ENDOMETRIOSIS

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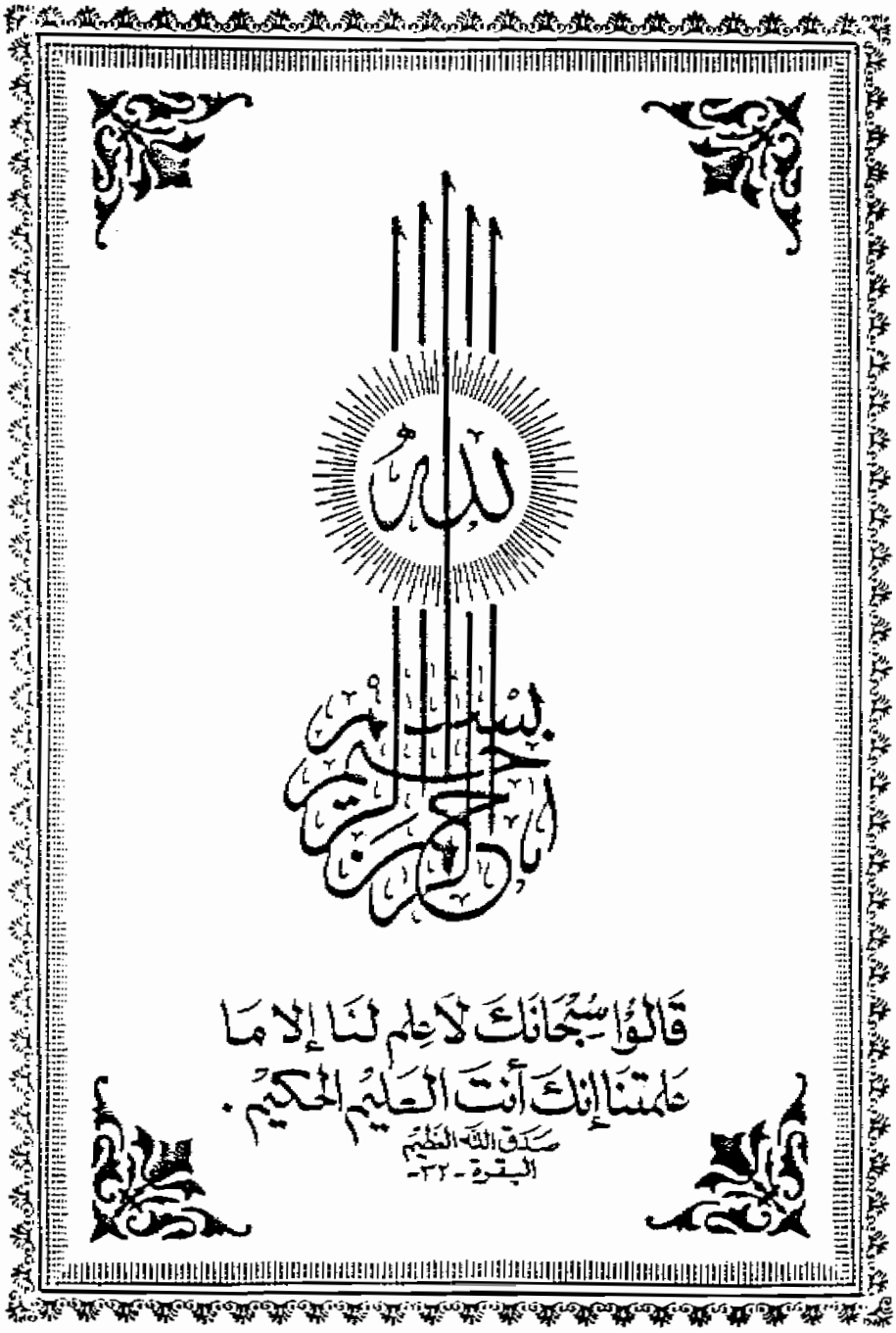
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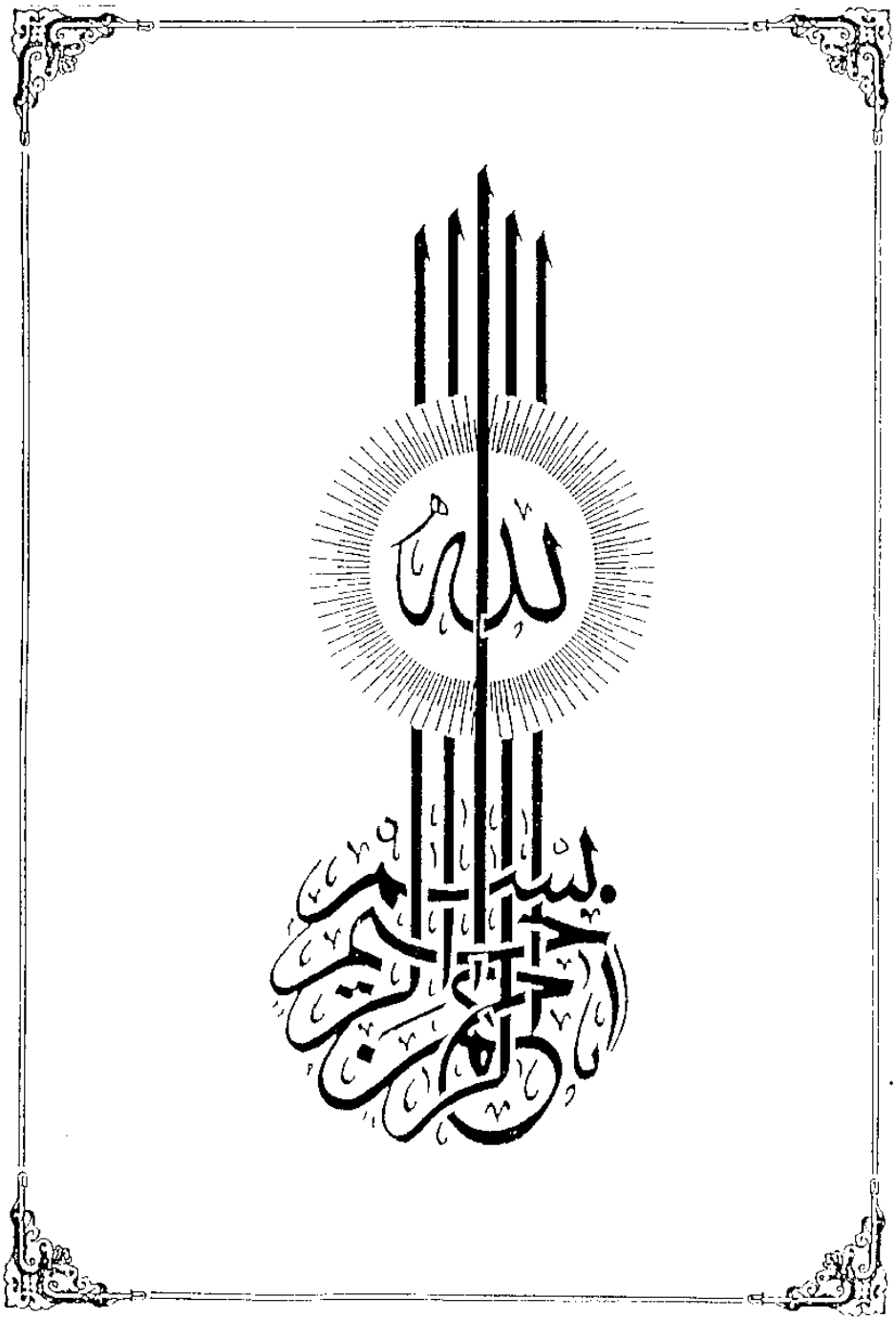

INTRODUCTION

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INTRODUCTION

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During a meeting of the Cambridge Medical Society on 14 April 1882, professor James Paget reported the first record of the disease entity now known as endometriosis (O'Connor, 1987).

This term is used to describe the presence of functioning endometrial glands and stroma outside the uterine cavity (Sakata et al., 1990).

The condition is not a neoplasm and because it often manifests itself by multiple and scattered lesions, the old term endometrioma, coined by Blair-Bell, in now supplanted by the term endometriosis (Jeffcoate, 1986).

Endometriosis is a disease of the reproductive years and may be associated with pelvic pain, dysmenorrhoea, dyspareunia and infertility (Marona et al., 1990). Endometriosis can occur any where in the body unusually, however, it is confined to the organs and tissues of the abdomen and pelvis, at or below the level of the umbilicus (Jeffcoate, 1986).

Its exact aetiology is unknown, however more than a dozen of theories have been offered in attempt to explain

its occurrence, but none have satisfactorily explained all the features of the disease (Muse and Wilson, 1987).

The association of infertility and endometriosis is established but it is difficult to say which is the cause and which is the effect (O'Connor, 1987).

During the last two decades, the hormonal and surgical management of endometriosis has been transformed radically by the introduction of a host of sophisticated treatment modalities (Barbieri, 1990).

AIM OF THE WORK

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The aim of this essay is to compare the different classifications of the disease, and to review the relation between endometriosis and infertility.

Also to throw lights on the recent advances in diagnosis and treatment of the disease either by medical therapy comparing the results of treatment with Progestogen with that of Danazol and Gonadotropin releasing hormone agonists, or by surgical treatment using electro-microsurgical techniques and LASER either endoscopic or through open surgery.

REVIEW OF THE LITERATURE

REVIEW ON ETIOLOGY, RISK FACTORS AND PATHOLOGY OF ENDOMETRIOSIS

Endometriosis is one of the most frequently encountered pathologic findings in gynaecology, affecting an estimated 10-15% of all premenopausal women (Deborah, 1988).

Endometriosis is a benign process characterized by the presence and proliferation of endometrial tissue in sites outside the endometrial cavity (Albert, 1988). Such ectopic endometrium demonstrates the ability to grow, infiltrate and even disseminate in a manner similar to malignant tissue (Merrill et al., 1978).

Etiology:

There are so many theories as to the etiology of endometriosis that obviously no single theory covers all clinical presentations of the disease. Either there are multiple disease processes each with a unique etiology, or one disease process with a multifactorial etiology (O'Connor, 1987).

(I) Celomic Metaplasia:-

The first complete theory of histogenesis was advanced by Robert Meyer early in this century. It stated that certain cells, in response to poorly defined stimuli, might change their character and even physiologic function. More

specifically, ectopic endometrium may arise from totipotential cells of the peritoneal mesothelium, either as a result of an inflammatory process or by some undefined inductive influence (Deborah et al., 1988).

In 1980 El-Mahgoub and Yaseen described a case that they believed proved the celomic metaplasia theory because their patient had primary amenorrhoea and endometriosis.

(II) Embryonic cell Rests:-

This theory was predicated on the assumption that in areas adjacent to the developing Mullerian ducts, cell rests of Mullerian origin may be present with the potential to form Functioning endometrium.

During embryogenesis, the urogenital ridges originally reach into the thoracic region where the Mullerian ducts first appear (Deborah, 1988).

(III) Retrograde Menstruation:-

Sampson in 1922 concluded that endometrial cells regurgitated through the fallopian tubes during menstruation explained the vast majority of endometriosis (Deborah, 1988).

Prostaglandin F2 (PGF2) appears to be important in the initiation of menstruation as well as in the production of

rhythmic uterine contractions, which elevate the pressure within the uterus and aid in the expulsion of menses (Vijayakumar, 1981). Of the possible routes of regress for the menstrual effluent, the cervical canal normally has the largest caliber and therefore less resistance than the fallopian tubes, affording the greatest volume of menstrual flow in this direction (Deborah, 1988).

Ayers and Freidenstab observed relative hypotonia of the uterotubal junction in women with endometriosis (Ayers et al., 1985).

The evidence to support the entry of viable endometrial cells through the fallopian tubes are: the presence of endometrial cells in the fallopian tube has been demonstrated on fixed sections of intact fallopian tubes and by perfusing excised human oviducts (Deborah, 1988), as well as the presence of endometrial cells in the peritoneal cavity has been demonstrated by cytologic evaluation of peritoneal fluid obtained through endocentesis and at the time of diagnostic laparoscopy (Bartosik et al., 1986).

(IV) Lymphatic, Hematogenous, and Iatrogenic Dissemination:-

Endometrial tissue spread outside the uterus by lymphatic drainage was first suggested by Halban in 1925 (Deborah, 1988) Vascular spread of endometriosis was proposed by Sampson and later confirmed by Javert. Since