INTRODUCTION

INTRODUCTION

Pre-eclampsia is a pregnancy-specific disorder characte-rized by increased blood pressure, urinary loss of proteins, edema, and activation of the hemostatic system (Brown, 1989)

Chesley and Cooper (1986), examined the possibility that susceptibility to preeclampsia is dependent upon a single regressive gene.

Dietary deficiency especially for calcium have been suspected as a cause of pre-eclampsia (Belizan and Villar, 1980).

Prostaglandins are implicated in the causation of pre-eclampsia prostacyclin (PGI_2); the vaso-dilating factor may account for hemo-dynamic changes in pregnancy while increasing "thromboxane A_2" the vaso-constricting, platelet aggregating factor is seen in pre-eclmpsia (Stevena, 1988).

Recently; Kaaja et al. (1995), have found that, the metabolic changes hypertriglyceridemia, hyperinsulinemia, low HDL_2 cholesterol and hyperuricemia in pregnancy induced hypertension resemble the main features of "Insulin Resistance Syndrome". This may result in endothelial cell dysfunction as evidenced by prostacyclin suppression.

An interesting analogy of the concept of insulin resistance can be observed in pregnancy induced hypertension (Kaaja et al., 1995).

Patients with hypertension and insulin resistance often have elevated serum triglycerides and decreased high-density lipoprotein (HDL).

It has been suggested that insulin resistance might be the common etiologic factor causing hypertension, hyperinsulinemia, hypertrigylceridemia and low serum HDL cholesterol.

Endothelial cell dysfunction in preeclampsia may account for the altered vascular reactivity, activation of the coagulation cascade, and loss of vascular integrity that accompany the disease (Roberts et al., 1989).

Normal human pregnancy results in a pronounced physiologic hyperlipidemia involving a gestational rise in blood triglycerides and cholesterol (Potter and Nestel, 1979).

Women with preeclampsia display additional alterations in blood lipids, reflecting a disordered lipid and lipoprotein metabolism (Kokia et al., 1990 and Kaaja et al., 1995).

Circulating triglycerides and free fatty acids are dramatically elevated in nulliparous women with uncomplicated pregnancies (Hubel et al., 1996).

Metabolic patterns resembling "Syndrome X" or "Insulin Resistance Syndrome" are more common in preeclampsia (Kaaja et al., 1995).

Normal pregnancy is characterised by a gestational increase in total and LDL cholesterol followed by a progressive decrease during the puerperium (Potter and Nestel, 1979).

Hyperinsulinemia and insulin resistance are common findings in hypertensive individuals (Ferrannini et al., 1987). Patients with hypertension and insulin resistance often have elevated serum triglyceride and decreased high density-lipoprotein (HDL) cholesterol levels.

The mechanism by which insulin resistance could cause an alteration in lipid metabolism is unclear (Hubel et al., 1996)

However, there is evidence that insulin plays an important role in lipid metabolism (Steinberger et al., 1995).

Hyperinsulinemia has been documented to enhance hepatic very low density lipoprotein synthesis and thus may directly contribute to the increased plasma triglycride and low density lipoprotein cholesterol (LDL-C) levels (Stalder et al., 1981 and Orchard et al., 1983).

Lipoprotein is known to increase during pregnancy but the factors responsible for the change have not been established. In addition the lipoprotein concentration in preeclamptic pregnancy is significantly higher than in normal pregnancy (Kobayoshi et al., 1992).

The apolipoproteins are an important determinant of metabolism and the structure of plasma lipoproteins. The concentration of apolipoprotein B in preeclamptic pregnancy was significantly higher than in normal pregnancy (Kobayaski et al., 1992).

In preeclampsia Apolipoprotein AI and HDL cholesterol concentrations were reduced (Rosing et al., 1989).