INTRODUCTION

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

Preeclampsia (PE) 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 (abruptio placentae, HELLP syndrome, eclampsia.) and perinatal death. Its prevention, therefore, is of particular importance. The latter must be determined together with the group of women who would benefit from it ***(Desvaux and 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 146,320 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 remain a threat. ***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, 1991).***

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 use of low dose aspirin ***(Caritis et al, 1998)*** and antioxidants ***(Chappell et al, 1999).***

Recent 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 gestational hypertension-preeclampsia prior to 35 weeks' gestation and in those women with previous preeclampsia and/or pre-existing vascular disease. Also it was found that epidural anesthesia is safe in parturients receiving low-dose aspirin in pregnancy and in women with severe preeclampsia ***(Sibai et al, 2003).***

During the past two decades numerous clinical, biophysical, and biochemical tests have been proposed for the early detection of pre-eclampsia. Some of these tests are simple, whereas others are invasive. Some have been studied extensively; while others are still under clinical investigation. For example, Single urine calcium to creatinine ratio was suggested as an effective method for screening women at greatest risk for pre-eclampsia ***(Kazerooni and Hamze-Nejadi, 2003).*** Also, microproteinuria of more than 375 mg/l was suggested as a cut-off value and as a screening test for the early detection of women at risk of developing pre-eclampsia. On the other hand, serum uric acid and creatinine had

no predictive value as a screening test for pre-eclampsia ***(Weerasekera and Peiris, 2003).***

Furthermore, protein/creatinine ratio did not exclude adequately the presence of significant proteinuria or predict severe proteinuria and therefore should not be used as an alternative to 24-hour total protein evaluation ***(Durnwald and Mercer, 2003).*** Likewise, second-trimester homocysteine concentration levels were not helpful in the prediction of preeclampsia in chronically hypertensive women ***(Zeeman et al, 2003).***

Measurement of hepatocyte growth factor in peripheral blood between 14 and 21 weeks gestation may offer new possibilities in the early diagnosis and prediction of fetal birth weight but not of preeclampsia (**Tjoa et al, 2003)**.

Serum cystatin C is used as a marker, not only for impaired renal function, but also for the degree of glomerular endotheliosis and increase in glomerular volume in pregnancy. Therefore it was recently suggested to be of value in the monitoring of pregnancies complicated by pre-eclampsia (**Strevens et al, 2003).** Finally, plasma adiponectin concentrations were not found to be elevated in normal human pregnancy and paradoxically elevated (by 47%) in women with PE. This may be secondary to exaggerated nonspecific adipocyte lipolysis or as a physiological response to enhance fat utilization and attenuate endothelial damage. Future studies should determine whether adiponectin concentrations help improve prediction of PE (**Ramsay et al, 2003)**.

Tests proposed for early detection of P.I.H disease include ***(Dekker and Sabai, 1991):***

Standards methods of antenatal care :

* Blood pressure increase.
* (MAP) Mean arterial pressure in second trimester.
* Proteinuria by dipstick, microalbuminuria.

Vasoconstrictive tests :

* Cold pressor test.
* Isometric exercise test.
* Roll-over test.

Biochemica markers :

- Maternal Serum inhibin A

The clinical, biochemical and biophysical tests will be discussed and compared. Their relative importance will be evaluated as methods for prediction of preeclampsia.