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Pregnancy is a curious normal physiological condition where no one can foretell about its outcome. So every pregnancy is termed at risk, but there are several factors which highlight this risk and are termed as high risk pregnancy.

Toxemia of pregnancy is one of these obstetrical conditions in which there is increased perinatal morbidity and mortality of both mother and foetus. It is a threat to the well being of the mother and the unborn child.

Various obstetrical studies have documented the vascular, haematological and biochemical abnormalities in the pregnant mother with severe hypertension but the effect of this disease process on the foetus and neonates is incompletely characterized. Characterization of the neonatal consequences of maternal hypertension is complicated by the administration of antihypertensive and anti-epileptic medications to the mother. Thus the foetal outcome may be influenced by both the maternal disease and pharmacological intervention.

In toxemia of pregnancy, intense vasospasm of all the vessels occur particularly kidney and uterus, leading to hypertension and reduced uteroplacental perfusion, which is proved by observing the clearance rate of dihydro-isoandrosterone and clearance rate of radio sodium from uterine muscle. Clearance rate of
these substances decreased markedly in these patients indicating reduced uteroplacental perfusion. This reduced uteroplacental perfusion results in poor placenta tion, placental infarction, small placenta and retroplacental haemorrhage. Placental dysfunction due to toxemia of pregnancy was noted by Schule et al (1971) and played an important role in the etiology of intrauterine growth retardation.

Foetal villous trophoblast are remarkably efficient in extracting or sequestrating essential nutrients from the maternal circulation and foetus is said to be a demanding but efficient parasite, which cannot survive without oxygen. So nature has provided the foetus with the unique potential of sustaining the life in anoxic conditions in the form of foetal Hb, active erythropoiesis, increased erythropoietin level and efficient placental circulation, therefore newborn suffering from chronic anoxia in foetal life as in the toxemia of pregnancy show increased Hb%, foetal Hb% and increased number of R.B.C.. There is no controversy about compromised uteroplacental flow in toxemia of pregnancy leading to intrauterine asphyxia to newborn but whether this intrauterine hypoxia results in reduced leucocyte count and thrombocytopenia is controversial.

Brazy et al (1982) noted neutropenia, leucopenia and thrombocytopenia in infants of toxemic mother
while Sibai et al (1983) concluded that incidence of abnormal lab findings in study and control groups were similar. The frequent occurrence of abnormal lab findings in control premature infants suggest that a factor other than eclampsia (probably sepsis, hypoxia, acidosis) might be responsible for these abnormalities.

Jone B Brazy (1982) and Sibai et al (1983) noticed that newborns of hypertensive mothers had a significantly higher incidence of somatic growth retardation, microcephaly, low birth weight and prematurity. It is thought that the compromised placental perfusion from uterus vasospasm is almost certainly a major culprit in the genesis of increased perinatal morbidity and mortality associated with pregnancy induced hypertension.

Intrauterine growth retardation was present in premature infants only. Moreover, intrauterine growth retardation was symmetrical only, hence there is no head sparing when eclampsia develops early in the 3rd trimester. They concluded that these infants of eclamptic mother are at increased risk for prematurity, intrauterine growth retardation and perinatal asphyxia. Most of the immediate neonatal complications are related to prematurity and growth retardation.

Most of the world literature available is related to the managed cases of pre-eclampsia and eclampsia so we cannot decide whether these effects are
due to disease per se or due to antihypertensive drugs. On the other hand our study was planned in Bundelkhand, where more often no antenatal care is taken by pregnant mothers and unmanaged cases of toxemia land up in obstetrics unit. Moreover in most of the cases, patients were put on conservative therapy because of unavailability of anti-hypertensive therapy in form of hydralazine or magnesium sulphate, hence we have not considered these drugs as a cause of the above noted in the neonates.

Present study was undertaken with the hope and aim that it will throw some more light regarding the controversy of effect of toxemia of pregnancy on foetal outcome in our present circumstances.