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Comparative Studies of Serum Uric Acid and Lipid Profile in Pregnant and Non Pregnant Women Attending Vasundhara Hospital and Fertility Research Centre Jodhpur City, Rajasthan, India

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Abstract: Changes in lipid profile during pregnancy has being normally observed and these changes mostly differ according to trimester. An increased maternal morbidity and mortality was recorded in history due to severe or uncontrolled Gestational hypertension (GH) and leads to more than 14% of early or preterm births. During pregnancy, its documented that Dyslipidaemia hyperuricemia are strongly believed to have pathophysiological role. A total of 150 women among which 100 pregnant women and 50 non pregnant women have participated in the study. Serum T. cholesterol (TC), triacylglyceride (TG), high density lipoprotein (HDL) and low density lipoprotein (LDL) were tested and estimated in the study. It was found in this study that Total cholesterol, triglycerides and high density lipoprotein in the three trimesters when compared to the control subjects were found to be high ($p< 0.05$). The change inlow density lipoprotein was not significantly high ($p> 0.05$) in the first trimester but becamesignificant ($p< 0.05$) in the second and third trimester when compared with the control. Comparison between first, second and third trimesters showed that TC, TG and LDL in the 2nd and 3rd trimesters were significantly higher than in the 1st trimester. Serum uric acid Serum lipid profile were analysed and estimated by enzymatic methods. The serum uric acid showed was found to be strongly correlated with systolic blood pressure, TGL ($r = 0.293$, $p < 0.01$;) TGL/HDL ($r = 0.549$, $p < 0.001$; $r = 0.434$, $p < 0.001$ respectively) and a negative correlation with HDL/VLDL ($r = -0.423$, $p < 0.001$). Triglyceride showed significant positive relation with diastolic blood pressure ($r = 323$, $p = 0.001$); uric acid ($r = 0.553$, $p < 0.001$), TGL/HDL ($r = 0.759$, $p < 0.001$) and negative relation with HDL/VLDL ($r=0.691$, $p < 0.001$). In the early 2nd trimester, estimation of lipid profile and serum uric acid can be advisable to prevent complications of Gestational Hypertension.

Keywords: Pregnancy, Lipid Uric acid.

INTRODUCTION

Pregnancy is accompanied by significant variations in maternal lipid metabolism. It is associated with significant changes in the functions of the normal liver and understanding these changes is essential to a proper clinical evaluation of liver abnormalities during pregnancy (Boyd, E, 1934; Stock, M. J. and Metcalfe, J, 1994). Pregnancy greatly increases demand for metabolic fuels that are needed for growth and development of the foetus and its support structures. The total gestation related energy cost has been estimated at approximately 83000 kcal. The major change in energy expenditure and in the accumulation of fat occurs at different times during pregnancy (Deepak P. and Digisha P, 2011). Physiologically, the mother becomes almost a new person during the period of pregnancy. Profound local and systemic changes in maternal physiology are initiated by conception and continued throughout pregnancy, it is obvious that the hormonal interplay immediately after fertilization makes the hormones to be the initial primary changes in pregnancy, the human chorionic gonadotropin (hCG) secreted by the corpus luteum of pregnant women, progesterone, relaxin and human chorionic somatomammotropin (hCS) secreted by syncytiotrophoblast all increase to maintain the possible changes in pregnancy (Kortenoever M., 1960). The advancement of pregnancy is accompanied by the extra demand of energy. As pregnancy progresses, a well-integrated metabolic shift occurs to ensure an adequate supply of nutrients to a constantly feeding foetus from an intermittently fasting and feeding mother. During early pregnancy, maternal metabolic environment is modified by a rise in serum levels of estrogen and progesterone, pancreatic beta-cell hyperplasia occurs and there is an increase in the secretion of insulin, (Kalkhoff RK., 1982). In this stage, there is increased body fat accumulation associated with both hyperphagia and increased lipogenesis while in late pregnancy there is an accelerated breakdown of fat depots, which plays an important role in foetal development (Herrera E., 2002). Increase in blood cholesterol levels impair insulin-mediated entry of glucose into the cells thus resulting in hyperglycemia of variable severity. (Alwan. *et al*; 2009, Andersson. *et al*.; 1993). Hyperinsulinemia leads to an increase in peripheral glucose utilization, a decline in fasting

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plasma glucose levels, increased tissue storage of glycogen, increased storage of fats and decreased lipolysis. Freinkel N., (1964) was the first to describe the maternal metabolic changes of late Pregnancy as ―accelerated starvation, when food is unavailable and ―facilitated anabolism‖ when food is ingested. Maternal fuel adjustments during late pregnancy include a sparing of glucose (for the foetus) and an increased concentration of fatty acids in plasma. It is known that high concentrations of many of the steroids occur as normal pregnancy advances. Since cholesterol is the source of most of the steroids found in increased amounts in the circulation of normal pregnant women, the part played by lipid metabolism in pregnancy, becomes all the more intriguing, as cholesterol is a major factor for the development of atherosclerosis. Chauffard. *et al*., (1911) undertook the first chemical study of blood lipids during pregnancy and suggested that an increase occurs in the cholesterol level. With the development of more modern techniques various studies observed an increase in various lipid fractions, though the increase was neither consistent in time of appearance nor proportion of changes in various fractions.

MATERIAL AND METHODS

This prospective observational clinical study was conducted in Vasundhara Hospital and Fertility Research Centre Jodhpur City, Rajasthan, India, between April 2016 to July 2016 after obtaining the Ethical Committee clearance.

Sample Collection

All subjects under study were made sure to fast overnight at for not less than 8 hrs. Approximately, 5ml of fasting blood sample, under aseptic condition, was collected from pregnant and non-pregnant women attending Vasundhara Hospital and Fertility Research Centre Jodhpur into a plain tube. The blood sample undergone centrifugation at 4000rpm for 5 minutes and the serum is collected and stored at 4°C for estimation of serum uric acid and lipid profile. Serum Triglycerides (TG), Total cholesterol (TC) and HDL cholesterol (HDL-C) and Uric Acid were analyzed by enzymatic methods with the help of Glaxo kits on ERBA Chem-5 semi-auto analyzer. A total of 150 women were enrolled and the study groups are control group: comprised 50 non-pregnant, healthy women having normal menstrual function with no evident hormonal deficiency. Study group: Comprised 100 pregnant women and their gestational ages.

Parameters Assessed

The blood was drawn into plain tubes and subjected to estimation of lipid profile by semiautoanalyser (Serum Cholesterol by modified method of (Roeschlau. *et al*., 1974), Serum triglyceride using modified method by (Mcgowan. *et al*., 1982), HDL – C using modified method by (Bursteim. *et al*., 1970) and the VLDL and LDL–C was calculated by Friedewald‘s formula.

Serum Uric Acid was assessed using semi-autoanalyser (enzymatic method).

Lipid Profile

Total Cholesterol

(CHOD-PAP) into three (3) test tubes labelled blank, standard and test, 1000 µl of reagent was added to each test tube. Distilled water (10µl), standard solution (10 µl) and serum (10 µl) were added respectively to each test tube. Mixed and incubated for 5 minutes at 37°C. Absorbance of each mixture was taking and the absorbance of test (sample) was against reagent blank at 500 nm (normal value: 100 – 250mg/dl).

Triglycerides (TG)

Into three (3) test tubes labelled test, standard and blank, 1000µl of reagent was added into the three test tubes. 10µl of sample, standard and distilled water were added in the test tubes respectively. Mixed and incubated for 5 minutes at 37°C. Absorbances of test, standard and blank were taking at 500nm. Absorbance of test (sample) was measured against reagent blank (normal value: 60 – 150 mg/dl).

High Density Lipoproteins-Cholesterol (HDL-C) Precipitation Reaction

250µl of sample and 500µl of precipitating reagent were added to a fresh test tube. Mixed and allowed the reaction mixtures to stand for 10 minutes at room temperature and then centrifuged at 4000rpm (1800Xg) for 10 minutes to obtain the clear supernatant. And the supernatant was used to estimate the concentration of HDL-C in the sample.

Cholesterol Determination

50µl of the supernatant and 1000µl of the working reagent were also added to a fresh test tube.
Mixed well and incubated for 10 minutes at 37°C.
Absorbance were taken
Normal values: 35 – 70 mg/dl

Low Density Lipoprotein- Cholesterol (LDL-C):

LDL- cholesterol (mg/dl) = Total cholesterol – HDL-cholesterol – (TG/5)

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Normal values: 80-150 mg/dl
Estimation of Very Low Density Lipoproteins (VLDL): **VLDL** = TGL/5 or 20% of TG
Normal values: ≤ 30mg/dl

Serum Uric Acid
Three (3) test tubes were labelled test, standard and blank. 500µl was added to each test tube, 20µl of serum, standard and distilled water were added to the tubes respectively. The mixtures were incubated at 37°C for 5 minutes. Absorbance of each mixture was taken and the concentration of uric acid in the sample was determined.
Normal values: 2 – 7mg/dl

Anthropometric Data Collection Measurement of Body Mass Index
Mean values of age, blood pressure, BMI and other anthropometric parameters were measured. Body weight of the subject and control was measured in kilogram scale using Bathroom Scale Hana BR-9011 UK. The height was measure in meters using a standard stadiometer. The body Mass Index (BMI) was calculated using the formula weight/height.

Measurement of Blood Pressure
After a rest period of close to 30 minutes in the hospital, then both the systolic and diastolic pressures were measured in each subject using auscultatory method on the brachial artery.

Exclusion Criteria
Patients with gestational diabetes, diabetes Mellitus, chronic gestational hypertension (hypertension arising before 20 weeks of gestation), complicated and/or multiple pregnancies, women consuming antihypertensive medication, aspirin and / or corticosteroids hepatitis B infections, human immunodeficiency virus (HIV) infection, coronary artery disease, chronic obstructive pulmonary disease (COPD) are not included in this study.

Statistical Analysis
The SPSS software was used during the analyses. Mean ± standard deviation was employed when presenting the data using Microsoft excel 2010. The student t- test was used for analyses of data and $p < 0.05$ were considered statistically significant. Using paired t test, the mean serum lipid profile and serum uric acid concentrations of the cases and controls were compared. $P < 0.05$ was the significance obtained. Lipid fractions and uric acid were correlated using Pearson's correlation coefficient, when only the test was considered.

RESULT
In this study, the TC, TG, LDL-C and VLDL-C were found to be significantly elevated in pregnant women in second and third trimester when compared with control ($p< 0.05$). It was also found that HDL-C was significantly lowered in test group compared to control ($p< 0.05$). table depicts the results as mean SD. Serum uric acid was also found to be significantly elevated in gestational hypertension ($p< 0.05$).

Table 1: Biochemical parameters as mean ± SD

Test parameters	Pregnant women (n=100)	Control (n=50)	p value
TC (mg/dl)	172.10 ± 30.16	148.1 ± 10.3	<0.05
TGL (mg/dl)	190.48 ± 37.95	106.9 ± 15.1	<0.05
HDL-C (mg/dl)	57.50 ±7.90	40.3 ± 8.9	<0.05
LDL-C (mg/dl)	76.50 ± 30.22	86.7 ± 12.9	<0.05
VLDL-C (mg/dl)	38.096 ± 7.59	29.4 ± 3.48	<0.05
UA (mg/dl)	4.928 ± 2.13	4.076 ± 0.79	<0.05

TC, TGL, HDL-C, LDL- C, VLDL-C, Uric acid,

Table 2: Represents Pearson's coefficient of determination (r) of serum uric acid with blood pressure and lipid parameters in pregnant women

Lipid parameters	(r)	p value
TC	0.145	0.149
TG	0.549	<0.0001
LDL-C	- 0.090	0.375
VLDL-C	0.561	<0.0001
HDL-C	- 0.090	0.372
Systolic blood pressure	0.291	< 0.01
Diastolic blood pressure	0.233	< 0.05

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In pregnant test group, all the parameters were correlated using Pearson's correlation (**Table2**). That showed significant positive correlation of uric acid with systolic blood pressure, TGL, and TGL/HDL ($r = 0.291$, $p < 0.01$; $r = 0.549$, $p < 0.001$; $r = 0.429$, $p < 0.001$ respectively) and significant negative correlation with HDL/VLDL ($r = -0.415$, $p < 0.001$). TGL showed significant positive correlation with diastolic blood pressure ($r = 313$, $p = 0.001$); uric acid ($r=0.$, $p < 0.001$), TGL/HDL ($r = 0.762$, $p < 0.001$) and significant negative correlation with HDL/VLDL ($r = - 0.701$, $p < 0.001$).

Table 3: showing the normal values and methods used for each parameter

Parameters	Methods	Normal values
Total cholesterol	Enzymathic method	125-250 mg/dL
Triglyceride	GPO-PAP method	35-165 mg/dL
HDL-Cholesterol	Enzymathic method	35-65 mg/dL
LDL-Cholesterol	Frieswaldformule	80-150 mg/dL
VLDL	Frieswaldformule	≤ 30mg/dl
Serum ueic acid	Enzymatic method	2 – 7mg/dl

Table 4: TC, TG, HDL AND LDL levels (Mean ± S.D in mg/dl) of pregnant women in their first, second and third trimesters and control

Parameters mg/dl	First trimester n=30	Second trimester n=40	Third trimester n=30	Control n=50
TC	5.11 ± 3.164*	8.12 ± 4.191*	1.9 ± 4.231*	148.1 ± 10.3
TG	1.21 ± 9.180*	5.34 ± 5.217*	3.26 ± 1.211*	106.9 ± 15.1
HDL-C	1.4 ± 6.45*	4.6 ± 4.44*	8.3 ± 9.47*	40.3 ± 8.9
LDL-C	± 4.82 21.4 *	.16 ± 5.1033*	6.8 ± 2.141*	86.7 ± 12.9
VLDL-C	35.536 ± 4.55	05.8 ± 696.37*	59.7 ± 932.38*	29.4 ± 3.48
Uric Acid	03.2 ± 21.4*	1.2 ± 33.4*	0.3 ± 93.4*	3.758 ± 0.75

** = Significantly different from control, n= frequency, TC= Total Cholesterol, TG=Triglycerides, HDL-C=High Density Lipoprotein Cholesterol, LDL-C=Low Density Lipoprotein Cholesterol, VLDL-C=Very Low Density Lipoprotein Cholesterol*

The result analysis shows a significant increase ($p<0.05$) in the TC, TG and HDL level during the first trimester of pregnancy when compared with control as shown in table 1. There was a significant increase ($p<0.05$) in the TC, TG, HDL and LDL levels during the second trimester of pregnancy when compared with that of the control subjects. Also, from table 1, the result shows a significant increase ($p<0.05$) in the TC, TG, HDL and LDL levels during the third trimester of pregnancy when compared with the control subjects.

Table 5: TC, TG, HDL and LDL levels (Mean ± S.D in mg/dl) of pregnant women in their first trimester as compared with second trimester

Parameter (mg/dl)	First Trimester n=30	Second Trimester n=40
TC	164.3 ± 11.5	8.12 ± 4.191**
TG	180.9 ± 21.1	5.34 ± 5.217**
HDL-C	45.6 ± 4.1	4.6 ± 4.44**
LDL-C	82.4 ± 12.4	3.16 ± 5.103**
VLDL-C	35.536 ± 4.55	05.8 ± 696.37**
Uric Acid	4.21 ± 2.03	1.2 ± 33.4**

***Significantly different from 1st trimester, n= frequency, TC= Total Cholesterol, TG= Triglycerides, HDL= Highdensity lipoprotein cholesterol, LDL= Low density lipoprotein cholesterol, VLDL = Very Low Density Lipoprotein*

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Table 6: TC, TG, HDL and LDL levels (Mean ± S.D in mg/dl) of pregnant women in their first trimester as compared with third trimester

Parameters mg/dl	First trimester n=30	Third trimester n=30
TC	164.3 ± 11.5	1.9 ± 4.231***
TG	180.9 ± 21.1	3.26 ± 1.211***
HDL-C	45.6 ± 4.1	8.3 ± 9.47***
LDL-C	82.4 ± 12.4	6.8 ± 2.141***
VLDL-C	35.536 ± 4.55	59.7 ± 932.38***
Uric Acid	4.21 ± 2.03	0.3 ± 93.4***

****Significantly different from 1st trimester, n= frequency, TC= Total Cholesterol, TG= Triglycerides, HDL=High density lipoprotein cholesterol, LDL= Low density lipoprotein cholesterol, VLDL = Very Low Density Lipoprotein*

Table 7: TC, TG, HDL and LDL levels (Mean ± S.D in mg/dl) of pregnant women in their second trimester as compared with third trimester

Parameters mg/dl	Second Trimester n=40	Third trimester n=30
TC	191.4 ± 12.8	1.9 ± 4.231****
TG	217.5 ± 34.5	3.26 ± 1.211****
HDL-C	44.4 ± 6.4	8.3 ± 9.47****
LDL-C	103.5 ± 16.3	6.8 ± 2.141****
VLDL-C	37.696 ± 8.05	59.7 ± 932.38****
Uric Acid	4.33 ± 2.1	0.3 ± 93.4****

**** Significantly different from 2nd trimester, n= frequency, TC= Total Cholesterol, TG= Triglycerides, HDL=*
High density lipoprotein cholesterol, LDL= Low density lipoprotein cholesterol, VLDL= Very Low Density Lipoprotein

DISCUSSION

In our sample, in the case of triglycerides and VLDL, all lipid sub-fractions except for HDL were significantly elevated (p value < 0.05) and more than that of non-pregnant women. Our research findings are consistent with most preceding studies (Enquobahrie. *et al*., 2004, Cekmen. *et al*., 2003). The hormonal imbalance is a prime factor for the aetiopathogenesis of high lipid profile in pregnant women compared to non-pregnant women which is a common cause of pregnancy induced hypertension (PIH) or gestational hypertension (GH) among pregnant women. Gestational hypertension is a hyperestrogenemic condition (Jayanta. *et al*., 2006This finding also holds true in our research. In this study, the concentration of serum total cholesterol, serum triglyceride, cholesterol and low density lipoprotein cholesterol throughout normal pregnant women increased with a rise in gestational age, while HDL dropped slightly in the 2nd trimester with serum triglyceride concentration showing a very significant increase, while in the third trimester of normal pregnancy than in the non-pregnant women, the mean value being raised to two folds. Similar findings were recorded in the studies of Fahraeus. *et al* (1995) Jimenez. *et al.*, (1988) and Potter and Nestel (1979). Oestrogen is the main modulator of this hypertriglyceridemia, since pregnancy is associated with hyperoestrogenaemia. Oestrogen causes endogenous triglyceride hepatic biosynthesis, which is being pursued by VLDL. The hyperinsulinism observed during pregnancy can modulate this process (Adegke. *et al*., 2003, Glueck. *et al*., 1975).

This study also showed that the test subjects ' total cholesterol, high density lipoprotein, and triglyceride levels in the first trimester were higher than those of the control subjects. This is in line with Klovich M's. Hallman B (1979), In which they found that zygote formation in the uterine wall occurs during the first trimester of pregnancy. It accounts for high First Trimester cholesterol and triglyceride levels. In the second trimester, total

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cholesterol, triglyceride, HDL and LDL of the test subjects were found to be higher than those of the control subjects. That is consistent with the results of Wald and Guckle, (1988), who observed that Increased maternal lipid profile reflects the maternal transition from carbohydrate to fat metabolism, which is an alternative route to generating energy due to high energy demand. In the third trimester the total cholesterol, triglyceride, HDL, and LDL levels of the test subjects were higher than those of the control subjects. This is in line with results of Russell and Copper, (1989), in which they reported that there is development of foetal organ in the third trimester. This study also showed a significant increase ($p<0.05$) in total cholesterol, triglyceride, and LDL levels compared to the second trimester during the first trimester of pregnancy. This is in accordance with Munoz. *et al.,* (1995), who, after 25th week of gestation, gradually increased total cholesterol, triglyceride and LDL during pregnancy with significantly higher values. We also observed that HDL was slightly lower in the second trimester relative to the first trimester of pregnancy, in line with Desoye. *et al.,* (1987) research, in which they observed a decline in HDL after weeks 22 to 24 which coincided with the onset of increased insulin resistance and increased plasma insulin concentration. This study also showed significant increases in total cholesterol, triglyceride, HDL, and LDL levels in the third trimester relative to the first trimester of pregnancy ($p<0.05$). This is in line with Desoye. *et al.,* (1987) analysis, in which they found that LDL levels peaked at approximately week 36, HDL 2 and 3 levels peaked at approximately 28 weeks and remained unchanged till delivery Decreased utero-placental blood flow, the major pathophysiological phenomenon in gestational hypertension, leads to dysfunction of fetal adrenal glands in the production of Dehydroepiandrosterone sulphate (DHEAS). DHEAS is the major source of oestrogen during pregnancy, i.e. 90 percent of oestrogen in maternal circulation is from fetal DHEAS which is transformed into placenta estriol (Gratacos. *et al*., 2003). Hypoestrogenemia also contributes to decreased expression of VLDL / apo E receptors resulting in reduced transportation of VLDL to the fetal compartment and hypertriglyceridemia in the mother. More LDL taken up by the foetus for DHEA synthesis is reduced due to reduced foeto-placental perfusion that leads to increased LDL. Triglyceride is a significant biomarker of cardiovascular disease (CVD) risk because of its association with atherogenic remnant particles and apo CIII (Michael. *et al*., 2011). The elevated triglycerides in gestational hypertension result in increased atherogenic small dense LDL and reduced HDL rates. This can be due to increased TGL exchange between LDL and HDL (Vanessa AR., 2006). The changes in lipoprotein fractions observed in this research were consistent with changes found in coronary artery disease (Kaaja. *et al* 1995, sattar. *et al* 1997). In our research, elevated triglyceride dyslipidemia and reduced HDL were close to that of many other studies (Vidyabati. *et al*., 2010, Adiga and Adiga, 2010, Shalini. *et al*., 2011). However, uric acid level was also significantly elevated in in pregnant women with high blood pressure which is in consistent with some workers (Laughon. *et al* 2009). The currently favored concept is that increased circulating uric acid is secondary to reduced renal urate clearance, as can be seen with hypovolemia. Elevated serum uric acid in in pregnant women with PIH is associated with poor perinatal outcomes including small gestational age (SGA) for infants and preterm birth (PTB) (Redman. *et al*., 1976, Roberts. *et al*., 2005). Uric acid is the end product of purine catabolism catalyzed by the enzyme xanthine oxidase/dehydrogenase. The oxidase form of the enzyme producing uric acid and superoxide will be increased proportionally by hypoxia. Therefore, increased uric acid production occurs in a setting of hypoxia, local acidosis, or increased tissue breakdown or with reduced renal function and can increase oxidative stress—all of which would indicate more severe pre-eclampsia (Roberts. *et al*., 2005, Anil. *et al*., 20011). Uric acid is also found to be associated with carotid atherosclerosis and its increase is an independent risk factor for cardiovascular diseases that mediates altered vascular function and inflammation. The elevation could be due to the difference between radicals free of oxygen and NO. Since uric acid has antioxidant activity in the serum too The level may rise as a compensatory mechanism for counteracting increased oxidative stress under metabolic or atherosclerosis conditions (Nieto. *et al*., 2000; Aparna. *et al*., 2012, Hansel. *et al*., 2004). Dyslipidemia is apparent during the first and second trimesters, often exceeding pre-eclampsia clinical symptoms. At the earliest test (20 weeks), the HDL-C declines gestation in women that later develop the syndrome and implicating dyslipidemia in the pathophysiology (Frauke. *et al*., 2007, Chappell. *et al*., 2002). Hyperuricemia is associated with poor perinatal results, and atherosclerosis as well. Both

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dyslipidemia (elevated triglycerides and decreased HDL) and elevated uric acid predict atherosclerosis and are also known to play a pathophysiological role in the pre-eclampsia clinical manifestations seen in this study as well.

CONCLUSION

In our study, it was found out that Total Cholesterol, TG, HDL, LDL and VLDL were significantly increased compared to control. And among the parameters HDL-C was found to have the lowest values. The same applied to Uric Acid which was also significantly increased. So, it was concluded that lipid profile and serum uric acid measurements at different trimesters of pregnancy can be suggested as cost-effective markers that may help in the prevention of pregnancy complications like gestational hypertension, pre-eclampsia/eclampsia, intrauterine growth retardation, HELLP syndrome, future cardiovascular risk of the mother and stroke.

RECOMMENDATIONS

In this present study, the outcomes of the pregnancies are not studied, hence limited only to period of pregnancy. Basically, it is recommended that:

Similar studies on large number of groups with results of the outcome of the pregnancies should be carried out

Similar study should be carried of pregnant women according to their ages and gestational ages.

Similar studies should be conductedon the basis of diet (either vegetarian or non-vegetarian).

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