

شبكة المعلومات الجامعية التوثيق الإلكتروني والميكروفيلو

بسم الله الرحمن الرحيم





MONA MAGHRABY



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جامعة عين شمس التوثيق الإلكتروني والميكروفيلم قسم

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Sonographic myometrial thickness measurement as a predictor of latency period in preterm premature rupture of membranes

A Thesis

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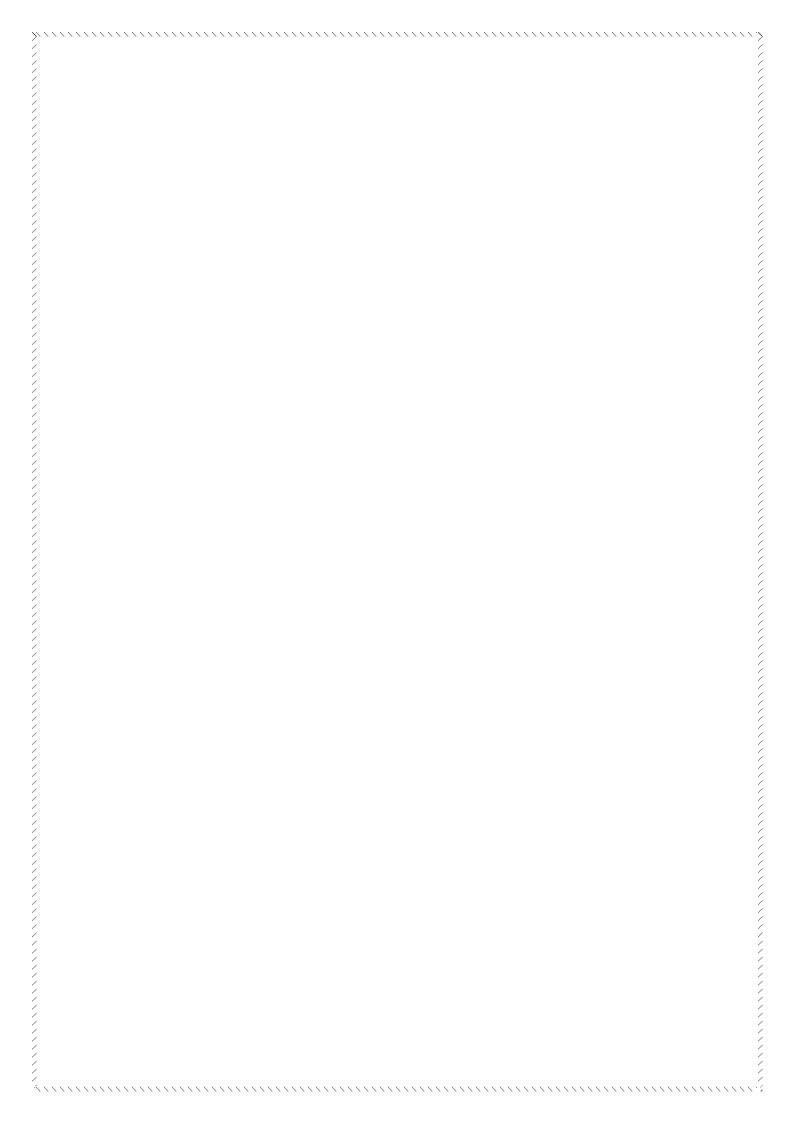


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List of abbreviations

Abbreviation	Meaning
AC	Abdominal circumference
AFP	Alpha fetoprotein
AFI	Amniotic fluid index
AFV	Amniotic fluid volume
BPD	Biparietal diameter
CRP	C reactive protein
ELISA	Enzyme linked immunosorbent assay
FBM	Fetal breathing movement
FFN	Fetal Fibronectin
FHR	Fetal heart rate
FL	Femur length
FTM	Fetal trunk movement
GBS	Group B streptococci
GCC	Genomics Coordination Centre
HC	Head circumference
HCG	Human chorionic gonadotropin
IGFBP-1	Insulin-like growth factor binding protein-1
IL	Interleukin
IUFD	Intrauterine fetal death
IUGR	Intrauterine growth restriction
LOX	Lysyl oxidase
MFM	Maternal fetal medicine
MPH	Massive pulmonary hemorrhage
MT	Myometrial thickness
NICHD	National institute of child health and human development
NO	Nitric oxide
PA	Plasminogen activators
PPROM	Preterm premature rupture of membranes
PROM	Premature rupture of membranes
RDS	Respiratory distress syndrome
ROS	Reactive oxygen species
SEFW	Sonographic estimation of fetal weight
TAT	Thombin-antithrombin
TIMP	Tissue inhibitors of metalloproteinases
TNF	Tumour necrosis factor
US	Ultrasonography
VAF	Vaginal amniotic fluid

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Introduction

Preterm premature rupture of membranes (PPROM) and preterm delivery are very challenging obstetric problems. The main problem for an obstetrician in the management of PPROM is to predict when such a patient is likely to go in labor especially in cases of expectant management. Expectant management gives us the time for fetal lung maturity but with a risk of infection to both mother and the baby (*Gupta and Nagarsenkar*, 2016).

Preterm premature rupture of membranes (PPROM), is defined as spontaneous rupture of membranes before 37 weeks of gestation and the onset of contractions, affects 2–3% of pregnancies in the United States and accounts for approximately one third of all preterm births. More than 90% of gravid women who experience PPROM prior to 34 weeks deliver in less than one week and PPROM is a significant cause of neonatal morbidity and mortality (*Noor et al.*, *2007*).

One of the characteristic features of PPROM is the brief time from rupture of membranes to delivery, with the duration of time increasing with decreasing gestational age. Given that neonatal morbidities are strongly correlated with gestational age at delivery, strategies to extend latency for those pregnancies that may benefit from the delay in delivery are often employed (*Horton et al.*, 2015).

An effect of uterine stretch on the duration of human pregnancy has been suggested by the strong association of multiple gestations with preterm delivery. It was found that 54% of twin pregnancies were delivered preterm compared to only 9.6% who were delivered preterm in a comparison singleton pregnancy group (*Algeri et al.*, 2018).

The term latency refers to the time between membrane rupture and delivery. Latency is an important factor for neonatal survival in these patients. Studies showed that latency period after PPROM is associated with a higher infant mortality rate specially when occur before 30 weeks' gestation, with pulmonary disease being the major cause of death. There is an urgent need for a thorough evaluation of expectant management of PPROM. The risk of chorioamnionitis increases with increasing latency period which worsens the neonatal outcomes (*Aziz et al.*, 2009).

PPROM is associated with several factors that lead fetal morbidity and mortality. Amnionitis, advanced labor and non-reassuring fetal status usually force the clinician to affect delivery despite fetal immaturity. Also, chorioamnionitis is one of the various causes of neonatal brain damage in this period. Having adequate knowledge about latency period after PPROM and conducting appropriate management such as early referring to well-equipped center, clinicians can resolve mater and fetus (*van Teeffelen et al.*, *2014*).

Abdominal and transvaginal ultrasound scans has been used as a valuable method for measurement of myometrial thickness in prediction of maternal conditions (*Bergeron et al.*, 2009) or pregnancy outcome (*Sfakianaki et al.*, 2008).

Our objective is to evaluate the accuracy of sonographic measurement of myometrial thickness in the prediction of latency interval in women with premature rupture of membranes in order to help the clinicians in better decision making.

Aim of the work

This study aims to assess the accuracy of myometrial thickness measurement as an accurate method in prediction of latency period in women with preterm premature rupture of membranes.

Premature Rupture of Membranes (PROM)

Definition:

Premature rupture of membranes (PROM) is defined as rupture of amniotic membranes prior to the onset of labor, regardless of gestational age (*Ehsanipoor*, 2016).

It is unfortunate that "premature" also carries the connotation of preterm pregnancy, so when PROM is noted in a pregnancy prior to 37 completed weeks of gestation, the term preterm premature rupture of membranes (PPROM) is usually applied (*Goya et al., 2013*).

The time interval between rupture of membranes and the onset of labor is termed the latency period; the time interval between rupture of membranes and delivery is termed the interval period (*Test et al.*, 2011).

Romero et al., 2015 recommended using the more specific title Prelabor spontaneous rupture of membranes (SROM) and suggested that the ambiguous term premature to be dropped, since the clinical approach to management is very different when the fetus is immature (Romero et al., 2015).

Incidence:

The incidence of PROM ranges from 4.5 to 7.6% of total deliveries and approximately 70% of cases of PROM occur in pregnancies at term (*TC et al.*, 2014).

Etiology and pathogenesis of PPROM

The etiology of PROM is multifactorial. Maternal enzymes, maturational and mechanical forces, phospholipids

content, collagen disruption, amniotic-cell cytokines induced by fetal signals and bacterial phospholipases and collagenases all play major and inter related role (*Fortner et al.*, 2014).

Support for the concept that reactive oxygen species (ROS) are involved in the pathogenesis of PPROM is derived from several lines of investigation; ROS is an atom or molecule with one unpaired electron in its outer orbit that is capable of existing independently for a brief period of time. Examples of ROS include super oxide (0²), hydrogen peroxide (H²0²), hydroxyl ion (HO), nitric oxide (NO), and hypochlorous acid (HCIO). ROS are generated continuously through leakage from the electron transport chain in the process of cellular respiration within the mitochondria. ROS also play crucial roles in destruction of microorganisms by phagocytic cells (*Tsakiridis et al.*, 2018).

Monocytes, neutrophils, and macrophages release ROS during phagocytosis and microbial killing. Leakage of ROS is capable of damaging collagen and amniotic epithelium in the amniotic membranes. However, in the majority of cases, there is no obvious cause (s). Subclinical intrauterine infections have been proposed as a predisposing factor for PROM (*Caughey et al.*, 2008; Waters and Mercer, 2009).

Risk factors of PPROM

1 -Infection:

A strong body of evidence suggests that infection plays a role in the pathogenesis of PPROM and preterm delivery.