Effect of the enhanced Lipoperoxidation Process in pre - eclampsia on the fetal oxidant - antioxdant balance

Thesis
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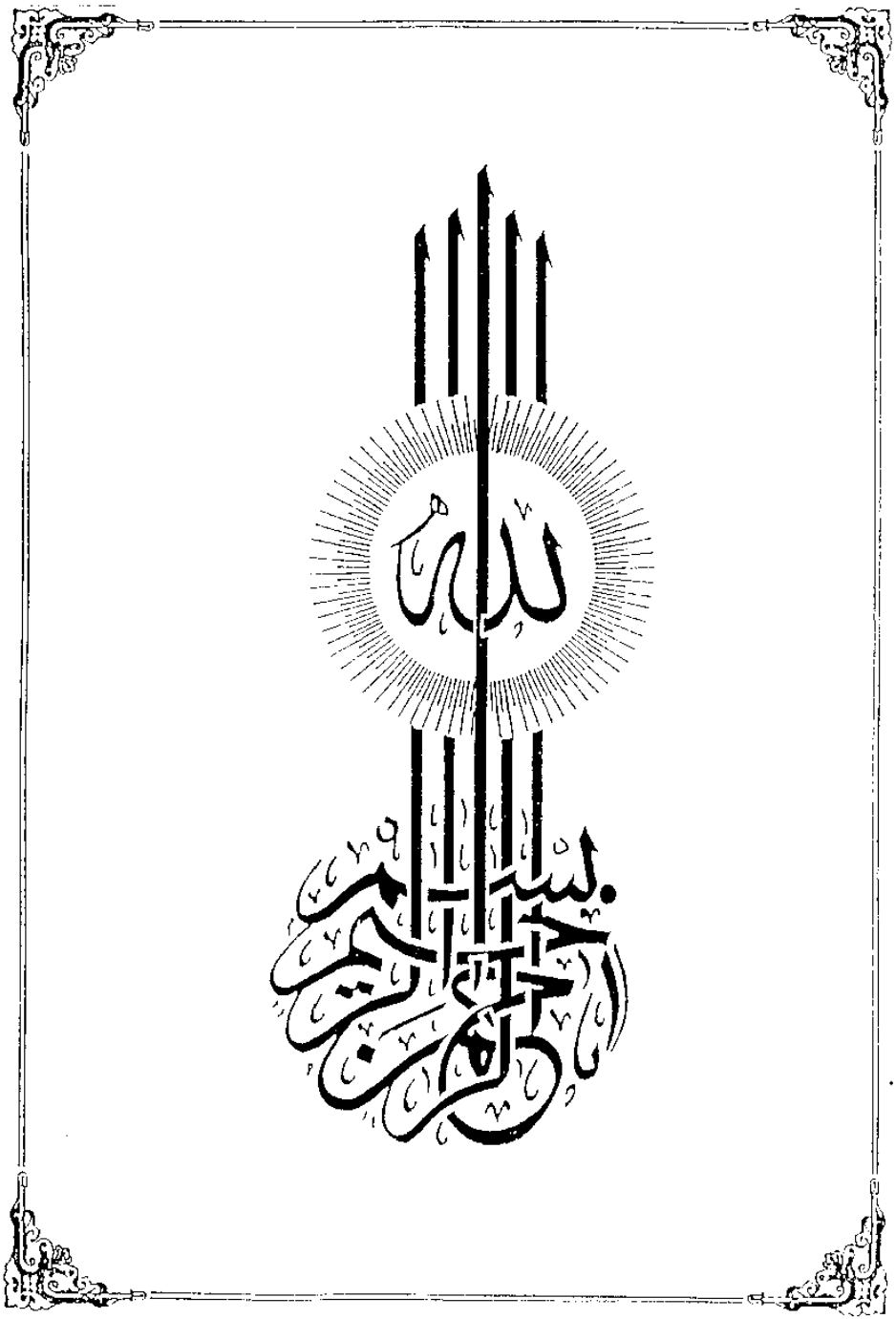
بسم الله الرحمن الرحيم



بسم الله الرحمن الرحيم

« إنا فتحنا لك فتحا مبينا »

صدق الله العظيم

(سورة الفتح آية ١)

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INTRODUCTION

Hypertensive disorders complicating pregnancy are common and form one of the great triad, along with hemorrhage and infection, that continues to be responsible for large number of maternal death (Roberts, et al, 1989). It is a multifacted syndrome with variable involvement of several organ system. The classic triad of hypertension, edema and proteinuria is still the most common presentation (Entmar, 1983).

Most investigative efforts have focused on the hypertensive component of this disorder with reduced attention given to other equally important characteristics. Many studies indicate that pathologic and pathophysiologic changes in preeclamptic women are not secondary to increased blood pressure (Chesley 1978; Reberts, et al., 1989).

Theories about pathophysiologic changes as activation of the coagulation cascade, increased sensitivity to pressors; reduced plasma volume, and abnormalities of renal proximal tubular function, all antedate increased blood pressure (Roberts, et al., 1989).

One of such theories explained the physiologic abnormalities of preeclampsia by dysfunction of vascular endothelial cells (Roberts, et al 1989).

Hubel, et al. (1989) have suggested that lipoperoxide levels in patients with preeclampsia increase beyond normal pregnancy levels and there is conflicting evidence regarding the role of the reduced uteroplacental perfusion which intensifies the release of placental lipid peroxidation products into the circulation. Lipid peroxides are highly

reactive and very damaging compounds. They are very toxic to enzymes, cells, and proteins (Mead, et al., 1986, Hennig et al., 1988). Although they affect many cellular components, the primary sites involve membrane associated polyunsaturated fatty acids and protein thiols. (Freeman and Crapo, 1982).

In view of its potentially destructive character, uncontrolled lipid peroxidation has been suggested as an etiologic factor in preeclampsia (Roberts et al., 1989). Impaired function of the vascular endothelium may, in turn, cause vasospasm, the general increase in sensitivity to vasopressors, and associated cardiovascular complications occurring in the disease (Rodegers et al., 1988).

Antioxidant mechanisms normally control lipid peroxidation and protect against its propagation. Deficiency of physiological free radical scavengers such as vitamin E, Selenium, glutathione and uric acid, causes oxidative stress with resultant overwhelming peroxidation process. (Warso and Lands, 1985).

The placenta is a rich source of polyunsaturated fatty acids . (Ogburn et al., 1988), so the combination of increased oxygen radicals generated from thromboxane production with the high placental content of fatty acids would result in increased placental formation of lipid peroxides. Placental lipid peroxides apparently contribute to maternal circulating levels because plasma lipid peroxide levels decrease precipitately after delivery (Wickens et al., 1981).

The placental tissue actively transports a small quantity of lipid by pinocytosis from the maternal blood to the cord blood and at the sametime, it is possible that superoxide and lipoperoxides also reach the fetal tissue and affect its development (Yamaguchi et al., 1964).

Yoshioka, et al., 1987, reported that maternal , fetal and placental levels of lipoperoxides and antioxidant change during pregnancy.

Takehara et al (1990) confirmed that the lipoperoxide concentration in blood of pregnant women increased as gestation progressed, but it was kept lower in the cord blood than in the maternal blood.

Diamant et al. (1980) suggested that the placental tissue supresses lipoperoxide formation and protects the fetus from any kinds of radicals. Yoshioka et al., (1979) observed that antioxidant activity was higher in the maternal blood than the cord blood, however the difference was not statistically significant.

AIM OF THE WORK

This study was carried out to investigate the effect of the enhanced lipid peroxidation process in preeclamptic pregnancies on the fetus by comparing the lipoperoxide levels and the total plasma antioxidant activities of maternal versus fetal blood, and to explore the relation of the lipoperoxide level of the patient to the pregnancy outcome.

REVIEW

Pregnancy Induced Hypertension

Pregnancy may induce hypertension in previously normotensive women or aggravate hypertension in women who have underlying hypertension. Hypertensive disorders complicating pregnancy are common and form one of the great triad, along with hemorrhage and infection, that continues to be responsible for large number of maternal death. (Roberts, 1989).

Definitions and classification :

Pregnancy induced hypertension is hypertension that develops as a consequence of pregnancy, and regresses postpartum (The American college of Obstetricians and Gynecologists, 1986).

Pregnancy induced hypertension is divided into three categories :-

1. Hypertension alone : (without proteinuria or pathological edema).

Hypertension is defined as a 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 30 mm Hg. the blood pressure readings cites must be obtained on at least two occasions 6 hours or more apart (Villar et al, 1988).

2. Preeclampsia : It is the development of hypertension with proteinuria and or pathological edema after the 20th week of pregnancy and sometimes earlier when there are extensive hydatidiform changes in the chorionic villi. Preeclampsia is almost exclusively a disease of nulliparous women (Chesley, 1985).

Proteinuria is defined as 300 mg or more of urinary protein during a 24-hour period or 100mg/dl or more in at least two random urine specimens collected 6 hours or more apart (Chesley, 1985).

The combination proteinuria and hypertension during pregnancy markedly increases the risk of perinatal mortality (Friedman and Neff, 1976).

Proteinuria is a sign of worsening hypertensive disease, especifically preeclampsia and when it is overt and persistent, the maternal and Fetal risks are increased even more (Chesley, 1985).

3. Eclampsia : It is diagnosed when convulsions, not caused by any coincidental neurological disease such as epilepsy, develop in a woman who also has clinical criteria for preeclampsia (Brown et al, 1987).

The seizures are grand mal and may first appear before labor, during labor, or post partum. Any seizure that develops more than 48 hours post partum is more likely to be the consequence of some other lesion of the central nervous system. However, Brown et al, 1987have encountered otherwise typical up to 10 days postpartum.