CANCER BREAST AS A TEAM MANAGEMENT

ESSAY

Submitted in Partial Fulfilment of
The Requirement of the
Master Degree in
(GENERAL SURGERY)

By

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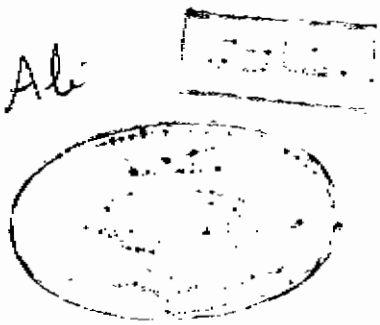
M. B., B. Ch.



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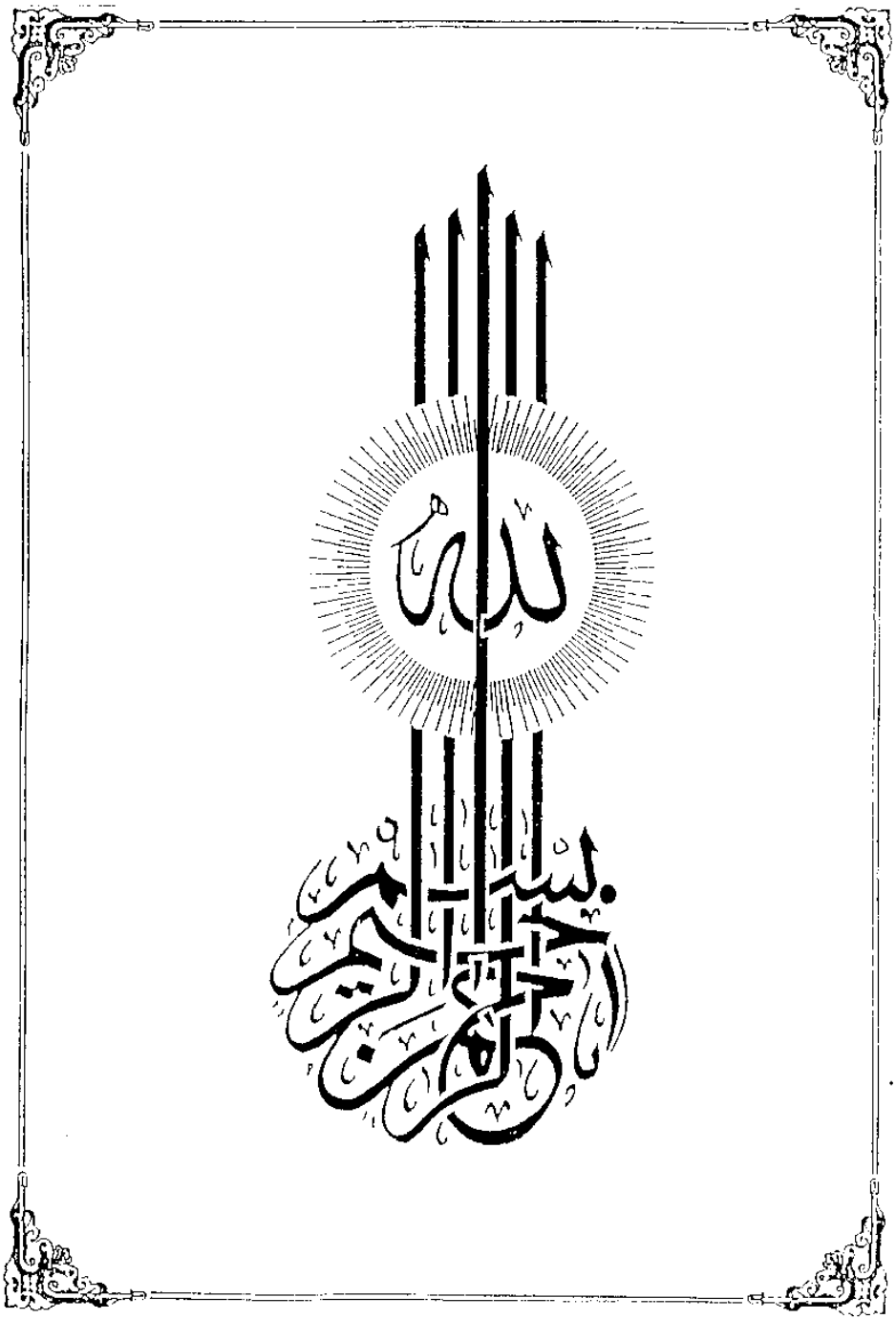
1985

ACKNOWLEDGEMENT

It is a great pleasure for me to take the opportunity to record my sincere thanks and deep gratitude to Professor Dr. REDA MAHMOUD MOUSTAFA, Assistant Prof. of Surgery, Ain Shams University, who gave me much of his golden time and his unlimited knowledge and experience to revise all the review and with his continuous guidance, advise, encouragement and supervision to get a complete representing subject as this. I will stay through out my life indebted to him.

A feeling of gratitude is expressed to Dr. MOHAMED ABDEL MONEM, Lecturer of General Surgery, Ain Shams University.

Last, but not least, I wish to thank every one who contributed with help in this essay.



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INTRODUCTION

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In this work we will study the pathology , diagnosis and team management of cancer breast.

We intended in this study to show that for diagnosis of cancer breast, it should be detected as early as possible. This understanding has led us to provide the different techniques used in diagnosis of breast cancer, with a comment on the limitations and falacies of these techniques.

Cancer of the breast is perplexing to be treated. The different approaches and alternatives of treating the disease, simply indicate that none of them is completely satisfactory.

The aim of this work is to implement the concept of team management of cancer breast, instead of being separate surgeon and oncologist.

PATHOLOGY OF CANCER BREAST

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ETIOLOGY OF CANCER BREAST

A considerable amount of knowledge concerning the causation of breast cancer has been accumulated. We do not yet know its ultimate cause but we have identified a number of factors which influence its occurrence.

In 1936 Bittner made the remarkable discovery that in mice the development of mammary carcinoma is dependent upon a factor transmitted by the mother in her milk to her offspring. It soon became apparent that the milk factor was a virus.

Moore and his associates Charney and Sarkar (1970),have recently described the chemical and morphologic characteristics of the virus, and their methods of isolating, purifying, and transmitting it. Benjamin F. Rush (1974) found that the milk of thirty-nine percent of parsi women in India also were found to contain these particles.

The group of parsi women is of particular interest because of their endogenous history over centuries, resulting in an inbred population.

At present viral etiology is debatable. Three animal models exist for the viral induction of breast cancer, namely:

a) The mouse mammary tumor virus (MMTV),

b) The Mason Pfizer Monkey Virus (MPMV), and

c) The Rat Mammary tumor virus (R-35 virus).

a- Mouse Mammary Tumor Virus (MMTV):

MTV variants infect their host, mostly via the mothers milk. However infection can be transmitted also through both female and male reproductive tracts and by parasites such as fleas. Most exogenous MTVs are highly oncogenic. It is now accepted that mouse mammary cancer, occurring spontaneously in certain suceptible mice strains, is a systemic disease produced by diverse factors such as genes or genetic factors, hormones, a virus (or viruses) and by enviromental factors (Dmochowski, 1971).

These factors are closely interrelated and in their interplay lead eventually to clinical appearance of breast cancer in these animals.

It was found that virus of characteristic morphology, the so called type B particle transmitted in mothers milk, was the causative factor in the existence of all the previously mentioned factors. Also, the virus was detected in the mammary gland and milk, long before the observation of clinical symptoms of neoplasia (Bittner, 1936).

The virus or type B particle was also present in breast cancer cells of mice.

Schlom et al.(1973) reported that MTV antigens, viral RNA and even infectivity can be demonstrated not only in the mammary gland and in mammary tumors but also in epididymis, in blood cells, and in the blood forming organs. Notable is the expression of large amounts of MTV antigen in the cells of spontaneous leukemias of some mouse strains (Calafat et al., 1974).

b- Mason Pfizer Monkey Virus (MPMV):

MPMV has the morphological and biochemical characteristics of an RNA tumor virus (Schlom and Spiegelman 1971). MPMV seems to be common in rhesus monkey, but it is not an endogenous virus since no viral information is regularly found in the DNA of rhesus monkey cells (Colcher et al.,1975).MPMV has also been isolated from cultures of human cells (Nelson Rees et al.,1974). Although MMTV and MPMV are morphologically similar, they share no protein antigens and their polynucleotide chains do not hybridize with one another.

c- Rat mammary Tumor Virus (R-35-Virus)

Oncogenicity of this C-particle-related virus, is isolated from a sprague-Dawley rat mammary tumor, was

demonstrated when female neonates inoculated with it developed mammary cancers. R-35 is not a "B" type particle, unlike MMTV.

Cancer of the breast is the most common cancer among Egyptian females and constitutes 27.2% of all female cancer incidence (Aboul-Nasr and Boutros 1976). However, statistics from the National Cancer Institute, Cairo (1970 - 1980) show that breast cancer accounts for 34.8% of the total malignancies among Egyptian females (Ibrahim et al., 1982).

Lactation effectively inhibits ovarian function. Mothers who breast fed their babies were found to be two to three times less at risk of developing breast cancer than those who did not nurse, (Zippin, 1969), the longer the period of lactation, the lower the risk of breast cancer. However, other investigators reported that the protective effect of lactation seems independent of parity and total time of nursing (Haagensen, 1971), whereas many authors beleive that lactation protects from breast cancer (Bromeis, 1939).
However, studies in the late decade indicated little or no difference in the lactation experience in cancer patients and controls (Wynder et al., 1960, and Mac Mahon et al.,1973).

Parity protects women against mammary carcinoma, and the disease is more frequent in unmarried women. Clemmesen (1951), and Smithers et al., (1952) reported an association between multiparity and a decreased likelihood of developing breast cancer is inversally proportional to the number of children born (Shapiro et al., 1968). The most important aspect seems to be the age at which the women have their first term child, (Mac Mahon et al., 1973).

Woods et al.(1980) reported an association between age at the time of diagnosis of breast cancer and the number of pregnancies carried to term. Their study showed that the average age at diagnosis was higher in nulliparous patients than in those who carried at least one pregnancy to term. Moreover, the average age decreased with the increase in number of births.
Women who first deliver before age 18 have about one-third the breast cancer risk of those who first delivery is delayed until age 35 or older (Lilienfeld et al., 1975; MacMahon et al., 1973).
Vorherr (1979) reported that breast cancer risk appears to be fourfold greater in women whose first pregnancy occurs after 30-35 years of age than in nulliparous women of the same age group. However, the assumption of a

protective effect of early pregnancy and the deleterious effect of late pregnancy is not uniformly agreed upon. Zinns (1979) reported that the incidence of breast cancer during pregnancy is rare. The average age of patients affected with breast cancer while pregnant is 35-36 years. He suggested that the quick progress of the disease during pregnancy may be due to increased hormonal stimulation, which cause major alterations in breast physiology.

Lilienfeld (1963) indicated that female relatives of women with breast cancer have 2-3 times the general population breast cancer rate. This increased risk appears evident for paternal relatives and for those on the maternal side and Haagensen, (1971) suggested that daughters of affected women experience the disease at younger age than did their mothers.

Cady (1970) also reported an excess bilateral disease among young patients with a family history of breast cancer. The risk of breast cancer for women with a family history of breast cancer is increased 1.8 fold (Anderson, 1973), when the family history showed that breast cancer occurred in premenopausal life. The risk is increased three folds and only a 1.5 fold increase is observed when there is a family history of postmenopausal breast cancer (Anderson, 1973).

Anderson (1971) found that relatives of women with bilateral breast cancer had 3 times the risk of relatives of patients with unilateral diseases.

The importance of genetic factors has been shown by Henston (1958) and his associates, who freed high mammary cancer strain mice of the virus by caesarian delivery and foster nursing by mothers of a strain that did not carry the virus. Nevertheless, 38 percent of these mice developed breast carcinoma and transmitted it to successive generations. In further studies of the gene control over the transmission of mouse mammary carcinoma Henston (1960) and his associates presented evidence that several genes are concerned. In human being a genetic factor in breast cancer, if it exists, is not concentrated, and defined by long continued inbreeding, and we should expect it to be less apparent. Nevertheless, carefull studies of the occurrence of the disease in special families have revealed some very convincing data. For example, in 1966 the distinguished surgeon, Paul Broca, who had a deep interest in anthropology and heredity, reported the incidence of cancer in his own family. He traced the cause of death of all 38 members of five generations of his family who died between 1768 and 1856. Ten of the 24 women died of cancer of the breast. Since then

many family histories have been reported in which breast cancer is abnormally frequent (Haagensen 1971).

Murphy and Abbey, in 1959, reported a study of the frequency of mammary cancer in two generations of women. They concluded that their study revealed no statistical significant difference in frequency of breast cancer in mothers and sisters of the patients in their series.

Harvald and Hauge in 1963 reported a study of a number of Danish twins. Their study did not reveal a statistically significant support for the concept that genetic factors play an important role in the transmission of cancer. In his data concerning the age of onset of mammary carcinoma in both paternal and maternal aunts. Haagensen (1971) concluded that mammary carcinoma is a familial disease. When it appears in a family the succeseding generation of women are not only predisposed to develop it, but they develop it approximately 10 to 12 years earlier.

Bucalossi (1957) and his associates compaired the total duration of menstrual life in 4000 patients with breast cancer in their Milan Cancer Institute with the duration of menstrual life in 2788 patients who did not have breast cancer and found it to be slightly longer for the former. MacMahon and Feinleib, in 1960, published a study of the age at which menopause occurred in