INTRODUCTION
INTRODUCTION

Pregnancy is a physiological state which brings significant changes in metabolic process. Metabolism of carbohydrate, protein and fat are altered in presence of placental hormones which brings about significant changes in serum total cholesterol and serum triglyceride levels. It has been observed that increase in serum total cholesterol and serum triglycerides occurs steadily till term and then abruptly falls after delivery (Boyd, 1934; Dieckmann & Wegner, 1934; Waston, 1957). This denotes that the placenta is the principal organ responsible for the elevated Serum Total Cholesterol (STC) and Serum Triglyceride (STG) during pregnancy. It is also true that the levels of hormones which are affected during pregnancy also changes with the expulsion of placenta. Therefore, these hormones are indirectly responsible for the altered lipid profile during pregnancy (Berzin & Von Studintz, 1957; Indu Bala et al, 1983). It is known that high concentration of many of the sex steroids occurs as normal pregnancy advances. These sex hormones are necessary for maintenance and continuation of pregnancy. Since cholesterol is the precursor of these sex hormones part played by lipid metabolism in pregnancy become more intriguing.
Among the sex hormones regulating serum lipid levels, important are oestrogen, progesteron, human placental lactogen and human chorionic gonadotrophin. During normal pregnancy plasma progesterone increases to about 25 ng/ml 9 weeks after ovulation which is about twice that of non-pregnant cycle and remains relatively constant until about 10 weeks of gestation when placental secretions taken over. At about 12 weeks of pregnancy plasma level of progesterone gradually increases from a mean of 25 ng/ml to about 80 ng/dl at 28 weeks of gestation. Thereafter there is steeper increase in plasma levels to the concentration of about 180 ng/ml with the level being relatively constant during the last 4-6 weeks of gestation. Similarly, oestrogen concentration also increases till term (Klopper and Billiwick, 1963; Beisher et al, 1969). Oestriol level in the plasma at 30 weeks is 200 n mol/L and 660-700 n mol/L at 40 weeks. Urinary oestriol rises progressively during pregnancy from 5.59 mg/24 hrs at 20 weeks to 17.5 mg/24 hrs at 32 weeks and 33 mg/24 hrs at term and if the level is less than 12 mg/24 hours during later months of pregnancy it suggests serious foetal compromise in utero.

It has also been shown from various studies that in 50% of cases showing sustained low level of oestrogen there is evidence of growth retardation. So normal values are necessary for normal pregnancy outcome.
Unfortunately some of the pregnancies does not continue to term and results is abortion and even if it continue, the foetal growth is not satisfactory and there is restriction of foetal growth, resulting in intra-uterine growth retardation (IUGR). In these cases alteration in the hormones levels may be responsible for these adverse outcome and as the precursor for all these hormones is cholesterol it becomes very important to analyse the changes in the lipid parameters to know the outcome of pregnancy and in these cases there may be alteration in lipid profile.

Cholesterol is an important component of cell membrane. It is necessary for cell division so it is important for synthesis of structural component of foetus and the elevation of all lipid plasma carrier during pregnancy correlates well with the increase in the fetal caloric demand. Lipid for foetal growth and development are transferred across the foetal membrane or synthesized in the placenta. Triglycerides and fatty acids are directly transported from the mother to foetus in early pregnancy but probably are synthesized in foetus later in pregnancy. Cholesterol is capable of direct transfer from mother to the foetus. So we can estimate maternal total serum cholesterol, serum triglyceride and other parameters to know about the development and growth of foetus.
The implantation of the conceptus, the support of embryonic development and continuation of pregnancy depends on a complex interaction and hormonal effects on hypothalamic pituitary uterine ovarian axis. When a woman presents with three or more first trimester spontaneous miscarriage, it become important to rule out a persistent or recurrent endocrinial defect, beside the other causes like cytogenic, immunological, anatomical defects and infections. The most common of endocrine defect is luteal phase defect and other causes includes hypothyroidism, hyperthyroidism and diabetes mellitus. All these affects lipid parameters in the pregnant female. While diabetes & hyperthyroidism are investigated by routine investigations. Luteal phase insufficiency is not investigated so well, so it become an important cause to be ruled out for habitual abortion. As the precursor for placental progesterone biosynthesis is maternal low density lipoprotein (LDL) cholesterol (Simpson & associates, 1954; Hellig and associates, 1970; Casey, 1992). So by estimating maternal low density lipoprotein cholesterol placental production of progesterone can be predicted which is important for maintenance of pregnancy. This study includes changes in lipid fraction brought by hormonal dysbalance causing habitual abortion.

Intra-uterine growth and its aberrations are major concern of modern obstetrics because birth weight
is the strongest indicator of perinatal mortality. The ability to reach an optimal birth weight results from the interaction between the fetal growth potential and the environment.

The birth weight depends both on gestational age and fetal growth. The importance of analysing birth weight as a function of gestational age is confirmed by the fact that within fixed gestational age strata perinatal mortality is related inversely to birth weight. Although perinatal mortality is an outcome variable that is both clinically relevant and readily ascertainable, the morbidity associated with intra-uterine growth retardation is also significant (Koops and associates, 1982).

But the problem in diagnosing and managing pregnancies with impaired fetal growth are substantial. With the development of ultrasound scanning obstetrician become capable of diagnosing growth in uterus and the term - intra uterine growth retardation is designated to indicate the fetuses with birth weight less than 10th percentile or below 2 standard deviation of the mean for that period of gestational age. Intra uterine growth retardation may be defined as a pathological decrease in the rate of fetal growth. There are many factors associated with intra uterine growth retardation. Apart from fetal causes like chromosomal abnormalities, congenital malformations and
fetal infection and placental abnormalities maternal causes are very important. Among various maternal causes, important are maternal vascular diseases like pregnancy induced hypertension, chronic hypertension and advanced diabetes mellitus, maternal nutrition, besides environment & haematological causes. All these can alter lipid profile of mother so we aim to see these alteration in lipid profile in cases of intra uterine growth retardation.