

HERPES SIMPLEX TYPE-2 INFECTION AS A RISK FACTOR OF PREMATURE RUPTURE OF FETAL MEMBRANES

THESIS

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BY

MONA GAMAL EL- DIN ABD EL- RAHMAN
M.B., B. CH.

Under Supervision of

Prof. Dr. MOHAMED YEHIA

Ass. Prof. Of Obstetrics & Gynaecology
Ain Shams University

Prof. Dr. HAZEM ELZENINI

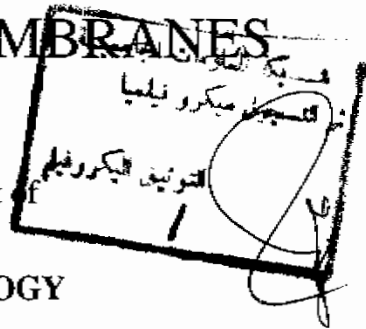
Ass. Prof. Of Obstetrics & Gynaecology
Ain Shams University

Prof. Dr. HADIA H. BASSIM

Ass. Prof. Of Clinical Pathology
Ain Shams University

FACULTY OF MEDICINE
AIN SHAMS UNIVERSITY

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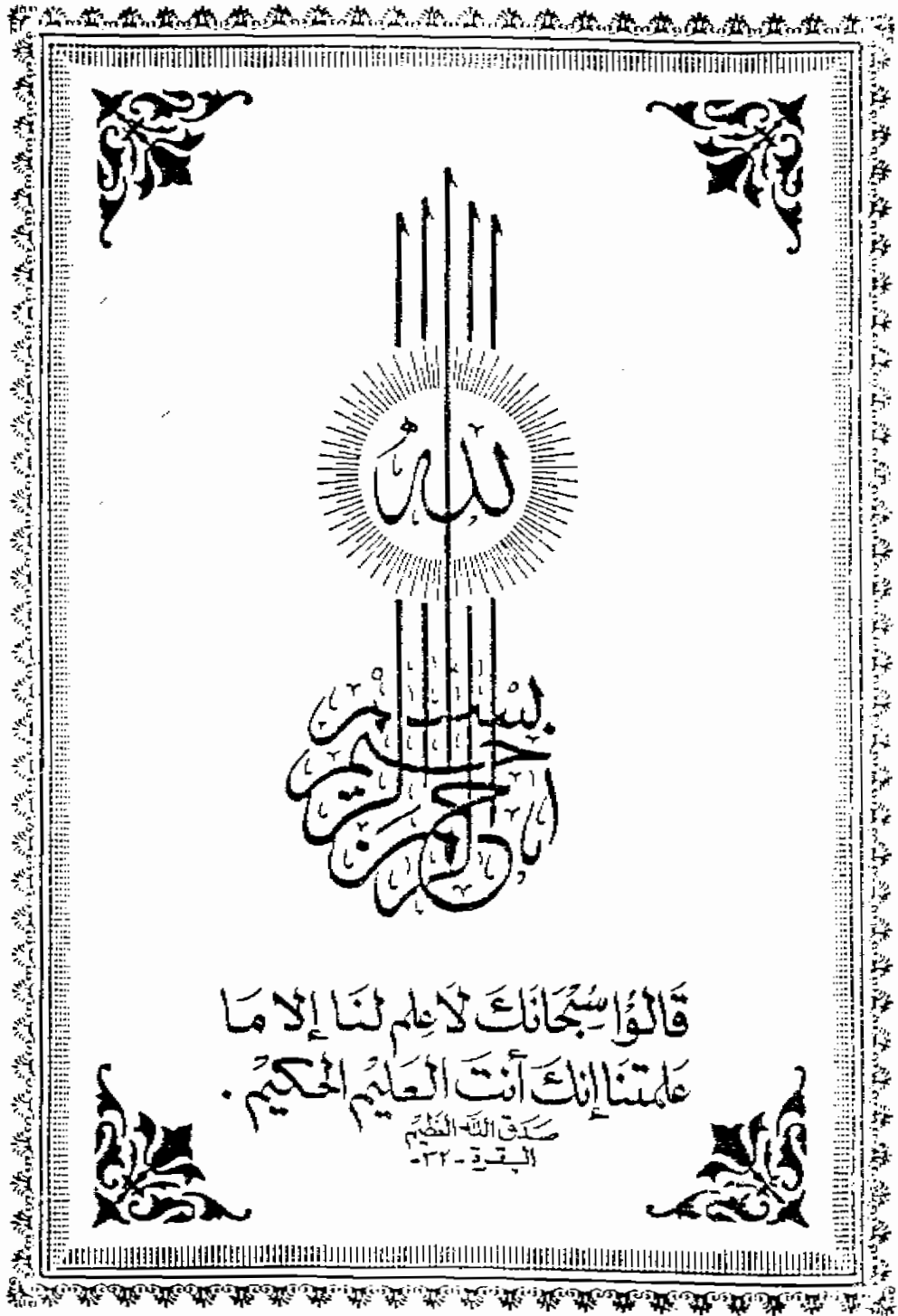
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INTRODUCTION



AIM OF
THE WORK

Introduction

Premature rupture of membranes is a major obstetric complication occurring in approximately 10% of all pregnancies. (*Mead 1980*). It remains a major contributor to perinatal morbidity and mortality as a result of complication of prematurity and sepsis (*Gibbs and Blanco 1982*).

Now it is well established that the standard definition of premature rupture of membranes is the disruption of the amnion and chorion with resultant leakage of amniotic fluid prior to the onset of labor, while the term "Preterm", will refer to pregnancy with gestational age less than 37 weeks of amenorrhea, calculated from the first day of last menstrual period (*Pritchard et al., 1985*).

The exact etiology of PROM is not fully explained (*Naeye et al 1980*) although genital infections constitute a major risk. These infections either by aerobic and anaerobic bacteria, yeast, viruses, parasites or mycoplasma. (*Gibbs and Blanco 1982*).

Herpes simplex type 2 virus was recorded as one of these genital infections which is about three times more common in pregnant women than non pregnant

women and this is because the host defense mechanisms could be modified by the action of progesterone during pregnancy. (*Hanshow 1973*). Also there was increased prematurity rate in infants of women with cytologically detected herpes genitalis, and in infants of women with serologic evidence of primary infection. (*Nahmias and Coworkers. 1971*).

Aim of the work

The aim of the present study is to evaluate the role of Herpes Simplex virus type 2 as a risk factor in cases of premature rupture of fetal membranes.

REVIEW OF LITERATURE

Anatomy of fetal membranes & liquor

- * *Gross appearance of fetal membranes.*
- * *Embryological origin of fetal membranes.*
- * *Microscopic picture of fetal membranes.*

*** Gross appearance of fetal membranes :-**

The fetal membranes consist of an inner thin glistening tough layer called the amnion, and an outer thicker but more friable layer called the chorion.

The amnion is a thin, translucent, avascular, double layered membrane which lies in a direct contact with the inner surface of the chorion, it lines the uterine cavity and surrounds the fetus completely as a membranous sac. The chorion is the specialized fetal envelope, which is provided by complex branching villi, in direct contact with free maternal blood, these arrangements provide for the nutrition of the ovum during the first 8-12 weeks of development. The amnion and chorion, although adherent, are never connected intimately and usually can be separated easily from one another at all periods of pregnancy. (Pritchard *et al.*, 1985).

It is well established that the amnion is not a mere passive lining of the uterine cavity, but it is the main load bearing component of the chorioamniotic membrane, and its ability to resist intra-amniotic pressure throughout pregnancy is due to the collagen-rich connective tissue layers which are found to decrease to a nadir at parturition.

As regards the chorion it forms a good deal of connective tissue thickness

of the placenta on its fetal aspect and is the structure in and through which the major branching umbilical vessels travel on the surface of the placenta. (Pritchard *et al.*, 1985).

The two membranes slide upon each other readily, indeed there is only trivial connective tissue attachment between them with modest collection of fluid occasionally accumulates between them. The two, merely stick together, and are not organically united. This looseness of attachment between amnion and chorion may not only provide some safety to the fetus in the course of its growth but also may permit differential sliding in the event that the chorion ruptures while the amnion remains intact in the course of labor and delivery. (Alger and Pupkin, 1986).

*** Embryological origin of fetal membranes :-**

a- Development of the amnion :-

By the time the human embryo has become implanted, the amniotic cavity has already appeared (Hamilton *et al.*, 1982).

It appears as a small cleft between the ectoderm of the inner cell mass and the trophoblast.

As the amniotic cavity enlarges, a layer of flattened cells called the amnioblast develops from the inner surface of the trophoblast and forms the roof of the amniotic cavity. At this stage, the floor of the amniotic cavity is formed by ectodermal germ layer of the embryonic disc.

The amnioblast lies in close contact with the inner layer of the trophoblast and becomes continuous with the ectodermal germ layer at the margins of the embryonic disc.