Predictive value of Serum CA125 In Patients with Ovarian Masses

Thesis

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Presented by

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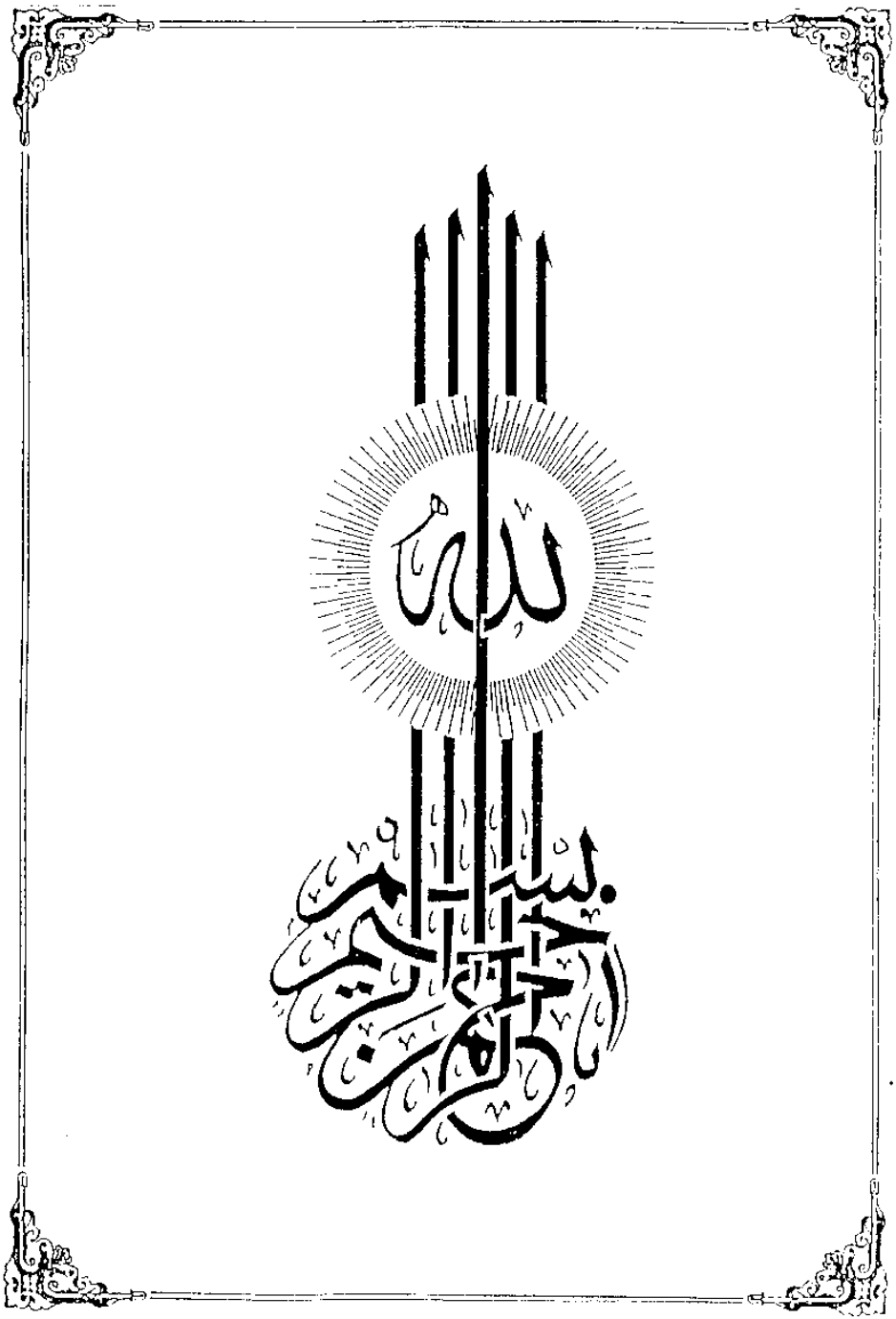
This work is dedicated to

My Wife



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INTRODUCTION

Ovarian cancer accounts for more deaths than cancer of the uterine cervix and corpus *(Hudson, 1985)*. Indeed, 70% of patients have metastases beyond the pelvis at the time of diagnosis *(Piver, 1983)*. The 5-year survival rate is about 20% in advanced disease (stage III and IV) compared with almost 80% in patients with stage I disease (The overall 5-year survival rate has not changed over the past 20 year and remains at approximately 25-30% *(Piver, 1989)*.

This poor survival is mainly attributable to late diagnosis due to the insiduous nature of the disease, to the unreliability of clinical examination and to the lack of an effective early screening technique *(DiSaia and Creasman, 1989)*.

The existence of effective therapy nowadays accentuates the need for a sensitive tumour marker in this concealed disease. The value of tumour markers has already been established in gynaecological oncology. In ovarian germ cell tumours and gestational choriocarcinoma, estimation of alpha fetoprotein (AFP), human chorionic gondotrophin hCG and Schwangerschaft's protein 1 (SPI) provide a quantitative assessment of viable tumour load *(Bagshawe, 1976)*.

In the context of epithelial ovarian cancer several potential markers have been investigated *(Sepala et al., 1975; Knauf and Urbach, 1978)*. Of these, the antigenic determinant CA 125 appears to be the most useful clinically with a reported sensitivity of 82% and a concordance with disease states of 93% *(Bast et al., 1983; Dodd et al., 1985)*. CA 125 was first defined by *Bast et al. in 1981* by a murine IgG1 monocolonal antibody raised against the serous ovarian carcinoma cell line OVCA 433 *(Bast et al., 1981)*.

AIM OF THE WORK

Evaluation of the significance of serum CA 125 levels in diagnosis, management and follow up of patients with ovarian masses.

Review of Literature

OVARIAN TUMOURS

Neoplasms of the ovary present an increasing challenge to the physician. They are the cause of more deaths than any other female genital tract cancer.

The gynecologic oncologist is frustrated by the limitation of knowledge available about etiologic factors in ovarian cancer and by the failure to achieve a significant reduction in mortality from these neoplasms over the past six decades *(DiSaia and Creasman, 1989).*

CLASSIFICATION

The student of ovarian pathology is often confused by the enormous variation in histologic structure and biologic behaviour. For the purpose of standardization for comparing results in different centres, the World Health Organization (WHO) adopted a pathological classification in 1973. The WHO classification is based on the histological cell of origin and no account is taken of the gross characteristics or functional activity of the tumour.

The WHO classification of ovarian tumours is as follows:

I. Common "epithelial" tumours

A. Serous Tumours

1. Benign

a. Cystadenoma and papillary cystadenoma.

b. Surface papilloma.

c. Adenofibroma and cystadenofibroma.

2. Of borderline malignancy (of low malignant potential)

a. Cystadenoma and papillary cystadenoma.

b. Surface papilloma.

c. Adenofibroma and cystadenofibroma.

3. Malignant

a. Adenocarcinoma, papillary adenocarcinoma and papillary cystadeno-carcinoma.

b. Surface papillary carcinoma.

c. Malignant adenofibroma and cystadenofibroma.

B. Mucinous tumours

1. Benign

a. cystadenoma

b. adenofibroma and cystadenofibroma.

2. Of border line malignancy (of low malignant potential)

a. cystadenoma.

b. adenofibroma and cystadenofibroma.

3. Malignant

a. Adenocarcinoma and cystadenocarcinoma.

b. Malignant adenofibroma and cystadenofibroma.

C. Endometrioid tumours

1. Benign

a. Adenoma and cystadenoma.

b. Adenofibroma and cystadenofibroma.

2. Of border line malignancy (of low malignant potential)

a. Adenoma and cystadenoma.

b. Adenofibroma and cystadenofibroma.

3. Malignant

a. Carcinoma

1. Adenocarcinoma

2. Adenoacanthoma
3. Malignant adenofibroma and cystadenofibroma.

b. Endometrioid stromal sarcomas.

c. Mesodermal (mullerian) mixed tumours, homologous and heterlogous.

D. Clear cell (mesonephroid) tumours

1. Benign: adenofibroma.
2. Of borderline malignancy (of low malignant potential).
3. Malignant: carcinoma and adenocarcinoma.

E. Brenner tumours

1. Benign
2. Of borderline malignancy (proliferating).
3. Malignant

F. Mixed epithelial tumours

1. Benign
2. Of borderline malignancy.
3. Malignant

G. Undifferentiated carcinoma

H. Unclassified epithelial tumours.

II. Sex cord stromal tumours

A. Granulosa-Stromal Cell Tumours

1. Granulosa cell tumour
2. Thecoma-fibroma group of tumours.
   a. Thecoma
   b. Fibroma
   c. Unclassified.

B. Androblastomas: Sertoli-Leydig cell tumours

1. Well differentiated
    a. Tubular androblastomas: Sertoli-cell tumour (tubular adenoma of Pick).
    b. Tubular androblastoma with lipid storage (Sertoli-cell tumour with lipid-storage) (folliculome lipidique of Lecene).
    c. Sertoli-Leydig cell tumour (tubular adenoma with Leydig cells).
    d. Leydig cell tumour (hilus cell tumour).
2. Of intermediate differentiation.
3. Poorly differentiated (sarcomatoid).
4. With heterogenous elements.

C) Gynandroblastoma.

D) Unclassified.

III. Lipid cell tumours

IV. Germ Cell tumours

A. Dysgerminoma.

B. Endodermal sinus tumour.

C. Embryonal carcinoma.

D. Polyembryoma.

E. Choriocarcinoma.

F. Teratomas.
    1. Immature
    2. Mature
        a. solid
        b. cystic
            1. Dermoid cyst (mature cystic teratoma).
            2. Dermoid cyst with malignant transformation.