INTRODUCTION

Prelabor rupture of membranes (PROM), previously known as premature rupture of membranes, refers to spontaneous rupture of membranes at least 1 hour before the onset of uterine contractions. Preterm PROM (PPROM) refers to rupture of membranes before 37 weeks 0 days' gestation. PROM complicates 8–10% of all pregnancies. PPROM is associated with 30–40% of preterm deliveries and is the leading identifiable cause of preterm delivery. It occurs in about 1–3% of all pregnancies.\(^1\) PPROM can lead to significant perinatal morbidity associated with prematurity such as respiratory distress syndrome, neonatal sepsis, umbilical cord prolapse, placental abruption and fetal death.\(^2\) It contributes to about 20% of all perinatal deaths and is also associated with maternal morbidity. The pathogenesis and management of patients with PPROM are presented in this chapter in line with current evidence.

Terminology

- **Prolonged rupture of membranes** is any rupture of membranes that persists for more than 24 hours and prior to the onset of labor
- **Previable premature rupture of membranes** is rupture of membranes before viability limit, usually **before** 23 weeks. Complicates 0.1–0.7% of pregnancies.
- **Latent period** is the time from membrane rupture until delivery
- **Expectant management**: management of patients with the goal of prolonging gestation ('watchful waiting until delivery indication arises')
PATHOGENESIS

The etiology of PPROM is multifactorial. The final common pathway in the occurrence of PROM is disruption of fetal membranes. Fetal membranes are complex in structure with two components: the choriodecidua, which is relatively thick and cellular, and the amnion, which is thinner and stronger. The chorion and amnion are closely adherent consisting of several cell types, including epithelial cells, mesenchymal cells, and trophoblast cells, embedded in a collagenous matrix. The amnion accounts for approximately 20% of the thickness of the fetal membranes, but dominates the mechanical response of the fetal membranes. The physical integrity of these membranes maintains the pregnancy until term, and in a majority of women, they would only rupture after the onset of contractions or after artificial intervention during labor.

Rupture of the fetal membranes is precipitated by stretch forces acting upon biochemically mediated, pre-weakened fetal membranes. Fetal membranes weaken in late gestation as a result of biochemical changes involving extracellular matrix remodeling and apoptosis. The pre-weakening could also occur before term due to defective collagen, infections or other inflammatory processes. Acute inflammation is associated with increased proteolytic enzymes and activation of cytokines, which trigger the cascade of matrix breakdown. Hemorrhage in early pregnancy can cause membrane weakness as it also triggers the inflammatory cascade. Decidual hemorrhage or abruptio placenta results in decidual production of thrombin to initiate hemostasis. Like cytokines, thrombin has been demonstrated to induce dose-dependent weakening of the fetal membranes in vitro with concomitant remodeling and apoptosis which mimic that seen in the physiological weak zone of term fetal membranes. Increased mechanical stretch of fetal membranes may also result in loss of integrity and resultant rupture of membranes remote from term. Examples of conditions that could cause increased stretching of the membranes are twin gestation and polyhydramnions. Sac exposure due to isthmus–cervix incompetence may also result in PPROM, since the mechanical forces on the herniating membranes are higher, as they are concentrated on a small surface area. The herniated membranes are also exposed and have higher chance of infection.

RISK FACTORS

Although there appears to be no single etiology for PPROM, there are a number of risk factors that have been identified. Studies indicate that the presence of certain risk factors increase the possibility of PPROM occurrence compared with others. The factors that contribute to PPROM can be broadly classified as maternal, uteroplacental or fetal in origin (Table 1). The common factors at play include intrauterine infection, decidual hemorrhage (placental abruption), excessive uterine stretch, and maternal or fetal stress. These factors set off mechanisms that cause activation of factors and cascades that inhibit uterine quiescence which lead to PPROM.

Table 1 Risk factors for spontaneous preterm premature rupture of membranes (PPROM)

<table>
<thead>
<tr>
<th>Maternal factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>• PPROM in a prior pregnancy (recurrence risk is 16–32% as compared with 4% in women with a prior uncomplicated term delivery)</td>
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<tr>
<td>• Antepartum vaginal bleeding</td>
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<tr>
<td>• Chronic steroid therapy</td>
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<tr>
<td>• Collagen vascular disorders (such as Ehlers-Danlos syndrome, systemic lupus erythematosus)</td>
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<tr>
<td>• Direct abdominal trauma</td>
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<tr>
<td>• Preterm labor</td>
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<tr>
<td>• Cigarette smoking</td>
</tr>
<tr>
<td>• Illicit drugs (coca)</td>
</tr>
<tr>
<td>• Anemia</td>
</tr>
<tr>
<td>• Low body mass index (BMI &lt;19.8 kg/m^2)</td>
</tr>
<tr>
<td>• Nutritional deficiencies of copper and ascorbic acid</td>
</tr>
</tbody>
</table>
- Low socioeconomic status
- Unmarried status

**Uteroplacental factors**

- Uterine anomalies (such as uterine septum)
- Placental abruption (may account for 10–15% of preterm PROM)
- Advanced cervical dilatation (cervical insufficiency)
- Prior cervical conization
- Cervical shortening in the second trimester (<2.5 cm)
- Uterine overdistention (polyhydramnios, multiple pregnancy)
- Intra-amniotic infection (chorioamnionitis)
- Multiple bimanual vaginal examinations (but not sterile speculum or transvaginal ultrasound examinations)

**Fetal factors**

- Multiple pregnancy (preterm PROM complicates 7–10% of twin pregnancies)

History of PPROM in a prior pregnancy has been a consistent risk factor for PPROM.\(^\text{10,11}\) The risk of recurrence is 16-32%, as compared with approximately 4% in women with a prior uncomplicated term delivery.\(^\text{14}\) Black women also have been shown to have increased the risk of PPROM due to the increased likelihood of placenta abruption compared to other ethnicities.\(^\text{15,16}\) Decreased collagen content is linked to it, as well as collagen vascular disorders like Ehlers-Danlos and systemic lupus erythematosus.\(^\text{11}\) Factors resulting in uterine distension have been associated with PPROM including conditions such as multiple pregnancies and polyhydramnios. Other risk factors include chronic steroid therapy, anemia, abdominal trauma, smokers, illicit drug use, history of sexually transmitted infections, lower body mass index, procedures (cerclage and amniocentesis) and vaginal hemorrhage. Invasive uterine procedures performed during pregnancy (such as amniocentesis, chorionic villus sampling, fetoscopy and cervical cerclage) can damage the membranes, causing them to leak, but these are rare causes of PPROM. Most cases of PPROM occur in otherwise healthy women without identifiable risk factors.\(^\text{11}\) Epidemiological and historical factors that are known not to be associated with PPROM include sexual intercourse, speculum examinations, maternal exercise and parity.\(^\text{13}\)

### CLINICAL FEATURES

**Clinical history**

Typically, a woman presents with a history of a large gush of fluid down the legs, followed by a steady trickle or a feeling of “wetness”. The color, consistency and smell are important factors to consider during history taking and examination. Other features typical in presence of chorioamnionitis include abdominal pain, fever, and a foul smelling vaginal discharge.

**Examination findings**

An assessment of vital signs is necessary to rule out infection. Maternal tachycardia and elevated temperature are signs of infection as well as abdominal tenderness. Palpable fetal parts could be encountered due to reduced amniotic fluid. A sterile speculum examination is essential in patients with suspected PPROM. The examiner should look for pooling of liquor in the posterior fornix. The patient can be told to cough or perform Valsalva maneuver during the speculum exam if liquor is not obviously seen to trickle out of the cervical os. If there is no pooling, vaginal fluid should be obtained for the confirmatory tests discussed below. The pool can also be collected and sent for determination of fetal lung maturity, if the gestational age is greater than 32 weeks. The pooled liquor is collected into a specimen bottle, which is then taken to the laboratory. The test done depends on what is available. There are various tests for lung maturity such as lamellar body count undertaken by a standard hematology analyser, Lecithin/Sphingomyelin ratio done on paper
During the speculum examination, the patient's cervix should be assessed for cervical dilatation and for the presence of cord prolapse. Cervical secretions should also be sent for culture.

It is important to rule out possible differential diagnoses from both clinical history and examination. These may include leakage of urine (urinary incontinence); excessive vaginal discharge, such as physiological discharge or bacterial vaginosis; and cervical mucus (show) as a sign of impending labor.\[13\]

## DIAGNOSIS

The diagnosis of PPROM is mainly clinical. The patient usually presents with a history of leakage of fluid vaginally. This should be followed by a sterile speculum examination to document rupture of membranes by visualization of amniotic fluid pooling in the posterior fornix or clear fluid flowing from the cervical canal. Laboratory tests can then be used to confirm the clinical diagnosis or to make the diagnosis when it is uncertain. Sonographic examination is also undertaken to assess amniotic fluid volume, identify the presenting part, estimate gestational age (if not previously performed), and estimate fetal weight. The specific confirmatory tests are discussed below.

### Nitrazine paper test

Nitrazine or phenaphthazine is a pH indicator dye. Amniotic fluid usually has a pH range of 7.0–7.3 compared with the normally acidic vaginal pH of 3.8–4.2 and the normal acidic pH of urine of 5.0–6.0.\[17\] This test involves putting a drop of fluid obtained from the vagina onto paper strips containing nitrazine dye. The strips change color depending on the pH of the fluid. The strips will turn from yellow to blue if the pH is greater than 6.0 (Figure 1). A blue strip means it is more likely that the membranes have ruptured. False positive test results may occur with co-existent blood, semen, soap, or bacterial vaginosis, while false negatives may result from scanty amniotic fluid within the vaginal canal.

![Figure 1](image)

**Figure 1** The nitrazine paper test: (A) normal; (B) bacterial vaginosis; (C) pregnant woman with premature rupture of membranes

### Arborization or ferning test

Fluid from the posterior vaginal fornix is swabbed onto a glass slide and allowed to dry for at least 10 minutes. Amniotic fluid produces a delicate ferning pattern, in contrast to the thick and wide arborization pattern of dried cervical mucus.\[18\] Well-estrogenized cervical mucus or a fingerprint on the microscope slide may cause a false-positive fern test. False negatives may occur due to inadequate amniotic fluid on the swab or heavy contamination with vaginal discharge or blood.
**AmniSure**

AmniSure is a rapid slide test that uses immunochromatography methods to detect trace amounts of placental alpha microglobulin-1 (PAMG-1) protein in vaginal fluid. The concentration of this protein in amniotic fluid is 1,000–10,000 times higher than in cervicovaginal fluid. Consequently, the presence of high concentrations of PAMG-1 in cervicovaginal fluid is considered evidence of rupture of membranes, and the threshold of the test for the diagnosis has been set at 5.0 ng/mL. AmniSure test is not expected to be positive when fetal membranes are intact since the concentration of PAMG-1 in cervical vaginal secretions is less than 0.25 ng/mL. The sensitivity and specificity of PAMG-1 test in the diagnosis PROM are 97.3% and 98.7%, respectively.

Steps in carrying out the AmniSure test:

1. A sterile swab is inserted into the vagina for 1 minute, and then placed into a vial containing a solvent for 1 minute;
2. AmniSure test strip is dipped into the vial;
3. The test result is revealed by the presence of one or two lines within the next 5–10 minutes. One visible line means a negative result for amniotic fluid, two visible lines is a positive result, and no visible line is an invalid result (Figure 2).

![Figure 2 Process of the AmniSure test](image)

**Actim PROM**

This test is similar AmniSure but employs the identification of placental protein-12 also known as insulin-like growth factor binding protein-1 (IGFBP-1) in the vaginal fluid. This test has a positive predictive value (PPV) and negative predictive value (NPV) of 83.8% and 88.6%, respectively, in the diagnosis of PROM.

**Transabdominal intrauterine injection of dye**

Amniocentesis is performed and indigo carmine dye instilled into the amniotic cavity, under ultrasound guidance. This procedure may be used to help in the diagnosis of rupture of membranes when the diagnosis remains difficult despite using the standard non-invasive methods (Figure 3).

![Figure 3 Process of the transabdominal intrauterine injection](image)
Fetal fibronectin
Fetal fibronectin is a protein produced in pregnancy that functions as a ‘glue’ attaching the chorion to the decidua. A negative fetal fibronectin result strongly supports absence of membrane rupture, but a positive result only indicates disruption of the interface between chorion and decidua, which can occur with intact membranes as in the case of preterm labor.\textsuperscript{21}

Other tests
1. An ultrasound examination to check for reduced amniotic fluid volume and for assessment of fetal well-being can also be undertaken to support the diagnosis or aid in management as well as to ascertain fetal dating if not already done previously;
2. A full blood count to have a baseline white blood cell count is also performed since a new elevation of leukocyte count could be a sign of maternal infection. Other laboratory markers of infection include C-reactive protein (CRP) and procalcitonin;
3. If the antenatal profile of the woman has not been previously undertaken, it would be prudent to perform;
4. **Amniosense** is a pad commercially available in the UK, which looks like a panty liner, can detect the difference between urine and amniotic fluid by changing to a blue/green color when amniotic fluid is detected. This pad can be worn for up to 12 hours.

**MANAGEMENT**

A number of factors are considered where PPROM management is concerned. Some of the most important factors being gestational age and presence of infection. Other factors may include fetal status and well-being, fetal weight, maternal comorbidities, and presence of contractions or cervical dilation as well as availability of neonatal intensive care unit. Prematurity is the biggest concern in women with PPROM. Other complications include chorioamnionitis, oligohydramnions, necrotizing enterocolitis, cord compression, abruption placenta, respiratory distress syndrome, neurologic impairment and antepartum fetal death.\textsuperscript{10} In the absence of complications, management is centered on watchful waiting until 34 weeks, while in those beyond 34 weeks, delivery is considered after ascertainment of fetal lung maturity.

**Management based on gestational age**
In PPROM, there are several categories:

- 34–36 weeks
- 32–34 weeks
- 24–31 weeks
- Preivable PROM at <24 weeks.

**Gestational age: 34–36 weeks**
This group, usually, has attained lung maturity. Studies show that there is no benefit in conservative management in this group.\textsuperscript{22} Prolonging the pregnancy may serve to increase the risk of infection in both the mother and the neonate. Antibiotics are therefore used for prophylaxis, including group B streptococcus prophylaxis as discussed later in this chapter. Upon membrane rupture, the patient possesses a risk of ascending infection.\textsuperscript{13} This should therefore be balanced against the risk of prematurity. It is essential to monitor both the mother and fetus closely to rule out all possibilities of ascending infection. This would include daily monitoring of vital signs such as temperature and pulse, and assessing for uterine tenderness and foul discharge. Other investigations may include a full blood count with differential blood count. PROM is not a contraindication to vaginal delivery and all other factors including fetal status and obstetric...
indications (obstetric hemorrhage or cord accidents) may be considered in determining the mode of delivery.

**Gestational age 32–34 weeks**

In this group, fetal lung maturity has to be confirmed before delivery is considered. Conservative management is considered where infection has been ruled out, both mother and fetus are stable and where lung maturity is yet to be established. There is no benefit in prolonging pregnancy where lung maturity is confirmed, and this only serves to increase the risk of infection. Where lung maturity is not confirmed, steroids are given, as well as antibiotic prophylaxis. Magnesium sulfate for neuroprotection is also considered. Close monitoring for infection to rule out evidence of chorioamnionitis is key.

**Gestational age 24–31 weeks**

Conservative management is strongly advised unless contraindicated, i.e. in presence of infection, hemorrhage and fetal compromise. Administration of corticosteroids to ensure fetal lung maturity, antibiotic prophylaxis and magnesium sulfate for neuroprotection are advised. Monitoring for contractions in anticipation of preterm birth, close monitoring for infection and fetal monitoring by ultrasound and cardiotocography are essential. Transfer to facilities with NICU services and the involvement of a neonatologist/pediatric team is important.

**Preivable PROM**

Management in this scenario is highly individualized. Counselling of parents/families is important in ensuring that they are aware of the prognosis regardless of the interventions.

Even with prolongation of pregnancy and absence of infection, the fetus still has the risk of complications like Potter's syndrome, chronic lung disease, neurologic abnormalities and cerebral palsy.

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**MEDICAL MANAGEMENT IN PPROM**

The three major aspects of medical management in PPROM include corticosteroid use, tocolysis, magnesium sulfate and antibiotics use.

**Corticosteroid use for fetal lung maturity**

Administration of corticosteroids has been made standard practice for lung maturation in preterm birth and evidence supports the continued use of a single course of antenatal corticosteroids to accelerate fetal lung maturation in women at risk. It has been shown that treatment with antenatal corticosteroids reduces the risk of perinatal death, neonatal death, intraventricular hemorrhage, respiratory distress syndrome, necrotizing enterocolitis, requirement for respiratory support and NICU admission, even with presence of advanced neonatal care.

According to the World Health Organization, antenatal corticosteroid therapy is recommended for women at risk of preterm birth from 24 to 34 weeks of gestation provided that preterm birth is imminent, with accurate gestation dates and where maternal infection has been ruled out. This has to be in a setting where adequate care may be provided at childbirth and for the baby. According to this guideline, antenatal corticosteroid therapy is recommended in women with PPROM with no clinical signs of infection. Therefore its use is not recommended in women with features of chorioamnionitis.

**Dexamethasone and betamethasone dosage**

Betamethasone 12 mg given intramuscularly in two doses (once daily for 48 hours)

Dexamethasone 6 mg given intramuscularly in four doses (twice daily for 48 hours)

A single repeat course of antenatal corticosteroid is recommended by WHO if preterm birth does not occur within a week after the initial dose, and a subsequent clinical assessment demonstrates that there is a high risk of preterm birth in the coming week.
Presence of diabetes mellitus should not be a contraindication to administration of steroids for lung maturation. Additional monitoring should be undertaken and glucose control maintained.\textsuperscript{25,26} Corticosteroid use at 34–36 weeks remains controversial but could be administered in cases of confirmed absence of fetal lung maturity.

**PROPHYLACTIC ANTIBIOTIC THERAPY**

In conservative management of PPROM, the use of antibiotics has been recommended as it prolongs latency (delays the onset of preterm labor) and improves neonatal outcomes as well as reduces the risk of maternal infection.\textsuperscript{10} In a review by Kenyon,\textsuperscript{27} the use of antibiotics following PPROM was associated with statistically significant reductions in chorioamnionitis and a reduction in the numbers of babies born within 48 hours and 7 days. The following markers of neonatal morbidity were reduced: neonatal infection, use of surfactant, oxygen therapy and abnormal cerebral ultrasound scan prior to discharge from hospital. The demonstrated delay in onset of labor may also allow sufficient time for effective prophylactic corticosteroids.

The choice and regimen of antibiotics are such that they cover for common pelvic pathogens.\textsuperscript{27} The NICE guideline in 2015, recommends offering women with PPROM oral erythromycin 250 mg 4 times a day for a maximum of 10 days or until the woman is in established labor, whichever is sooner.\textsuperscript{23} The National Institute of Child Health and Human Development (NICHD) Maternal-Fetal Medicine Units (MFMU) Network and ACOG 2018 recommend intravenous ampicillin 2 g every 6 hours and erythromycin 250 mg every 6 hours for 48 hours followed by oral amoxicillin 250 mg every 8 hours and erythromycin 333 mg every 8 hours for 5 days.\textsuperscript{28} Co-amoxiclav is associated with an increased risk of neonatal necrotizing enterocolitis and is therefore avoided.\textsuperscript{27,29} The ORACLE Children Study, evaluating children’s health at older ages, found that antibiotics seemed to have little effect on the health of children.\textsuperscript{30} Acceptable antibiotic dosages and duration are as illustrated in Table 2 below.

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Acceptable antibiotic dosages and duration in PPROM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Azithromycin 1 g orally upon admission, PLUS ampicillin 2 g intravenously every 6 hours for 48 hours, FOLLOWED BY amoxicillin 500 mg orally three times daily or 875 mg orally twice daily for an additional 5 days.\textsuperscript{31}</td>
<td></td>
</tr>
<tr>
<td>Ampicillin 2 g every 6 hours and erythromycin 250 mg every 6 hours for 48 hours followed by oral amoxicillin 250 mg every 8 hours and erythromycin 333 mg every 8 hours for 5 days.\textsuperscript{29}</td>
<td></td>
</tr>
<tr>
<td>A first generation cephalosporin such as cefazolin or cephalexin can be used for those with penicillin allergy in combination with a macrolide for those with low risk penicillin allergy. For those with high risk, clindamycin and a macrolide can be used for a total duration of 5 days.\textsuperscript{31}</td>
<td></td>
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</tbody>
</table>

**Group B streptococcus prophylaxis**

The use of intrapartum group B streptococcus (GBS) antibiotic prophylaxis in women with preterm PPROM who are test positive for GBS is recommended, even if the patient has previously received a course of antibiotics after PPROM.

**TOCOLYSIS FOR INHIBITION OF PRETERM LABOR**

Tocolytic use, although associated with longer overall latency with fewer births within 48 hours and 7 days, has no significant effect on perinatal mortality.\textsuperscript{32} The principal indication for tocolysis in the setting of PPROM is to delay delivery for 48 hours to allow administration of corticosteroids. Women most likely to benefit from use of a tocolytic drug are those who are in early preterm labor, requiring in utero transfer for NICU or for completion of steroid dose. As a rule, tocolytics should not be used for more than 48 hours. Tocolytics are contraindicated in patients who are in advanced labor (>4 cm dilation) or who have any findings suggestive of subclinical or overt chorioamnionitis.

WHO guideline recommends nifedipine as a preferred tocolytic agent for preterm labor for a brief interval to allow for steroid administration. Although betamimetics may be effective in tocolysis, it is not recommended due to higher risk of
Regimens for tocolysis

1. Nifedipine an initial oral dose of 20 mg followed by 10–20 mg three to four times daily, adjust dose as per tocolytic effect for up to 48 hours.
2. Atosiban of an initial bolus dose of 6.75 mg over 1 minute, followed by an infusion of 18 mg/h for 3 hours, then 6 mg/h for up to 45 hours (to a maximum of 330 mg).
3. Avoid use of multiple tocolytics due to adverse effects.

MAGNESIUM SULFATE FOR NEUROPROTECTION

Evidence supports the use of antenatal magnesium sulfate administration in women at risk of preterm birth as neuroprotection against cerebral palsy for the baby. The protective effects of magnesium sulfate on neurological complications are likely to be increased at earlier gestational ages.

NICE recommends offering intravenous magnesium sulfate for neuroprotection of the baby to women between 24+0 and 29+6 weeks (and consideration for women at 30+0 and 33+6 weeks) of pregnancy who are in established preterm labor or having a planned preterm birth within 24 hours.

Dosing regimens for magnesium sulfate neuroprotection

1. Intravenous bolus of 4 g over 20 minutes, then 1 g/h until delivery or for 24 hours, whichever occurs first.
2. Intravenous bolus 4 g over 30 minutes as single dose.
3. Intravenous bolus 6 g over 20–30 minutes, followed by intravenous maintenance of 2 g/h.

*For women on magnesium sulfate, monitor for clinical signs of magnesium toxicity at least every 4 hours by recording pulse, blood pressure, respiratory rate and deep tendon (for example, patellar) reflexes and oliguria.

In summary, management of PPROM is largely influenced by gestational age (see Table 3). Conservative management is recommended until 34 weeks unless there are contraindications such as chorioamnionitis, fetal distress or active labor.

Table 3  Summary of management of PPROM

<table>
<thead>
<tr>
<th>Gestational age</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>34 weeks or more</td>
<td>Proceed to delivery, by induction of labor or a by cesarean section if indicated</td>
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<tr>
<td></td>
<td>Group B prophylaxis is recommended</td>
</tr>
<tr>
<td>32–33 completed weeks</td>
<td>Expectant management unless fetal lung maturity is documented</td>
</tr>
<tr>
<td></td>
<td>Group B streptococcus prophylaxis is recommended</td>
</tr>
<tr>
<td></td>
<td>Corticosteroids should be administered</td>
</tr>
<tr>
<td></td>
<td>Antibiotics to prolong latency</td>
</tr>
<tr>
<td>21–31 completed weeks</td>
<td>Group B streptococcus prophylaxis is recommended</td>
</tr>
<tr>
<td></td>
<td>Corticosteroids should be administered</td>
</tr>
<tr>
<td></td>
<td>Antibiotics to prolong latency</td>
</tr>
<tr>
<td></td>
<td>Magnesium sulfate should be administered incase delivery is imminent for neuroprotection</td>
</tr>
<tr>
<td>Before 24 weeks</td>
<td>Patient counseling</td>
</tr>
</tbody>
</table>
Complications

One of the most common complications of PPROM is early (premature) delivery. There exists an inverse relationship between the latent period, the time from membrane rupture until delivery, and the gestation at which PPROM occurs. A large study in Canada, United Kingdom and Australia demonstrated that slightly more than half of the women who had PPROM at 16–26 weeks' gestation had a latent period of 1 week with almost a quarter delivering after 4 weeks. In comparison, up to 95% of women with PROM near term had a latent interval of approximately 24 hours. There is a 3–4 fold risk in neonatal or perinatal mortality.

Neonates who survive PPROM are often affected by numerous complications, both in utero and after delivery. The following are likely complications in utero malpresentation, oligohydramnios, intrauterine infection which occurs in 15–30% of women with preterm PROM and accounts for 3–20% of neonatal deaths, umbilical cord compression, umbilical cord prolapse and abruption placenta. Immediately after delivery a fetus is at risk of necrotizing enterocolitis, neurological impairment, intraventricular hemorrhage, respiratory distress syndrome which occurs in 10–40% of neonates, retinopathy of prematurity, patent ductus arteriosus, limb restriction deformities which complicate approximately 12% of preterm PROM, pulmonary hypoplasia which develops in 26% of preterm PROM prior to 22 weeks and fetal asphyxia. Infection, cord accident and other factors contribute to the 1–2% risk of intrauterine fetal demise (stillbirth) after preterm PROM.

Potential maternal effects include, postpartum endometritis, chorioamnionitis and funisitis and septicemia in up to 33% of mothers, coagulopathy and delivery by cesarean section.

PRACTICE RECOMMENDATIONS

- A woman presenting with a history of gush of fluid vaginally must have a sterile speculum examination to confirm the diagnosis of PROM. Digital cervical examinations should be avoided since they decrease the latent period and increase risk of infection without added clinical benefit.
- Antibiotics should be administered to patients with preterm PROM because they prolong the latent period and improve outcomes.
- Corticosteroids should be given to patients with preterm PROM between 24 and 32 weeks' gestation to decrease the risk of intraventricular hemorrhage, respiratory distress syndrome, and necrotizing enterocolitis.
- Long-term tocolysis is not indicated for patients with preterm PROM, although short-term tocolysis may be considered to facilitate maternal transport and the administration of corticosteroids and antibiotics.
- Multiple courses of corticosteroids and the use of corticosteroids after 34 weeks' gestation are not recommended.
- When the diagnosis of chorioamnionitis is made, expeditious delivery should be done and intravenous antibiotics administered.

CONFLICTS OF INTEREST

The authors of this chapter declare that they have no interests that conflict with the contents of the chapter.
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