

**“MATERNAL AND PERINATAL OUTCOME IN CASES OF
PRETERM PREMATURE RUPTURE OF MEMBRANES
(pPROM) - A PROSPECTIVE STUDY”**

Dissertation submitted for

M.S., DEGREE EXAMINATION

M.S. OBSTETRICS AND GYNAECOLOGY

BRANCH II



**CHENGALPATTU MEDICAL COLLEGE,
CHENGALPATTU**

**THE TAMIL NADU DR.M.G.R. MEDICAL UNIVERISTY
GUINDY, CHENNAI – TAMILNADU**

MAY – 2018

CERTIFICATE

This is to certify that the dissertation titled “**MATERNAL AND PERINATAL OUTCOME IN CASES OF PRETERM PREMATURE RUPTURE OF MEMBRANES (pPROM) - A PROSPECTIVE STUDY**” is a bonafide work done by **Dr. A. DEVI** in **Chengalpattu Medical College**, during the academic year 2016 – 2018 under my direct supervision and guidance, submitted to the **Tamilnadu Dr. M.G.R. Medical University** in partial fulfilment of University regulation for **M.S degree Branch – II Obstetrics and Gynaecology** degree examination of the Tamilnadu Dr. M.G.R. Medical University.

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DECLARATION BY THE CANDIDATE

I, **Dr. A. DEVI**, solemnly declare that the dissertation titled **“MATERNAL AND PERINATAL OUTCOME IN CASES OF PRETERM PREMATURE RUPTURE OF MEMBRANES (pPROM) - A PROSPECTIVE STUDY”** has been prepared by me. I also declare that this bonafide work or a part of this work was not submitted by me or any other for any award, degree, diploma to any other university board in India or abroad. This is submitted to the **Tamilnadu Dr. M.G.R. Medical University**, Guindy, Chennai in partial fulfilment of the rules and regulation for the award of **M.S degree Branch – II Obstetrics and Gynaecology**.

Place: Chengalpattu

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I commence with the holy name of GOD benevolence and beneficence that enabled me to complete this research.

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" With modicum and civility this book is dedicated at the lotus feet of my parents"

INSTITUTIONAL ETHICAL COMMITTEE

CHENGALPATTU MEDICAL COLLEGE, CHENGALPATTU

Title of Work : **Maternal and Perinatal Outcome in Cases of Preterm Premature Rupture of Membrane (PPROM)- a Prospective Study**

Principal Investigator : Dr.A. Devi, MD.,

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The request for an approval From the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 25.10.2016 at the Medical Education Unit, Government Chengalpattu Medical College, Chengalpattu at 12.00 PM.

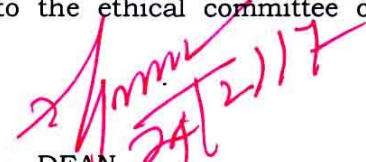
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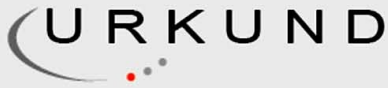
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| 9 | <ul style="list-style-type: none">• ANNEXURES• ABBREVIATIONS• BIBLIOGRAPHY• PROFORMA• INFORMATION SHEET• INFORMED CONSENT FORM• MASTER CHART | |

INTRODUCTION

Preterm premature rupture of fetal membranes (pPROM) is defined as the onset of amniotic fluid leakage from the vagina before the onset of uterine contractions at less than 37 weeks of gestational age [1]. Preterm premature rupture of membranes (pPROM) occurs in 2–3% of all pregnancies leading to 30–40% of preterm births.

pPROM is a multifactorial process including certain risk components such as pPROM in previous pregnancy, smoking, socioeconomic status, poor nutrition (e.g. body mass index below 19.8 kg/m², copper and ascorbic acid deficiencies), prior cervical conization, cervical cerclage, second- and third-trimester bleeding, acute pulmonary disease and prior episodes of preterm contractions, infection (bacterial vaginosis), amniocentesis, polyhydramnios and multiple gestation but in most of the cases, the cause remains unknown and is not apparent at the time of membrane rupture[2].

Fetal membrane rupture is a physiologic process at term, but when it occurs preterm, it results from abnormal structural weakening of the membranes in the region of the internal cervical os where it is initiated by membrane stretch and involves local inflammation and ascending bacterial colonization [1]. The weakening of membranes is directly caused by bacterial collagenases and proteases, but a number of other pathways are also involved like increased maternal cytokines or an imbalance in MMPs and TIMPs in response to microbial colonization, trauma, and uterine over-distension [3]. Genital tract

pathogens that have been associated with pPROM include *Neisseria gonorrhoeae*, *Chlamydia trachomatis*, *Trichomonas vaginalis*, and group B β -hemolytic streptococcus (GBS). When fluid leakage occurs after amniocentesis, resealing of the membranes is usual (86-94%), but it is usually uncommon after preterm premature rupture of membranes.

The latent period from membrane rupture to delivery is typically brief after pPROM. If pPROM occurs before 34 weeks of gestation, more than 90% of women will deliver within 1 week. Near the limit of viability, about two-thirds of women will deliver within 1 week of membrane rupture, but with expectant management, a latency of four weeks or more can be achieved in one in five cases [1].

Currently most authorities accept a plan of active management which includes prevention of infection, delay of delivery until fetal maturity is achieved and active intervention by induction if labor is no longer preventable or if early infection is suspected [4].

The present study undertaken is to identify the risk factors causing pPROM and to study fetal and maternal outcome associated with pPROM.

REVIEW OF LITERATURE

History:

Greek and Roman obstetric literature provide various references of preterm premature rupture of membranes (pPROM). pPROM was commonly associated with difficult labour and overcome by the use of powerful shaking sternuateries, encouragement, holding of breath and bearing down and strong-smelling things [5]. Rupture of membranes long before the labor may be called dry labor where gentle cervical dilatation is lost causing injury to the cervix and increased pain due to the hard head pressing on cervix [6]. The uterine wall applies itself to the fetal contour which irritates the muscle to cause irregular contractions and thereby forming contraction rings which leads to prolonged labour.

According to Natale et al., [7] and Jairam et al., [8] expectant management of pPROM did not reduce the incidence of caesarean birth and newborn requirements for neonatal intensive care. Administration of antibiotics in pPROM patients also showed reduced neonatal and maternal mortality [9].

Incidence:

Preterm prelabour rupture of membranes (pPROM) occurs in 2–3% of all pregnancies leading to 30–40% of preterm births [10].

PROM occurs approximately in 10% of all pregnancies and in 70% of cases, it occurs in pregnancy at term. Most Indian studies from Mumbai report an incidence of PROM between 7 and 12% [11]. Gunn et al., [12] observed the incidence of PROM to range from 2-17%.

Latent Period:

The time between the rupture of membranes and onset of labour is called latent period. Generally, the shorter the gestation period, the longer the latent period. In pPROM, labour sets within 24 hours in 35-50% patients and within 72 hours in 70% patients. Almost 90% patients deliver within the next two weeks [10].

Fetal membranes:

There are two fetal membranes- Amnion and chorion.

Amnion:

Amnion, the inner of the two fetal membranes, is a tough membrane derived from ectoderm that lines the fetal surface of placenta and umbilical cord. It is in contact with the contents of amniotic sac, namely the amniotic fluid and the fetus. Amnion has no blood vessels, nerves or lymphatics. It is elastic and expands to accommodate the growing fetus. There are five layers in amnion:

- Inner layer of cuboidal epithelium: The epithelial cells synthesize fibronectin, prostaglandin, interleukin, vasoactive peptides like endothelin and parathyroid-hormone-related peptide(PTH-RP) and corticotropin releasing hormone(CRH). Recently it has been implicated that the amniotic epithelium helps in exchange of fluid and electrolytes that is not known to occur between the amniotic sac and mother.
- Basement membrane: It is a band of reticular tissue adherent to the epithelial cells. It is well defined over the placental and reflected parts of amnion.
- Acellular compact layer made of collagen: It is adherent to the basement membrane composed of randomly scattered reticular fibrils.
- Fibroblast like mesenchymal layer: It consists of fibroblast and Hofbauer cells that synthesize collagen, interleukins and prostaglandins.
- Outer spongy layer of acellular loose connective tissue between amnion and chorion.

The thickness of normal amnion is 0.02-0.5mm which varies as a result of alteration in the mucin and fluid content.

Chorion:

The chorion is in contact with the amnion on its inner aspect and maternal decidua on its outer aspect. The chorionic membrane is derived from chorion leave. It is initially separated from amnion by the chorionic cavity that later

disappears and fuses with the amnion. In early pregnancy, the chorion leaflet possesses actively functioning chorionic villi. As pregnancy advances, the villi develop into the placenta and simultaneously the villi atrophy. The blood vessels supplying these atrophic villi also degenerate.

On histological examination, the chorion is made up of 4 layers:

- Cellular layer: It consists of interlacing fibroblast network similar to that present in the fibroblast layer of amnion.
- Reticular layer: It forms the major part of reticular tissue of chorion in which fibroblasts and Hofbauer cells are embedded.
- Basement membrane: It is a narrow band of reticular tissue forming the basement membrane of the trophoblast which lies upon its deeper surface.
- Trophoblast: It contains trophoblast cells about 4-6 cells in thickness, but is extremely variable ranging from 0.04-0.40 mm. In some areas, the chorion is healthy and functionally active whereas in other areas, there is evidence of cellular degeneration and pyknosis of cell nuclei.

Amniotic fluid:

Amniotic fluid fills the amniotic cavity and surrounds the fetus from early pregnancy. The terms 'liquor amnii' and amniotic fluid, both imply a relation between the membrane and the fluid, beyond mere anatomical containment, and many have believed the membranes to be the origin of the amniotic fluid and the regulator of its water and solute content.

Early in pregnancy, the amniotic cavity is surrounded by the amnion and the exocoelomic cavity, also called chorionic cavity, by the chorion, containing coelomic fluid. The amniotic cavity gradually increases in size while the chorionic cavity decreases in size. By 14th week of gestation, the amnion and the chorion fuse leaving only the amniotic cavity filled with amniotic fluid.

The major sources of production of amniotic fluid include fetal urine and fetal lung fluid, while the major routes of clearance include fetal swallowing and intermembranous transfer of fetal blood. The minor sources of production include fetal oral-nasal cavities, while the minor routes of clearance include trans membranous transfer to maternal blood.

The composition of the amniotic fluid varies in the two halves of pregnancy. In the first half, the concentration of major solutes is closely related to those in fetal than in the maternal serum. At about 20 weeks of gestation, the fetal skin becomes impermeable and hence there is a fall in osmolarity and sodium concentration. There is a rise in urea and creatinine concentrations reflecting the maturation of fetal renal function.

The volume of amniotic fluid increases rapidly with the growth of the fetus. At 12 weeks of pregnancy, it is about 50 ml while increases to 400 ml at 20 weeks of gestation. At 35 weeks, it reaches a peak of one liter. During the last few weeks of pregnancy, the volume decreases and at about 43 weeks, the volume ranges from 100 to 600 ml.

Etiopathogenesis:

The amniotic membranes are a connective tissue structure and their tensile strength depends on the synthesis, degradation and quality of the collagen.

Under normal circumstances, the tensile strength of the membranes increases until 20 weeks, plateaus until 39 weeks after which it dramatically reduces [13].

The following factors are responsible for the decreased tensile strength of the membranes:

- Inherited intrinsic weakness of the collagen matrix.
- Acquired degradation of the collagen matrix.
- Increased levels of matrix metalloproteinases(MMP-2, MMP-3, MMP-8, MMP-9).
- Decreased levels of tissue inhibitors of matrix metalloproteinases(TIMP-1, TIMP-3) [14], [15].
- Bacterial invasion produces proteases that stimulate the host inflammatory response resulting in release of cytokines and prostaglandins [16], [17].

According to Barabas et al., 1966[18], pPROM was found to be common among the women affected by connective tissue disorders like Ehlers-Danlos syndrome where the tensile strength of the membranes is found to be decreased. Acquired degradation of collagen matrix due to tobacco smoking, nutritional deficiencies of copper and ascorbic acid etc., where there is abnormal cross-linking of collagen, were also found to be predisposing to pPROM.

Membrane activation is one of the three components of final pathway of parturition. Membrane activation is due to increased collagenolysis which occurs due to the predominance of MMPs over TIMPs.

According to McGreogor et al., 1987[19], intra amniotic infection was found to produce alterations in the tensile strength of the fetal membranes due to the proteolytic enzymes present in the microorganisms that weaken the fetal membranes.

K. Lowndes et al., 2006 [20], found that the relaxin gene was overexpressed in patients with pPROM than in those women in preterm labor with intact membranes. Later Millar et al., [21] found that the relaxin mediated pathway of pPROM was independent of infection.

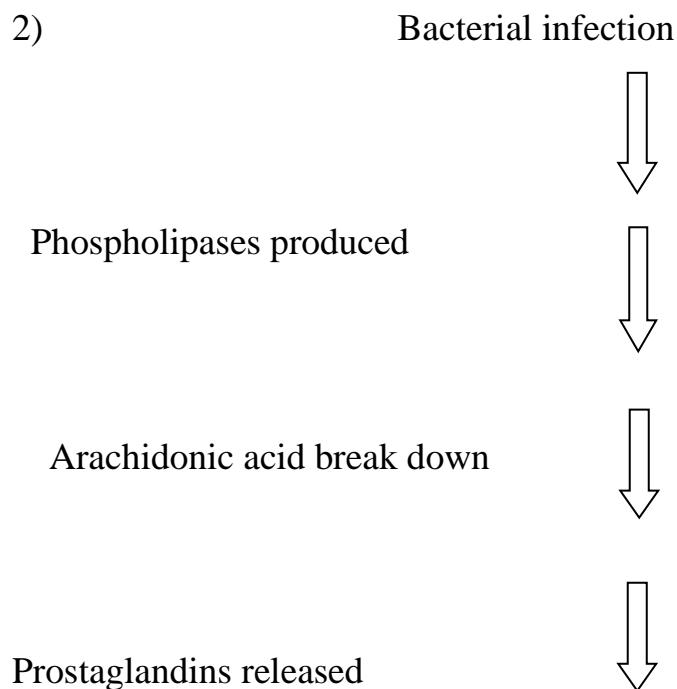
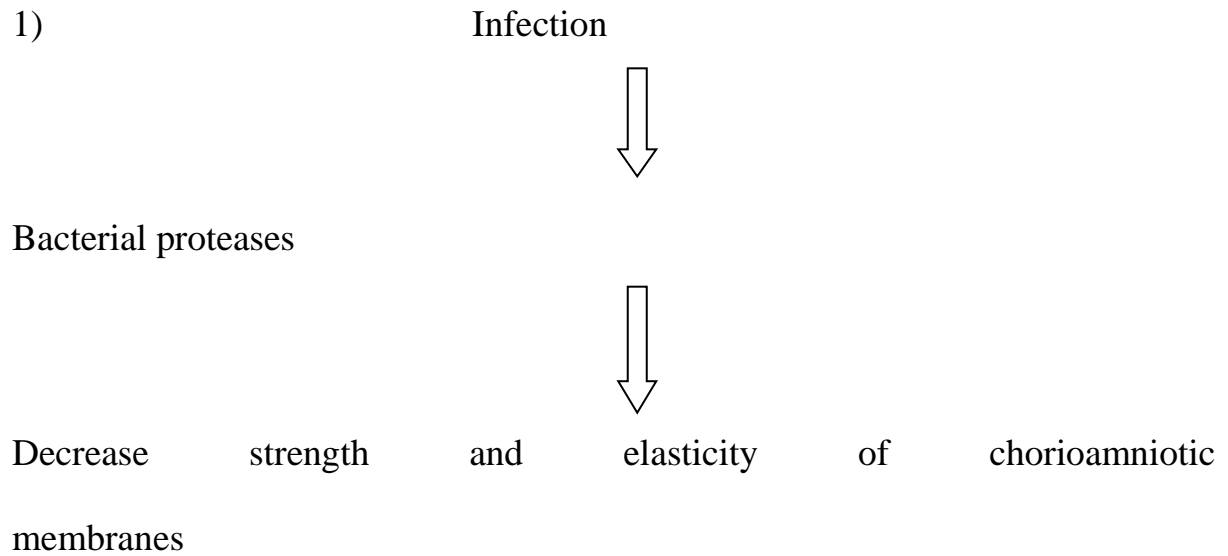
Risk factors:

pPROM is a complex and multifactorial entity where a large number of clinical factors are associated.

Infection:

The greatest risk factor for pPROM is infection [2], [16] where the incidence is 38.3% according to Romero et al., 1993[22].

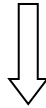
The processes by which bacterial invasion leads to pPROM include:



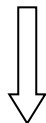
Preterm contractions initiated

3)According to Maymon et al., 2000[14] and Park et al., 2003[23],

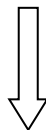
Infection



Release of pro inflammatory cytokines and mediators



Extracellular matrix disrupted and MMPs released



Weakening of fetal membranes

Genetic:

Several genetic polymorphisms with distinct racial distributions are associated with an increased risk of pPROM[24],[25].According to Ferrand et al., 2002[26], polymorphism of the MMP-9 region was more frequently found in African- American population who had pPROM than those who delivered at term.

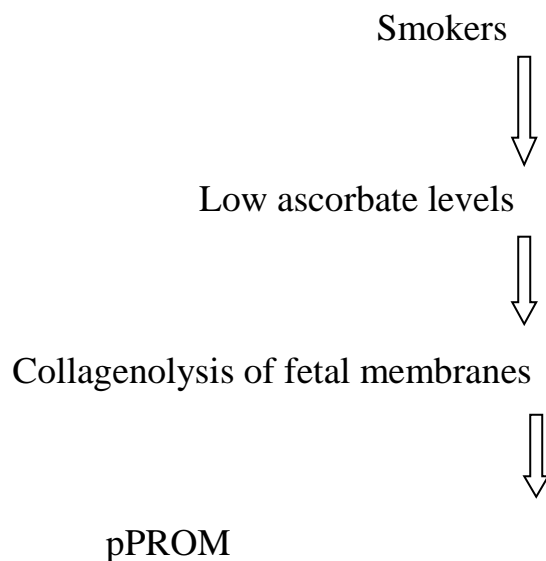
Previous preterm delivery:

According to Naeye et al., 1982[27] and Asrat et al., 1991[28], women who have had previous pPROM are estimated to have a 21% to 32% recurrence

risk in subsequent pregnancies which may be due to endogenous maternal genetic factors or persistence of exogenous environmental factors.

Nutrition:

Vitamin C acts as a coenzyme for collagen cross linking in the extracellular matrix of fetal membranes.



Repetitive stress:

Lavery et al., 1982[29] stated that the uterine activity causes tissue fatigue that reduces the ability of the membranes to tolerate normal increases in pressure.

Complications:

Maternal:

- Acute chorioamnionitis
- Subclinical chorioamnionitis

- Premature placental separation
- Postpartum endometritis
- Risk of operative delivery

Acute chorioamnionitis:

25% of all cases of pPROM develop chorioamnionitis. The clinical diagnosis of chorioamnionitis requires the presence of fever (>100 F or 37.8⁰C) and two or more of:

- Maternal pulse > 100 bpm
- Fetal heart rate > 160 bpm
- Uterine tenderness
- Foul smelling vaginal discharge
- Leukocytosis > 15000
- C-reactive protein >2.7 mg/dl
- with no other infectious site involved.

The risk of intrauterine infection increases with the duration of membrane rupture and with declining gestational age [30], due to decreased antibacterial activity of amniotic fluid remote from term [31], [32].

Burchell et al., 1964[33] found that 1.7% of his patients developed fever within 24 hours, 7.5% between 24 and 48 hours and 8.6% beyond 48 hours. In

another study, the prevalence of chorioamnionitis was 2.7% before 12 hours, 6.3% between 12 and 24 hours and 26.4% after 24 hours of latency [34].

Subclinical chorioamnionitis:

Bacteriologic studies of the amniotic fluid of the patients with pPROM admitted to the hospital revealed an incidence of infection in 40% of patients but only a few of them presented with signs and symptoms of overt infection in a study conducted by Romero et al., [23]. The only symptom of chorioamnionitis is uterine contractions while other signs include absence of respiratory movements in the biophysical profile(BPP) and a change in the pattern of non-stress test from reactive to non-reactive.

Placental separation:

Placental abruption affects 4-12% of pregnancies with pPROM[35]. Placental abruption and hemorrhage occur in 10% of the cases with pPROM secondary to uterine decompression and inflammation, as opposed to 1% of the general obstetric population. According to Rotschild et al., [36] and Fortunato et al., 1994[37], placental abruption occurs in upto 50% of pPROM prior to 20 weeks' gestation.

Postpartum endometritis:

Postpartum endometitis is a frequent maternal complication in women with pPROM, particularly if they develop chorioamnionitis (40%) and are

delivered by caesarean section. Shumway et al., [38] reported that the incidence of postpartum maternal sepsis was between 0% and 3%, but sepsis leading to death had an incidence of 0.14%.

Risk of operative delivery:

Caesarean delivery rates significantly increase in pPROM, secondary to obstetric complications such as fetal malpresentations, non-reassuring fetal heart patterns, cord compression and placental abruption.

Fetal/neonatal complications:

Infection:

Fetal infection is a major complication of mid trimester pPROM(<26 weeks gestation), where neonatal mortality was reported to be approximately 35-40% according to Dinsmoor et al., 2004[39]. The risk of perinatal mortality rate was found to be correlating with the residual volume of amniotic fluid. In pregnancies with pPROM between 20 and 25 weeks of gestation, the group with the largest vertical pocket of <2 cm had a greater neonatal mortality rate than those with the vertical pocket >2 cm.

Alexander et al., [40], in his study involving very low birth weight infants, found that the infants born with infection had a higher incidence of sepsis, respiratory distress syndrome(RDS), early onset seizure, intraventricular

hemorrhage and periventricular leucomalacia which makes it obvious that chorioamnionitis is a major risk factor for neurological injury in the newborn.

In 1999, Yoon et al., [41] found that fetal inflammatory response syndrome[FIRS] was associated with cerebral palsy in which IL-6 was elevated in the fetal plasma.

Pulmonary hypoplasia:

It is a respiratory sequel of pPROM that is to be feared of. In pPROM, pulmonary hypoplasia is due to the altered pressure gradient between amniotic cavity and the alveoli where there is loss of fetal lung fluid into the amniotic cavity.

An incidence of 50% of neonates suffering from pulmonary hypoplasia at 19 weeks was observed, while it was 10% at 25 weeks and rare after 26 weeks. It was also observed that these fetuses with pulmonary hypoplasia were born to mothers whose median amniotic fluid pocket was less than 2 cm [42]. Pulmonary hypoplasia was rarely lethal after 24 weeks because of the alveolar growth that is adequate to support postnatal development [35].

Hyaline membrane disease(HMD):

The greatest threat to the newborn in pPROM is Hyaline membrane disease.

As reported in various studies, at all gestational ages, the risk of respiratory distress is greater than the risk of infection.

The National Neonatal Database gives an incidence of RDS of 100%.

| Gestational age | RDS (%) | Sepsis (%) |
|------------------------|----------------|-------------------|
| 24 weeks | 100 | 36.4 |
| 27-28 weeks | 97.8 | 24.4 |
| 31-32 weeks | 58.1 | 1.6 |
| 33-34 weeks | 30.9 | 0.5 |

The following data was taken from Mercer 2003:

| Gestational age | RDS (%) | Sepsis (%) |
|------------------------|----------------|-------------------|
| 24 weeks | 100 | 40 |
| 28 weeks | 85 | 32 |
| 32 weeks | 25 | 4.5 |
| 34 weeks | 10 | 3.0 |

It seems clear from the above data that expectant management to improve fetal pulmonary maturity should dominate other considerations before 36 weeks, while infection is an important concern especially before 38 weeks.

Cerebral Palsy:

The common complications that frequently occur in pPROM include acute or subclinical chorioamnionitis, severe intraventricular bleeding, intrapartum fetal acidosis and hypoxia that lead to cerebral palsy in the newborn.

The pathogenesis involved is cerebral ischemia which leads to decreased delivery of energy substrates to the brain tissue, accumulation of lactate and inorganic phosphate due to anaerobic metabolism, increased free radical and cytokines production and intracellular accumulation of calcium and phosphorus in neural cells. The cerebral vascular system of a preterm infant is immature because of increased vulnerability to hypoxia that eventually leads to cerebral palsy.

Congenital abnormalities:

Another important factor to be considered that leads to death among infants born to mothers with pPROM is congenital abnormality. According to a study conducted by Berkowitz et al., 1976[43], 4 out of 20 non-RDS deaths following pPROM were caused by congenital abnormalities.

Fetal deformities:

Facial and skeletal deformities occur as a result of severe, prolonged oligohydramnios in pPROM as the fetuses lose the protective cushion against

compression and have a severe limitation in the ability to move the limbs. Most of these cases occur with pPROM before 26 weeks and after a latency of 5 or more weeks [44].The risk of fetal deformities increases when the duration of pPROM exceeds 14 days.

Intrauterine fetal demise:

As mentioned by Morales and Talley 1993[45], the incidence of intrauterine fetal demise decreases as gestational age increases. The most common factors leading to fetal demises include fetal infection, cord prolapse, placental abruption and cord compression. The incidence of cord prolapse is reported to be around 1.9% where the main risk factor is non-cephalic presentation of the fetus, especially before 26 weeks' gestation [46].Placental abruption occurs in almost 50% cases of pPROM prior to 20 weeks' gestation.

Diagnosis:

Diagnosis of pPROM is a stepwise process, where in most of the cases, the patient herself gives a history of membrane rupture in the absence of uterine contractions. During speculum examination, usually copious amounts of amniotic fluid are seen in the vagina which makes the diagnosis obvious.In case of absence of amniotic fluid in the vagina, the patient can be asked to cough or strain down, or gentle moving of the fetus and slight pressure on the uterus will provoke leaking of amniotic fluid from the cervix. In spite of all these, if the nature of the fluid is uncertain, a small amount can be collected over the lower

blade of the speculum for further tests that include fern test and nitrazine test. This sample can also be used for assessing the fetal maturity by measurement of phosphatidyl glycerol and should be sent for culture and sensitivity.

Fern test:

Principle: Drying out of salts containing the amniotic fluid produces a fern pattern.

Procedure:

A small amount of fluid is placed on a glass slide and allowed to dry, where it forms a crystallization pattern that resembles a fern plant under a microscope. If the dry slide is heated, it remains white if it is an amniotic fluid sample, otherwise it turns brown.

Accuracy of the test is affected by blood and meconium.

Cervical mucus sample also produces a similar arborization pattern that leads to false positive results. Tricomi et al., 1966[47], in his study obtained 4.4% false positive results and 4.8% false negative results.

Nitrazine test:

It is a simple bedside test. Normal vaginal pH is 4.5 - 5.5. The amniotic fluid has a pH of 7.0 - 7.5.

Principle:

The pH of the vaginal secretions and urine is acidic while that of the amniotic fluid is alkaline.

Procedure:

A nitrazine paper or swab stick is dipped into the fluid collected over the speculum. If it turns deep blue, then the pH of the fluid is alkaline. If it remains yellow or changes to olive yellow, then the pH of the fluid is around 5.0 - 5.5.

Antiseptic solutions, seminal fluid, urine, blood, bacterial vaginosis and trichomoniasis alter the vaginal pH and cause false positive results.

If both the tests produce positive results, then the diagnosis of pPROM is 100% reliable.

Alpha feto protein:

Detection of Alpha feto protein(AFP) in the vaginal secretions is an accurate test for the diagnosis of pPROM with the specificity of 100%[48], since it does not exist in vaginal secretions or urine while it is present in higher concentrations in the amniotic fluid. However this test is unreliable at term as the concentration of AFP in amniotic fluid decreases with gestational age. Maternal blood contamination also affects the accuracy of the test.

Fetal fibronectin:

Fetal fibronectin is also present in large amounts in the amniotic fluid. In almost 93.8% of the women with pPROM, fetal fibronectin can be detected in the endocervix or vagina by means of an ELISA test. This test is highly accurate and is not affected by blood, but meconium may interfere [49].

Intra amniotic injection of Indigo Carmine:

This procedure is indicated in women with a clinical history consistent with pPROM and a negative fern and nitrazine tests. In this procedure, the amniotic cavity is injected with 2-3 cc of sterile solution of Indigo Carmine and a tampon is placed in the vagina. The presence of bluish discoloration in the tampon after 30 mins to 1 hour is diagnostic of pPROM.

AmniSure:

It is a new generation test, with a sensitivity of 99% and specificity of 100% [50], that is based on the detection of trace amounts of placental microglobulin-1(PAMG-1) produced by the cells of decidual part of the placenta. PAMG-1 can be detected in the amniotic fluid after the rupture of membranes. This test can be performed using a kit in 5-10 minutes.

Management:

Hospitalisation and monitoring:

Hospitalisation is mandatory in cases of pPROM because of the potential complications it leads to. For example, if chorioamnionitis is diagnosed, then expedient delivery is required to reduce the risk of neonatal sepsis and morbidity. Maternal monitoring for tachycardia, fever, uterine tenderness, blood stained or purulent discharge and fetal monitoring for tachycardia via non-stress test are recommended.

Corticosteroid therapy:

Corticosteroid therapy showed a decrease in incidence of RDS, IVH, necrotizing enterocolitis, and neonatal death. The National Institutes of Health Consensus Development Conference (2000) and American Congress of Obstetricians and Gynecologists (ACOG 2007) recommend the use of a single course of antenatal steroids for mothers with pPROM without the evidence of chorioamnionitis, especially between 24 and 34 weeks of gestation, though there is a high risk of perinatal infection, the neonatal benefits outweigh the risks.

Dosage:

- Betamethasone 12mg i.m. in two consecutive days 24 hours apart.
- Dexamethasone 6mg i.m. 12 hours apart for four doses.

Repeated weekly antenatal corticosteroids are not recommended as it carries potential risks. The benefits and risks of a single rescue dosage remote from the initial dose remain to be determined.

Tocolytics:

Short term tocolysis is used to enable the action of corticosteroids and antibiotics. It is also used in cases of transfer of the mother to a higher centre. Long term tocolysis has no role to play. Prophylactic tocolysis has no effect on fetal and maternal outcome although it is shown to increase overall latency.

- ▶ Nifedipine 20-30mg followed by 10-20mg every 6 hours. 500ml bolus of i.v. fluids can be administered before the loading dose to prevent hypotension.
- ▶ Magnesium sulphate 4g bolus followed by 2g per hour.
- ▶ Ritodrine- start infusion with 50mcg per minute, increase every 20 mins to a maximum of 350mcg per minute.
- ▶ Terbutaline-

(a) oral - 2.5-5mg every 4-6 hours.

(b) subcutaneous - 250mcg every 20-30 minutes for 4-6 hours.

(c) i.v.- 5-10mcg per min increased every 10-15 mins to a max of 80mcg per min.

▶ Indomethacin-

(a) oral - loading dose of 50-100mg

(b) rectal - loading dose of 100-200mg

followed by 25-50mg every 4-6 hours.

Tocolytics may mask infection if administered repeatedly. If betamimetics are combined with corticosteroids or calcium channel blockers, it will increase the risk of pulmonary edema, hence a strict control of fluid balance is necessary to prevent this complication. Prostaglandin synthetase inhibitors are to be avoided as they mask early signs of intrauterine infection and affect neonatal cardiovascular adaptation to extrauterine life.

Antibiotic prophylaxis:

Antibiotics are administered prophylactically in view of increasing the latency period and decreasing the rate of chorioamnionitis and newborn sepsis [2].

The National Institute of Child Health and Human Development Maternal Fetal Medicine Unit Research Network [2] and the Oracle 1 Randomized Trial [9] found that the combination of initial intravenous therapy (48 hours) with ampicillin and erythromycin, followed by oral therapy of limited duration (five days) with amoxicillin and enteric-coated erythromycin-base at 24-32 weeks of

gestation, decreased the likelihood of chorioamnionitis and delivery for upto three weeks.

In both the studies, it was found that the complications to the neonate were reduced with the use of ampicillin or erythromycin. However, there was an increased incidence of Necrotizing Enterocolitis among those treated with amoxicillin/clavulanic acid. According to Lewis et al., [46] both the three-day and seven-day courses of either ampicillin or ampicillin-sulbactam seem to be effective in reducing perinatal infection.

The most common microorganisms causing neonatal pneumonia, meningitis and sepsis include Group B Streptococcus. Hence women with pPROM should have genital tract cultures obtained.

Penicillin G should be started after cultures are obtained with a loading dose of 2.5×10^6 units every 4 hours. If Penicillin G is not available, a 2g loading dose of intravenous ampicillin should be started, followed by 1g every 4 hours.

The purpose of this management is to decrease the vertical transmission of GBS and the severe neonatal morbidity that may occur. For those allergic to penicillin, erythromycin is recommended. Erythromycin 250mg bid for 10 days can be given.

Management protocol of pPROM:

The following factors are to be considered for further management of **pPROM:**

- Intraamniotic infection
- Non-assuring fetal heart rate
- Cord prolapse
- Active labour
- Lethal fetal malformation
- Mature fetus

The presence of the any of the above factors recommends immediate termination of pregnancy. If the above-mentioned factors are absent, then the management of pPROM varies according to the gestational age.

Pregnancy <24 weeks:

Termination is recommended after counseling, rarely continuation may be attempted with strict vigilance.

Pregnancy 24-34 weeks:

Conservative management with antibiotics and corticosteroids. Fetal surveillance and maternal monitoring is mandatory.

Pregnancy 34-36 weeks:

Conservative management is recommended. Termination can be done after 36 weeks. If proper neonatal care is available, then earlier termination can also be done.

Pregnancy >36 weeks:

Termination is the choice. 24 hours can be awaited for spontaneous onset of labour.

AIMS AND OBJECTIVES

- To study the risk factors causing preterm premature rupture of membranes.
- To study the outcome of labour in preterm premature rupture of membranes.
- To find out the maternal and perinatal morbidity and mortality trends in preterm premature rupture of membranes.

MATERIALS AND METHODS

The present study on " Maternal and Perinatal outcome in cases of preterm premature rupture of membranes (pPROM) " was conducted in the Department of Obstetrics and Gynaecology, Govt. Chengalpattu Medical College and Hospital, with a study period of 10 months.

The study group includes patients admitted with pPROM under the Department of Obstetrics and Gynaecology, Govt. Chengalpattu Medical College and Hospital.

Sample size: 200 patients admitted with pPROM.

Sampling technique: Prospective study.

Inclusion criteria:

1. All pregnant women with pregnancy between 28-37 weeks of gestational age with preterm premature rupture of membranes.
2. Primi gravida/Multi gravida
3. Singleton/Twin pregnancy
4. Mal presentations
5. Polyhydramnios
6. Mother with diabetes mellitus
7. Mother with PIH/Preeclampsia
8. Confirmation of pPROM by a speculum examination

Exclusion criteria:

1. PROM more than 37 weeks.
2. Congenital anomalies.
3. IUD.

Sample specifications:

Clinical samples such as blood and urine were collected from the patients admitted with pPROM under the Department of Obstetrics and Gynecology, Govt. Chengalpattu Medical College and Hospital.

A detailed clinical examination was performed. In all cases, routine hematological investigations, urine examination, cardiotocograph and obstetric ultrasound examination was performed.

Methodology:

A detailed history was taken including age, booking, socio-economic status, parity, menstrual history, time of onset of draining, amount of fluid lost, its colour, odour, association with pain or bleeding per vagina and perception of fetal movements, history of previous similar episodes in other pregnancies and history suggestive of incompetent os.

General examination was done. Height and weight were noted. Pulse, BP, temperature was noted. Systemic examination included cardiovascular, respiratory systems and CNS systems.

In the obstetric examination, following were noted.

- Height of uterine fundus, lie, presentation and position of foetus, engagement of presenting part, condition of uterus, whether contracted or relaxed. Uterine tenderness was looked for as a sign of chorioamnionitis. Fetal heart sound was auscultated and its rate, rhythm and tone were noted.
- A sterile speculum examination was conducted and presence of liquor amni was noted. The amniotic fluid was collected in cases of frank leaking and sent for culture and sensitivity. When no amniotic fluid was seen in the vagina, the patient was asked to cough and the amniotic fluid was collected per speculum. In cases of doubt, fluid from vagina was collected in a glass slide and examined under microscope for ferning or subjected to litmus paper test. Cervical swab was taken and sent for Gram stain and culture sensitivity.
- A single pelvic examination was done to note the Bishop's score, adequacy of pelvis, assessment of CPD and to rule out cord prolapse.

Injection Ampicillin 500mg was given as a prophylactic antibiotic 6th hourly. A 4th hourly monitoring for pulse, BP, temperature was carried out. Fetal heart sounds were recorded every half an hour initially.

Depending upon the Bishop's score, the labour was allowed to progress spontaneously or induced with Cervi prime gel or misoprostol 25mcg according to RCOG guidelines.

The onset of complications like fetal distress, fetal heart rate variations, chorioamnionitis were looked for.

In cases of fetal jeopardy or any other obstetric complications, labour was cut short by caesarean section.

Mothers were watched for third stage complications like PPH and retained placenta and followed up in puerperal period. Foul smelling lochia and febrile illness postnatally were specifically asked for. Episiotomy and caesarean section wounds were followed up regularly and wound infections are looked for. Maternal complications like puerperal sepsis, urinary tract infections and respiratory tract infections were noted.

The babies were followed up in the postnatal period. Neonatal mortality and morbidity were noted. Neonates were monitored for the complications of birth injuries, signs of asphyxia, meconium aspiration and sepsis.

Both mother and baby were followed up till their stay in the hospital.

STATISTICAL ANALYSIS:

Variables like age, parity, socio economic status, duration of pregnancy, mode of delivery, maternal and fetal outcomes are recorded.

Values are expressed as prevalence rates. Conventional Chi squared test was used to analyze differences.

$P < 0.05$ was considered significant.

Statistical analysis was performed with SPSS statistical software with all the relevant data compiled and entered.

RESULTS AND OBSERVATIONS

The study was performed in the Department of Obstetrics and Gynaecology, Chengalpattu Medical College and Hospital. The study group included 200 patients admitted with pPROM.

The risk factors, outcome of labour, maternal and perinatal outcome of preterm premature rupture of membranes were investigated in this study.

DISTRIBUTION OF CASES AS PER AGE: (n=200)

Table 1.1: Age distribution:

| AGE | NUMBER | PERCENTAGE |
|---------------|---------------|-------------------|
| <20 years | 20 | 10 |
| 21 - 25 years | 110 | 55 |
| 26 - 30 years | 47 | 23.5 |
| >30 years | 23 | 11.5 |
| Total | 200 | 100 |

FIGURE 1.1: AGE DISTRIBUTION:

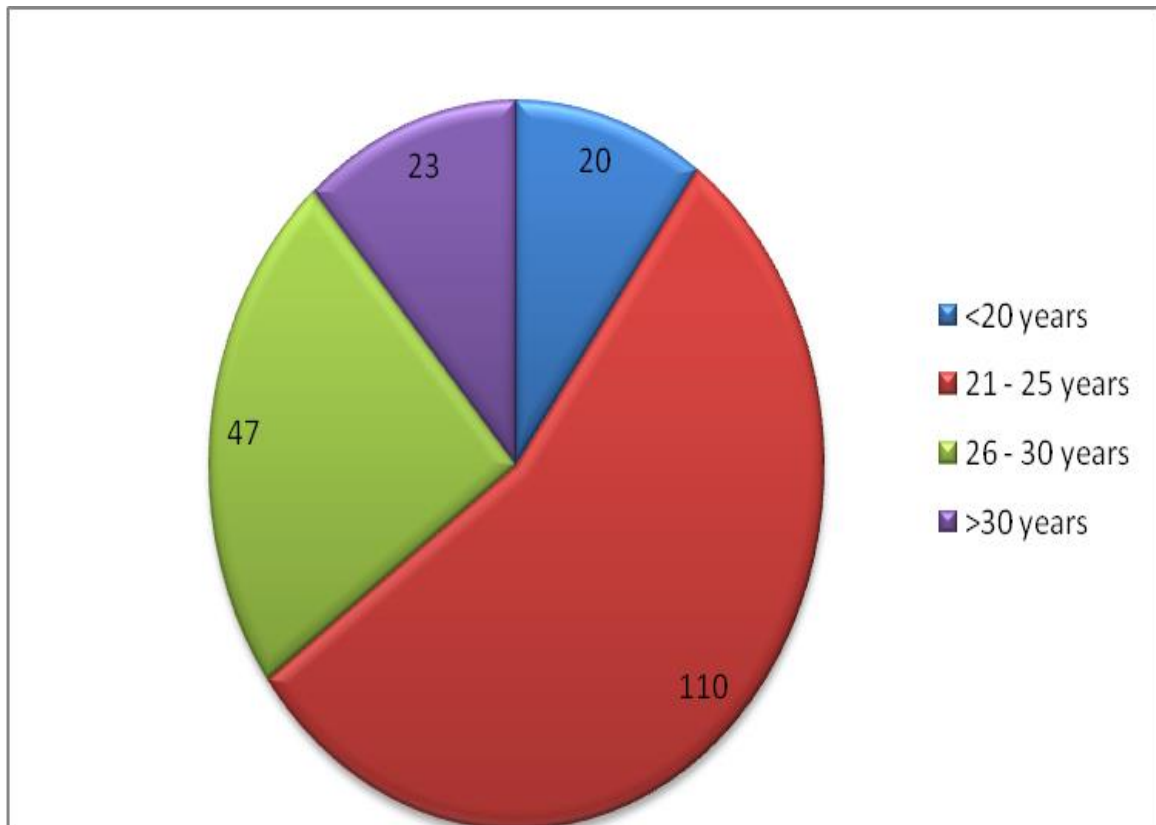


TABLE 1.1:

Among the selected cases, pPROM was noted in 20 (10%) mothers in the age group of <20 years, 110 (55%) mothers in the age group of 21-25 years, 47 (23.5%) mothers were in the age group of 26-30 years, and 23(11.5%) mothers above 30 years of age.

ANALYSIS OF pPROM AS PER SOCIOECONOMIC STATUS: (n=200)

Table 1.2: Socio economic status distribution:

| SES | NUMBER | PERCENTAGE |
|--------------|------------|------------|
| III | 12 | 6 |
| IV | 56 | 28 |
| V | 132 | 66 |
| Total | 200 | 100 |

FIGURE 1.2: SOCIO ECONOMIC STATUS DISTRIBUTION:

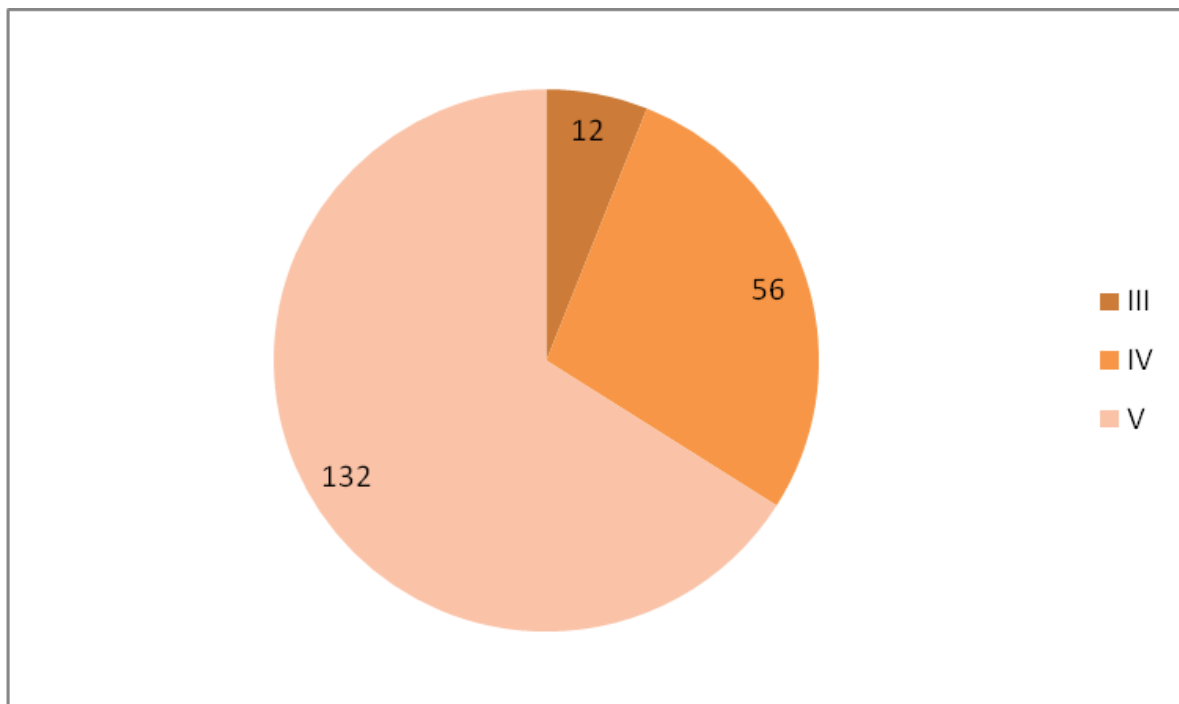


Table 1.2:

In the present study, majority of cases belong to socioeconomic status V with a total of 132 cases (66%), while 56 cases (28%) belong to socioeconomic status IV and 12 cases (6%) belong to socioeconomic status III.

DISTRIBUTION OF CASES AS PER OBSTETRIC SCORE: (n=200)

Table 1.3: Parity distribution:

| GRAVIDA | NUMBER | PERCENTAGE |
|----------------|---------------|-------------------|
| Multigravida | 74 | 37 |
| Primigravida | 126 | 63 |
| Total | 200 | 100 |

FIGURE 1.3: PARITY DISTRIBUTION:

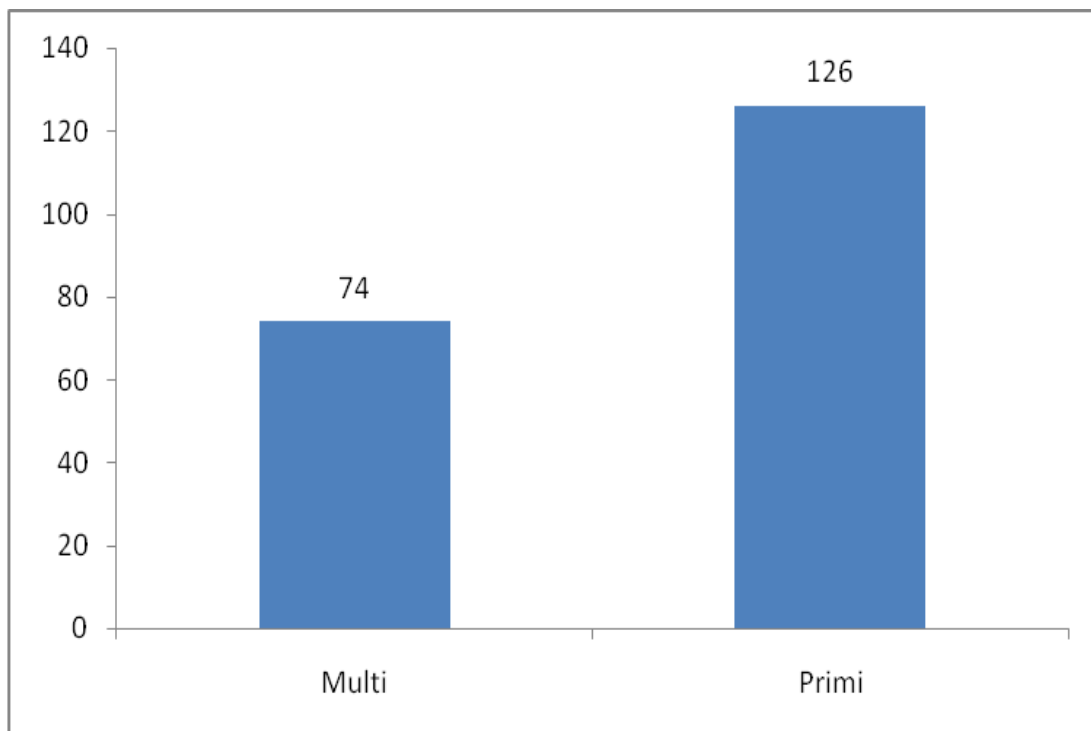


Table 1.3:

Number of multigravida in the study were 74 (37%) and primigravida were 126 (63%).

DISTRIBUTION OF CASES AMONG BOOKED AND UNBOOKED:

(n=200)

Table 1.4: Booked vs Unbooked:

| PPROM | NUMBER | PERCENTAGE |
|--------------|---------------|-------------------|
| Unbooked | 36 | 18 |
| Booked | 164 | 82 |
| Total | 200 | 100 |

FIGURE 1.4: BOOKED VS UNBOOKED:

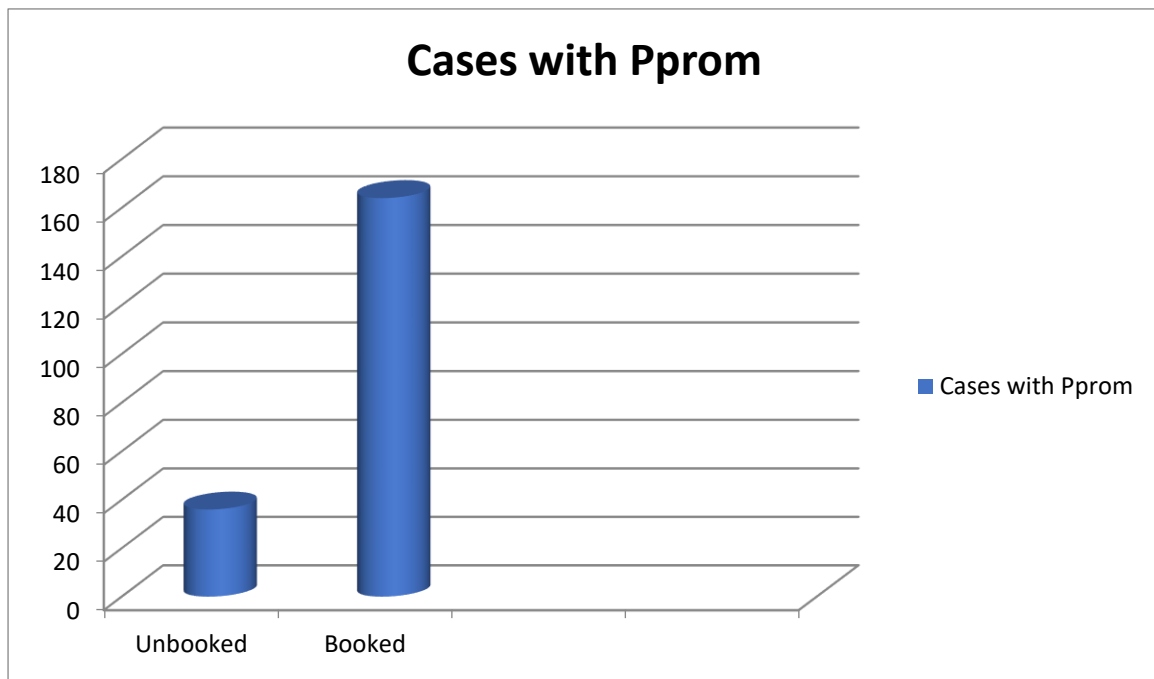


Table 1.4:

Among the 200 patients studied, 164 cases (82%) were booked and 36 cases (18%) were unbooked.

ANALYSIS OF pPROM ACCORDING TO GESTATIONAL AGE:

(n=200)

Table 1.5: Gestational age distribution:

| GESTATIONAL AGE | NUMBER | PERCENTAGE |
|------------------------|---------------|-------------------|
| 28-31weeks | 21 | 10.5 |
| 32-34 weeks | 66 | 33 |
| 35-36 weeks | 113 | 56.5 |
| Total | 200 | 100 |

FIGURE 1.5: GESTATIONAL AGE DISTRIBUTION:

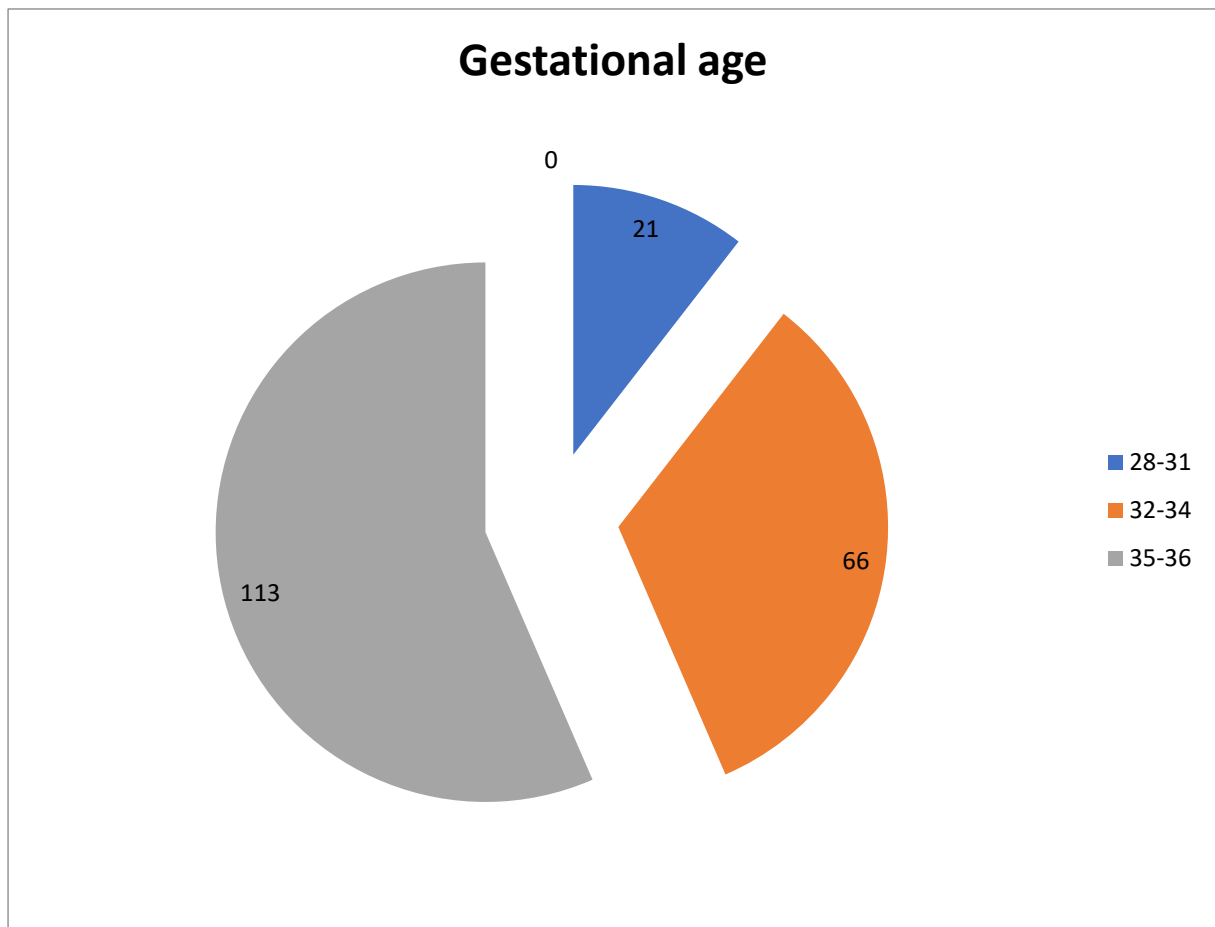


Table 1.5:

In our study, pPROM more commonly occurred in patients with gestational age of 35-36 weeks with a frequency of 113 patients (56.5%), 66 patients (33%) in 32-34 weeks and 21 patients (10.5%) in 28-31 weeks, which signifies that 43.5% had early pPROM and 56.5% had late pPROM.

ANALYSIS OD pPROM AS PER LATENT PERIOD: (n=200)

Table 1.6: Latent period distribution:

| LATENCY (in hrs) | NUMBER | PERCENTAGE |
|-------------------------|---------------|-------------------|
| 0-24 | 121 | 60.5 |
| 25-72 | 57 | 28.5 |
| >72 | 22 | 11 |
| Total | 200 | 100 |

FIGURE 1.6: LATENT PERIOD DISTRIBUTION:

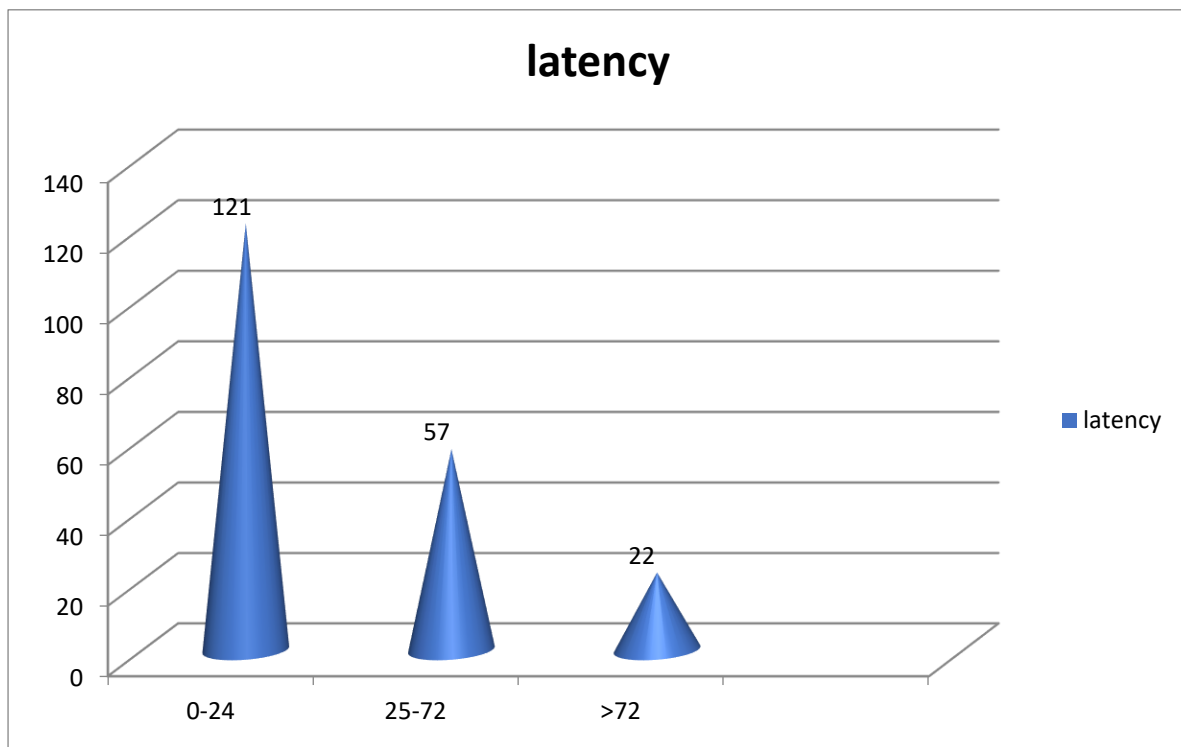


Table 1.6:

60.5%(121 cases) of my study population had delivery within 24 hours of membrane rupture. Only 11%(22 cases) had a latent phase of >3 days. The rest of 28.5%(57 cases) delivered within 25-72 hours.

DISTRIBUTION OF CASES ACCORDING TO MODE OF DELIVERY:

(n=200)

Table 1.7: Mode of delivery vs pPROM:

| MODE OF DELIVERY | NUMBER | PERCENTAGE |
|-------------------------|---------------|-------------------|
| Vaginal | 117 | 58.5 |
| Assisted Breech | 12 | 6 |
| Twins by vaginal | 5 | 2.5 |
| LSCS | 66 | 33 |
| Total | 200 | 100 |

FIGURE 1.7:MODE OF DELIVERY VS PPRM

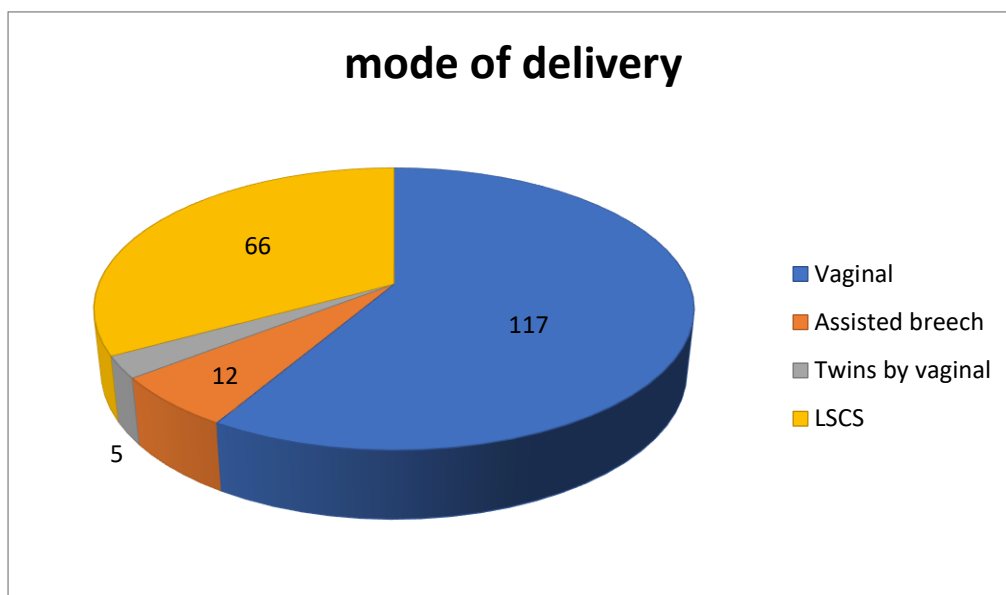


Table 1.7:

Out of the 200 cases in my study, only 66 cases (33%) delivered by Lower segment caesarean section. The rest 117 cases (58.5%) delivered by normal vaginal delivery.

DISTRIBUTION OF LSCS CASES AS PER INDICATIONS: (n=66)

Table 1.8:

| INDICATIONS FOR LSCS | NUMBER | PERCENTAGE |
|-----------------------------|---------------|-------------------|
| Previous LSCS | 13 | 6.5 |
| Breech | 3 | 1.5 |
| Fetal Distress | 29 | 14.5 |
| CPD | 5 | 2.5 |
| Severe oligohydramnios | 16 | 8 |
| Total | 66 | 33 |

FIGURE 1.8: DISTRIBUTION OF LSCS CASES AS PER INDICATIONS

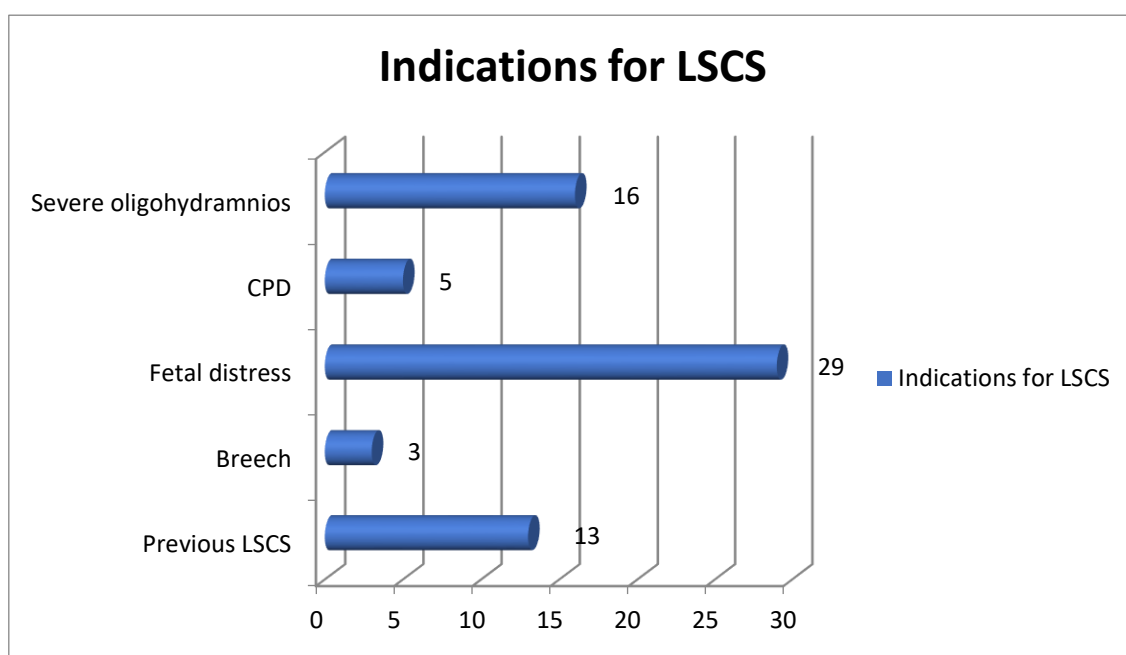


Table 1.8:

The most common indication for LSCS was fetal distress (14.5%), followed by severe oligohydramnios (8%) and previous LSCS (6.5%). Cephalopelvic disproportion (2.5%) and breech presentation (1.5%) also contributed as indications for LSCS in this study.

ANALYSIS OF CHILD BIRTH WEIGHT IN pPROM PATIENTS:

(n=200)

Table 1.8:

| BIRTH WEIGHT (in kg) | NUMBER | PERCENTAGE |
|-----------------------------|---------------|-------------------|
| <1.5 | 12 | 6 |
| 1.5-2.0 | 24 | 12 |
| 2-2.5 | 97 | 48.5 |
| >2.5 | 67 | 33.5 |
| Total | 200 | 100 |

FIGURE 1.9 ANALYSIS OF CHILD BIRTH WEIGHT IN pPROM

PATIENTS:

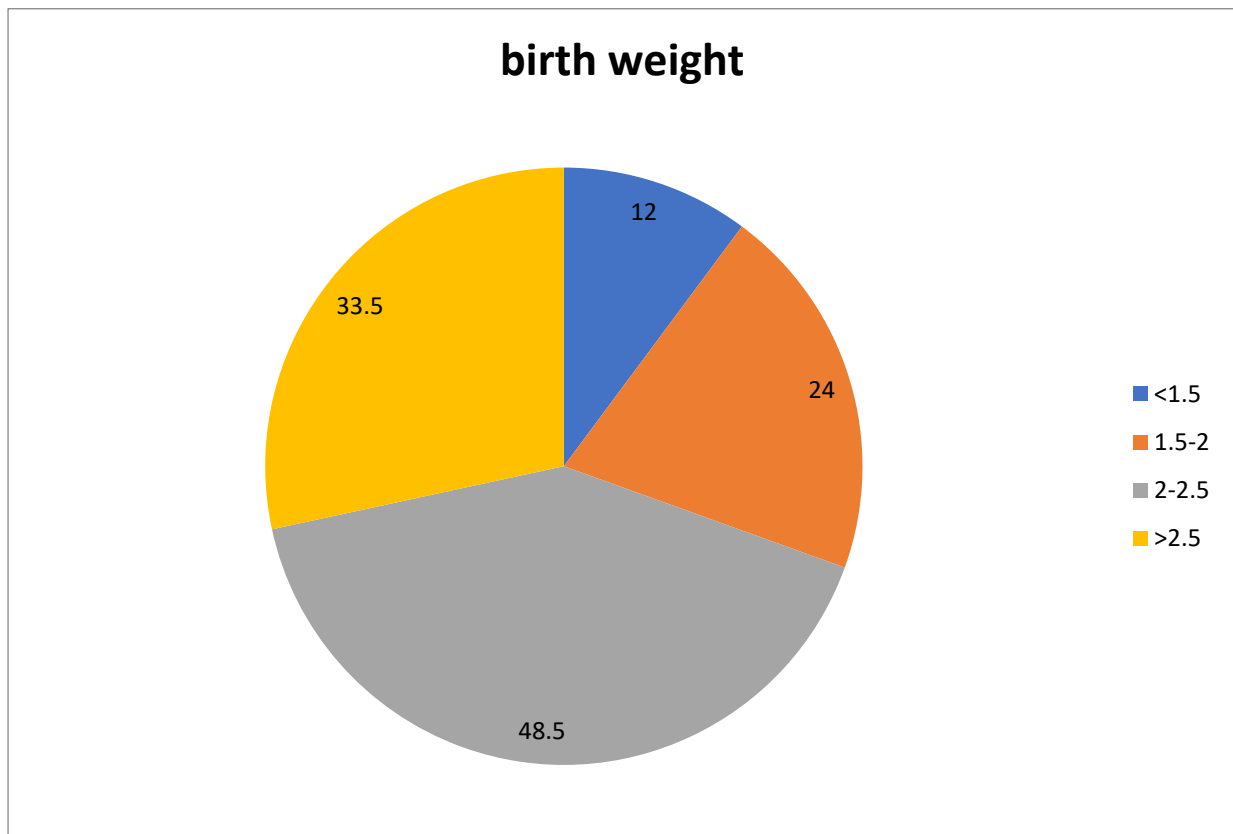


Table 1.9:

Among 200 cases in the study, 97 patients (48.5%) delivered babies whose birth weight was between 2-2.5 kg. 67 patients (33.5%), 24 patients (12%) and 12 patients (6%) delivered babies of >2.5 kg, 1.5-2 kg and <1.5 kg birth weights respectively. These results imply that only 6% delivered very low birth weight babies, 12% delivered low birth weight babies and 82% delivered babies weighing >2kg.

**DISTRIBUTION OF CASES AS PER MODE OF INDUCTION AMONG
VAGINAL DELIVERY IN pPROM PATIENTS: (n=134)**

Table 1.10: Mode of induction distribution:

| MODE OF INDUCTION IN VAGINAL DELIVERY | NUMBER | PERCENTAGE |
|--|---------------|-------------------|
| Spontaneous | 72 | 36 |
| Cervi prime | 25 | 12.5 |
| Misoprostol | 37 | 18.5 |
| Total | 134 | 67 |

FIGURE 1.10: MODE OF INDUCTION DISTRIBUTION:

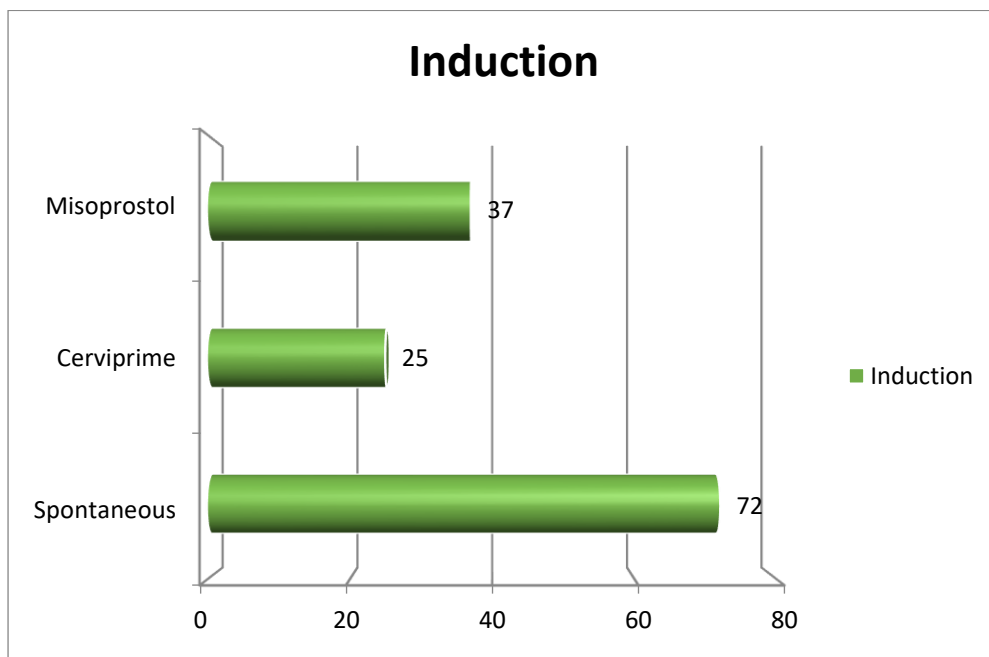


Table 1.10:

Among 134 patients delivered vaginally, 72 patients (36%) had spontaneous delivery, Misoprostol induction done for 37 patients (18.5%) and Cervi prime gel induction done in 25 (12.5%) of them which signifies that 31% had induced labour.

ANALYSIS OF RISK FACTORS ASSOCIATED WITH pPROM: (n=200)

Table 1.11: Risk factors distribution:

| RISK FACTORS | NUMBER | PERCENTAGE |
|--------------------------|---------------|-------------------|
| No risk factors | 140 | 70 |
| Breech | 15 | 7.5 |
| History of recent coitus | 12 | 6 |
| Previous history of PROM | 14 | 7 |
| Polyhydramnios | 11 | 5.5 |
| Twins | 4 | 2 |
| UTI | 4 | 2 |
| Total | 200 | 100 |

FIGURE 1.11: RISK FACTORS DISTRIBUTION:

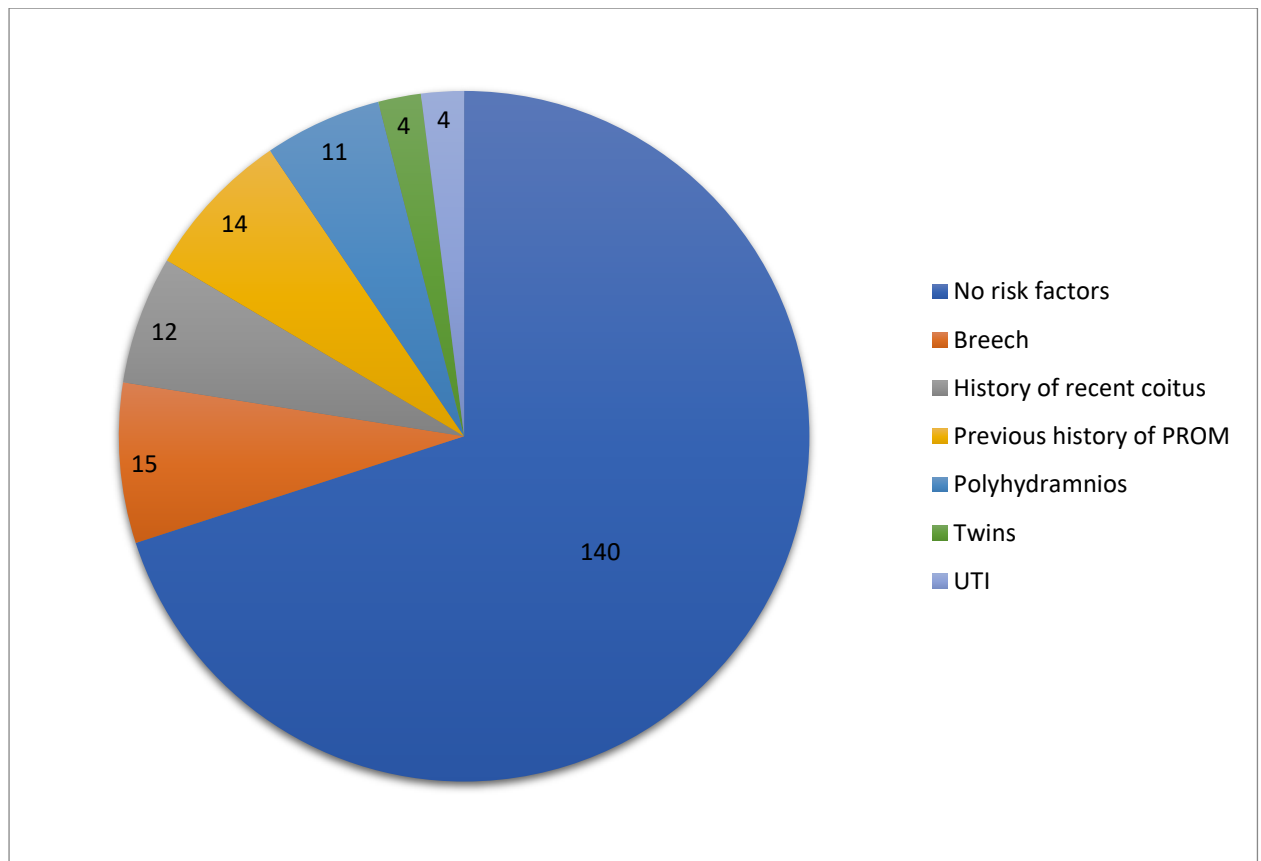


Table 1.11:

70% of the population in the present study had no risk factors. The commonest risk factor was breech presentation (7.5%) followed by patients with previous history of PROM (7%), history of recent coitus (6%), polyhydramnios (5.5%) and multiple gestation and UTI each contributing to 2% respectively.

ORGANISMS ISOLATED BY AMNIOTIC FLUID CULTURE

REPORTS: (n=200)

Table 1.12:

| AMNIOTIC FLUID | NUMBER | PERCENTAGE |
|------------------------|---------------|-------------------|
| No growth | 158 | 79 |
| E. coli | 24 | 12 |
| Klebsiella | 9 | 4.5 |
| Pseudomonas Aeruginosa | 2 | 1 |
| Proteus | 6 | 3 |
| S.Aureus | 1 | 0.5 |
| Total | 200 | 100 |

FIGURE 1.12:

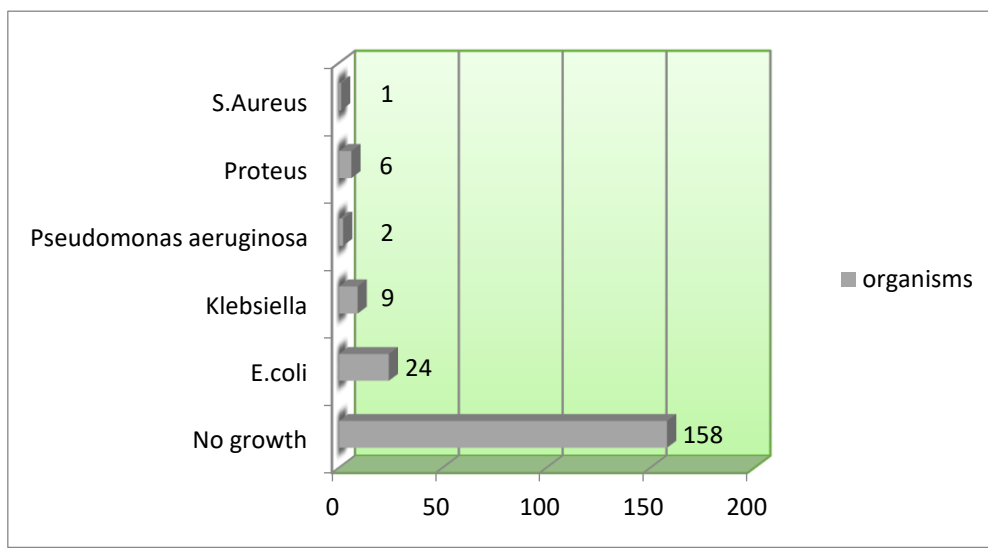


Table 1.12:

Among the culture and sensitivity reports, there was no growth in culture in 79% of cases. E. coli was the most common organism found in cultures, which was present in 12% of pPROM cases, followed by Klebsiella in 4.5% of cases, Proteus in 3%, Pseudomonas aeruginosa in 1% and S.Aureus in 0.5%.

ANALYSIS OF MATERNAL COMPLICATIONS IN pPROM: (n=200)

Table 1.13:Maternal complications distribution:

| MATERNAL COMPLICATIONS | NUMBER | PERCENTAGE |
|-------------------------------|---------------|-------------------|
| No complications | 167 | 83.5 |
| Chorioamnionitis | 8 | 4 |
| Abruption | 7 | 3.5 |
| Puerperal pyrexia | 7 | 3.5 |
| Wound infection | 11 | 5.5 |
| Total | 200 | 100 |

FIGURE 1.13:MATERNAL COMPLICATIONS DISTRIBUTION

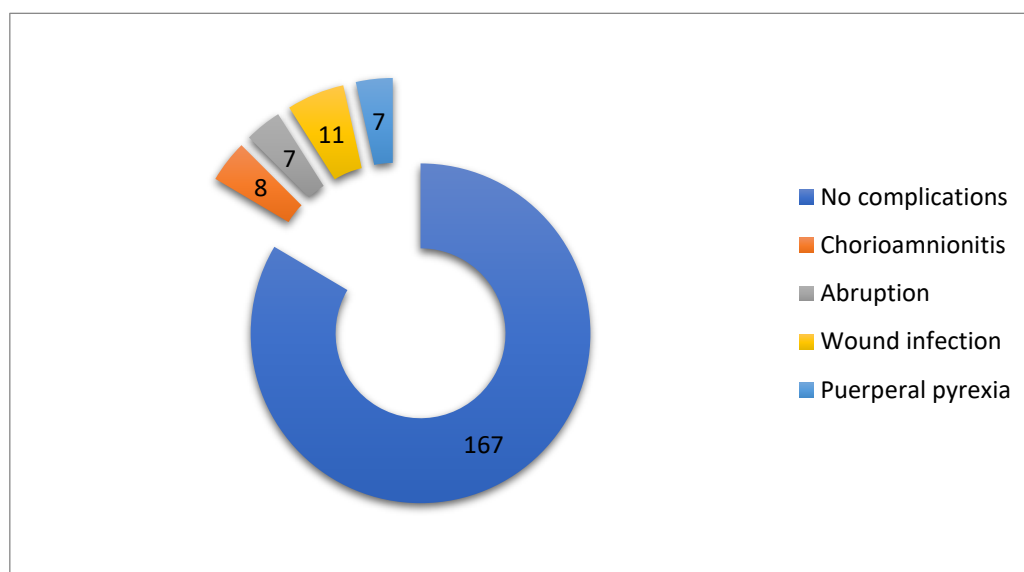


Table 1.13:

In the present study, no maternal complications were seen in 167 cases (83.5%) of pPROM. Only 16% had complications. Wound infection was the most common maternal complication in 11 cases (5.5%) followed by chorioamnionitis (4%), puerperal pyrexia and abruption each contributing to 3.5%.

ANALYSIS OF NICU ADMISSIONS IN PPROM: (N=200)

TABLE 1.14:

| NICU ADMISSIONS | NUMBER | PERCENTAGE |
|------------------------|---------------|-------------------|
| No | 152 | 76 |
| Yes | 48 | 24 |
| Total | 200 | 100 |

FIGURE 1.14:

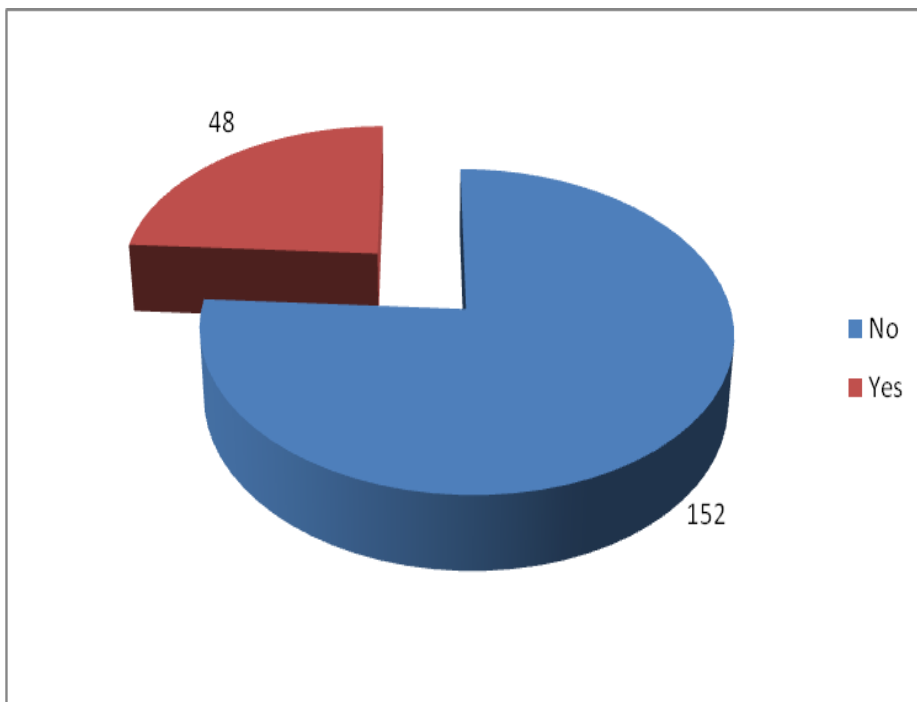


Table 1.14:

Out of my study population, babies born to 48 mothers (24%) had NICU admissions.

ANALYSIS OF NEONATAL COMPLICATIONS IN PPROM: (N=200)

TABLE 1.15:

| NEONATAL COMPLICATIONS | NUMBER | PERCENTAGE |
|-------------------------------|---------------|-------------------|
| No complications | 151 | 75.5 |
| RDS | 25 | 12.5 |
| Septicemia | 10 | 5 |
| Jaundice | 8 | 4 |
| IVH | 6 | 3 |
| Total | 200 | 100 |

FIGURE 1.15:

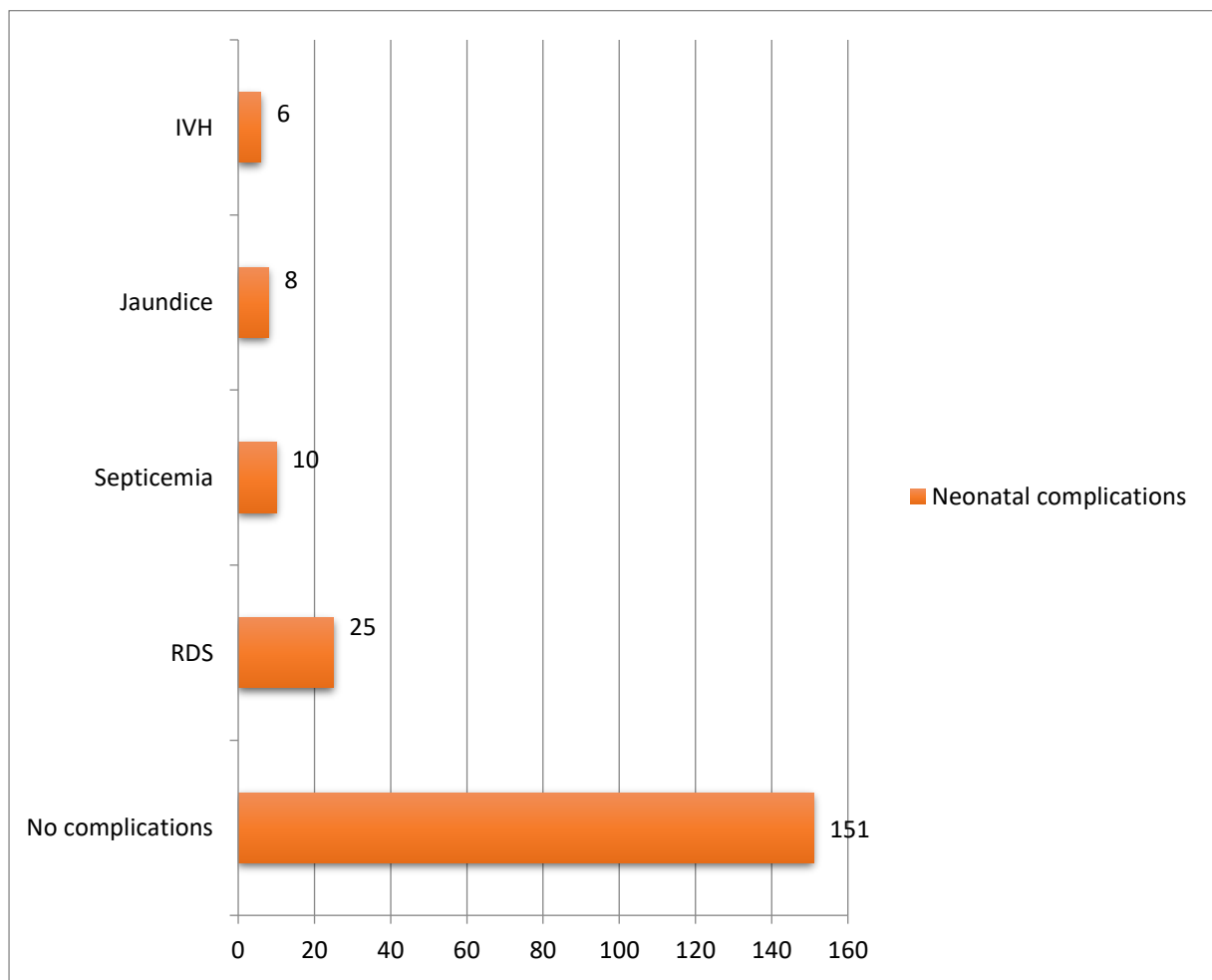


Table 1.15:

Almost 75.5% of the newborn had no complications. 12.5% suffered from respiratory distress syndrome, 5% had septicemia, 4% suffered from septicemia and 3% had intraventricular hemorrhage.

DISTRIBUTION OF NEONATAL DEATH IN pPROM CASES
ACCORDING TO GESTATIONAL AGE: (n=200)

Table 1.16:

| NEONATAL DEATH ACC. TO GESTATIONAL AGE | NUMBER | PERCENTAGE |
|---|---------------|-------------------|
| No deaths | 187 | 93.5 |
| 28-30 | 4 | 2 |
| 30-34 | 7 | 3.5 |
| 35-36 | 2 | 1 |
| Total | 200 | 100 |

FIGURE 1.16:

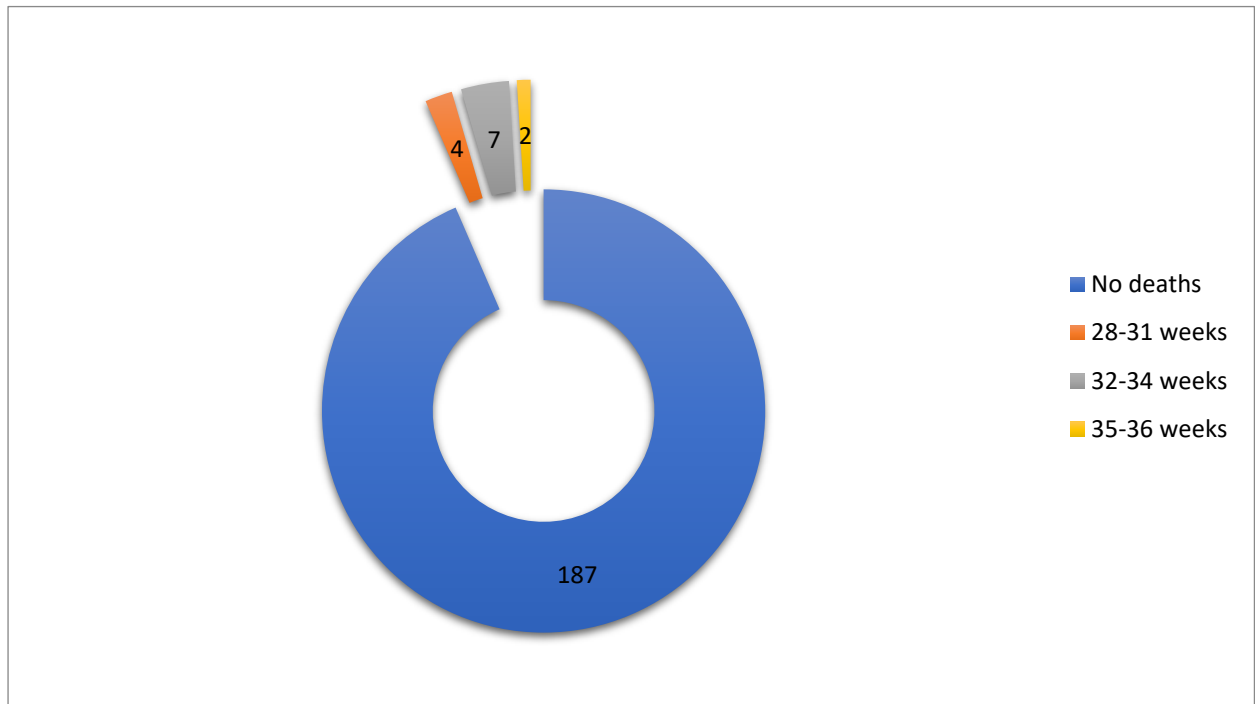


Table 1.16:

In my study, there were 13 neonatal deaths of which 11 were early preterm babies.

TEST OF SIGNIFICANCE:

TABLE 1:

| Neonatal Morbidity | Gestational Age (in weeks) | | | |
|---------------------------|-----------------------------------|--------------|--------------|--------------|
| | 28-31 | 32-34 | 35-36 | Total |
| No morbidity | 14 | 47 | 90 | 151 |
| RDS | 4 | 12 | 9 | 25 |
| Septicemia | 1 | 2 | 7 | 10 |
| Jaundice | 2 | 5 | 1 | 8 |
| IVH | 0 | 0 | 6 | 6 |
| Total | 21 | 66 | 113 | 200 |

Chi-square value-16.83; P value- 0.03

Out of 24.5% of neonatal morbidity, complications were maximum in 32-34 weeks group.

STATISTICALLY SIGNIFICANT

TABLE 2:

| NICU admissions | Gestational Age (in weeks) | | | |
|------------------------|-----------------------------------|--------------|--------------|--------------|
| | 28-31 | 32-34 | 35-36 | Total |
| No | 3 | 42 | 107 | 152 |
| Yes | 18 | 24 | 6 | 48 |
| Total | 21 | 66 | 113 | 200 |

Chi-square value- 71.02; P value- 0.0001

NICU admissions were more common in 28-31 weeks group. Out of 21, 18 had NICU admissions which was 85%.

STATISTICALLY SIGNIFICANT

TABLE 3:

| Maternal complications | Gestational Age (in weeks) | | | |
|-------------------------------|-----------------------------------|--------------|--------------|--------------|
| | 28-31 | 32-34 | 35-36 | Total |
| Nil complications | 14 | 60 | 93 | 167 |
| Chorioamnionitis | 4 | 6 | 5 | 15 |
| Abruption | 2 | 0 | 5 | 7 |
| wound infection | 1 | 0 | 10 | 11 |
| Total | 21 | 66 | 113 | 200 |

Chi-square value- 17.27; P value- 0.008

Maternal morbidity was more common in 28-31 weeks of gestation which was about 33%.

STATISTICALLY SIGNIFICANT

TABLE 4:

| Amniotic fluid Culture & sensitivity | Gestational Age (in weeks) | | | |
|---|-----------------------------------|--------------|--------------|--------------|
| | 28-31 | 32-34 | 35-36 | Total |
| No organisms | 13 | 53 | 92 | 158 |
| E.coli | 4 | 11 | 9 | 24 |
| Klebsiella | 2 | 0 | 7 | 9 |
| Pseudomonas Aeruginosa | 0 | 0 | 2 | 2 |
| Proteus | 1 | 2 | 3 | 6 |
| S.Aureus | 1 | 0 | 0 | 1 |
| Total | 21 | 66 | 113 | 200 |

Chi-square value- 19.67; P value- 0.03

Sepsis was more common in 28-31 weeks group of which E. coli was the most common organism.

STATISTICALLY SIGNIFICANT

TABLE 5:

| Risk factors | Gestational Age(in weeks) | | | |
|--------------------------|----------------------------------|--------------|--------------|--------------|
| | 28-31 | 32-34 | 35-36 | Total |
| No risk factors | 15 | 36 | 89 | 140 |
| Breech | 0 | 10 | 5 | 15 |
| History of recent coitus | 2 | 6 | 4 | 12 |
| Previous history of PROM | 2 | 3 | 9 | 14 |
| Polyhydramnios | 1 | 6 | 4 | 11 |
| Twins | 1 | 3 | 0 | 4 |
| UTI | 0 | 2 | 2 | 4 |
| Total | 21 | 66 | 113 | 200 |

Chi-square value- 23.54; P value- 0.02

Risk factors were commonly found among 32-34 weeks group with 45%.

STATISTICALLY SIGNIFICANT

TABLE 6:

| Birth Weight (in kg) | Gestational Age (in weeks) | | | |
|-----------------------------|-----------------------------------|--------------|--------------|--------------|
| | 28-31 | 32-34 | 35-36 | Total |
| <1.5 | 11 | 0 | 1 | 12 |
| 1.5-2.0 | 5 | 15 | 4 | 24 |
| 2-2.5 | 3 | 47 | 47 | 97 |
| >2.5 | 2 | 4 | 61 | 67 |
| Total | 21 | 66 | 113 | 200 |

Chi-square value- 145.5; P value-0.0001

Among 28-31 weeks of gestational age, 52% of babies were <1.5kg. In 32-34 weeks gestational age, 75% were 2-2.5kg, and in 35-36 weeks, 53% were >2.5kg.

STATISTICALLY SIGNIFICANT

TABLE 7:

| Induction in vaginal delivery | Gestational Age | | | |
|--|------------------------|--------------|--------------|--------------|
| | 28-31 | 32-34 | 35-36 | Total |
| Spontaneous | 7 | 28 | 36 | 72 |
| Cervi prime | 3 | 6 | 16 | 25 |
| Misoprostol | 10 | 14 | 13 | 37 |
| Total | 21 | 48 | 65 | 134 |

Chi-square value- 9.93; P value- 0.13

Spontaneous delivery commonly occurred in patients with 35-36 weeks of gestation of about 50%.

STATISTICALLY NOT SIGNIFICANT

TABLE 8:

| Indications for LSCS | Gestational Age(in weeks) | | | |
|-----------------------------|----------------------------------|--------------|--------------|--------------|
| | 28-31 | 32-34 | 35-36 | Total |
| Vaginal delivery | 20 | 49 | 65 | 134 |
| Previous LSCS | 0 | 4 | 9 | 13 |
| Breech | 0 | 0 | 3 | 3 |
| Fetal Distress | 0 | 6 | 23 | 29 |
| CPD | 1 | 2 | 2 | 5 |
| Severe oligohydramnios | 0 | 5 | 11 | 16 |
| Total | 21 | 66 | 113 | 200 |

Chi-square value- 18.5; P value- 0.04

Fetal distress was the most common indication for LSCS in all gestational age groups. Previous LSCS was the second most common indication. LSCS was more in 35-36 weeks of gestation (42%).

STATISTICALLY SIGNIFICANT

TABLE 9:

| Mode of Delivery | Gestational Age(in weeks) | | | |
|-------------------------|----------------------------------|--------------|--------------|--------------|
| | 28-31 | 32-34 | 35-36 | Total |
| Vaginal | 18 | 36 | 63 | 117 |
| Breech | 0 | 10 | 2 | 12 |
| Twins by vaginal | 2 | 3 | 0 | 5 |
| LSCS | 1 | 17 | 48 | 66 |
| Total | 21 | 66 | 113 | 200 |

Chi-square value- 34.09; P value- 0.0001

Almost 95% of women 28-31 weeks of gestational age group, 74% of women in 32-34 weeks of gestation delivered vaginally. In 35-36 weeks of gestation, almost 57% delivered vaginally and 43% delivered by caesarean section.

STATISTICALLY SIGNIFICANT

TABLE 10:

| Latency (in hrs.) | Gestational Age (in weeks) | | | |
|--------------------------|-----------------------------------|--------------|--------------|--------------|
| | 28-31 | 32-34 | 35-36 | Total |
| 0-24 | 2 | 38 | 81 | 121 |
| 25-72 | 10 | 20 | 27 | 57 |
| >72 | 9 | 8 | 5 | 22 |
| Total | 21 | 66 | 113 | 200 |

Chi-square value- 38.95; P value-0.0001

Out of 11% who had latent phase of >3 days, 7% of them were of <34 weeks of gestational age. Almost 66% of women in 35-36 weeks delivered within 24 hours.

STATISTICALLY SIGNIFICANT

TABLE 11:

| Age of the mother | Gestational Age(in weeks) | | | |
|--------------------------|----------------------------------|--------------|--------------|--------------|
| | 28-31 | 32-34 | 35-36 | Total |
| <20 years | 7 | 5 | 8 | 20 |
| 21 - 25 years | 8 | 33 | 69 | 110 |
| 26 - 30 years | 2 | 24 | 21 | 47 |
| >30 years | 4 | 4 | 15 | 23 |
| Total | 21 | 66 | 113 | 200 |

Chi-square value- 25.53; P value- 0.001

55% of the women were in the age group of 21-25 years.

STATISTICALLY SIGNIFICANT

TABLE 12:

| pPROM | Gestational Age (in weeks) | | | |
|--------------|-----------------------------------|--------------|--------------|--------------|
| | 28-31 | 32-34 | 35-36 | Total |
| Unbooked | 16 | 10 | 10 | 36 |
| Booked | 8 | 49 | 107 | 164 |
| Total | 24 | 59 | 117 | 200 |

Chi-square value- 13.54; P value- 0.001

18% of the cases were unbooked. Out of which, 27% came for their first antenatal checkup at 35-36 weeks.

STATISTICALLY SIGNIFICANT

TABLE 13:

| Latent Period (in hrs) | Neonatal Mortality | | | | Total |
|-----------------------------------|---------------------------|-------------------|-----------------|------------|--------------|
| | RDS | Septicemia | Jaundice | IVH | |
| 0-24 | 13 | 3 | 5 | 4 | 25 |
| 25-72 | 7 | 5 | 1 | 2 | 15 |
| >72 | 5 | 2 | 2 | 0 | 9 |
| Total | 25 | 10 | 8 | 6 | 49 |

Chi-square value- 4.865; P value- 0.56

Among the babies delivered within 72 hrs of rupture of membranes, 80% of them suffered from respiratory distress syndrome. Sepsis was common among the babies born within 24hrs of latent period (70%).

STATISTICALLY NOT SIGNIFICANT

TABLE 14:

| Neonatal Deaths | Gestational Age | | | |
|------------------------|------------------------|--------------|--------------|--------------|
| | 28-31 | 32-34 | 35-36 | Total |
| No deaths | 17 | 57 | 113 | 187 |
| 28-31 | 4 | 0 | 0 | 4 |
| 32-34 | 0 | 7 | 0 | 7 |
| 35-36 | 0 | 2 | 0 | 2 |
| Total | 21 | 66 | 113 | 200 |

Chi-square value- 53.59; P value- 0.001

Out of 11 early neonatal deaths, 7 were in 32-34 weeks and 4 in 28-31 weeks.

STATISTICALLY SIGNIFICANT

TABLE 15:

| Maternal complications | Latent Period | | | |
|-------------------------------|----------------------|--------------|---------------|--------------|
| | 0-24 | 25-72 | >72 | Total |
| Chorioamnionitis | 0 | 7 | 8 | 15 |
| Abruption | 5 | 0 | 2 | 7 |
| wound infection | 6 | 1 | 4 | 11 |
| Total | 11 | 8 | 14 | 33 |

Chi-square value- 16.215; P value- 0.003

Chorioamnionitis was more common in patients who had >72 hrs of latent period and abruption was common among those whose latent period was <24hrs.

STATISTICALLY SIGNIFICANT

TABLE 18:

| Latency (in hrs) | Maternal Morbidity | | |
|-------------------------|---------------------------|------------|--------------|
| | Yes | No | Total |
| 0-24 | 11 | 110 | 121 |
| 25-72 | 12 | 45 | 57 |
| >72 | 16 | 6 | 22 |
| Total | 39 | 161 | 200 |

Chi-square value- 48.15; P value- 0.0001

19.5% had maternal morbidity of which 41% had >72hrs of latent period.

STATISTICALLY SIGNIFICANT

DISCUSSION

Preterm premature rupture of membranes is a fair complication of pregnancy that leads to various maternal and neonatal complications.

The present study was undertaken to identify the risk factors causing pPROM, the outcome of labour and the fetomaternal complications associated with pPROM.

In the present study, 200 patients admitted with pPROM were evaluated. In this study, pPROM was present in 55% of cases in the age group of 21-25 years. Similar results were obtained in a study conducted by Akter et al., [51] (40.33%).

Patients belonging to socio economic status V were observed to be the most common class to get admitted with pPROM with 66% which is comparable with the study conducted by Swathi Pandey [52] which is 61%.

Studies have shown a correlation between low socio-economic status and defects in the amniotic membrane. The factors that lead to pPROM in low socio-economic status include poor hygiene, malnutrition, anemia, stress, over exertion, high parity, recurrent genitourinary infections etc. These factors lead to a decreased antibacterial activity in the amniotic fluid of patients that in turn leads to pPROM.

The major factor that leads to an increase in cases of pPROM among mothers belonging to low socio-economic status is malnutrition. Malnutrition in

turn leads to increased risk of infections that eventually leads to pPROM. Hence the cause of pPROM involves a vicious cycle of malnutrition and infections.

It was noted in the present study that 63% of the patients admitted with pPROM were primigravida. In a study conducted by Swathi Pandey [52] (multigravida 48% and primigravida 52%), and Fatemeh Tavassoli [53] (multigravida 44.1% and primigravida 55.9%), similar results were obtained.

The percentage of booked cases in the present study was found to be 82% while that of unbooked cases was noted to be 18%. These results are comparable to a study conducted by Shwetha Patil et al., [54] where the percentage of unbooked cases was accounting to 31% and booked cases to 69%. There was no significant correlation between the antenatal care and incidence of pPROM which was in contrast to a study done by Shweta Anant Mohokar et al., [55] where there was a strong correlation between the unbooked cases (84%) and the incidence of pPROM.

The unbooked cases receive poor antenatal care that ultimately leads to increased risk of infection to the mother which is a major risk factor for pPROM.

In my study, 10.5% of the study population were in the gestational age of 28-31 weeks, 33% in 32-34 weeks while the majority was observed in the gestational age of 35-36 weeks which was noted to be 56.5%. In a study conducted by Shweta Patil et al., [54] the percentage of pPROM in 28-31 weeks

was 7%, that between 32-34 weeks was 18% and 75% between 35-36 weeks of gestational age, whose results correlate with the present study.

The present study showed that 43.5% of the mothers had early pPROM and 56.5% had late pPROM, which implies that the risk of pPROM increases with increasing gestational age. This can be justified with the fact that pPROM occurs due to mechanical stretching of membranes with increasing gestational age.

In the present study, 60.5% of the population had delivery within 24 hours, which was similar to the results obtained in a study conducted by Shweta Patil et al., [54] (64%) and also in a study conducted by Russels[56] (80%). Only 11% had a latent phase of >3days, 28.5% delivered within 25-72 hours in my study which also correlated with the above-mentioned studies.

Most of the cases (67%) had vaginal delivery while only 33% delivered by caesarean section. In a study conducted by Tahir S et al., [57], the rate of caesarean section was 20%. Out of the 67% of the patients who delivered by vaginal route, 58.5% had a normal vaginal delivery while 6% delivered by assisted breech method and 2.5% of them delivered twins vaginally.

Among the 67% of patients who delivered vaginally, 36% went into spontaneous labour, while induction was done to the rest of 31%. Out of the 31% of the patients induced, 18.5% were induced with misoprostol and 12.5% induced with cerviprime gel.

Fetal distress was found to be the most common indication for LSCS in the present study, which accounted for 14.5%, followed by severe oligohydramnios (8%), previous LSCS (6.5%), CPD (2.5%) and breech presentation (1.5%). In studies conducted by Swathi Pandey [52] and Singhal [58], fetal distress was the most common indication for LSCS.

In this study, 82% of the patients with pPROM gave birth to children weighing >2kg, of which 48.5% of them were in the birth weight of 2-2.5kg. Only 6% had very low birth weight babies and 12% had low birth weight babies. These results obtained were nearly similar to the results in the study by Swetha Anant Mohokar et al., [55] where 26% gave birth to babies weighing 2-2.5kg.

Assessing the risk factors causing pPROM, 70% of the study population had no risk factors while the most common risk factor among others was found to be breech presentation (7.5%). Gunn et al., [12] also showed similar results in his study where breech presentation was the most common risk factor. In the present study, previous history of pPROM was the second commonest risk factor with (7%), followed by history of recent coitus (6%), polyhydramnios (5.5%), twins and UTI (2% each).

Amniotic fluid culture sensitivity was done in all the patients and there was no growth in cultures in 79% of them. Among the 21% of the positive cases, E. coli was found to be the most common organism (12%). Klebsiella(4.5%), Proteus (3%), Pseudomonas aeruginosa (1%) and S. aureus

(0.5%) were the other organisms that were isolated. The commonest organism isolated by Swathi Pandey [52] in cervical swab was E. coli and by Kamala Jayaram [59] was E. coli, Staphylococci, Streptococci and atypical coliforms.

Among 200 cases, maternal complications were present only in 16% of the population of which wound infection was predominating (5.5%). Puerperal pyrexia was present in 3.5% of my study population and 4% had chorioamnionitis. A study by Artal K [60] showed the incidence of puerperal pyrexia to be 13% and chorioamnionitis to be 3%.

24% of the babies born to pPROM mothers were admitted in NICU for various complications in my study. These results correlated with Shweta Patil et al., [54] where the percentage of NICU admissions was 36%. NICU admissions of 24% included babies born by normal vaginal delivery and LSCS.

Out of the 24% babies admitted, the most common cause for neonatal morbidity was respiratory distress syndrome (12%), followed by septicemia (5%), jaundice (4%), IVH (3%).

Out of 200 cases, 13 neonatal deaths were seen of which 11 of them were early preterm babies. A study by Swetha Anant Mohokar[55] showed 15% mortality among neonates.

SUMMARY AND CONCLUSION

- pPROM is a fair complication of pregnancy.
- In the present study, the most common age group to suffer from pPROM was 21-25 years.
- Mothers belonging to socioeconomic status V had more risk of developing pPROM.
- Primigravida was found to be another factor contributing to pPROM.
- Most of the antenatal cases were booked and hence there was no correlation between the booked status and chance of developing pPROM.
- 44% of the unbooked cases came for first antenatal checkup only in gestational age of 35-36 weeks.
- Mothers with gestational age of 35-36 weeks suffered from pPROM more commonly compared to other gestational ages.
- The time interval between the rupture of membranes and delivery was <24 hours in most of the cases. A very few cases delivered after 72 hours of membrane rupture.
- The short latent period of less than 24 hours was observed to be common in 35-36 weeks of gestational age.
- The babies who were born with latent period of <24 hours suffered from respiratory distress syndrome more commonly and those born with latent period of >72 hours were found to be suffering from sepsis.

- Vaginal mode of delivery was common among 67% of the cases. Out of the 67%, 36% of the mothers had spontaneous delivery, while the rest of them had to undergo vaginal induction with cerviprime gel or misoprostol.
- Spontaneous progression of labour was more common in 35-36 weeks gestational age group while induction had to be done to mothers with lesser gestational age.
- Only 33% had to undergo LSCS of which the most common indication was fetal distress in all gestational age groups.
- Previous LSCS was the second common indication for LSCS which was common among 35-36 weeks of gestational age.
- The babies born to mothers admitted with pPROM were in the birth weight of 2-2.5 kg more commonly. Only 6% of the babies born were of <1.5 kg.
- Very low birth weight babies were born to mothers with less gestational age while mothers with 35-36 weeks of gestational age gave birth to babies weighing >2.5 kg.
- Out of 200 cases in the study, a very few had risk factors while 70% had no risk factors to develop pPROM.
- Of the risk factors evaluated, breech presentation, previous history of pPROM were common among the mothers. Others included, history of recent coitus, polyhydramnios, twins and urinary tract infection.
- Mothers with gestational age of 32-34 weeks had more risk factors compared to mothers with gestational age of 28-31 weeks and 35-36 weeks.

- Amniotic fluid culture sensitivity results showed no growth in 79% of the cases. E. coli was the commonest organism to be isolated while culture results also showed growths of Klebsiella, Pseudomonas aeruginosa, Proteus and S. aureus.
- Sepsis was common in 28-31 weeks group.
- Most of the mothers with pPROM had no complications. The most common complication was chorioamnionitis followed by puerperal pyrexia.
- Maternal morbidity was seen commonly in mothers with gestational age of 28-31 weeks.
- Only 24% of the babies born had NICU admissions for complaints of respiratory distress, jaundice, septicemia and intraventricular hemorrhage.
- Most of the babies born to mothers with gestational age of 28-31 weeks required NICU admissions.
- Respiratory distress syndrome was the most common neonatal complication followed by septicemia.
- Neonatal complications were observed to be more in the mothers with gestational age of 32-34 weeks.
- There were 13 neonatal deaths in this study, while 11 of them were early preterm babies. Most of them died due to respiratory distress syndrome.

SUGGESTIONS

- Though pPROM is a common complication of pregnancy, its consequences can be prevented by the use of antibiotics, corticosteroids etc.
- There were no risk factors in most of the mothers, but the risk of breech presentation can be avoided by external cephalic version, avoidance of coitus in the later weeks of pregnancy reduces the risk of pPROM.
- Mothers diagnosed to have polyhydramnios or with multiple gestation are prone to suffer from pPROM, hence adequate rest and proper care can reduce pPROM to an extent.
- Urinary tract infections can be treated by administration of antibiotics that will reduce pPROM.
- The use of antibiotics in the latent period can reduce the maternal complications like chorioamnionitis and puerperal pyrexia. Septicemia in the neonates can also be prevented by the use of antibiotics.
- Administration of corticosteroids in pPROM <34 weeks reduces the neonatal morbidity that includes respiratory distress syndrome which is the most common cause of neonatal deaths.
- Neonatal care facilities can be improved to manage neonatal emergencies so as to reduce neonatal deaths.

ABBREVIATION

pPROM – Preterm Pre mature Rupture of the membrane

SES – Socio Economic Status

S.AUREUS – Staphylococcus Aureus

P.AEURUGINOSA – Pseudomonos Aeuruginosa

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H/o recent coitus:

Immunized/not:

H/o suggestive of infection:

H/o recurrence of PROM:

Past H/o

Medical

Surgical

HT

DM

Any cervical Sx.

General Examination

Ht

Weight

Nutritional status

Anemia

PR

BP

CVS

RS

Obstetric Examination

P/A

Speculum Examination

P/V

Lab Investigations:

Hb%

TC

DC

Urine Alb.

Urine sugar Urine C/S

Cervical Swab C/S

Amniotic Fluid C/S

Fetal Blood C/S (selected)

Mode of delivery:

Vaginal

Assisted breech

Twins vaginal delivery

LSCS

Mode of Vaginal delivery

Spontaneous

Induced

Cerviprim

misoprostol

Indication for LSCS

Outcome:

Maternal:

Any Complications

Fetal:

Preterm/Term

Wt. of Baby

APGAR 1min/5min

NICU Admission

Any complications

INFORMATION SHEET

Place of the study: Chengalpattu medical college

Name of the investigator: Dr. A. DEVI

Name of the participant:

Age: Hospital No:

- We are conducting a study on “**Maternal and Perinatal Outcome in Cases of Preterm Premature Rupture of Membrane (PPROM) - A Prospective Study**”
- The purpose of the study is to identify the risk factors for Rupture of Membrane and fetomaternal outcome.
- The privacy of the patient in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.
- Taking part in the study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time. Your decision will not result in any loss of benefits to which you are otherwise entitled.
- The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Principal investigator:

Dr. A. DEVI,

PG Student,

Department of Obstetrics and Gynaecology

Chengalpattu medical college,

Chengalpattu.

Signature of investigator

Signature of patient /guardian

Date:

Chengalpattu

INFORMED CONSENT FORM

Place of the study: Chengalpattu medical college

Title of the study: Maternal and Perinatal Outcome in Cases of Preterm Premature Rupture of Membrane (PPROM) - A Prospective Study

Name of the investigator: Dr. A. DEVI

Name of the participant: Age: Hospital No:

I have read and understood the patient information sheet provided to me regarding my participation in the study.

I have been explained about the nature of the study and had my questions answered to my satisfaction.

I have been explained about my rights and responsibilities by the investigator.

I will cooperate with the investigator and undergo clinical tests subjected during the study whole heartedly.

I have been advised about the risks associated with my participation in this study.

I am aware of the fact that I can opt out of the study at any time without having to give any reason and this will not affect my future treatment in this hospital.

I hereby give permission to the investigator to release the information obtained from me as result of participation in this study to medical journals, conference proceedings.

I understand that my information will be kept confidentially if my data are publicly presented / published.

I have decided to participate in the research study I am awrwe that if I have any question during the study I should contact the investigator.

By signing this consent form, I attest that the information given in this document has been clearly explained to me and understood by me, I will be given a copy of this consent document.

Name and Signature / thumb impression of the patient / guardian:

Name:

Signature:

Date:

Name and Signature of the investigator:

Name:

Signature:

Date:

புறநாடு நடைபெற்று வருகின்றது.

புறநாடு நடைபெற்று வருகின்றது.

புறநாடு நடைபெற்று வருகின்றது.

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செங்கல்பட்டு அரசு பொது மருத்துவமனையில் மகப்பேறு மற்றும் மகளிர் நல துறையில் ஆராய்ச்சி நடைபெற்றுவருகின்றது.

நீங்களும் இந்த ஆராய்ச்சியில் பங்கேற்க நாங்கள் விரும்புகிறோம். இதனால் உங்களுக்கு எந்த பாதிப்பும் ஏற்படாது என்பதை தெரிவித்துக் கொள்கிறோம்.

முடிவுகளை அல்லது கருத்துகளை வெளியிடும் போது அல்லது ஆராய்ச்சியின் போது உங்களது பெயரையோ அல்லது அடையாளங்களையோ வெளியிடமாட்டோம் என்பதையும் தெரிவித்துக்கொள்கிறோம்.

இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களுடைய விருப்பத்தின்பேரில் தான் இருக்கிறது. மேலும் நீங்கள் எந்தநேரமும் இந்த ஆராய்ச்சியிலிருந்து பின்வாங்கலாம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

இந்த சிறப்பு பரிசோதனைகளின் முடிவுகளை ஆராய்ச்சியின் முடிவின் போது தங்களுக்கு அறிவிப்போம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

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|----|--------------|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 9 | Suganya | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 10 | Ramzan | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 11 | Mary | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 0 |
| 12 | Ponni | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 13 | Nagavalli | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 14 | Jamuna | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 |
| 15 | Nasreen | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 16 | Anitha | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 17 | Sivagami | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 18 | Aarthi | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 19 | Revathi | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 20 | Valli | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 1 |
| 21 | Govindammal | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 22 | Devaki | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 |
| 23 | Anjalai | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 24 | Priya | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 |
| 25 | Rani | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 |
| 26 | Kala | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 27 | Mallika | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 28 | Rajeshwari | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 29 | Chithra | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 30 | Shameem | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 1 |
| 31 | Sumathi | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 32 | Kamakshi | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 33 | Vellachi | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 34 | Suganthi | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 35 | Nithya | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 |
| 36 | Ellammal | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 37 | Kuttyammal | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 1 |
| 38 | Maheshwari | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 39 | Naseema | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 40 | Aaysha | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 0 |
| 41 | Saritha | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 |
| 42 | Fathima | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 43 | Buvaneshwari | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 44 | Ambika | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 45 | Vasanthi | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 |
| 46 | Mallika | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 |
| 47 | Sangeetha | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 48 | Saritha | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 |
| 49 | Anjalai | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 50 | Jothi | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 51 | Nirmala | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 52 | Meenakshi | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 53 | Kalyani | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |

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|----|------------|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 54 | Renuga | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 55 | Priya | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 |
| 56 | Ratha | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 57 | Latha | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 58 | Komala | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 |
| 59 | Marry | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 60 | Ganga | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 61 | Muniyammal | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 62 | Chithra | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 |
| 63 | Sasikala | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 |
| 64 | Malini | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 |
| 65 | Selvi | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 66 | Veerammal | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 67 | Prema | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 |
| 68 | Sumathi | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 69 | Prathiba | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 70 | Sulochana | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 71 | Malar | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 |
| 72 | Sankari | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 73 | Karthika | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 74 | Sivagami | 0 | 1 | | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 75 | Brindha | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 76 | Kalaivani | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 77 | Indra | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 78 | Suganya | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 79 | Bharathi | 0 | 0 | | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 80 | Kumutha | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 81 | Pattammal | 1 | 0 | | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 |
| 82 | malathi | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 83 | Rasathi | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 84 | Papitha | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 1 |
| 85 | Karpagam | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 86 | Valli | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 87 | Kokila | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 |
| 88 | Bhavani | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 |
| 89 | Indhumathi | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 90 | Vijaya | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 91 | Meena | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 92 | Chithra | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 93 | Geetha | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 94 | Shuba | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 95 | Akila | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 96 | Fathima | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 97 | Shobana | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 1 |
| 98 | Rani | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 1 | 0 | 0 | 0 | 0 |

