Introduction

Hypertensive disease is the most common medical complication of pregnancy, with a reported incidence 12-22% of pregnancies, and it is directly responsible for 17, 6% of maternal deaths in the United States (US) **(ACOG 2002).**

Preeclampsia (PE) is a major cause of maternal and prenatal morbidity and mortality worldwide **(Ascarelli et al 2005).** It's a pregnancy specific disease defined classically as the development of hypertension, proteinuria and/or edema in the second half of pregnancy in a women who has previously been normotensive and otherwise clinically well **(Wanger 2004).**

Maternal complications of PE can be severe and include abruption placenta, thrombocytopenia, hepatic hemorrhage and rupture, eclampsia, disseminated interavascular coagulation, stroke, intracerebral hemorrhage, cardiopulmonary failure, adult respiratory distress syndrome hemolysis, elevated liver enzymes and low platelet count (HELLP syndrome) **(Schlenzing et al 2000).**

Preeclampsia is a form of hypertension that is unique to human pregnancy, occurs in about 2-10% of all pregnancies **(Fugate and Meyer 2001).** From these patients 2-10% will progress to HELLP syndrome **(Schelenzing et al 2000).** Eclampsia occurs in about 1/2-2% of cases of preeclampsia i.e. about one out of 1500 pregnancies. Eclampsia accounts for approximately 50,000 maternal deaths worldwide annually **(Omu et al 2004).**

Twenty five percent of eclampsia cases occur before labour, 50% during labour and 25% after labour and has been developed without prior development of preeclampsia. The incidence is increased in women of low socioeconomic status, extremes of age and primigravid state **(Fugate and Meyer 2001)**.

The exact cause of PE has not been identified. Numerous theories of potential causes, ranging from genetic, immunologic, endocrinologic, dietary, vascular, neurological factors and even infectious agents has been reported as possible aetiological causes **(Fugate and Meyer 2001)**.

Resolution of PE occurs only with delivery and subsequent removal of functioning trophoblastic tissue. These trophoblastic cells produce a factor that is cytotoxic to endothelial cells and is responsible for the multiplicity of clinical expression of preeclampsia pathophysiology **(Rodgers et al 1988)**.

The observation that postpartum curettage leads to a more rapid normalization of the elevated blood pressure of PE was reported in 1961 in a nonrandomized study **(Hunter et al 1961)**.

A randomized American study concluded that immediate postpartum curettage of women with severe preeclampsia resulted in a faster clinical recovery than those who did not undergo curettage **(Magann et al 1993)**.