INTRODUCTION
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Various studies have been done about the hormonal changes brought about by pregnancy but still pregnancy is a metabolic curiosity. However it brings significant changes in metabolic process. It is known that high concentration of many of the sex steroids occur as normal pregnancy advances and since cholesterol is the precursor of most of these steroids, part played by lipid metabolism in pregnancy becomes more and more intriguing.

It has been observed that increase in serum total cholesterol and serum triglycerides occur steadily till term and then falls abruptly after delivery (Boyd, 1934; Dieckmann & Wegner, 1934; Waston 1957). This was also corroborated by studies previously done in our department (Arora and Indu, 1985; Arora and Vinita, 1986). This denotes that placenta is the principal organ responsible for the elevated serum total cholesterol (STC) and serum Triglyceride (STG) during pregnancy. The incidence of atherosclerosis is less in Indian women even in repeated pregnancies, where the cholestrol level is elevated. This is probably because the elevated Oestrogen might be preventing the deposition of cholestrol in the intima of arteries and veins (Chaturvedi et al., 1978). These metabolic changes are rather exaggerated in Toxaemia of pregnancy (a syndrome complex characterise by hypertension to the extent of 140 mm hg or more with oedema or proteinuria or both induced by pregnancy usually after 20th week of gestation). The toxaemia of pregnancy is an inadequate term which still frequently used to cover a condition peculiar to pregnancy whose aetiology is unknown but was formerly attributed to the action of a hypothetical toxin since it is no longer believed that this disorder is caused by toxin, a better term in pre-eclampsia and is accompanied by fetal and maternal hypercholesterolaemia (Indu Bala, 1983; lall and Sinha, 1983). Ghosh reported that the value of total cholesctrol rises significantly in pre-eclamptic patients. Brat Vold and De Averez (1961) noted some increase in serum total cholesterol in pre-eclampsia as compared with values in normal pregnancy. The difference however was not thought to be significant. On the contrary Arsoa and Kretowicz (1963) reported that such difference was statistically significant. The serum cholestrol level falls gradually in the post partum period both in toxaemia and in normal pregnancy. However in toxaemia cases such levels do not return to normal as quickly as it does in the cases of normal pregnancy. (Lall and Sinha).

Cholesterol is necessary for cell division so it is important for synthesis of structural component of foetus and elevation of all lipid plasma carrier during pregnancy correlates well with the increase in
the fetal caloric demand. 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. Placental production of progesterone can be estimated which is important for maintenance of pregnancy and also as the precursor for placental progesterone biosynthesis is maternal low density lipoprotein (LDL) cholesterol (Simpson & associates, 1954; Hellig & associates, 1970; Casey, 1992) so by estimating maternal LDL.

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. Among the sex hormones important are oestrogen, progesterone, human placental lactogen and human chronic Gonadotrophin. During normal pregnancy plasma progesterone increases to about 25 ng/ml 9 weeks after ovulation and remain relatively constant until about 10 weeks of gestation when placental secretions taken over. Plasma level of progesterone rises rapidly from 28th week onwards and reaches to approximately 180 ng/ml with the level being relatively constant during the last 4-6 weeks of gestation. Similarly oestrogen concentration also increases till term (Kloppler and Billwiicz, 1963; Beisher et al, 1969) urinary oestrial level also rises progressively during pregnancy from 5.5 g mg / 24 hr at 20 weeks to about 33 mg / 24 hrs at term and if the level is less than 12 mg /24 hrs. during later months, it suggests serious foetal compromise in utero. It has also been shown from various studies that in 50% cases showing sustained low level of oestrogen there is evidence of growth retardation and also as the precursor for placental progesterone is maternal LDL c (Simpson & Associates, 1954, Hellig & Associates, 1970, Casey, 1992) So by estimating maternal LDL placental production of progesterone can be estimated which is important for maintenance of pregnancy Intrauterine growth and its aberrations are major concern of modern obstetrics because birth weight is the strongest indicator of perinatal mortality.

The birth weight depends upon both the gestational age and foetal growth. Although perinatal mortality is an outcome variable that is both clinically relevant and readily ascertainable, the morbidity associated with intrauterine growth retardation (IUGR) is also significant (Koops and associates, 1982).

The term intrauterine growth retardation is designated to indicate the fetus with birth weight less than 10th percentile or below 2 standard deviation of the mean for that period of gestational age. There are many factors associated with intrauterine growth retardation. Apart from fetal causes like chromosomal
abnormalities, congenital malformations, fetal infections and placental abnormalities, maternal causes are also very important. Among various maternal causes, important are maternal vascular diseases like pregnancy induced hypertension, chronic hypertension and advanced diabetes Mellitus, nutrition besides environment & Haematological causes.

Although studies have been done on lipid lipoproteins profile in female sof various age groups and their relationship with different hormones, little attention has been given to its relationship with pregnancies complicated by toxaemia and intrauterine growth retardation.

Before studying the changes in lipoprotein profile it is necessary to have some background knowledge about lipoprotein. Lipoproteins are of mainly five types - High density lipoprotein (HDL), Low density lipoprotein (LDL), very low density lipoprotein (VLDL), serum triglycerides (STG) and total serum cholestrol (STC). Lipids remain in plasma in complex form other than chylomicrons. Triglyceride transport is the major recognised function of serum lipoproteins. VLDL tells the function rate of hepatic triglyceride synthesis which in turn depends upon :-

- Rate of free fatty acid uptake from blood
- Fatty acid synthesis from glucose
- Extent of fatty acid oxidation
- Conversion of fatty acid to triglycerides.

VLDL transports triglycerides to muscle and other tissue as a source of energy. Ingestion of glucose decreases VLDL whereas increased level of VLDL has been related with obesity, high carbohydrate or low fat diet, ingestion of food in need and diminished level of HDL and LDL.

LDL is mainly derived from breakdown of VLDL in the circulation. LDL is increased by fat saturated fatty acids and cholestrol. function is uncertain.

HDL is synthesized in the liver. HDL lipid serve as major substrate for lecithin cholestrol acyl transferase. HDL also function to transport cholestrol from peripheral tissues to the liver. So with all these informations we decided to study the changes in lipoprotein profile in pregnancies complicated by PIH(pre-eclampsia and eclampsia) and IUGR during their antepartum, intrapartum and postpartum period in a group of females from Bundelkhand region.