1. INTRODUCTION

The intrauterine existence of fetus solely depends on one organ “The Placenta”. Placenta is a vital organ playing central role in pregnancy. It maintains pregnancy and promotes normal fetal development and serves as a major organ for transfer of essential elements between mother and fetus.\(^1\) It has a significant role in health of the fetus because it is the sole source of nutrients for the fetus and first line of defense from the external world. Placental function is highly influenced by its anatomical structure.\(^2\) One of the key roles of placenta is the transport of maternal nutrients to the fetus. Many placental diseases result in fetal growth restriction or even fetal death.\(^3\) Placental efficiency is improved by increase in placental nutrient transfer capacity and thus permits increase in the number of grams fetal weight. Placental insufficiency is major cause of impaired fetal growth.\(^1\) Therefore placenta has been implicated with aberrant fetal growth which is associated with pregnancy pathologies. Thus histology and morphology of placenta is considered essential.

There are many well established causes of intrauterine growth restriction (IUGR), and preeclampsia being one of them. Preeclampsia is a systemic disorder defined as development of hypertension and proteinuria after 20 weeks of gestation in previously normotensive woman. Preeclampsia affects 5 to 7 percent of women worldwide and is a major cause for maternal and neonatal morbidity and mortality.\(^4\) It is one of the disorders of pregnancy which is accompanied by pathological changes in placenta and is associated with high perinatal morbidity and mortality.\(^5\) Preeclampsia contributes to complications like preterm birth, perinatal death, IUGR and is directly associated with 10 to 15 % of maternal deaths. The incidence being 3 to 7 % in nulliparas and 1 to 3 % in multiparas. Pathophysiology of this multisystem disorder characterized by abnormal vascular response to placentation still remains unclear.\(^6\)

Preeclampsia is a major unsolved problem in feto-maternal medicine and is a primary cause of placental insufficiency.\(^7\) Despite decades of research on this condition there is no significant improvement to predict preeclampsia prior to the onset of symptoms.\(^8\) Hypertension in pregnancy is found to be associated with various histological changes in placenta. These changes eventually lead to poor fetal outcome.\(^9\) Abnormal
cytotrophoblast invasion leads to placental ischemia and endothelial dysfunction which characterizes preeclampsia.⁸

Etiology of preeclampsia remains unknown, currently accepted hypothesis are placental ischemia hypothesis, genetic hypothesis, immune maladaptation hypothesis and hypothesis of imbalance between free oxygen radicals and scavengers in favor of oxidants.¹⁰ A long standing hypothesis suggests that preeclampsia develops as a consequence of immune maladaptation between mother and fetus leading to impaired tissue and arterial invasion by trophoblastic cells followed by worsened placental perfusion. This results in chronic hypoxia in intervillous space and is expected to trigger placental necrosis.¹¹

Pathogenesis of preeclampsia is believed to be multifactorial although it remains a subject of extensive research. It is accepted that the presence of placenta rather than fetus is responsible for development of preeclampsia, thus placenta plays a vital role in development of preeclampsia but the severity and progress is significantly affected by maternal response to factors and proteins derived from placenta.⁴ Although the cause of preeclampsia is unknown evidence strongly implicates placenta and anatomical examination shows that the part of placenta that is most affected by this syndrome is fetal maternal interface.¹²

Preeclampsia is a disorder of vascular endothelial malfunction and vasospasm.¹³ Placenta is the key organ in pathogenesis of preeclampsia and its removal abolishes the disease. Pathological examination of placentae of preeclamptic women show abnormalities like infarcts, atherosis, thrombosis and chronic inflammation.¹⁴ A fetus is not a requisite for preeclampsia although chorionic villi are essential. Preeclampsia is characterized by abnormalities like vascular endothelial damage subsequently leading to vasospasm.⁹

Previously demonstrated histochemical observations indicate changes in intensities and distribution of important placental enzymes. Trophoblastic dysfunction may lead to placental insufficiency in preeclampsia. Disappearance of the disease immediately after delivery indicates that preeclampsia is a “placental disease”. ⁷

This study reviews the importance of morphological and histochemical changes of placentae associated with preeclampsia.