تحديد كمية ألفا واحد مضاد التربسين فى السائل الأمنيونى
فى حالات تمزق الغشاء الامنيونى المبكر

رسالة

مقدمة من

الطبيبة / هويدا محمود محمد حسن عبيد

توطئة للحصول على درجة الماجستير

فى

أمراض النساء والتوليد

اشراف

الدكتور / خالد الهضيبى
استاذ مساعد امراض النساء والتوليد
كلية الطب - جامعة عين شمس

الدكتور / عصام عمار
مدرس أمراض النساء والتوليد
كلية الطب - جامعة عين شمس

الدكتور / يحيى عبدالرؤوف
مدرس علم البكتيريا والمناعة
كلية الطب - جامعة عين شمس

كلية الطب
جامعة عين شمس

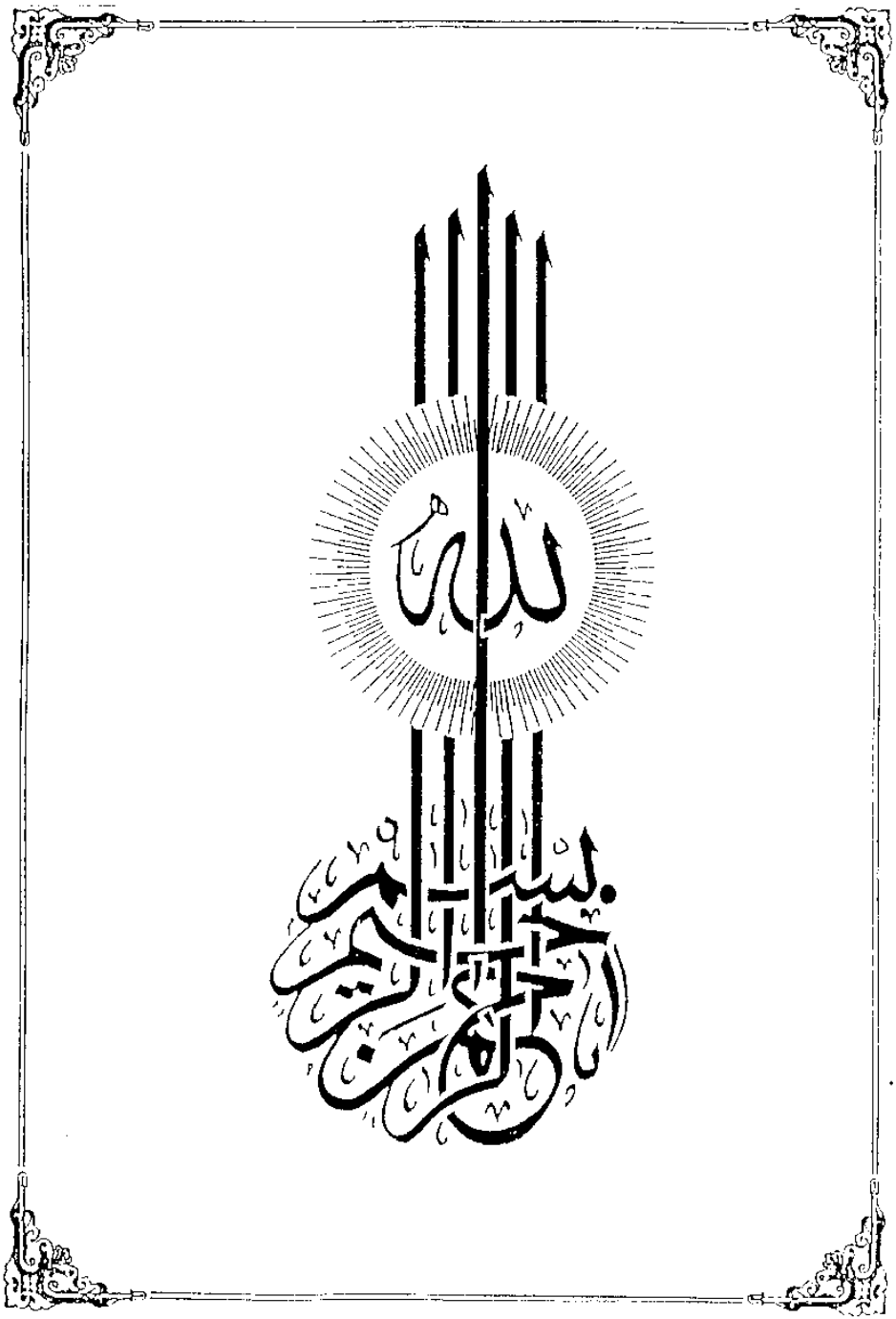
١٩٨٨ م

بسم الله الرحمن الرحيم

"وفوق كل ذي علم عليم"

صدق الله العظيـــــم

سورة يوسف الآيه ٧٦



ABSTRACT

Twenty five cases of premature rupture of membrane (PROM) and 15 cases without PROM are involved in our study.

The concentration of α_1-antitrypsin in amniotic fluid was measured in patients with and without premature rupture of membranes. The concentration of α_1-antitrypsin in amniotic fluid with premature rupture of membranes (21.42±11.34 IU/ml) was significantly lower than in cases without premature rupture (54.28±42.52 IU/ml) ($P < 0.001$). These study suggests that low concentration of α_1-antitrypsin in amniotic fluid is one cause of premature rupture of membranes.

ACKNOWLEDGEMENT

I would like to express my deepest gratitude to **Dr. Khalid El-Hodeiby,** Ass. Prof. of Obstetrics and Gynecology Ain Shams University for his valuable guidance, kind help, constant advice and supervision of this thesis.

I wish also to express my sincere thanks and appreciation to **Dr. Essam Ammar,** Lecturer of Obstetrics and Gynecology. Ain Shams University for his helpful advice, great cooperation and for his reviews of the manuscript and helpful suggestions and comments.

I indebted to **dr. Yehia Abd Al-Raouf,** Lecturer of Microbiology and Immunology, Faculty of Medicine Ain Shams University, for his helpful support, providing accurate unfailing advice and supervision during the practical work of this study.

Lastly, I thank the staff of the antenatal and labor wards, Obstetrics and Gynecology Hospital Ain Shams University for their great cooperation.

DEDICATION

This thesis is dedicated to my father , my son and my husband .
Their continual love and support made this work possible .

CONTENTS

° ° ° ° °

LIST OF TABLES

LIST OF FIGURES

LITERATURE REVIEW

DEVELOPMENT AND ANATOMY

The human amnion develops either by delamination from the cytotrophoblast about the seventh or eight day of development of the normal ovum or else it develops essentially as an extension of the fetal ectoderm.

Initially, a minute vesicle, the amnion develops into a small sac that covers the dorsal surface of the embryo. As the amnion enlarges, the growing embryo is gradually engulfed and prolapses into the amniotic cavity. Distension of the amniotic sac eventually brings it into contact with the interior of the chorion; a position of the mesoblasts of chorion and amnion near the end of the first trimester results in the abliteration of the extraembryonic coelom. The amnion and chorion, although adherent, are never connected intimately and usually can be separated easily, even at term, (Pritchard et al., 1985).

By the time the human embryo has become implanted, the amniotic cavity has already appeared, (Hamilton, et al.,1962). Trophoblastic cells lined with mesoderm constitute the chorion. Amniogenic cells delaminate

from the inner surface of the trophoblast to form the amnion. Ectoderm continuous with that of the embryonal disc grows out to line the amniotic cavity.

By the third trimester, the amnion consists of a single layer of epithelial cells superimposed on a layer of dense connective tissue containing a high concentration of collagen filaments. These two layers together are about 0.05 to 0.11 mm thick (Donforth and Hull, 1958; and Polishuk et al., 1962). Reticular stains demonstrate a heavy reticulum in the fibrous layer, and it is this connective tissue that provides the major strength to the fetal membranes. Loose areolar tissue is very weakly attached to the fibrous layers and makes fingerlike projections into the underline chorion. The chorion, in turn, lies directly apposed to uterine decidua. Since no mitotic activity is demonstrable in the latter half of pregnancy, the amniotic cavity must enlarge by stretching and hypertrophy of already-existing cells. The epithelial cells of the reflected surface of the amnion that line the uterus are cuboidal, while those on the placental surface are columnar. This may be the consequence of constant growth stress, aggrevated by uterine contraction, applied to the reflected surface cells, as compared with the reduced stress generated over

the placental surface, which is splinted by the underlying tissue mass. In normal pregnancy, a well-described system of amnion cell shedding can be observed with the scanning electron microscope (Polland et al., 1976). During this process, the basement membrane is exposed, demonstrating the importance of the basement membrane and its supporting connective tissue in maintaining fetal membrane integrity. Immediately beneath the basement membrane is a compact network of randomly scattered reticular fibrils (Bou-Resli et al., 1981), which become progressively less dense at sites further from the placental bed. The major portion of the connective tissue zone is the fibroblast layer compsoed of collagenous bundles and reticular fibrils in crossing sheets with embedded fibroblasts. Again as the distance progresses from the placenta this arrangement becomes looser, and the sheets are disrupted. There are lesser amounts of both collagen and reticular fibrils, (Alger and Pupkin, 1986).

The chorion itself averages four to six cells in thickness but is extremely variable, ranging from 0.04 to 0.40 mm (Daonforth and Hull, 1958; and Polishuk et al., 1962). In some areas the chorion is healthy and