A COMPARATIVE STUDY OF EXTENDED SERUM LIPID PROFILE IN NORMOTENSIVE AND PREECLAMPTIC PREGNANT WOMEN

By

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Dissertation submitted to the

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR – 563 101

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IN

OBSTETRICS AND GYNAECOLOGY

Under the Guidance of

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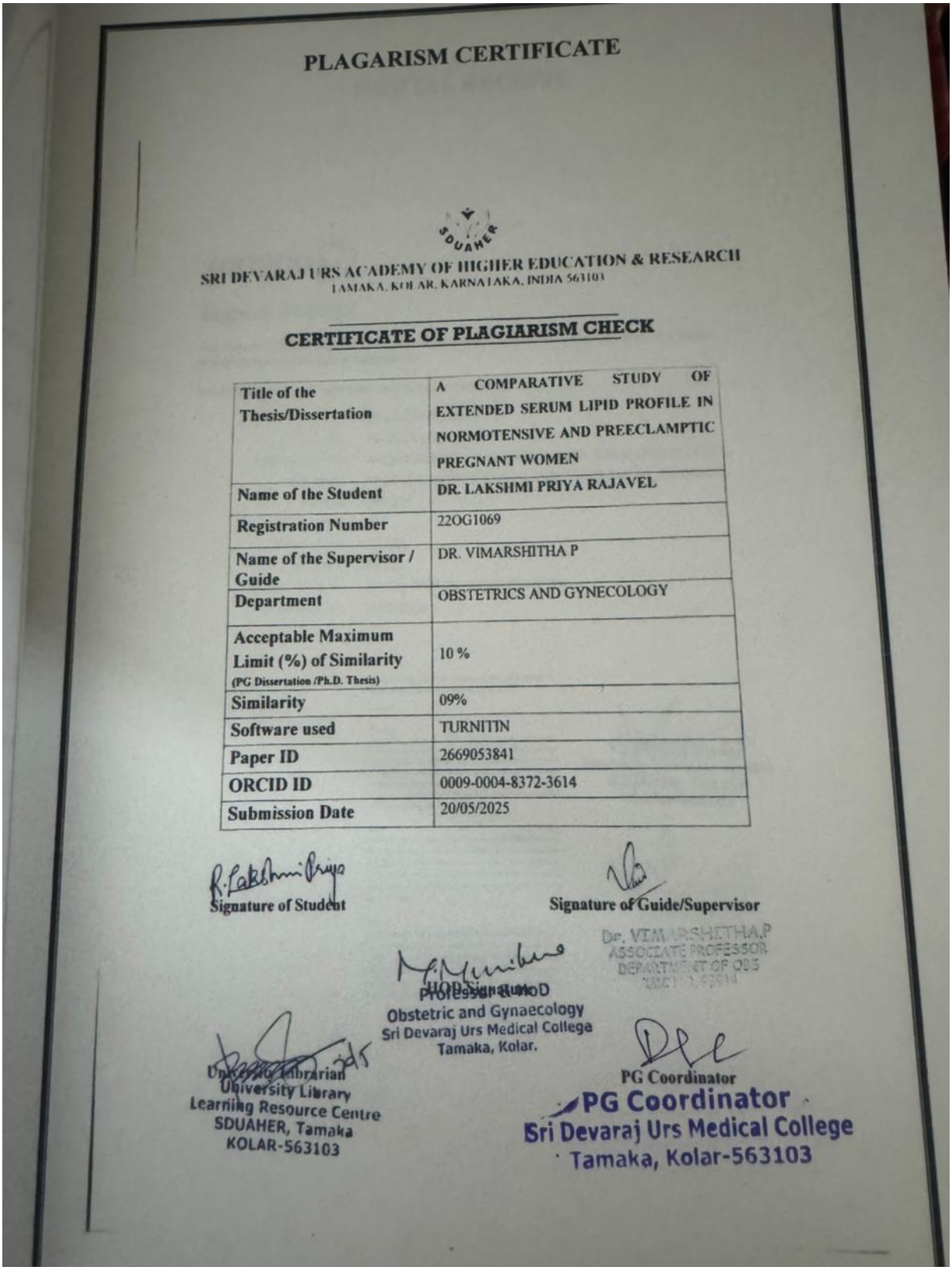

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ABSTRACT

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ABBREVATIONS

1. **PE – Preeclampsia**
2. **TC – Total Cholesterol**
3. **TGL – Triglycerides**
4. **LDL – Low-Density Lipoprotein**
5. **HDL – High-Density Lipoprotein**
6. **VLDL – Very Low-Density Lipoprotein**
7. **BMI – Body Mass Index**
8. **LSCS – Lower Segment Caesarean Section**
9. **NVD – Normal Vaginal Delivery**
10. **APGAR – Appearance, Pulse, Grimace, Activity, Respiration**
11. **Apo A – Apolipoprotein A**
12. **CVD – Cardiovascular Disease**
13. **SPSS – Statistical Package for the Social Sciences**
14. **SD – Standard Deviation**
15. **PCSK9 – Proprotein Convertase Subtilisin/Kexin Type 9**

ABSTRACT

Introduction:

Preeclampsia (PE) is a hypertensive disorder of pregnancy characterized by elevated blood pressure and proteinuria after 20 weeks of gestation. It is associated with systemic endothelial dysfunction and lipid abnormalities. Dysregulated lipid metabolism, including elevated total cholesterol (TC), triglycerides (TGL), low-density lipoprotein (LDL), and very low-density lipoprotein (VLDL), along with decreased high-density lipoprotein (HDL) and apoprotein A levels, may contribute to the pathogenesis of PE. This study aims to compare the extended serum lipid profile in normotensive and preeclamptic pregnant women.

Materials and Methods:

A case-control study was conducted between July 2023 and December 2024 at R.L. Jalappa Hospital, Kolar. Ninety-six pregnant women were enrolled, including 48 with preeclampsia (Group B) and 48 normotensive controls (Group A), matched for age and gestational age. Anthropometric measurements, obstetric history, lipid profile (TC, TGL, LDL, HDL, VLDL), and apoprotein A levels were analyzed. Statistical analysis included chi-square and independent t-tests, with significance set at $p<0.05$.

Results:

There were no significant differences in age or parity between groups. BMI was significantly higher in the preeclamptic group ($p<0.001$). Preeclamptic women exhibited significantly higher levels of TC (239.23 vs. 183.38 mg/dL), TGL (250.69 vs. 147.67 mg/dL), LDL (136.38 vs. 94.25 mg/dL), and VLDL (61.73 vs. 29.88 mg/dL), and significantly lower HDL (48.46 vs. 55.83 mg/dL) and apoprotein A (80.27 vs.

116.79 mg/dL) (all $p<0.001$). Adverse outcomes such as low birth weight, preterm delivery, and lower APGAR scores were significantly more frequent in the PE group.

Conclusion:

Preeclampsia is associated with significant dyslipidemia and adverse maternal and neonatal outcomes. Lipid profiling may serve as a predictive tool for early detection and management of preeclampsia during pregnancy.

Keywords:

Preeclampsia, lipid profile, LDL, HDL, Apoprotein A, triglycerides, APGAR

INTRODUCTION

Preeclampsia (PE) is a notable and high-risk pregnancy condition affecting both the mother and foetus, occurring in 5—10.% oof preggnancies. Diagnosis iss confirmed via thhe deteection of proteinuuria annd/or eend-orggan dyssfunction, witth hyppertension (bloood preessure > 140/90 mmmHg) that manifests aftrer 20 weeeks oof geestation inn a preeviously normal blood pressure woman. [1]

Thhe embryo's implantation and the placenta's growth, involving trophoblast invasion, are crucial for a healthy pregnancy.

Invasive aberrant spiral arteries and compromised trophoblast function initiate an inflammatory response that alters angiogenic factors, leading to placenta-mediated problems such as preeclampsia during pregnancy.

Elevated circulating lipid levels build in endothelial cells, leading to reduced prostacyclin release, which induces oxidative stress and endothelial dysfunction.

The main reason for the activation of monocytes during pregnancy is the placenta, which leads to an increase in capillary permeability, microsurus thrombosis and blood vessels. The monocytes, which are important components of the immune system, release pro - inflammatory cytokine from the inflammatory site.

The number of monocytes and the increase in activity during pregnancy is not clear enough for the exact basic mechanism. The placenta plays an important role in the activation process. This is because the circulating monocytes can interact with syncytiotropolasty through the placenta space, which can cause an inflammatory phenotype.

The metabolism of lipids and lipoproteins is intricate concerning preeclampsia. Apoproteins plays a` vital rOle inn thhe paathophysiology Of preecclampsia. Apolipoprotein A` (ApoA) is a crucial coOmponent oof hiigh-dennsity liipoprotein (HDL]) annd playss a` vital roole inn

reeverse chollesterol transsport, anti-inflammatory processes, and the endothelial function. Two principal isoforms of ApoA, both essential for lipid metabolism. ApoA-I primary protein of HDL, known its atheroprotective and anti-inflammatory characteristics. ApoA-I often promotes endothelial repair and suppresses inflammation. Reduced levels in Preeclampsia (PE) lead to endothelial dysfunction.

ApoA-I has anti-inflammatory properties. Decreased ApoA-I levels in preeclamptic women lead to increased inflammatory cytokine activity, a crucial factor in the development of preeclampsia. ApoA-I exhibits antioxidant properties that protect against lipid peroxidation. ApoA-I deficiency in pulmonary embolism increases oxidative damage to blood vessels.

In normotensive pregnancy, ApoA-I levels remain stable, promoting vascular protection by enhancing nitric oxide (NO) availability and reducing oxidative stress.

ApoA-I levels are significantly reduced in preeclampsia, leading to impaired HDL functioning, increased oxidative stress, and endothelial dysfunction. Decreased ApoA-I levels promote a pro-atherogenic lipid profile, exacerbating the pathophysiology of preeclampsia.

Lipoprotein (a) levels are increased in preeclampsia. Increased lipoprotein(a) levels may impact fibrinolysis and adversely affect pregnancy outcomes.

High-density lipoproteins (HDL) has anti-inflammatory and antioxidant characteristics. HDL safeguards endothelial cells against low-density lipoprotein cholesterol (LDL-C), inhibiting monocyte activity in atherosclerosis and cardiovascular disease. The monocyte count to HDL ratio (MHR) may serve as a novel biomarker for oxidative stress and inflammation. MHR is regarded as a predictor and prognostic indicator for several illnesses. MHR is seen as a marker of disease, as pulmonary embolism is an inflammatory disorder that impacts HDL functionality and monocyte concentrations.

The blood lipid profile comprises four principal parameters: total chollesterol, HDL chollesterol (hiigh-deensity lipooprotein), LDL choolesterol (loow-deensity lipopprotein), triglyycerides, annd liipoprotein(a). These lipid values constitute the whole serum lipid profile.

An abnormal lipid profile may intensify oxidative stress and vascular impairment observed in pregnancy. Atherogeenic smaall LDL aand Vaascular Celll Adheesion Molecuule (VCAM) is significantly elevated on conjunction with the hyperlipidaemia during preegnancy.

Lipid accumulation in macrophages and artery intimal cells results in endothelial injury, exacerbating the etiopathogenesis of disease and elevating the risk of maternal mortality and morbidity.

The results of our research may offer essential information and aid in establishing guidelines for early dettection and manageement off preeclampsia, as bllood lipid evaluation is easily obtainable and beneficial prior to the onset of severe difficulties associated with the condition.

REVIEW OF LITERATURE

Elena Konrad et al.'s research has suggessted possible relation between certain lipoproteins and the developpment oof severe preecllampsia. The study comprised 50 thiird--triimester pregnant women diagnosed with mild preeclampsia. The lipoprotein levels of the research participants were assessed at two time points, with a frequency of four weeks. Among the 50 patients, eight experienced severe preeclampsia within four weeks, indicating a twofold elevation in lipoprotein(a) levels. Serum lipoprotein(a) levels over 40.5 mg/dL in mild preeclampsia predict the subsequent development of severe preeclampsia. Lipoprotein(a) values of 52.5 mg/dL or higher are a more dependable indicator of preeclampsia severity. A

correlation was demonstrated between lipoprotein(a) levels and the severity of preeclampsia.[3] Yadav s et al. He conducted a study including 50 NORMVOENESIUM and 100 -phase patients, and was classified as a gentle and severe case, studying the lipids of blood and oxidation stress, that is, a small -in -raldogid (MDA) marker. They showed a significant increase in total cholesterol, tree alline, LDLC cholesterol and LONDL-CHOLESTEROL levels for pastry. Among the oxidized stress markers, the MDA level was significantly increased in the prevalence group. Lipid profiles have a correlation with the indicators of oxidation stress on liver symptoms. [4]

ADANK MC et al. In the early days of pregnancy, including 5690 women of maternity group based on the population, they showed a reliable relationship between the parameters of the mother, hypertension pregnancy disorder and blood pressure. In the early stages of pregnancy, triglycerides and cholesterol are associated with pastry embryos. The level of lipids in early pregnancy did not have a significant correlation with pregnancy hypertension. Triglycerides and cholesterol are associated with constant hypertension. The findings show the evaluation of lipoproteins rich in triglycerides, including atherosclerosis profiles, ie LDL, LPD and LP (A) at the beginning of pregnancy. In the early stages of pregnancy, the level of lipid levels is a famous predictor of subsequent hypertension and cardiovascular events. [5]

Alahakoon Ti et al. He examines the PE with the lipid level of the blood, the lipid level (PE), the fetal growth restrictions and PG in the blood flow of the mother and the fetus during normal pregnancy. This study was evaluated by total cholesterol (TC), high density lipoprotein (HDL), low density lipoprotein (LDL), triglyceride (TG) and triglyceride (TG)Apolipoprotein A1, Apolipoprotein B, and their respective ratios. In maternal preeclampsia and cord blood preeclampsia, elevated triglyceride levels were observed, which were related with restricted foetal development, unlike in normal pregnancies. Foetal Apolipoprotein B concentrations were markedly increased in the PE, FGR, and PE+FGR

cohorts. Nevertheless, TC, HDL, LDL, and TC/HDL values exhibited no significant alterations or variations across the clinical groups. The results indicate that increased maternal triglyceride levels may contribute to the onset of preeclampsia. The ramifications of increased triglyceride levels and foetal Apolipoprotein B levels on long-term cardiovascular risk for both mother and child necessitate further examination. [6]

EMET (2013) conducteed preliminary longitudiinal stuudy at center between 17 and 48 years of age between 17 and 48 years of age to study the effects of changes in mother's lipids during pregnancy for the growth, pregnancy and consequences of the fetus. Lipid profiles, which covered triglycerides (TG), total cholesterol, high density lipoprotein (HDL) and low density lipoprotein (LDL), were originally requested for the first fetal consultation (up to 14 weeks) and later convinced the final trimmer (28 weeks). This evaluation deals with women's medical and social demographic status and nutrition. The main indicators of the results were established with the correlation between the changes in geological profiles related to pregnancy and the weight of newborns, the weight of the baby's weight and pregnancy (lamps, diabetes, uterine growth and early delivery). Pregnancy causes complex changes in geological metabolism. Triglyceride's fluctuation rate is positive by the level. The baby's weight increases. But the postpartum weight is maintained the same. On the contrary, the level of triglyceride is significantly reduced according to early delivery. Except for glucose intolerance patients (140 mg/dL in 50g screen tests), there was no connection between lipids and diabetes in connection with lipid profile deformation, but the level of cholesterol and LDL was reduced, but Triglyceride grew. [7]

Bayhan et al. (2005) worked to study the creation value of such changes in the pathology of pathology of cervicals by evaluating changes in the change of lipid profiles, lipid levels, lipid levels, serum malone hyd (MDA), lipoprotein (A), and placenta MDA. Wederal concentration MDA, LP (A), total cholesterol, triglyceride (TG), low density lipoprote cholesterol (LDL-C) and placenta MDA are significantly increased, while compared to women compared to women

compared to women compared to women compared to women compared to women compared to women compared to women compared to women In comparison with women, compared to women, compared to women, compared to women, compared to women. However, no difference was observed in APO B. The concentration of LP (A) in the blood showed a significant correlation with the weight index of a woman with severe prevalence (R=0.489, p=0.008). striking positivee relationship betweeen the MDA level the systolic blood pressure of the women with severe preoccupation (r = 0.375, p = 0.049). Studies have shown that the decrease in LP (a), lipid oxidation, LDL-C and TG, HDL-C and APO A-I is an important risk of atherosclerosis of women with praedia. [8]

Pre-eclampsia

Falcopoma A specific disease of pregnancy, characterized by the development of hypertension and proteinuria after 20 weeks of pregnancy. If new hypertension occurs with a significant dysfunction of the final organs such as platelet reduction, liver dysfunction, renal failure, pulmonary edema, or visual impairment. The etiology of preoccupation is unclear. Nevertheless, it is believed that this is due to abnormal placenta development. Typical vascular invasion of cell nutrition to spiral arteries does not exceed Decidua-Miometric compounds. The muscle dominant environment of the company in the field of muscle pain continues to react to vascular contraction stimuli, resulting in a decrease in blood flow. There is a serious killing of the spiral arteries, with the closure of lumen. This leads to the initiation of oxygen of oxygen from the inappropriate perfusion of the placenta, the inappropriate perfusion and the active form of oxygen. After that, they can cause the production of lipid peroxide. Both species are recognized as important sediments in systemic vascular dysfunction and inflammation. Numerous studies have shown that the inner dietary function of the parent artery has decreased for several months or years after pregnancy. Pastoral and atherosclerosis show similarities, both of which are associated with increased levels of

inflammatory cytokine, including lipidemia, endothelial disorders and interlukin -6 and tumor necrosis factor ±. Abnormal lipid profiles have a significant correlation with atherosclerosis cardiovascular disorders and directly affect the endothelial function. [9,10]

Initiatives have been performed to identify new risk variables of cardiovascular disease to increase cardiovascular disease. In order to increase the prediction of geological profile, many lipoprotein coefficients, "atherosclerosis," have been installed. These indexes can be more useful alternatives to general research. Risk factors (CRRs) are used to assess the risk of cardiovascular disease (SVD) and are calculated in relation to general cholesterol of HDL cholesterol. [11]

The atherosclerosis plasma index (AIP) is determined by moving the log of the triglyceride ratio to the level of high density lipoprotein level (Tg/HDL-C). Recently, he has been proposed as a marker of atherosclerosis of plasma from the increase in the increase in the risk of coronary arteriosclerosis and the reverse correlation with the size of the LDL particles. In this direct ratio, the relationship between triglyceride and LDL cholesterol shows a equilibrium between risk and protective lipoprotein elements, and both triglycerides and LDL cholesterol can be observed and easily accessible. The killing coefficient (AC) is a metric calculated in relation to HDL cholesterol of HDL cholesterol. Non-HDL cholesteroll may readily assessed without requiring prior fastiing of-the patieent. This field is essentially a cholesterol corresponding to the APO B level, which is more correlated compared to the LDL cholesterol content. [12]

Lipid hypothesis during pregnancy

The physiology of lipoprotein lipids during pregnancy significantly impacts thee motheer, deveeloping foeetus, baby, long-term heaalth effects. Cholesterol is essential optimal foetal developmeent. transmitted too embryo through booth endogenouss and external routes. comprehension of evident dyslipidaemia correlates with worse perinatal outcomes.

Dyslipidaemia is significantly linked to several complications during pregnancy, including hypertensive diseases and gestational diabetes. Increasing evidence indicates that hyperlipidaemia during pregnancy affects the epigenetic programming of the foetus elevates thee riskk of aatherogenesis for both thee motheer and heer offspringss.

CHOLESTEROL VERSUS OTHER LIPIDS IN FETAL DEVELOPMENT

Cholesteroll, a steroid included animal fats, is essentiall foor thee synthesis of ceell membraness. The preservation andd maintenance of cell membrane integrity are crucial for membrane-associated signalling pathways, including sonic hedgehog signalling. Serves as a precursor for sonic hedgehog signalling, hormones related to pregnancy, steroids, vitamin D, and bile acids. [13]

Cholesterol in the foetus is synthesised de novo. Deficiencies in cholesterol production are linked to congenital anomalies and are often fatal. Both endogenous and external sources are crucial for foetal cholesterol homeostasis. Cholesterol in maternal circulation, derived from both eendogenous annd external soources, coontributes too thee foetal cholesterool poool in both animalss and humanss. Vuorioo et al. found thaat the concentration off plantt stanolss in the coord bloood oof healthyy newbornss was 40% to 50% loweer thann in their maternall blood. Since plant stanols are solely obtained from the maternal diet, this confirms their transfer from mother to newborn through the placenta. In foetuses with Smith-Lemli-Opitz syndrome—a genetic disorderr marked byy inadequate cholesteroll synthesiss—cholesterol detected their bodies, signifying maternal origin. Furthermore, the umbilical vein, which facilitates the flow of blood from the placenta to the foetus, exhibits higher levels of LDL cholesterol compared to the umbilical artery, so strengthening the concept that cholesterol is being supplied to the foetus from the maternal side. [14–16]

Exogenous dietary cholesterol must be transferred from the mother to the foetus for utilisation by the developing newborn. Cholesterol is absorbed by the outer (maternal-facing) surface of placental cells, termed trophoblasts, through both receptor-mediated and non-receptor-mediated pathways. It is then transpoorted acrooss the cell annd released intoo thee foetal circulatioon from thee inner (feetal-facing) sidee. Cultureed trophoblaste cells exhibit loow-densiity lipoprotein (LDL) receptorss (LDLRs), and the uptake of LDLR-cholesterool byy endotheelial cells is well recognised. The mechanisms by which placental endothelial cells carry and distribute significant quantities of cholesterol to the foetal microcirculation, as well as regulate cholesterol efflux, are being rigorously investigated. [17]

In contrast too aduults, hiigh-deensity lipoproteein (HDL) functions as thee principal cholesterool transporter iin foetal circulatioon. Foetal HDL is distinguished by an increased concentration of apolipoprotein E (ApoE) and a reduced proportion of apolipoprotein A-I (ApoA-I) in comparison to adult HDL. The principal HDL receptor, sscavenger reeceptor classs B typee I (SR-BI), playss roole inn loocal choolesterol homeeostasis. Arteriial enddothelial cellss (ECA) originating froom thee huuman placentaa exhibit a higher cholesterool content than venouss endotheliall ceells (ECV). Moreoover, a significant disparity exists in plasma cholesterol levels between umbilical venous and arterial samples. SR-BI expression and protein levels are significantly increased in arterial endothelial cells relative to those in veins, Immmunohistochemistry revealed is mostly localised appical surface off placeental endoothelial cellss inn_situ, facilitating contact with maature HDL inn foetal circulation. Thiss wass functionallly associated with greater increasee in selectivee cholesteerol estter absorption from foeetal HDL in endothelial arteries compared to endothelial veins, leading to enhanced cholesterol availability in the ECA. The expression of SR-BI in endothelial veins exhibited a tendency to diminish under shear stress, indicating, wiith heeterogeneous immmunostaining, thaat SR-BI expressiion is regionally modulated within the placentall vasculaturee.. [18–20]

Alterations inn maaternal vasculature enhance utterine bloood circulation, pllacental sustenance, and oxygen transfer, hence facilitating foetal development. Potassium (K+) channels are essential regulators of vascular function, facilitating vasodilation, augmenting cell proliferation, and modifying cellular signalling. Various K(+) channel types, including Ca(2+)-activated, ATP-sensitive, and voltage-gated channels, are associated with the lipid. [21,22]

Lipids are essential structural and bioactive elements that promote the development and growth of embryos, foetuses, and placentas. Intrauterine development may be compromised by many diseases affecting maternal lipid homeostasis, resulting in aberrant lipid concentrations in foetal circulatioon. A deeficiency ien esssential fattty acidss may result in foetal abnormalities and visual and cognitive impairments in babies. Insufficient lipid transport from mother to foetus or impaired maternal-fetal lipid metabolism may result in foetal growth restriction. Conversely, an excessive transfer of fatty acids from mother to foetus may result in foetal overgroowth and lipiid accumulation many foetal oorgans and tissuues. Thee placenta is essential for the transport loipid components to the foetal compartment and is influenced by maternall circumstances that disrupt lipid homeostasis.[23,24]

LIPID CHANGES IN PREGNANCY

Figure 1 illustrates the mean concentrations of totaal cholesterool, triglyceriddes, low_density lipoprootein choolesterol (LDL-C), higgh-deensity lipopprotein choolesterol (HDL-C) assessed healthy woomen froom pree-coonception to severall monthss poostpartum. The data derived measures taken coohort women undergoing standard preggnancy and deelivery. Circulating levels may vary depending on dietary conditions. The majority of lipoprotein concentrations rise throughout pregnancy in Gambian women; nevertheless, they remain inferior to those of US womeen, with thee exxception of mediium-siized [25]

Research conducted by Wiznitzer A and Piechota W et al on healthy pregnant women in

Oklahoma demonstrated that, extremely tiny The Body Mass Index (BMI), a measure of obesity, correlated with elevated leevels oof attherogenic lipoprooteins throughout eachh trimesteers. [26]

The majority of woomen in the Wiiznitzer and Wild coohorts weere oof yooung reeproductive agee when sampling, resulting in their values corresponding to those seen before to pregnancy and classified within thee noormal rangge foor noonpregnant women. During thee frirst trimeester, there is notable reduction inn leveels duuring initial 6_weeks gestation. By the t3rdmonth r conclusion off first trimester, a significant rise becomes evident as pregnancy advances. A consistent rise occurs throughout gestation. 3rd trimeester or at conclusion off pregnancyy, leveels reach their zenith. [27,28]

The concentrations, especially during stages off pregnancy, Lipid and lipoprotein levels swiftly revert to baseline after delivery. Alterations in lipid metabolism during pregnancy provide adequate nutrient transfer to the foetus; nevertheless, the gradual escalation throughout gestation correlates with heightened insulin resistance in the mother. Despite variations in dietary cholesterol, plasma cholesterol levels during late pregnancy are around 50% elevated compared to pre-pregnancy levels, while triglyceride levels increase by two to thrice. These modifications are essential for improving substrate availability for the foetus. Lipid abnormalities correlate with adverse pregnancy outcomes and are likely linked to enduring vascular damage following severe obstetrical complications, hence increasing the mother's susceptibility to future cardiovascular disease. [29]

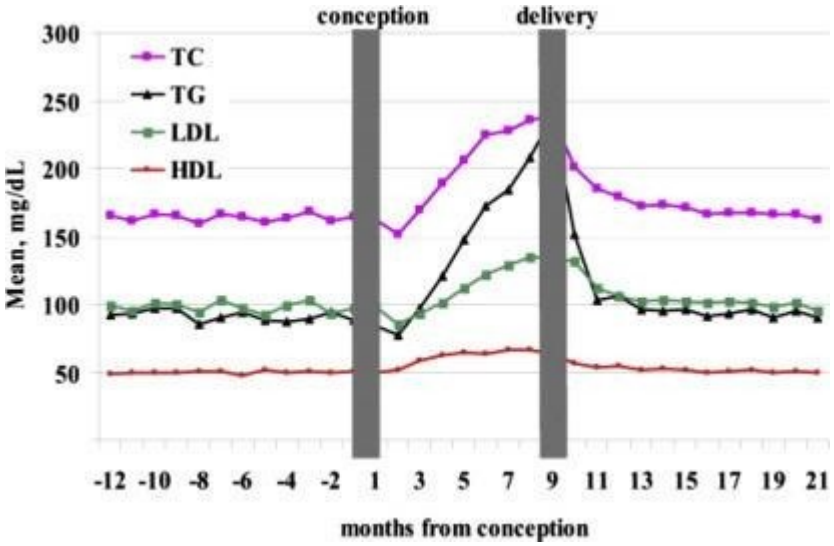


Figure 1.

In clinical care, it is essential to recognise that maxximum pllasma choolesterol levels often notsurpass 250 mg/dL throughout normaal preegnancy, despite significant rise in triglyceridee levelss occurs as pregnanccy advances. In cross-sectional studies of complicated pregnancies, cholesterol levels often surpass 300 mg/dL. Elevated levels are linked to several detrimental maternal pregnancy problems. In typical pregnaant women, athherogenic indeex, LDL/HDL, remainss mostly stable durring prregnancy. indicates when totaal lippoprotein leevels rise, the distribution of cholesterol - containing lippoprotein frractions remains stable. Physiological hyperlipidemia/hypertriglyceridemia is characterised by a concurrent elevation in HDL-C in healthy women during pregnancy, distinguishing it from pathological dyslipidaemias. During gestation, both LDL and HDL levels, along with triglycerides, are elevated. elevation in largee HDL during latee gestatioon reduction ina mediuum. [30,31]

Table 1.

Elevated concentrations of lipids, lipoproteins, and apolipoproteins

Triglycerides	2.7-fold increase
Total Cholesterol	43% increase
LDL Cholesterol	36% increase
HDL Cholesterol	25% increase
Lipoprotein (a)	190%*
Apolipoprotein B	56% increase
Apolipoprotein AI	32% increase

All increases originate the third trimester, with the exception of the elevation in HDL cholesterol.

During gestation, levels of small dense LDL rise, especially in individuals with significantly elevated triglyceride concentrations. Alongside the rise in triglycerides, LDL-C, and HDL-C, there is also an increase in apolipoprotein B and A-I levels. In the majority of cross-sectional studies, Lp(a) levels remain stable during pregnancy. Numerous studies assessing Lp(a) levels during pregnancy observed an elevation in Lp(a) concentrations as term neared. The inability detect variance in Lp_(a) levelss is mostly the significant heterogeneity in Lp(a) levels between people. Values fluctuate between 1 mg/dL and exceeding 200 mg/dL, mostly influenced by genetic determinants. [32,33]

Many physiological changes occur during pregnancy trees, geological mainly shows protein fairy tale activity. The synthesis of lipids and the storage of fat are expected to increase the demands of fetal energy in the late pregnancy. Increasing lipid production from 10 weeks of pregnancy to 30 weeks of pregnancy contributes to maternal humps in the early stages of pregnancy and increases sensitivity to insulin. Improved sensitivity to insulin contributes to the production of fatty acids in fat cells and increases the expression of lipoprotein lipase, increasing the absorption of fatty acids from the rich triglyglyceride of lipoprotein in blood flow. Increasing synthesis of progesterone, cortisol, leptin and prolactin helps to increase fat production. There is a significant increase in fat cells to promote the increase in fat accumulation.

In the third trimester of pregnancy, the body enters a predominantly catabolic metabolic state, largely due to a physiological decline in insulin sensitivity. This insulin resistance enhances the mobilization of triglycerides stored in fat cells through increased lipolysis. Rising levels of human placental lactogen (HPL) during this stage further stimulate the breakdown of fat within adipose tissue. At the same time, the decreased insulin responsiveness leads to a reduction in lipoprotein lipase activity in adipocytes, which limits the absorption of fatty acids from triglyceride-rich particles in the blood. Collectively, these adaptations result in a lowered capacity for fat storage and lipid synthesis in fat cells.[34]

During pregnancy, elevated triglyceride levels (hypertriglyceridemia) arise due to an increase in the production and a reduction in the clearance of lipoproteins rich in triglycerides. This imbalance is primarily driven by enhanced hepatic synthesis of these lipoproteins, while their breakdown and removal from circulation become less efficient. the liver results from heightened lipolysis of triglycerides in adipocytes, leading to an increase in free fatty acids delivered to the liver. The liver subsequently synthesises these free fattty aciids intoo VLDL and secretes them. Elevated oestrogen levels during the third trimester augment hepatic

lipogenesis and the synthesis of very low-density lipoprotein (VLDL). Insulin resistance may promote enhanced fattty aciid synthesis livers, the suppression glucosee productioon can bee resisttant too insuulin, whilee lipogenesiss remains unimpeded. Elevated insulin levels have been shown, particularly in animal studies, to stimulate fatty acid synthesis in the liver. Simultaneously, the impaired clearance of triglyceride-rich lipoproteins is linked to decreased activity of both lipoprotein lipase and hepatic lipase. The decline in hepatic lipase is attributed to increased estrogen concentrations, while the suppression of lipoprotein lipase appears to result from a combination of factors, including insulin resistance and elevated estrogen. This, coupled with reduced hepatic lipase activity, contributes to impaired triglyceride clearance from LDL and HDL particles. [35]

As pregnancy reaches full term, lipoprotein lipase activity increases within the mammary glands to enhance fatty acid uptake, supporting triglyceride production for breastfeeding. Rising plasma cholesterol levels during this period are likely driven by increased hepatic cholesterol synthesis. Moreover, elevated levels of proprotein convertase subtilisin/kexin type 9 (PCSK9) suggest an alternative regulatory mechanism, where PCSK9 potentially decreases hepatic LDL receptor expression, thereby contributing to increased LDL cholesterol concentrations. [36]

Estrogen increase	Inhibits Hepatic Lipase
	Stimulates VLDL production
Human Placental Lactogen increase	Stimulates lipogenesis in the liver Induces insulin resistance
	Increases lipolysis
Insulin Resistance	Decreases LPL activity
	Increases lipolysis
	Increase CETP Stimulates lipogenesis in the liver

IMPLICATIONSS OF THE FETUS AND MOTHER

A thorough research and metaa-analysis Ryckman KK et al has elucidated knowledge and ramifications dyslipidaemia during pregnancy on maternal and foetal outcomes. Maternal dyslipidaemia, defined by elevated triiglyceride decreased HDL-C levelss, is linked to seeveral negative periinatal outcoomes. Intervention studies aimed at reducing lipid levels, which also show a simultaneous decrease in negative perinatal outcomes, are crucial to establish that lipid abnormalities are causative. The assessment of pregnant women is hindered by legal protections, little funding, and limited research, mostly due to medical-legal considerations and the fundamental principle of non-maleficence.

Dyslipidaemia, albeit asymptomatic, is a critical component of metabolic syndrome (MetS). The presence of MetS significantly affects maternal vascular health, leading to several additional health issues for both the mother foetus.

Gestational Diabetes

Pregnancy naturally leads to a state of insulin resistance, which is considered a key factor in the development of gestational diabetes mellitus (GDM). This condition poses several risks to the fetus, including brachial plexus injury, low blood sugar levels, breathing difficulties, elevated bilirubin levels, and cardiac muscle abnormalities. For the mother, GDM increases the likelihood of developing pre-eclampsia and significantly raises the risk of progressing to type 2 diabetes after childbirth.

A comprehensive analysis combining data from thirteen cohort studies higher triglyceride levels during early pregnancy are associated with a greater likelihood of developing gestational diabetes mellitus (GDM). [37,38]

Elevated triglyceride concentrations during pregnancy have been consistently associated with an increased risk of developing gestational diabetes mellitus (GDM). In addition, maternal HDL-C levels below 51 mg/dL have been identified as a significant risk factor for GDM. Evidence from a meta-analysis further supports these associations, demonstrating that GDM is correlated with both higher maternal triglyceride levels and lower HDL-C concentrations in early to mid-gestation. In contrast, , characterized by elevated levels of triglycerides, total cholesterol, and LDL-C, along with reduced HDL-C levels. These lipid abnormalities underscore the importance of long-term lipid monitoring in this population. Furthermore, independent of progression to type 2 diabetes, women with a prior diagnosis of GDM are at an increased risk of developing cardiovascular disease later in life. [39,40]

Specific high-risk populations may exhibit lipid level irregularities that elevate their risk prior to pregnancy. The PPCOS II study, conducted by the Reproductive Medicine Network,

linked to improved rates of healthy live births but was associated with a decreased incidence of preeclampsia. [41]

Pre-Eclampsia

Pre-eclampsias rapidlyy progressing affecting. This disorder poses significant risks to the fetus, including impaired growth and serious complications related to both spontaneous and medically induced preterm births. Such complications can lead to outcomes like cerebral palsy, seizures, growth retardation, and even mortality.[42,43]

. While one meta-analysis found no significant relationship between LDL cholesterol and pre-eclampsia, another reported a positive correlation. Additionally, a longitudinal study by Enquobahrie et al. demonstrated.HDL-C levels were observed to be 7.0% lower in women with pre-eclampsia compared to the control group. A 3.6-fold increase in the frequency of pre-eclampsia was documented among women with total cholesterol levels over 205 mg/dL, in comparison to those with levels below 172 mg/dL, even after controlling for confounding variables. [44,45]

LIPID SCREENING

The National Lipid Association (NLA) recommends regular lipid screening, particularly for women without documented pre-pregnancy lipid levels. However, screening among women of reproductive age is often insufficient, partly due to disparities in healthcare access.. [46]

Recent guidelines suggest lipid testing at the initial prenatal visit and, if results are within normal ranges, again at the start of the third trimester. Those considered at higher risk should undergo cholesterol evaluations at their first visit, again at the beginning of the second trimester, and monthly during the third trimester. Triglyceride levels exceeding 250 mg/dL warrant regular lipid panel monitoring. While the timing and necessity of advanced lipid tests—such as nuclear magnetic resonance (NMR), ion mobility analyses for particles, apolipoproteins lipoprotein(a)—remain uncertain, initial measurement of lipoprotein(a) is advisable for patients without prior assessments. Postpartum lipid testing is also recommended when abnormalities are detected during pregnancy.[47]

Aim & Objectives of

the study Aim:

To compare the serum lipid and serum apoprotein(a) parameters (triglycerides, total cholesterol, LDL, HDL) of normal pregnant women and with the pre-eclamptic pregnant women.

OBJECTIVES

- To measure serum lipid profile in normotensive pregnant women and pre-eclamptic women.
- To assess the risk of developing hypertension with deranged lipid profile

Materials and Methods-

Source of data: The research will be carried out on pregnant women with normotensive, late-onset preeclampsia, and severe preeclampsia at the Department of Obstetrics and Gynaecology, RL Jalappa Hospital, Kolar, during the study duration.

Study design: CASE CONTROL STUDY

Study period: JULY 2023 – DECEMBER 2024(1 1/2 years)

Inclusion Criteria

- All pregnant women fulfilling the specified inclusion criteria will be recruited for this study.
- Age 19 - 45 years
- Singleton pregnancy
- Gestational age between 20 and 40 weeks, verified by ultrasonography performed during the first trimester.
- Women in pregnancy, between 20 and 40 weeks of gestation, exhibiting elevated blood pressure accompanied by protein in the urine.
- Pregnant women without comorbidities

Exclusion Criteria

- Multiple gestation and foetal demise
- Substantial foetal abnormalities (and postnatal surgical intervention).
- Chromosomal anomalies, hereditary conditions, and gross placental abnormalities

- Presence of maternal systemic problems - Gestational Diabetes Mellitus, Dyslipidaemia, Thyroid dysfunction, Autoimmune disorders, Cardiovascular diseases
- Administration of certain medicines include corticosteroids, nonsteroidal anti-inflammatory drugs (NSAIDs), antilipidemics, and immunosuppressants

Statistical analysis:

Data will be entered into Microsoft Excel and analyzed using SPSS software version 22. Categorical variables will be summarized as frequencies and percentages, with the Chi-square test applied to assess statistical significance. Continuous variables will be expressed as means with standard deviations, and the independent t-test will be used to compare means between two groups. A p-value of less than 0.05 will be considered statistically significant.

Sample Size:

The sample size calculation was based on the difference in mean LDL cholesterol levels between normal pregnancies and those complicated by preeclampsia, as reported by Aghade SM et al., with values of 123.2 ± 16.4 mg/dL and 133.8 ± 13.3 mg/dL, respectively. Utilizing the specified methodology and MedCalc software for sample size determination, a minimum of 32 participants per group was required to achieve 80% statistical power at a 95% confidence level. Accounting for an anticipated 10% attrition rate, the sample size was adjusted to approximately 35 participants per group. Therefore, the total sample size for the study was set at 96 individuals.

Sample Size Estimation Formula:

$$N = \frac{2\,SD^2(Z_{\alpha/2} + Z_{\beta})^2}{d^2}$$

- Where $Z_{\alpha/2}$ is the critical value of the Normal distribution at $\alpha/2$ (e.g. for a confidence level of 95%, $\boldsymbol{\alpha}$ is 0.05 and the critical value is 1.96).
- Z_{β} is the critical value of the Normal distribution at β (e.g. for a power of 80%, ß is 0.2 and the critical value is 0.84),
- SD is the standard deviation from previous study population variance, and
- d is the largest difference between two mean

Method of collection of data:

All participants are required to give their written informed permission before engaging in the study. This study included participants diagnosed with early and late-onset severe preeclampsia from July 2023 to December 2024 at R L Jalappa Hospital Tamaka Kolar, affiliated with Srii Devaraj URS Medical College under Srii Devaraj Urs Academy of Higher Educatiion annd Reseaarch, all of whom provided written informed consent before participation. We will do a comprehensive lipid profile analysis, in conjunction with standard laboratory tests, on blood samples obtained from these people at 20-40. The control group for the study will comprise normotensive pregnant women of comparable age and gestational age who received care at our hospital throughout the same timeframe. Proteinuria is identified when protein levels above 300mg iin a 24_hourr uriine colleection,

oor, in the absence oof such a collection, by a dipstick test indicating +2 protein annd/orr prottein/creatiinine ratiio off 0.3 iin a random uriine sample. Severe preeclampsia is diagnosed when a pregnant woman meets the criteria for preeclampsia and exhibits systoliic bloodd pressurre off 160,mmHg orr hiigher and/(or) diastolic blood pressure of 110 mmHg or higher on at least two separate measurements taken at least four hours apart, or when there is evident end-organ dysfunction. Venous blood samples will be obtained from participants following a 12-hour fasting interval during gestation. The Abbott Architect C8000 system (Abbott Diagnostics, USA) will utilise the original reagent to assess totaal choleesterol (TC), triiglycerides, and HDL choolesterol, with HDL cholesterol meassured by a direct eenzymatic technique that obviates precipitation. LDL cholesterol will be computed utilising the Friedewald equation (TC = LDL cholesterol + HDL cholesterol + Triglycerides).

Data Analysiis

- Datta was initiially enterred iinto Micrrosoft Exceel annd lateer exported for statisstical anallysis usiing SPSS versiion 22,, a robust statistiical software widely employed in medical research for both descriptive and inferential statistics.
- Categorical data (e.g., age group, BMI classification, delivery mode, parity, and birthweight classification) were summarised using frequencies and percentages.
- Continuous variables (e.g., total cholesterol, triglycerides, HDL, LDL, and APGAR scores) were described using their mean and standard deviation.
- The chi-square test was utilised to examine categorical data to identify significant correlations between groups (e.g., variations in BMI categories between normotensive and preeclamptic populations).
- An iindependent t_test was employed too compare thee means off contiinuous variiables across tthe 2 research groups (e.g., cholesterol levels in normotensive and preeclamptic women).
- A p-value below (0.05) waas consiidered stattistically signiificant, suggestiing

thatt thee obserrved differencees weree unliikely too havee arisen bby chaance.

Thee study employed traditional and appropriate statistical methods within a case-control paradigm. The utilisation of SPSS software enabled reliable analysis, and the integration of both parametric (t-test) and non-parametric (Chi-square) methods accommodated the traits of continuous and categorical data. The sample size calculation based on prior data improves the statistical power and validity of the findings. The procedure is comprehensive, statistically sound, and replicable

Dr Lakshmi Priya - Results

Table 1: Comparison of Age Group between the groups

Group Age Group	Group A (Normal)	Group B (Preeclampsia)	Total
18 - 26	38	37	75
27 - 35	10	11	21
Total	48	48	96
	p-value = 0.80		

Table 1 shows that the majority of participants in both groups are in the 18-26 age range, with 38 individuals from Group A (Normal) and 37 from Group B (Preeclampsia), totaling 75 participants in this category. The 27-35 age group includes 10 individuals from Group A and 11 from Group B, making up 21 participants. The total sample consists of 96 participants, with an equal distribution of 48 individuals in each group.

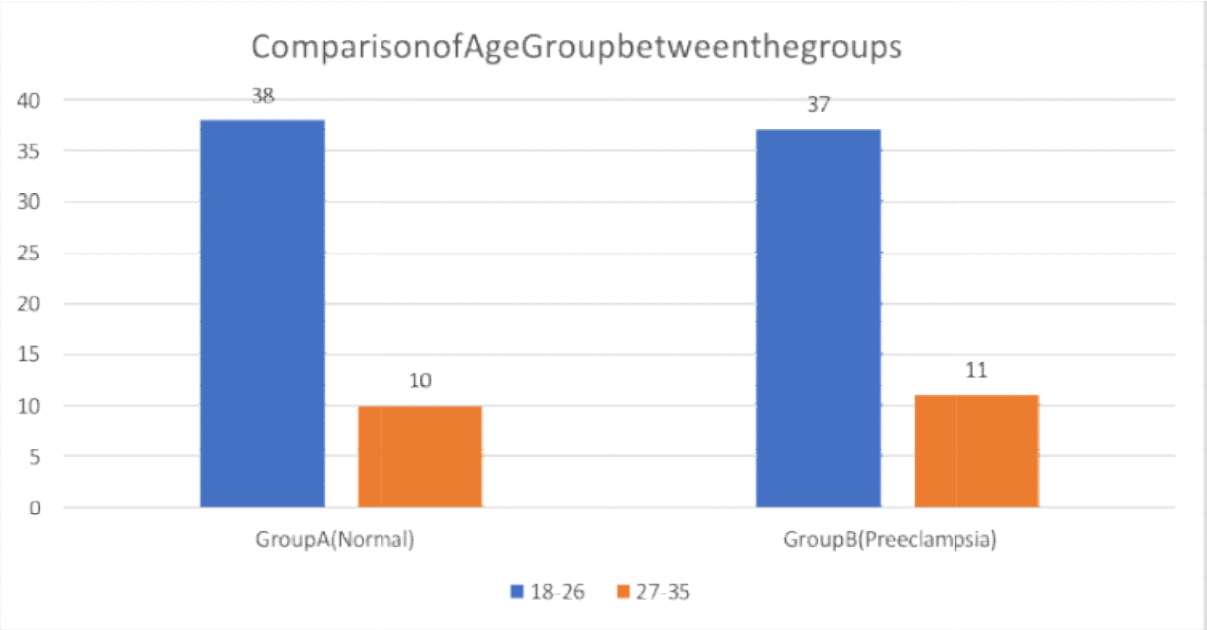


Table 2: Mean age comparison between the groups

Age in years		
	Group A (Normal)	**Group B (Preeclampsia)**
Mean	24.86	24.10
Std. Deviation	3.81	4.56
	p-value = 0.98	

Table 2 compares the mean age between Group A (Normal) and Group B (Preeclampsia). The mean age for Group A is 24.86 yearss with a standdard deviiation in of 3.81, while Group B has a mean age of 24.10 years with a standard deviation of 4.56..

Table 3: Comparison of anthropometry

	Weight		Height in metres		BMI	
Group A (Normal)		**Group B (Preeclampsia)**	**Group A (Normal)**	**Group B (Preeclampsia)**	**Group A (Normal)**	**Group B (Preeclampsia)**
Mean	64.23	68.33	1.58	1.60	24.18	25.53
Std. Deviation	6.42	11.85	0.02	0.06	2.33	2.80
p-value		0.26		0.28		0.39

Table 3 compares the anthropometric measurements (weight, height, and BMI). weight for Group A is 64.23 kg, while for Group B, it is 68.33 kg. In terms of height, Group A has a mean of 1.58 meters, and Group B has a mean of 1.60 meters. The mean BMI for Group A is 24.18, while for Group B, it is 25.53. The standard deviations for weight, height, and BMI are higher in Group B compared to Group
A. this indicate no significant differences between the two groups in these anthropometric measures.

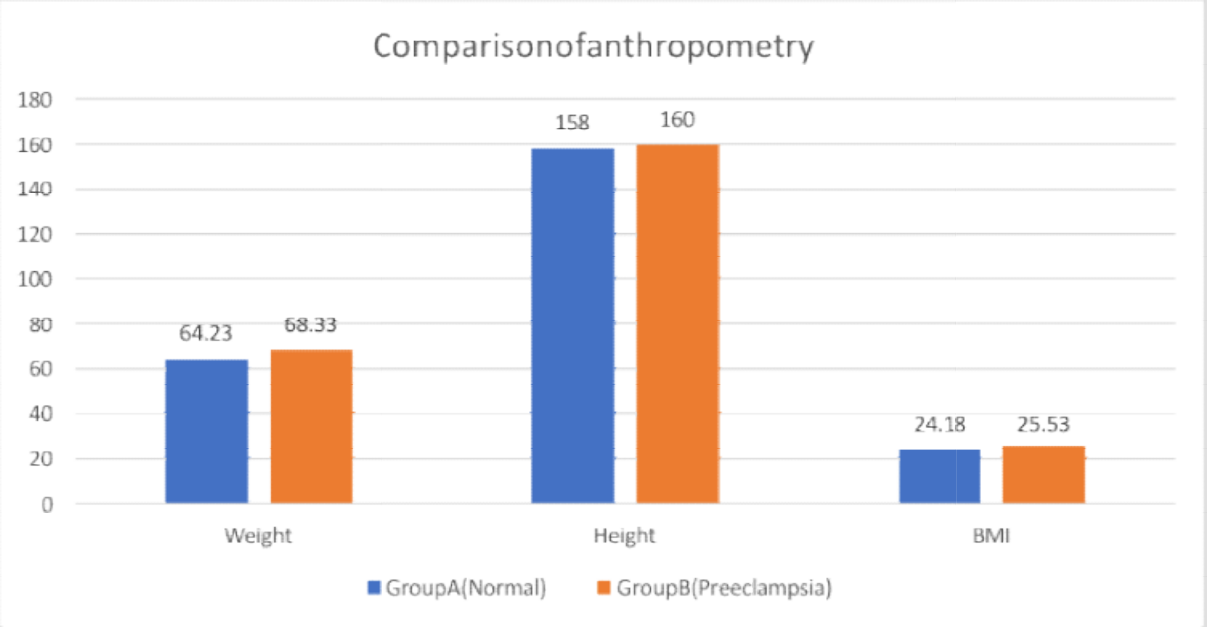


Table 4: Comparison of BMI Classification

Group BMI Classification	Group A (Normal)	Group B (Preeclampsia)	Total
Normal wt (18.5 – 23.00 kg/m²)	25	5	30
Overwt (23-24.9 kg/m²)	13	18	31
Obesity Class I (25-29.9 kg/m²)	10	25	35
Total	48	48	96
	p-value < 0.001		

Table 4 compares the BMI classification between A (Normal) and B (Preeclampsia). In A, 25 participants are classified as having normal weight, 13 as overweight, and 10 as obese class I. In Group B, 5 participants are classified as having normal weight, 18 as overweight, and 25 as obese class I. The total sample includes 96 participants, with 48 in each group.

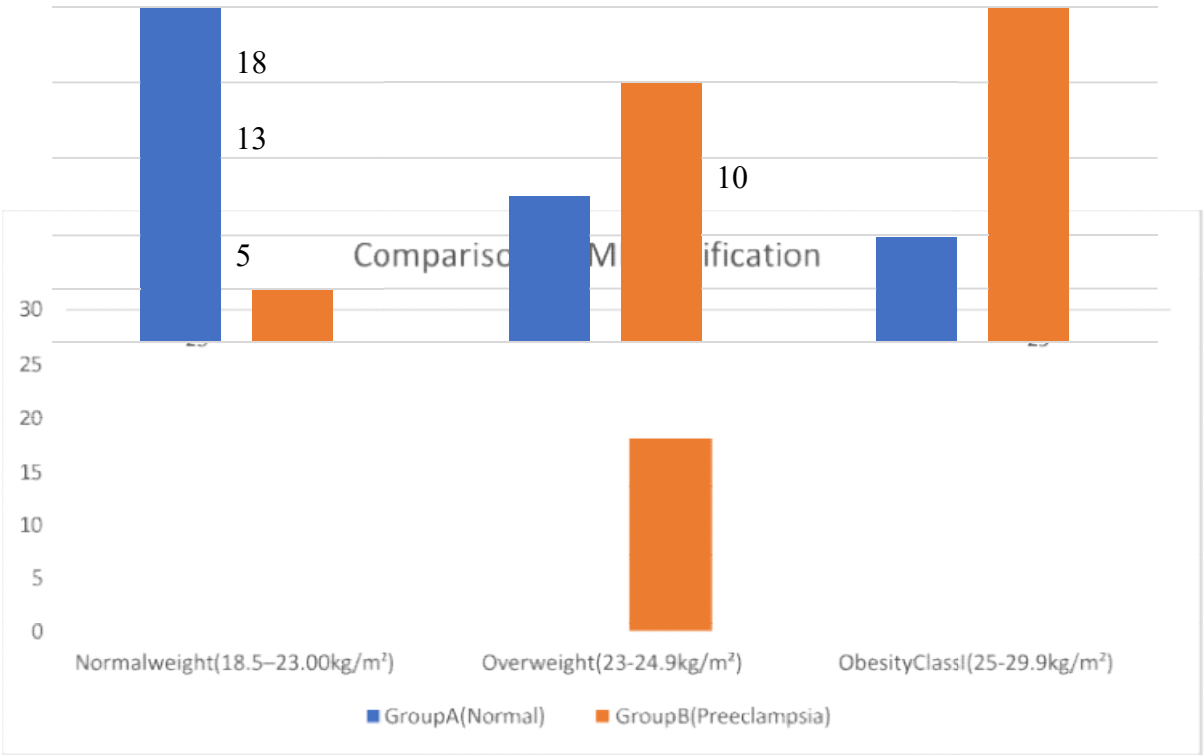


Table 5: Comparison of parity status

	Group		
Para	**Group A (Normal)**	**Group B (Preeclampsia)**	**Total**
Primigravida	29	33	62
Multigravida	19	15	34
Total	48	48	96
	p-value = 0.39		

Table 5 compares the parity status between A (Normal) and B (Preeclampsia). In A, 29 participants are primigravida (first pregnancy), while 19 are multigravida (having had more than one pregnancy). In Group B, 33 participants are primigravida, and 15 are multigravida. The total sample consists of 96 participants, with 48 in each group.

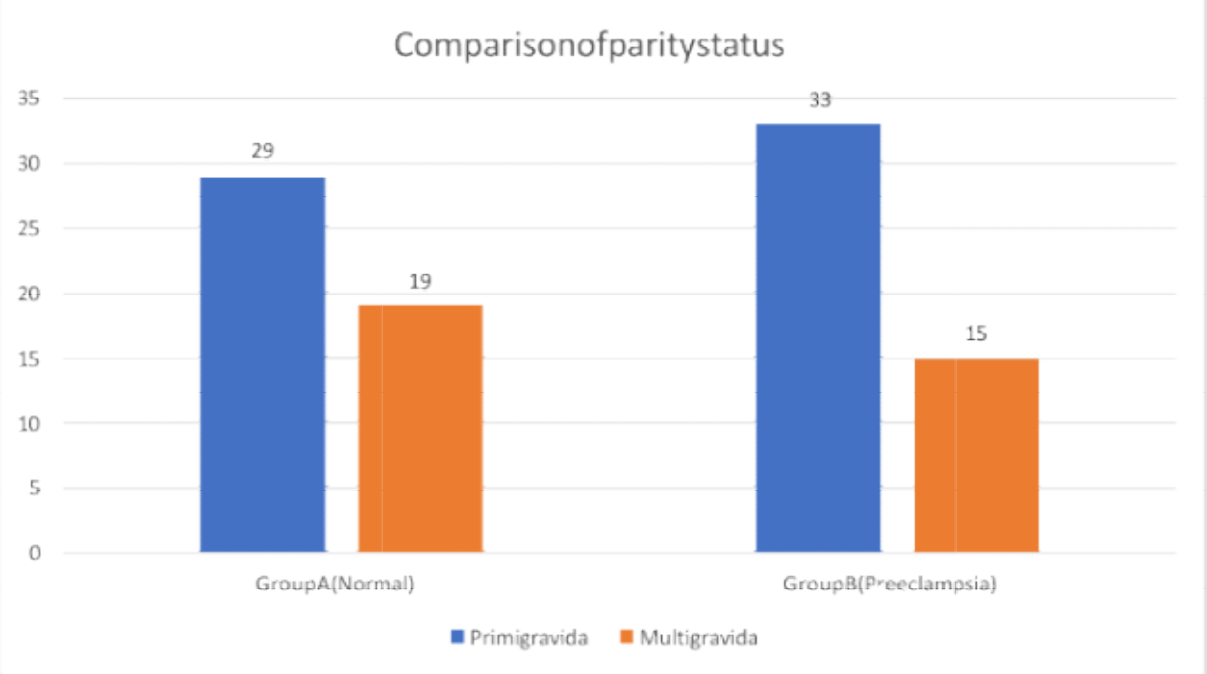


Table 6: Comparison of mode of delivery

	Group		
Mode of Delivery	**Group A (Normal)**	**Group B (Preeclampsia)**	**Total**
LSCS	21	44	65
NVD	27	4	31
Total	48	48	96
	p-value < 0.001		

Table 6 compares the mode of delivery between Normal and Preeclampsia. In A, 21 participants delivered via LSCS (caesarean section), and 27 had a normal vaginal delivery (NVD). In Group B, 44 participants underwent LSCS, while only 4 had a normal vaginal delivery. The total sample includes 96 participants, with 48 in each group.

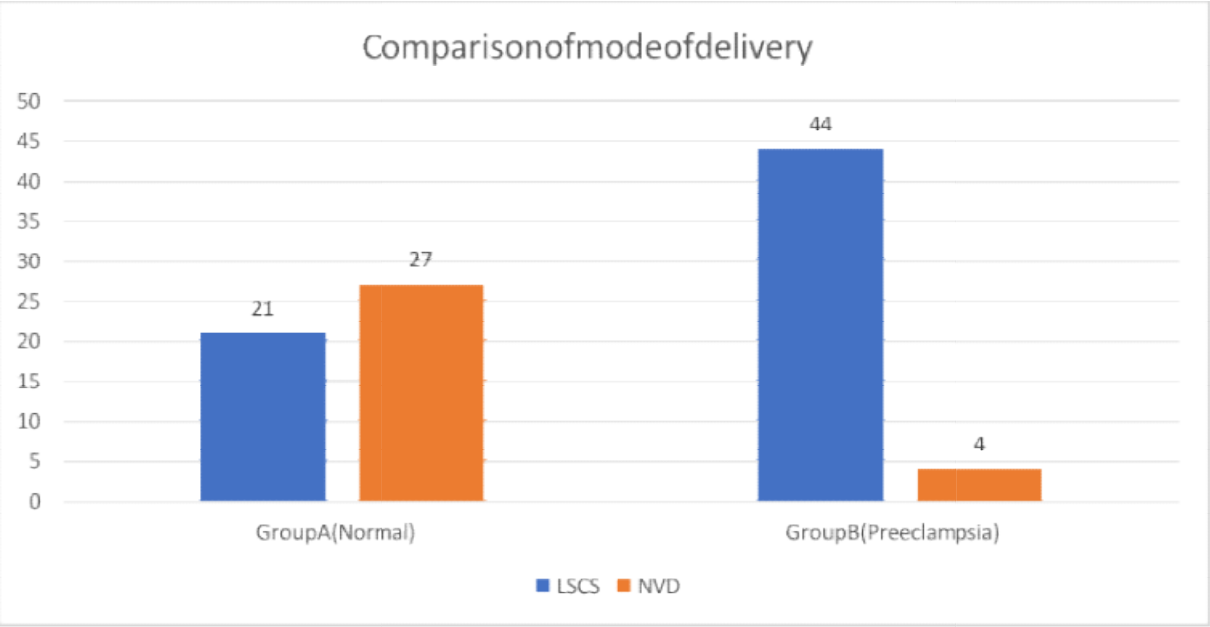


Table 7: Comparison of Gestational week at delivery

Gestational week at Delivery		
	Group A (Normal)	**Group B (Preeclampsia)**
Mean	37.75	33.35
Std. Deviation	1.86	3.56
	p-value < 0.001	

Table 6 compares the mode of delivery normal and preeclampsia. In A, 21 participants had a caesarean section (LSCS), and 27 had a normal vaginal delivery (NVD). In Group B, 44 participants underwent LSCS, while only 4 had NVD. The total sample includes 96 participants, with 48 in each group. the mode of delivery, with Group B having a substantially higheer rate of caesarean sections compared to Group A.

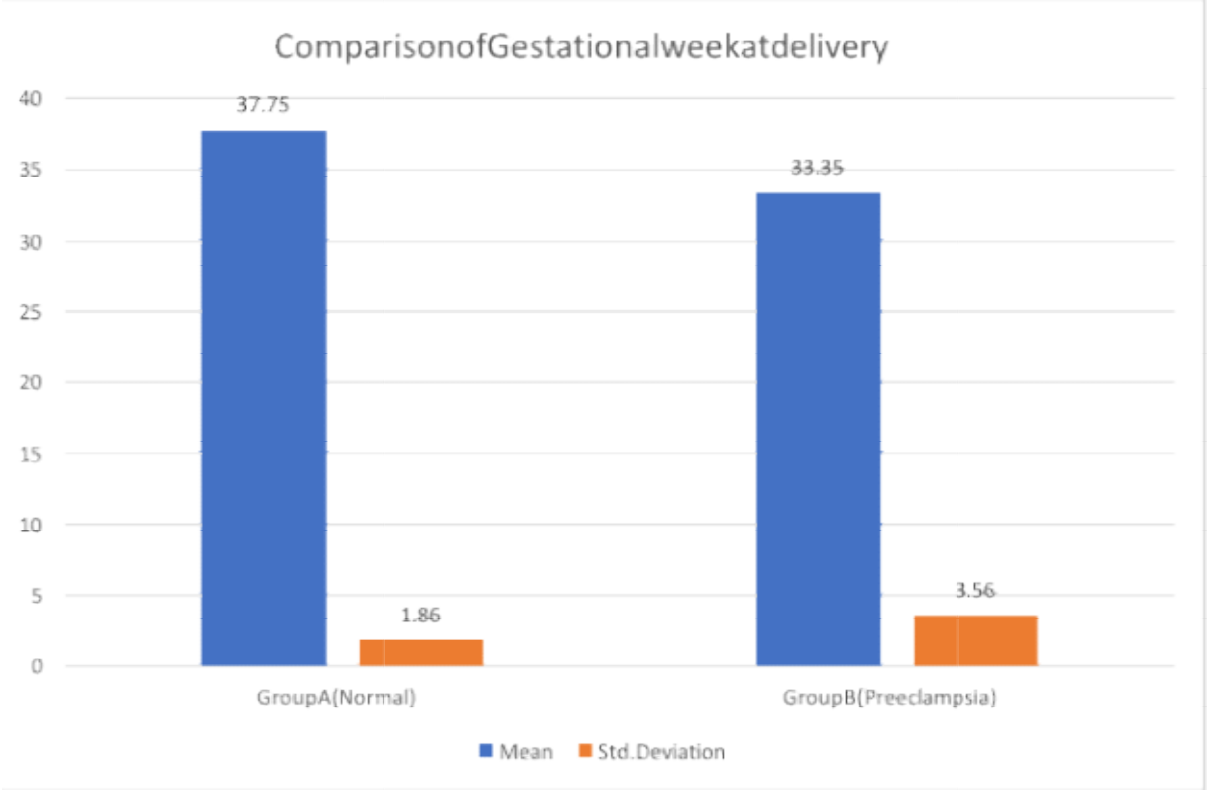


Table 8: Comparison of birthweight classification

Group Birthweight Classification	Group A (Normal)	Group B (Preeclampsia)	Total
Low Birth Wt (< 2.5 Kgs)	7	46	53
Nor Wt (2.5 – 3.9 Kgs)	40	2	42
Macrosomiia (> 4.0 Kgs)	1	0	1
Total	48	48	96

p-value < 0.001

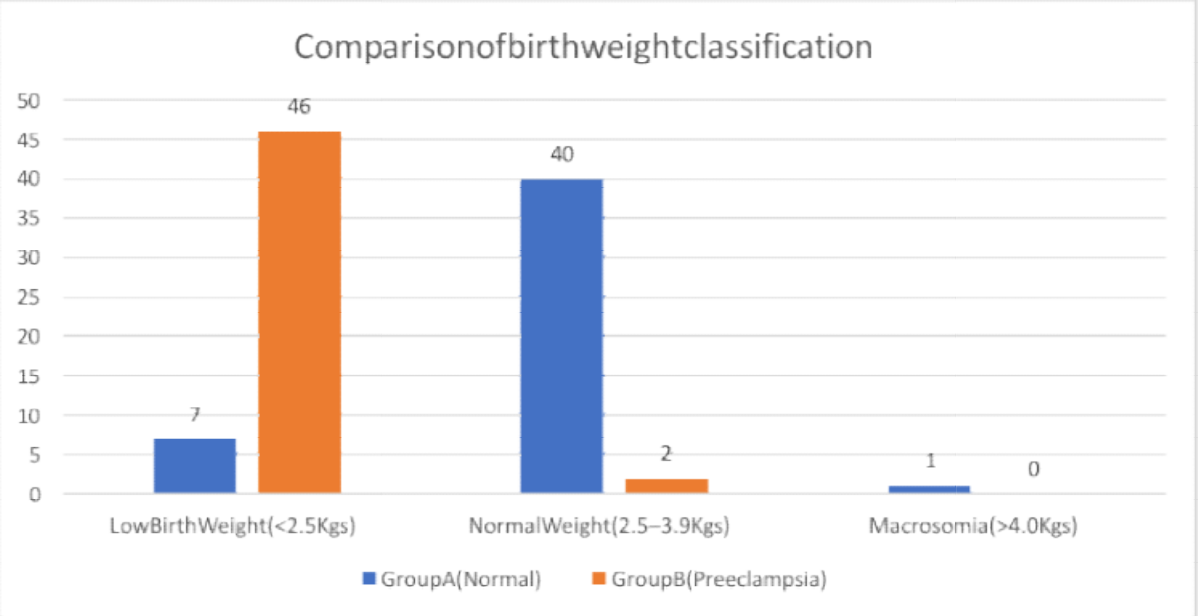


Table 8 compares the birthweight classification between normal and preeclampsia. In A, 7 participants had low birth weight (< 2.5 kg), 40 had normal birth weight (2.5 – 3.9 kg), and 1 had macrosomia (> 4.0 kg). In Group B, 46 participants had low birth weight, and 2 had normal birth weight, with no cases of macrosomia. The total sample includes 96 participants, with 48 in each group.

Table 9: Comparison of APGAR score

APGAR score at 5th minute		
	Group A (Normal)	**Group B (Preeclampsia)**
Mean	8.46	6.69
Std. Deviation	0.97	0.97
	p-value < 0.001	

Table 9 compares the APGAR score at the 5th minute among normal and preeclampsia. The mean APGAR score for Group A is 8.46, with a standard deviation of 0.97, while Group B has a mean score of 6.69, also with a standard deviation of 0.97. APGAR scores, with Group A having a notably higher score compared to Group B.

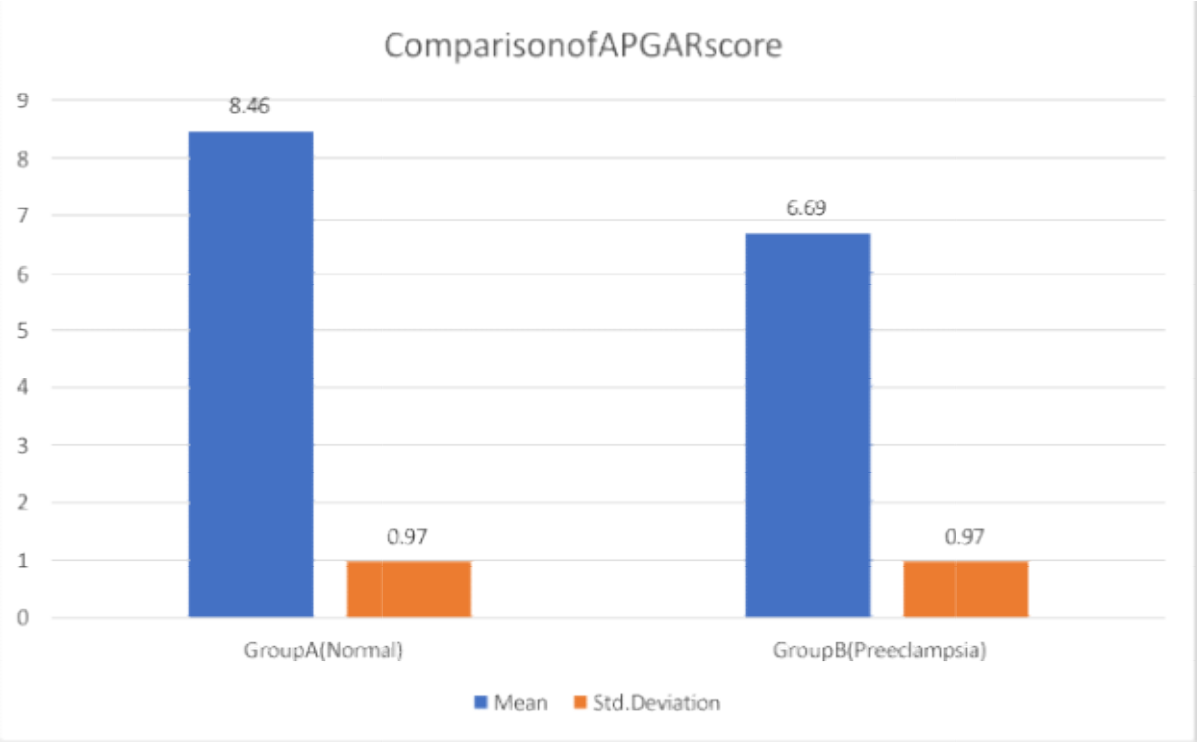


Table 10: Comparison of Total Cholesterol

Total Cholesterol		
	Group A (Normal)	**Group B (Preeclampsia)**
Mean	183.38	239.23
Std. Deviation	26.61	39.33
	p-value < 0.001	

Table 10 compares the total cholesterol levels mormal and preeclampsia. total cholesterol for Group A is 183.38 mg/dL, with a standard deviation of 26.61, while Group B has a higher mean of 239.23 mg/dL and a standard deviation of 39.33. total cholesterol levels between the two groups, with Group B showing significantly higher cholesterol levels.

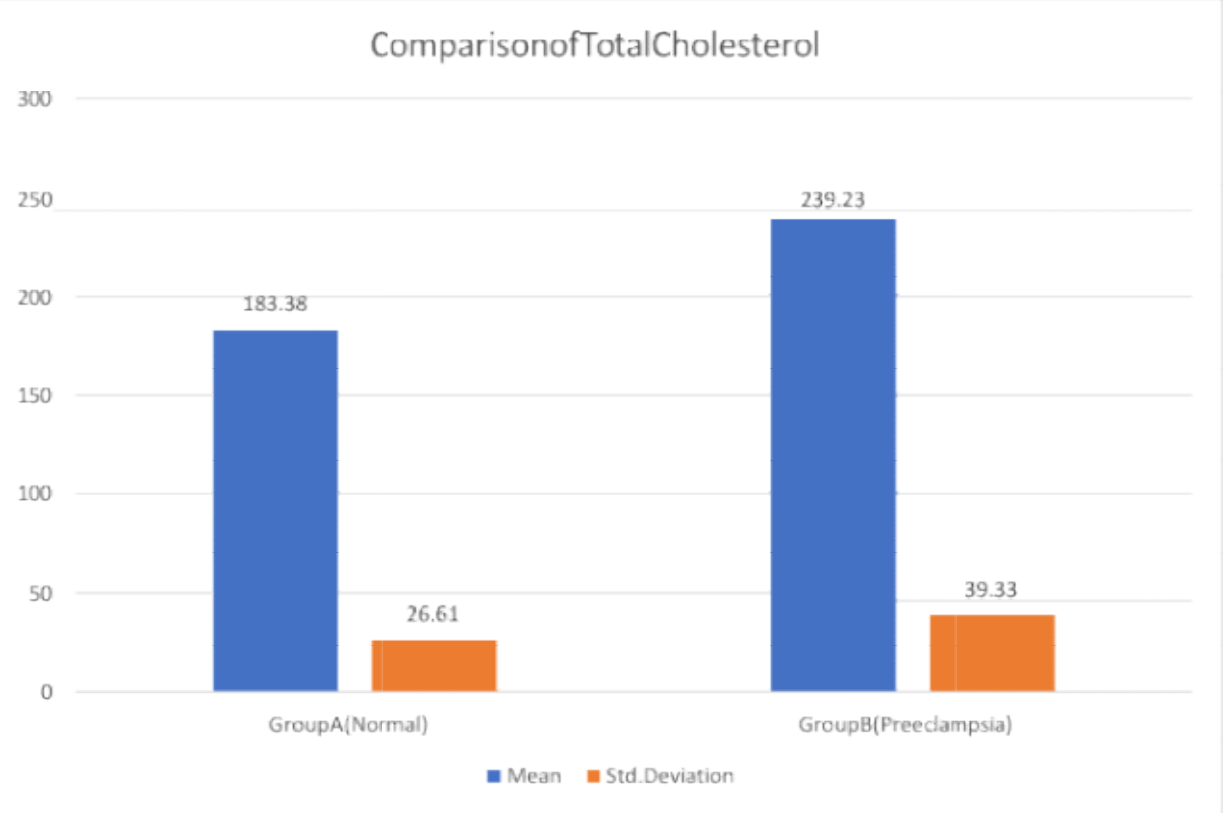


Table 11: Comparison of Total Cholesterol Classification

Group Total Cholesterol Classification	Group A (Normal)	Group B (Preeclampsia)	Total
Acceptable (< 200 mg/dL)	38	8	46
Mild High (200 - 239 mg/dL)	8	12	20
Increase (> 240 mg/dL)	2	28	30
Total	48	48	96

p-value < 0.001

Table 11 compares the total cholesterol classification among normal and preeclampsia. In A, 38 participants have desirable cholesterol levels (less than 200 mg/dL), 8 have slightly high levels, and 2 have increase cholesterol. In Group B, 8 participants have desirable cholesterol levels, 12 have borderline high levels, and 28 have high cholesterol. The total sample includes 96 participants, with 48 in each group. the cholesterol classification between the two groups, with a notably high proportion of participants in B having high cholesterol levels.

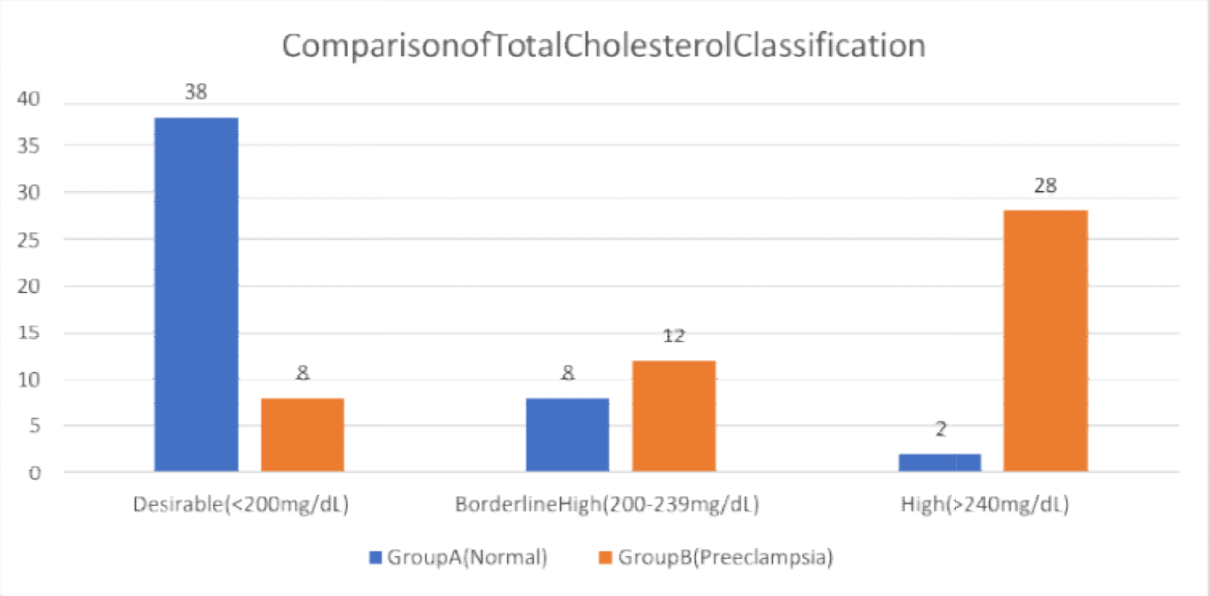


Table 12: Comparison of TGL

TGL		
Group A (Normal)		**Group B (Preeclampsia)**
Mean	147.67	250.69
Std. Deviation	21.13	90.72
	p-value < 0.001	

Table 12 compares TGL level for Group A is 147.67 mg/dL, with a standard deviation of 21.13, while Group B has a significantly higher mean of 250.69 mg/dL and a standard deviation of 90.72 TGL levels between two groups, with Group B showing much higher triglyceride levels compared to Group A.

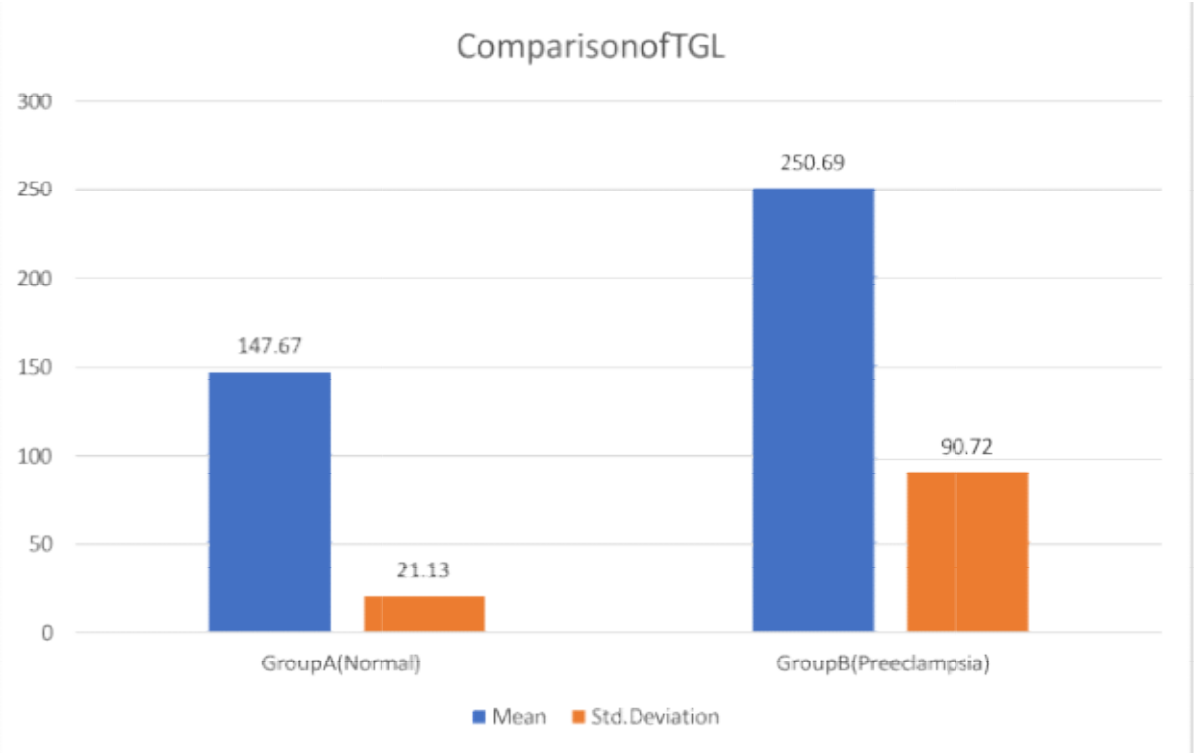


Table 13: Comparison of TGL Classification

Group **TGL Classification**	**Group A (Normal)**	**Group B (Preeclampsia)**	**Total**
ordinary	24	10	34
Slight increase	24	8	32
Increase	0	30	30
Total	48	48	96
p	**-value < 0.001**		

Table 13 compares the triglyceride (TGL) classification Normal , Preeclampsia. In A, 24 participants have normal TGL levels (<150 mg/dL), and 24 have borderline high levels (150 - 199 mg/dL). In Group B, 10 participants have normal levels, 8 have borderline high levels, and 30 have high TGL levels (200 - 499 mg/dL). The total sample consists of 96 participants, with 48 in each group. TGL classification, with a much higher proportion of participants in Group B having high triglyceride levels.

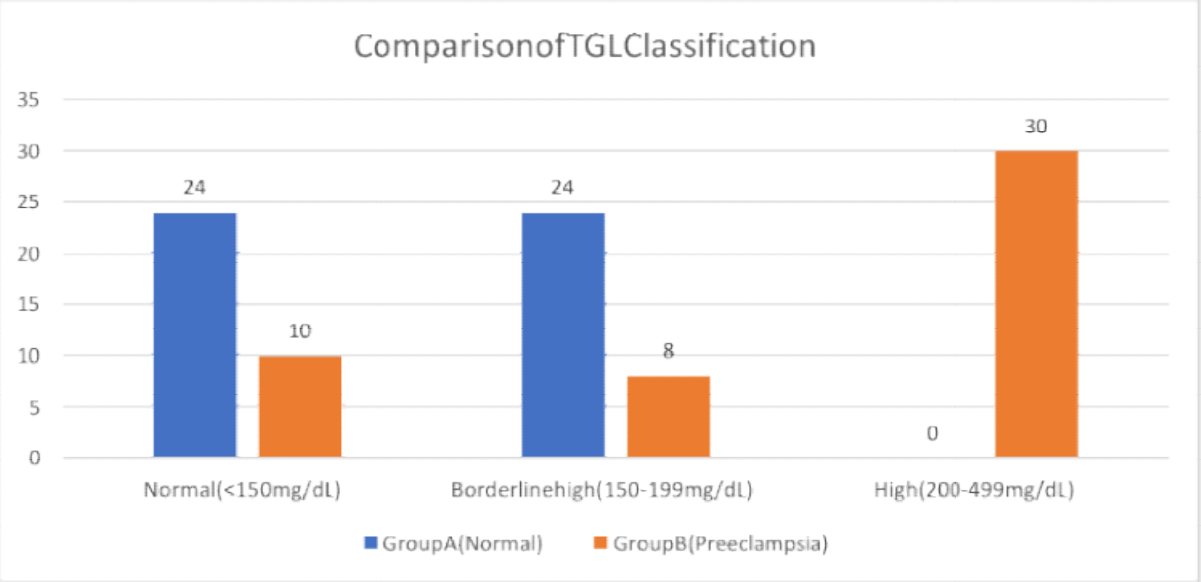


Table 14: Comparison of HDL

HDL		
Group A (Normal)		**Group B (Preeclampsia)**
Mean	55.83	48.46
Std. Deviation	8.51	9.14
	p-value < 0.001	

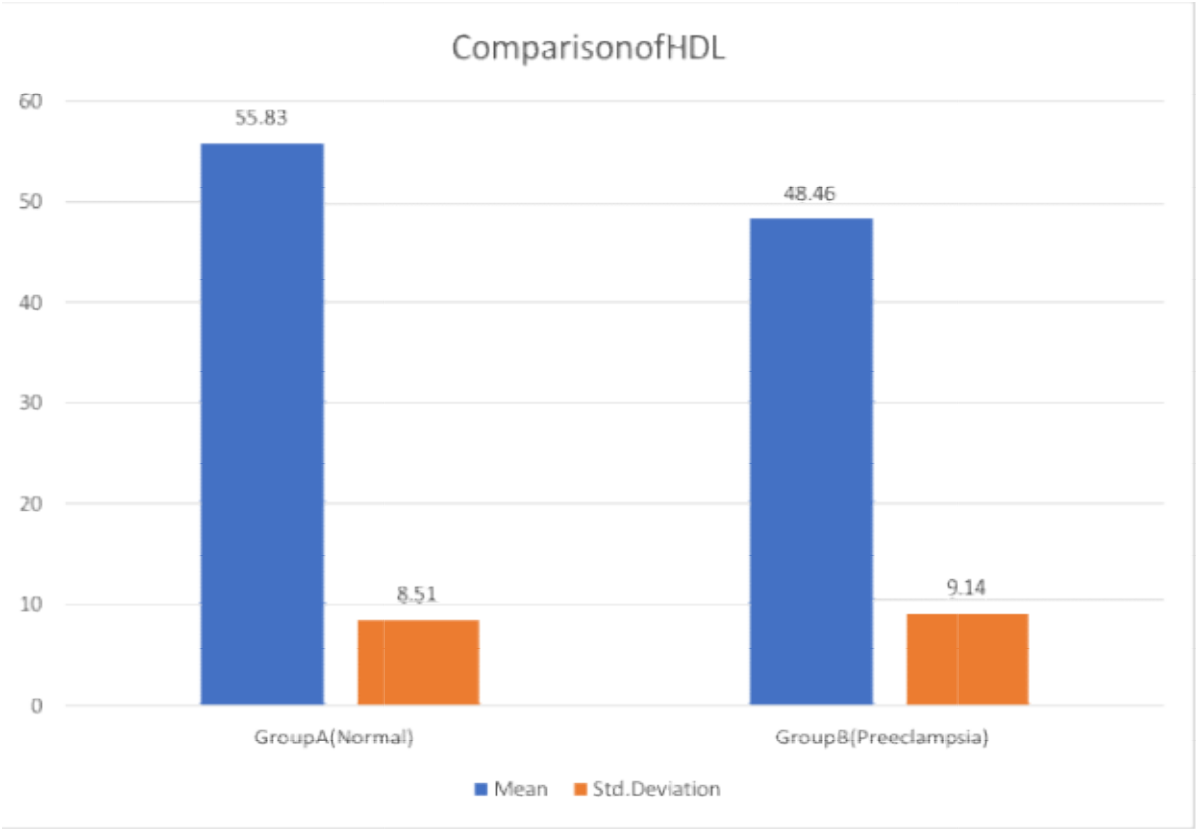


Table 14 compares the HDL (high-density lipoprotein) levels between Group A (Normal) and Group B (Preeclampsia). The mean HDL level for Group A is 55.83 mg/dL, with a standard deviation of 8.51, while Group B has a mean of 48.46 mg/dL and a standard deviation of 9.14. HDL levels between two groups, with Group A showing higher HDL levels compared to Group B.

Table 15: Comparison of HDL Classification

Group HDL Classification	Group A (Normal)	Group B (Preeclampsia)	Total
Normall	31	41	72
increased	17	7	24
Total	48	48	96
	p-value = 0.02		

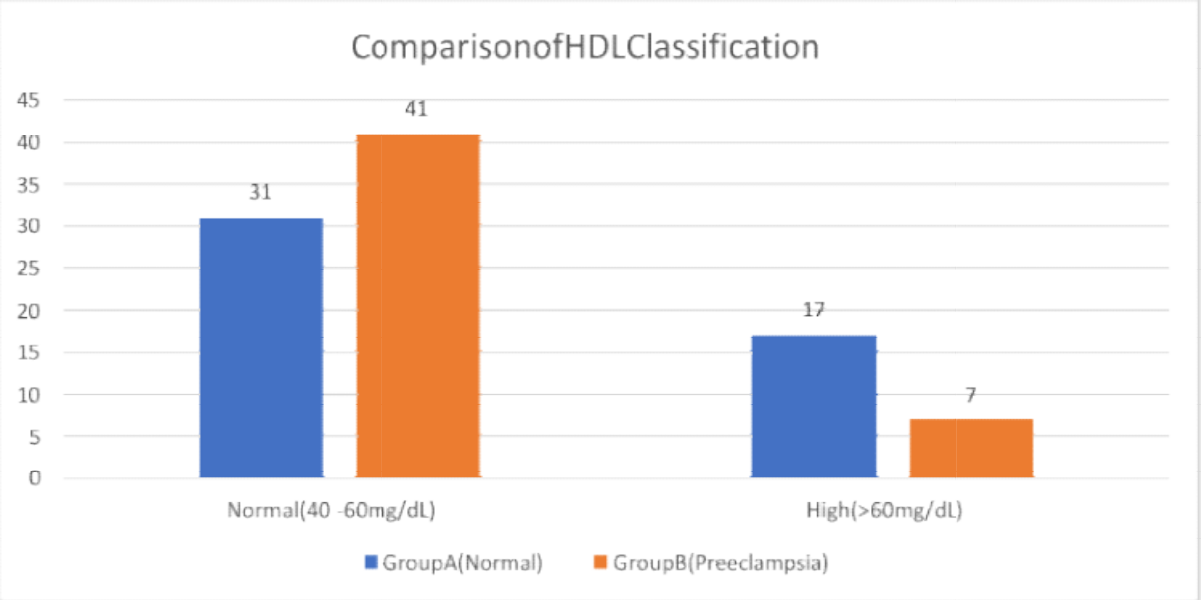


Table 15 compares the HDL (high-density lipoprotein) classification (Normal) and (Preeclampsia). In A, 31 participants fall within the normal HDL range (40 - 60 mg/dL), and 17 have high HDL levels (> 60 mg/dL). In Group B, 41 participants have normal HDL levels, and 7 have high HDL levels. The total sample consists of 96 participants, with 48 in each group. HDL classification, with a higher proportion of participants in Group A having high HDL levels compared to Group B.

Table 16: Comparison of mean LDL

	LDL	
	Group A (Normal)	**Group B (Preeclampsia)**
Mean	94.25	136.38
Std. Deviation	13.58	20.62
	p-value < 0.001	

Table 16 compares the mean LDL (low-density lipoprotein) levels (Normal) B (Preeclampsia). LDL level for Group A is 94.25 mg/dL, with a standarrd deviiatiion of 13.58, while Group B has a higher mean of 136.38 mg/dL and a standarrd deviiationn of 20.62. LDL levels between two groups, with Group B showing significantly higher LDL levels compared to Group A.

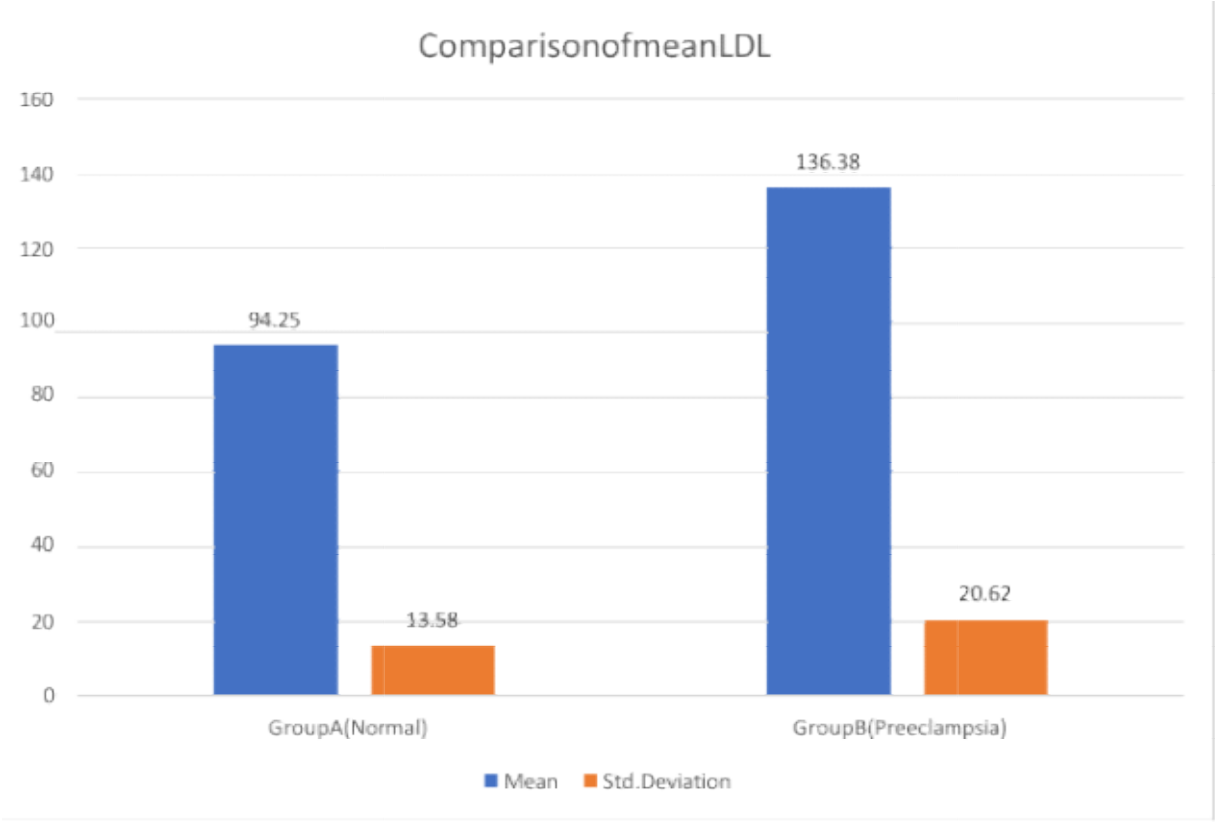
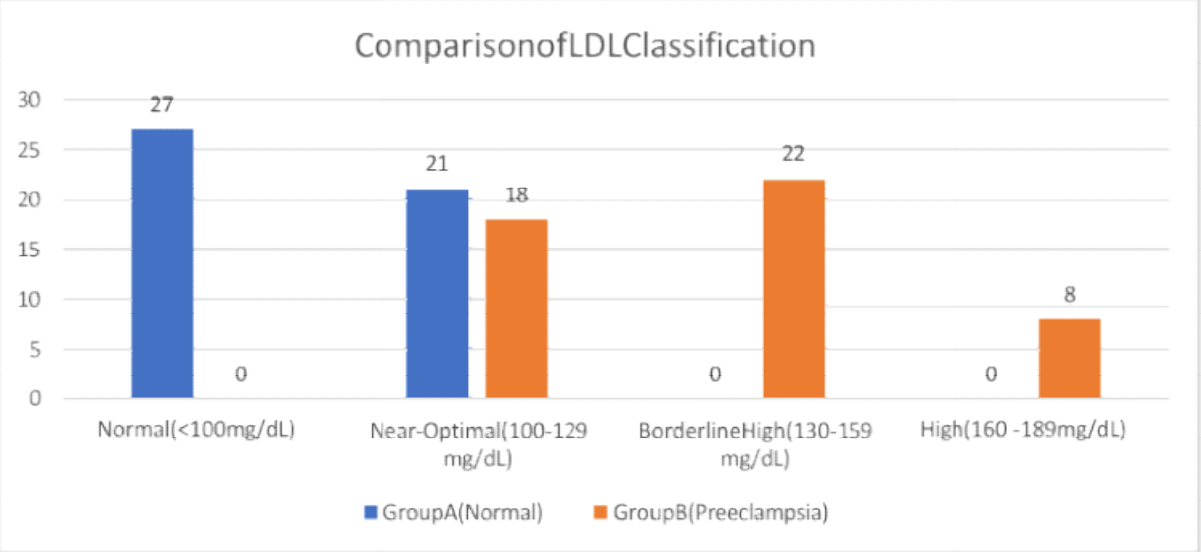


Table 17: Comparison of LDL Classification

Group LDL Classification	Group A (Normal)	Group B (Preeclampsia)	Total
Normal (<100 mg/dL)	27	0	27
Optimal	21	18	39
Slightly High	0	22	22
Increased	0	8	8
Total	48	48	96
p	**-value < 0.001**		

Table 17 compares the LDL (low-density lipoprotein) classification among (Normal) and (Preeclampsia). In A, 27 participants have normal LDL levels (<100 mg/dL), and 21 have near-optimal levels (100 - 129 mg/dL). In Group B, no participants have normal LDL levels, 18 have near-optimal levels, 22 have borderline high LDL levels (130



- 159 mg/dL), and 8 have high LDL levels (160 - 189 mg/dL). The total sample consists of 96 participants, with 48 in each group. This shows changes in LDL classification between two groups, with a higher proportion of participants in Group B having higher LDL levels.

Table 18: Comparison of VLDL

VLDL		
Group A (Normal)		**Group B (Preeclampsia)**
Mean	29.88	61.73
Std. Deviation	5.14	7.17
	p-value < 0.001	

Table 18 compares the mean VLDL (very low-density lipoprotein) levels among Group-A (Normal) Group-B (Preeclampsia). VLDL level for Group A is 29.88 mg/dL, with a standard deviation of 5.14, while Group B has a significantly higher mean of 61.73 mg/dL and a standard deviation of 7.17. VLDL levels between two groups, with Group B showing notably higher VLDL levels compared to Group A.

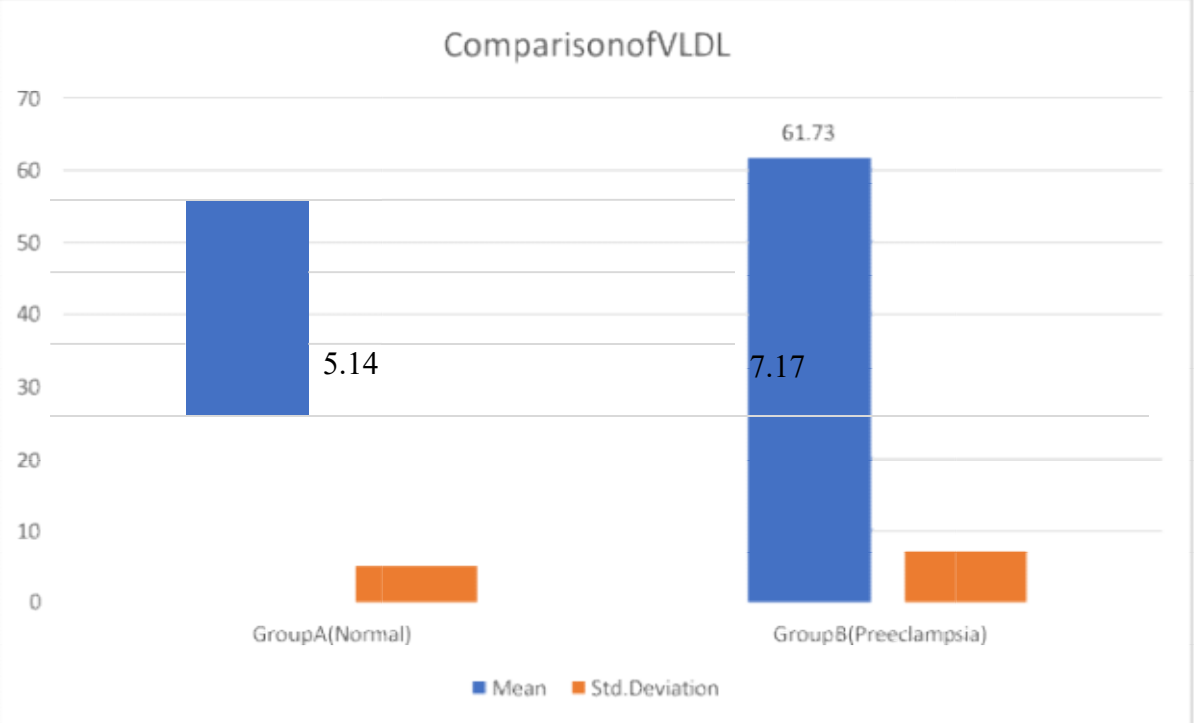
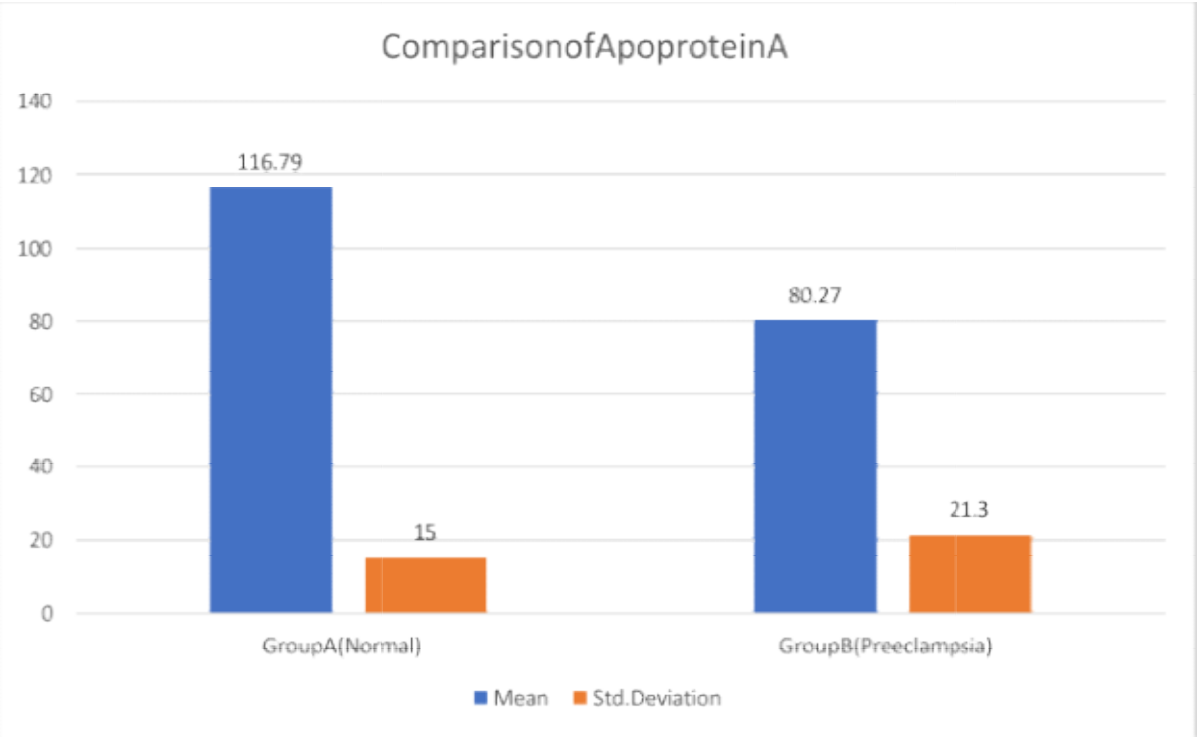


Table 19: Comparison of Apoprotein A

Apoprotein A		
	Group A (Normal)	**Group B (Preeclampsia)**
Mean	116.79	80.27
Std. Deviation	15.00	21.30
	p-value < 0.001	

Table 19 compares the mean Apoprotein A levels among Group-A (Normal) Group-B (Preeclampsia). Apoprotein A level for Group A is 116.79 mg/dL, with a standard deviation of 15.00, while Group B has a significantly lower mean of 80.27 mg/dL and a standard deviation of 21.30. By this Apoprotein A levels between the two groups, with Group A showing notably higher levels compared to Group B.



DISCUSSION

This research analysed maternal and neonatal characteristics in normotensive pregnant women (Group A) in comparison to those with preeclampsia (Group B). The results indicated significant discrepancies in BMI, lipid profiles, delivery procedures, neonatal outcomes, and gestational age at birth. The results were compared with similar studies from the literature.

1. Age Distribution and Mean Age

The present study revealed no statistically significant difference in age distribution (p=0.80) (or) mean age (p=0.98) bettween two group, with a predominance of young primigravidae among participants. This aligns with the research by Jeyabalan et al., which also found no significant age difference between preeclamptic and normotensive women, suggesting that while younger age is a risk factor, it may not consistently achieve statistical significance in cross_sectional study. [48]

2. BMI and Obesity

BMI was significantly increased in the preeclampsia group (p<0.001). This corresponds with the findings of Bodnar et al., who demonstrated that obesity significantly increases the risk of preeclampsia through mechanisms related to chronic inflammation and endothelial dysfunction. A research by Sebire et al. identified BMI as a substantial independent risk factor for preeclampsia. [49,50]

3. Parity Status

No significant difference in parity was observed (p=0.39), however primigravidae were considerably more common in the preeclampsia group. A meta-analysis by Duckitt and Harrington identified primiparity as a known risk factor for preeclampsia; however, this correlation may not consistently reach statistical significance in smaller studies like the present one. [51]

4. Mode of Delivery

The preeclampsia group had signiificantly increassed rate off caesarean sections (p<0.001), supporting conclusions of van Dadelszen et al., who associated preeclampsia with an augmented probability of surgical delivery for both maternal and foetal reasons. [52]

5. Gestational Age at Delivery

Group B experienced significantly earlier delivery (mean 33.35 weeks versus 37.75 weeks, p<0.001), highlighting the risks of iatrogenic or spontaneous preterm birth linked to preeclampsia. Sibai et al. highlighted that preeclampsia is a primary contributor to anticipated preterm birth. [53]

6. Birth Weight and Neonatal Outcome

Group B had a significantly increased prevalence of low birth weight ($p<0.001$), and the APGAR scores at 5 minutes were correspondingly reduced ($p<0.001$). These results correspond with the findings of Xiong et al., who revealed that preeclampsia is associated with intrauterine growth restriction and worse neonatal health outcomes. [48]

7. Lipid Profile and Apoprotein A

Total cholesterol, LDL, VLDL, and triglycerides were significantly elevated in women with preeclampsia, but HDL and apolipoprotein A levels were substantially reduced. The results align with those of Enquobahrie et al., who recognised dyslipidaemia, namely increased triglycerides and decreased HDL levels, as notable features of preeclampsia. Modified lipid metabolism is believed to have a role in endothelial dysfunction, oxidative stress, and placental vascular anomalies, which are essential to the pathophysiology of preeclampsia. [44]

8. Lipid Classification

The study revealed that a higher percentage of women in the preeclampsia cohort were categorised as high-risk concerning LDL, triglycerides, and cholesterol levels. In contrast, normotensive pregnant people demonstrated more favourable lipid profiles. These findings correspond with those of Wiznitzer et al., who established significant connections between lipid abnormalities and the start of preeclampsia. [27]

This study highlights the correlation between metabolic alterations, obstetric outcomes, and neonatal variables in pregnancies affected by preeclampsia compared to normotensive pregnancies. Although sociodemographic parameters such as age and parity are comparable across the groups, the existence of clinical differences underscores the systemic implications of preeclampsia.

This investigation indicates substantial metabolic dysregulation in the preeclamptic population, particularly regarding lipid metabolism. Elevatted levells of totall cholestterol, LDL, VLDL, annd triiglycerides, together wiith decreased HDL, and apoprotein A, suggest that preeclampsia may reflect underlying endothelial dysfunction driven by lipid abnormalities. This dyslipidemic profile supports the concept that preeclampsia shares pathophysiological traits with metabolic syndrome and cardiovascular disease. Altered lipoprotein levels may provoke oxidative stress, therefore damaging the vascular endothelium, a critical pathogenic event in preeclampsia.

The adverse perinatal outcomes seen in the study—specifically preterm delivery, increased caesarean section rates, and low birth weight—indicate both iatrogenic and disease-related issues. The repeated requirement for surgical delivery likely reflects obstetricians' attempts to reduce maternal and foetal danger associated with worsening preeclampsia. Similarly, early

gestational termination, sometimes necessitated by deteriorating maternal health or signs of foetal distress, elevates the prevalence of preterm birth and its related consequences.

The insufficient APGAR scores in neonates born to preeclamptic mothers underscore the heightened risk of prenatal hypoxia and the need for enhanced neonatal resuscitation and surveillance. These newborns are more susceptible to delayed transition due to prematurity and potential intra uterine growtth restriiction, frequently associiated wiith impaired placental perfusion iin hypertensive pregnancies.

The observed anthropometric differences, including increased maternal weightt and Body Mass Index in preeclamptic groups, support the growing evidence linking obesity and excess adiposity to the aetiology of preeclampsia. Adipose tissue operates as an endocrine organ, releasing pro-inflammatory cytokines and adipokines that can exacerbate systemic inflammation and insulin resistance, both linked to the development of hypertension issues during pregnancy.

Thesse findingss highlights need of earlly identiification oof high-riisk women by preconception counselling, consistent prenatal monitoring of metabolic markers, and weight management treatments within the realm of public health. The ongoing dyslipidaemia observed in preeclampsia necessitates the integration of lipid profiling into routine prenattal caree forr womeen wiith riskk factor suchh as increased Body Mass Index, a famiily hiistory of cardiovascular disease, or previous hypertensive pregnancies.

The study provides substantial insight into the many dimensions of preeclampsia, elucidating the interplay of metabolic, vascular, and obstetric factors that affect maternal and foetal outcomes. It emphasises the need for multidisciplinary treatment approaches and the development of predictive models utilising biochemical and clinical indicators to improve early diagnosis and therapy.

Indeed. This section provides a detailed comparison of the findings related to Apolipoprotein A (ApoA) from the current study with those from previous referred studies:

Apolipoprotein A (ApoA) in Preeclampsia

Apolipoprotein A, particularly ApoA-I, plays a crucial protective role during pregnancy by offering anti-inflammatory, antioxidant, and endothelium-stabilizing effects. This study demonstrated that ApoA levells reduced preeclampsia underscoring its involvement in the pathogenesis of preeclampsia. This observation aligns with previous studies.

1. Bayhan et al. (2005): This study evaluated lipid profiles and apolipoproteins in women with preeclampsia, demonstrating that ApoA-I levels were significantly lower in preeclamptic women compared to healthy controls. This reduction coincided with elevated level of

malondialdehyde (MDA), Lp(a), totall chollesterol, triiglycerides, and LDL-C, all suggestive of an atherogenic and oxidative stress-driven environment.[8]

The current study's finding that ApoA levels are reduced in preeclampsia supports Bayhan et al.'s claim that decreased ApoA-I may play a role in the endothelial dysfunction and oxidative stress observed in preeclamptic pregnancies.

2. Alahakoon et al. (2020): This prospective case-control research evaluated lipid profiles in maternal and foetal circulations, demonstrating that ApoA1 levels were reduced in preeclamptic pregnancies, particularly in instances worsened by foetal growth restriction (FGR). The study found no significant differences in total cholesterol or LDL levels across the subgroups; nevertheless, the ongoing reduction in ApoA1 underscores its specificity and potential as a sensitive diagnostic marker for preeclampsia.

The results of our investigation support this conclusion and further highlight the significance of ApoA1 as a predictive biomarker for vascular dysfunction in hypertensive pregnancies.[6]

3. Konrad et al. (2020):

This longitudinal study emphasised relation among lipoprotein(a) and Apo fractions with severiity of preecllampsia, indicating that patients progressing to severe disease had increased Lp(a) and altered Apo levels. While ApoA-I levels were not the central focus, their data contextually supports the idea that apolipoproteins act as dynamic indicators of vascular and inflammatory status throughout pregnancy.

This study enhances the current results by quantifying the reduction in ApoA levels and directly contrasting them across normotensive and preeclamptic cohorts, therefore substantiating the atherogenic shift linked to preeclampsia.[3]

4. Emet et al. (2013): This study primarily investigated changes in lipid markers during gestation and their effects on pregnancy outcomes. Pregnancies complicated by glucose intolerance demonstrated increased triglycerides and altered HDL/LDL profiles, indirectly suggesting later impacts on apolipoproteins. Considering that ApoA-I is the primary component of HDL, the reduced HDL levels seen in preeclampsia—both in their study and ours—can be reasonably deduced to align with decreasing ApoA-I concentrations.[7]

Pathophysiological Implications: • Reduced ApoA-I leads to compromised HDL functioning, crucial for reverse cholesterol transport and protection against oxidative stress. This disruption in preeclamptic people results in endothelial dysfunction and inflammation.

• ApoA-I enhances nitric oxide availability and promotes vascular relaxation; thus, its deficiency may exacerbate the hypertensive state seen in preeclampsia. • The concurrent elevation of atherogenic lipids and reduction in ApoA-I creates a pro-inflammatory, pro-

thrombotic milieu detrimental to both maternal and foetal health.

The reduction in ApoA levels seen inn thiis study is consistent with result of Bayhan et _al., Alahakoon et_al., and other referenced research. The consistent findings highlight the importance of ApoA-I as a biomarker for endothelial and inflammatory conditions during pregnancy and suggest its potential application in early preeclampsia risk assessment. Future interventional research should evaluate whether elevating ApoA-I levels might mitigate the risk or severity of pulmonary embolism.

Limitations

The study provides important insights into the metabolic and clinical differences between normotensive and preeclamptic pregnancies; nevertheless, several limitations must be acknowledged:

1. **Sample Size and Single-Center Design**: The study had 96 participants and was conducted at a single tertiary care institution. This may affect the generalisability of the findings to a broader population, especially across different geographies and healthcare settings.
2. **Cross-Sectional Nature**: The study strategy is observational and cross-sectional, gathering data at a single point during pregnancy. This constrains the ability to ascertain causality or examine temporal relationships between lipid abnormalities and the onset or progression of preeclampsia.
3. **Uncontrolled Confounding Variables**: Many potential confounders, such as dietary habits, physical activity, socioeconomic status, genetic predisposition, insulin resistance, and pre-pregnancy lipid levels, were neither controlled nor adjusted for. These variables may have influenced the lipid profile and obstetric outcomes.
4. **Lack of Longitudinal Follow-up**: Neonatal outcomes were limited to birth measurements and APGAR scores. No follow-up was performed to assess long-term morbidity or growth trajectories of the neonates, which might have provided more comprehensive insights into infant health.
5. **Absence of Inflammatory and Oxidative Stress Markers**: The study may have been improved by include markers such as C-reactive protein, serum uric acid, or indicators of oxidative stress, as preeclampsia is associated with systemic inflammation and endothelial dysfunction, to clarify the pathophysiological mechanisms more clearly

Conclusion

This study highlights a strong association between preeclampsia and impaired lipid metabolism, demonstrating significantly elevated levels of total cholesterol, triglycerides, LDL, and VLDL, and reduced levels of HDL and apoprotein A. Preeclamptic pregnancies were associiated wiith increaseed rates off caesarean delivery, preeterm biirth, low-birth-weight, and reduced APGAR scores, underscoring thee substaantial effects oof thiis condition on the mateernal annd neonatal heealth.

The results support existing research that recognises obesity and dyslipidaemia as critical contributors to the aetiology of preeclampsia. Finding underscore necessity foor identiification management off metabolic rissk facttors during gestation. Integrating lipid profile and BMI evaluations into routine prenatal care may enable the early detection of women at increased risk.

Summary

This cross-sectional comparative study evaluated 96 pregnant women, evenly distributed between normotensive (Group A) and preeclamptic (Group B) groups, to examine differences in clinical, obstetric, and biochemical attributes. No significant differences were seen in age or parity across the groups. Preeclamptic women had significantly raised BMI, lipid abnormalities (total cholesterol, LDL, VLDL, triglycerides), and worse pregnancy outcomes, characterised by a higher incidence of caesarean sections, earlier gestational age at birth, reduced neonatal weights, and lower APGAR scores.

The study demonstrates that preeclampsia is associated with significant maternal metabolic disturbances and worse neonatal outcomes. These findings highlights thhe necessity for compreheensive prenatal screening, riisk assessmeent, and multimodal treatment strategiies too mitiigate issues associated with preeclampsia.

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