

ISSN - 2347-5536 Case Report

A CASE STUDY ON 3 WEEKS PREMATURE RUPTURE OF MEMBRANES CAUSED BY OROPHARYNGEAL MICROBIOTA

AREEFA SM ALKASSEH1*, SHAIMAA JAMAL ALKHATIB²

¹Department of Midwifery, Faculty of Nursing College, Islamic University of Gaza, Gaza, Palestine, ²Department of Nursing, Faculty of Nursing College, Islamic University of Gaza, Gaza, Palestine. Email: abahri@iugaza.edu.ps

Received: 21 January 2021, Revised and Accepted: 19 February 2021

ABSTRACT

Premature rupture of membrane (PROM) is produced when amniotic membranes tear before labor onset and is recorded in around 8% of full-term gestations. Preterm PROMs (PPROMs) take place before the 37th week of gestation, with an incidence of 2–4% of pregnancies, and it is associated with higher maternal and perinatal morbidity and mortality, mainly related to infectious processes and prematurity. Among maternal complications, which include postpartum infection, premature placental detachment, and maternal sepsis, we highlight clinical chorioamnionitis for its incidence and severity. Of decreasing frequency, perinatal complications include respiratory distress, neonatal sepsis, intraventricular hemorrhage, necrotizing enterocolitis, and neurological lesions. Full-term PROM frequently has a physiological cause and is a consequence of uterine contractions; however, PPROM usually has a multifactorial etiology that is often unknown, although the most frequently reported cause is an infection, observed in up to 60% of cases. Therefore, the etiology of PPROM, although probably infectious, remains unknown in most cases. The obstetric approach varies as a function of gestational age, actively inducing the pregnancy in full-term PROM but performing an overall evaluation of maternal-fetal status in PPROM. In the latter situation, an assessment is made of the relative risks and benefits of a wait-and-see attitude versus pregnancy induction, considering signs of infection and/or prematurity, and ordering antibiotic treatment when PPROM is diagnosed. Multiple combinations of antimicrobial drugs have been proposed and better perinatal and maternal outcomes have been reported for the prophylactic administration of some new combinations. This study describes a case of PPROM caused by urinary tract infection.

Keywords: Maternal, Membrane, Premature, Rupture.

© 2021 The Authors. Published by Innovare Academic Sciences Pvt Ltd. This is an open access article under the CC BY license (http://creativecommons. org/licenses/by/4.0/) DOI: http://dx.doi.org/10.22159/ijhs.2021v9i2.40854. Journal homepage: https://innovareacademics.in/journals/index.php/ijhs

INTRODUCTION

Preterm premature rupture of membranes (PPROM) before the 37th week of gestation usually has a multifactorial etiology that is often unknown [1-4]. Although the most frequently reported cause is infection by group B Streptococcus with incidence 4-8% [1-4]. PPROM is the commonest precursor of preterm birth, and can lead to death, neonatal disease, and long-term disability [5-8]. The previous studies showed that, small trials of antibiotics for PPROM suggested some health benefits for the neonate, but the results were unsatisfying [5-8]. Moreover, maternal genitourinary infection during pregnancy might be prevented with appropriate screening and treatment programs before and during early pregnancy that need to be supported by ministry of health agenda [7-8]. This case describes a PPROM caused by urinary tract infection.

CASE REPORT

We report the case of a 32-year-old pregnant woman from khan Younis in her 32nd gestational week with a history of two previous full-term eutocic deliveries, and one C/S delivery due to antepartum hemorrhage (placenta previa).

The reported case arrived at the emergency obstetrics and gynecology department due to the vaginal discharge of clear fluid, with no uterine contractions or bleeding. According to the case clinical records, she had no previous an ultrasonography examination during her pregnancy period. The gestational age was estimated as 32 weeks+5 days by ultrasonic fetal biometry. Mother during her first admission showed conscious and oriented X3. The routine V/S was within normal range, recorded blood pressure was 100/80 mmHg taken from the left arm at supine position, temperature 37c axillary, respiratory rate was 19 b/m with shallow regular, pulse 75 bpm, and intravenous (IV) cannula was fixed at right. There was no sign of inflammation on the IV site. The mother was C/O mild abdominal pain, low back pain, mother advised

to stay at complete bed rest. Examination with a sterile speculum in the emergency department revealed the release of clear amniotic fluid, while tran-svaginal ultrasound showed a closed cervix, abdominal ultrasound showed that a normal amount of amniotic fluid and biometrics indicated 32+0 weeks of gestation. Urine, endocervical, and vaginal-rectal cultures were taken, and complete blood and urine analyses were performed. The cardiotocographic record at admission showed good fetal reactivity and the absence of uterine contractions in that time.

Post-admission, the hospital protocol for preterm premature rupture of membrane (PPROM), was followed, administering antibiotic therapy with erythromycin (250 mg orally) and corticosteroids as dexamethasone (4 mg IM) for lung maturation, tocolytic medication as $MgSo_4$ (4 g/100 ml Nacl/20 min loading dose) these used to stop preterm labor. Although the mother remained clinically stable and afebrile, the labor was induced with IV oxytocin followed by eutocic delivery in gestation week 33+4 after the patient signed her informed consent.

The motive for the induction was suspicion of subclinical chorioamnionitis, based on her increasing leukocytosis, neutrophilia, and C-reactive protein (CRP) levels since admission (leukocytosis of 11030 at admission vs. 14650 at induction, neutrophilia of 83% at admission: vs. 81% at induction, and CRP of 11.27 mg l⁻¹ at admission vs. 37 mg l⁻¹ at induction). The cardiotocography remained normal, with no uterine tenderness. The newborn was female and weighed 1970 g, with Apgar scores at 1 and 5 min of 9 and 10, respectively, and she was admitted to the neonatal intensive care unit (NICU). During her NICU stay, the newborn remained clinically stable, with no need for vasoactive drugs or other advanced support measures, and she received prophylactic antibiotherapy in accordance with the hospital protocol. Cultures were taken at admission and throughout the NICU stay were negative for infections, the newborn was discharged at 13 days of life with a weight of 2180 g, cephalic perimeter of 32 cm and length of 47 cm, and no abnormal clinical findings.

DISCUSSION

PPROM of infectious etiology is an entity with potentially severe maternal-fetal morbidity and mortality and an added risk of prematurity. In the majority of cases, the pathogen derives from urinary tract infection, our experience of epidemiologic changes in the etiology of PPROM means that a more complete diagnostic procedure must be followed, with wider analytic studies, adapting available diagnostic procedures to clinical needs. New culture-independent tools sometimes yield positive results even when vaginal and endocervical cultures are negative [9-12]. These new techniques can help to detect a possible unusual bacterial infection when vaginal and endocervical cultures are negative in cases of chorioamnionitis, preterm birth, or PPROM.

Among multiple antimicrobial combinations proposed for PPROM treatment, those based on erythromycin and/or cephalosporins have been the most widely used. Novel combinations were recently found to be efficacious and were reported to improve perinatal and maternal outcomes in comparison to the classic regimen [13,14]. A meta-analysis, in 2015, also proposed a prophylactic role for bacterial infection in limiting the prolongation of pregnancy in PPROM; however, there is insufficient evidence to support the routine use of antibiotics during pregnancy to prevent infectious adverse effects, and some antibiotherapies may cause additional newborn morbidity [8].

The bacteria identified, here, are habitual colonizing opportunistic pathogens. In the present patient, the aim of this study to protect mother life and survive her pregnancy [14].

In my case, we give the mother medications according the therapeutic protocol as: Antibiotic erythromycin (250 mg orally) as and corticosteroids as dexamethasone (4 mg IM) for lung maturation and tocolytic medication as MgSo4 (4 g/100 ml Nacl /20 min loading dose) these used to stop preterm labor.

I think this protocol give positive results for the mother in my case because: 1. We treat the infection to avoid further complications as abortion

- We give dexamethasone for lung maturation because of premature labor
- 3. We give (Mgso₄) as tocolytic agent to delay premature labor for weeks then the mother deliver C/S AT 35 weeks from gestational age.

CONCLUSION

Preventive antibiotic therapy should consider: Opportunistic infections by urinary tract infection.

CONSENT FORM

A consent form was applied by hospital archive registration department and by senior doctor, nurse, and midwife directors at the same hospital.

CONFLICTS OF AUTHORS

The authors declare that they have no competing interests.

AUTHORS DETAILS

Faculty of Nursing, Islamic University of Gaza, P.O. Box 108, Gaza, Gaza Strip, Palestine.

REFERENCES

- ACOG Committee on Practice Bulletins-Obstetrics. ACOG Practice Bulletin No. 80: Premature rupture of membranes. Clinical management guidelines for obstetrician-gynecologists. Obstet Gynecol 2007;109:1007-19.
- RCOG Green-Top Guideline No. 44. Preterm Prelabour Rupture of Membranes; 2010.
- Romero R, Quintero R, Oyarzun E, Wu YK, Sabo V. Intraamniotic infection and the onset of labor in preterm premature rupture of the membranes. Am J Obstet Gynecol 1988;159:661-6.
- Cobo T, Palacio M, Martínez-Terrón M, Navarro-Sastre A, Bosch J, Filella X, et al. Clinical and inflammatory markers in amniotic fluid as predictors of adverse outcomes in preterm premature rupture of membranes. Am J Obstet Gynecol 2011;205:126.e1-8.
- Dare MR, Middleton P, Crowther CA, Flenady VJ, Varatharaju B. Planned early birth versus expectant management (waiting) for prelabour rupture of membranes at term (37 weeks or more). Cochrane Database Syst Rev 2006;1:CD005302.
- ACOG practice bulletin No. 107: Induction of labor. Obstet Gynecol 2009;114:386-97.
- 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;357:979-88.
- 9. Vytla S, Mendz G, Quinlivan J. Dental bacterial DNA are present in the amniotic cavity of healthy pregnant women at term. Transl Med 2016;6:181.
- Kaakoush N, Quinlivan J, Mendz G. Bacteroides and Hafnia infections associated with chorioamnionitis and preterm birth. J Clin Gynecol Obstet 2014;3:76-9.
- Mendz GL, Petersen RW, Quinlivan JA, Kaakoush NO. Potential involvement of *Campylobacter curvus* in preterm birth. BMJ Case Rep 2014;10:1136.
- Lee J, Romero R, Kim SM, Chaemsaithong P, Yoon BH. A new antibiotic regimen treats and prevents intra-amniotic inflammation/ infection in patients with preterm PROM. J Matern Fetal Neonatal Med 2016;29:2727-37.
- 13. Lee J, Romero R, Kim SM, Chaemsaithong P, Park CW, Park JS, *et al.* A new anti-microbial combination prolongs the latency period, reduces acute histologic chorioamnionitis as well as funisitis, and improves neonatal outcomes in preterm PROM. J Matern Fetal Neonatal Med 2016;29:707-20.
- 14. Available from: https://www.emedicine.medscape.com/article/261137-overview.