

INTRODUCTION

Preeclampsia/eclampsia is one of the most common complications of pregnancy. It is a cause of high morbidity for both mother and fetus, especially in developing countries in a recent survey. eclampsia was found to be a major cause of maternal mortality (24.2%) second only to obstetric hemorrhage (*Vanderjagt et al., 2004*)

Despite the considerable research carried out on EPH-gestosis, the aetiology of this condition remains unsettled. Most of the new information is piecemeal and an overall explanation is lacking (*D'Anna et al., 2004*).

Abnormalities within the methionine- Homocysteine Pathway have been implicated in the development of placental disease and defects within the placental vascular bed which associated with placental infarction or abruption and EPH-gestosis (*Ray and Laskin, 1999*).

The presence of high maternal Homocysteine concentration has been associated with pregnancy complication as placental abruption, EPH-gestosis and increase the risk of poor pregnancy outcome (*Scoll and Johnson, 2000*).

Homocysteine results from the transmethylation of methionine. Its metabolism depends primarily on three enzymes and several vitamin cofactors (*Aubard et al., 2000*).

Hyperhomocysteinaemia in the maternal or fetal circulation is associated with placental vascular disease with either the maternal

syndrome of pre-eclampsia and/ or fetal syndrome of growth restriction. Maternal plasma homocysteine levels were significantly higher in pregnancies complicated by pre-eclampsia, pregnancies with evidence of umbilical placental vascular disease, and pregnancies with both complications compared with the normal pregnancy (*Wang et al., 2000*).

The maternal syndrome of pre-eclampsia and the fetal syndrome of intrauterine growth restriction have been associated with vascular disease in the maternal uteroplacental and fetal umbilical placental circulations (*Trudinger and Cook, 1990*).

Placental infarction or abruption, recurrent pregnancy loss and pre-eclampsia are thought to arise due to defects within the placental vascular bed. Deficiencies of vitamin B12 and folate, or other abnormalities within the methionine-homocysteine pathway have been implicated in the development of such placental diseases (*Ray and Lasken, 1999*).

Disturbances in homocysteine metabolism have also been reported as risk factor for early pregnancy loss and for other congenital birth defect. However, besides embryonic or foetal consequences, hyperhomocysteinaemia has also described as a cause of maternal obstetric complications such as pre-eclampsia. This showed the role of hyperhomocysteinaemia in human reproduction (*Nelen, 2002*).

Women who develop severe pre-eclampsia have higher plasma homocysteine levels in early pregnancy than women who remain normotensive throughout pregnancy and elevated plasma homocysteine level in early pregnancy can increase the risk of developing severe pre-eclampsia by almost three folds (*Cotter et al., 2001*).

AIM OF THE WORK

The aim of this study is to assess serum level of homocysteine in carefully diagnosed women with mild pre-eclampsia, eclampsia and to find a relation between homocysteine level and pre-eclampsia and eclampsia.