Introudction

Pre-eclampsia. (PE) is a major cause of fetal and maternal mortality. The condition does not usually manifest itself until late in pregnancy, but the underlying pathology is present much earlier ***(Roberts and Redman, 1993; Redman, 1995).***

Pre-eclampsia is defined as "the rise of blood pressure 140/90 mmHg or more, after 20 weeks gestation in women with no history of hypertension or renal disease, with the presence of proteinuria 300mg/day, or more than +2 or +1 in a catheterized random urine specimen ***(Bartha et al., 2001).***

There are many risk factors for the development of PE. Among these are prmigravidity, race, low social class and smoking.

Race is considered as a possible risk factor for the development of PE. The incidence was reported to be more common in black races than in white races, mostly due to a genetic factor and greater prevalence of development of chronic hypertension ***(MC Gillvary, 1993).***

Some authors found an association between PE and low social class, while others did not ***(Kilpark et al., 1998).***

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Pre-eclmpsia is more common below the age of 17 years and above 35 years. This may be due to poor immune capacity at that ages ***(Arias, 1993).***

The chances of developing pre-eclampsia are greater if pregnancy is a first pregnancy, or if it is a first pregnancy with a new partner. PE has been traditionally regarded as a disease of primigravidae ***(Chesly, 1984).***

Obesity is also a risk factor for PE. A systematic review reported that, at antenatal booking a body mass index (BMI) more than 35 kg/m², a systolic BP of 130 mmHg, or a diastolic BP of 80 mmHg were predictive of the development of PE ***(Duckitt et al., 2006).***

The pathophysiological changes that are thought to be responsible for pre-eclampsia appear to begin between 10 and 20 weeks gestation ***(Schardifi and Rtatus, 1987).***

Being able to predict which pregnant women, are at great risk for the development of preeclampsia would be of great value in preventive studies, because it would be possible to distinguish a high-risk population that could benefit from more condensed treatment and intense observation.

A proper screening test for prediction of PE should be sensitive, reliable, cheap and easy to perform. Early prediction of pre-eclampsia can be done by; a family history of PE, blood pressure measurement in mid trimester, roll-over test and hand grip test and angiotensin sensitivity test. Now, Doppler flowmetry measurements in the uterine artery at mid trimester (16-24 week) found by many authors to be associated with the development of PE.

The normal process of trophoblastic invasion is completed by 20 weeks gestation. Hence, the initiating placental pathology for PE exists prior to this stage of pregnancy and long before the onset of the clinical syndrome. Therefore, it might be possible to develop new plasma/ serum

biochemical markers for identifying subjects at increased risk of developing pre-eclampsia. Several laboratory markers have been proposed to predict the development of pre-eclampsia. These tests include; renal tests (as uric acid, calcium excretion, micro albuminuria and kallirein), endothelial dysfunction markers (fibronectin, plasminogin activator inhibitor, thrombomodulin, cell adhesion molecules, endothelins, free fatty acids, vascular endothial growth factors, and placental growth factor, coagulation factors, and other markers, (atrial naturetic peptides, lysosomal enzymes, B_2-microglubulin, B-hCG, number of erythroblasts, tumor necrosis-factor, plasma placental isoferritn, fetal DNA, oxidative stress, , and others tests as prostacycline metabolites, activine, inhibin A and corticosteroid releasing hormone. However, many of these tests are complicated, need special equipments to do, expensive and were not practical for mass prediction of pre-eclampsia.

Several studies have confirmed that elevated second trimester maternal serum hCG was associated with subsequent development of pre-eclampsia ***(Gonen et al., 1992 Sorensen et al, 1993a;Wenstrom et al., 1994;Ashour et al,1973, Van Rjn et al., 1999; Muller et al., 2000; Ong et al., 2000; Chandra et al., 2003; and Lepagee et al., 2003).*** Other authors, used maternal serum human chorionic gonadotropoin in their studies and came to the same conclusion ***(Hsu et al., 1994; Yaran et al., 1996; Benn et al., 1999; Roiz Hernandez, 2006; Yaron et al., 1996). Bahdo-Singh et al., (1998),*** used mid trimester maternal urine human chorionic gonadotopin beta-subunit core fragment they found that clinically normal patients, with elevated mid trimester levels of urine beta-core fragment of hCG, were more at increased risk for subsequent development of pe-eclampsia.

These previous studies stimulated us to undertake this study on the predictive value of a single measurment of serum B-hCG, measured at 16-20 weeks gestation, in Egyptian primigravidae as apredictor for development of pE.