"MATERNAL AND FETAL OUTCOME IN PREGNANCY COMPLICATED BY PREMATURE RUPTURE OF MEMBRANES"

BY

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Under the guidance of

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ABSTRACT

Background: Premature rupture of membranes (PROM) refers to rupture of fetal membranes prior to the onset of labor and can occur at any gestational age even after 42 weeks of gestation. For this reason it is also called as pre labor ROM.

A number of risk factors predisposing for spontaneous PROM:

- Infection- Chorioamnionitis, urinary tract Infection, lower genital tract infection
- Multiple gestation
- Polyhydramnios
- Travelling
- Invasive procedure like amniocentesis, chorionic villus sampling, cervical encirclage

Maternal complications include intra amniotic infections occurring in 13% to 60%, abruption placentae of approximately 6%, post partum endometritis in 2% to 13% and the risk of cesarean section and its associated surgical complications.

Neonatal complications relate primarily to the gestational age of rupture of membranes. PROM is associated with 4 fold increase in perinatal mortality and 3 fold increase in neonatal morbidity, including respiratory distress syndrome (RDS), occurs in 10-40% and responsible for 40 -70% neonatal death. Other complications like fetal pulmonary hypoplasia, skeletal

deformities complicate 12%, cord prolapse cesarean delivery for presentation. Infection, cord prolapse, cord accident and other contribute to 1%-2% of still birth after PROM.

Objectives: This study was designed to evaluate the various maternal complications due to PROM and the effect of premature rupture of membranes on fetal outcome.

Method: It is a Randomized Prospective study conducted in 120 pregnant women with premature rupture of membranes from gestational age 34 weeks onwards attending department of Obstetrics and Gynecology of BLDEU's Shri B M Patil Medical College, Bijapur

After taking in to consideration the inclusion and exclusion criteria, all 120 patients were included into the study during the period from October 2009 to September 2011. Clinical details were noted, thorough general physical and systemic examination and laboratory examination was done. Maternal and fetal outcome was analyzed in each case

Result: A total of 120 patients of proved PROM were studied. Incidence of PROM was 7.44%. The incidence of prematurity was 14.16% , significantly higher than the overall incidence.

Conservative management could be undertaken in only 3.33%. Active termination of pregnancy had to be undertaken in 25.83% of the cases, while 70.84% of the patients went into labour spontaneously.

Overall Caesarean section rate in PROM was 34.16%, while the corrected rate was 20%.

The overall incidence of amnionitis was 6.66% and postpartum maternal morbidity was 15.83%.

Incidence of perinatal mortality in PROM was 5.73%. Perinatal infection was responsible for 42.86% of the perinatal deaths, while RDS was responsible for 57.14% of perinatal deaths. Prematurity with its hazards - infection and RDS were responsible for 85.72% of perinatal deaths.

Conclusion: Premature rupture of membranes is associated with maternal complications like amnionitis, increased incidence of LSCS, puerperal fever, and wound infection. PROM has effect on fetal outcome in terms of perinatal morbidity in the form of prematurity, RDS, cord prolapse, infections. And also effects the perinatal mortality.

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INTRODUCTION

Premature rupture of membranes (PROM) is one of the most common complications of pregnancy that has major impact on fetal and maternal outcome, it is one of the commonest clinical events where a traditional pregnancy can turn into a high risk situation for the mother as well as fetus. The obstetrician is invariably in a dilemma regarding the future plan of management and PROM still remains controversial and challenging.

A prospective study of the patients presenting with spontaneous premature rupture of the membranes was undertaken at BLDEU's Shri B.M Patil Medical College and Research centre, Bijapur, to learn more about this common obstetric complication, the way it presents and the maternal and fetal outcome in pregnancies complicated by premature rupture of membranes.

AIMS

1) To identify and study the various maternal complications due to PROM.
2) To study the effect of PROM on fetal outcome.

REVIEW OF LITERATURE

Spontaneous rupture of membranes before the onset of true labor pains is well known entity in obstetrics. The earliest contribution to the subject comes from Margaret Schulze in 1929. She found the association of premature rupture of membranes with increased fetal mortality, after analyzing 600 cases. She also found an increased incidence of maternal morbidity associated with premature rupture of membranes.

DEFINITION

The Definition of premature rupture of membranes (PROM) itself is controversial. By and large there are two sets of definitions. Many authors including Aaron B[1], Atkins[4], Calkins[5], Gunn[6], Naeye[8] ,Patrick Duff[9] establish the diagnosis when spontaneous rupture occurs prior to the onset of labour at any gestational age even at 42 weeks gestation. Also referred to as pre labor rupture of membranes (ROM). When PROM occurs before 37 weeks of gestational age called as preterm premature rupture of membranes (PPROM), responsible for 30% of preterm deliveries[10]. Prolonged PROM referred to when PROM greater than 24 hours which is associated with increased risk of ascending infection.

Other authors like Breese[11], BurcheH[12], Lanier[13], Friedman[14], Jiwane[15], and James Harger[16] diagnose premature rupture of the fetal membranes only when a specified latent period varying from 1 hour to 12 hours had elapsed following rupture of membranes before the onset of labor.

INCIDENCE

Because of the various definitions used in establishing the diagnosis of PROM, the incidence reported in the literature varies widely from 2.6% to 21%. Bhalerao and Desai[17] reported an incidence between 7 and 12%. Friedman and McElin[14] in their review article quoted the incidence as 6.6 to 13.9% of all patients with spontaneous rupture of the membrane at least one hour prior to the onset of labour at term or earlier. Gunn[6] et al in their review article reported the range of incidence as 2.7 to 17% with the majority failing between 7 to 12%, while in Gunn's own series incidence of spontaneous PROM was 10.7% of total number of deliveries. The incidence of PROM according to other authors ranged from 2.6% to 21% Lenihan[18] , Patrick Duff[9] , Vintzileos[20] , Kodkany[19] , Jiwane[15] , Anjana Devi[21] and Dare[22]

AETIOL OGY

Various suggestions are made in the literature as the etiological factors for spontaneous PROM. Evaldson G et al[23] reported an increased risk of preterm PROM in women whose first pregnancy was voluntarily terminated by suction curettage.

Knox and Hoerner[24] suggested that infections cause some premature rupture by weakening the membranes. This is supported by Naeye and Peters[25] findings that acute chorioamnionitis was twice as frequent in membranes that ruptured just before the onset of preterm labor as in those that ruptured after the labour began.

Richard Naeye[8] analyzed five risk factors – Cigarette smoking, coitus, parity, prior surgery of the cervix and chorioamnionitis, for their possible roles in PROM. His results indicate that the PROM had a recurrence rate of 21% in the next

pregnancy. Similarly coitus and chorioamnionitis together were strongly predisposing to PROM in preterm as well as full term pregnancies. Prior damage to the cervix: - cervical canal by surgery or other instrumentation predisposes to. The relation between smoking and PROM was not exactly proved.

Lenihan[18] postulated that a digital pelvic examination could possibly result in inoculation of bacteria into the endocervical canal. This could conceivably increase the incidence of PROM K.A. Jiwane[15] vowed a definite relationship between routine antenatal pelvic examination and premature rupture of membranes.

Bourne[26] suggested that localized connective tissue necrosis is usually present near the site of premature rupture. Artal[27] et al postulated that the damage is caused by enzymatic depolymerization of the collagen fibres, which leads to localized damage of the membranes.

In a study conducted by Harger[16] et al, bleeding in the first or third trimester, smoking more than 10 cigarettes per day and previous gynecologic surgery were found to be associated with an increased risk of preterm premature rupture of membranes.

According to Iams[28] symptoms like menstrual like cramps, backache, pelvic pressure and an increased amount of vaginal discharge, visualization of the flow of the amniotic fluid from the cervical os and/or its pooling in the posteriorivaginal fornix following fundal pressure. Either of these findings established a definitive diagnosis.

LABORATORY TESTS

Whenever clinical diagnosis of PROM is in doubt, there are numerous laboratory procedures described in establishing the presence of amniotic fluid.

These tests were based on certain guidelines:

a) Study of changes in vaginal pH.

b) Staining techniques to identify fetal fat globules within or outside fetal cells.

c) Cytological identification of fetal fat cells.

d) Recognition of crystallization pattern of amniotic fluid.

e) Heating of endocervical specimen over an alcohol burner to evaporate water.

MICROSCOPIC FETAL CELL IDENTIFICATION:

A literature review by Friedman et al.[14] described the first microscopic technique for identification of fetal particles in amniotic fluid, developed by Philipp et al in 1929 and published in the German literature. In this technique, fetal lanugo hairs were identified in amniotic fluid. This was presumed to be incontrovertible evidence of membrane rupture when identified in vaginal secretions. However, because of the scanty amounts of fetal lanugo hair in amniotic fluid and the fact that such hair was present in amniotic fluid only later in pregnancy, this method never gained popularity.

Kittrich introduced the **Nile Blue test** for diagnosis of PROM. The test is based on the staining of neutral fat by oxazone present in commercial Nile Blue Sulphate stain. This test was later used by Brosens and Gorden[29] .Fetal cells in the vaginal smear stained with Nile Blue Sulphate stain are identified as anucleate orange stained cells occurring either singly or in clusters; other cells, Vaginal Squames, pus

cells or erythrocytes stain blue. Maternal cells occasionally contain fat droplets, but their background color remains blue. Brosens and Gorden[29] further stated that in the presence of intact membranes, no false positive results were recorded. False negative results are likely to occur before 36 weeks of gestation. At this time of pregnancy the percentage of orange anucleate cells in the liquor amnii is less than 10% and less than 1% before 32 weeks. After 36 weeks the relative as well as the absolute number of these cells rapidly increases, and at term the great majority of cells in the liquor are anucleate orange staining cells.

Friedman and McElin[14] found the test to be positive in 86.4% of cases. Additionally since less than 10% of fetal fat-containing cells are present before 36 weeks; Nile blue test was useless before that time. In a study conducted by Gaucherand P[30] the Nile blue test was positive in 88% of cases with frank rupture of membranes and in 90% cases where membrane rupture was clinically doubtful. Bourgeois[31] used Sudan III stain for the identification of fat drop in the vernix caseosa. Hopman[32,33], reported that the fetal epithelial cells as they occur in the vernix caseosa were made up of translucent polygonal, anucleate cells, although in places nuclear remnants were still visible as uncolored residue. The protoplasm was slightly granular and furrowed by a network of fine little canals which communicate with each other.

Freidman and McElin[14] analyzing vaginal smears stained by Papanicolaou technique reported that Pap smear test had an accuracy of 96.7% for diagnosing PROM. They described that immediately after rupture of membranes smear showed blue squamous cells with gray white or light ye)low polygonal, translucent anucleate fetal cells. If the membranes were intact, primary acidophilic staining vaginal epithelial cells predominated.

Averette[34] introduced another method for identification of fetal squamous cells. The cervical smear was fixed with 95% ethyl alcohol for at least 60 seconds and then covered immediately with 0.25% solution of Pinacyanole Chloride in 50% Methyl alcohol for 20 seconds. They observed that polygon shaped fetal squamous cells (vernix caseosa cells) stained blue white or pale lavender with Pinacyanole Chloride, while vaginal cells stained dark blue or purple. According to Averette[34] et al, differentiation of fetal cells from hypercornified vaginal cells posed no problem as the vernix caseosa cells are more transparent as compared to denser, compact and opaque vaginal cells. Further a delicate canal system is visible in vernix caseosa ceils when examined under a high power microscope. A canal system, if present in the hypercornified vaginal cells, is coarse and difficult to outline. They claimed the method to be simple, quick (needed only 80 seconds) and the accuracy was 97 %. Brosens and Gorden[29] commented that the vernitest (Pinacyanole Chloride) though simple to perform, the difference in staining reaction between fetal and maternal squames is marginal, and false positive results were obtained by the contamination of the specimen with vulval secretions. Friedman and McElin[14] in their study reported accuracy of 79% with Pinacyanole Chloride. Kushner et al[35] using the 10 seconds acridine orange fluorescent stain of Riva and Turner observed with ultraviolet microscopy that the anucleate fetal squamous cells stained green or reddish green and possessed a distinct morphology. Friedman and McElin[14] reported 65.5% accuracy with this test.

Goldfine[36] suggested the use of litmus paper sticks soaked in 0.2% alcohol bromothymos blue solution, which changes to green colour in the presence of amniotic fluid. Because of the difference in pH of vaginal secretions (4.5 to 5.5) and

amniotic fluid (7.0 to 7.5), it was rightly assumed that the pH of vaginal secretions would rise when contaminated by escaping amniotic fluid.[37,38]. This assumption prompted preliminary experiments with bromthymol blue dye and, later, nitrazine applicators. Although similar in principle to bromthymol blue, a reported advantage of nitrazine is the complete change in colour of the applicator when exposed to amniotic fluid in vaginal secretions.[36] Impregnated with sodium-dinitro-phenylozonapthol-disulphonate, nitrazine paper showed promising early results in detecting membrane rupture, with accuracies of 100%[37] and 98.9%[38] in clinically ruptured cases, and similarly high rates of accuracy among intact cases. This preliminary high optimism regarding nitrazine testing decreased by the 1960s,[39,40] because of false positive results from vaginal infections, blood, semen, alkaline urine or alkaline antiseptics, and false negatives in cases of minimal leakage from chronic membrane rupture or "high leak" of the membranes.[41]

Colour charges are interpreted as follows :

Probably intact membranes

Yellow	pH	5.0
Olive yellow	pH	5.5
Olive green	pH	6.0

Ruptured membranes -

Blue green	pH	6.5
Blue gray	pH	7.0
Deep blue	pH	7.5

According to Abe[38], the accuracy of the Nitrazine test was 97.7%. In a study conducted by Friedman and McElin[14], accuracy with Nitrazine test was 93.3%. They also reported that the Nitrazine test was often false positive in the presence of cervicitis, vaginitis, semen, alkaline urine, soap, antiseptic solutions and blood. The test was often false negative after several hours or days of rupture of membranes.

Papanicolaou[42] was the first person to describe the fern phenomenon or arborizatian test in dried cervical mucus. Kardos et al described the crystallization pattern of the amniotic fluid and described its utility in the diagnosis of ruptured membranes. Marcel Ferron[43] stated that the crystalline pattern apparently depends upon the relative concentration of sodium chloride and protein present in the amniotic fluid. They also stated that the crystallization takes place in any trimester of pregnancy. Arborization was defined as tree-like branching typified by that seen in cervical mucus pattern. Amniotic fluid forms more discrete crystallization patterns which may coalesce, although separate crystallization centers can be distinguished. They also noted that cervical mucus did show crystallization in a few patients during pregnancy with intact membrane but the pattern is atypical which could be easily distinguished from discrete crystallization pattern of amniotic fluid. Marcel[43] reported an accuracy of 98.5% with this test for detecting PROM. It was simple, quick and required no staining or cytology. Anjaneyulu and Likhite[44] in quoted an accuracy of 86.6% with this test. They stated that occasionally it is possible that contamination with cervical mucus could be responsible for a false positive crystallization test during pregnancy. To distinguish it, they stated that cervical mucus forms heavy, dark and wide Arborization pattern, whereas with amniotic fluid the pattern is thin, delicate and discrete.

In a study conducted by Gupta and Gupta[45] this test had 92.5 % accuracy. Friedman and McElin[14] reported 96.4 % accuracy with the crystallization test, however, the test was false negative in the presence of blood, meconium and heavy discharge and occasionally false positive with cervical mucus and urine crystals. Gaucherand[30] reported 87% accuracy with crystallization test.

Max Borten[46] suggested that ferning of amniotic fluid was demonstrated as early as 6,1/2 weeks of gestation and also stated that crystallization is a property of amniotic fluid throughout pregnancy.

This test might give false negative results especially when amniotic fluid is mixed with blood (particularly when the ratio of blood to fluid exceeds 1:10) and surgical soap when mixed in equal parts with amniotic fluid. Urine may show false positive test which can be excluded by use of speculum to expose cervix and vaginal vault and collecting fluid from cervix or posterior vaginal fornix. The time between rupture of the membranes and obtaining the smear may be important according to Ruck. In his series, the accuracy of the test decreased to 90% or less with a greater than 4 hour interval between rupture and obtaining the smear.

Roger Smith[47] introduced a new technique for detection of PROM. Amniocentesis was performed in a routine manner using a 22 gauge spinal needle. Once the amniotic cavity was entered, approximately 2 to 5 cc of a sterile 10% solution of sodium fluroscein was slowly introduced into the amniotic sac. At 15 and 45 minutes after the injection, a speculum examination of the cervix was carried out using a long-wave ultraviolet light. The presence of yellow-green fluorescent fluid leaking from the cervix or in the vaginal vault is positive evidence of rupture of the membranes. Direct visualization of the leaking fluid is occasionally facilitated by

fundal pressure or having the patient bear down. He reported 100% accuracy with this test.

Iannetta[48] described a new test in diagnosing PROM. This test was called '**Evaporation test'**. It was based on the heating of endocervical material on a glass slide to evaporate water, thus leaving a white residue if amniotic fluid is present and a brown precipitate if it is not. The brown colour is due to the charring of the protein in the cervical mucus.

Schiotz[49] evaluated the above test and found an accuracy of 89.5%. He also stated that, this test could be used as early as 26 weeks.

This test was later confirmed by Chitra Sarin[50] who showed a accuracy of 99%.

Manning et al[51] initially described a technique for measuring by ultrasound the deepest vertical pool of amniotic fluid in patients with intrauterine growth restriction. This method was later used to assess membrane rupture; it was shown that ultrasound quantification of the deepest amniotic fluid pocket is of poor quality in confirming membrane rupture.[52] No significant difference was found in the mean depth of amniotic fluid pocket between 100 patients with confirmed term PROM and 51 patients with intact membranes.[52] Oligohydramnios may not be detected in patients with confirmed PROM, possibly because drainage may become intermittent or even stop once the presenting part descends and acts as a plug, preventing further drainage.[52] Robson et al. suggested that a significant amount of amniotic fluid needs to drain rapidly and continuously for oligohydramnios to occur, especially because the fluid is replaced to varying degrees by the fetus.[52] Erdemoglu et al.[53] showed that a reduction in the four-quadrant AFI below 80 mm did not reliably identify cases of

suspected membrane rupture by history with negative visualization of fluid by speculum examination. The measurement of AFI offers no advantage over measurement of a single vertical pocket of fluid in cases where ultrasound is used to evaluate possible membrane rupture.

In 1970, amniocentesis with injection of dye to confirm amniotic membrane rupture had become a commonplace procedure; it was thought to be safe and had high patient acceptability rates.[54] Prior to amniocentesis, intravenous injection of radioisotope was performed for placental localization, and the amnio-injection was performed under local anaesthesia. Interestingly, the two reported disadvantages of the procedure at that time were related to difficulty in diagnosis in the presence of meconium-stained fluid and the possibility of neonatal skin staining for 48 hours after dye injection.[54] Several types of stains have been used for amnio-injections, with safety hazards reported only for methylene blue. Although ultrasonographically guided transabdominal instillation of indigo carmine dye (1 mL of dye in 9 mL of sterile normal saline) and observation for fluid passage transvaginally is designated an "unequivocal" diagnostic method for confirmation of membrane rupture,14 this invasive test carries increased maternal and fetal risk. Inherent risks of intra-amniotic dye injection include trauma, bleeding, infection, and preterm labour.[55] While strengthening diagnostic certainty, a "negative dye test" may occur if the membranes seal after previous amniotic fluid leakage.

Initially isolated in Moscow in 1975[56] (Placental Alpha-Microglobulin-1)PAMG-1 has undergone recent evaluation for diagnostic testing in PPROM. This 34kDa placental glycoprotein is abundant in amniotic fluid (2000–25 000 ng/mL), with much lower concentrations in maternal blood (5–25 ng/mL). The protein is present in negligible amounts in cervicovaginal secretions with intact membranes

(0.05–0.2ng/mL).[57] The 1000- to 10 000-fold difference in concentration between amniotic fluid and cervicovaginal secretions stimulated interest in a PAMG-1 immunoassay. Marketed as **AmniSure** (AmniSure International, Cambridge, MA), the assay's minimum detection threshold for PAMG-1 is 5 ng/mL, sufficient for 99% accuracy with minimal false negatives. PAMG-1 can be detected with as little as 0.25 μL of amniotic fluid in 1 mL of vaginal secretions.[57] In the presence of blood or vaginitis, the background level of PAMG-1 can occasionally reach a maximum of 3 ng/mL.[57] False-positive results with use of the AmniSure assay seem very unlikely, although these may appear with increased use.[58] Further, assay of PAMG-1 appears to be reliable over a wide range of gestational ages (11 to 42 weeks), and proved superior to conventional combined clinical tests involving visualization of fluid pooling in the posterior fornix, arborization, and nitrazine testing.[58] This test is currently available in Europe and was recently approved by the Food and Drug Administration for use in the United States. AmniSure is a novel rapid, non-invasive bedside test that may be very helpful in diagnosis of difficult cases without visible leakage. Further studies are needed to assess the reliability of the test according to the time from membrane rupture.

Efforts to be able to confirm chorioamniotic membrane rupture with minute amounts of amniotic fluid have recently led to the development of the absorbent pad, **AmnioSense**. This 12 _ 4 cm pad has a central strip that changes colour on contact with fluid with a pH > 5.2.15,[59] After contact with urine, the strip reverts to its riginal colour when dry. This is due to the detachment of conjugate- based nitrazine molecules by the urine ammonium ions. AmnioSense has undergone cytotoxicity and skin irritation and sensitization testing, and it complies with the US Pharmacopoeia

Guidelines. In a study of 34 women presenting with suspected membrane rupture, the AmnioSense pad initially showed 100% sensitivity; overall specificity was 75%, but when women with bacterial vaginosis or Trichomonas vaginalis were excluded from analysis, the specificity increased to 90%.

In a recent study, Mulhair et al.[59] compared the reliability of the absorbent pad test with a standard of amniotic fluid pooling in the posterior fornix on speculum examination in a cohort of 139 women. They found a specificity of 65.0% and a sensitivity of 98.3% for the AmnioSense pad. The two studies of the absorbent pad currently available[59] suggest that a negative AmnioSense result indicates intact membranes in term and preterm gestations in 99% of cases.

A positive result, however, suggests only a 70% chance of ruptured membranes, and thereby warrants confirmation or further investigations to identify infections[59]. It remains unknown whether potential confounding substances such as semen, blood, or meconium may be distinguished from amniotic fluid by the AmnioSense pad test. As women with negative pad checks are unlikely to have ruptured membranes, this would imply decreased need for an uncomfortable and intrusive speculum examination.

EFFECTS OF PROM ON LABOUR

Sacks[60] suggested that PROM is followed by spontaneous labor within 48 hours in 70 to 90 percent of the cases.

After premature rupture of membranes labor occurred spontaneously within the first 24 hours in 51 to 95% of the cases reported in the literature, but most authors reported figure between 80 and 90% Calkins[5], Breese[11], Gunn[6,7] et al, reported

spontaneous labor within 24 hours following PROM in 81% of women with mature birth weight infants in 51% of those with premature birth weight infants and 26% of those whose infants were immature. According to Kodkany[19] spontaneous onset of labor was seen in 77% of cases with PROM. In general, -the latent period was longer than 24 hours as the length of gestation decreased. (Gunn[6,7].)

EFFECT OF PROM ON THE STAGES OF LABOUR

Calkins[5] and Breese [11] found the first stage of labour to be moderately shorter in both primi and multigravidae when PROM had occured but the second stage of labor did not appear significantly altered. Gunn[6,7] et al noted that only 2.6% of the patients had prolonged first stage of labor (greater than 20 hours), while 3% had a prolonged second stage of labor (more than 2 hours). According to Anjana Devi[21] there was no difference in the duration of first and second stages of labour between PROM and control group.

CAESAREAN SECTION

The incidence of caesarean section in patient with PROM reported in the literature ranged from 1 to 19% [Breese[11], Gunn[6,7] , Kodkany[19]]. Gunn[6] noted the caesarean section rate of 4.1%. The majority of these operations were performed because of a history of prior caesarean.

Kodkany[19] reported a caesarean section rate of 19%, but most of them were for other obstetrical conditions like C.P.D., fetal distress, malpresentation, deep transverse arrest. Anjana Devi and Reddi Rani[21] , in a study of 104 patients with PROM reported the caesarean section rate to be 45.2%, main indication being

prolonged labour with unfavorable cervix. LSCS for undiagnosed CPD due to big babies was the indication in 4.8% of the cases.

MANAGEMENT

The management of the patient with premature rupture of membranes remains a major dilemma for the obstetrician. It is difficult to make a decision between the risks of prematurity associated with prompt delivery and the progressive risk of infection associated with prolonged observation. This is quite evident from the published literature on the subject.

Calkins[5] in his study of PROM realized the importance of a long lag period (latent period) and wrote that the management was largely concerned with the prevention of long lag period. He further advised that if labour failed to follow spontaneous PROM promptly, it should be induced medically with I.V. Oxytocin in those cases in which gestation was at or near term. All patients of PROM were hospitalized and kept under close observation and adequate amount of penicillin alone or in combination with other antibiotics were administered.

Varner[61] in suggested that in predominantly middle-class, well-nourished population, the risk of perinatal infection associated with conservative management of preterm premature rupture of the membranes was low. Furthermore with careful in-hospital management, the incidence of amnionitis prior to labour was not significantly different than in those patients delivered prior to 24 hours of rupture of the membranes. He further stated that the major cause of neonatal morbidity and mortality in his study was the respiratory distress syndrome. The percentage of neonates with RDS was less after rupture of the membranes of more than 24 hours. He concluded that in patients with PROM, a conservative management plan

may be employed with minimal risk of perinatal infection. However prior to treating patients conservatively, the clinician should be aware of both the incidence of infection in his or her patient population and hospital setting and the signs and symptoms of early amnionitis, so that serious errors in management can be minimized.

In his study of management of premature rupture of membranes and unfavorable cervix in term pregnancy Patrick Duff[9], stated that the patients managed by intervention had longer labour and a higher incidence of cesarean delivery. He concluded that carefully selected indigent patients at term who have PROM and a cervix unfavorable for induction of labour may be safely managed in a conservative manner without increased risk of maternal-neonatal infection and without prolongation of hospitalization. He also cautioned that the findings of his investigations may not be applicable to term patients with obstetric or medical complications or to patients who had a cervix favorable for induction of labour.

In his article on management of spontaneous rupture of the membranes in the absence of labour in primigravida women at term David Conway[62] demonstrated that a conservative approach to spontaneous rupture of the membranes in primigravida women at 37 weeks gestation or beyond was not associated with an increase in maternal or neonatal infection. He therefore recommended allowing up to 24 hours to elapse after spontaneous rupture of the membranes before considering induction.

In a comparison of induction versus expectant management in PROM with mature amniotic fluid at 32 to 36 weeks, Brian Mercer[63] concluded that induction of labour in patients with PROM and identifiable pulmonary maturity at 32 to 36 weeks was associated both with less frequent neonatal evaluation and antimicrobial therapy

for anticipated infection and with a shorter duration of neonatal antimicrobial therapy. Expectant management was associated with prolonged maternal hospitalization and separation of mothers from their infants without evidence of neonatal benefit. Further expectant management was associated with a high incidence of chorioamnionitis and fetal heart rate abnormalities that occur before labor. He proposed that the women with preterm PROM and confirmed fetal pulmonary maturity near" term should be considered candidates for labor induction. He also suggested that future protocols aimed at prolongation of pregnancy for the reduction of infant morbidity and mortality should actively exclude those patients who had little to gain from expectant management.

Cunningham and MacDonald[64] in Williams Obstetrics outlined management of PROM at Parkland Hospital.

1) One sterile speculum examination was performed to identify fluid coming from the cervix. Demonstration of visible fluid or a positive Nitrazine test was indicative of ruptured membranes.
2) Ultrasound examination was performed to help confirm gestational age, identify the presenting part and assess amniotic fluid volume.
3) If the gestational age was 34 completed weeks or less and if there were no maternal or fetal indications for delivery, the women was observed closely in Labour and Delivery, with continuous fetal heart rate monitoring.
4) If there was no evidence of fetal jeopardy, or if labour did not begin, the woman was transferred to the High Risk Pregnancy Unit for close observation for signs of labour, infection or fetal jeopardy.

5) If the gestation age was greater than 34 weeks and the labour did not begin spontaneously in 12 hours, labour was induced with intravenous Oxytocin. If induction failed, caesarean delivery was performed.
6) Dexamethasone 5 mg was given intramuscularly every 12 hours for 4 doses for enhancement of fetal maturation. This dosage was repeated every 7 days.
7) When labour was diagnosed, Ampicillin 2 gm was given IV every 6 hours prior to delivery for prevention of group B streptococcus infection in the neonate.

ANTIBIOTIC PROPHYLAXIS

Calkins[5] administered penicillin prophylactically to all patients presenting with PROM. He wrote that though penicillin seemed to have reduced the incidence of fetal infection it had not eliminated this complication. Breese[13] also reported the reduction of fetal mortality rate from 37 to 29% with antibiotic administered in labour.

Sperling[65] et al demonstrated lower rate of maternal and neonatal complications when antibiotics were given before delivery rather than postpartum.

There is no antibiotic treatment of choice and different number types, number, and combinations are used. Also the antibiotic should be effective against Group B streptococci, E coli, anaerobic bacteria, which are some of the most frequent causes of severe infectious morbidity.

Kenyon[66] et al in his study, demonstrated monotherapy with third generation cephalosporin or Ampicillin- sulbactum. However Ampicillin- sulbactum have been associated with high incidence of necrotizing enterocolitis in new born. Others use

double antibiotics such as Ampicillin plus cleocin and still others use triple antibiotic therapy with gentamycin or aztreonam plus clindamycin plus Ampicillin.

Burchell[12] reported a reduction in the neonatal death rate by one-third when the mothers were given prophylactic antibiotics during labour. There was no evidence in the literature to demonstrate that antibiotics given during the latent period would decrease the incidence of amnionitis.

Johnstone[67] and associates reported that in the patients with PROM treated with antibiotics the incidence of chorioamnionitis and endometritis-myometritis significantly decreased. Treated infants remained in utero twice as long and experienced three times the weight gain than that of the placebo group. Infants from the treatment group had significantly higher 1-minute Apgar scores and significantly decreased frequency of intraventricular hemorrhage and clinically suspected sepsis. There was a trend towards less RDS. Furthermore, the treatment group infants had a decreased chance of prolonged hospitalization.

Kodkany and Telang[19] reported that antibiotics according to culture and sensitivity combined with steroids may help to reduce neonatal morbidity. Anjana Devi and Reddi Rani[21] in reported a lowered incidence of chorioamnionitis due to routine use of prophylactic antibiotics in patients with PROM. She however also added that despite the modern antibiotics postpartum infection remains high in patients with chorioamnionitis.

ROLE OF CORTICOSTEROIDS IN PATIENTS WITH PROM

Young[68] and Colleagues in their prospective study concluded that intravenous dexamethasone therapy reduced the incidence and severity of RDS among infants delivered at 28 to 33 weeks gestations. Prior to 28 weeks, there was no significant benefit from dexamethasone. This was likely due to immaturity of fetal lung alveoli at that gestational age. They also concluded that beyond 34 weeks gestation, mortality from RDS is uncommon and routine treatment with dexamethasone seemed to be unwarranted.

Morales[69] and colleagues in their study demonstrated that the ante partum administration of corticosteroids in pregnancies with PROM resulted in a significant decrease in the incidence of respiratory distress syndrome without an increased risk of maternal or neonatal infection. Further, the use of corticosteroids also resulted in a substantial reduction of intraventricular hemorrhage, neonatal hospital stay and cost. However Iams[28] and colleagues showed no significant decrease in neonatal respiratory distress syndrome or mortality when steroid therapy was used, compared to conventional therapy. Also steroid-treated mothers had an increased incidence of postpartum febrile morbidity.

Lewis[70] and colleagues in a randomized trial of steroids after treatment with antibiotics indicated that if patients are treated with a broad-spectrum antibiotic and then given steroids the fetus benefited without an increased risk of infectious morbidity.

COMPLICATIONS OF PROM

1) PREMATURITY:

Prematurity has been one of the major complications of PROM. The incidence of premature infants associated with PROM reported in the literature varies from 9 to 40% with the majority approximately 20% [Calkins[5], Gunn[6,7] et al reported the incidence of prematurity associated with PROM as 17%, while overall incidence of premature infants for all deliveries was 7%. Some of the other investigators and their incidence of premature birth weight infants are Daljit Singh[71] and colleagues 32.9 %.Anjana Devi and Reddi Rani[21] 21. 2%.

2) PROLAPSED CORD :

The incidence of prolapse of the cord in all deliveries reported in the literature varied from 0.3 to 0.6%. (Gunn[6] et al). The incidence was only slightly higher in patients with PROM and ranged between 0.3 to 1.7%, Ballard[72]. Gunn[7] et al) reported an incidence of 0.7% of cord prolapse associated with PROM. In all cases prolapse occurred during labour and 60% of the infants died due to this complication.

3) BREECH PRESENTATION :

The incidence of Breech presentation at term is 3.5% (Cunnigham and MacDonald[21],). However in patients with PROM incidence of breech delivery increases slightly with a. range of 3.3 to 8.9% (Gunn[6,7] et al. Breese[11] found an incidence of 27.6% “in premature infants with PROM. When they were delivered vaginally, the perinatal mortality was 51%. By contrast in his series Gunn[7] found that there was 0.26% perinatal mortality in premature PROM when the vertex presented at

birth. Gunn [7] et al reported an incidence of 6.3% for breech presentation with PROM in his series, and the overall perinatal mortality rate was 24.6% of that group ten were immature and all died. The incidence of breech presentation in the infants weighing 1000 to 2499 grams was 16.8%, and 36.2% of these died. The uncorrected perinatal mortality in the 1000 to 2499 gram infants who were delivered by vertex was 11.2%. Perinatal mortality rate for breech deliveries with mature birth weight infants was only 3.2%.

4) AMNIONITIS :

Acute chorioamnionitis occurs frequently in women with PROM. The diagnosis of chorioamnionitis is clinical. It requires the presence of fever (>100F or 37.8 c) and atleast 2 of the following: maternal tachycardia (>100/ min), fetal tachycardia (> 160 bpm), uterine tenderness, foul odour of the amniotic fluid or maternal Leucocytosis (> 15,000) or C reactive protein > 2.7 mg/dl.

The true incidence of amnionitis developing in all patients with premature rupture of the fetal membranes is difficult to obtain from the literature. Russell[73] reported an incidence of 5% amongst the patients with ruptured membranes who were hospitalized and not in labour. In a separate study from the same institute Clark and Anderson[74] found close to 11% incidence. Gunn[6] et al observed incidence of amnionitis as 9.1% and further noted that the risk of developing amnionitis increased proportionately with the increase in the length of the latent period, irrespective of the weight of the infant or the length of gestation, rising to an overall 26.4% for latent period longer than 24 hours. Some of the other investigators and the incidence of amnionitis reported by them are Miller[75] and colleagues 38%. Schreiber and

Benedetti[76]27%,Varner and Galask[61] 5.2%, Moretti and Sibai[58] 39.3%, Kodkany and Telang[19] 5%, Morales and Talley[77] 24%, Anjana Devi and Reddi Rani[21] 5.76%.

Varner and Galask[61] in their article on conservative management of PROM observed that the rate of infection for patients with PROM of less than 24 hours was 0%, for patients with PROM of more than 24 hours but less than 48 hours was 6.6% for those with PROM of more than 48 hours and less than 72 hours was 4.2%, while patients with PROM more than72 hours had a 7.3% infection rate. Sanyal and Mukherjee[78] also agreed that the severity of chorioamnionitis was directly proportional to duration of rupture of the membranes and delivery interval. However, Moretti and Sibai[79] reported that the majority of the patients who had amnionitis did so within the first 3 days of rupture. They also concluded that there was no significant difference in the incidence of amnionitis between patients who did or did not receive steroids. Miller[75] and colleagues found that the most common organisms isolated from the cultures were group B beta-hemolytic streptococci, E-Coli and Klebsiella pneumonia, Ureaplasma urealyticum, lactobacillus). However, no correlation could be found between colonization of the amniotic fluid and the number of pelvic examinations, the duration of labour or the duration of the rupture of the membranes. Varner and Gallask[61] found -Coli, group B beta-hemolytic streptococci and peptostreptococci from the endocervical cultures. According to Shukla and Mishra[78] the most common bacteria isolated were E-Coli and Klebsiella.

Amnionitis appears to influence the perinatal mortality significantly in patients with PROM. Russell and Anderson[73] found that 32% of the perinatal mortality in patients with PROM was associated with amnionitis- In Gunn's[6] series the perinatal mortality rate was 16% when amnionitis developed. The risk of perinatal death was significantly increased when amnionitis developed after the latent period exceeded 24

hours (20.7 %). When amnionitis developed after the latent period of less than 24 hours, the perinatal mortality rate was only 2.1%.

5) ABRUPTIO PLACENTA :

Nelson[80] et al reported an increased risk (4%) for the development of abruptio placentae during the course of expectant management of patients with prolonged preterm PROM. Vlntzileos[81] and colleagues found that the expectant management was associated with the development of abruptio placentae in 19 of the 298 patients (6.3%). They concluded that the 'disproportion[1] created between the reduced size of the intrauterine surface area and the unchanged area of the chorionic placenta's attachment surface may lead to disruption of the placental attachment site in the decidua spongiosa layer. The higher frequency (52.6%) of severe oligohydramnios after PROM who developed abruptio placentae is compatible with the theory of "disproportion". They concluded that the patients with severe oligohydramnios are at high risk for developing abruptio placentae during the course of expectant management. Moretti and Sibai[79] also reported an increased incidence (6.8%) of abruptio placentae in the patients managed conservatively after PROM.

MATERNAL MORTALITY:

In 1962, Russell and Anderson[73] concluded that 'amnionitis' was the common factor for the 5 maternal death (3.8%) associated with premature rupture of the membranes. Webb[82] reviewed 54 maternal deaths associated with PROM. He calculated the risk of maternal mortality in PROM to be one in 5451. Sepsis was present in 46 of the patients and was listed as the primary cause of death in 38. Although most serious infections were related to patients being neglected, he noted

that in 15 cases less than 24 hours had elapsed between amniorrhexis and onset of fever or delivery. The other causes of maternal death in his series were amniotic fluid embolism, hemorrhage, ruptured uterus and cardiac arrest. There was no maternal death reported by Gunn[6,7] in his series. Sacks and Baker[60] reported one maternal death associated with PROM, but cause of death was Placenta accreta. Moretti and Sibai[79] reported one maternal death from sepsis associated with PROM. Sanyal and Mukherjee[78] reported 2 cases of maternal death in the whole series during puerperium with clinical evidence of bacteriaemic shock. Thus sepsis was the major cause of maternal death in patients with PROM.

MATERNAL MORBIDITY:

Only a few authors have reported the incidence of maternal morbidity associated with PROM. Margaret Schulze reported that though there was no maternal death associated with PROM, morbidity was increased by approximately 20 %, when the labour was delayed by more than 24 hours. However, Calkins[5] reported no demonstrable maternal morbidity in his series. Burchell[16] found an increasing incidence with increasing length of latent period, 1.7% upto 24 hours increasing to 8.6% over 48 hours. Using the data of Sacks and Baker[60] the incidence of maternal morbidity was calculated to be 1.3% when the latent period was less than 24 hours and 4,8% when it was longer than 24 hours. Breese[11] reported an increased incidence in cases associated with prematurity (7.8%).

During conservative management of preterm pregnancies (between 28 and 36 weeks) associated with PROM, Varner and Galask[61] reported 5.2% incidence of amnionitis while 7% of the patients developed endometritis post partially. He further noted that there was no correlation between the risks of endometritis and the duration

of PROM. Beydoun[5] and Yasin in their article found that nine (13.0%) of the 69 patients developed postpartum endomyometritis, with five of them following caesarean section. Six patients (8.7%) had a retained placenta. Other maternal complications included two abruptio placentae and one each of postpartum hemorrhage, post-cesarean section atelectasis, wound infection and cervical laceration. One patient with a previous cesarean section had a placenta accreta necessitating an abdominal hysterectomy.

Sanyal and Mukherjee[78] noted puerperal pyrexia in 17.5% of cases. They, however, claimed that the incidence of puerperal sepsis and PPH was not influenced by PROM. Kodkany and Telang[19] found that maternal morbidity was present in 21 cases (21%), 19 cases (90.47%) of which had PROM for >24 hours. Puerperal pyrexia was present in 16 cases (16%) as compared to 3% in control.

Anjana Devi and Reddi Rani[21] noted puerperal fever in 21 (20.19%) of cases with PROM. Wound infection was responsible for 8 cases, puerperal sepsis for 9 and urinary tract infection for 4 cases.

Lebherz[83] and associates used prophylactic antibiotics in a double blind prospective study and found significant reduction in postpartum morbidity. They found 7.3% puerperal morbidity rate in patients with PROM who did not receive prophylactic antibiotics compared to 3.5% morbidity in patients who received demethylchlortetracycline. This was due to reduction in the incidence of endometritis, the primary cause for puerperal morbidity. The incidence of parametritis, pyelonephritis, was also reduced. Similar observation of reduction in maternal morbidity rates by use of prophylactic antibiotics have been made by Russell and Anderson[73] , Johnston[67] and colleagues.

PERINATAL MORTALITY

The overall incidence of perinatal mortality reported in the literature ranges form 2.6 to 11 % Calkins[5], Breese[11] , Burchell[12], Varner and Galask[61], Sanyal and Mukherjee[78], Kodkany and Telang[19], Anjan Devi and Reddi Rani[21]]. When calculated according to birth weight the incidence of fetal and neonatal death was found to be as high as 31% for the premature infants [Breese[11] , Gunn[6,7] et al].

Gunn[6] et al reported perinatal mortality rate of 4.1%. All infants of immature birth weight (500 to 999 gms) died. The perinatal mortality rate for premature infants (1000 to 2500 gms) was 15.1% while in infants weighing more than 2500 gms it was 0.8%. The overall incidence of perinatal mortality during the same period was 2.7%. Thus 16.1% of perinatal deaths were associated with PROM. Breese[11] and Gunn[6,7] et al have shown that perinatal mortality rates rise markedly with prolongation of latent period and rupture delivery interval. In the Gunn's series 10,2% of the infants between 1000 and 2500 gm died when the latent period was less than 24 hours but when the latent period exceeded 24 hours perinatal mortality rate rose to 22.5%. When the birth weight was 2500gms and over, the perinatal mortality rate was -0,5 and" 2% for latent period under and over 24 hours. The rise in perinatal mortality with prolonged latent period was chiefly because of increased incidence of infection. The aggressive management of Russell and Anderson[73] using caesarean section to ensure termination of all pregnancies within 24 hours after rupture of the membranes raised the rate of caesarean section from 4.5 to 12 %, but reduced fetal deaths by two third. According to Eastman and Hellman[84] the uncorrected survival rate for newborns weighing approximately 1125 gms (a weight corresponding to 29 weeks of gestation) is about 50%, and for a weight of 1500 gm (a weight corresponding to 34-35 weeks) about 70%.

Sacks and Baker[60] while analyzing the various causes of death of infants state that immaturity was the major cause of perinatal mortality responsible for death of 17 out of 23 infants. The other causes were Hyaline membrane disease, subarachnoid hemorrhage, meningitis and congenital anomalies.

Moretti and Sibai[79] in their series reported 17 still births and 67 neonatal deaths, accounting for a total perinatal mortality of 67.7% They also found that in patients with PROM <23 weeks, the perinatal survival rate was 13.3%, while it was 50% in patients with PROM at 24 to 26 weeks.

Evaluating the effect of prophylactic antibiotics administered to the mother during labour, Breese[11] claimed that the fetal mortality rate was reduced from 37 to 29%. However, Russell and Anderson[73] , Lebherz[83] and associates, Johnston[40] and colleagues have demonstrated lack of effectiveness of prophylactic antibiotic therapy following PROM in decreasing perinatal mortality rates.

Hadi[85] and colleagues in a study of role of amniotic fluid volume in perinatal outcome of patients with PROM between 20 and 25 weeks found that the survival rate of infants before 25 weeks was 6.7%, whereas between 26 and 34 weeks, it was 89.4%. He concluded that the following information can be used while counseling patients with PROM at 20 to 25 weeks :

1. Women at 20-25 weeks gestation with PROM with inadequate amniotic fluid volume have a dismal chance for neonatal survival. If amniotic fluid reaccumulates, the chance for survival significantly improves. In pregnancy at < 23 weeks where oligohydramnios persists, pregnancy outcome is extremely poor.

2. Pregnancies with adequate amniotic fluid volume that continue beyond 25 weeks are associated with a survival rate of 89%.
3. Pregnancies with inadequate amniotic fluid volume that expend beyond 25 weeks of gestation carry a perinatal death risk of 69% versus 2.1% in those with adequate amniotic fluid volume.
4. The risk of bronchopulmonary dysplasia and orthopedic deformities is very low. However, the risk of stillbirth and neonatal death is substantially higher in pregnancies with inadequate amniotic fluid volume than in those with adequate amniotic fluid volume.

They concluded that inadequate amniotic fluid volume had a strong correlation with poor neonatal survival.

NEONATAL OUTCOME IN PROM

Shubeck[86] et al stated that there was a 4-fold increase of percentage of premature infants over the range of intervals from membrane rupture to birth.

Moretti and Sibai[79] while reviewing perinatal outcome in expectant management of PROM found that most of the complications were related to extreme prematurity. Out of the 68 neonates admitted in the neonatal I.C.U., 70% had respiratory distress, 50% had intraventricular hemorrhage, 29.4% had sepsis, 22% had broncho-pulmonary dysplasia, 13.2% had necrotizing enterocolitis, 11.8% had congenital pneumonia.

Nimrod[87] and colleagues in their article on the effect of very prolonged ruptured membranes on fetal development found pulmonary hypoplasia and skeletal deformities associated with prolonged PROM. They found that the risk of

development of pulmonary hypoplasia was quite low when rupture occurred after 26 weeks. The probable explanation was that significant number of terminal air sacs had already developed after 26 weeks gestation. Hence inhibition of further lung growth after that time left the fetus with a sufficient number of alveoli to support adequate respiratory function.

They also demonstrated that patients with prolonged PROM before 26 weeks gestation and with a latent period of 5 weeks or more had a 47% risk of fetal skeletal deformity. They concluded that optimum management should be directed towards preservation of amniotic fluid. Rotschild[88] and colleagues (1990) diagnosed pulmonary hypoplasia in 16% of the infants. They confirmed the findings of Nimrod[87] and colleagues. They also found a strong association between the presence of pulmonary hypoplasia and the extent of skeletal deformities.

Daljit Singh[71] and colleagues in their article on neonatal infection following PROM found that the commonest neonatal superficial infection was conjunctivitis. Other infections included pyoderma, umbilical sepsis, septicemia, purulent meningitis, pneumonia. They also found that PROM less than 12 hours was not associated with neonatal infection while the incidence was 30.8% with PROM more than 72 hours.

Kodkany and Telang[19] in their study found that neonatal morbidity was present in 39.8% of patients with PROM. Birth asphyxia was the commonest; it was followed by RDS and congenital anomalies. They also stated that neonatal morbidity was common in preterm babies and the incidence increased-as the duration of PROM increased.

Anjana Devi and Reddi Rani[21] in their study noted neonatal morbidity in the following order in patients with PROM: conjunctivitis (22.1%), prematurity (21.2%), respiratory distress (18.3%), septicemia (11.5%), pneumonitis, umbilical sepsis, superficial skin infection (5.8% each), meconium aspiration, convulsions (4.8% each) and meningitis (2.9% cases).

MATERIAL AND MATERIALS

SOURCE OF DATE:

All the patients presenting with spontaneous premature rupture of membranes (PROM) and admitted in BLDEU's Shri B M Patil Medical College, Bijapur between October 2009 to September 2011 were studied. Spontaneous premature rupture of membranes was diagnosed when spontaneous leakage of amniotic fluid occurred prior to the onset of labour anytime from 34 weeks of pregnancy onwards.

SAMPLE SIZE: 120

SELECTION CRITERIA:

1. All patients with PROM admitted to BLDEU's Shri. B.M.Patil Medical College & Research Centre

EXCLUSION CRITERIA:

1. Anomalous babies
2. Elderly Primiparity
3. Hydramnios
4. H/O Antepartum hemorrhage

METHOD OF COLLECTION OF DATA:

A complete history including duration of amenorrhea, duration of leaking, duration of labour pains (when patient presented in labour) and presence of suspected predisposing factors like history of PV examination 1 week before leak, history of leaking in previous pregnancies, history of prior cervical instrumentation (D & C, S &

E, cervical biopsy, encirclage, other surgery on cervix) history suggestive of chronic cervicitis/vaginitis, history of antecedent coitus within 1 week before leak, history of travelling were recorded. Socio- economic status of the patient was assessed by recording the monthly income, size of the family and educational status of the women. Menstrual, obstetric past, family and personal history of the patient were recorded.

All patients were examined, including general examination, systemic examination and speculum examination. Sterile per speculum examination without using any antiseptic was undertaken to reveal presence or absence of amniotic fluid leak through cervix, with or without application of fundal pressure and fluid collected for specific laboratory tests. If speculum examination was negative for fluid leak, then fluid from the posterior vaginal fornix was collected and subjected to laboratory tests. Digital per vaginal examination was not done in those patients in whom conservative line of management was planned, in rest of the patients a sterile digital per vagina examination was also undertaken to evaluate cervical dilatation, effacement, presence or absence of cord prolapse. The fluid obtained was subjected to four laboratory tests.

CRYSTALLIZATION TEST

A drop of fluid was taken on a clean glass slide and allowed to air dry and subsequently examined under low power of microscope. Presence of discrete crystallization pattern or ferning was labeled as positive test.

LITMUS PAPER TEST

Both litmus papers were dipped in the amniotic fluid. If the colour of the paper changed from red to blue, the test was labeled as positive.

EVAPORATION TEST

A drop of fluid was taken on a clean glass slide and heated for 30 seconds. If a white precipitate is left behind, the test was labeled as positive. A brown precipitate indicated a negative test.

Patients were diagnosed to have 'high leak[1] when initial examination on admission revealed frank leakage of amniotic fluid (with laboratory confirmation) but subsequently were found to have intact bag of fore waters at or before delivery. All other cases of PROM not having high leak were termed to have low leak. Latent period was defined as the time interval between onset of leaking and onset of labour. Total duration of leak was defined as the Lime between onset of leaking and delivery.

Gestational age was assessed by the knowledge of the date of last menstrual period, findings of initial prenatal examination and ultrasonographic examination and subsequent application of Dubowitz[22] criteria to the newborn.

Clinical diagnosis of amnionitis was made when patients with PROM developed fever with Leucocytosis with uterine irritability and tenderness with or without foul smelling liquor. C - reactive protein estimation was done in patients suspected to have amnionitis. Placenta was examined histopathological in patients diagnosed to have amnionitis.

Perinatal mortality included all fetal and neonatal deaths from 28th completed weeks of pregnancy and upto first 7 days of neonatal life. Post partum morbidity was defined as a temperature greater than 100.4° F, on two separate occasions greater than

6 hours apart, after the first 24 hours post-partum, as well as any documented systemic complication with or without fever such as endometritis, parametritis, wound sepsis, peritonitis or urinary tract infection (Gunn[6])

All patients with ruptured membranes for more than 6 hours were given antibiotics - Ampicillin/ Amoxicillin/ third generation cephalosporins with or without Gentamycin / Amikacin.

In the management, with gestational age more than 36 weeks, if spontaneous labour did not occur within 6 hours, the labour was induced with Oxytocin by intravenous drip. In some cases when the contractions were not good, the labour was accelerated with Oxytocin. In patients presenting with clinical amnionitis, pregnancy was terminated promptly with Oxytocin induction or caesarean section. Steroids or tocolytic therapy was not used in this group.

OBSERVATION TABLES

Table 1:. Incidence of Premature Rupture of Membranes

Total No. of Cases	No. of Cases with PROM	Incidence
1612	120	**7.444169**

Out of 1612 patients during the study period between October 2009 to August 2011 , 120 cases had Premature rupture of membranes giving the incidence of 7.44 % in our hospital.

Table 2: Age wise Distribution of Cases

Maternal age in years	No. of cases	Percentage
16 to 20	33	27.50
21 to 25	57	47.50
26 to 30	29	24.16
31 to 35	1	0/84
36 to 40	Nil	0.00
Total:	**120**	**100.00**

Table 2. Reveals that 21 to 25 years was the common age group as 47,50 % of the patients were between these ages.

Graph 1: Age wise Distribution of Cases

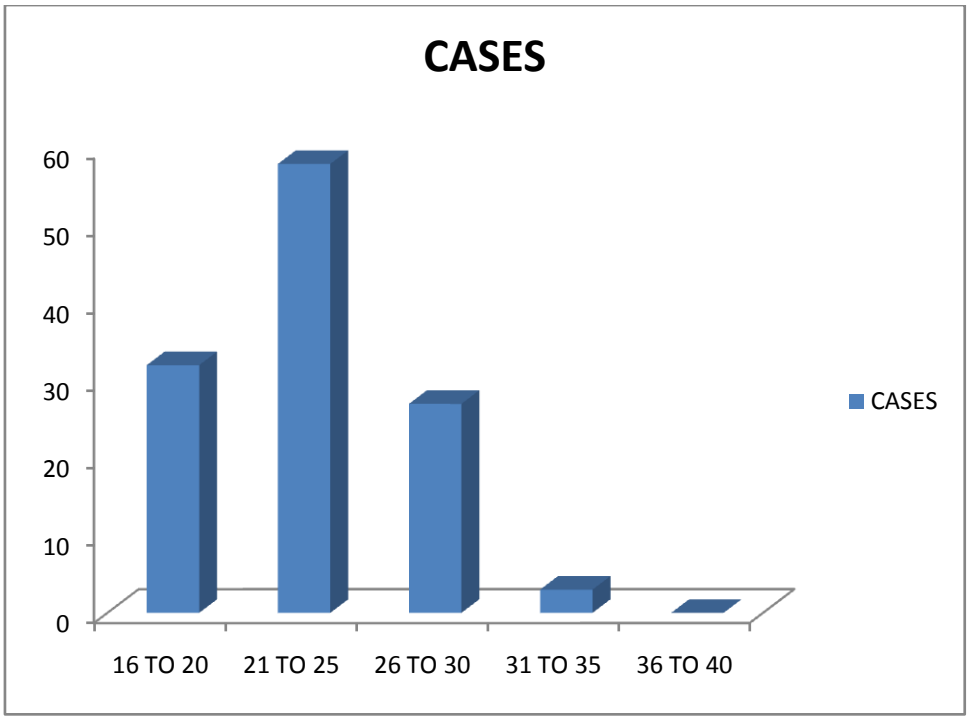


Table 3: Parity wise Distribution of Cases.

Gravida	No. of Cases	Percentage
PRIMI	60	50.00
2nd to 4th	58	48.33
5 and more	2	1.67
Total:	**120**	**100.00**

Table 3 reveals that 50 % of the patients were Primigravida and 50 % were Multigravidae.

Graph 2: Parity wise Distribution of Cases.

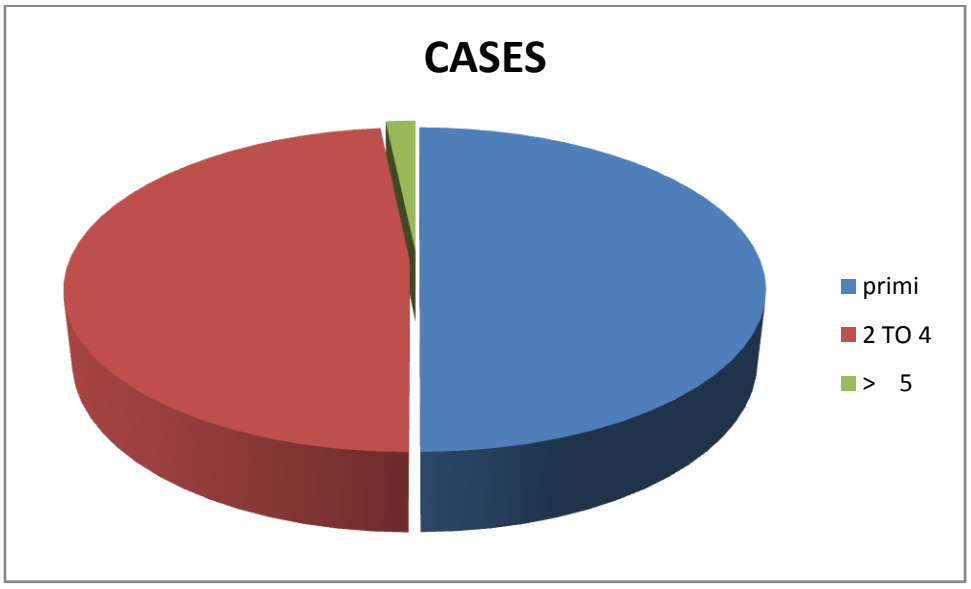


Table 4: Incidence of Singleton and Multiple pregnancy

Pregnancy	No. of cases with PROM	Incidence in PROM
Singleton	118	98.33%
Twins	2	1.67%
Total	**120**	**100.00%**

Table 4 shows that 98.33 % of the patients with PROM had singleton pregnancy while the incidence of twins in PROM was 1.67 %. .

Table 5: Analysis of various presentations

Singleton Pregnancy

Presentation	No. of cases with PROM	Incidence in PROM (Percentage)	Overall Incidence of various presentations (Percentage)
VERTEX	105	88.98	96.03
BREECH	12	10.18	3.33
BROW	0	0.00	0.00
SHOULDER	1	0.84	0,64
FACE	0	0.00	0.00
TOTAL	**118**	**100.00**	**100.00**

There were 2 cases of Twin pregnancy. The presentation was 1st Breech and 2nd Vertex in one case and 1st Vertex and 2nd Breech in other case.

Table 5 shows that presentation other than vertex had a higher incidence in PROM.

Table 6 : Presence of suspected predisposing factors

Factors	No. of cases	Percentage
Past cervical operations	16	13.33
Leaking in previous pregnancy	16	13.33
Chronic cervicitis / vaginitis	8	6.67
PA/ examination	9	7.50
Antecedent coitus within 1 week	24	20.00
Travelling	15	12.50
Presence of more than one factor	18	15.00
None of the factors present	54	45.00

Table 6 shows that in 55 % (66 out of 120) of the cases one or more predisposing factor were present.

13.33 % (16 out of 120) had history of past cervical' operations, 13.33 % (16 out of 120) had leaking in previous pregnancy. 6.67 % (8 out of 120) had history suggestive of chronic cervicitis / vaginitis, 7.50 % (9 out of 120) had P/V examination,20.00 % (24 out of 120) had antecedent coitus within 1 week, 12.50 % (15 out of 120) had history of travelling.

15.00 % (18 out of 120) had presence of more than one factor while 45.00 % (54 out of 120) had none of the factors present.

Table 7: Duration of gestation

Duration of pregnancy in completed weeks	No. of cases	Percentage	Overall incidence of Prematurity during study period
PREMATURE			
34-36	17	14.16%	14.16667
Total	**17**		
MATURE			
37weeks onwards	103	85.8333%	
GRAND TOTAL	**120**	**100.00**	

Table 7 reveals that 14.16 % of the cases were preterm while 85.83 % had term pregnancy.

The overall incidence of Prematurity during the study period was 14.16%.

Graph 3: Duration of gestation

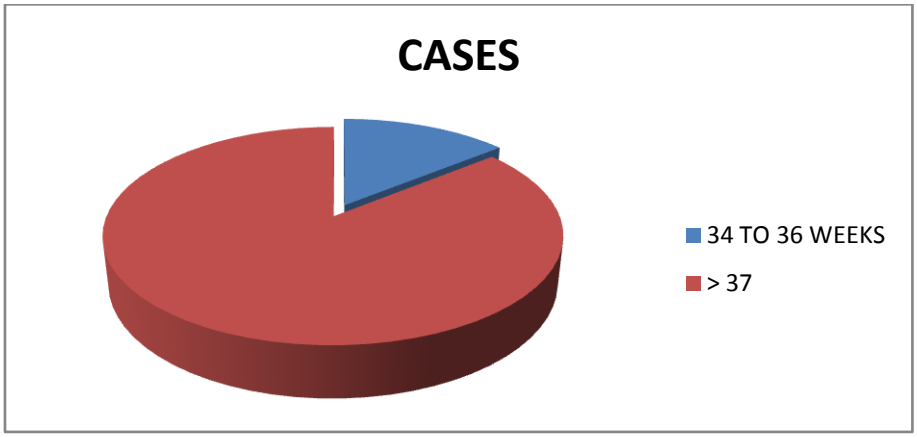


Table 8: Associated complications in patients with PROM

Complications	No. of cases	Percentage	Overall incidence during study period (Percentage)
C.P.D.	7	5.83	1.55
Previous L.S.C.S.	15	12.50	6.81
Post date	15	12.50	7.53
Cervical incompetence	3	2.50	2.00
Rh incompatibility	5	4.16	5.61
Pre-Eclampsia	9	7.50	4.15
Diabetes	2	1.66	2.72
Malpresentation	13	10.83	4.28

Table 9: Diagnosis of PROM

Test	No. of cases with +ve test	Percentage
Direct visualization	116	96.66
Crystallization	116	96.66
Litmus paper	116	96.66
Evaporation	116	96.66

Table 9. shows that Direct visualization, Crystallization, Litmus paper and Evaporation test were all positive in 96.66 % of cases.

Graph 4: Diagnosis of PROM

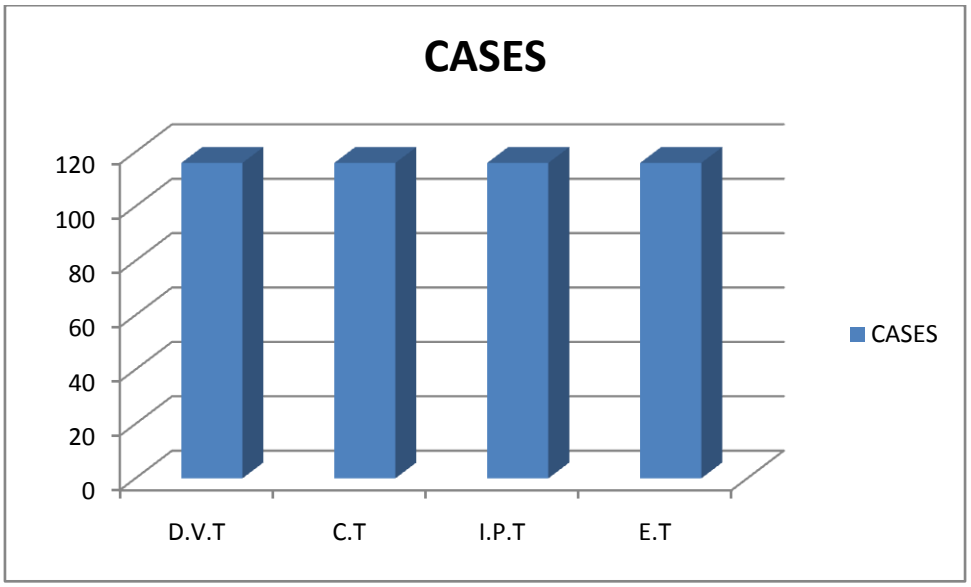


Table 10: Distribution of cases according to the type of leak

Type of Leak	No. of Cases	Percentage
High leak	26	21.67
Low leak	94	78.33
Total	**120**	**100.00**

The incidence of High leak was 21.67 %, whereas the incidence of Low leak was 78.33 %.

Graph 5: Distribution of cases according to the type of leak

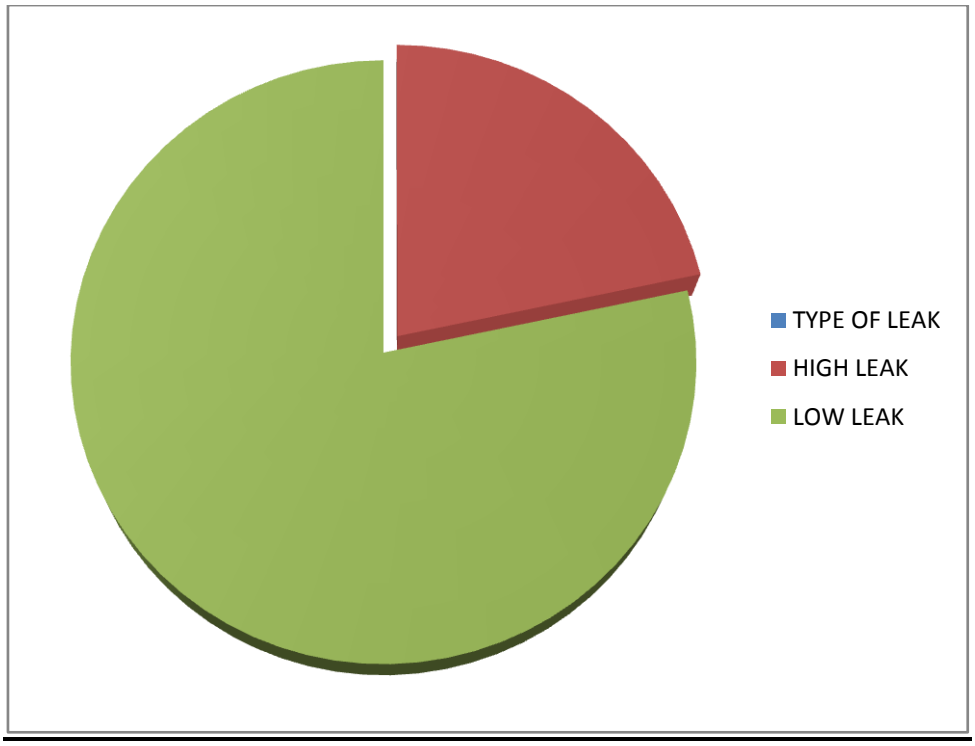


Table 11: Management of PROM

Management	No. of cases	Percentage
Conservative	4	3.33
Active termination of Pregnancy	31	25.83
Spontaneous onset of labour	85	70.84
Total	**120**	**100.00**

It is obvious from Table: 11 that 70.84 % of the patients developed spontaneous onset of labour, 3.33 % of the cases were treated conservatively and pregnancy was terminated actively in 25.83 % of cases,

Table 12: Mode of delivery

Type	Spontaneous Vaginal delivery		Others			Total
	With Oxytocin	Without Oxytocin	Forceps delivery	Ventouse delivery	LSCS	
Spontaneous onset	33	18	3	1	30	85
Active Termination	18	-	1	1	11	31
Conservative	2	2	-	-	-	4
Total	**53**	**20**	**4**	**2**	**41**	**120**
Incidence in PROM	60.34 %		3.34 %	1 .66 %	34.16%	100%

Table: 12 Shows that the incidence of LSCS in PROM was 34.16%, the incidence of Forceps delivery was 3.34%, Ventouse delivery was 1.66%, while 60.84% of the patient delivered vaginally spontaneously.

Table 13.. Indication for L.S.C.S.

Indications	No. of cases	Percentage
Fetal distress	5	58.54
Failure to progress	15	
Dysfunctional labour	3	
Twin pregnancy with PROM with P.I.H.	1	
C.P.D.	3	41.46
Primigravida with Breech Presentation.	5	
Transverse lie	1	
Previous L.S.C.S. for recurrent cause	7	
Cervical fibroid	1	
Total :	**41**	**100.00**

Table: 13. Shows indications for Caesarean section. Some of them (17/41) would have had Caesarean section even if they never had PROM.

Table 14: Distribution of cases according to latent period

Type of Leak	Latent Period	No. of Cases	Percentage
High	24 hours or less	21	80.77
	More than 24 hours	5	19.23
	Total :	**26**	**100.00**
Low	24 hours or less	80	85.11
	More than 24 hours	14	14.89
	Total :	**94**	**100.00**

Table 14 shows that 15.83 % of total cases (both high and leak) had latent period more than 24 hours.

Table 15: Interval between Onset of leak and delivery

(Total duration of leak)

Type of Leak	Total duration of leak	No of Cases	Percentage
High	24 hours or less	17	65.38
	More than 24 hours	9	34.62
	Total :	**26**	**100.00**
Low	24 hours or less	71	75.54
	More than 24 hours	23	24.46
	Total :	**94**	**100.00**

Table : 15 shows that in total 26.66 % of patients (High and Low leak considered together) had leaking for more than 24 hours 34.62 % of the patients from high leak group and 24.46 % of patients from low leak group had total duration of leak more than 24 hours.

Table 16: Relation of amnionitis to latent period

Latent Period	High Leak			Low Leak			Overall incidence in PROM
	No. of cases with amnionitis	Total cases	Percent	No. of cases with amnionitis	Total cases	Percent	
24 hours or less	0	21	0.0	2	80	2.5	6.66%
More than 24 hours	1	5	20.00	5	14	35.7	
Total	**1**	**26**	**3.84**	**7**	**94**	**7.44**	

Table: 16 shows overall incidence of amnionitis as 6.66 %. However, in patients with high leak it is zero percentage, in patients with latent period less than 24 hours and 20.00 % in patients with latent period more than 24 hours. In those with low leak the incidence of amnionitis was 2.5 % in patients with latent period less than 24 hours and 35.7 % in those with latent period more than 24 hours.

When high and low leak were considered together 2 out of 101 cases with latent period of 24 hours or less developed amnionitis for an incidence of 1.98 % and 6 out of 19 cases with latent period of more than 24 hours, i.e. 31.58 % developed amnionitis.

Table 17: Relation of post-partum morbidity to latent period

Latent Period	High Leak			Low Leak			Overall incidence in PROM
	Post partum infection	Total cases	Inci-dence	Post partum infection	Total cases	Inci-dence	
24 hours or less	0	21	0.00%	10	80	12.5%	15.83%
More than 24 hours	1	5	20.00%	8	14	57,14%	
Total	**1**	**26**	**3.84%**	**18**	**94**	**19,14%**	

Table: 17 shows 15.83 % as overall incidence of post-partum morbidity in PROM. No patient in high leak group with latent period less than 24 hours had post-partum morbidity, whereas the incidence of post-partum morbidity was 20 % when latent period was more than 24 hours.

In the low leaks group, the incidence of post-partum morbidity was 12.5 %, when the latent period was 24 hours or less and 57.14 % when the latent period was more than 24 hours.

When high leak and low leak were considered together, the incidence of post-partum morbidity was 9.90 %, when the latent period was less than 24 hours and 47.36 % when it was more than 24 hours.

Table 18: Relation of amnionitis to Total duration of leak

Total duration of leak	High leak			Low leak			Overall inci-dence in PROM
	No. of cases with amnionitis	Total cases	Percentage	No. of cases with amnionitis	Total cases	Percentage	
24 hours or less	0	17	0.0	2	71	2.81	6.66 %
More than 24 hours	1	9	11.11	5	23	21.73	
Total	**1**	**26**	**3.84**	**7**	**94**	**7.44**	

Table: 18 shows overall incidence of amnionitis as 6.66 %. However, in patients with high leak, it is zero %, in patients with total duration of leak less than 24 hours and 11.11 % in patients with total duration of leak more than 24 hours.

In those with low leak, the incidence of amnionitis was 2.81 % in patients with total duration of leak less than 24 hours and 21.73 % in those with total duration of leak more than 24 hours.

When high leak and low leak were considered together 2 out of 88 cases with total duration of leak of 24 hours or less developed amnionitis for an incidence of 2,27 % and 6 out of 32 cases with total duration of leak of more than 24 hours, i.e. 18.75 % developed amnionitis.

Table 19: Relation of Post-partum morbidity to Total duration of Leak

Total Duration of leak	High leak			Low leak			Overall incidence in PROM
	Post-partum infection	Total cases	Percen-tage	Post-partum infection	Total cases	Perce-ntage	
24hours or less	0	17	0.0	10	71	14.08	15.83%
More than 24 hours	1	9	11.11	8	23	34.78	
Total	**1**	**26**	**3.84**	**18**	**94**	**19.14**	

Table: 19 shows 15.83 % as overall incidence of post-partum morbidity in PROM. No patient in high leak group with total duration of leak less than 24 hours had post-partum morbidity, whereas the incidence of post-partum morbidity was 11.11 % when the total duration of leak was more than 24 hours.

In the low leak group, the incidence of post-partum morbidity was 14.08 %, when the total duration of leak was 24 hours or less and 34.78 % when the total duration of leak was more than 24 hours.

When high leak and low leak were considered together, 10 out of 88 cases with total duration of leak less than 24 hours developed post-partum morbidity for an incidence of 11.36 % and 9 out of 32 cases (i.e. 28.12 %) with total duration of leak more than 24 hours developed post-partum morbidity.

Table 20:

Relation of perinatal mortality to the total duration of leak

	High leak		Low leak		Total
	Total duration of leak		Total duration of leak		
	24 hours or less	More than 24 hours	24 hours or less	More than 24 hours	
No. of Perinatal deaths	1	0	3	3	7
Total birth	17	9	73*	23	122
Incidence	5.88%	0.00	4.10%	13.04%	5.73 %
Overall perinatal mortality in the total no. of deliveries - 6.4 %					

* Two set of twins

Table: 20. Shows that, in the high leak group, when the total duration of leak was less than 24 hours, the incidence of perinatal mortality was 5.88 %. and when it was more than 24 hours, it was zero %.

In the low leak group, when the total duration of leak was less than 24 hours, the incidence of perinatal mortality was 4.1 % and when it was more than 24 hours it was 13.04 %.

When high leak and low leak were considered together, the perinatal mortality was 4 in 90 births (i.e. 4.44 %) when the total duration of leak was less than 24 hours, and 3 in 32 births (i.e. 9.37 %) when the total duration of leak was more than 24 hours.

Table 21: Relation of perinatal mortality to latent period

	High leak		Low leak		Total
	Latent Period		Latent Periods		
	24 hours or less	More than 24 hours	24 hours or less	More than 24 hours	
No. of Perinatal death	1	0	3	3	7
Total birth	21	5	82*	14	122
Incidence	4.76 %	0	3.65%	21.42%	5.73 %
Overall perinatal mortality in the total no. of deliveries - 6.4 %					

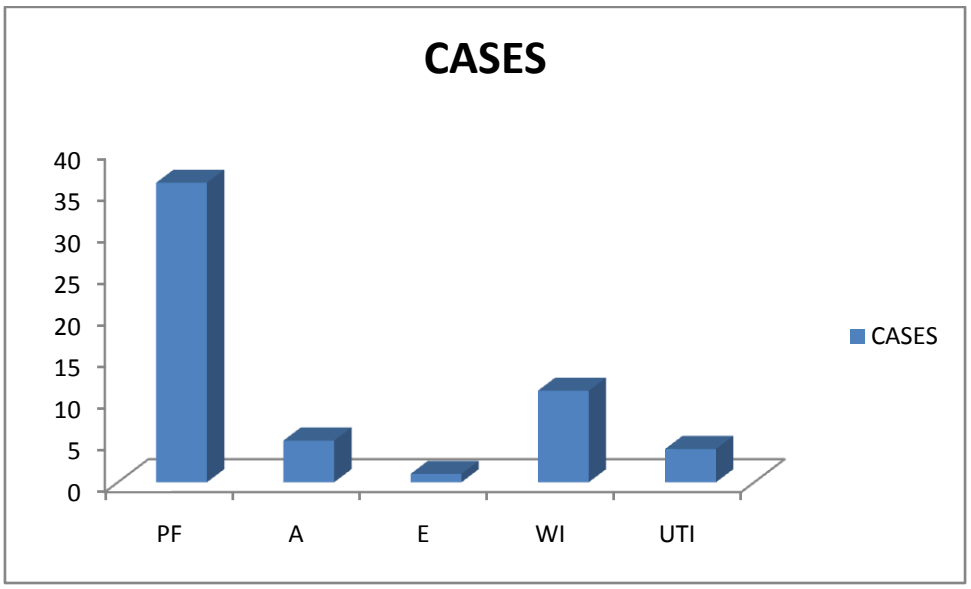
*Two set of twins.

Table 21 gives 5.73 % as the incidence of perinatal mortality in PROM. In the high leak group, when the latent period was less than 24 hours, the perinatal mortality was 4.76 %, while there was no perinatal mortality when the latent period was more than 24 hours.

In the low leak group the incidence of perinatal mortality was 3.65 % when the latent period was less than 24 hours and 21.42 % when the latent period was more than 24 hours.

When high leak and low leak were considered together, the perinatal mortality was 4 in 103 (3.88 %) when the latent period was 24 hours or less and 3 in 19 (15.78 %) when the latent period was more than 24 hours.

Graph 6: Analyses of Causes of Maternal Morbidity:



Graph 6 indicates that maternal morbidity in the form of puerperal fever was 63.15%, amnionitis 6.66 %, endometritis 1.75%, wound infection 19.29% and Urinary tract infection was 7.01%

Table 22: Analysis of causes of maternal morbidity

Maternal Complications	Cases	Percentage %
Puerperal Fever	36	63.15789
Amnionitis	5	8.77193
Endometritis	1	1.754386
Wound Infection	11	19.29825
Urinary Tract Infection	4	7.017544

Table: 23. Analysis of Causes of Perinatal mortality

Cause	No. of Deaths		Total	Percentage
	Premature	Mature		
Infections	2	1	3	42.86%
Congenital anomalies	0	0	0	0.00
R.D.S.	4	0	4	57.14
Cord Prolapse	0	0	0	0.00
Total	6	1	7	100.00
Percentage	85.72	14.28	100.00	

Table: 23 indicate that 42.86 % of the perinatal deaths were due to infection and 57.14 % of the deaths were due to R.D.S. 85.72 % of the perinatal deaths were amongst premature infants while 14.28 % were in mature infants.

DISCUSSION

Out of 1612 patients during the study period between Oct 2009 to Sept 2011, 120 cases had premature rupture of membranes giving the incidence of 7.44 % in our hospital (Table: I).

This is quite comparable with the incidence reported by various workers, as shown in the following table.

Authors	**Incidence of PROM (Percentage)**
Breese[13]	6.4
Anjana Devi et al [21]	7.2
Lanier[13]	6,2
Gunn[6,7]etval	10.7
Lenihan[18]	11
Patrick Duff[9]	6.1
Vintzileo's[81]	10
Present study	7.44

The maternal age associated with spontaneous PROM in this study ranged from 16 to 40 years (Table: I) and 21 to 25 years was the commonest age group (47.5 %). These findings correlate with those of Anjana Devi and Reddi Rani[21] and Varner and Galask [61] who reported the age range as 16 to 41 years, and the mean age was 25 years.

As for the parity, 60 out of 120 patients (50 %) were prirnigravidae and 60 (50%) were multigravidae. Of the multigravidae, only 2 patients had more than 4 pregnancies. (Table: 3)

The incidence of twin pregnancy in PROM in present study was 1.67 %, and the overall incidence was 1.62 % (Table: 4).

Of the 118 cases of Singleton pregnancy (98.33 %) associated with PROM (Table), 88.98% presented with vertex and 11.02% had malpresentation. The overall incidence of malpresentation when all deliveries were considered, during the study period was 3.97 %. Among malpresentation in PROM the incidence of breech was 10.18 %, but the overall incidence of breech was 3.33 %. This shows significantly higher incidence of malpresentation in association with PROM. The increased incidence of breech presentation in PROM was also noted by some of the other workers like Breese[11] (27.6 % in premature infants), Gunn [6] et al (3.3 to8.9%) and Kodkany [19] 31 %.

Six predisposing factors,viz history of:

I. Antecedent coitus within a period of 1 week before spontaneous PROM,
II. Past cervical operations (dilatation and curettage, Punch biopsy, Cervical encirclage),
III. Leaking in previous pregnancy,
IV. Chronic Cervicitis / Vaginitis,
V. Antenatal Pelvic examination
VI. Travelling, were studied for their possible etiological role in spontaneous PROM (Table: 6).

Out of the 120 patients, none of the factor was found positive in 45 % of patients and in 55 %, one or more factors were present. Out of these six factors studied, history of antecedent coitus within a period of 1 week prior to PROM was present in 20 % of the patients, past cervical operations were present in 13.33 %, leaking in previous pregnancy was present in 13.33 %. These findings are in agreement with those of Naeye[8]. History of antenatal pelvic examination was present in 7.50 % of patients. This is in agreement with the findings of Lenihan [18] and Jiwane [15]. 6.67 % of the patients had history of Chronic cervicitis Vaginitis while 12.50 % of the patients had history of travelling.

The incidence of prematurity in PROM in the present study was 14.66%, while the overall incidence of prematurity (when all deliveries were considered).This shows higher incidence of prematurity in association with PROM and is comparable with those of other workers like Calkins[5] (22 % in primi and 32 % in rnultiparae), Burchell[12] (23%), Anjana Devi[21] (21%). This also confirms the earlier findings made by many authors that PROM is one of the important cause of premature labour.

When associated complications in patients with PROM in this study were analyzed (Table : 8) the incidence of C.P.D. (5.83 %), Previous L.S.C.S.(12.50 %), Prematurity (19.16%), Postdate (12.50 %), Cervical incompetence (2.50 %), Rh incompatibility (4.16 %), Preeclampsia (7.50 %) and representation (10.83 %) are significantly higher than overall incidence in total number of deliveries during the study period.

Analyzing the methods of diagnosis (Table; 9), a sterile speculum examination for direct visualization of fluid leak from the cervix was positive in 96.66 % of the patients with PROM. Out of the four laboratory tests performed for diagnosis, Crystallization test was positive in 96.66 % of cases. These tests were negative in only

four cases in which the patients were referred after 24 hours with dry labour and intranatal infection. These patients had several hours of leaking prior to the onset of labour. The results of some of the other workers with this test are Marcel Ferron et al[43], 98.5% accuracy, Friedman and McElin[14] , 96.4 % accuracy,Anjaneyulu and Likhite [44], 86.6 % accuracy, Gupta and Gupta [41], 92.5 % accuracy. Godhara and Pendse[30] reported this test to be positive in 100 % cases when there was frank PROM, while in doubtful cases the test was positive 87 % of the times. The findings of various workers are almost identical with those obtained during the present study.

Evaporation test was positive in 96.66 % of cases. All the four patients in which the test was negative were referred after 24 hours with dry labour and intranatal infection. The accuracy of this test according to some other workers is Schiotz[49] 89.5%, Chitra Sarin[50] 99%.. The findings of the various workers are similar to those obtained in present study.

The litmus paper test was positive in 96.66% of cases. All the four patients in which the test was negative were referred after 24 hours with dry labour and intranatal infection.

The diagnosis of high leak was made when speculum examination revealed fluid escaping through the cervix with positive laboratory tests but subsequently the bag of membranes was found to be present at or before delivery. Out of the 120 patients 21.67% had high leak (Table: 10).

Out of the 120 patients with spontaneous PROM, 85. (70.84%) developed spontaneous onset of labour, 4 (3.33%) were treated conservatively with pregnancy continuing for variable length of time, while in 31 patients (25.83%) pregnancy was terminated either by Oxytocin induction or caesarean section (Table : 9).

34.16% of the patients with PROM were delivered by caesarean section in the present study and 65.84% had vaginal delivery with or without Oxytocin and / or forceps and Ventouse. (Table: 12). The overall rate of caesarean section in the general population during the study period was 10.64%. Therefore, the incidence of caesarean section in PROM is significantly higher.

Of the 41 cases delivered by caesarean section (Table 13) 17 (41.46%) have had to be delivered by caesarean section irrespective of their association with PROM. For the remaining 24 deliveries by caesarean section, PROM was one of the important factors leading to caesarean section. This gives the corrected rate of caesarean section in PROM as 20% (24 out of 120). The incidence of caesarean section in patients with PROM reported in the literature are 1to7% [Eastman[84], Breese[11], Gunn[6] , Kodkany[19] - 19% and Anjana Devi[21] - 45.2%. The higher incidence of caesarean section delivery in the present series as compared to that reported in the literature is probably due to the fact that this center is one of the referral hospitals and therefore the overall incidence of complicated labour is higher.

When latent periods where analyzed (Table : 14) 19.23% of the patients with high leak had latent period of more than 24 hours , while in the low leak group the corresponding figure was 14.89%.

Similarly when total duration of leak was analyzed (Table : 15) , it was found that in the high leak group 34.62% cases where associated with total duration of leak more than 24 hours, while in the low leak group, the corresponding figure was 24.46%.

Eight out of 120 patients developed amnionitis giving an incidence of 6.66% of amnionitis in patients with PROM (Table: 16). When cases with amnionitis were

analyzed according to latent period, amongst the high leak group no patients developed amnionitis when the latent period was less than 24 hours and 1 patient (20%) developed amnionitis when it was more than 24 hours. However in the low leak group the incidence of amnionitis was 2.5% when the latent period was 24 hours or less and 35.7 % when the latent period exceeded 24 hours. When both high and low leak were considered together, 1,98% (2out of 101) developed amnionitis when latent period was 24 hours or less, but when it was more than 24 hours, 31.58% developed amnionitis. This confirms the findings that longer the latent period, more is the incidence of amnionitis made by number of workers.

The incidence of post-partum morbidity during the present study was 15.83 % (Table: XVII). When post-partum maternal morbidity was compared to the latent period, it was found that longer the latent period, higher was the post- partum morbidity. None of the patients in the high leak group developed post-partum morbidity when the latent period was less than 24 hours, but when the latent period was more than 24 hours; the incidence of post-partum morbidity was 20.00%. In the low leak group, the incidence of post-partum morbidity was 12.5% when the latent period was 24 hours or less and 57.14% when the latent period was more than 24 hours.

When amnionitis was compared with the total duration of leak, amongst the high leak group no patient developed amnionitis when the total duration of leak was less than 24 hours and 1 patient (11,11%) developed amnionitis when it was more than 24 hours (Table : 18). However in the low teak group the incidence of amnionitis was 2.81% when the total duration of leak was 24 hours or less and 21.73% when the total duration of leak exceeded 24 hours. When both high and low leak were considered together, 2.27% (2 out of 88) developed amnionitis when the

total duration of leak was 24 hours or less, but when the total duration of leak was more than 24 hours, 18.75 %developed amnionitis.

When the post-partum maternal morbidity was compared with the total duration of leak, (Table : 19) it was found that longer the total duration of leak, higher was the post-partum morbidity. None of the patients in the high leak group developed post-partum morbidity when the total duration of leak was less than 24 hours, but when the total duration of leak was more than 24 hours, the incidence of post-partum morbidity was 11.11%. In the low-leak group, the incidence of post-partum morbidity was 14.08% when the total duration of leak was 24 hrs or less and 34.78% when the total duration of leak was more than 24 hours.

When causes of maternal morbidity was analyzed (table 22), it indicated puerperal fever in 63.157%, Amnionitis is 6.66%, endometritis in 1.75%, wound infection in 19.29% and urinary tract infection in 7.01%.

The perinatal mortality in PROM in this study was 5.73%, but the overall incidence of perinatal mortality in the study period was 6.4% (Table: 20). When perinatal mortality was compared to the total duration of leak (Table; 20), the incidence of perinatal mortality was 4.44%, when the total duration of leak was 24 hours or less and 9.37% when it was more than 24 hours. This confirms the findings of various workers that the perinatal mortality is directly proportional to the total duration of leak.

When perinatal deaths were analyzed according to the latent period (Table; 21)**,** it is obvious that longer the latent period, higher is the perinatal mortality. These findings are in agreement with those reported by number of authors like Breese [11], Gunn [6] et al, Sanyal and Mukherjee[78] and Anjana Devi and Reddi Rani [21].

When various causes for the perinatal deaths were analyzed (Table : 22), it was found that 42.86% of the perinatal deaths were due to neonatal infection. Respiratory distress syndrome was responsible for 57.14% of the perinatal deaths. When the data was analyzed according to maturity 85.72% of the perinatal deaths occurred in premature and 14.28% of the perinatal deaths occurred in mature infants.

In premature infants 33.33% (2 out of 6) deaths were due to perinatal infection and 66.66% (4out of 6) deaths were due to RDS. Thus neonatal infection and RDS were important causes of deaths in premature. These findings are in agreement with those reported by Sanyal and Mukherjee[78] and Anjana Devi and Reddi Rani [21].

SUMMARY

1) Incidence of PROM at BLDEU's Shree B M Patil Medical College, Bijapur was 7.44%.
2) Incidence of malpresentation and twins in PROM was 11.02 % and 1.67 % respectively. Incidence of breech presentation was also higher (10.18%).
3) None of the suspected predisposing factors were present in 45% of cases, while one or more suspected predisposing factors were present in 55% of cases.
4) The incidence of prematurity was 14.16% , significantly higher than the overall incidence.
5) The efficacy of different diagnostic tests for PROM are
    a) Direct Visualization test 96.66%
    b) Crystallization test 96.66%
    c) Litmus paper test 96.66%
    d) Evaporation test 96.66%
6) Incidence of high leak was quite significant - 21,67%.
7) Conservative management could be undertaken in only 3.33% . Active termination of pregnancy with Oxytocin or Caesarean section had to be undertaken in 25.83% of the cases, while 70.84% of the patients went into labour spontaneously.
8) Overall Caesarean section rate in PROM was 34.16% , while the corrected rate was 20%.
9) The duration of latent period and total duration of leak were directly related to the development of amnionitis, maternal morbidity and perinatal mortality.

10) The incidence of amnionitis, maternal morbidity and perinatal mortality was comparatively less in patients with high leak.

11) The overall incidence of amnionitis was 6.66% and postpartum maternal morbidity was 15.83%. When latent period was 24 hours or less amnionitis and maternal morbidity were present in 1.98% and 9.90% respectively. But when the latent period exceeded 24 hours, incidence of amnionitis and maternal morbidity rose to 31.58% and 47.36% respectively.

Similarly when the total duration of leak was 24 hours or less, amnionitis and maternal morbidity were present in 2.27% and 11.36% respectively. However when the total duration of leak exceeded 24 hours, incidence of amnionitis and maternal morbidity rose to 18.75% and 28.12% respectively.

12) When causes of maternal morbidity was analyzed (table 22), it indicated puerperal fever in 63.157%, Amnionitis is 6.66%, endometritis in 1.75%, wound infection in 19.29% and urinary tract infection in 7.01

13) Incidence of perinatal mortality in PROM was 5.73%. When the latent period was 24 hours or less the incidence of perinatal mortality was 3.88% , but when the latent period exceeded 24 hours , the incidence of perinatal mortality sharply rose to 15.78%.

Similarly when the total duration of leak was less than 24 hours, the incidence of perinatal mortality was 4.44% . But when the total duration of leak exceeded 24 hours, the incidence of perinatal mortality sharply rose to 9.37%.

14) Perinatal infection was responsible for 42.86% of the perinatal deaths, while RDS was responsible for 57.14% of perinatal deaths. Prematurity with its hazards - infection and RDS were responsible for 85.72% of perinatal deaths.

CONCLUSION

A total of 120 patients of proved PROM were studied. Detailed history, Clinical examination and laboratory tests were undertaken to confirm the diagnosis of PROM. Effects of age, parity, singleton and multiple pregnancy and presentation on incidence of PROM were studied. Six suspected predisposing factors viz - coitus, past cervical operations, leaking in previous pregnancy, chronic cervicitis/vaginitis, travelling, Antenatal pelvic examination were also studied.

Patients with pregnancy between 34 to 36 weeks, not in labour and not having amnionitis were managed conservatively. Pregnancy was terminated promptly in those who developed amnionitis, or went into labour. All patients with pregnancy more than 37 weeks were terminated by induction with Oxytocin or by caesarean section depending on the case.

Latent period and total duration of leak, their effect on pregnancy and its relation to amnionitis, maternal morbidity and perinatal mortality were also studied. The various causes of perinatal mortality were also analyzed.

BIBLIOGRAPHY

1. Aaron B Caughey MD, MPP, PhD, Julian N Robinson, MD and Errol R Norwitz, MD. Contemporary diagnosis and management of Premature Rupture of Membranes. Rev Obstet Gynecol 2008, 1 (1):11-22.
2. American College of Obstetrician and gynecologists, author. Premature Rupture of membranes. Washington DC: American College of obstetricians and Gynecologists; 1998. (ACOG practice bulletin No. 1)
3. ACOG committee on Practice Bulletin- Obstetrics authors. Clinical Management guidelines for obstetrician- gynecologist. (ACOG practice bulletin No. 80: Premature Rupture of membranes) Obstet Gynec. 2007;109: 1007-1019.
4. Atkins H J. Premature Rupture of membranes and prematurity. Am J Obstet Gynec 1949;58:565.
5. Calkins, L. Premature Spontaneous Rupture of Membranes. Am. J. Obst. and Gynec 1952; 64:871-877.
6. Gunn GL, Mishell DR, Morton DG. Premature rupture of membranes of fetal membranes: a review. Am J Obstet Gyneccol 1970;106:469-83.
7. Gunn GL, Mishell DR, Morton DG. Incidence of PROM. Am J Obstet Gynecol 1970;106:469.
8. Naeye RL, Peter EC. Factors that predispose to premature rupture of membranes. Obstet Gynecol 1982;60:1982.
9. Duff P, Huff RN, Gibbs S RS. Management of premature rupture of membranes and unfavourable cervix in term pregnancy. Obstet Gynecol 1984; 63 :69.
10. Arias F, Tomich PH. Etiology and outcome of low birth weight and preterm infants. Obstet gynecol 1982;60:277-8.

11. Breese M W. Spontaneous premature rupture of the membranes. Am J Obstet Gynec 1961;81:1086.
12. Burchell RC. Premature spontaneous rupture of membranes. Am J Obstet Gynecol 1964;88:251.
13. Lanier LR Jr, Scarbrough RW Jr et al. Incidence of maternal and fetal complications associated with rupture of membranes before onset of labor. Am J Obstet Gynecol 1965 oct 1;93:398-404.
14. Friedman ML, McElin T W. Diagnosis of ruptured fetal membranes. Am J Obstet gynecol 1969;104:544-50.
15. Jiwane K A. Antenatal vaginal examination as a cause of premature rupture of membranes. J Obstet Gynec India 1991; 41:337.
16. Harger JH, Hsing AW, Tuomala RE, et al. Risk factors for preterm premature rupture of fetal membranes: a multicentre case-control study. Am J Obstet Gynecol 1990;163:130.
17. Bhalerao S, Desai A. Premature rupture of membranes. In:Saraiya UB, Rao KB, Chatterjee A, eds. Principles and practice of Obstetrics and gynecology 2 nd edition. An FOGSI publications. New Delhi: Jaypee Brothers, 2003:125.
18. Daftary SN, Desai SV. Preterm labour and premature rupture of membranes. In:Daftary SN, Desai SV eds. Selected topics in obstetrics and gynecology 2nd edition. New delhi: BI Publications, 2006:128.
19. Lenihan John P Jr MD. Relationship of antepartum pelvic examinations to premature rupture of membranes. Obstet gynecol 1984; 63:33.
20. Kodkany BS, Telang MA. Premature rupture of membranes: A study of 100 cases. J Obstet and Gynecol India 1991;41:492.

21. Vintzileos AM, Campbell WA, Nochimson DJ, Weinbaum PJ. The use of non stress test in patients with premature rupture of membranes. Am J Obstet Gynec 1986;155(1);149-53.
22. Devi Anjana, Reddi Rani. Premature rupture of membranes: A clinical study. J of Obstet Gynec India; 1996;46:63-67.
23. Dare MR, Middleton P, Crowther C.A, Flenady V.J, et al. Planned early birth versus expectant management for prelabour rupture of membranes at term (37 weeks). Cochrane Database of systemic reviews 2006, Issue 1. Art No: CD005302. DOI: 10.1002/ 14651858. CD005302. Pub2 level 1.
24. Evaldson G, Largelius A et al. Premature rupture of membranes. Acta Obstet Gyneacol scand 1980;59:385.
25. Knox I C Jr, Hoerner JK. The role of infection in premature rupture of the membranes. Am J Obstet Gynec 1995; 173:951.
26. Naeye R L. Causes and consequences of premature rupture of membranes of fetal membranes. Lancet 1980; 1:192.
27. Bourne G. The Human Amnion and Chorion. London, Llyod luke 1962:175
28. Artal R, Burgeson RE, Hobel SG, Hollister D. An in vitro model for the study of enzymtically mediated biochemical changes in the chorioamniotic membranes. Am J Obstet Gynec 1979;133:656.
29. Iams JD, Stilson R, Johnson FF, Williams Ra, Rice R. Symptoms that precede preterm labour labour and preterm premature rupture of the membranes. Am J Obstet Gynec 1990 Feb;162 (2):486-90.
30. Brosens I and Gorden H. The cytological diagnosis of ruptured membranes using Nile Blue sulphate staining. Obstet Gynec J of Br comm. 1965;72:342.

31. P. Gaucherand, S Guiband, RC Rudigoz et al. Laboratory investigations in the rapid diagnosis of premature rupture of membranes. Acta Obstet Gynec Scand 1994;73 (6):456-59.
32. Bourgeois GA. Identification of fetal squames and diagnosis of ruptured membranes by vaginal smear. Am J Obstet Gynec 1942;44:80-7.
33. Hopman BC. A method of detection of ruptured membranes through examination of vaginal cytology. Am J Obstet Gynec 1952;63:1342.
34. Hopman BC, Wargo JD, SC. Cytology of vernix caseosa cells. Obstet gynecol 1957;10:656-9.
35. Averette HE, hopman BC, Ferguson JH. Cytodiagnosis of ruptured fetal membranes. Am J Obstet Gynecol 1963;87:226-30.
36. Kushner DH, Chang IW, Vercruvsse JM. Flouroscence microscopy for determination of ruptured fetal membranes by vaginal smear Obstet Gynecol 1964;23:196–9.
37. Goldfine S. The detection of ruptured membranes by vaginal smear. Am J Obstet Gynecol 1955;70:109-14.
38. Baptisti A. Chemical test for determination of ruptured membranes. Am J Obstet Gynecol 1938;35:688-90.
39. Abe T. Detection of rupture of fetal membranes with nitrazine indicator. Am J Obstet Gynecol 1940;39:400-4.
40. Gall SA, Spellacy WN. Cytologic diagnosis of ruptured membranes. Obstet gynecol 1964;24:732-5.
41. Tricomi V, Hall JE, Bittar A, Chambers D. Arborization test for the detection of ruptured fetal membranes- clinical evaluation. Obstet gynecol 1966;27:275-9

42. Bornstein J, Geva A, Solt I, Fait V, Schoenfeld A, Shoham HK, et al. Nonintrusive diagnosis of premature ruptured amniotic membranes using a novel polymer. Am J Perinatol 2006;23:351–4
43. Papanicolaou GN. A general survey of the vaginal smear and its use in research and diagnosis. Am J Obstet gynecol 1946;51:316
44. Marcel Ferron and Rolland Bilodeau. Amniotic Fluid Crystallisation test for ruptured membranes. Can med Assoc 1963; 89:1064-1067
45. Anjaneylu R and Likhite MG. The Fern Phenomenon or arborisation test of amniotic period. J of Obstet Gynec india 1967;17:170
46. Gupta P L and Gupta P. Arborization test of amniotic fluid. J Obstet Gynecol India 1977;27:315
47. Borten M and freidman E A. Amniotic fluid ferning in early gestation. Am J Obstet Gynec 1986;154: 628
48. Smith R P. A technique for the detection of rupture of membranes. A review and preliminary report. Obstet Gynecol 1976;48:172
49. Ianetta Odilon. A new simple test for detecting rupture of fetal membranes. Obstet Gynecol 1984;63:575
50. Schiotz H. The evaporation test for detecting rupture of fetal membranes. Acta Obstet Gynecol Scand 1987;66:245
51. Sarin Chitra. A simple technique for the detection of ruptured fetal membranes. J of Obstet Gynecol India 1987;37:57
52. Manning FA, Hill FM, Platt LD. Qualitative amniotic fluid volume determination by ultrasound. Antepartum detection of intrauterine growth retardation. Am J Obstet Gynecol 1981;139:254–8.

53. Robson MS, Turner MJ, Stronge JM, O'Herlihy CO. Is amniotic fluid quantitation of value in the diagnosis and conservative management of prelabour membrane rupture at term? Br J Obstet Gynaecol 1990;97:324–8.

54. Erdemoglu E, Mungan T. Significance of detecting insulin-like growth factor binding protein-1 in cervicovaginal secretions: comparison with nitrazine test and amniotic fluid volume assessment. Acta Obstet Gynecol Scan 2004;83:622–6.

55. Atlay RD, Sutherst JR. Premature rupture of the fetal membranes confirmed by intra-amniotic injection of dye (Evans blue T-1824). Am J Obstet Gynecol 1970;108:993–4.

56. Gibbs RS, Blanco JD. Premature Rupture of the membranes. Obstet Gynecol 1982;60:671–9.

57. Petrunin DD, Griaznova IM, Petrunina IuA, Tatarinov IuS. Immunochemical identification of organ specific human placental alpha-microglubulin and its concentration in amniotic fluid [article in Russian]. Akush Ginekol (Mosk) 1977;1:64–5.

58. Cousins LM, Smok D, Lovett SM, Poeltler DM. AmniSure placental alphamicroglobulin-1 rapid immunoassay versus standard diagnostic methods for detection of rupture of membranes. Am J Perinatol 2005;22:1–4.

59. Lee SE, Park JS, Norwitz ER, Kim KW, Park HS, Jun JK. Measurement of placental alpha-microglobulin-1 in cervicovaginal discharge to diagnosis rupture of membranes. Obstet Gynecol 2007;109:634–40.

60. Mulhair L, Carter J, Poston L, Seed P, Briley A. Prospective cohort study investigating the reliability of the AmnioSens method for detection of spontaneous rupture of membranes. BJOG 2009;116:313–8.

61. Sacks Morton and Baker TH. Spontaneous premature rupture of membranes. A prospective study. Am J Obstet Gynec 1967;97:888
62. Varner Michael and Galask Rudolph. Conservetive management of premature rupture of membranes. Am J Obstet Gynec 1981;140:39
63. Conway D I, Prendiville W J, Morris A, Speller D C E. Management of spontaneous rupture of the membranes in the absence of labour in primigravida women at term. Am J Obstet Gynec 1984;150:947.
64. Mercer BM, Crocker L, Boe N et al. Induction versus expectant management in premature rupture of membranes with mature amniotic fluid at 32 to 36 weeks: a randomized trial. Am J Obstet Gynecol 1993;82:775-82
65. Cunningham F G and Mac Donalds PC. Williams Obstetrics. 20 th edition. 1997
66. Sperling RS, Ramamurthy RS, Gibbs RS. A comparison of intrapartum versus immediate poatpartum treatment of intraamniotic infection. Obstet gynecol 1987;70:801-5
67. Kenyon SL, Tauylor DS, Tarnow Mordi W et al. Broad spectrum antibiotic for preterm premature rupture of membranes: the ORACLE 1 randomized trial. Lancet 2001;357:979-88
68. Johnston MM, Sanchez- Ramos L, Vaughn AJ, Todd MW et al. Antibiotic therapy in preterm premature rupture of membranes: a randomized, prospective, double blind trial. Am J Obstet Gynecol 1990 seo;163 (3):743-7
69. Young BK, Klein SA, Kartz M, Wilson SJ, Douglas GW. Intravenous dexamethasone for prevention of neonatal respiratory distress: A prospective controlled study. Am J Obstet Gynecol 1980;138(2):203-9

70. Morales WJ, Diebel ND, Lazar AJ, Zadrozny D. The effect of antenatal dexamethasone administration on the prevention of RDS in preterm gestations with premature rupture of membranes. Am J Obstet gynecol 1986 Mar;154:591-5
71. Lewis D F, Brody K, Edwards M S. Preterm premature ruptured membranes. A randomized trial of steroids after treatment with antibiotics. Obstet Gynecol 1996;88:801-5.
72. Singh Daljit. Neonatal infection following premature rupture of amniotic membranes. J of Obstet Gynec India 1989;39:232
73. Ballard M B. Premature rupture of membranes. Am J Obstet Gynec 1936;32:455
74. Russell K P. The aggressive management of ruptured membranes. Am J Obstet Gynecoln1962;83:930
75. Clark D M and Anderson G V. Perinatal mortality and amnionitis in a general hospital population. Obstet Gynec 1964;31:714
76. Miller J H. bacterial colonization of amniotic fluid in the presence of ruptured membranes. Am j Obstet Gynecol 1980;137:451
77. Schreiber James. Conservative management of preterm premature rupture of fetal membranes in a low socio economic status. Am J Obstet Gynecol 1980;136:92
78. Morales W J, Angel JL, et al. A randomized study of antibiotic therapy in idiopathic preterm labor. Obstet gynecol 1988;72:829-33
79. Sanyal M K and Mukherjee T N. Premature rupture of membranes: An assessment from a rural medical college of west Bengal. J Obstet Gynecol India 1990;40:623
80. Moretti M, Sibai BM. Maternal and perinatal outcome of expectant management of premature rupture of membranes in Midtrimester. Am J Obstet gynecol 1988;159:390-6

81. Nelson L H , Anderson R L, O'shea TM et al. Expectant management of preterm premature rupture of membranes. Am J Obstet Gynecol 1994;171:350
82. Vintzileos AM, Campbell WA, Nochomson DJ et al. Preterm rupture of the membranes: a risk factor for the development of abruptio placenta. Am J Obstet Gynecol 1987;156:1235-8
83. Webb G A. Maternal death associated with premature rupture of membranes. Am J Obstet Gynecol 1967;98:594
84. Lebherz T B. Double blind study pf premature rupture of membranes: A report of 1896 cases. Am J Obstet Gynecol 1963;87:218
85. Eastman N J and Hellman L M. Williams Obstetrics, 13th edition, New York Appleton century crofts, 1966
86. Hadi N A. Premature rupture of membranes between 20 and 25 weeks gestation: Role of amniotic fluid in perinatal outcome. Am J Obstet Gynecol 1994;170:1139
87. Shubeck F, Benson RC, Clarke WW Jr,et al. Fetal hazards after rupture og the membranes. A report from the collaborative project 1966;28:22-31
88. Nimrod C, Varela- Gittings F, Machin G et al. The effect of very prolonged ruptured membranes on fetal development. Am J Obstet Gynecol 1984;148:540-3
89. Rotschild AVI. Neonatal outcome after prolonged preterm rupture of membranes. Am J Obstet Gynecol 1990;162:46.

ANNEXURE A

TO STUDY THE MATERNAL AND FETAL OUTCOME IN PREGNANCY COMPLICATED BY PREMATURE RUPTURE OF MEMBRANES

PROFORMA

SI NO :

NAME :

AGE :

ADDRESS :

DOA : **DOD:**

RELIGION :

EDUCATION :

SOCIO ECONOMIC STATUS:

CHIEF COMPLAINTS:

H/O P V LEAK

COLOR

QUANTITY

H/O PAIN IN ABDOMEN

H/O FEVER

H/O WHITE DISCHRARGE PV

PRESENTING COMPLAINTS :

OBSTETRIC HISTORY :

Married Life :

Obstetric Score :

Details of Previous Pregnancy:

1.	
2.	
3.	
4.	

History of Contraception **:**

MENSTRUAL HISTORY: PaMC:

Regular/ Irregular

Flow of Scanty/ Moderate/ Severe

Dysmenorrhoea

LMP:

EDD:

Period of Gestation:

PAST HISTORY:

H/O Cervical Incompetence

H/O Antecedent coitus

H/O Travelling

H/O PV Examinations

H/O chronic cervicitis

H/O Leaking in previous pregnancies

H/O Cervical operation

H/O Trauma

H/O Amniocentesis

H/O External cephalic Version

Family History:

Personal History:

Diet:

Sleep:

Bladder/ bowel

Addiction

GENERAL PHYSICAL EXAMINATION

Built and Nourishment : Vitals :

Height : Pulse :

Weight : BP :

Pallor : Temp :

Edema :

SYSTEMIC EXAMINATION

CVS

RS

PER ABDOMEN

PER SPECULUM EXAM

PER VAGINAL EXAM

DIAGNOSIS

INVESTIGATIONS

Hb%

TC

DC L/N/E/M/B

BLOOD GROUPING WITH Rh TYPING

URINE ANAYSIS

CRP

HBsAg

HIV

FERN TEST

LITMUS TEST CRYSTALLIZATION TEST:

USG EVAPORATION TEST:

MODE OF DELIVERY:

INDICATION FOR LSCS:

TOTAL DURATION OF LABOUR

MATERNAL COMPLICATIONS:

FETAL COMPLICATIONS:

ASSOCIATED COMPLICATIONS:

ANNEXURE B

RESEARCH INFORMED CONSENT FORM

TITLE OF THE TOPIC: Maternal and fetal outcome in pregnancy Complicated by Premature Rupture of Membranes

PRINCIPAL INVESTIGATOR : Dr. SHRUTI SRINIVAS

PG GUIDE NAME : Dr.V.R.GOBBUR
Professor

PURPOSE OF RESEARCH

I have been informed that this study is to evaluate the maternal and fetal outcome in pregnancy complicated by premature rupture of membranes. I have also been given a free choice of participation in this study.

PROCEDURE

I understand that I will be a part of this study. My history and physical findings will be taken from the case paper and will be evaluated in a systematic way. I will not be asked for any follow up.

RISK AND DISCOMFORTS

I understand that this procedure is not expected to aggravate any side effect or cause detrimental effect to me or my child.

BENEFITS

I understand that my participation in the study will help to study the maternal and fetal morbidity and mortality in pregnancy complicated by Premature Rupture of Membranes.

CONFIDENTIALITY

I understand that the medical information produced by this study will become a part of hospital records and will be subject to the confidentiality and privacy regulation of BLDE University's Shri .B. M .Patil Medical college. Information of a sensitive personal nature will not be a part of the medical records, but will be stored in the investigator's research file and identified only by a code number. The code key connecting names to numbers will be kept in a secured location**.**

If the data are used for publication in the medical literature or for teaching purpose no names will be used**.**

I understand that the relevant designated authority and permitted to have an access to

my medical record and to the data produced by the study for audit purpose. However, they are required to maintain confidentiality**.**

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at any time and understand that I will be informed of any significant new finding discovered during the course of the study, which might influence my continued participation.

If during the study or later I wish to discuss my participation or concerns regarding this study with a person not directly involved I am aware that the other staff members are available to talk with me.

This 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 to my present of future care in the hospital and also understand that the researcher may terminate my participation in the study if at any time he feels the need and explain me the reason to do and help to arrange for my further appropriate treatment.

ANNEXURE C

INJURY STATEMENT:

I understand that in the unlikely event of injury to me resulting directly from my participation in this study, if such injury were reported promptly, the appropriate treatment would be available to me. 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 Mrs._____the purpose of the research, the procedures required and the possible risks to the best of my ability in her own language

INVESTIGATOR DATE:

(Dr.SHRUTI SRINIVAS)

I confirm that Dr. SHRUTI SRINIVAS ,has explained to me the purpose of research, the study procedure, that I am will to undergo the investigation and the possible discomforts as well as benefits. I have been explained all the above in detail in my own language and I understand the same. Therefore I agree to give consent to participate as a subject in this research project.

PARTICIPANT DATE:

ANNEXURE E

KEY TO MASTER CHART

PVL : Per vaginal leaking

PIA : Pain in Abdomen

SPF : Suspected predisposing factors

PCO : Past cervical operations

LPP : Leaking in previous pregnancy

CC : Chronic cervicitis

V : Vaginitis

PVE : Per vaginal examination

AC : Antecedent Coitus

T : Travelling

fTP : Full Term Pregnancy

DVT : Direct Visualisation test

CT : Crystallization test

LPT :Llithmus paper test

ET : Evaporation test

CA : Conservative approach

AOL : Accelerartion of labour

IOL : Induction of labour

LSCS : Lower segment cesarean section

SD : Spontaneous Delivery

FTVD : Full term vaginal delivery

PTVD : Pre term vaginal delivery

FTPAVD: Full term pitocin accelerated Vaginal delivery

FTPIVD: Full term pitocin induced vaginal delivery

VeD : Ventouse delivery

FoD : Forceps delivery

ABD : Assisted Breech delivery

FD : Fetal Distress

FTP : Failure to progress

DL : Dysfunctional labour

TP : Twin pregnancy

PROM : Premature rupture of membranes

PIH : Pregnancy induced Hypertension

PWB : Primi with Breech

S : Shoulder Presentation

CPD : Cephalopelvid disproportion

TL : Transverse Lie

PC : Previous Section

CF : Cervical Fibriod

A : Amnionitis

PF : Puerperal fever

WI : Wound Infection

UTI : Urinary tract infection

E : Endometritis

PM : Prematurity

PD : Post datism

CI : Cervical Incompetence

RI : Rh incompatibility

PE : Pre eclampsia

MP : Malpresentation

C :Conjuctivitis

CV : Congenital Varicella

In : Infection

RDS : Respiratory distress syndrome

FSB : Fresh still born

MA : Meconium Aspiration

BE : Baby expired

Sl. No	Name	Age	I.P.D	P.V.I Since	P.I.A Since	S.P.F	Gravida	GEST AGE	D.V.T	C.T	I.P.T	E.T	Management	M.O.D	Ind for L.S.C.S
1	M.T	20	92732	4 HRS	2 HRS	-	Primi	fTP	+	+	+	+	A.O.L	F.T.V.D	-
2	M.S	32	92808	1 HRS	-	-	Primi	fTP	+	+	+	+	L.S.C.S	L.S.C.S	C.F
3	A.S.	20	92819	4.5 HRS	4 HRS	-	2	fTP	+	+	+	+	S.D	F.T.V.D	-
4	R.M.	20	92930	6 HRS	-	V	Primi	fTP	+	+	+	+	S.D	F.T.V.D	-
5	V.J.	27	62405	1 HRS	-	L.P.P	4	fTP	+	+	+	+	L.S.C.S	L.S.C.S	PC (2)
6	S.P.	22	93685	9.5 HRS	6 HRS	-	2	fTP	+	+	+	+	A.O.L	FTPAVD	-
7	S.B.	26	94000	3.5 HRS	3 HRS	A.C	2	fTP	+	+	+	+	A.O.L	FTPAVD	-
8	K.M.	19	94021	4 HRS	-	V	3	fTP	+	+	+	+	A.O.L	FTPAVD	-
9	S.M.	20	94096	27 HRS	24 HRS	-	Primi	fTP	+	+	+	+	L.S.C.S	L.S.C.S	FTP
10	S.S.	27	94374	48 HRS	8 HRS	L.P.P	8	35 wks	-	-	-	-	S.D	PTVD	-
11	S.G.	20	94574	7.5 HRS	4.5 HRS	-	2	fTP	+	+	+	+	A.O.L	FTPAVD	-
12	M.D.	25	94580	4 HRS	-	-	Primi	fTP	+	+	+	+	A.O.L	FTPAVD	-
13	S.V.	27	94677	3 HRS	-	-	Primi	fTP	+	+	+	+	I.O.L	FTPAVD	-
14	S.G.	20	94730	14 HRS	12 HRS	T.P.C.O	2	fTP	+	+	+	+	A.O.L	FTPAVD	-
15	S.S.	25	53046	4 HRS	-	-	2	fTP	+	+	+	+	A.O.L	FTPAVD	-
16	M.S.	22	94901	62 HRS	-	A.C	Primi	fTP	-	-	-	-	L.S.C.S	L.S.C.S	PWB
17	M.B.	26	94756	14 HRS	-	L.P.P	2	fTP	+	+	+	+	I.O.L	L.S.C.S	FTP
18	L.C.	22	77010	12 HRS	-	-	2	fTP	+	+	+	+	I.O.L	L.S.C.S	FTP
19	L.M.	19	95260	6 HRS	4 HRS	V.P.C.O	2	fTP	+	+	+	+	S.D	F.T.V.D	-
20	R.K.	27	95258	10 HRS	4 HRS	V.P.C.O	3	fTP	+	+	+	+	S.D	F.T.V.D	-
21	S.B.	26	95271	5 HRS	3 HRS	-	3	fTP	+	+	+	+	S.D	ABD	-
22	A.K.	20	79684	2 HRS	1 HRS	A.C	2	fTP	+	+	+	+	A.O.L	FTPAVD	-
23	D.K.	23	95964	5 DAYS	-	P.C.O	3	36 wks	-	-	-	-	C.A	P.T.V.D	-
24	L.M.	19	96001	9 HRS	7 HRS	A.C	Primi	fTP	+	+	+	+	A.O.L	FTPAVD	-
25	A.K.	28	38789	3 HRS	2 HRS	P.C.O	4	34 wks	+	+	+	+	S.D	PTVD	-
26	S.M.	18	91452	9 HRS	8 HRS	-	Primi	fTP	+	+	+	+	A.O.L	FTPAVD	-

Sl. No	Name	Age	I.P.D	P.V.I Since	P.I.A Since	S.P.F	Gravida	GEST AGE	D.V.T	C.T	I.P.T	E.T	Management	M.O.D	Ind for L.S.C.S
27	V.S.	22	95987	1 HRS	-	-	Primi	fTP	+	+	+	+	L.S.C.S	L.S.C.S	TP (PIH)
28	S.N.	26	77854	7 HRS	-	P.C.O	4	fTP	+	+	+	+	A.O.L	FTPAVD	-
29	S.K.	25	91076	5 HRS	2 HRS	-	Primi	fTP	+	+	+	+	A.O.L	FTPAVD	-
30	S.M.	28	96116	7 HRS	4 HRS	P.C.O	Primi	36 wks	+	+	+	+	S.D	PTVD	-
31	J.M.	32	96146	4 HRS	3 HRS	P.C.O	3	fTP	+	+	+	+	L.S.C.S	L.S.C.S	CPD
32	S.P.	19	96302	17 HRS	15 HRS	-	Primi	34 wks	+	+	+	+	S.D	PTVD	-
33	S.I.	25	96325	8 HRS	-	A.C.T	2	fTP	+	+	+	+	I.O.L	L.S.C.S	FD
34	V.J.	20	96544	18 HRS	7 HRS	P.C.O	Primi	fTP	+	+	+	+	L.S.C.S	L.S.C.S	FTP
35	S.G.	22	96695	6 HRS	5 HRS	P.C.O	3	fTP	+	+	+	+	A.O.L	FTPAVD	-
36	S.P.	19	88305	6 HRS	3 HRS	-	Primi	fTP	+	+	+	+	A.O.L	VED	-
37	A.B.	23	96769	8 HRS	-	-	Primi	fTP	+	+	+	+	I.O.L	FTPAVD	-
38	S.K.	25	96794	12 HRS	-	-	Primi	fTP	+	+	+	+	I.O.L	FOD	-
39	S.M.	19	96823	24 HRS	14 HRS	-	Primi	fTP	+	+	+	+	S.D	F.T.V.D	-
40	J.S.	23	96848	4 DAYS	-	P.C.O	2	fTP	+	+	+	+	I.O.L	FTPAVD	-
41	V.T.	26	96891	3 HRS	-	-	2	fTP	+	+	+	+	I.O.L	VED	-
42	A.J.	30	97119	6 HRS	5 HRS	-	2	fTP	+	+	+	+	I.O.L	F.T.V.D	-
43	V.P.	18	97199	2 DAYS	8 HRS	-	Primi	fTP	+	+	+	+	S.D	FOD	-
44	C.D.	27	96978	1 HRS	-	P.C.O	4	35 wks	+	+	+	+	I.O.L	PTVD	-
45	S.P.	19	97912	12 HRS	8 HRS	-	Primi	fTP	+	+	+	+	L.S.C.S	L.S.C.S	CPD
46	A.K.	21	97743	5 HRS	3 HRS	-	Primi	fTP	+	+	+	+	A.O.L	FTPAVD	-
47	V.M.	20	97781	12 HRS	-	A.C.V	Primi	35 wks	+	+	+	+	I.O.L	PTVD	-
48	S.S.	20	96712	5 HRS	3 HRS	-	Primi	fTP	+	+	+	+	A.O.L	FTPAVD	-
49	K.I.	25	97833	4 HRS	-	A.C.V	Primi	36 wks	+	+	+	+	A.O.L	PTVD	-
50	V.P.	23	98221	7 HRS	6 HRS	-	Primi	fTP	+	+	+	+	A.O.L	FTPAVD	-
51	R.D.	24	82651	1 HRS	-	P.C.O	2	fTP	+	+	+	+	L.S.C.S	L.S.C.S	PWB
52	A.J.	28	98676	1 HRS	-	A.C.V	Primi	fTP	+	+	+	+	A.O.L	L.S.C.S	FTP

Sl. No	Name	Age	I.P.D	P.V.I Since	P.I.A Since	S.P.F	Gravida	GEST AGE	D.V.T	C.T	I.P.T	E.T	Management	M.O.D	Ind for L.S.C.S
53	N.M.	21	97956	6 HRS	-	P.V.E	Primi	fTP	+	+	+	+	A.O.L	F.T.V.D	-
54	A.B.	21	98407	2 HRS	-	A.C.P.V.	3	fTP	+	+	+	+	S.D	F.T.V.D	-
55	S.R.	24	98524	3 HRS	-	A.C.T	Primi	fTP	+	+	+	+	A.O.L	FTPAVD	-
56	M.A.	20	98556	1 HRS	-	A.C.T	Primi	fTP	+	+	+	+	A.O.L	FTPAVD	-
57	S.P.	23	84434	40 HRS	-	-	2	fTP	+	+	+	+	L.S.C.S	L.S.C.S	PC
58	M.B.	26	98746	7 HRS	1 HRS	V	Primi	fTP	+	+	+	+	A.O.L	L.S.C.S	DL
59	S.D.	22	98840	3 HRS	-	T	2	36 wks	+	+	+	+	A.O.L	PTVD	-
60	M.S.	24	99017	2 DAYS	4 HRS	A.C.V	Primi	fTP	+	+	+	+	L.S.C.S	L.S.C.S	CPD
61	S.K.	27	55463	2 HRS	1 HRS	A.C.V	2	fTP	+	+	+	+	S.D	FTVD	-
62	P.P	29	99036	7 HRS	4 HRS	A.C.T	2	fTP	+	+	+	+	S.D	FTVD	-
63	S.Y	22	99055	2 HRS	-	A.C.T	Primi	fTP	+	+	+	+	I.O.L	FTPIVD	
64	U.M	20	99133	13 HRS	8 HRS	A.C.V	Primi	fTP	+	+	+	+	A.O.L	FTPAVD	-
65	J.C	19	99196	7 HRS	-	-	Primi	fTP	+	+	+	+	A.O.L	LSCS	FTP
66	U.P	25	99036	2 HRS	-	-	2	fTP	+	+	+	+	L.S.C.S	LSCS	PC
67	S.K.	19	99566	2 DAYS	-	-	Primi	fTP	+	+	+	+	I.O.L	FTPAVD	FTP
68	B.P	25	99684	11 HRS	8 HRS	V.P.V.E	2	fTP	+	+	+	+	A.O.L	LSCS	-
69	A.P	22	99739	10 HRS	8 HRS	V.P.V.E	2	fTP	+	+	+	+	L.S.C.S	LSCS	FD
70	S.M.	25	99782	16 HRS	10 HRS	-	Primi	fTP	+	+	+	+	L.S.C.S	LSCS	PWB
71	B.K	19	99792	4 HRS	-	-	Primi	fTP	+	+	+	+	I.O.L	LSCS	FD
72	V.S.	22	100153	2 HRS	-	-	4	fTP	+	+	+	+	L.S.C.S	LSCS	FD
73	M.K	22	100163	2 HRS	-	-	4	fTP	+	+	+	+	L.S.C.S	LSCS	FD
74	S.M.	35	100661	10 HRS	6 HRS	-	2	fTP	+	+	+	+	L.S.C.S	LSCS	CPD
75	M.P	24	100712	12 HRS	-	P.C.O	2	fTP	+	+	+	+	A.O.L	LSCS	FTP
76	L.M.	25	101015	4 HRS	-	P.C.O	2	35 wks	+	+	+	+	C.A	PTVD	-
77	M.C	25	100907	3 HRS	-	P.C.O	2	35 wks	+	+	+	+	C.A	PTVD	-
78	S.M.	30	101312	14 HRS	3 HRS	-	2	fTP	+	+	+	+	I.O.L	FTPIVD	-

Sl. No	Name	Age	I.P.D	P.V.I Since	P.I.A Since	S.P.F	Gravida	GEST AGE	D.V.T	C.T	I.P.T	E.T	Management	M.O.D	Ind for L.S.C.S
79	S.P.	22	101398	12 HRS	-	-	Primi	fTP	+	+	+	+	A.O.L	FTPAVD	-
80	L.S	24	101666	6 HRS	2 HRS	P.C.O	2	fTP	+	+	+	+	A.O.L	FOD	-
81	A.P	28	101730	20 HRS	-	L.P.P	Primi	fTP	+	+	+	+	L.S.C.S	LSCS	FTP
82	B.D	21	101929	2 DAYS	-	-	Primi	fTP	+	+	+	+	I.O.L	LSCS	FTP
83	P.P	22	101934	3 DAYS	-	-	Primi	fTP	+	+	+	+	S.D	FTVD	-
84	U.H	22	102199	2 DAYS	1 HRS	-	2	35 wks	+	+	+	+	A.O.L	PTVD	-
85	J.D	23	102262	48 HRS	-	A.C	Primi	fTP	+	+	+	+	I.O.L	FTPIVD	-
86	S.P.	24	102536	4 DAYS	-	A.C.V	2	fTP	+	+	+	+	L.S.C.S	LSCS	PC
87	S.P.	23	102555	2 HRS	-	A.C.V	Primi	fTP	+	+	+	+	A.O.L	FTPAVD	-
88	Y.J	22	102843	10 HRS	-	A.C	Primi	fTP	+	+	+	+	S.D	FTVD	-
89	U.G	25	103067	12 HRS	-	P.C.O	2	35 wks	+	+	+	+	A.O.L	PTVD	-
90	J.B	26	103002	5 HRS	2 HRS	A.C	Primi	fTP	+	+	+	+	I.O.L	FTPIVD	-
91	R.D.	23	103334	2 HRS	-	P.C.O	3	fTP	+	+	+	+	I.O.L	FTPIVD	-
92	B.K	22	103340	2 HRS	-	A.C	2	fTP	+	+	+	+	A.O.L	LSCS	FTP
93	A.K.	22	103417	2 HRS	-	A.C.A	2	fTP	+	+	+	+	L.S.C.S	LSCS	CPD
94	S.M.	28	103548	20 HRS	16 HRS	A.C	3	fTP	+	+	+	+	S.D	FTVD	-
95	B.K	27	103573	8 HRS	-	-	Primi	fTP	+	+	+	+	A.O.L	LSCS	FTP
96	R.J	30	103619	8 HRS	-	P.C.O	3	fTP	+	+	+	+	I.O.L	FTPIVD	-
97	M.K	27	103689	16 HRS	-	-	3	34 wks	+	+	+	+	I.O.L	PTVD	-
98	J.D	23	103876	1.5 HRS	-	A.C	Primi	fTP	+	+	+	+	I.O.L	FTPIVD	-
99	A.K.	20	103993	8 HRS	-	-	Primi	fTP	+	+	+	+	I.O.L	FTPIVD	-
100	A.T	24	104171	1.5 HRS	-	-	Primi	fTP	+	+	+	+	I.O.L	LSCS	FTP
101	S.K.	22	104521	6 HRS	4 HRS	A.C	Primi	fTP	+	+	+	+	S.D	FOD	-
102	B.K	19	104622	40 HRS	6 HRS	P.C.O	Primi	fTP	+	+	+	+	A.O.L	FTPAVD	-
103	S.D.	22	104680	2 HRS	-	-	4	fTP	+	+	+	+	L.S.C.S	LSCS	PC
104	P.P	22	104798	8 HRS	-	L.P.P	2	fTP	+	+	+	+	A.O.L	FTPIVD	-

Sl. No	Name	Age	I.P.D	P.V.I Since	P.I.A Since	S.P.F	Gravida	GEST AGE	D.V.T	C.T	I.P.T	E.T	Management	M.O.D	Ind for L.S.C.S
105	S.B.	22	104965	3.5 HRS	-	-	2	fTP	+	+	+	+	I.O.L	FTPIVD	-
106	M.A.	20	105414	14 HRS	4 HRS	A.C	Primi	fTP	+	+	+	+	A.O.L	LSCS	FTP
107	H.M	30	105969	54 HRS	30 HRS	-	2	fTP	-	-	-	-	L.S.C.S	LSCS	DL
108	S.P.	20	106047	18 HRD	6 HRS	A.C.V	Primi	fTP	+	+	+	+	L.S.C.S	LSCS	FTP
109	R.M.	25	106453	24 HRS	4 HRS	-	Primi	fTP	+	+	+	+	L.S.C.S	LSCS	PWB
110	R.D.	21	104875	24 HRS	-	P.C.O	2	34 wks	+	+	+	+	L.S.C.S	PTVD	-
111	S.K.	20	106463	6 HRS	2 HRS	-	Primi	fTP	+	+	+	+	S.D	FTVD	-
112	S.T	25	106894	72 HRS	12 HRS	-	Primi	fTP	+	+	+	+	A.O.L	FTPAVD	-
113	R.S	24	106963	10 HRS	2 HRS	-	3	fTP	+	+	+	+	L.S.C.S	LSCS	FD
114	R.S	22	107024	2 HRS	-	-	Primi	fTP	+	+	+	+	I.O.L	LSCS	FTP
115	S.T	27	107321	4 HRS	-	P.C.O	5	35 wks	+	+	+	+	C.A	PTVD	-
116	R.S	19	107556	22 HRS	-	-	Primi	fTP	+	+	+	+	L.S.C.S	LSCS	PWB
117	P.N	27	107669	14 HRS	13 HRS	-	3	fTP	+	+	+	+	L.S.C.S	LSCS	PC
118	R.S	20	108266	2 HRS	-	P.C.O	3	fTP	+	+	+	+	A.O.L	FTPAVD	-
119	S.T	18	108321	9 HRS	7 HRS	-	Primi	34 wks	+	+	+	+	S.D	PTVD	-
120	M.M	23	108429	4 HRS	-	-	Primi	33 wks	+	+	+	+	A.O.L	PTVD	-

Type of Leak	Distribution of Labour			I.P	T.D.L	Materna 1 Comp	Foetal Comp	Asso Comp
	Ist S	2nd S	3rd S					
High	6 hrs	15 min	5 min	2 hrs	8 hrs	-	-	-
Low	-	-	-	-	3.5 hrs	-	C.V	R.I.C.F
Low	7.5 hrs	25 min	5 min	30 min	8.5 hrs	-	C	PC
Low	8.5 hrs	25 min	5 min	1 hrs	9.5 hrs	-	-	-
Low	-	-	-	-	2.5 hrs	PF.WI	-	PC,MP
High	21 hrs	20 min	5 min	3.5 hrs	24.5 hrs	-	-	-
Low	3 hrs	15 min	5 min	30 min	3.5 hrs	-	-	RI
Low	4 hrs	15 min	5 min	9 hrs	13.5 hrs	PF.WI	C	MP-
Low	-	-	-	3 hrs	28 hrs	PF	C	-
Low	8 hrs	10 min	5 min	36 hrs	44 hrs	PF	FSB (IN)	PC,PM
High	5 hrs	20 min	5 min	3 hrs	8 hrs	-	-	-
Low	18 hrs	30 min	10 min	4 hrs	22 hrs	-	-	-
Low	10 hrs	30 min	5 min	5 hrs	15 hrs	-	-	-
Low	25 hrs	30 min	5 min	2 hrs	28 hrs	PF	RDS,MA	RI
Low	7 hrs	30 min	5 min	5 hrs	12 hrs	PF	-	-
High	-	-	-	-	62 hrs	PF.WI	-	MP
Low	-	-	-	17 hrs	26 hrs	-	C	PD
Low	-	-	-	10 hrs	26 hrs	PF.WI	C	-
Low	5 hrs	30 min	5 min	2 hrs	8 hrs	-	-	-
Low	13 hrs	20 min	5 min	6 hrs	19 hrs	-	-	MP-
Low	6 hrs	30 min	5 min	2 hrs	8 hrs	-	-	MP
Low	3 hrs	15 min	5 min	30 min	4 hrs	PF	-	-
High	12 hrs	30 min	10 min	5 days	5.5 days	PF,A	RDS	PM
Low	5 hrs	30 min	5 min	9 hrs	14 hrs	PF,UTI	-	PE
Low	3 hrs	15 min	5 min	1 hrs	4 hrs	-	C	PM,CI
Low	13 hrs	15 min	5 min	1 hrs	14 hrs	-	C	-

Type of Leak	Distribution of Labour			I.P	T.D.L	Materna 1 Comp	Foetal Comp	Asso Comp
	Ist S	2nd S	3rd S					
Low	-	-	-	-	3.5 hrs	-	-	PIH,TP
Low	2.5 hrs	15 min	5 min	10 hrs	13 hrs	-	C	CI
Low	8 hrs	30 min	5 min	3 hrs	11.5 hrs	-	-	-
Low	15 hrs	30 min	5 min	3 hrs	18 hrs	PF,UTI	BE (RDS)	PM,PE
Low	-	-	-	1 hrs	6 hrs	PF.WI	-	CPD,PC
Low	20 hrs	20 hrs	10 min	2 hrs	22 hrs	PF,A	-	PH
Low	-	-	-	10 hrs	16 hrs	W.F	-	-
Low	-	-	-	11 hrs	18 hrs	PF.WI	C	RI
Low	5 hrs	25 min	5 min	1 hrs	6 hrs	-	-	-
Low	9 hrs	2 hrs	10 min	3 hrs	13 hrs	PF	-	-
Low	5.5 hrs	30 min	10 min	8.5 hrs	14 hrs	PF	-	-
High	7.5 hrs	2 hrs	10 min	13 hrs	20 hrs	PF	RDS	-
Low	14 hrs	20 min	5 min	10 hrs	24 hrs	PF,A	-	-
Low	6 hrs	30 min	5 min	4 days	4 days	-	-	-
High	7 hrs	1 hrs	10 min	3.5 hrs	11 hrs	-	C	PM
Low	2.5 hrs	15 min	5 min	6 hrs	9 hrs	PF	-	PM
High	10 hrs	2 hrs	5 min	36 hrs	48 hrs	-	-	-
High	12 hrs	20 min	10 min	1 hrs	13 hrs	-	-	PM
Low	-	-	-	4 hrs	16 hrs	PF,A	-	CPD,PD
Low	8 hrs	15 min	5 min	2 hrs	10 hrs	-	-	-
Low	8 hrs	25 min	5 min	12 hrs	20 hrs	-	-	PM
Low	8 hrs	15 min	5 min	2 hrs	10 hrs	-	-	-
High	5 hrs	20 min	5 min	5 hrs	10 hrs	E	-	PM
Low	10 hrs	30 min	5 min	1 hrs	11 hrs	-	-	PD
Low	-	-	-	1 hrs	3 hrs	-	-	MP
High	12 hrs	-	-	4 hrs	19 hrs	-	-	-

Type of Leak	Distribution of Labour			I.P	T.D.L	Materna 1 Comp	Foetal Comp	Asso Comp
	Ist S	2nd S	3rd S					
Low	8 hrs	30 min	5 min	6 hrs	14 hrs	-	-	-
High	7 hrs	30 min	5 min	2 hrs	9 hrs	-	-	PD
Low	7 hrs	30 min	5 min	20 min	8 hrs	-	-	-
Low	3 hrs	30 min	10 min	30 min	4 hrs	-	-	PE
Low	-	-	-	-	42 hrs	PF	-	CPD,MP
Low	-	-	-	6.5 hrs	15 hrs	PF	RDS	-
High	13 hrs	5 min	5 min	5 hrs	18 hrs	-	RDS	PM
Low	-	-	-	48 hrs	50 hrs	PF,A	RDS	CPD,PD
LOW	2 hrs	10 min	5 min	1 hrs	3 hrs	-	-	-
LOW	4 hrs	15 min	5 min	3 hrs	7 hrs	-	-	-
LOW	18 hrs	30 min	5 min	2 hrs	21 hrs	-	-	-
LOW	12 hrs	20 min	5 min	5 hrs	17 hrs	-	-	PD
LOW	-	-	-	10 hrs	18 hrs	-	-	-
LOW	-	-	-	1 hrs	8 hrs	PF	-	PC
High	-	-	-	48 hrs	56 hrs	PF	-	PM
LOW	10 hrs	10 min	5 min	3 hrs	13 hrs	-	-	-
LOW	-	-	-	2 hrs	18 hrs	PF	MA	PC
LOW	-	-	-	6 hrs	18 hrs	-	-	MP
LOW	-	-	-	6 hrs	14 hrs	PF	-	-
LOW	4 hrs	25 min	5 min	50 hrs	54 hrs	-	RDS	PM,MP
LOW	-	-	-	3 hrs	4 hrs	PF	RDS,C	PM,MP
LOW	-	-	-	10 hrs	11 hrs	PF	RDS,MA	CPD,PD
High	-	-	-	14 hrs	24 hrs	WI	-	PM,C1
High	3 hrs	15 min	5 min	96 hrs	100 hrs	-	-	PM
High	4 hrs	15 min	5 min	5 days	6 days	PF	C	PM
LOW	3 hrs	10 min	5 min	11 hrs	14 hrs	-	C	-

Type of Leak	Distribution of Labour			I.P	T.D.L	Materna 1 Comp	Foetal Comp	Asso Comp
	Ist S	2nd S	3rd S					
High	5 hrs	10 min	5 min	12 hrs	17.5 hrs	-	-	-
High	11 hrs	60 min	5 min	4 hrs	15 hrs	PF	-	PIH
LOW	-	-	-	-	24 hrs	-	-	PD
LOW	-	-	-	-	2 days	-	RDS	PD
High	4 hrs	25 min	5 min	2 days	2 days	-	-	PM
High	6 hrs	30 min	5 min	48 hrs	54 hrs	-	-	PM
LOW	5 hrs	20 min	5 min	48 hrd	53 hrs	PF	-	-
LOW	-	-	-	-	96 hrs	WI	-	PM,MP
LOW	5 hrs	30 min	5 min	2 hrs	7 hrs	-	-	-
High	14 hrs	40 min	10 min	2 hrs	16 hrs	PF	-	-
High	31 hrs	15 min	5 min	12 hrs	44 hrs	-	-	PC,PM,M
High	8 hrs	20 min	5 min	3 hrs	11 hrs	-	-	-
LOW	3 hrs	30 min	5 min	10 hrs	13 hrs	-	-	PC,PM
LOW	-	-	-	2 hrs	12 hrs	-	-	PC
LOW	-	-	-	-	4 hrs	-	-	CPD,PD
LOW	20 hrs	30 min	5 min	4 hrs	24 hrs	-	RDS	-
High	-	-	-	8 hrs	22 hrs	WI	-	PD
LOW	6 hrs	20 min	5 min	8 hrd	14 hrs	-	-	PD
LOW	5 hrs	15 min	5 min	16 hrs	21 hrs	-	RDS	PM, TP
LOW	7 hrs	30 min	5 min	2 hrs	9 hrs	UTI	-	-
LOW	7 hrs	30 min	5 min	9 hrs	16 hrs	-	-	-
LOW	-	-	-	3 hrs	12 hrs	WI	BE(RDS)	PD
LOW	6 hrs	20 min	10 min	2 hrs	8 hrs	-	FSB (IN)	PM
LOW	10 hrs	20 min	5 min	34 hrs	44 hrs	-	-	-
LOW	-	-	-	2 hrs	4 hrs	PF	-	PC
High	3 hrs	20 min	5 min	9 hrs	12 hrs	-	-	PIS

Type of Leak	Distribution of Labour			I.P	T.D.L	Materna 1 Comp	Foetal Comp	Asso Comp
	Ist S	2nd S	3rd S					
LOW	5 hrs	20 min	5 min	3 hrs	8 hrs	-	-	MP
LOW	-	-	-	10 hrs	20 hrs	PF.A	RDS	-
LOW	-	-	-	24 hrs	57 hrs	PF.A	FSB (IN)	MP-
High	-	-	-	12 hrs	19 hrs	-	-	PD
LOW	-	-	-	20 hrs	25 hrs	-	-	CPD
LOW	12 hrs	20 min	5 min	24 hrs	36 hrs	-	-	PM
LOW	6 hrs	15 min	5 min	4 hrs	10 hrs	-	-	-
LOW	14 hrs	30 min	5 min	60 hrs	74 hrs	UTI	RDS	PE
LOW	-	-	-	8 hrs	12 hrs	-	-	PC
LOW	-	-	-	4 hrs	12 hrs	-	-	PD
LOW	3 hrs	15 min	5 min	72 hrs	75 hrs	-	BE(IN)	PM
LOW	-	-	-	-	26 hrs	-	-	MP
LOW	-	-	-	1 hrs	20 hrs	PF	-	PC,MP(S)
LOW	5 hrs	20 min	5 min	2 hrs	7 hrs	-	-	MP
LOW	7 hrs	20 min	5 min	2 hrs	9 hrs	-	-	-
LOW	4 hrs	20 min	5 min	2 hrs	6 hrs	-	-	PM