EVALUATION OF CLINICAL & LABORATORY FINDING IN PREGNANCY HYPERTENSION

Thesis Submitted by

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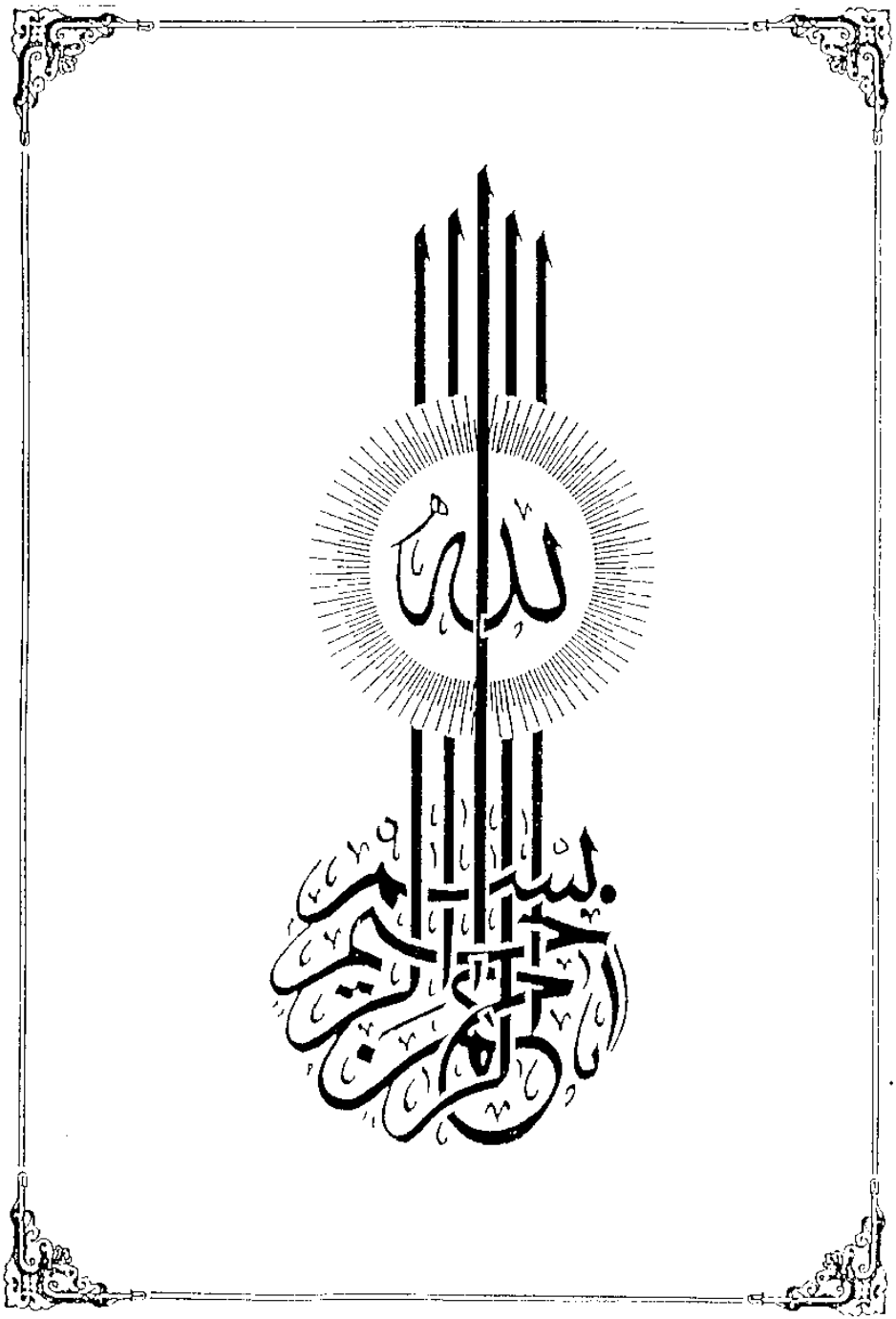

CONTENTS

AIM OF THE WORK

Pre-eclampsia/eclampsia, a syndrome peculiar to pregnancy, has been the subject of controversies and research for a long time. Different openions regarding its cause, pathophysiology and treatment are reported in the literature. In this study we are interested in:

(1) Evaluation of the clinical and laboratory findings of pregnancy induced hypertension (pre-eclampsia/eclampsia) and pregnancy aggravated hypertension (superimposed pre-eclamsia) and their correlation with the severity of the disease and the pregnancy outcome.

(2) To determine the significance of the level of the blood pressure in the mid-trimester together with other parameters in predicting ultimate development of pre-eclampsia.

REVIEW OF LITERATURE

Toxaemia of pregnancy attracted the attention of researchers since a long time. The clinical syndrome of eclampsia has been described in some articles in the ancient history. It was not,however, described as a separate entity. In fact, most articles confused eclampsia with epilepsy. Eclampsia parturientum was first used in 1739 by Sauvage who was the first to distinguish the condition from epilepsy. Just after it became differentiated from epilepsy,eclampsia was then confused with nephritis. Lever in 1843 was then able to differentiate eclampsia from nephritis by disappearance of proteinuria due to eclampsia after delivery. The recognition of hypertension in those patients was not possible until the end of the last century, when the devices

for measuring arterial pressure were invented. The entity of hypertension, non fatal, non convulsive, later known as pre-eclampsia, was then introduced. Again,many of these cases were confused with essential hypertension, which was only recognized as a part of hypertensive disorders of pregnancy by Herrick in 1936 (Chesley 1978).

This big bulk of historic confusion must not surprise us, simply if we realize the fact that now, few years from the year 2000, the same bulk of confusion and even more still exists. Pre-eclampsia/eclampsia is now confused with renal, vascular, cerebral, cardiac, haematologic and even hepatic disorders. A lot of research was conducted in an attempt to identify the aetiology of this peculiar disease of pregnancy. After so many years of

extensive research, pre-eclampsia is still a disease of theories. As an alternative, clinicians were satisfied by research that helped them to recognize the disease early and to treat it accordingly.

Early Recognition:

For so many years prediction of pre-eclampsia was based only on identification of a list of high risk factors that may predispose to the disease.

Factors that are described in the text books include:

Nulliparity: It has long been accepted that pre-eclampsia is a disease of primigravidas and that a normotensive first pregnancy gives some sort of protection for subsequent pregnancies. Whether the duration of the first pregnancy is important or not is controversial. Some believe that pregnancy ending in abortion gives a protection which is,

however, less than pregnancy continuing to term (MacGilliveray 1958). Others deny any protective effect of first pregnancy terminating before 37 weeks (Campbell et al. 1985). Interestingly is that multiparous women who conceive for the first time by a new partner seem to have an increased risk of pre-eclampsia i.e. they behave as virtual primigravidae (Feeney 1980).

Family history: This factor had been described in the literature for over a hundred years. In a small population area pre-eclampsia and eclampsia were diagnosed 11 times in 8 members of the same family over 4 generations. Similar diagnoses were made only once in other members of the community in 10 years (Bracklehurst & Ross 1960). In another study 48.4% of pre-eclamptics had pre-eclampsia in their sisters' first pregnancies compared to 28.2% in

normotensives. The mothers of patients with pre-eclampsia in their first pregnancy had a significantly higher blood pressure than those whose daughters were normal. Comparable results were seen in the siblings (Adams & Finlayson 1961). The authors concluded that there is a familial tendency to pre-eclampsia, probably, in that group of patients that would, anyway, become hypertensive later on in life. Chesley studied daughters, grand-daughter, and daughters-in-law (as control) of eclamptic mothers. He observed a familial predisposition to toxemias that is, at least partly, independent of the hypertensive diathesis (Chesley et al, 1968).

Diabetes: White in her extensive study of diabetic patients found that this population had a higher incidence of eclampsia than the general population (White 1935).

Vedra found also a higher incidence of pre-eclampsia in diabetics than non diabetics. This was obvious in long standing cases, he concluded that the vascular sequels rather than the metabolic disorder is responsible for this high incidence (Vedra 1980).

Chronic hypertension: As quoted from the older literature the incidence of pregnancy toxemia is higher in patients with chronic hypertension.

Pre-eclampsia/eclampsia superimposed on chronic hypertension was observed in two thirds of patients by some (Chesley 1978), and in 86% of patients by others (Chesley 1978). MacCartney, however believed that pre-eclampsia/eclampsia is overdiagnosed in chronic hypertensive patients, since he found in

his electron microscopic studies of renal biopsies, a lower incidence of the pathognomonic lesion of pre-eclampsia, i.e. glomerular endotheliosis (MacCartney 1964).

Age: The age incidence of pre-eclampsia eclampsia is either girls in their teens, or women above 35 years. It is important to realize that most cases lie between 20-25 years of age simply because most first pregnancies cluster in this age group.

Multiple pregnancy: It is believed that twin first pregnancy has a higher chance for pre-eclampsia (5 times more than singlton). Twin subsequent pregnancy may also increase the chance for pre-eclampsia (MacGillivray 1958).

Molar pregnancy: The association of toxaemia

with molar pregnancy has been described in the literature since a long time. The incidence of toxaemia is around 40% in molar pregnancies (Chesley et al., 1946). Page pointed out the importance of the size of the tumour. The incidence of toxaemia was more in late stage tumours (reaching above the umbilicus) (Page 1939). These tumours are rarely seen nowadays. It is important to emphasize that molar pregnancy is associated with early onset severe form of the disease.

Fetal malformation: The association of molar changes, chromosomal abnormalities and pre-eclampsia (Broekhuizen et al 1983), lead to the belief by some that pre-eclampsia may be evoked by fetal genes. The earlier reports of higher male/female ratio in pre-eclamptic patients (Scott et al., 1978) suggests a responsible Y chromosome. Later

studies by Redman and others disagreed with the above findings and pointed out that pre-eclampsia may develop as a result of homozygosity for recessive maternal immune response genes linked to HLA, which could predispose to severe pre-eclampsia (Redman et al 1978). These data are supported by the data of Chesley who found a higher incidence of pre-eclampsia eclampsia in the daughter and granddaughter when compared to the daughters-in-law of eclamptic women (Chesley et al., 1968).

Thus, it seems that pre-eclampsia results from an absent or deficient maternal immune response to the fetus, which is necessary for normal pregnancy, and is more likely to be imperfectly developed on the first exposure to the fetal antigens (Scott et al., 1978).

Other factors related to the disease are described in the literature and include: fetal hydrops & body built. Less important is illegitimate pregnancy, seasonal peaks and low social classes (Chesley, 1978).