INTRODUCTION

The human placenta is a hemochorial villous organ that is essential for transfer of nutrients and gases from the mother to the fetus and for removal of fetal waste products ***(Blackburn, 2007).***

Placentation begins with implantation of the blastocyst beneath the uterine epithelium and differentiation into embryonic and extra-embryonic tissues ***(Benirschke and Kaufmann, 2000).***

During the physiological changes that occur in the first and in the beginning of the second trimester of pregnancy, spiral arteries of the placental bed are converted into the uteroplacental arteries ***(Pijnenborg et al., 1983).***

The essence of this conversion consists of losing the muscular elements in the vessel walls, making them unable to respond to vasomotor influences. Cells that infiltrate the walls of spiral arteries and replace their normal elements are called migratory, non-villous, or intermediate trophoblastic cells ***(Pijnenborg et al., 1981).***

Beside infiltrating and replacing the anatomic structures of spiral arteries, intermediate trophoblastic cells also penetrate into the lumina of these vessels, forming endovascular plugs ***(Kos et al., 2005).***

By term the placenta weighs approximately 480 gm (give or take 135 gm) with a diameter of 18-22 cm. and thickness of 2-2.5 cm , although considerable variation is seen ***(Blackburn ,2007).***

Pre-eclampsia is a major cause of maternal morbidity and mortality, complicating 7–10% of pregnancies ***(Anderson and Ren, 2002).***

Pre-eclampsia is a syndrome defined as the onset of hypertension and proteinurea after 20 weeks of gestation in previously normotensive and

nonproteinuric women. Although the precise mechanism of disorder remained elusive but according to new emerging consensus it is a complex polygenetic trait in which maternal and foetal genes as well as environmental factors are involved ***(Laivuori, 2007).***

Though pathogenesis is not yet clear, several theoretical mechanisms have been proposed which result in uteroplacental insufficiency ***(Bartha et al., 2001).***

Common pathological features in preeclampsia include small placentas with decidual arteriopathy, infarcts in central portions of the placenta, abruption placentae, and intervillous thrombosis. Arteriopathy is commonly found in PE, as well as intrauterine growth restriction, whereas the other findings are nonspecific ***(Kaufmann et al., 2003).***

Some studies, especially those dealing with the morphological analysis of placental bed, have recorded a partial or complete lack of physiological changes in uteroplacental vessels in pregnancies complicated by preeclampsia or eclampsia ***(Kos et al., 2005).***

When the whole placentas are examined, these changes are also noticed in the basal plate and especially in the amniochorial membranes (because physiological changes in the blood vessels are not developed there, due to the lesser degree of invasion of intermediate trophoblast) ***(Khong et al.,1986).***

Two endogenous antiangiogenic proteins of placental origin—circulating soluble fms-like tyrosine kinase 1 (sFlt1) and soluble endoglin have been

suggested to play a causal role in the pathogenesis of preeclampsia ***(Hladunewich et al., 2007).***

Endoglin, a co-receptor for transforming growth factor β1 and β3 (TGF-β1 and TGF-β3, respectively), is highly expressed on cell membranes of vascular endothelium and syncytiotrophoblasts. Placental endoglin is up-regulated in preeclampsia, releasing soluble endoglin into the maternal circulation. Soluble endoglin is an antiangiogenic protein that may inhibit TGF-β1 signaling in vasculature ***(Levine et al., 2006)***.