# ADHESIONS IN OBSTETRICS AND GYNAECOLOGY

Thesis
submitted for partial fulfillment for
The Master Degree

In
OBSTETRICS AND GYNAECOLOGY

Presented by

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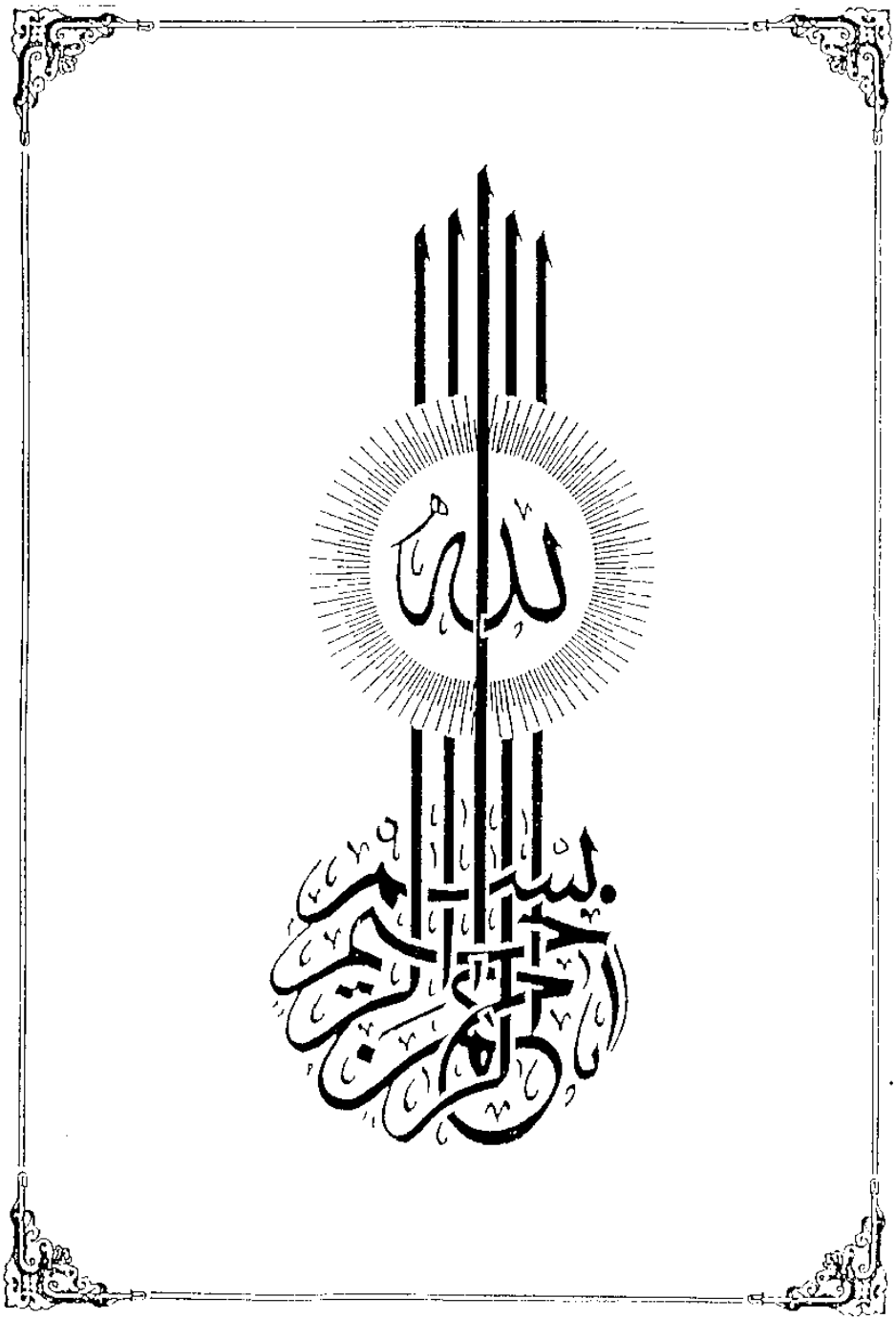
1989

بسم الله الرحمن الرحيم

# وما أوتيتم من العلم إلا قليلاً

سورة الإسراء ( آيه ٨٥)



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## ACKNOWLEDGEMENT

It was indeed an honour to have been supervised by one of the most notable professors of Obstetrics and Gynaecology namely, Professor Dr. Sobhi Khalil Abo-Louz professor of Obstetrics and Gynaecology , Faculty of Medicine, Ain Shams University. His incessant encouragment and fruitful remarks throughout the different phases of this work were of the utmost value to me,along with the precious time he offered to me in the course of this thesis,all this and more leave me greatly indebted.

Indeed words do fail me when I come to express my sincerest appreciation to Dr. Essam Ammar, lecturer of Obstetrics and Gynaecology, Faculty of Medicine, Ain shams University for without his dynamic efforts and ceaseless enthusiasm, this work would have never been completed. How he offered me of his valuable time denying me none of it, day or night; truly his magnanimity over whelmed me through this work.

An immensely gratifying experience it was, to work under the supervision of an outstanding and able scientist namely Dr Samia Ammar, Assistant professor of Pathology, Faculty of Medicine, Ain Shams University, were it not for her untirring efforts and her expanding experience on the field this thesis would have never been completed.

# INTRODUCTION AND AIM OF THE WORK

# INTRODUCTION

One of the characteristics of living tissue is its ability to react to injury as in case of the local response where the adjacent living tissues undergo changes which enable phagocytic cells and circulating plasma to enter the area of damage, this phase is known as acute inflammation and continues so long as tissue damage continues. When the causative agent is removed, the debris of the inflammatory reaction is removed by scavenger cells.

The tissue may then return to normal, but when tissue loss has been sustained, there is a final stage of healing by repair, regeneration or both.

In acutely inflammed tissue in which cellular damage has been relatively slight, the cellular and tissue changes are reversible and necrosis does not occur. The demolition phase results in removal of the excudate and the organ returns to normal, to this process the term resolution is applied. Resolution thus means the complete return to normal of a tissue following acute inflammation. Sometimes removal of the excudate is delayed, then the fibrin becomes invaded by granulation tissue and fibrosis is the result. It often occurs in fibrinous excudate of inflammed serous cavities, in this manner fibrous adhesions are formed.

In case of mesothelial lining of peritoneum and other serous cavities, the denuded areas are healed by metaplasia of the underlying connective tissue cells which take on the form and function of flattened mesothelium. Hence , following abdominal surgery raw

areas are rapidly covered by newly formed mesothelium. Adhesions do not develop unless healing is complicated by inflammation caused by infection or the presence of irritant chemicals such as talc or starch in glove powder likewise sutures can cause inflammation and adhesion formation.

Thus knowledge of the factors that induce adhesions development and of surgical techniques and ancillary modalities to reduce adhesion formation is essential if optimal results are to be obtained by the reconstructive surgeon.

## AIM OF THE WORK

The aim of this study is to write a full review on adhesions both pelvic and abdominal, before and after gynaecological or obstetrics procedures.

In addition, histopathological study of adhesions removed from 20 cases during gynaecological or obstetrics surgery was done.

# REVIEW OF LITERATURE

## Anatomy Of The Pelvic Peritoneum :

According to Romanes (1977), the parietal abdominal preitoneum passes directly into the lesser pelvis over he margins of the superior aperture. It covers the superior surfaces of the pelvic organs and dipping between them producing peritoneal pouches and fossae. Anteriorly and posteriorly the arrangement of this peritoneum is virtually identical in the two sexes, but it differs in the intermediate region because of the presence of the large genital septum and its contained structures in the female.

The postero- superior surface of the lesser pelvis is covered with peritoneum down to the second peice of the sacrum, excpet where the medial limb of the Sigmoid mesocolon is attached. Inferior to this the rectum intervenes between the sacrum and the peritoneum. The peritoneum covers the front and sides of the upper part of the rectum, but leaves it by turining forwards from the anterior surface of its middle third to run upward over the genital septum. Thus the peritoneum forms the recto-uterine pouch between the rectum and the contents of the genital septum. Then the peritoneum in the median plane passes from the recto-uterine pouch on to the superior part of the posterior vaginal wall; then covers the superior surfaces of the cervix and body of the uterus and turns over the fundus to cover the inferior surface of the body of the uterus. On this surface, at the junction of the body and cervix, the peritoneum bends forwards over the superior surface of the bladder, thus forming the uterovesical pouch of peritoneum. It continues forwards on the bladder and passes directly on to the posterior surface of the anterior abdominal wall.

Lateral to the uterus, the peritoneum passes : 1) from the cervix as the recto-uterine fold on the uterosacral ligament; 2) from the body of the uterus as a double layer (superior and inferior) enclosing the connective tissue of the septum (parametrium); the round ligament of the uterus; the ligament of the ovary; and the uterine tubes in its free anterior margin.

The broad ligament of the uterus is thickened laterally where the utero-sacral ligament passes posteriorly and the round ligament of the uterus curves anteriorly. Its superior layer of peritoneum covers the ovary close to the attachment of the ligament to the lateral pelvic wall. The part of the broad ligament lateral to the ovary is the suspensory ligament of the ovary. The part between the ovary and the uterine tube is the mesosalpinx (mesentery of the tube), while the remainder is mesometrium (mesentery of the uterus). Thus the uterus and parametrium lie in a transverse peritoneal fold with a free anterior margin. This allows it to expand upwrads into the abdominal cavity during pregnancy without disturbing its supporting structures which are composed of the connective tissue in the base of the broad ligament. This holds the cervix to the walls of the pelvis and is especially thickened (Transverse ligaments of the cervix) around the uterine arteries as the uteroscaral ligament. Anterior to the broad ligament and lateral to the bladder, the peritoneum passes forwards as the floor of a shallow paravesical fossa on each side. This is limited laterally by the ridge produced by the round ligament curving forwards to the deep inguinal ring , and has the oblitrated umblical artery passing forwards in its floor.

According to Ten teachers (1985), the pelvic peritoneum is reflected from the lateral borders of the uterus to form on either side a double fold of peritoneum known as the broad ligament. This is not a "ligament" but a peritoneal fold and it does not support the uterus. The fallopian tubes run in the upper free edge of the broad ligament as far as the point at which the tube opens into the peritoneal cavity, the part of the broad ligament which is lateral to the opening is called the infundibulo- pelvic fold, and in it the ovarian vessels and nerves pass from the side wall of the pelvis to lie between the two layers of the braod ligament. The portion of the broad ligament which lies above the ovary is known as the mesosalpinx, and between its layers are to be seen any wolffian remnants which may be present. Below the ovary the base of the broad ligament widens out and contains a considerable amount of loose connective tissues, called the parametrium. The ureter is attached to the posterior leaf of the broad ligament at this point. The ovary is attached to the posterior layer of the broad ligament by a short mesentery (mesovarium) through which the ovarian vessels and nerves enter the hilum. The vagina does not have any peritoneal covering infront, behind it is in contact with the rectovaginal pouch for about 2cm, where the vagina is separated from the abdominal cavity only by the peritoneum and thin fascia. The peritoneal cavity can be opened by posterior colpotomy at this point.

## Inflammation:

### Definition

Marchesi(1985) reported that inflammation is the characteristic response of the living tissue to injury. Whenever tissue is injured, there follows at the site of injury a series of events that tend to destroy or limit the spread of the injurious agent. The early events in this so called inflammatory response are mainly vascular and are usually succeeded by repair and healing of the injured tissue.

The agents that injure tissues and therefore evoke the inflammatory response include bacteria and other types of micro-organisms and non living agents such as trauma, heat, cold, radiant, electrical energy and chemicals. Inflammation is described as acute or chronic according to its duration.

Acute inflammation is characterized by three main vascular events (Marchesi, 1985).

I- **Vasodilatation and changes in the blood flow:** the mechanism responsible for the local dilatation of capillaries and venules was examined by Lewis (1927) in a series of experiments, the results of which indicate that injury leads to the local release of a substance responsible for the dilatation of the capillaries and venules e.g. histamine and other susbtances like kinins. According to Schachter (1969), kinins attracted attention because 1) kinin forming enzymes appear to be associated with physiologic vasodilatation and 2) kinins accumulate in inflammatory exudates (Webster, 1968).

## II- Excudation of plasma :

Under physiologic conditions, the endothelium of the capillaries and proximal portion of the venules permits free movement of water and small molecules to and fro across the endothelium but normally restricts the passage of plasma proteins. In inflammation, there is a net movement of fluid into the tissues that has been estimated to be five to seven times greater than that from a normal vessel with similar levels of hydrostatic pressure and plasma proteins. The fluid excudate of inflammation characteristically contains one to six grams of plasma protein per 100 ml (Marchesi 1985).

## III- Emigration of neutrophilic leukocytes:

Pullinger and Florey (1935) said that emigration of neutrophilic leukocytes through the wall of blood vessels into the adjacent tissues represents the main cellular phase of acute inflammation.

Waltr and Israel (1967) reported that the first cells involved are the neutrophil polymorphs and these take two to nine minutes to penetrate the vessel wall, several hours later the monocytes predominate and in tissues these large cells become phagocytic and are called macrophages , their emigration continues after the polymorphs have been stopped. Lymphocytes have not yet been observed to migrate in acute inflammation, some red cells may also escape from the vessels usually following a white cell.