

Evaluation of Serum Uric Acid and Lipid Profile In Gestational Hypertension, A Cross Sectional Study in Burdwan Medical College and Hospital

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Abstract

Aim: Study of lipid profile and uric acid in gestational hypertension and with normotensive pregnant mothers. **Material & methods:** A cross-sectional study on gestational hypertension and normotensive pregnant mothers as a control group attending at out-patient department in Burdwan Medical College & Hospital. **Results:** Serum uric acid was significantly elevated in study group & total Cholesterol, Low density lipoprotein (LDL), Very low density lipoprotein (VLDL), Triglycerides and High density lipoprotein (HDL) levels were significantly lower in study groups in comparison to control groups. The mean serum lipid and uric acid concentration of the study and control groups were compared using paired t test. Significance was set at $P < 0.05$. Correlations between various lipid fractions, uric acid and fasting blood sugar were made using Pearson's correlation coefficient, considering study & control groups.

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I. Introduction

The deadly triad of complications in pregnancy includes hypertention, haemorrhage and infection contribute greatly to the maternal morbidity and mortality¹ Hypertensive disorders complicate 5-10% of all pregnancies. Pregnancy induced hypertension (PIH) remains the major cause of both maternal and foetal morbidity and mortality. While dealing with PIH women, the obstetricians have to be very careful to diagnose and properly manage the patients to prevent further progression of PIH disorders and complications. In India the incidence of preeclampsia is reported to be 8-10% of the pregnancies² The incidence in primigravidae is about 10% and in multigravidae about 5%³. It causes IUGR leading to low birth weights. Low birth weight child is prone to suffer from diabetes, hypertension, and coronary vascular disorders in their later life⁴.

Working group of the National High Blood Pressure Education Program (2000) describes four types of hypertensive disorders: 1) Gestational hypertension (formerly Pregnancy Induced Hypertension), 2) Preeclampsia and Eclampsia syndrome, 3) Preeclampsia syndrome superimposed on chronic hypertension and 4) Chronic hypertension¹.

According to the norms of American college of obstetrics and Gynaecologists, the diagnostic criteria for gestational hypertension [Pregnancy Induced Hypertension (PIH)] is :-

- (1) Systolic Blood pressure ≥ 140 mm/Hg
- (2) Diastolic Blood pressure ≥ 90 mm/Hg Or
- (3) Increase of ≥ 30 mm/Hg in Systolic pressure Or
- (4) Increase of ≥ 15 mm/Hg in Diastolic pressure, in a previously normotensive woman⁵. Placental implantation with abnormal trophoblastic invasion of uterine vessels and immunological maladaptive tolerance between maternal, placental and fetal tissues are some of the etiological factors for the hypertensive disorders. Placental ischemia is the main factor responsible for the disease process. This may be due to various factors like vasospasm, vascular endothelial cell activation and dysfunction, increased presser responses, and decreased prostacyclin:Thromboxane A2 ratio¹.

It is suggested that the disease is present till the placenta is present; once it is removed the situation improved⁵. This leads to delivery of premature babies. Endothelial activation and altered platelet counts are the markers of the disease progression before the development of signs and symptoms⁵. Hypoestrogenemia, predominance of smaller and denser serum LDL and significant concentration of soluble vascular cell adhesion molecule-1 are important contributors for endothelial dysfunction in gestational hypertension⁶. In lipid mediated endothelial dysfunction an essential step is oxidation of low-density lipoprotein⁷⁻⁹. The atherogenesis itself may be initiated by hypertriglyceridemia. Hypertriglyceridemia leads to elevated levels of small dense LDL particles

which is atherogenic. Marked dyslipidaemia occurs with gestational hypertension. In many ways this dyslipidaemia represents an accentuation of the lipid changes noted in normal pregnancy. Mean plasma triglyceride and Free Fatty Acid (FFA) concentrations increase about 2-fold on average in women with gestational hypertension relative to women with uncomplicated Pregnancy¹⁰.

Elevated uric acid is another component of the preeclampsia syndrome that was recognized many years ago. It is one of the most consistent and earliest detectable changes in preeclampsia and has been cited as a better predictor of fetal risk than blood pressure¹¹.

There are several potential origins for uric acid in preeclampsia; these are - abnormal renal function, increased tissue breakdown, acidosis and increased activity of the enzyme xanthine oxidase/dehydrogenase^[21].

However, despite hyperuricemia antedating other clinical findings of preeclampsia, it has historically been ascribed to impaired renal function. Outside of pregnancy, hyperuricemia is considered a risk factor for hypertension, cardiovascular and renal disease^[34]. This evidence, as well as the observation that severity of preeclampsia increases with increasing uric acid, questions whether uric acid may play a role in the pathophysiology of preeclampsia. While this concept is largely unstudied, we expand upon ideas forwarded by Kang^[35] to share in the hypothesis that an elevated concentration of uric acid in preeclamptic women is not simply a marker of disease severity but rather contributes directly to the pathogenesis of the disorder.

We hypothesize that uric acid acts adversely upon both the placenta and maternal vasculature. In this presentation we will discuss the potential effects of uric acid on placental development and placental function. Further, we will examine the potential negative impact of uric acid on the maternal cardiovascular system with specific emphasis on its effects upon endothelial function and repair, inflammation and vascular tone.

Aims & Objective

Assessments of lipid profile & uric acid in gestational hypertension with normal control. Measurement of serum total cholesterol (TC), triglycerides (TG), high density lipoprotein (HDL), low density lipoprotein (LDL), very low density lipoprotein (VLDL) cholesterol by enzymatic methods in hypertensive and normotensive mothers. Measurement of serum uric acid by enzymatic methods & plasma fasting blood sugar of both hypertensive and normotensive pregnant mothers.

II. Materials & Methods

The study has been done in Burdwan Medical College in the Department of Obstetrics & Gynecology attending as out-patient with pregnancy during February 2015 to July 2016

A cross-sectional study on Gestational Hypertension patients and normotensive pregnant mothers as a control group attending at out-patient department in Burdwan Medical College & Hospital.

INCLUSION AND EXCLUSION CRITERIA:

The inclusion criteria are:

- The patients must have systolic blood pressure >140mm of Hg and Diastolic Blood Pressure >90 mm of Hg.
- Age between 18 to 45 years old.
- Gestational age >20 weeks and <37weeks of gestations are included in my study group who has newly onset hypertension diagnosed during pregnancy.

The exclusion criteria-

- Patients with preexisting hypertension, renal, cardiovascular disease and diabetics.
- Patients with preexisting hyperuricemia.
- Preexisting hyperlipidemia.
- Patients with gestational age <20 weeks and >37 weeks are excluded from my study groups.

The patients with preexisting hypertension, hyperuricemia, hyperlipidemia and hyperglycemia are excluded by taking history and by previous medical records

SAMPLE SIZE:

50 study group and 50 of control group.

STUDY DESIGN:

Hospital based cross sectional study

PARAMETERS TO BE STUDIED:

Serum lipid profile-Total Cholesterol, Triglycerides, HDL, LDL and VLDL.

- Serum Uric Acid.
- Plasma glucose- Fasting Blood Sugar (FBS).

STUDY TOOLS:

- Auto analyzer XL-600
- Centrifuge machine REMI R-8C.
- Routine laboratory glass wares and PVC Wares.
- Sphygmomanometer, □Stethoscope.
- Plain blood collection vials, oxalo-fluride vials, disposable syringes.
- Rubber tourniquet.
- Spirit and cotton swabs.

PLAN FOR ANALYSIS:

Data entry was done right after collection of relevant data (oral questionnaires and blood samples) for a given patient. Data analysis has been done after completion of data collection from all patients. Data has been analyzed for any significant correlation between the parameters from the data collected at regular intervals. Parameters have also been analyzed for any correlation with gestational hypertension and age of gestation. The interrelationship between gestational hypertension, lipid profile, uric acid and fasting blood sugar has also been assessed. SPSS software version 20, for windows 7 has been used for data analysis.

ETHICAL CONSIDERATIONS:

The study proposal along with other relevant documents was submitted to the Institutional Review Board for review and approval. The study commenced after such approval is obtained in written.

COLLECTION OF SAMPLE:

5 ml of Venous Blood samples were drawn from all the hypertensive and non-hypertensive (control group) with proper aseptic techniques following a fast of 12 hours. Blood was collected in a container having no anticoagulant. The collected clotted blood then centrifuged at 2000 rpm speed for 5 minutes for separation of serum.

All the tests were done with serum obtained from clotted blood and analyzed for Serum Uric acid (UA), Triglycerides (TG), Total cholesterol (TC) and HDL cholesterol (HDL-C) by enzymatic methods with the help of Coral clinical system on Auto analyzer XL-600.

LDL is calculated by Frederickson-Friedwald's formula according to which LDL cholesterol = Total cholesterol - (HDL cholesterol + VLDL cholesterol). VLDL cholesterol (VLDL-C) was calculated as 1/5 of Triglycerides.

Data were statistically analyzed by Student's 't' test and significance was expressed in term of 'P' value.

Gestational hypertension is determined by brachial arterial blood pressure more than 140mm/90 mm of Hg on sitting position.

STUDY TECHNIQUES:-

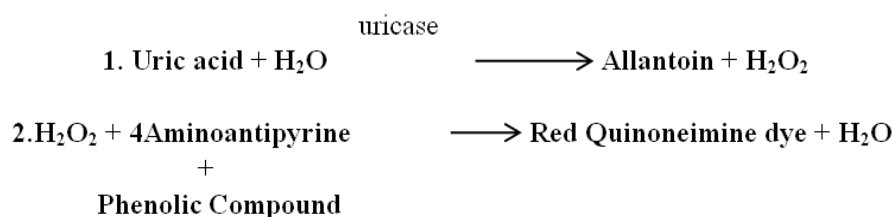
1. Serum uric acid estimation:-

Done by Uricase / PAP method.

Reagent kit- Coral clinical system.

Principle-

Uricase converts uric acid to allantoin and hydrogen peroxide. The hydrogen peroxide formed further reacts with a phenolic compound and 4-aminoantipyrine by the catalytic action of peroxidase to form a red coloured quinoneimine dye complex. Intensity of the color formed is directly proportional to the amount of uric acid present in the sample.



Sample material - Serum, plasma.

Uric acid is reported to be stable in the sample for 3-5 days when stored at 2-8°C.

Procedure-

Wavelength/filter : 520 nm/ Yellow green

Temperature : 37°C.

Light path : 1 cm.

Substrate start assay:

Pipette into clean dry test tubes labeled as Blank (B), Standard (S) and Test (T).

Additional Sequence	B (ml)	S (ml)	T (ml)
Working reagent	1.0	1.0	1.0
Distilled water	0.02	-	-
Uric Acid Standard (S)	-	0.02	-
Sample	-	-	0.02

Mix well and incubate at 37°C for 5 min. or at R.T. at (25°C) for 15 min. Measure the absorbance of the Standard (Abs.S), and Test Sample (Abs.T) against the Blank , within 30 min.

Calculation-Uric acid in mg/dl = Abs.T/Abs.S X 8

Linearity-This procedure is linear upto 20mg/dl. If values exceed this limit, Dilute with normal saline (NaCl 0.9%) and repeat the assay. calculate the value using the proper dilution factor.

Normal Reference values-

Serum/ Plasma- Males- 3.4 – 7.0 mg/dl

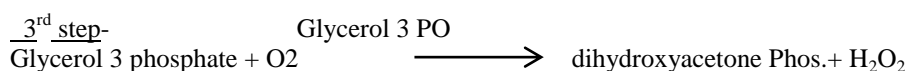
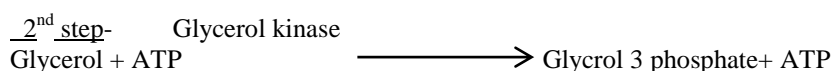
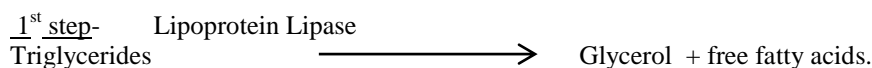
Females – 2.5 – 6.0 mg/dl

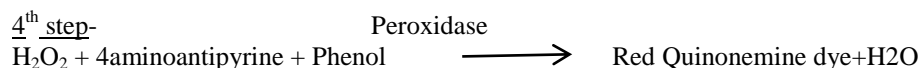
2.Triglycerides:-

Done by GPO/PAP method_

Principle-

Lipoprotein Lipase hydrolyses triglycerides to glycerol and free fatty acid. The glycerol formed with ATP in the presence of glycerol kinase forms glycerol 3 phosphate which is oxidised by the enzyme glycerol phosphate oxidase to form hydrogen peroxide. The hydrogen peroxide further reacts with phenolic compound and 4-aminoantipyrine by the catalytic action of peroxidase to form a red coloured quinoneimine dye complex. Intensity of the colour formed is directly proportional to the amount of triglycerides present in the sample.





Procedure-

Wavelength/filter : 505 nm/ Green

Temperature : 37⁰C./R.T.

Light path : 1 cm.

Substrate start assay:

Pipette into clean dry test tubes labeled as Blank (B), Standard (S) and Test (T).

Additional Sequence	B (ml)	S (ml)	T (ml)
Working reagent	1.0 0.01	1.0 -	1.0 -
Distilled water	-	0.01	-
Triglycerides Standard (S)	-	-	0.01
Sample			

Mix well and incubate at 37⁰C for 5 min. or at R.T. at (25⁰C) for 15 min. Measure the absorbance of the Standard (Abs.S), and Test Sample (Abs.T) against the Blank , within 60 min.

Calculation-

Triglycerides in mg/dl = Abs.T/Abs.S X 200

Linearity-

This procedure is linear upto 1000 mg/dl. If values exceed this limit, Dilute with normal saline (NaCl 0.9%) and repeat the assay. calculate the value using the proper dilution factor.

Special precaution- fasting samples of 12 to 14 hours are preferred. Patient should not drink alcohol for 24 hours before the test.

System parameters
Reaction : End point
Wavelength : 505 nm
Sample vol: 0.01ml
Zero setting : reagent
Blank Reagent Vol- 1.00 ml
Incub. Time: 37 ⁰ C
Standard: 200 mg/dl
Incub. Time : 5 min./15 min
Reaction slope: increasing
Linearity : 1000mg/dl
Units : mg/dl

Normal Reference values-

Serum/ Plasma- Suspicious :- 150 mg/dl and above

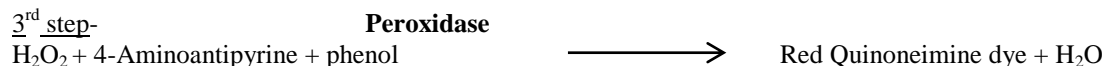
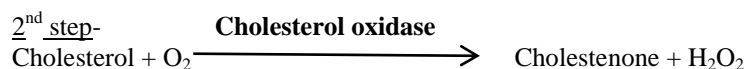
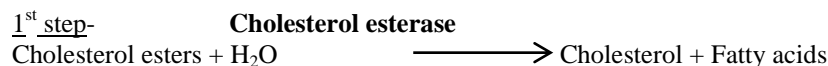
Elevated :- 200 mg/dl and above.

3.Total Cholesterol-

Method- CHOD / PAP method.

Principle:-

Cholesterol esterase hydrolyses esterified cholesterol to free cholesterol. The free cholesterol is oxidized to form hydrogen peroxide which is further reacts with phenol and 4-aminoantipyrine by the catalytic action of peroxidase to form a red coloured quinoneimine dye complex. Intensity of the colour formed is directly proportional to the amount of cholesterol present in the sample.



Procedure-

Wavelength/filter: 505 nm/ Green
 Temperature: 37⁰C./R.T.
 Light path: 1 cm.

Substrate start assay:

Pipette into clean dry test tubes labeled as Blank (B), Standard (S) and Test (T).

Additional Sequence	B (ml)	S (ml)	T (ml)
Working reagent	1.0	1.0	1.0
Distilled water	0.01	-	-
Triglycerides Standard (S)	-	0.01	-
Sample	-	-	0.01

Mix well and incubate at 37⁰C for 5 min. or at R.T. at (25⁰C) for 15 min. Measure the absorbance of the Standard (Abs.S), and Test Sample (Abs.T) against the Blank , within 60 min.

Calculation-

Cholesterol in mg/dl = Abs.T/Abs.S X 200

Linearity-

This procedure is linear upto 750mg/dl. If values exceed this limit, Dilute with normal saline (NaCl 0.9%) and repeat the assay. calculate the value using the proper dilution factor.

Note-Anticoagulants such as fluorides and oxalates results in false low values. The test is not influenced by Hb values upto 20 mg/dl and bilirubin upto 10 mg/dl.

System parameters
Reaction : End point
Wavelength : 505 nm
Sample vol: 0.01ml
Zero setting : reagent Blank
Reagent VOL- 1.00 ml
Incub. Time: 37 ⁰ C
Standard: 200 mg/dl
Incub. Time : 5 min./15 min
Reaction slope: increasing
Linearity : 1000mg/dl
Units : mg/dl

Normal Reference values-

Serum/ Plasma- Suspicious :- 220 mg/dl and above
 Elevated :- 260 mg/dl and above.

4. High Density Lipoprotein Cholesterol:-

Method – Direct method.

Principle –The direct HDL Cholesterol assay is a homogenous method for directly measuring serum HDL-C level without the need for any pretreatment and centrifugation steps. First step , substances with high affinity to

Reference value of LDL.⁸⁴

□ Male- 20-69 years=66-186 mg/dl □ Female- 20-69 years=57-206 mg/dl

Risk of coronary heart disease with LDL:-

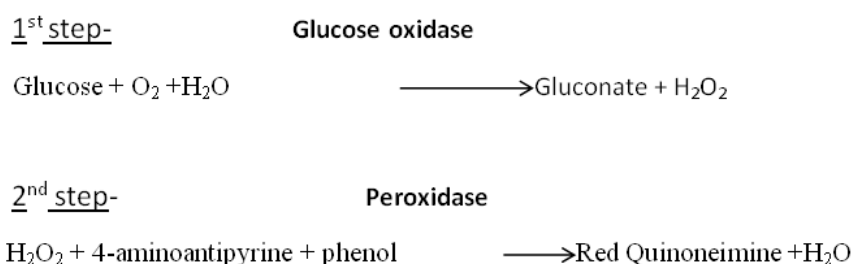
- Optimal less than 100mg/dl
- Near/above optimal=100-129 mg/dl
- Border line=130-159mg/dl
- High =160-189mg/dl
- Very high=more than 189mg/dl.

7.plasma fasting glucose –

Method- Glucose Oxidase/Peroxide Method.

Procedure-

glucose is oxidized to gluconic acid and hydrogen peroxide in the presence of glucose oxidase. Hydrogen peroxide further reacts with phenol and 4-aminoantipyrine by the catalytic action of peroxidase to form a red coloured quinoneimine dye complex. Intensity of the color formed is directly proportional to the amount of glucose present in the sample.



Procedure-

Wavelength/filter : 505 nm/ Green Temperature: 37⁰C./R.T.

Light path : 1 cm.

Substrate start assay:

Pipette into clean dry test tubes labeled as Blank (B), Standard (S) and Test (T).

Additional Sequence	B (ml)	S (ml)	T (ml)
Working reagent	1.0	1.0	1.0
Distilled water	0.01	-	-
Triglycerides Standard (S)	-	0.01	-
Sample	-	-	0.01

Mix well and incubate at 37⁰C for 5 min. or at R.T. at (25⁰C) for 30 min. Measure the absorbance of the Standard (Abs.S), and Test Sample (Abs.T) against the Blank , within 60 min. Calculation- cholesterol in mg/dl = Abs.T/Abs.S X 100

Linearity-This procedure is linear upto 500mg/dl. If values exceed this limit, Dilute with normal saline (NaCl 0.9%) and repeat the assay. calculate the value using the proper dilution factor.

Note-To avoid glycolysis the serum should be separated from the clot as soon as possible, and plasma should be collected in an EDTA + Fluoride bulb (0.5 mg +1mg per ml of blood ⁸⁵

System parameters	
Reaction	: End point
Wavelength	: 505 nm
Sample vol	: 0.01ml
Zero setting	: reagent Blank
Reagent VOL:	1.00 ml Incub. Time : 37°C/ R.T.
Standard	: 100 mg/dl
Incub. Time	: 10 min./30 min
Reaction slope:	increasing
Linearity	:500mg/dl
Units	: mg/dl

Normal Reference values-

Serum/ Plasma- (Fasting):- 70-110 mg/dl and above
(2 hrs.P.P) :- upto200 mg/dl.

III. Review of Literature

Hypertension during pregnancy is a major health problem. It is one of the leading causes of perinatal morbidity and mortality.¹² Preeclampsia (PE) is a theoretical disease with a pathogenesis that is not clearly understood yet. Lately vascular system pathology and vasoconstriction have been blamed as causes for preeclampsia with growing acceptance. Lipid values in normal pregnancies change with gestational age. In a normotensive pregnancy, serum triglyceride, total and HDL-cholesterol increase during pregnancy, but lipoprotein A levels decrease. These changes are reported to be secondary to hormonal changes during pregnancy.¹²⁻¹⁴ In this study we aimed to demonstrate the lipid profile.

The association of alteration of serum lipid profile in essential hypertension is well documented. An abnormal lipid profile is known to be strongly associated with atherosclerotic cardiovascular diseases and has a direct effect on endothelial dysfunction. The most important feature in toxemia of pregnancy is hypertension which is supposed to be due to vasospastic phenomenon in kidney, uterus, placenta and brain⁽¹²⁾.

Altered lipid synthesis leading to decrease in PGI2 : TXA2 ratio is also supposed to be an important way of pathogenesis in pregnancy induced hypertension⁽¹³⁾. Thus abnormal lipid metabolism seems to be important in the pathogenesis of pregnancy induced hypertension (PIH) too. Obviously the association of serum lipid profile with gestational proteinuric hypertension is highly suggested to reflect some new diagnostic tools. Moreover, the hormonal imbalance is a prime factor for the etio-pathogenesis of PIH and this endocrinal imbalance is well reflected in alteration of serum lipid profile.

There are several potential origins for uric acid in preeclampsia; these are-

- abnormal renal function,
- increased tissue breakdown,
- acidosis
- And increased activity of the enzyme xanthine oxidase/dehydrogenase^[15].

Gestational hypertension, also referred to sometimes as pregnancy induced hypertension (PIH) is a condition of high blood pressure during pregnancy. This can lead to a serious condition called preeclampsia, also sometimes called as toxemia of pregnancy. Hypertension during pregnancy affects about 6-8% of all pregnant women.

The different types of hypertension during pregnancy:

High blood pressure can present itself in a few different ways during pregnancy. The following are the 3 common types of gestational hypertension:

- **Chronic Hypertension-** Women who have high blood pressure (over 140/90) before pregnancy, early in pregnancy (before 20 weeks), or carry it on after delivery.
- **Gestational Hypertension-** High blood pressure that develops after 20 weeks in pregnancy and goes away after delivery.
- **Preeclampsia-** Both chronic hypertension and gestational hypertension can lead to this severe condition after week 20 weeks of pregnancy. Symptoms include high blood pressure and protein in the urine and can lead to serious complications for both mother and baby, like convulsion, unconsciousness etc. in mother and in baby like growth retardation, premature delivery etc. if remain untreated.

Several epidemiological studies have shown that hyperuricemia is a risk factor for cardiovascular diseases (CVDs) in the general population⁽¹⁶⁻¹⁸⁾. The contribution of serum uric acid (UA) to atherosclerotic vascular disease however remains controversial⁽¹⁹⁾. Some studies argue that the observed association between UA and atherosclerotic vascular disease is attributable to an indirect association of hyperuricemia with

cardiovascular risk factors or clustering of these metabolic and hemodynamic risk factors, designated metabolic syndrome (MS) (20,21).

Recent evidence suggests that UA stimulates vascular smooth muscle proliferation and induces endothelial dysfunction. UA has been shown to decrease endothelial nitric oxide production and lead to endothelial dysfunction and insulin resistance^(22,23). Consequently UA induces vascular inflammation and artery damage, which in turn leads to an increased risk of atherosclerosis.

Although many studies in adults have focused on the relationship between UA, metabolic syndrome, and carotid atherosclerosis⁽²⁴⁻²⁶⁾, very little is known about this association in children and adolescents. Because atherosclerosis often begins in childhood or young adulthood⁽²⁷⁾, studies in this age group are important. One additional advantage of examining children is that a diminished potential confusion exists with adult-onset complications. Previous cross-sectional data have shown a close relationship between UA concentrations and cardiovascular risk factors in obese children and adolescents⁽²⁸⁻³¹⁾. However the role of UA as an independent risk factor for CVD remains to be clarified in children. Carotid intima-media thickness (IMT) measured noninvasively by ultrasonography is a well-established index (32,33).

Uric Acid

Uric acid is a product of purine degradation catalyzed by the enzyme xanthine dehydrogenase/xanthine oxidase (XDH/XO). XDH is converted to its oxidase form XO by several stimuli including ischemia^[34]. Purine metabolism by XO couples the production of uric acid with the production of the free radical superoxide (O_2^-), and is implicated as a contributor to oxidative stress^[34]. XDH/XO is found in most tissues but is concentrated in the liver and gut. Recently, a circulating population of XO has been identified that increases dramatically following ischemic tissue damage^[35]. Increased circulating uric acid accompanies similar insults^[36]. It is speculated that circulating XO can bind to endothelium and lead to local oxidative injury^[37]

Most mammals possess the enzyme uricase, that breaks down uric acid into allantoin, a nontoxic product excreted by the kidney. Humans and great apes however do not possess uricase and uric acid clearance is reliant largely upon renal excretion^[38]. Uric acid is minimally soluble and its concentration is maintained relatively low in healthy individuals (<6.0 mg/dL). However even low concentrations of uric acid possess biological function. Uric acid is a plasma antioxidant capable of scavenging superoxide, hydroxyl radical and singlet oxygen^[39]. It also reduces nitrosylation of tyrosine residues on proteins by peroxynitrite and is capable of maintaining superoxide dismutase activity^[40] shown to have beneficial effects in some settings. Conversely, uric acid itself can become a pro-oxidant (urate radical) in a setting of compromised antioxidant availability, particularly reduced ascorbate availability^[41]. Uric acid is also a mediator of inflammation stimulating the production of monocyte chemoattractant protein-1, IL-1 β , IL-6 and TNF- α ^[42, 44].

Uric Acid during Pregnancy

Uric acid concentrations are influenced by diet (i.e. high protein, and fructose), alcohol consumption, increased cell turnover, enzymatic defects in purine metabolism or altered kidney function [42]. Estrogen is uricosuric and uric acid concentrations are higher in men and post-menopausal women [43]. In pregnancy uric acid concentrations initially fall 25-35% due to the effects of estrogen, expanded blood volume and increased glomerular filtration rate[44]. However, concentrations slowly rise to those observed in non-pregnant women by term gestation (4-6 mg/dl) [43].

Hyperuricemia and Preeclampsia

Elevated uric acid concentrations were first noted in preeclampsia women in the late 1800s. Since that time numerous reports have demonstrated a relationship between uric acid concentrations and severity of disease [48, 49]. Nonetheless, the clinical utility of hyperuricemia in the management of preeclampsia is controversial. Recently it has been examined the relationship of uric acid elevations in pregnant hypertensive women to the endpoints of preterm birth (largely indicated preterm birth for the management of preeclampsia) and growth restriction [49]. Hyperuricemia was present in about 16% of women with gestational hypertension without proteinuria and 75% of women with clinically diagnosed PE. Pregnancy induced hypertension with hyperuricemia was associated with an excess of these adverse fetal outcomes. The increased frequency of preterm birth and growth restriction was present in hypertensive women with elevated concentration of uric acid even in the absence of proteinuria.

In women who go on to develop preeclampsia, uric acid concentration is elevated as early as 10 weeks of gestation [49], a time much earlier than the clinical presentation of the disorder. Increased uric acid often precedes clinical manifestations of the disease, including reduced glomerular filtration rate [49]. Nonetheless, hyperuricemia has historically been attributed to reduced renal clearance. Uric acid is filtered, reabsorbed and secreted by the kidney. hypervolemia, an early change in preeclampsia, increases uric acid reabsorption which

could increase serum uric acid concentrations. However, increased uric acid precedes the reduction in plasma volume [50]. Increased uric acid production from maternal, fetal or placental tissues through heightened tissues breakdown (ie. increased substrate availability) and/or increased XO activity could also explain the increased concentration. The specific stimuli responsible for increased XO activity in preeclamptic women are unclear.

Uric Acid as a Pathogenic Vascular Factor

Evidence for a pathogenic role of uric acid is increasing. In the non-pregnant population hyperuricemia is an independent predictor of cardiovascular and renal disease in both the general population and in subjects with chronic hypertension [51]. Uric acid is also a marker for adverse cardiovascular events in patients with established cardiovascular disease [51]. Experimental studies also support a pathogenic role for uric acid. Rats rendered experimentally hyperuricemic through administration of oxonic acid, an uricase inhibitor,

Potential Effects of Uric Acid on Placental Vascular Development, Structure and Function

1. Uric Acid and Placental Development-

The placental trophoblast modifies its phenotype over the course of gestation, switching from a highly proliferate to a highly invasive cell subtype. This allows for adequate placental development and invasion of maternal decidua and spiral arterioles. To date we do not find any studies examining the effects of uric acid on trophoblast cells. Our discussion of the impact of uric acid on placental growth and development is speculative and based upon phenotypical and functional similarities between endothelial cells and trophoblast cells [53]. Uric acid has a profound effect on both endothelial cell proliferation and migration, inhibiting serum-induced proliferation in HUVEC cells by 50% and inhibiting HUVEC migration by as much as 75% [54]. These effects are a direct result of uric acid uptake by endothelial cells as treatment with probenecid, an inhibitor of organic anion transport, attenuated these effects [54].

Invasive extra villous cytotrophoblast cells invade maternal spiral arterioles with associated vascular remodeling. This results in large diameter, flaccid vessels with no responsive smooth muscle in the vessel wall. In preeclampsia this tightly regulated re-modeling process does not take place adequately, resulting in compromised oxygen and nutrient delivery to the placenta. Nitric oxide (NO) production facilitates trophoblast migration and invasion both in-vitro and in animal models [55, 56]. Moreover, eNOS is present in invading cytotrophoblast cells [57]. Uric acid reduces NO production in endothelial cells [54] and a similar effect in trophoblast could modify the migratory and invasive phenotype of trophoblast cells. Since uric acid is increased in women destined to develop preeclampsia prior to 10 weeks gestation it is plausible that uric acid could contribute to inadequate trophoblast invasion and spiral arteriole remodeling. Furthermore, localized increased uric acid production is also possible as elevations in XO enzyme and activity have been observed in invasive cytotrophoblast cells of preeclamptic women [58].

2. Uric Acid and Placental Vascular Structure and Function

Normal placental vascular structure and transport function are ultimately responsible for transporting required oxygen and nutrients to the developing fetus. Uric acid is capable of damaging adult vasculature [51] and could have similar effects in the placentae of preeclamptic women. It enters smooth muscle cells through organic anion transporters and activates intracellular mitogen-activated protein kinases (i.e. p38) and nuclear transcription factors (i.e. NFκ-B) [44]. In vitro uric acid stimulates the production of: platelet derived growth factor [58], the vasoconstrictors thromboxane and angiotensin II [59,60], and markers of inflammation such as C-reactive protein [54]. Thus, uric acid treatment of smooth muscle cells results in a proliferative and pro-inflammatory phenotype. Interestingly some of these effects of uric acid on smooth muscle cells are attenuated in the presence of antioxidants [44] suggesting a pathogenic role for the urate radical.

The placental vasculature lacks autonomic innervation [61], relying entirely upon locally produced or circulating substances for hemodynamic control. The primary vasoactive compound responsible for the maintenance of optimized placental perfusion is endothelial derived NO. In hyperuricemic rats uric acid decreases eNOS activity limiting NO availability [54] and up-regulates COX-2 expression with increased generation of the potent vasoconstrictor thromboxane [59]. A similar vasoconstrictive effect of uric acid in the placentae of women with preeclampsia would compromise placental perfusion, and could inhibit fetal growth.

2. Uric Acid and Placental Redox Balance

Increased placental oxidative stress is a well-documented feature of preeclampsia. Oxidative imbalance results from increased pro-oxidant generation coupled with insufficient antioxidant capacity. Under normal circumstances uric acid scavenges oxidizing agents known to play a role in the placental pathologies of preeclampsia [39]. This anti-oxidant function of uric acid results in the transformation of uric acid into a free radical, a urate radical [62]. And again under normal circumstances, urate is quickly recycled back to its anti-oxidant state through the actions of ascorbate [41]. In a setting of reduced ascorbate availability, as is present in

preeclamptic women [63], the urate radical persists and can potentially oxidatively modify placental proteins and lipids

Potential Effects of Uric Acid on Maternal Vasculature

1. Uric Acid and Maternal Hypertension:-

The potential pathogenic effects of uric acid upon the placental vascular bed are also relevant to the maternal vasculature. Preeclamptic women have elevated concentrations of circulating vasopressors, such as thromboxane and endothelin, with parallel decreases in vasodilators such as prostacyclin [64, 65]. The maternal vasculature of preeclamptic women also exhibits increased sensitivity to vasopressor agents [66]. It is proposed that this is in part due to reduced availability of nitric oxide secondary to the endothelial dysfunction [67]. As stated above, elevated uric acid concentration could participate in reduced production of NO and may in part explain the altered endothelial contribution to vascular tone in preeclamptic women. Decreasing uric acid with allopurinol improved endothelial dependent vasodilation in diabetic and congestive heart failure patients [68, 69]. Reducing uric acid concentration might be a potential therapeutic strategy for preeclamptic women as well.

2. Uric Acid and Inflammation

The preeclamptic environment of heightened inflammation results in endothelial dysfunction and vascular lesions. Uric acid is a potent mediator of inflammation. In vascular smooth muscle cells uric acid increases the concentration of the chemokine monocyte chemo attractant protein-1 (MCP-1) mRNA and protein in a time and dose dependant fashion [44]. Uric acid also stimulates human monocytes to produce the pro-inflammatory cytokines IL-1 β IL-6 and TNF- α , [51, 44] which are also elevated in the circulation of experimentally induced hyperuricemic animals [70] as well as preeclamptic women [71]. In preeclamptic women the increased concentration of circulating TNF- α was positively correlated to circulating uric acid concentrations [72].

3. Uric Acid and Endothelial Repair

Dramatic elevations in circulating uric acid follow an acute ischemia-reperfusion event [73]. This acute rise in uric acid concentrations in animal models induces endothelial progenitor cell (EPC) mobilization [73]. Uric acid is posited to act as a signal for endothelial damage promoting repair of damaged vessels through EPC mobilization. However, this protective role of uric acid is only present with acute increases of circulating uric acid. Increased mobilization of EPCs is not observed in rats rendered chronically hyperuricemic following an induced renal ischemic-reperfusion injury [73]. Thus chronically increased uric acid is associated with blunted release of EPC's. It is of note that these progenitor cells are reduced in preeclampsia [74]. This may be the result of increased usage of these cells in damaged vascular beds or, based on the data outlined above, decreased mobilization of the cells.

3. Uric Acid and Maternal Renal Dysfunction

Renal dysfunction is a consistent finding in preeclamptic women. Renal anatomic changes include juxtaglomerular hyperplasia, macula densa atrophy, afferent arteriopathy, glomerular hypertrophy and glomeruloendotheliosis [54]. The mildly hyperuricemic rat model demonstrates remarkably similar renal changes [60, 54] including afferent arteriopathy, mild tubulointerstitial fibrosis, glomerular hypertrophy and eventually glomerulosclerosis with subsequent albuminuria and proteinuria [52]. These uric acid induced pathologies are independent of uric acid crystal formation and can be inhibited by lowering uric acid concentrations. Interestingly, over 20 years ago Nochy reported that the renal lesions observed in preeclamptic women are seen only in hyperuricemic preeclampsics [75]

Forward Cycle of Uric Acid Production in Preeclampsia

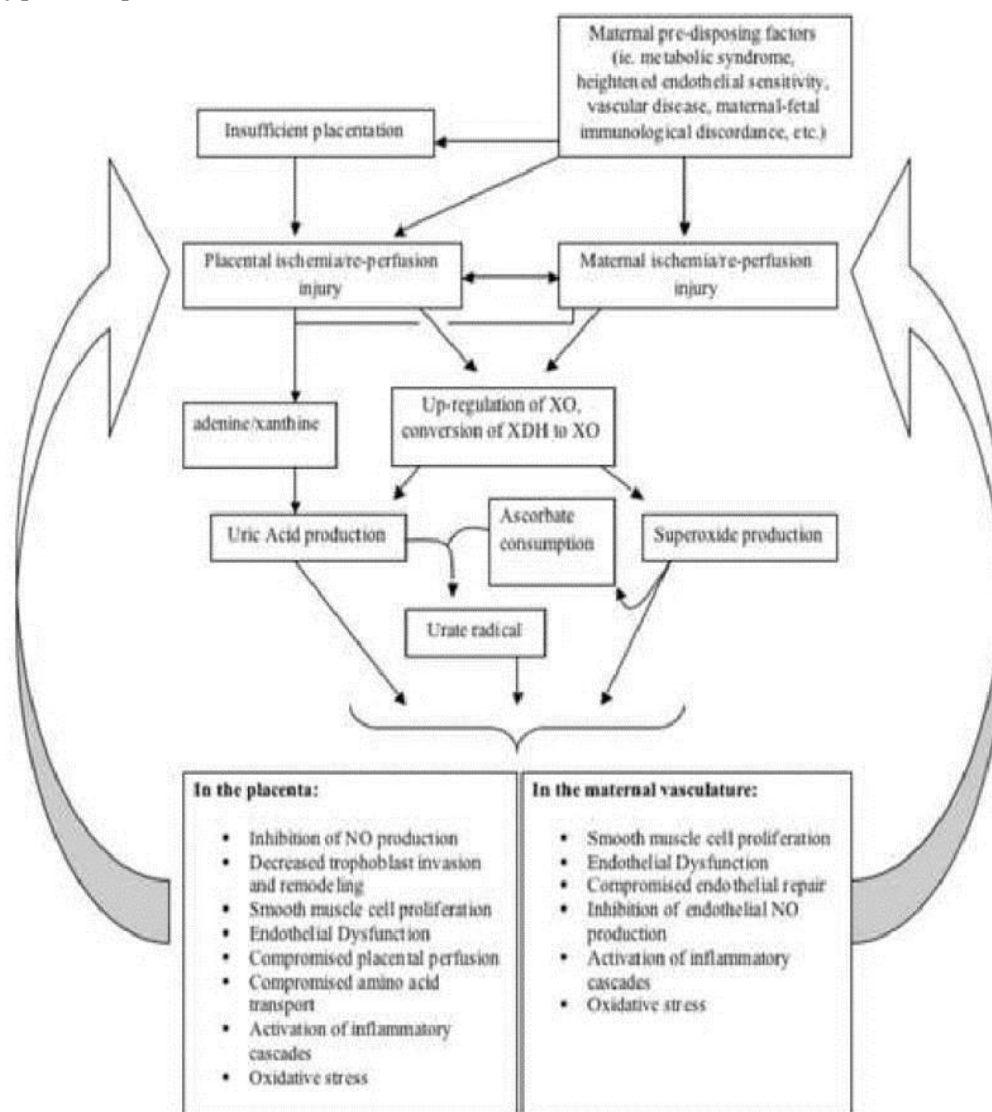
How is the hyperuricemia of preeclampsia initiated?

The increase in uric acid antedates the reduction of glomerular filtration and hypovolemia [51, 43]. Thus it seems unlikely that these two features of preeclampsia, that potently reduce uric acid excretion, are initially responsible. It is possible that women destined to develop preeclampsia come into pregnancy with elevated uric acid as part of the metabolic syndrome or that uric acid production is increased in early pregnancy. There are several plausible sources for increased uric acid in women with preeclampsia, including the fetus, placenta, and maternal organs and vasculature.

Uric acid could contribute to failed placental bed vascular remodeling by impeding trophoblast invasion with resultant reduced placental perfusion, setting the stage for ischemia reperfusion injury to the placenta and oxidative stress. Maternal tissues may also experience ischemic injury due to vasospasm secondary to endothelial dysfunction ,also plausibly related to increased uric acid. Ischemic injury and oxidative stress

promotes a feed-forward cycle of uric acid production. With tissue injury, purines are liberated and with hypoxia ATP is degraded to both adenine and xanthine (substrate). Additionally hypoxia is a potent inducer of the holoenzyme xanthine oxidase/dehydrogenase and preferentially increases the oxidase form of the enzyme [34]. With the parallel increases in both substrate and enzyme concentrations, uric acid production will increase. Furthermore vasospasm and loss of fluid secondary to endothelial dysfunction also stimulate renal reabsorption of uric acid [76]. Thus hyperuricemia leads to more uric acid production and less uric acid excretion in a feed forward loop.

The feed forward mechanism of uric acid production and potential pathogenic roles of uric acid in the context of preeclampsia-



Gestational hypertension with lipid status

E. J. Roccella et al say that ,changes in the plasma lipids during pregnancy have been recognized, described & are thought to be done mostly to alterations in hormonal milieu. Plasma lipids & lipoproteins undergo both qualitative & quantitative changes during pregnancy. These changes revert towards normal shortly after delivery (77).

The association of alteration of serum lipid profile in essential hypertension is well documented. An abnormal lipid profile is known to be strongly associated with atherosclerotic cardiovascular diseases and has a direct effect on endothelial dysfunction.

M. D. Lindheimer et al say that, the most important feature in toxemia of pregnancy is hypertension which is supposed to be due to vasospastic phenomenon in kidney, uterus, placenta and brain (78).

L. A. Simmons et al say that, altered lipid synthesis leading to decrease in PGI:TxA2 ratio is also supposed to be an important way of pathogenesis in pregnancy induced hypertension (PIH). (79)

Thus abnormal lipid metabolism seems important in the pathogenesis of pregnancy induced hypertension too. Obviously the association of serum lipid profile with gestational proteinuric hypertension is highly suggested to reflect some new diagnostic tools. Moreover, the hormonal imbalance is a prime factor for the etiopathogenesis of PIH and this endocrinal imbalance is well reflected in alteration of serum lipid profile.

N. F. Gant et al say that, early pregnancy dyslipidemia is associated with an increased risk of Pre-eclampsia. (80)

J. M. Davison et al say that, Women with a history of pre-eclampsia have significant differences in lipid parameters and an increased susceptibility to lipoprotein oxidation when compared with women who had normal pregnancy. Disorders of lipoprotein metabolism are reported to be a major cause of hypertension and proteinuria in Pre-eclampsia. (81)

Therefore, simple measurements of serum lipid parameters may be of good predictive value in toxemia of pregnancy, avoiding the costly endocrinal investigations. Despite considerable research, the causes of preeclampsia remain unclear and there are no clinically useful screening tests to identify women in whom it will develop.

STATISTICAL ANALYSIS

All the data, thus collected, was put into Microsoft Excel sheet. The analyses were performed using SPSS version 20.0 software. The mean serum lipid concentrations and uric acid concentration of the cases and controls were compared using paired t test. Significance was set at $P < 0.05$. Correlations between various lipid fractions, uric acid and fasting blood sugar were made using Pearson’s correlation coefficient, considering the group with gestational hypertension which is study group and who are normal blood pressure pregnant mother are control groups.

IV. V Results

Table 1: Demographic characteristics of control and gestational hypertension groups.

	Gestational Hypertension (n=50) ±SD	Control (n=50) ±SD
Age (years)	25.24 ± 3.826	26.34 ± 3.826
Gestational age(weeks of pregnancy)	25.04 ± 4.746	26.06 ± 4.635
Systolic BP (mm/Hg)	153 ± 520	110.60 ± 9.783
Diastolic BP (mm/Hg)	98.26 ± 5.710	76.04 ± 7.382

Figure 1: shows Demographic characteristics of Hypertensive and control groups.

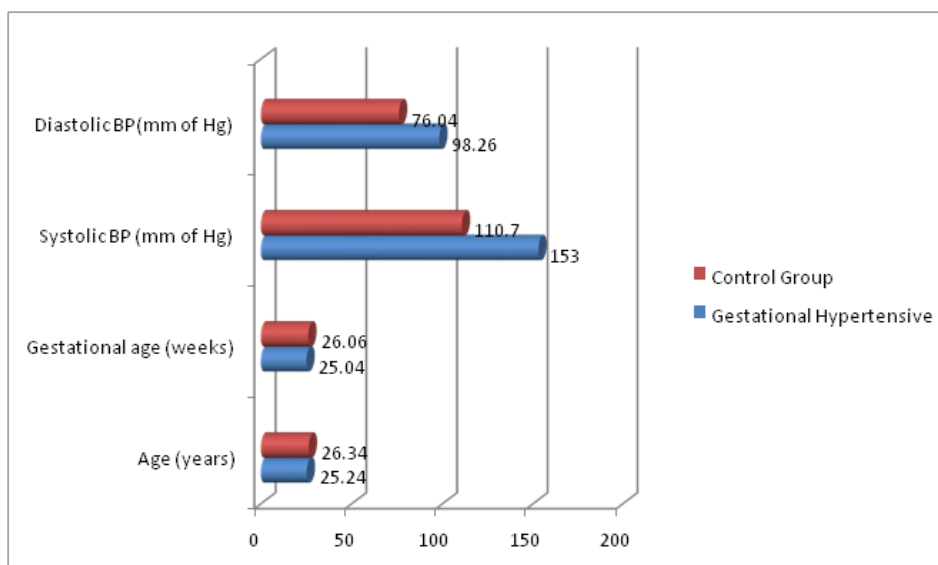


Table-2.Biochemical parameters as mean \pm SD

Tests	Tests Gestational Hypertension (n=50)	Control (n=50)	P value
TC (mg/dl)	215.74 \pm 21.963	154.20 \pm 16.479	0.049
TGL (mg/dl)	306.44 \pm 19.366	185.18 \pm 32.276	0.009
HDL-C (mg/dl)	24.26 \pm 2.841	53.90 \pm 5.765	<0.0001
LDL-C (mg/dl)	135.38 \pm 22.058	63.26 \pm 17.867	0.071
VLDL-C (mg/dl)	56.10 \pm 9.550	37.04 \pm 3.380	<0.0001
UA (mg/dl)	6.12 \pm 0.659	3.28 \pm 0.701	0.045
FBS (mg/dl)	93.58 \pm 8.741	82.66 \pm 10.72	0.064

[TC-total cholesterol, TGL-triglycerides, HDL-C-high density lipoprotein cholesterol, LDL- C-low density lipoprotein cholesterol, VLDL-C-very low density lipoprotein cholesterol, UA-uric acid, FBS-fasting blood sugar.]

Figure 2: Bar diagram shows Comparison of lipid parameters between Gestational hypertension (GH) and control groups-

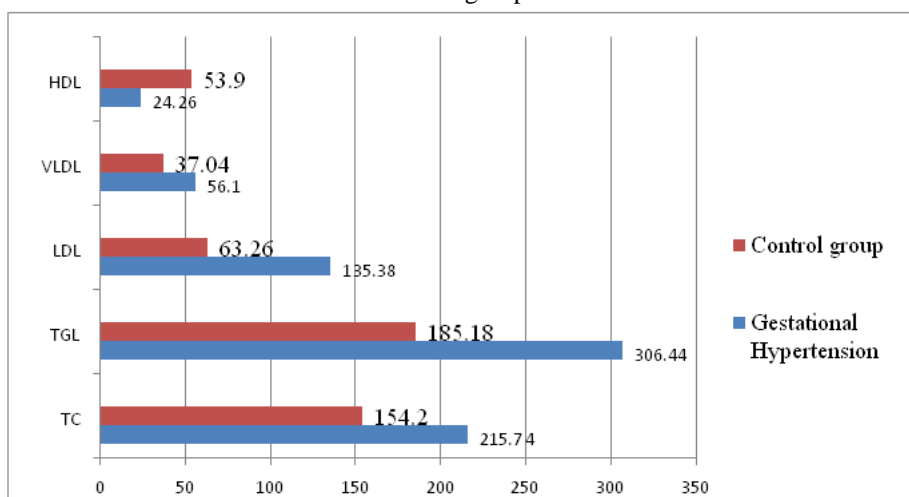


Figure3: Bar diagram shows Comparison of Total Cholesterol between Gestational hypertension (GH) and control groups. P < 0.05

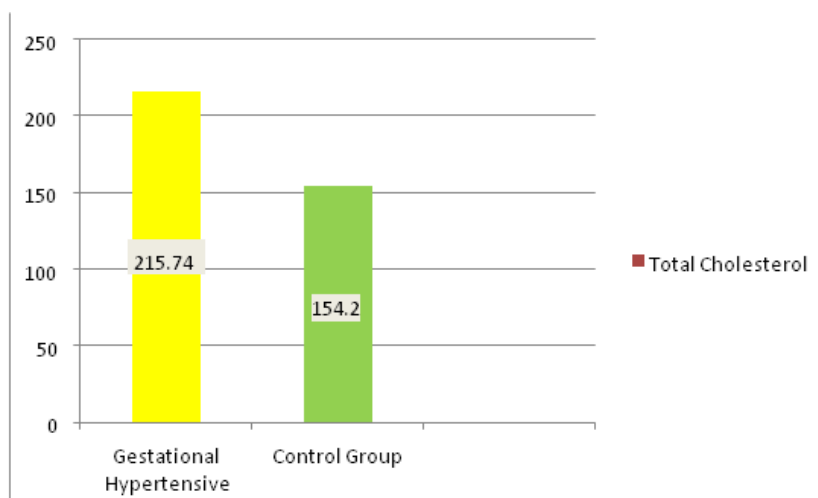


Figure 4: Bar diagram shows Comparison of Triglyceride between Gestational hypertension (GH) and control groups. $P < 0.05$

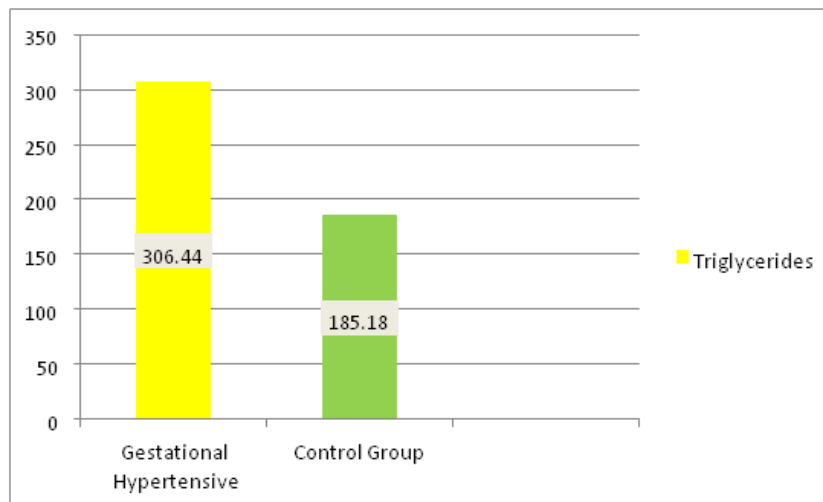


Figure 5: Bar diagram shows Comparison of High Density Lipoprotein Cholesterol (HDL) between Gestational hypertension (GH) and control groups. $P < 0.0001$

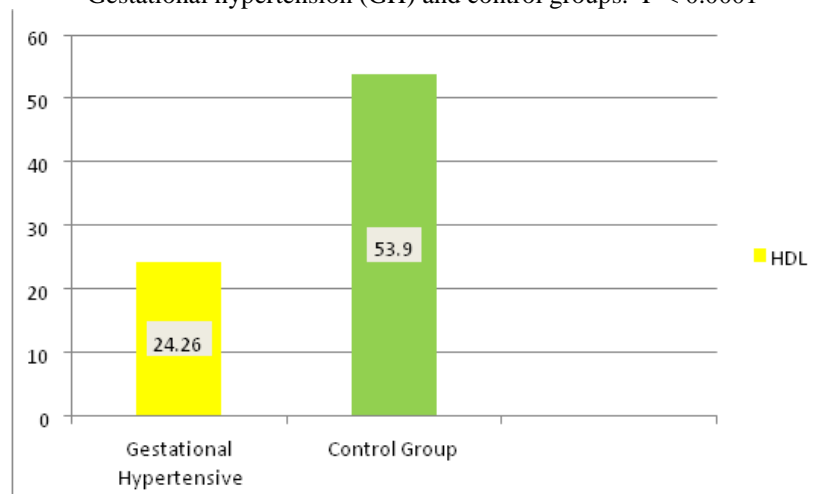


Figure 6: Bar diagram shows Comparison of Low Density Lipoprotein Cholesterol (LDLC) between Gestational hypertension (GH) and control groups. $P = 0.071$

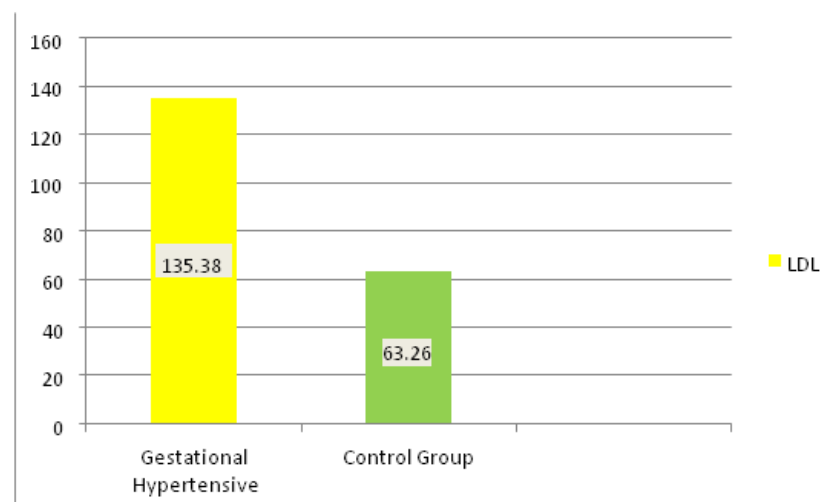


Figure 7: Bar diagram shows Comparison of Very Low Density Lipoprotein Cholesterol (VLDL-C) between Gestational hypertension (GH) and control groups. $P < 0.0001$

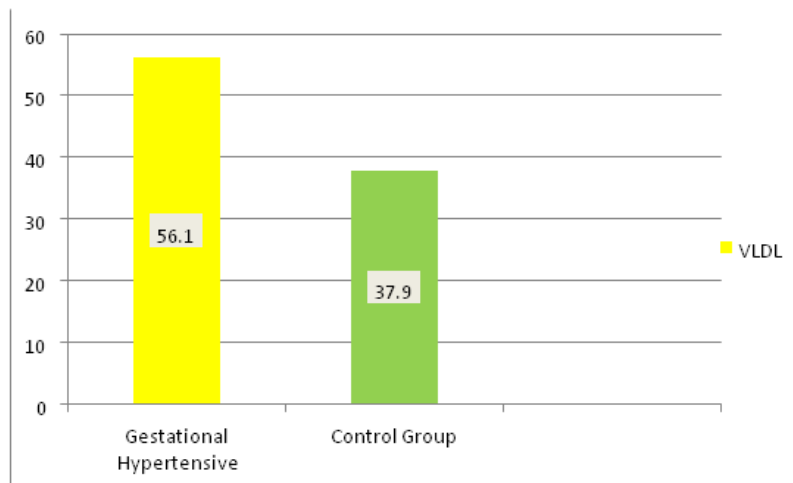


Figure 8: Bar diagram shows Comparison of Uric Acid between Gestational hypertension (GH) and control groups. $P < 0.05$

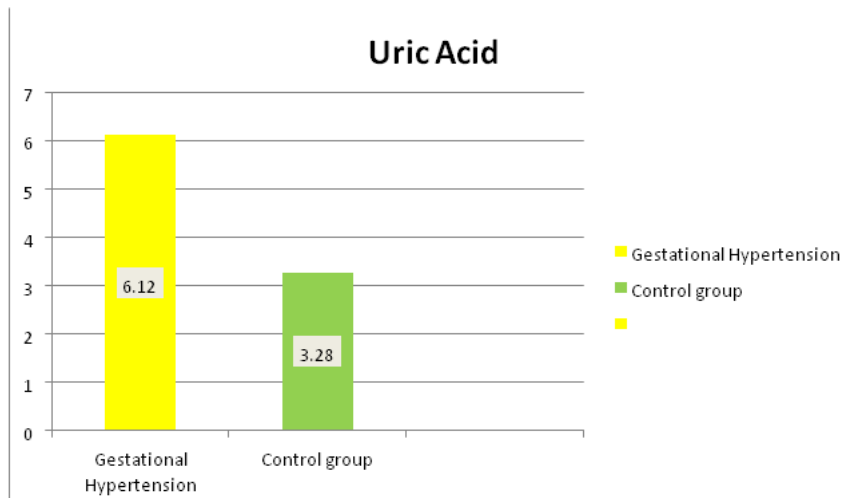


Figure 9: Bar diagram shows Comparison of Fasting Blood Sugar(FBS) between Gestational hypertension (GH) and control groups. $P = 0.064$

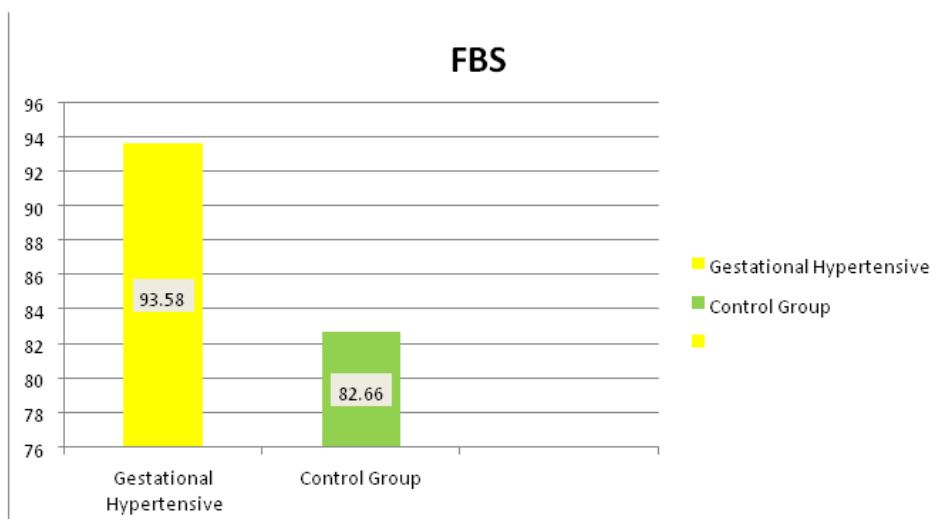


Table-3: Pearson's coefficient of determination (r) of serum uric acid with blood pressure and lipid parameters in Gestational hypertensive women-

	'r' value	P value(2 tailed)
Total Cholesterol	-0.198	0.168
Triglycerides	0.172	0.232
LDL-C	-0.236	0.099
VLDL-C	0.037	0.799
HDL-C	0.179	0.213
Systolic BP	0.072	0.621
Diastolic BP	0.122	0.398

V. Discussion

The present study was a hospital based, non-interventional, cross sectional study. This study was undertaken in the Department of Biochemistry of Burdwan Medical College @ Hospital, Burdwan in collaboration with the Department of Obstetrics and Gynaecology of Burdwan Medical College @Hospital .The patients with gestational hypertension who had come for treatment in the Obsterics and Gynaecology department in indoor and outdoor of Burdwan Medical College @ Hospital were selected as study cases. Age and period of gestation- matched controls were selected from the pregnant mothers who attended in the antenatal clinic of Burdwan Medical College.

The objective of this study was based on the assumption that the gestational hypertensive mothers may suffer from increased uric acid and deranged lipid profile due to pregnancy induced hypertension.

The study showed serum lipid elevations in gestational hypertensive patients and the results obtained from the present study showed significantly higher levels ($p < 0.05$) of plasma total cholesterol, TGL, LDL-C, and VLDL-C and significantly lower levels of HDL-C levels in hypertensive pregnant women compared to the controls. The findings in our study are in consistent with most of the previous studies^(86, 87)

Gratacos et al. (1996) showed a significantly higher levels of triglycerides, LDL-C, HDL-C, Total Cholesterol (TC), apoA and apoB in hypertensive pregnancy. In our study, the results were similar to Gratacos et al's study, but were different for HDL-C levels, which are decreased and LDL-C are not statistically significantly increased ($p=0.071$). This is probably due to my low sample size.

The total cholesterol level in gestational hypertensive cases was found to be higher ($215.74\text{mg/dl} \pm 21.96$) than controls ($154.20\text{mg/dl} \pm 16.47$) and the difference was statistically significant ($p < 0.05$). These findings are in agreement with the studies reported by Cekmen MB et al. (2003) and Sattar et al. (1997).

In the present study, the mean serum TGL levels of hypertensive cases ($306.44\text{mg/dl} \pm 19.36$) are higher than controls ($185.18\text{mg/dl} \pm 32.27$). This increase is statistically significant ($p < 0.05$). It was found that serum TGL levels was more higher in hypertensive pregnancy cases than that of controls, which have also been reported in previous studies by Cekmen MB et al. (2003).

The mean VLDL-C in gestational hypertensive cases ($56.10\text{mg/dl} \pm 9.55$) rise significantly increased, compared to the controls ($37.04\text{mg/dl} \pm 3.38$), which is due to hypertriglyceridemia leading to enhanced entry of VLDL that carries endogenous triglyceride into circulation. The increased VLDL-C levels in the hypertensive pregnant women in the present study correlates with the findings of other researchers (Sattar et al., 1997).

In the present study, the serum LDL-C levels of gestational hypertensive cases ($135.38\text{mg/dl} \pm 22.05$) are higher ($p=0.071$) and shows a 46.7% rise over the control group ($63.26. \text{mg/dl} \pm 17.86.$) which corroborated with the findings of other studies like Hubel et al. (1998), Sattar et al., (1997) and DR. P.JOSEPHINE LATHA et all (2013). But in my study 'p' value for LDL-C is statistically not significant ($p > 0.05$). This is probably due to my low sample size ($n=50$) in comparison to other studies, where sample sizes are higher in number.

In addition, in the present study the mean serum HDL-C levels in cases are low

($24.26\text{mg/dl} \pm 2.84$) while in controls were high ($53.90\text{mg/dl} \pm 5.76$), ($p < 0.0001$) and this is statistically highly significant. The HDL-C levels decreased in the present study to about 45%. These findings are similar to the studies done by Kaaja R et al. (1995) and Ware JS et al. (1999). The low levels of HDL-C in hypertensive pregnancy is not only because of hypoestrogenaemia but also due to insulin resistance (Kaaja R et al., 1995)..

No significant differences were exhibited between gestational hypertension (GH) group (93.58 ± 8.74) and controls (82.66 ± 10.72) regarding FBS ($p = 0.064$). this is also similar finding in DR.P.JOSEPHINE LATHA et all⁽⁸⁹⁾.

In gestational hypertension group, all the parameters were correlated using Pearson's Correlation (Table-3). That showed significant positive correlation of uric acid with systolic and diastolic blood pressure, TGL, VLDL, HDL ($r = .072, .024, .172, .037, .179$) and have negative correlation with TC and LDL ($r = -.198, -.236$) respectively.

In the present study uric acid level was also significantly elevated in gestational hypertensive group (6.12 ± 0.65) against the control groups (3.28 ± 0.70). Which is consistent with some workers like S. Katherine Laughon et al^(87,89)

Therefore it is evident that dyslipidemia and hyperuricemia was found in gestational hypertensive patients. The elevated TC, TGL, VLDL-C, & LDL-C levels and reduced HDLC levels may be due to exaggerated insulin resistance and low estrogen levels which may contribute to the pathogenesis.

The hormonal imbalance is a prime factor for the aetiopathogenesis of gestational hypertension. Gestational hypertension is a state of hypoestrogenemia⁽⁹⁰⁾. Decreased uteroplacental blood flow which is the main pathophysiological event in gestational hypertension leads to impairment in the formation of Dehydroepiandrosterone sulphate (DHEAS) by fetal adrenal glands. DHEAS is the important source of estrogen in pregnancy, (i.e.) 90% of estrogen in maternal circulation is from fetal DHEAS which is converted to estradiol in placenta⁽⁹¹⁾. Hypoestrogenemia also leads to decreased expression of VLDL/apo E receptors resulting in reduced transport of VLDL to fetal compartment and so there is maternal hypertriglyceridemia. Further LDL taken up by the fetus for the synthesis of DHEA is decreased due to reduced fetoplacental perfusion leading to increased LDL.

Triglyceride represents an important biomarker of CVD risk because of its association with atherogenic remnant particles and apo CIII⁽⁹²⁾₁₄. The elevated triglycerides result in increased atherogenic small dense LDL and decreased HDL levels in Gestational hypertension. This may be the result of increased exchange of TGL into LDL and HDL⁽⁹³⁾. The changes in lipoprotein sub fraction seen in this study were compatible with changes seen in coronary artery disease (Kaaja et al 1995, satar et al 1997). The dyslipidemia of elevated triglycerides and lowered HDL in our study was similar to that of many other studies (94-96)16-18

In the present study uric acid level was also significantly elevated in gestational hypertensive group which is consistent with some workers⁽⁹⁷⁾. The currently favored concept is that increased circulating uric acid is secondary to reduced renal urate clearance, as can be seen with hypervolemia. Elevated serum uric acid in hypertensive pregnant patients is associated with poor perinatal outcomes including small for gestational age (SGA) infants and preterm birth (PTB)^(98,99). 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 with hypoxia. Thus, 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 preeclampsia^(100,101).

Uric acid is also found to be associated with carotid atherosclerosis and its increase is an independent risk factor for cardiovascular diseases which mediate altered vascular function and inflammation. Its elevation may be due to imbalance between oxygen free radicals and NO⁽¹⁰²⁾. Because uric acid is also known to have antioxidant activity in the serum, its level may rise as a compensatory mechanism to counteract the increased oxidative stress under the conditions of metabolic syndrome⁽¹⁰³⁾ or atherosclerosis⁽¹⁰⁶⁾.

Dyslipidemia is evident during the first and second trimester, far preceding the clinical manifestations of preeclampsia⁽¹⁰⁴⁾. HDL-C is reduced at earliest measurement (20 weeks gestation) and then throughout gestation in women who later develop the syndrome, again implicating dyslipidemia in the pathophysiology⁽¹⁰⁵⁾.

Hyperuricemia is associated with poor perinatal outcomes and also associated with atherosclerosis. Both dyslipidemia (elevated triglycerides and decreased HDL) and elevated uric acid predict atherosclerosis and also considered to be having pathophysiological role in the clinical manifestations of preeclampsia which was seen in the present study too..

VI. Conclusion

The present hospital based, non-interventional, cross sectional, case control study was undertaken in the Department of Biochemistry of Burdwan Medical College @ Hospital, 50 Patients, who attended the Department of Obstetrics and Gynaecology, Burdwan Medical College @ Hospital were selected as the cases for the present study and the period of gestation – matched 50 control were selected from the pregnant mothers attended the antenatal clinic of Burdwan Medical College Hospital. The cases were selected by simple random method.

The data obtained were analyzed by SPSS software for study of mean, standard deviation, Student pair t test to find out the significance of difference between the cases and controls.

After result analysis following observations were obtained,

1. Serum uric acid was significantly elevated in cases groups
2. Serum lipid profile was significantly elevated in cases of Total Cholesterol (TC), LDL, VLDL, and Triglycerides and HDL levels were significantly lower in study groups in comparison to control groups.
3. There is no significant difference found in plasma Fasting Blood Glucose levels in both groups.

Lipid profile and serum uric acid measurements in the early 2nd trimester can be suggested as cost-effective markers that may help in the prevention of the complications of gestational hypertension like, eclampsia, intrauterine growth retardation, HELLP syndrome, future cardio vascular risk of the mother for the future development of Hypertension and stroke.

A clinical trial of life style and dietary modification would help in cases of altered lipid metabolism. It has already been suggested in one study that including uric acid in the research diagnosis of preeclampsia identifies a more severe group that is likely to have a more homogeneous pathophysiology than when this marker is not included⁽¹⁰⁷⁾.

However our study is limited, since the outcomes of the pregnancies are not studied. In future similar studies could be conducted on large study groups for a longer duration period with results of the outcome of the pregnancies for better evaluation of the role of serum uric acid and lipid profile are needed to establish their usefulness as reliable and cost-effective biomarkers in gestational hypertension.

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