Original Research

Comparative study of serum lipid profile levels in normotensive and hypertensive pregnant women in third trimester of pregnancyand assessment of correlation between blood pressure and lipid profile level among these subjects

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Abstract

Background: Pregnancy-Induced Hypertension Is One Of The Most Prevalent Diseases During Pregnancy That Leads To Maternal And Fetal As Well As Neonatal Morbidity And Mortality All Over The Globe.Hence, The Present Study Was Undertaken With Aim To Measure Serum Lipid Profile In Mild And Severe Hypertensive Pregnancy And To Compare With Normal Pregnancy.

Materials And Method: The Present Cross-Sectional Study Was Conducted Among 150 Female Subjects Who Were Divided Into Three Groups With 50 Subjects In Each Group For Assay Of Lipid Profile. Group 1 Comprised Of Healthy Non Pregnant Volunteers As Control. Group 2 Comprised Of Apparently Healthy Pregnant Women In 3rd Trimester Of Pregnancy Having Uncomplicated Pregnancy Without Hypertension.Group 3 Comprised Ofpregnant Women In 3rd Trimester Of Pregnancy Having Pregnancy Induced Hypertension (PIH). Post-Hoc Test And Chi-Square Test Was Used For Analysis With *P* Value <0.05 Considered As Significant Value.

Result: TheMost Significant Increase Was In Levels Of Triglyceride (TG) And Total Cholesterol (TC). There Was A Positive Correlation Between Blood Pressure (BP) And Triglycerides Level, BP (Systolic) Increased Corresponding TC Value That Increased Significantly (R = 0.333 And P = 0.000). Similarly, BP (Diastolic) Increased Corresponding TC Value That Increased Significantly. (R = 0.281 And P = 0.001). **Conclusion:**In Hypertensive Pregnant Women, Hyperlipidemia Is More Profound Than In The Normotensive Group. Hence, As A Result We Conclude That Lipids Have An Important Contributory Role In Pre-Eclampsia, And Must Not Be Underestimated During The Hypertensive Diseases Of Pregnancy.

Keywords: Cholesterol; Maternal Hyperlipidemia;Pregnancy-Induced Hypertension (PIH)

Introduction

Pregnancy-Induced Hypertension Is One Of The Most Prevalent Diseases During Pregnancy That Leads To Maternal And Fetal As Well As Neonatal Morbidity And Mortality All Over The Globe [1].MaternalHyperlipidemia Is One Of The Most Consistent And Striking Changes To Take Place In Lipid Metabolism During Late Pregnancy. The

Hepatic Effect Of Estradiol And Progesterone Also Play An Important Role [2].DuringPregnancy, There Is An Increase In The Hepatic Lipase Activity And Decrease In Lipoprotein Lipase Activity[3,4].Therefore, Biochemical Profile Of Such Patients Is Pivotal To Provide Required Adequate Evidence To Clinicians Who Are Endeavouring For Potential Interventions To Reduce This Burden[1].Moreover, Pre-Eclampsia Incidence And Severity May Be Predicted By Plasma Lipid Profiles During The First Trimester Of Pregnancy [5]. Pregnancy-Induced Hypertension (PIH) Appears To Have A Role In The Pathophysiology Of Abnormal Lipid Metabolism. It Is Clear That Lipid Profile Analysis May Have A Role In Diagnosis Given The Correlation Between Blood Lipids And Gestational Proteinuric Hypertension [6]. Pregnant Women's Blood Pressure (BP) Has Historically Been Determined Mostly By A Few Measures Obtained At The Doctor's Office. Even In The Third Trimester Of Pregnancy, These Haphazard Time-Unspecified Data Do A Poor Job Of Selecting A Group For Possible Preeclampsia Diagnosis. Nonetheless, The Foundational Method For Diagnosing Pre-Eclampsia Remains Isolated Blood Pressure Measurement [7]. There Is Ongoing Discussion Over The Function And State Of Serum Lipids In Expectant Mothers [8]. Therefore, The Purpose Of The Current Study Was To Compare Serum Levels Of HDL, LDL, Triglycerides, And Cholesterol In Moderate And Severe Hypertensive Pregnancies To Those In Normal Pregnancies.

Materials And Method

The Present Cross-Sectional Study Was Conducted In The Department Of Biochemistry, In Collaboration With Thedepartment Of PathologyAnd The Department Of Gynaecology In A Tertiary Hospital Over A Period Of One Year. A Total Number Of 150 Female Subjects Aged Between 18 To 35 Years Were Taken For Study And Were Divided Into Three Groups. Group 1 Comprised Of 50 Subjects, Healthy Non Pregnant Volunteers As Control. Group 2 Comprised Of 50 Apparently Healthy Pregnant Women In 3rd Trimester Of Pregnancy Having Uncomplicated Pregnancy Without Hypertension.Group 3 Comprised Of 50 Pregnant Women In 3rd Trimester Of Pregnancy Having Pregnancy Induced Hypertension (PIH).ThePresent Study Was Approved By The Ethical Committee Of The Tertiary Medical Institute. The Approval Was On The Agreement That Patient Anonymity Must Be Maintained, Good Laboratory Practice, Quality Control Ensured, And That Every Finding Would Be Treated With Utmost Confidentiality And For The Purpose Of This Research Only. All Work Was Performed According To The International Guidelines For Human Experimentation In Biomedical Research. Approval Was Obtained From The Subjects By Taking The Informed Consent. Inclusion Criteria Consisted Of Only Those Subjects Who Were Willing To Participate In The Study, Healthy Non Pregnant And Pregnant Women Between The Age Of 18 To 35 Years, Patients With History Of Pregnancy Induced Hypertension (PIH) I.E., BP≥ 140/90 Mm Hg, Proteinuria Of 300 Mg/24 Hour Or More Or 1+ Dipstick Response, Generalized Edema.Exclusion Criteria Consisted Of Pregnant Women With Gestational Diabetes Mellitus, History Of Hypothyroidism, Anemia, Smoking, Alcoholism, HIV And Other Chronic Diseases.Informed Written Consent Both In English As Well As Vernacular Language, Was Taken From The Participants Included In The Study.A Detail History Including Obstetric History Was Taken And A Complete Physical Examination Including Blood Pressure (BP), Weight, Height Was Recorded And Clinical Evaluation Of Each Subject Was Done And Details Were Entered In The Pre Designed Proforma. 5 Ml Of Fasting Venous Blood Was Collected From The Antecubital Vein Under Aseptic Conditions From Each Subject Into Plain Vials. The Blood Was Centrifuged And Serum Separated For Assay Of Lipid Profile.Serum Triglycerides (TG), Total Cholesterol (TC) And HDL Cholesterol (HDL-C) Were Analyzed By Enzymatic Methods With The Help Of GlaxoKits On ERBA Chem-5. Serum LDL Cholesterol (LDL-C) Was 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.During Data Collection Completed Questionnaires Were Checked Regularly To Rectify Any Discrepancy, Logical Errors Or Missing Information. The Statistical Analysis Was Carried Using Statistical Package For Social Services -2.Post-Hoc Test And Chi-Square Test Was Used For Analysis With *P* Value <0.05 Considered As Significant Value.

Results:

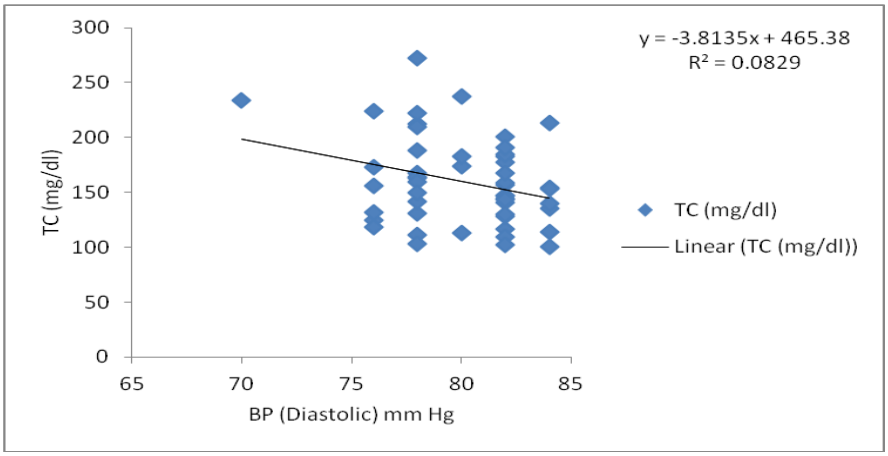
Table/Figure1: Observations Obtained In The Present Study Among Study And Control Group

	Normal Non-Pregnant Women (N=50) (Mean ± SD)	**Normal Pregnant Women (N=50) (Mean ± SD)**	**Hypertensive Pregnant Women (N=50) (Mean ± SD)**	Significance Between Groups
Age (Years)	25.32 ± 5.90	23.26 ± 2.64	24.60 ± 2.77	0.039*
Age Of Menarche (Years)	12.72 ± 1.12	13.20 ± 1.49	13.14 ± 1.45	0.166

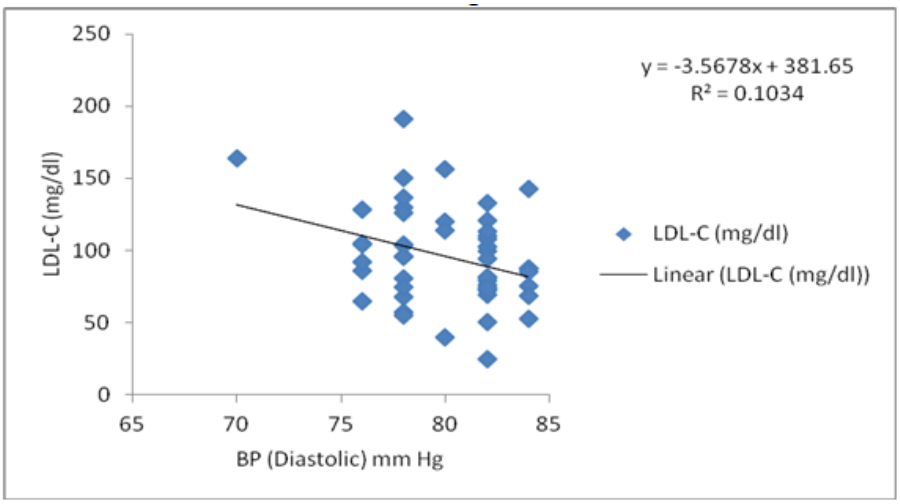
Systolic Blood Pressure (Mmhg)	119.92± 3.74	112.64± 9.42	147.56± 4.54	0.000
Diastolic Blood Pressure	80.04± 2.98	76.40± 5.43	94.28± 2.58	0.000
TC (Mg/Dl)	160.15 ± 39.51	198.34 ± 46.99	230.58 ± 47.82	0.000
TG (Mg/Dl)	104.94 ± 48.25	188.18 ± 36.70	336.03 ± 123.52	0.000
HDL-C (Mg/Dl)	43.08 ± 10.77	52.64 ± 14.09	53.63 ± 13.06	0.000
LDL-C (Mg/Dl)	96.08 ± 33.09	108.06 ± 38.86	109.76 ± 40.93	0.147
VLDL-C (Mg/Dl)	20.98 ± 9.65	37.63 ± 7.34	67.20 ± 24.70	0.000

Table/Figure 2: Correlations Among Various Parameters Of The Present Study

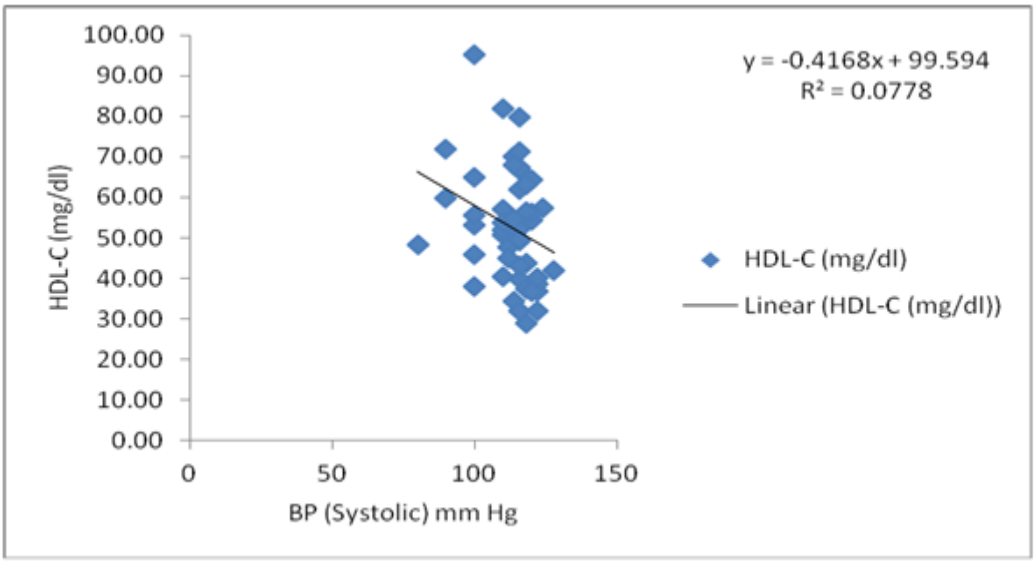
Correlations						
		TC (Mg/Dl)	**TG (Mg/Dl)**	**HDL-C (Mg/Dl)**	**LDL-C (Mg/Dl)**	**VLDL-C (Mg/Dl)**
BP (Sys)	**Pearson Correlation (R)**	0.333(**)	0.581(**)	0.090	0.054	0.581(**)
	Sig. (2-Tailed)	0.000	0.000	0.272	0.514	0.000
BP (Dias)	**Pearson Correlation (R)**	0.281(**)	0.545(**)	0.066	0.013	0.545(**)
	Sig. (2-Tailed)	0.001	0.000	0.419	0.876	0.000
** Correlation Is Significant At The 0.01 Level (2-Tailed).						
* Correlation Is Significant At The 0.05 Level (2-Tailed).						



Table/Figure 3: Correlation Between Blood Pressure (Diastolic) And Total Serum Cholesterol In Normal Non-Pregnant Women



Table/Figure 4: Correlation Between Blood Pressure (Diastolic) And Serum Low Density Lipoprotein-Cholesterol Level In Normal Non-Pregnant Women



Table/Figure 5: Correlation Between Blood Pressure (Systolic) And Serum High Density Lipoprotein-Cholesterol Level In Normal Pregnant Women

Table/Figure 1 Demonstrates Observations Obtained In The Present Study Among Study And Control Group. Mean Blood Pressure (Systolic) Of The Normal Non-Pregnant Women Was 119.92 ± 3.74 Mmhg, Normal Pregnant Women Was 112.64 ± 9.42 Mmhg And In Hypertensive Pregnant Women Was 147.56± 4.54 Mm Hg Which Is Much Higher And The Increase Is Highly Statistically Significant (P=0.000).MeanBlood Pressure (Diastolic) Of The Normal Non-Pregnant Women Was 80.04 ± 2.98 Mmhg, Normal Pregnant Women Was 76.40 ± 5.43 Mmhg And In Hypertensive Pregnant Women Was 94.28 ± 2.58 Mmhg Which Is Much Higher And The Increase Is Highly Statistically Significant (P=0.000).Mean TC (Mg/Dl) For The Normal Non-Pregnant Women Was 160.15 ± 39.51, Normal Pregnant Women Was 198.34 ± 46.99 And In Hypertensive Pregnant Women Was 230.58 ± 47.82. There Was A Highly Significant Difference In These Three Groups (P=0.000).Mean TG (Mg/Dl) For The Normal Non-Pregnant Women Was 104.94 ± 48.25, Normal Pregnant Women Was 188.18 ± 36.70 And In Hypertensive Pregnant Women Was 336.03 ± 123.52. There Was A Highly Significant Difference In These Three Groups (P=0.000).Mean HDL-C (Mg/Dl) For The Normal Non-Pregnant Women Was 43.08 ± 10.77, Normal Pregnant Women Was 52.64 ± 14.09 And In Hypertensive Pregnant Women Was 53.63 ± 13.06. There Was A Highly Significant Difference In These Three Groups (P=0.000).Mean LDL-C (Mg/Dl) For The Normal Non-Pregnant Women Was 96.08 ± 33.09, Normal Pregnant Women Was 108.06 ± 38.86 And In Hypertensive Pregnant Women Was 109.76 ± 40.93. The Difference Was Not Significant In These Three Groups (P=0.147).Mean VLDL-C (Mg/Dl) For The Normal Non-Pregnant Women Was 20.98 ± 9.65, Normal Pregnant Women Were 37.63 ± 7.34 And In Hypertensive Pregnant Women Was 67.20 ± 24.70. There Was A Highly Significant Difference In These Three Groups (P=0.000).Table/Figure 2 Demonstrates Correlations Among Various Parameters Of The Present Study. There Was Positive Correlation Between BP (Systolic) And TC Level. Result Shows That When BP (Systolic) Increased Corresponding TC Value Also Increased Significantly (R = 0.333 And P = 0.000). There Was Positive Correlation Between BP (Systolic) And TG, VLDL-C Levels. It Was Found That When BP (Systolic) Increased Corresponding TG And VLDL-C Values Also Increased Significantly (R = 0.581 And P = 0.000). On Evaluating The Correlation Between BP (Systolic) And HDL-C Level, We Found That There Was A Positive Correlation Between Two Variables But Correlation Was Not Statistically Significant (R = 0.090 And P = 0.272). On Further Evaluating The Correlation Between BP (Systolic) And LDL-C Level, We Found That There Was A Positive Correlation Between Two Variables But Correlation Was Not Statistically Significant (R = 0.054 And P = 0.514)ThereWas Positive Correlation Between BP (Diastolic) And TC Level. Result Shows That When BP (Diastolic) Increased Corresponding TC Value Also Increased Significantly. (R = 0.281 And P = 0.001). There Was Positive Correlation Between BP (Diastolic) And TG, VLDL-C Levels. Result Shows That When BP (Diastolic) Increased Corresponding TG And VLDL-C Values Also Increased Significantly. (R = 0.545 And P = 0.000). When We See The Correlation Between BP (Diastolic) And HDL-C Level We Found That There Was A Positive Correlation Between Two Variables But Correlation Was Not Statistically Significant (R = 0.066 And P = 0.419). When We See The Correlation Between BP (Diastolic) And LDL-C Level We Found That There Was A Positive Correlation Between Two Variables But Correlation

Was Not Statistically Significant (R = 0.013 And P = 0.876)CorrelationBetween Blood Pressure (Diastolic) And Total Serum Cholesterol In Normal Non-Pregnant Women As Illustrated In Table/Figure 3 Reports The Value Of 'R' As -0.29. Although Technically A Negative Correlation, The Relationship Between These Variables Is Only Weak. The Value Of R^2, The Coefficient Of Determination, Is 0.082. The P Value Is 0.041. The Result Is Significant At P<0.05.Correlation Between Blood Pressure (Diastolic) And Serumlow Density Lipoprotein-Cholesterol Level In Normal Non-Pregnant Women As Illustrated In Table/Figure 4 Reports That The Value Of 'R' As -0.32. Although Technically A Negative Correlation, The Relationship Between These Variables Is Only Weak. The Value Of R^2, The Coefficient Of Determination, Is 0.103. The P Value Is 0.023. The Result Is Significant At P<0.05. Correlation Between Blood Pressure (Systolic) And Serum High Density Lipoprotein-Cholesterol Level In Normal Pregnant Women As Illustrated In Table/Figure 5 Reports Value Of 'R' As -0.28.Although Technically A Negative Correlation, The Relationship Between These Variables Is Only Weak. The Value Of R^2, The Coefficient Of Determination, Is 0.077. The P Value Is 0.048. The Result Is Significant At P<0.05.

Discussion

Reduced Uteroplacental Perfusion Is The Main Cause Of Fetal Compromise; Preeclampsia Is The Largest Cause Of Maternal Mortality In Affluent Nations And Is Linked To A Five-Fold Increase In Perinatal Mortality [9]. The Prevalence Of PIH Across The Country In India Is 15.2%. As Far As We Now Know, Dyslipidemia And Elevated Oxidative Stress Play A Significant Role In The Preeclampsia Pathogenesis. Metabolic Disorders Common To Preeclampsia And Cardiovascular Disease (CVD) Can Cause Preeclampsia And CVD At Different Stages Of A Woman's Life. On The Other Hand, Preeclampsia Alone May Cause Vascular And Metabolic Alterations That Raise The Affected Women's Total Future Risk For CVD. In Preeclampsia, Serum Lipid Levels Rise Noticeably [10].AllLipid Components Rise Throughout Pregnancy In Step With The Growing Gestational Age. According To Reports, The Rise In Progesterone And Estrogen Levels During Pregnancy Is What Caused This Surge. Pregnant Women With Hypertension Have Larger Lipid Increases Than Pregnant Women With Normotension, Although There Is No Relationship Between Lipid Levels And The Prognosis Of The Condition [11]. In Comparison To Typical Pregnant Women In Their Third Trimester, We Found A Substantial Increase In The Fasting Triglyceride (P=0.000), Total Cholesterol (P=0.000), And VLDL-C Levels In PIH (P=0.000) In Our Study. Pregnancy Raises The Content Of All Lipoprotein Components, According To Knopp RH Et Al. [12]. With Peak Levels At Term, VLDL Cholesterol, Triglycerides, And LDL Cholesterol Increase 2.5-Fold And 1.6-Fold, Respectively, Over Pre-Pregnancy Levels. Midway Through Pregnancy, HDL Cholesterol Reaches Its Maximum Elevation Of 1.45 Times And Then Starts To Decrease To 1.15 Times At Term. In A Research By Lima VJ Et Al. [13], The Preeclamptic Patients Had Triglyceride And VLDL Contents That Were Noticeably Higher Than Those Of The Healthy Women. In A Related Study, Yadav S Et Al. [14] Found That The Study Group, Or Women With Preeclampsia, Had A Significant Decrease In HDL-C Compared To Controls, Or Women With Normotensive Pregnancy, And A Significant Increase In Serum Total Cholesterol (TC), Triglycerides (Tgs), Low-Density Lipoprotein-Cholesterol (LDL-C), High-Density Lipoprotein-C (HDL-C), And Very LDL-C (VLDL-C). According To Maksane S Et Al. [15], Pre-Eclampsia Was Associated With Considerably Higher Levels Of TG And VLDL-C, LDL-C, And HDL-C Compared To Normal Pregnancy. TG, VLDL-C, HDL-C, And LDL-C Values Were Lower In Normotensive Pregnant Women Than In Healthy Non-Pregnant Women. However, There Was No Statistically Significant Change In Total Cholesterol Or HDL Cholesterol Levels. The Preeclampsia Group Was Linked To A Significant Increase In Triglyceride And VLDL Cholesterol And A Reduction In HDL Cholesterol Concentration, While Eclamptic Women Showed A Significant Decrease In HDL Cholesterol And An Increase In LDL Cholesterol. This Similar Study By De J Et Al. [16] Also Reported A Significant Elevation In Serum Triglyceride And VLDL Cholesterol Levels As Well As A Reduced LDL Cholesterol Level In Normal Pregnancy. In Comparison To Women Who Are Not Pregnant, There Is An Increase In Triacylglycerol And Cholesterol Levels In VLDL, LDL, And HDL During The Third Trimester Of Pregnancy. Increased Lipolytic And Decreased Lipoprotein Lipase Activity In Adipose Tissue Are The Mechanisms Causing These Alterations. Progesterone And Estradiol's Hepatic Effects Are Also Significant [2]. Though The Processes Underlying These Lipoprotein Alterations Remain Unclear In Humans, Hypertriglyceridemia In Animal Models Is Linked To Increased Circulation Of Low-Density Lipoprotein (VLDL). Furthermore, Although Triglyceride Transport Is Not Decreased During Pregnancy, Decreased Adipose Tissue Lipoprotein Lipase (LPL) Activity In Late Gestation May Lead Triglyceride Fatty Acids To Be Rerouted To Other Tissues, Such As Muscle And The Uterus, For Oxidation Rather Than Storage. It Seems That Sex Hormones Are The Cause Of All Of These Alterations [12]. In The Current Investigation, There Was A Positive Association Found Between TC, TG, And VLDL-C Levels And BP (Systolic). It Was Discovered That There Was A Significant Increase In TC, TG, LDL-C, And VLDL-C Values In Correlation With An Increase In BP (Systolic). The Diastolic

Blood Pressure And The Temperature Coefficient (TC) Also Exhibited A Positive Link, Meaning That When The Diastolic Blood Pressure Rose, The TC Value Likewise Climbed Noticeably. Comparing Pregnant Women With Pregnancy-Induced Hypertension To Those With Normotension, A Research By Areda BG Et Al [1] Found That Lipid Abnormalities And Higher Systemic Inflammatory Markers Were Detected In The Former Group. Furthermore, The Study's Findings That Women With PIH Who Had Significant Dyslipidemia Were More Likely To Develop Hypertension [1] Corroborated Our Findings. The Current Study Emphasizes That Evaluating These Possible Biomarkers During The Early Stages Of Prenatal Care Services May Offer A Benefit For Pursuing PIH Interventions.Given That Pregnancy Is Linked To Hyperoestrogenemia, Oestrogen May Be The Primary Modulator Of Hypertriglyceridemia. OestrogenPrevents The Liver From Oxidizing Lipids, Which Increases The Amount Of Free Fatty Acids That Are Delivered To The Liver For The Production Of Endogenous TG, Which Is Carried By VLDL. But As Oestrogen Levels Fall In Pregnancy, Hypertriglyceridemia In Preeclampsia Is Most Likely Not Caused By Hyperoestrogenaemia [17].Furthermore, There Is An Overall Rise In Plasma Lipids During Pregnancy Due To Hormonal Changes Brought On By Progesterone, Estrogen, And Human Placental Lactogen. Severe Hypertriglyceridemia Results From These Alterations In Females With Aberrant Lipoprotein Metabolism [17]. Thus, Oestrogen Is In Charge Of Serum Suppression And The Induction Of TG And HDL. Thus, Hyperoestrogenemia Can Account For The Rise In HDL-C And Fall In LDL-C In Normotensive Pregnant Women. Because Oestrogen Levels Drop During Hypertensive Pregnancy, Hypostrogenemia May Be The Cause Of Decreased HDL-C And Elevated LDL-C Levels. The Decreased Amount Of Prostacyclin Observed In Hypertension Could Potentially Be Attributed To This Decreased HDL-C Level. Reduced Antioxidative Protection For Other Lipoproteins May Also Result From Lower Serum HDL-C Levels [15].LipidsHave A Part In Endothelial Dysfunction, Which Is The Most Significant Event In The Pathophysiology Of Preeclampsia. Preeclampsia Is Characterized By Elevated Levels Of Free Fatty Acid Flow, Triglycerides, LDL, HDL, Total Lipid, Cholesterol, And VLDL. Triglycerides, LDL, And VLDL Are Stored In The Liver Along With Disruptions To Beta Oxidation.AnRise In Plasma Lipid Levels Is Accompanied By An Increase In Cytokines And Lipid Peroxidase. Vasoconstriction Happens Throughout The Body As A Result Of Direct Or Indirect Disruption Of Endothelial Cells [11]. Pregnant Women Who Have An Aberrant Lipemic Response But A Normal Pregnancy Outcome May Benefit From Endothelial Dysfunction-Preventing Factors (E.G., Superior Placentation Or Genes Favoring Endothelial Resistance).To What Extent An Aberrant Response To Pregnancy In Terms Of Carbohydrates Or Lipoproteins Can "Flag" Women Who Are At Risk Of Cardiovascular Dysfunction After Pregnancy Has To Be Investigated Further. If These Risk Factors Are Identified In Pregnant Or Postpartum Women, Early Therapies (Antioxidants, Exercise, Etc.) May Be Able To Reduce Or Postpone Future Cardiovascular Morbidity And Death [18].

Conclusion

We Conclude That In Both Normotensive And Hypertensive Patients, All Lipid Fractions Except LDL-C Increase During Pregnancy. In The Normotensive Group, Hyperlipidemia Is Considerable Throughout The Third Trimester. Triglyceride And Total Cholesterol Levels Have Increased The Greatest. Pregnant Hypertensive Women Experience More Severe Hyperlipidemia Than Their Normotensive Counterparts. This Is Consistent With The Disease's Prognosis As Well. Therefore, It Can Be Concluded That Fats Have A Significant Role In Pre-Eclampsia And Should Not Be Disregarded While Treating Hypertension Illnesses In Pregnancy.

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