INTRODUCTION

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Pre-eclampsia occurs in the second half of pregnancy and is characterized by hypertension, proteinuria and oedema.

Approximately 10% of pregnancy complications are due to some degree of pregnancy induced hypertension. This hypertension can produce severe perinatal morbidity and mortality ***(Tanfeiled ,1987)***.

At present the causes of pregnancy induced hypertension have not been identified, although concomitant changes in a number of factors e.g. increased levels of factor VIII related antigen, fibronectin, cellular fibronectin and thrombomodulin are reported in pre-eclampsia ***(Saleh and Bottoms, 1988).***

The vascular endothelial unit has attracted the attention of many investigators as an etiological factor for pregnancy induced hypertension ***(Minuz et al, 1987 and Roberts et al, 1989).***

Rodgers et al, (1988) have shown that serum obtained from pre - eclamptic women has a greater cytotoxic effect on cultured endothelial cells than serum from normotensive pregnant women.

Plasma levels of endothelins are elevated in pre-eclampsia which probably reflect extensive maternal endothelial damage ***(Kamoi, 1990 and Kraayenbrink, 1993).***

Recent studies have shown that **Tumour necrosis factor alpha (TNF-α)** is involved in the pathological processes that produce damage to the vascular endothelial cells of the placentae ***(Greer, 1994).***

Van der poll, (1991) stated that (TNF-α) enhances prostaglandin synthesis. It is a potent and multifunctional cytokine, described as a product of activated macrophages ***(Trinchieri, 1992).***

Tumour necrosis factor - alpha (TNF-α) has a wide range of effects on cellular functions, and interferes with carbohydrate and fat metabolism and D.N.A synthesis ***(Yui and Garcia,1994).*** It was shown to be produced by many types of cells in female reproductive organs and tissues ***(Terranova, 1995).***

At present the evidence concerning a role for (TNF-α) in pre - eclampsia is contradictory .

Hyperinsulinemia and insulin resistance are common findings in hypertensive individuals ***(Ferrannini et al, 1987).***

Kaaja et al, (1995) found that the metabolic changes hypertriglyceridemia, hyperinsulinemia, low H.D.L cholesterol and hyperuricemia in pregnancy induced hypertension, resemble the main features of "Insulin Resistance Syndrome ". This may result in endothelial cell dysfunction .

An interesting analogy of the concept of insulin resistance can be observed in pregnancy induced hypertension.

The study of (TNF-α) in serum of pre-eclamptic women and the metabolic changes of insulin in these cases may explain the patho- physiology of pre-eclampsia evidenced by histopathological examination of the placentae.