STUDY OF THE EFFECT OF HONEY BEE STINGS ON PRE-ECLAMPTIC PATIENTS

Thesis

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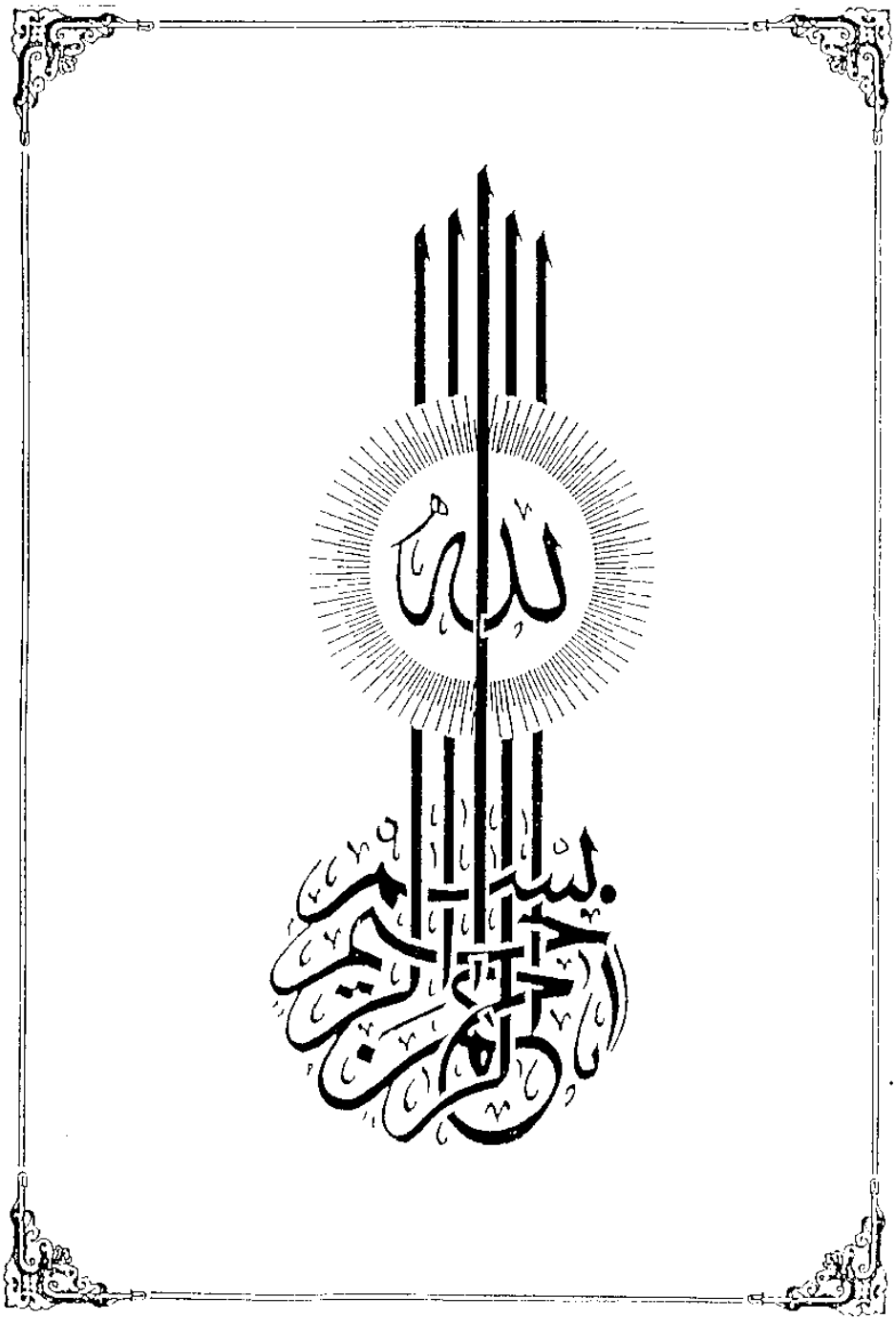
To whom who give me

love and kindness

my parents



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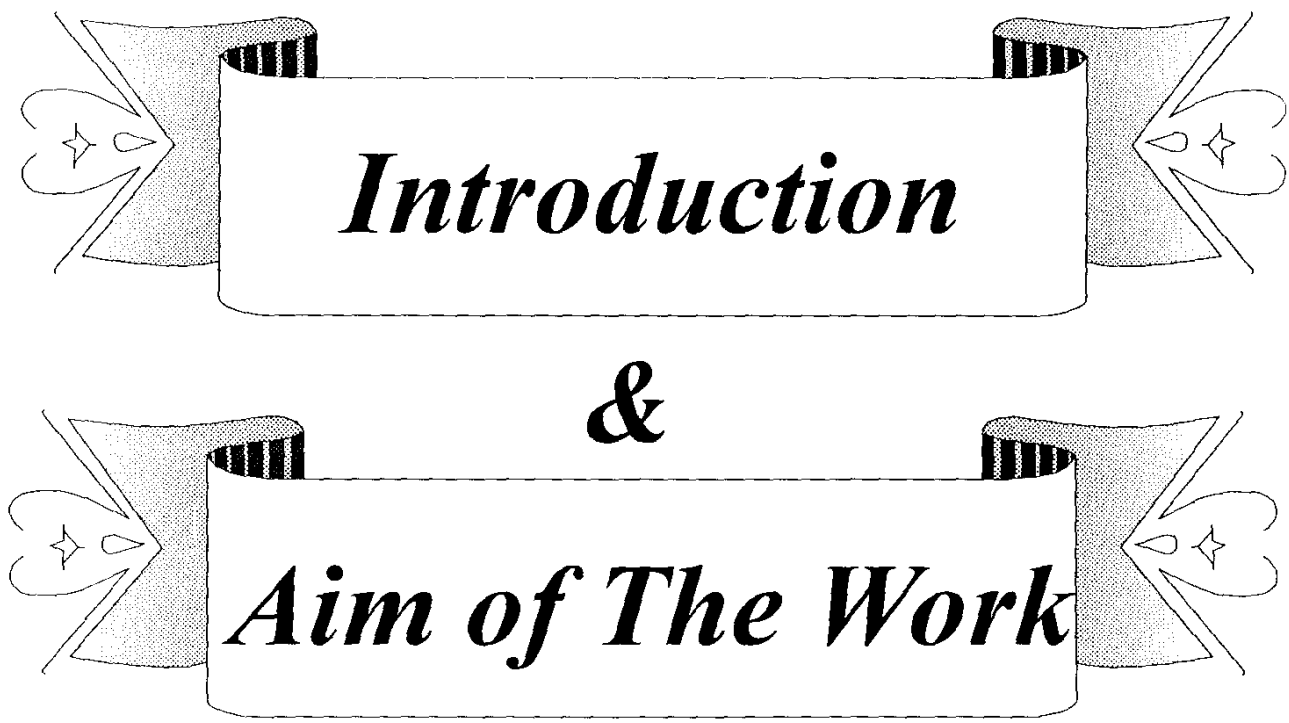

Introduction & Aim of The Work

Hypertensive disorders complicating pregnancy are common and form one of the great triad. along with hemorrhage and infection that continues to be responsible for a large number of maternal deaths. also hypertensive disorders are an important cause of prenatal mortality and sever morbidity.*(Cunningham et al., 1989)*

The presence of chorionic villi in certain women incites vasospasm and hypertension, ,and to effect a cure, these villi must be expelled or surgically removed. the vasospasm hypertensive state and related pathological changes somehow induced by the presence of chorionic villi may not be so great that pregnancy need to be terminated.
(pritchard 1985)

Kaunitz and colleagues *(1985*) reported that 20 percent of 2067 maternal deaths from (1974 - 1978) were caused by hypertensive disease. How pregnancy per se incites or aggravates hypertensive vascular disease remains unsolved despite decades of intensive research, and these disorders remain among the important unsolved problems in obstetrics.

Valeria J. et al., (1990) suggested that the causes of endothelial damage may be due to immune mechanism as the endothelial cells expresses variety of antigens that make them an important target.

Sever pre-eclampsia has traditionally been regarded as a disease of primigravida , with ***Macgillivry (1983)*** showing that the risk of sever disease was is times greater in the first than in a second pregnancy. this study suggested that a previous pregnancy was protective with regard to the development of sever pre-eclampsia.

Cooper et al., (1988) suggested that a fetal genetic component also contributed. Association between eclampsia and miscarriage was found in his study, there may be a genetic basis for disturbances of the normal feto-material interaction in the placental bed and this may point to immune mechanism.

Mauriella et al., (1984) suggested that honey bee stings induce humoral adaptive immune response in bee keeper

Hony bee stings stimulate the induction of natural human interferon in the body which will lead to improvement of the immune system (***Yehia M., et al 1988).***

Aim of The Work

This work aims to study the effect of honey bee stings on pre-eclamptic patients.

Review of Literature

Definitions

The American collage of ***Obstetricians and Gynecologists (1986)*** proposed the following definitions and classification of hypertension that develop during pregnancy or the puerperium ***(Cunningham et al 1989).***

- **Hypertension :-**

Diastolic blood pressure of at least 90 mm Hg or a systolic pressure of at least 140 mm Hg or a rise in diastolic pressure of at least 15 mm Hg or in systolic pressure of at least 30 mm Hg. The blood pressure reading cited must be obtained on at least two occasion 6 hours apart.

- **Pre-eclampsia :-**

It is the development of hypertension with proteinuria, edema or both induced by pregnancy after the 20^{th} weak of gestion and sometimes earlier when there are extensive hydatiform changes in the chorionic villi.

- **Eclampasia :-**

It is diagnosed when convulsion not caused by any coincidental neurological disease such as epilepsy develop in women who also has clinical criteria for pre-eclampsia.

- **Super imposed pre-eclampsia or eclampsia :-**

They are defined as pre-eclampsia or eclampsia that develops in a women with chronic hypertensive vascular or renal disease.

• **Chronic hypertensive disease :-**

It is persistent hypertension of whatever cause antedating pregnancy or detected before the 20th week of gestion in absence of hydatiform mole or extensive moler changes or hypertension that persists beyond 6 weeks post partum.

Earlier the committee on terminology of the American College of ***Obestetricians and Gynecologists (1972)*** proposed the following definitions ***(Hughes 1972)***.

• **Gestational hypertension :-**

It is elevated blood pressure that develops during the later half of pregnancy or during the first 24 hours following delivery. It is not accompanied by other evidence for pre-eclampsia or chronic underlying hypertension and it disappears within 10 days after delivery (it may represent a mild or incipient pre-eclampsia forestalled by delivery) ***(chesley 1985).***

• **Gestational edema**

It is abnormal fluid retention that regress after delivery.

Hypertension never developed.

• **Gestational proteinuria :-**

It is defined as proteinuria exceeding 300 mg/day in the absence of hypertension, edema, renal infection or other known renovascular disease.

These terms are not used by many teaching centers in the United states.

Pre-eclampsia is a multi systemic pregnancy specific disorder, defined clinically as a syndrome of gestional hypertension, excessive proteinuria and generalized edema ***(Taylor et al., 1991).***

A- hypertension :-

It is a measurable sign of pre-eclampsia, it is due to vasospasm. This vaso-spasm is a differential one i.e not affecting the different organs by the same degree.

As the decrease in blood flow due to vasospasm is compensated by the hypertension, the blood flow to different organs is not decreased except in the kidney and placenta ***(Doland 1979).***

Hypertension is a systolic blood pressure of at least 140 mm Hg or a diastolic blood pressure of at least 90 mm Hg or a rise of systolic blood pressure of at least 15 mm Hg over early pregnancy reading. A blood pressure of 140/90 mm Hg represent a mean arterial pressure **(MAP)** of 107 mm Hg which is indicator of cardiac work for it measures the resistance against which the heart works.

$$\text{MAP} = \text{diastolic pressure} + \frac{1}{3} \text{ pulse pressure}$$

The blood pressure cited above must be manifested on two occasions at least 6 hours apart ***(Knuppel and Drukker 1986).***

B- Proteinuria :-

Proteinuria is usually the last of triad to appear. It is diagnosed by one of three :

1) A 24 - hour urine collection containing ≥ 300 mg protein.
2) Two random mid stream urine or catheter specimens with

$$\text{a protein/createnine index} \quad \frac{(10 \times \text{protein mg/L})}{(\text{creatinine nmole/L})} \geq 300$$

3) 2 + (1g albumin / L) or more on reagent strip or sulphosalycilic acid (cold test).

Or 1 + (0.3 gm albumin/L) or more when urinary specific gravity <1030 because the dilution of urine affects protein concentration ***(Stirrat 1987).***

Perinatal mortality was four times higher when proteinuria complicated the hypertensive pregnancies and the level of proteinuria was early related to birth weight ***(Ferrazzani, et al 1990).***

The vascular damage in pre-eclampsia could be responsible for the plasmatic protein escape either in urine or in the interstitial space. Therefor, the vascular damage could explain protein loss, colloid osmotic pressure fall and intrauterin growth retardation ***(Bhatia et al.,1987).***

Proteinuria could be considered both a clinical late marker of the vascular damage and a negative factor for fetal outcome in hypertensive disease ***(Ferrazzani, et al.,1990).***