Introduction

Hypertensive disorders complicating pregnancy are common and form one of the deadly triad, along with hemorrhage and infection that result in much of the maternal morbidity and mortality related to pregnancy ***(Frias and Belfort, 2003)***.

Preeclampsia is a multisystem disorder that complicates 6% to 8% of pregnancies, with higher rates in women with preexisting hypertension, diabetes mellitus, or previous history of preeclampsia. Preeclampsia is associated with increased risk of adverse maternal outcome (abruption placentae, HELLP syndrome, eclampsia) and perinatal death. Its prevention, therefore, is of particular importance ***(Haddad, 2003)***. According to the ***National Center for Health Statistics (2000)***, hypertension associated with pregnancy was the most common medical risk factor ***(Ventura et al., 1998)***. It was identified in 146320 women, or 3.7 percent of all pregnancies that ended in live births. In 12.345 of these women eclampsia was diagnosed, and maternal deaths from this complication still remains a threat ***(Ventura et al., 1998)***. ***Berg and colleagues (1996)*** reported that almost 18 percent of 1450 maternal deaths in the United States from 1987 to 1990 were from complications of pregnancy-related hypertension.

The signs and symptoms of pre-eclampsia are usually apparent relatively late in pregnancy (late second to early third trimester). However the disorder results from abnormal interaction between fetal and maternal tissues much earlier in pregnancy, between 8-18 week's gestation ***(Dekker and Sibai, 1998)***.

How pregnancy incites or aggravates hypertension remains unsolved despite decades of intensive research, and hypertensive disorders remain among the most significant unsolved problems in obstetrics. Pregnancy induced hypertension (PIH) is a common disorder of pregnancy and a major cause of maternal and fetal morbidity and mortality. Thus its prevention would have a significant impact on maternal and perinatal outcome ***(Cunningham et al., 2001)***.

Prevention requires availability of methods of early detection. A variety of strategies have been used in attempts to prevent preeclampsia. Usually these strategies involve manipulations of diet ***(Knuist et al., 1998)*** and pharmacological attempts to modify the pathophysiological mechanisms thought to play a role in the development of preeclampsia. The latter includes the use of low dose aspirin ***(Caritis et al., 1998)*** and antioxidants ***(Chappell et al., 1999)***.

A large randomized trials have not shown a benefit in reducing the rate of preeclampsia or perinatal outcome from the use of low-dose aspirin. The majority of adverse pregnancy outcomes occurred in women who developed severe gestaional hypertension-preeclampsia prior to 35 weeks' gestation and in those women with previous preeclampsia and/or pre-existing vascular disease ***(Sibai et al., 2003)***.

Recent randomized trial in populations with low calcium intake demonstrates that while supplementation with 1.5 g calcium/day did not result in a statistically significant decrease in the overall incidence of pre-eclampsia, calcium significantly decreased the risk of its more serious complications ***(Villar and co-workers,2006).***

Early prediction of preeclampsia is difficult. Several maternal serum proteins such as PAPP-A, free β-HCG, placental growth factor, vascular endothelial growth factor and soluble fms-like tyrosine kinase-1 are reported to be useful markers for the prediction of preeclampsia. However, it is still difficult to predict the occurrence of hypoxia-related complications precisely ***(Spencer and colleagues,2005 ; Papaqeorqhiou and colleagues,2006).***