International Journal of Reproduction, Contraception, Obstetrics and Gynecology
Puttegowda SB et al. Int J Reprod Contracept Obstet Gynecol. 2026 Oct;15(10):4236-4242
www.ijrcog.org
pISSN 2320-1770 | eISSN 2320-1789

DOI: https://dx.doi.org/10.18203/2320-1770.ijrcog20263481

Review Article

Premature rupture of membranes: current concepts in diagnosis, management and prevention

SatishKumar B. Puttegowda[1], Chandana S. Ramakrishna[1]*, Shreyank H. Chandrashekara[1], Vijayalaxmi G. Sasavihalli[2], Ravindra B. Nagarajappa[1]

[1]Department of Pharmacy Practice, Sri Adichunchanagiri College of Pharmacy, Adichunchanagiri University, B. G. Nagara, Karnataka, India
[2]Department of Obstetrics and Gynaecology, Adichunchanagiri Hospital and Research Centre, Adichunchanagiri University, B. G. Nagara, Karnataka, India

Received: 22 July 2026
Accepted: 20 August 2026

***Correspondence:**
Dr. Chandana S. Ramakrishna,
E-mail: chandana10sr@gmail.com

ABSTRACT

Premature rupture of membranes (PROM) is an obstetric complication associated with adverse maternal and neonatal outcomes. Preterm PROM (PPROM) contributes substantially to preterm birth and unfavorable perinatal outcomes. This narrative review summarizes current evidence regarding the risk factors, diagnosis, management, and prevention of PROM and PPROM. A literature search was conducted using PubMed, Scopus, and Google Scholar for relevant publications from 2000 to 2026. Relevant clinical trials, systematic reviews, and clinical guidelines were included. Diagnosis of PROM requires clinical evaluation with the help of biochemical markers such as placental alpha microglobulin-1 (PAMG-1) and insulin-like growth factor binding protein-1 (IGFBP-1). The management strategies are based on gestational age and include antibiotic therapy, antenatal corticosteroids, magnesium sulfate for fetal neuroprotection, and careful assessment of maternal and fetal well-being. Early recognition, accurate diagnosis, and evidence-based management are essential for improving maternal and neonatal outcomes associated with PROM and PPROM. Management should be individualized according to gestational age and maternal and fetal clinical status, with appropriate use of antibiotics, antenatal corticosteroids, magnesium sulfate for fetal neuroprotection, and close monitoring when indicated. Preventive strategies addressing modifiable risk factors may further contribute to reducing the burden of PROM and PPROM.

Keywords: Preterm premature rupture of membranes, Diagnosis, Management, Corticosteroids, Antibiotic therapy

INTRODUCTION

Premature rupture of membranes (PROM) refers to rupture of the fetal membranes before the onset of labor. When membrane rupture occurs at or beyond 37 completed weeks of gestation, it is termed term PROM, whereas rupture before 37 completed weeks is referred to as PPROM.[1]

Preterm birth remains a leading cause of infant mortality worldwide, with PROM accounting for a substantial proportion of these births. In addition, PROM is associated with a perinatal mortality rate of approximately 18-20% and contributes to approximately 21.4% of adverse perinatal outcomes. Therefore, PROM is a major problem in resource-abundant and resource-limited healthcare facilities, and negatively impacts maternal, neonatal, and perinatal outcomes.[2]

A reduction in amniotic fluid volume following PROM, known as oligohydramnios, may trigger a spectrum of fetal complications, such as pulmonary hypoplasia, respiratory distress syndrome (RDS), intrauterine growth restriction, stillbirth, neonatal infection, amniotic fluid stained with

meconium, and low Apgar scores.[3] Even though the etiological processes underlying PPROM are varied and often unclear, a number of risk factors are clearly defined. They include maternal ethnicity, a history of poor pregnancy outcome, excessive overdistension of the uterus, cigarette smoking, low levels of pre-pregnancy body mass index (BMI), reproductive tract or urinary infection, emotional disruption before pregnancy (depression or stress), inappropriate nutrition, assisted reproduction, and oral infections (periodontal disease).[4]

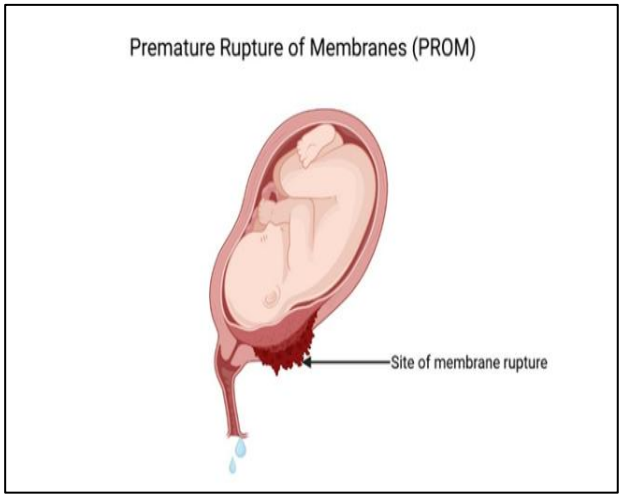

Figure 1: Schematic representation of PROM.

The pathogenesis of PROM is multifactorial and involves several biological and mechanical mechanisms. Maternal infection, particularly chorioamnionitis, is an important complication, while fetal and neonatal consequences include prematurity, respiratory complications, neonatal infection, umbilical cord compression, and other adverse perinatal outcomes.[5,6] Clinical outcomes are influenced by gestational age at membrane rupture, the duration of the latency period, and the severity of oligohydramnios.[7]

LITERATURE SEARCH STRATEGY

A narrative literature search was conducted using PubMed, Scopus, and Google Scholar to identify relevant literature published between January 2000 and 2026. Search terms included "prelabor rupture of membranes," "premature rupture of membranes," "PROM," "preterm prelabor rupture of membranes," "PPROM," "risk factors," "diagnosis," "management," "antibiotics," "antenatal corticosteroids," "tocolysis," "magnesium sulfate," and "prevention," individually and in appropriate combinations. Clinical guidelines, randomized controlled trials, observational studies, systematic reviews, meta-analyses, and relevant narrative reviews published in English were considered. Priority was given to clinical guidelines, systematic reviews, meta-analyses, and studies directly addressing the diagnosis, management, or prevention of PROM and PPROM. Publications not directly relevant to the objectives of the review and duplicate records were excluded.

EPIDEMIOLOGY

Globally, PROM occurs in approximately 8-10% of pregnancies, whereas PPROM is reported in approximately 2-3% of all pregnancies. PPROM contributes substantially to spontaneous preterm birth and is associated with increased neonatal morbidity and mortality. The disease burden may be greater in low- and middle-income countries, where limitations in access to timely antenatal and obstetric care may adversely influence maternal and neonatal outcomes.

PATHOPHYSIOLOGY

The pathophysiology of PROM involves weakening of the fetal membranes due to biochemical and mechanical processes. Infection-induced inflammation, oxidative stress, and activation of matrix metalloproteinases contribute to breakdown of the extracellular matrix within the amniotic membranes.

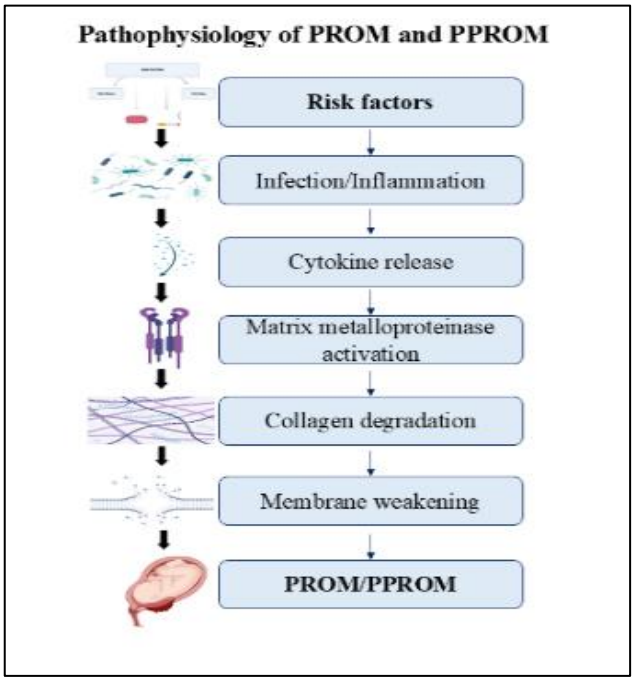

Figure 2: Proposed pathophysiological mechanisms involved in PROM and PPROM.

RISK FACTORS

Identifying risk factors early is important for effective prevention and management. Addressing these elements in advance plays a key role in prevention. Common risk factors for PROM include a history of miscarriage, previous preterm birth, previous PROM, tobacco use, polyhydramnios (excessive amniotic fluid), urinary or sexually transmitted infection, prolonged use of corticosteroids, multiple pregnancies, vaginal hemorrhage during pregnancy, abdominal trauma, connective tissue defects such as systemic lupus erythematosus or Ehlers Danlos syndrome, an extreme body mass index (low and

high), anemia, and socioeconomic hardship.[8,9] The latency period generally becomes shorter as gestational age increases. For example, when PROM occurs at term, most women typically enter labor on their own within 24 to 72 hours. Approximately 70% of them begin labor within the first 24 hours, 85% by 48 hours, and more than 90% within 72 hours.

The risk of infection increases with the duration of membrane rupture. The incidence of chorioamnionitis is estimated between 6-10 percent in full-term PROM, and the probability increases with rupture period longer than 24 hours. An extended membrane rupture period also predisposes the onset of postpartum complications, including endometritis.[10,11] Moreover, in full-term pregnancies, when rupture persists 24-48 hrs or more, the incidence of neonatal infections increases approximately 2.25 times.[12]

DIAGNOSIS

PROM should be considered in women who present with a sudden gush or persistent leakage of clear vaginal fluid. The first step in diagnosis is usually a sterile speculum examination. There are three clinical indicators that are usually measured: Evidence of a fluid in the vaginal canal or a visible discharge coming out of the cervical opening. A positive finding in the nitrazine test, with pH-sensitive paper turning blue due to the presence of alkaline amniotic fluid. A positive fern test, where the dried fluid, when observed using a microscope, has a fern-like crystalline structure.[8,13]

Although such classical diagnostic methods are quite common, their effectiveness decreases with time and may depend on the amount of blood, semen, antiseptics, or infections. Nitrazine test is highly sensitive with a range of 90%-97% whereas its specificity is low with a range of 16%-70%. On the other hand, the fern test shows variable sensitivity (51-98%) and specificity (70-88%).[8,10,14]

In order to increase the accuracy of diagnosis, a number of biochemical markers have been introduced. Several biochemical markers, such as AFP, beta-hCG, IGFBP-1, and PAMG-1, can also be detected in vaginal secretions. These biomarkers demonstrate high sensitivity and excellent negative predictive value.

Rapid diagnostic assays that can be used are AmniSure (PAMG-1), Actim PROM (IGFBP-1), and AmnioQuick Duo. These tests are rapid and help confirm membrane rupture. PAMG-1 and IGFBP-1 based tests are considered among the most accurate methods even in cases of vaginal bleeding or infection. Furthermore, an ultrasound assessment of the amniotic fluid index and the presence of intraamniotic debris (sludge) can be important in helping to diagnose PROM and the risks of the fetus.[14]

PROM diagnosis is especially difficult in cases where fluid leakage is not prominent. In such situations, rapid diagnostic assays are particularly useful. Both AmniSure and Actim PROM have a high sensitivity (95%-97%) and negative predictive value. Actim PROM is usually thepreferred one when vaginal bleeding is evident because AmniSure may produce false positive results in such situations.[15,16]

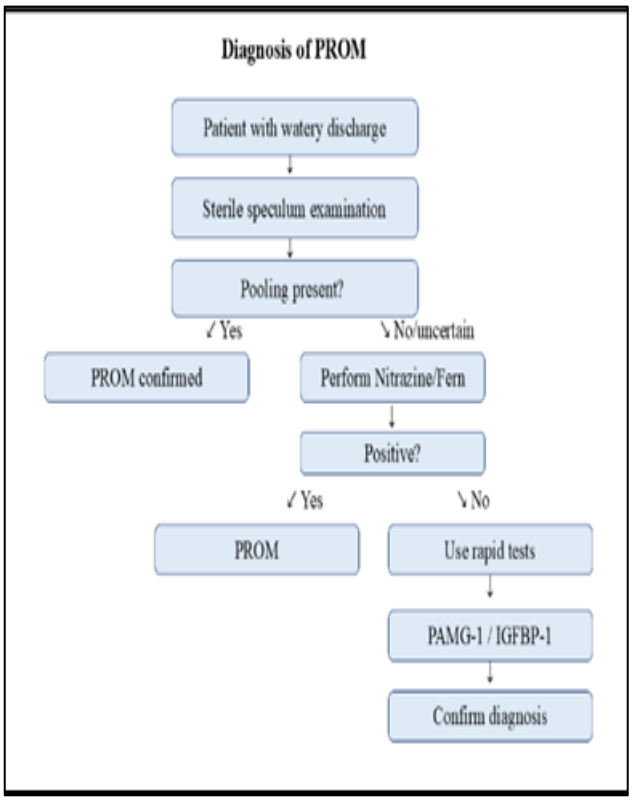

Figure 3: Diagnostic approach to suspected PROM.

Table 1: Diagnostic tests for PROM and their diagnostic characteristics.

Tests	Principle	Sensitivity	Specificity	Limitations
Sterile speculum exam	Visualization of fluid leakage	Variable	Variable	Operator dependent
Nitrazine test	pH detection (alkaline fluid)	90-97%	16-70%	False positives with blood/semen
Fern test	Crystallization pattern	51-98%	70-88%	Affected by contamination
PAMG-1 (AmniSure)	Placental protein detection	~95-99%	High	Costly
IGFBP-1 (Actim PROM)	Protein detection	~95-97%	High	Less available
Ultrasound AFI	Reduced fluid volume	Supportive	Supportive	Not confirmatory

MANAGEMENT

The management of PROM is determined primarily by gestational age, maternal and fetal condition, and the presence of infection or labor. Once PROM is confirmed, the decision between expectant management and delivery should be based on a comprehensive assessment of the risks associated with infection and prematurity. In term pregnancies, labor often begins spontaneously, while induction of labor may be considered when clinically appropriate. In PPROM, management should be individualized according to gestational age and maternal and fetal clinical status.[17]

In women with PPROM between 34 and 36+6 weeks of gestation, the decision between expectant management and delivery should be individualized according to maternal and fetal status and applicable clinical guidelines. Management includes close monitoring for signs of infection and fetal compromise, with Group B Streptococcus (GBS) screening and intrapartum antibiotic prophylaxis when indicated. Antenatal corticosteroids may be considered in eligible women at risk of preterm delivery according to current recommendations. Magnesium sulfate is used for fetal neuroprotection when early preterm delivery is anticipated, generally before 32 weeks of gestation. Routine tocolysis is not recommended in late-preterm PPROM, and any use of tocolytic therapy should be based on careful clinical assessment.[18]

INDUCTION OF LABOUR

For women presenting with PROM at or beyond 37 weeks of gestation, induction of labour is generally recommended to reduce maternal and neonatal complications. Oxytocin remains the intravenous agent most commonly used to induce uterine contractions. Misoprostol is another useful agent, and it can be given in an oral, vaginal, or sublingual form. Delivery through the vagina is normally preferred since it is related to less postpartum infection.

Labour should be induced in women with PROM occurring between 34 and 36+6 weeks of gestation when complications such as chorioamnionitis, fetal distress, or umbilical cord prolapse are suspected. The effectiveness of both techniques was proven by a randomized clinical trial that compared intravenous oxytocin with sublingual misoprostol of 25µg (introduced every four hours). Misoprostol was also linked to the second stage of labour being shorter and better neonatal outcomes, despite having more maternal side effects.[17]

ANTIBIOTICS

Preterm birth poses a serious risk to infant health, and undiagnosed infection is known to be one of the most important factors in the development of PPROM.[18] Previous studies have indicated that antibiotic treatment of women with PPROM can reduce infectious complications and prolong the duration of pregnancy.

Antibiotic use in PPROM should be considered according to its clinical indication. Latency antibiotics are administered during expectant management of PPROM to prolong pregnancy and reduce maternal and neonatal infectious complications. Recommended regimens vary among clinical guidelines and may include erythromycin- or penicillin-based therapy, depending on local recommendations and antimicrobial policies.[19] Evidence supporting latency antibiotic therapy is also derived from major clinical trials, including the ORACLE I trial and the National Institute of Child Health and Human Development Maternal-Fetal Medicine Units Network trial.[20,21]

Group B *Streptococcus* (GBS) intrapartum antibiotic prophylaxis should be considered separately from latency antibiotic therapy and administered when indicated according to maternal GBS status and applicable clinical guidelines. In contrast, when intra-amniotic infection (chorioamnionitis) is suspected or confirmed, therapeutic broad-spectrum antibiotics and appropriate obstetric management, including delivery when indicated, are required rather than continuation of latency antibiotic therapy alone.[1]

Evidence indicates that prophylactic antibiotic therapy after PPROM is associated with fewer infectious complications, a longer latency period, and improved neonatal outcomes.[18,20,21] A network meta-analysis evaluating different prophylactic antibiotic regimens, including erythromycin, clindamycin, gentamicin, penicillins, co-amoxiclav, and cephalosporins, reported differences in their effectiveness in reducing infectious complications.[19]

CORTICOSTEROIDS

Antenatal corticosteroids play an important role in the management of women at risk of preterm delivery, including those with PPROM. Their administration promotes fetal maturation and reduces the risk of neonatal complications, including RDS, intraventricular hemorrhage (IVH), and necrotizing enterocolitis (NEC).[22] A single course of antenatal corticosteroids is generally recommended for eligible women at risk of preterm delivery within 7 days. In selected women at risk of late-preterm delivery, antenatal betamethasone may also be considered to reduce the risk of neonatal respiratory complications.[23]

Common corticosteroid protocols include: Betamethasone had 12 mg intramuscularly, administered as two doses 24 hours apart. Dexamethasone had 6 mg intramuscularly, administered as four doses at 12-hour intervals.[24]

The decision to administer antenatal corticosteroids should be based on gestational age, the likelihood of preterm delivery, previous corticosteroid exposure, and the maternal and fetal clinical condition. Routine serial courses of antenatal corticosteroids are not recommended.

A repeat course may be considered in selected women at continued risk of preterm delivery according to applicable clinical guidelines; however, the role of repeat or rescue courses in PPROM remains uncertain.[25]

TOCOLYTICS

Routine tocolysis is generally not recommended in women with PPROM because the potential benefit of prolonging pregnancy must be balanced against the risk of maternal and fetal infection. Short-term tocolysis may be considered in carefully selected patients when a brief delay in delivery is required to complete a course of antenatal corticosteroids or facilitate maternal transfer to an appropriate healthcare facility, provided there is no evidence of intra-amniotic infection, placental abruption, or fetal compromise.[26]

Several classes of tocolytic agents, including calcium channel blockers, beta-adrenergic agonists, and nonsteroidal anti-inflammatory drugs (NSAIDs), have been used to delay preterm delivery. However, evidence supporting their routine use specifically in PPROM remains limited, and treatment should therefore be individualized according to gestational age, maternal and fetal clinical status, and applicable clinical guidelines.[26,27]

MAGNESIUM SULFATE

Magnesium sulfate is primarily administered for fetal neuroprotection in pregnancies complicated by PROM before 32 weeks of gestation.[8] The most common brain injury pattern associated with cerebral palsy in premature infants involves damage to the periventricular white matter.[28]

The neuroprotective effects of magnesium sulfate are believed to result from inhibition of N-methyl-D-aspartate (NMDA) receptors located on oligodendrocytes, cells particularly vulnerable to oxygen deprivation. The influx of Ca2+ into the cells is mediated by NMDA receptors, which are negatively regulated by magnesium sulfate ($MgSO_4$), which in turn inhibits Ca2+ mediated cytotoxicity during hypoxic or ischemic injury.[29]

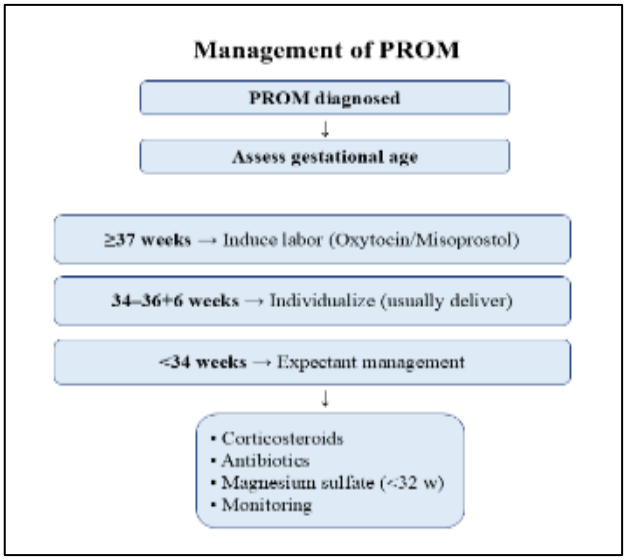

Figure 4: Gestational-age-based management approach for PROM and PPROM.

Table 2: Pharmacological management strategies for PROM and PPROM.

Drug class	Indication	Common regimen	Purpose
Corticosteroids	Eligible women at risk of preterm delivery	Betamethasone 12 mg IM×2 doses	Promote fetal lung maturation
Antibiotics	PPROM	Erythromycin ± ampicillin/ amoxicillin	Prevent infection, prolong latency
Magnesium sulfate	< 32 weeks	IV loading + maintenance	Neuroprotection
Tocolytics	Selected cases	Short-term therapy in carefully selected cases	Delay labor
Oxytocin	Term PROM	IV infusion	Induction of labor
Misoprostol	Term PROM	25 µg SL/vaginal	Cervical ripening

PREVENTIVE STRATEGIES FOR PROM/PPROM

Prevention of PROM and PPROM involves addressing potentially modifiable risk factors, including screening and appropriate treatment of genitourinary infections, smoking cessation, optimization of maternal nutrition, and assessment of cervical length in women at increased risk of preterm birth. Progesterone therapy may be considered in selected women at increased risk of spontaneous preterm birth according to current clinical recommendations. Several nutritional interventions have also been investigated for their potential role in preventing PROM and PPROM. Vitamin C supplementation has been evaluated because of its proposed role in collagen synthesis and maintenance of fetal membrane integrity. In a randomized trial involving 120 pregnant women, supplementation with 100 mg of vitamin C daily from 20 weeks of gestation was associated with a lower occurrence of PROM, with PROM reported in 7.7% of women receiving vitamin C compared with 24.5% in the control group.[30] A subsequent systematic review and meta-analysis also evaluated the potential role of vitamin C supplementation in reducing the occurrence of PROM and PPROM.[31] However, further high-quality evidence is

required before vitamin C supplementation can be considered a routine preventive strategy specifically for PROM or PPROM.

CONCLUSION

PROM, particularly PPROM, is an important obstetric complication associated with significant maternal and neonatal risks, emphasizing the need for early recognition and appropriate clinical management. Clinical evaluation, supported by appropriate diagnostic testing, plays a key role in establishing an accurate diagnosis. Management should be individualized according to gestational age and maternal and fetal clinical status, with appropriate use of antibiotics, antenatal corticosteroids, magnesium sulfate for fetal neuroprotection, and other interventions when clinically indicated. Preventive measures targeting modifiable risk factors may further contribute to reducing the burden of PROM and PPROM. Overall, an evidence-based and multidisciplinary approach is essential for optimizing maternal and neonatal outcomes.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Prelabor Rupture of Membranes: ACOG Practice Bulletin, Number 217. Obstet Gynecol. 2020;135(3):e80-97.
2. Tiruye G, Shiferaw K, Tura AK, Debella A, Musa A. Prevalence of premature rupture of membrane and its associated factors among pregnant women in Ethiopia: A systematic review and meta-analysis. SAGE Open Med. 2021;9:20503121211053912.
3. Idrisa A, Pius S, Bukar M. Maternal and neonatal outcomes in premature rupture of membranes at University of Maiduguri Teaching Hospital, Maiduguri, North-Eastern Nigeria. Trop J Obstet Gynaecol. 2019;36(1):15.
4. Addisu D, Melkie A, Biru S. Prevalence of Preterm Premature Rupture of Membrane and Its Associated Factors among Pregnant Women Admitted in Debre Tabor General Hospital, North West Ethiopia: Institutional-Based Cross-Sectional Study. Obstet Gynecol Int. 2020;2020:4034680.
5. Dars S, Malik S, Samreen I, Kazi RA. Maternal morbidity and perinatal outcome in preterm premature rupture of membranes before 37 weeks gestation. Pak J Med Sci. 2014;30(3):626-9.
6. Mercer BM. Preterm premature rupture of the membranes. Obstet Gynecol. 2003;101(1):178-93.
7. Sharma M, Agrwal M, Goudappagoudra H. Maternal and fetal outcome in preterm premature rupture of membrane. Int J Reproduct Contracept Obstet Gynecol. 2021;10(11):4178-82.
8. Garg A, Jaiswal A. Evaluation and Management of Premature Rupture of Membranes: A Review Article. Cureus. 2023;15(3):e36615.
9. Choudhary M, Rathore SB, Chowdhary J, Garg S. Pre and post conception risk factors in PROM. Int J Res Med Sci. 2015;3(10):2594-8.
10. Caughey AB, Robinson JN, Norwitz ER. Contemporary diagnosis and management of preterm premature rupture of membranes. Rev Obstet Gynecol. 2008;1(1):11-22.
11. Seaward PG, Hannah ME, Myhr TL, Farine D, Ohlsson A, Wang EE, et al. International Multicentre Term Prelabor Rupture of Membranes Study: evaluation of predictors of clinical chorioamnionitis and postpartum fever in patients with prelabor rupture of membranes at term. Am J Obstet Gynecol. 1997;177(5):1024-9.
12. Ghafoor S. Current and Emerging Strategies for Prediction and Diagnosis of Prelabour Rupture of the Membranes: A Narrative Review. Malays J Med Sci. 2021;28(3):5-17.
13. Abdelazim IA, Makhlouf HH. Placental alpha microglobulin-1 (AmniSure(®) test) for detection of premature rupture of fetal membranes. Arch Gynecol Obstet. 2012;285(4):985-9.
14. Tchirikov M, Schlabritz-Loutsevitch N, Maher J, Buchmann J, Naberezhnev Y, Winarno AS, et al. Mid-trimester preterm premature rupture of membranes (PPROM): etiology, diagnosis, classification, international recommendations of treatment options and outcome. J Perinat Med. 2018;46(5):465-88.
15. Palacio M, Kühnert M, Berger R, Larios CL, Marcellin L. Meta-analysis of studies on biochemical marker tests for the diagnosis of premature rupture of membranes: comparison of performance indexes. BMC Pregnancy Childbirth. 2014;14:183.
16. Elçi E, Güneş Elçi G, Sayan S. Comparison of the accuracy and reliability of the AmniSure, AMNIOQUICK, and AL-SENSE tests for early diagnosis of premature rupture of membranes. Int J Gynaecol Obstet. 2020;149(1):93-7.
17. Pourali L, Saghafi N, Eslami Hasan Abadi S, Tara F, Vatanchi AM, Motamedi E. Induction of labour in term premature rupture of membranes; oxytocin versus sublingual misoprostol; a randomised clinical trial. J Obstet Gynaecol. 2018;38(2):167-71.
18. Kenyon S, Boulvain M, Neilson JP. Antibiotics for preterm rupture of membranes. Cochrane Database Syst Rev. 2010;(8):CD001058.
19. Chatzakis C, Papatheodorou S, Sarafidis K, Dinas K, Makrydimas G, Sotiriadis A. Effect on perinatal outcome of prophylactic antibiotics in preterm prelabor rupture of membranes: network meta-analysis of randomized controlled trials. Ultrasound Obstet Gynecol. 2020;55(1):20-31.
20. Kenyon SL, Taylor DJ, Tarnow-Mordi W, ORACLE Collaborative Group. Broad-spectrum antibiotics for preterm, prelabour rupture of fetal membranes: the ORACLE I randomised trial. ORACLE Collaborative Group. Lancet. 2001;357(9261):979-88.
21. Mercer BM, Miodovnik M, Thurnau GR, Goldenberg RL, Das AF, Ramsey RD, et al. Antibiotic therapy for reduction of infant morbidity after preterm premature

rupture of the membranes. A randomized controlled trial. National Institute of Child Health and Human Development Maternal-Fetal Medicine Units Network. JAMA. 1997;278(12):989-95.
22. Roberts D, Brown J, Medley N, Dalziel SR. Antenatal corticosteroids for accelerating fetal lung maturation for women at risk of preterm birth. Cochrane Database Syst Rev. 2017;3(3):CD004454.
23. Gyamfi-Bannerman C, Thom EA, Blackwell SC, Tita ATN, Reddy UM, Saade GR, et al. Antenatal Betamethasone for Women at Risk for Late Preterm Delivery. N Engl J Med. 2016;374(14):1311-20.
24. Medina TM, Hill DA. Preterm premature rupture of membranes: diagnosis and management. Am Fam Physician. 2006;73(4):659-64.
25. Vidaeff AC, Doyle NM, Gilstrap LC. Antenatal corticosteroids for fetal maturation in women at risk for preterm delivery. Clin Perinatol. 2003;30(4):825-40.
26. Ubom AE, Vatish M, Barnea ER, FIGO Childbirth and Postpartum Hemorrhage Committee. FIGO good practice recommendations for preterm labor and preterm prelabor rupture of membranes: Prep-for-Labor triage to minimize risks and maximize favorable outcomes. Int J Gynaecol Obstet. 2023;163(2):40-50.
27. Berkman ND, Thorp JM, Lohr KN, Carey TS, Hartmann KE, Gavin NI, et al. Tocolytic treatment for the management of preterm labor: a review of the evidence. Am J Obstet Gynecol. 2003;188(6):1648–59.
28. Conde-Agudelo A, Romero R. Antenatal magnesium sulfate for the prevention of cerebral palsy in preterm infants less than 34 weeks' gestation: a systematic review and metaanalysis. Am J Obstet Gynecol. 2009;200(6):595-609.
29. Gupta N, Jahan U, Seep S, Singh CP, Shukla S. Role of magnesium sulphate as a neuroprotective agent on neonatal outcome in preterm deliveries. Int J Reproduct Contracept Obstet Gynecol. 2023;12(3):658-64.
30. Casanueva E, Ripoll C, Tolentino M, Morales RM, Pfeffer F, Vilchis P, et al. Vitamin C supplementation to prevent premature rupture of the chorioamniotic membranes: a randomized trial. Am J Clin Nutr. 2005;81(4):859-63.
31. Nath B, Gaikwad H, Roy H, Paul S, Kanti V. Role of Vitamin C Supplementation in the Prevention of Premature Rupture of Membranes (PROM) and Preterm PROM: A Systematic Review and Meta-Analysis. Cureus. 2024;16(6):e62445.

Cite this article as: Puttegowda SKB, Ramakrishna CS, Chandrashekara SH, Sasavihalli VG, Nagarajappa RB. Premature rupture of membranes: current concepts in diagnosis, management and prevention. Int J Reprod Contracept Obstet Gynecol 2026;15:4236-42.