 | 0 |
| 67 | MALE | >2.5 | 2.7 | 29 | 12-18 | 14 | NVD | > 37 | 38 | <5000 | 3650 | <3500 | 1642 | >0.2 | 0.3 | >10 | 128 | ENTER | KLEBSIELLA | PNEUMONIA | >2 | |
| 68 | MALE | >2.5 | 2.74 | 24 | <12 | 12 | LSCS | < 37 | 36 | 5000-2 | 7590 | >3500 | 4781 | <0.2 | 0.17 | <10 | 5 | STERIL | STERILE | NORMAL | <2.0 | 0 |
| 69 | MALE | >2.5 | 2.7 | 24 | 12-18 | 15 | NVD | > 37 | 39 | 5000-2 | 15050 | >3500 | 9030 | <0.2 | 0.13 | <10 | 5.7 | STERIL | STERILE | NORMAL | <2.0 | 0 |
| 70 | MALE | >2.5 | 2.6 | 23 | <12 | 12 | NVD | > 37 | 37 | 5000-2 | 17630 | >3500 | 12693 | <0.2 | 0.18 | <10 | 5 | STERIL | STERILE | NORMAL | <2.0 | 0 |
| 71 | MALE | 1.5- 2.5 | 2.4 | 22 | 12-18 | 16 | NVD | > 37 | 38 | 5000-2 | 7170 | <3500 | 2581 | <0.2 | 0.09 | >10 | 15 | STERIL | STERILE | NORMAL | >2 | 2 |