

**Early Induction of Labour versus
Delayed Management of Premature
Rupture of Membranes at Term**

Thesis

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Contents

	Page
 Introduction and Aim of the Work	1
 Review of Literature.....	
▪ Amnion.....	5
▪ Chorion	9
▪ Differential diagnosis of PROM	43
▪ Fetal and Neonatal complication of PROM	57
 Patients and Methods	68
 Results	72
 Discussion.....	86
 Summary and Conclusion	95
 References.....	99
 Arabic Summary.....	

List of Tables

Table No.	Table Title	Page No.
	Comparison between management groups as regards parity, smoking and the mean age.	73
	Comparison between management groups as regards mode of delivery.	74
	Comparison between management groups as regards the presence of complications with delivery.	74
	Comparison between management groups as regards the Presence of neonatal admission to NICU	75
	Comparison between parity (within the expectant management group) and the mode of delivery.	75
	Comparison between gravidity and occurrence of chorioamnionitis within expectant management group.	76
	Comparison between parity within the expectant management group and neonatal admission to ICU.	76
	Comparison between management groups as regards the mean interval till delivery (hours till delivery).	77

able o.	Table Title	age o.
	Comparison between management groups as regards the mean TLC and temperature on admission..	77
0	Comparison between management groups as regards the mean TLC and temperature after 48 hours from admission.	78

List of Figures

Figure No.	Figure title	Page No.
1	Comparison between management groups as regards the mean age.	9
2	Comparison between management groups as regards mode of delivery.	10
3	Comparison between management groups as regards presence of complication of delivery.	11
4	Comparison between management groups as regards admission to NICU.	12
5	Comparison between management groups as regards the mean interval till delivery (hours).	13
6	Comparison between management groups as regards the mean temperature and TLC on admission.	14
7	Comparison between management groups as regards the mean temperature and TLC after 48 hours.	15

Introduction

The membranes surrounding the amniotic cavity are composed of the amnion and the chorion, which are closely adherent layers consisting of several cell types, including epithelial cells, mesenchymal cells, and trophoblast cells. Embedded in a collagenous matrix. They retain amniotic fluid. Secrete substances both into the amniotic fluid and toward the uterus, and guard the fetus against infection ascending in the reproductive tract (*Sammuel et al., 1998*)

Premature rupture of membranes is defined as rupture of membranes prior to the onset of labour (*Seward, 1998*).

Premature rupture of membranes affects approximately 10% of pregnant women at term (37 weeks or more of gestation, resulting in an increased risk of maternal and neonatal infection (*Milasinovic, 1998*).

Many authorities believe that PROM at term is just variant regard the onset of labour. Risk factors for premature rupture of the membranes at term include over distention of the uterus due to multiple pregnancy or polyhydramnios. Cigarette smoking altered mechanical properties of the amniotic membranes, frequent digital

examination and infections (*Hannah 1998*).

The main problem with premature rupture of membranes is when it occurs before term and that has its own management and complications.

The main risk to the fetus after premature rupture of membranes at term is ascending infection. The risks to the mother are of uterine infections, via either chorioamnionitis or postpartum endometritis (*Mcparland, 2004*).

Most patients (90%) enter spontaneous labour within 24 hours when they experience premature rupture of membranes after reaching term (*Hodnett, 1997*).

However, if the woman does not start into labour within 24 hours, labour may be delayed up to seven days after premature rupture of membranes at term (*Hannah, 1998*).

The problem with premature rupture of the membranes at term is related to timing and methods of delivery.

PROM at term may be managed expectantly or by planned elective birth, usually by induction of labour (*El Khawad, 2005*).

Some reports have suggested that the risk of maternal and fetal infection increases proportionally with the time

between membranes rupture and birth (*Gafni, 1997*). Whether or not to induce labour may depend on the state of the cervix, Insufficiently ripe cervix is associated with increased length of labour and failed induction requiring caesarean section (*Romero, 1999*). After latent period for 24 hours induction of labour is an accepted standard treatment for most of cases.

There are conflicting conclusions from literature reviews assessing PROM at term. With a longer interval from admission to the onset of labour, there is an increased risk of neonatal intensive care unit admission, caesarean rates and more frequent maternal diarrhea and use of analgesia (*Akyol, 1999*).

Induction of labour reduces rates of chorioamnionitis, endometritis and neonatal infections. However it increases rates of caesarean birth (*Beer, 1999*).

Immediate induction without waiting the latent period carries a high incidence of caesarean section especially when cervix is unfavorable. However immediate induction is considered especially when there is high risk of infectious complications or in presence of additional complications such as preeclampsia. Expectant management is advisable in the current practice when the cervix is unfavorable or in the absence of any other

complications, strict monitoring is needed in such cases.

Aim of the work

The objective of this work is to study two options for management of premature rupture of fetal membranes at term. Comparison will be held between early induction of labour once diagnosis is made and delayed management by observing for up to 48 hours after rupture of membranes.

Comparison will be made by recording fetal outcome, neonatal outcome and maternal morbidity.

AMNION

It is the innermost fetal membrane and is contiguous with amniotic fluid this particular avascular structure occupies a role of incredible importance in human pregnancy. The amnion is the tissue that provides almost all of the tensile strength of the fetal membranes. Therefore, the development of the components of the amnion that protect against its rupture or tearing is vitally important to successful pregnancy outcome (*Jeffrey, 1991*).

Embryology of amnion:

Early in the process of implantation, a space develops between the embryonic cell mass and adjacent trophoblasts. Small cells that line inner surface of trophoblasts have been called amniogenic cells, the precursors of amniotic epithelium. The human amnion is first identifiable about the seventh or eighth day of embryo development. Initially a minute vesicle, the amnion develops into a small sac that covers the dorsal surface of the embryo. As the amnion enlarges, it gradually engulfs the growing embryo, which prolapses into its cavity. Distention of the amniotic sac eventually brings it into contact with the interior surface of the chorion leave. Apposition of the mesoblasts of chorion leave and amnion near the end of the first trimester causes

an obliteration of the extra embryonic coelom. The amnion and chorion leave, although slightly adherent, are never intimately connected and usually can be separated easily, even at term (*Benirschke et al., 2000*).

Anatomy of the amnion:

Amnion is a thin translucent membrane. The fetal surface of which is smooth and glistening.

The amnion extends from the fetal surface of the placenta above, to the internal uterine os below and wraps the umbilical cord. The part of the amnion that covers the inner aspect of the placenta is called the “placental Amnion”, while the remainder is referred to as the “reflected amnion”. A third area that directly overlies the internal os of the cervix and covers an area of only one or two square centimeters is known as the “**dependent amnion**” (*Cunningham et al., 2005*).

Bourne (1982) was unable to find blood vessels or nerves in the amnion at any stage of development and he could not identify distinct lymphatic channels.

Through the amnion, three umbilical vessels can be seen imbedded in Wharton jelly. These are two umbilical arteries and one umbilical vein. The amnion is loosely

attached to Wharton jelly except at the site of insertion of the umbilical cord in the placenta where they are firmly attached.

Histology of the amnion:

The human amnion is composed of five distinct layers. It contains no blood vessels or nerves; the nutrients it requires are supplied by the amniotic fluid (*Samuel et al., 1998*).

The Epithelium is the innermost layer, nearest the fetus, consists of single layer of non-ciliated cuboidal cells.

Amniotic epithelial cells secrete collagen types III and IV and noncollagenous glycoproteins (laminin, nidogen, and fibronectin) that form **the basement membrane**, the next layer of the amnion.

The Compact Layer of connective tissue adjacent to the basement membrane forms the main fibrous skeleton of the amnion. The collagens of the compact layer are secreted by mesenchymal cells in the fibroblast layer. Interstitial collagens (types I and III) predominate and form parallel bundles that maintain the mechanical integrity of the amnion. Collagen types V and VI form filamentous