"MATERNAL LIPID PROFILE DURING SECOND TRIMESTER ON PREGNANCY OUTCOMES AND ITS COMPLICATIONS -A ONE YEAR OBSERVATIONAL STUDY"

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ABBREVIATIONS

HDL- High density lipoprotein

LDL- Low density lipoprotein

VLDL- Very low density lipoprotein

GDM- Gestational diabetes Mellitus

TC- Total cholesterol

PTB- Preterm birth

DIPSI- Diabetes in Pregnancy Study group of India

GA - Gestational age

LMP- Last Menstrual Period

EDD- Expected date of delivery

POG- Period of gestation

CRL- Crown Rump Length

USG- Ultrasonography

BMI- Body mass index

PIH- Pregnancy induced hypertension

LGA- Large for gestational age

SGA - Small for Gestational Age

AGA - Appropriate for Gestational Age

EFW- Estimated Fetal Weight

USG- Ultrasonography

OPD- Outpatient department

MAPE- Mean absolute percentage error

DM- Diabetes Mellitus

WHO- World health organization

ACOG- American College of Obstetricians and Gynecologists

NIH - National Institute of Health

Sl.No.- Serial Number

KLE's- Karnataka Lingayat Educational Society

KAHER- KLE Academy of Higher Education and Research center

JNMC- Jawaharlal Nehru Medical College

SD- Standard Deviation

CI- Confidence interval

SE- Standard error

ABSTRACT

Background of the study:

Pregnancy is associated with hyperlipidemia. Problems arises if there is dyslipidemia surpassing the normal levels in pregnancy. Hyperlipidemia causes atherosis in the uteroplacental spiral arteries which results in decreased blood flow to the baby resulting in preeclampsia (PE). Insulin resistance is increased during pregnancy leading to gestational diabetes mellitus (GDM). Patients with dyslipidemia are known to have perinatal complications such as small for gestational age newborns, preterm birth and stillbirth.

Objectives

To study the association of serum lipid levels during the second trimester with the development of pregnancy-associated complications such as preeclampsia, GDM and preterm delivery.

Methods

All antenatal cases from 14- 28 weeks of gestation attending the outpatient Department of OBG in KLE's Dr Prabhakar kore hospital from 1st January 2020 to 30th june 2021. Fasting venous blood samples of the women were sent to lab for estimation of lipid profile which includes total cholesterol, triglycerides,low density lipoprotein(LDL), very low density lipoprotein(VLDL) and high density lipoprotein(HDL). All the patients were followed up at the time of delivery and categorized mainly into preeclampsia and preterm delivery.

Result- In our study, GDM patients had a higher level of triglycerides and VLDL i.e., 170±15.73 mg/dl and 34.02 ± 3.14 mg/dl respectively than the control group which had 144.39±22.13mg/dl and 28.8±4.43mg/dl with a p value of 0.0008 which is statistically significant. However, many of the preeclampsia patients in our study delivered late preterm with mean age of 35.40 ± 5.08 weeks.

Conclusion-

In conclusion, study population of 175 pregnant women serum triglycerides and VLDL were statistically significantly higher in GDM women compared to the control group. However, there was no difference between the GDM and control groups in terms of cholesterol, LDL or HDL. There was no difference in lipid markers between preterm and full-term deliveries. In my study of PE patients delivered at a much earlier gestational age than the normotensive patients and there was no difference in the lipid between the two groups. However larger sample size is required.

Key words – Triglycerides (TG),Very low density lipoprotein(VLDL),Low Density lipoprotein(LDL),High density lipoprotein(HDL),Gestational Diabetes Mellitus (GDM),Preeclampsia(PE)

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INTRODUCTION

Pregnancy is characterized by major adaptations in the maternal anatomy, physiology, biochemical and endocrine systems to achieve a successful pregnancy. These alterations influence every organ system and have an impact during pregnancy and after delivery. These adaptations are caused by various hormonal and genetic factors in order to meet the needs of the fetus and to establish favourable conditions for the fetal development and growth.[44]

There are various factors that are implicated in the pathogenesis of Preeclampsia, GDM and preterm delivery including genetic, immune, vascular, hormonal, endocrine and oxidative stress. These have led to identification of potential candidate markers from various cross-sectional studies for early diagnosis of maternal adverse outcomes.[70]The aim of this study is to find out any association between maternal lipid profile and different pregnancy outcomes.

Normal lipid changes in pregnancy

Lipid metabolism can be divided into two phases:

During the first two trimesters, primarily it is anabolic state. There is marked deposition and hypertrophy of maternal adipocytes with increased expression of insulin receptors such that the glucose is available to meet the metabolic demand of the growing fetus.[1]

There is increased lipid synthesis between 10 and 30 weeks of pregnancy promoted by maternal hyperphagia in early pregnancy and increase in insulin sensitivity which increases the activity of lipoprotein lipase in adipose tissue or it remains constant, controlling fat intake by the tissue.[1]

Lipid metabolism in the third trimester is a catabolic state caused by a decrease in insulin resistance. This enhances the lipolysis of stored triglycerides in

adipocytes. Insulin resistance results in a decrease lipoprotein lipase in adipocytes leading to a decrease in the uptake of fatty acids from plasma triglyceride rich lipoproteins. There is a role of hormones in inducing hyperlipidemia in the third trimester.[1]

Table 1: Role of Hormones in Inducing Hyperlipidemia in the Third Trimester

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

By the end of third trimester plasma cholesterol levels are 50% higher than routinely seen pre-pregnancy while triglyceride levels are doubled. These changes can be viewed as important for enhanced availability of substrates for the fetus[47].It is important to note that the maximum plasma cholesterol value never exceeds 250 mg/dL at any time during normal pregnancy even with the marked increases in triglyceride levels that occur normally. Even the total lipoprotein levels increase, the atherogenic index LDL/HDL remains unchanged[48].Physiological hyperlipidemia / hypertriglyceridemia is distinguished from pathological dyslipidemia by an increase in HDL-C in normal women as they progress through pregnancy.[49,42]

In women with pregnant hypertension, preeclampsia or DM has elevated LDL fractions with reduced HDL-C levels which appear to be more pronounced.[42]

DYSLIPIDEMIA IN PREGNANCY

Adverse pregnancy outcomes include both maternal and fetal effects

Pre-eclampsia and gestational diabetes are not only two of the most common adverse pregnancy outcomes, they also have short and long term consequences for both the mother and the fetus. Pre-eclampsia is a rapidly progressive condition that affects 5-8% of pregnancies. Preeclampsia refers to the new onset of hypertension and proteinuria **or** the new onset of hypertension and significant end-organ dysfunction with or without proteinuria after 20 weeks of gestation in a previously normotensive woman.[64]

Elevated plasma lipid and lipoproteins may induce endothelial dysfunction secondary to oxidative stress[56]. Dyslipidemia may impair trophoblast invasion thus contributing to a cascade of pathophysiologic events that lead to the development of preeclampsia. Higher levels of TG, lower levels of HDL-C and more fractionation of small dense atherogenic LDL particles are also associated with this gestations[42].Lipoprotein (a) levels are higher in preeclamptic women, but the relevance of this is unknown [43]. Several researches have proven proatherogenic trends in lipid concentrations that occur before preeclampsia.[42, 43]

Maternal risks of Hypertensive disorders in pregnancy

Fetal growth restriction, abruptio placentae and stillbirth can be sequelae of inadequate placentation due to preeclampsia. For the fetus, preeclampsia can lead to growth restriction and oligohydramnios as well as medically or indicated preterm birth.[65, 66]

Patients with pregnancy associated hypertension are at increased risk of coronary heart disease, stroke, heart failure and kidney disease later in life[51].The American Heart Association considers a history of preeclampsia or pregnancy-induced hypertension a major risk factor for development of CVD.[52]

Associations between preeclampsia and autism spectrum disorder (ASD) and possibly attention-deficit/hyperactivity disorder have also been observed.[53-55]

Effects of GDM

Elevated adipose tissue by the accumulation of free fatty acid levels may be a mechanism for markedly increased triglyceride levels in women with GDM, as increased free fatty acids can aggravate insulin resistance[11]. Insulin resistance also promotes abnormalities in very low-density lipoprotein cholesterol (VLDL-C), which are found in non HDL-C as well as LDL-C and intermediate-density lipoprotein.[11]

Consequences of GDM include large for gestational age, macrosomia, preeclampsia, gestational hypertension, polyhydramnios, stillbirth, increased chances of caesarean section, increased use of instrumental delivery and abruptio placenta.[17] Neonatal morbidity includes hypoglycemia, hyperbilirubinemia, hypocalcemia, hypomagnesemia, polycythemia and respiratory distress.[17] Long term consequences include type 2 diabetes mellitus and obesity in later life.[17]

Association of dyslipidemia with preterm delivery

Preterm birth was defined as gestational age at delivery <37 weeks. Cholesterol is required for uteroplacental vascularization, placental steroid production and placental transport. Hyperlipidemia, on the other hand is thought to be a cause of inflammation and oxidative stress. Inflammation is well known to be a substantial risk factor in PTB.[41]

Recently pravastatin has been used in the mid trimester to successfully reduce preeclampsia in persons with a prior history of preeclampsia.[59]

Though the obstetric care has improved in recent times. Pregnancy complications and perinatal morbidity are still present. Therefore, it is of clinical importance to prevent these adverse outcomes. One of the causational factors for perinatal morbidity and mortality could be maternal atherogenic lipid profile early in pregnancy. The aim of the study was to find out the association of lipid profile in second trimester with preeclampsia, diabetes mellitus and preterm delivery.

OBJECTIVES

To study the association of serum lipid levels during the second trimester with the development of pregnancy-associated complications such as preeclampsia, GDM and preterm delivery.

REVIEW OF LITERATURE

Lipids

In plasma, cholesterol and triglycerides are insoluble lipids. They are attached to lipoproteins which transport lipids to various tissues for energy use, lipid deposition, steroid hormone synthesis and bile acid generation making them soluble. Esterified and unesterified cholesterol, triglycerides and phospholipids, as well as protein make up lipoprotein.[2]

Structure of Lipoproteins

Cholesterol and triglycerides are insoluble lipids in water; they must be carried in the bloodstream in conjunction with proteins (lipoproteins).[2]

Lipoproteins are complex particles with a central hydrophobic core made up mostly of non-polar lipids (cholesterol esters and triglycerides). A hydrophilic layer comprising phospholipids, free cholesterol and apolipoproteins surrounds this hydrophobic core.[2]

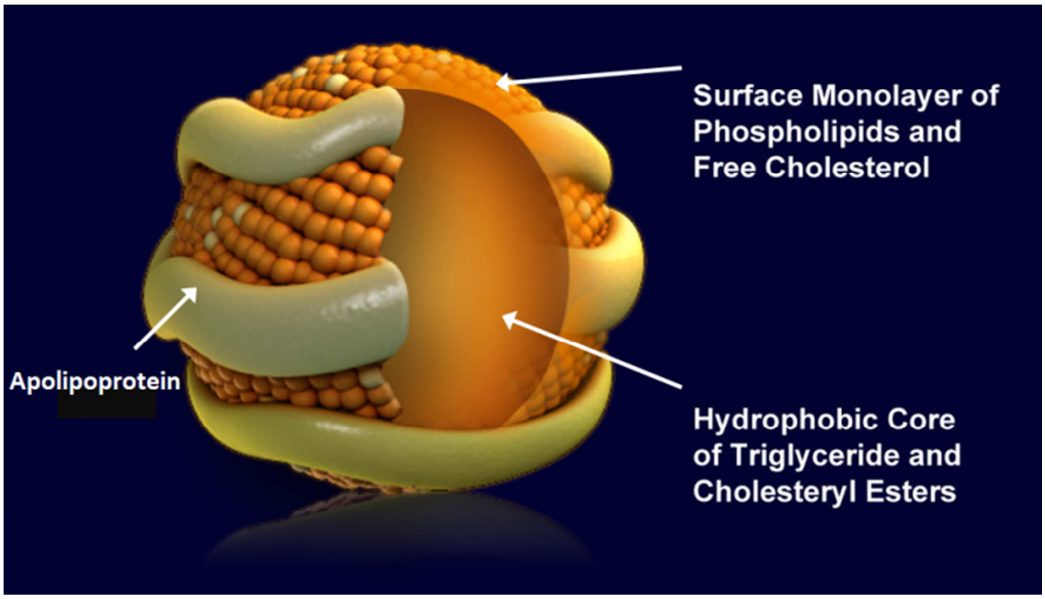


Figure 1 : Structure of Lipoprotein

They are majorly classified into Chylomicrons, Chylomicron remnants, very low density lipoprotein (VLDL), intermediate density lipoprotein (IDL), low density lipoproteins (LDL) and high density lipoproteins (HDL).

Chylomicrons

The intestine produces large triglyceride-rich particles that aid in the transfer of dietary triglycerides and cholesterol to peripheral tissues and the liver. Apolipoproteins A-I, A-II, A-IV, A-V, B-48, C-II, C-III and E are found in these particles. Each chylomicron particle contains one Apo B-48 molecule, which is the core structural protein. The size of chylomicrons fluctuates according to how much fat is ingested.[2]

Chylomicron Remnants

The removal of triglycerides from chylomicrons by peripheral tissues leads in chylomicron remnants, which are smaller particles. These particles are higher in cholesterol than chylomicrons and are pro-atherogenic.[2]

VLDL

These particles are produced by the liver and are triglyceride rich. They contain apolipoprotein B-100, C-I, C-II, C-III and E. Apo B-100 is the core structural protein. These VLDL particles are smaller than chylomicron.[2]

IDL

Triglycerides are removed from VLDL by muscle and adipose tissue, resulting in the production of cholesterol-rich IDL particles. Apolipoproteins B-100 and E are found in these particles. These IDL particles are proatherogenic.[2]

LDL

The majority of cholesterol in circulation is carried by LDL. Each LDL particle contains one molecule of Apo B-100. LDL is made up of a wide range of particle sizes and densities. Hypertriglyceridemia, low HDL levels, obesity, type 2 diabetes (i.e., people with the metabolic syndrome), viral and inflammatory conditions are all linked to an abundance of tiny dense LDL particles. Compared to big LDL particles, tiny dense LDL particles are more pro-atherogenic. Because small compact LDL particles have a lower affinity for the LDL receptor, they spend more time in the circulation. They also enter the artery wall more easily and bind to intra-arterial proteoglycans more strongly, trapping them in the arterial wall. Finally, small dense LDL particles are more prone to oxidation, which could lead to increased macrophage absorption[2].Since tiny LDL particles are retained longer in the artery wall, reactive oxygen species can modify surface phospholipids and unesterified cholesterol for longer[42]. Furthermore, the tiny LDL phenotype is linked to several other risk factors, including higher triglycerides, very low-density lipoprotein (VLDL), intermediate density lipoprotein (IDL), lower HDL,HDL2 concentrations, and insulin resistance.[44]

HDL

One possible mechanism through which HDL may be anti-atherogenic is that these particles play a significant role in reverse cholesterol transport from peripheral tissues to the liver. HDL particles also contain anti-oxidant, anti-inflammatory, anti-thrombotic and anti-apoptotic capabilities, which may help explain why they can prevent atherosclerosis. Cholesterol and phospholipids are abundant in HDL particles.[22]

Glucose is the most essential nutrient that crosses the placenta in terms of quantity, followed by amino acids. The fetoplacental unit comprises 500mg of protein in late gestation. Fat stores are preferentially utilized as a substrate for fuel metabolism during pregnancy and thus protein catabolism is reduced. The accumulation of lipids in maternal tissues and the development of maternal hyperlipidemia are the effects of lipid metabolism adaptation.[44]

Fat is the body's main source of energy storage and contains a lot of fatty acids. Subcutaneous tissue, adipose tissue, retroperitoneal areas and the omentum all retain extra fat. The body's fat serves to protect it from excessive cold and heat.[1] Cholesterol is created both endogenously exogenously and is required for optimal fetal development. It aids in the creation of cell membranes, as well as the maintenance of membrane integrity and the preservation of cholesterol rich domains required for most membrane associated signalling .It also serves as a precursor for a number of essential hormones, including steroids, vitamin D and bile acids.[4]

Lipoprotein

Lipids and proteins combine to form lipoproteins. Lipoprotein proteins are known as apolipoproteins. They have two main functions: they carry lipids to the liver and they supply various lipids to different organs via lipoprotein.[2]

Serum lipid profile

otal cholesterol, HDL cholesterol, LDL, VLDL cholesterol and Triglyceride are the elements of the serum lipid profile. Pre-pregnancy obesity directly causes metabolic dysregulation and impairs pregnancy's physiological adaptations, increasing the risk of adverse outcomes for both the mother and the fetus.

Dyslipidemia is increased by the following acquired condition [3]

1) Obesity, metabolic syndrome, type 2 diabetes, chronic renal failure, HIV, hepatocellular disease and chronic inflammatory diseases are all examples of insulin resistance.

2) Renal illness

3) Pregnancy

4) A high-calorie, high-saturated-fat or high glycemic index diet

5) Hypothyroidism

6) Alcohol consumption in excess of two drinks per day for males and one drink per day for women is considered excessive.

7) Certain medications such as glucocorticoids, thiazides, bile acid sequestrants, antineoplastic agents, anti-retroviral drugs and oral estrogens.

Atherosclerosis is caused by abnormal lipoprotein metabolism, which is a major risk factor.

Endothelial cells, macrophages, smooth muscle cells and T lymphocytes are among the cells within the artery that can oxidise LDL. Vitamin E inhibits the absorption of oxidised LDL by lowering CD36 receptor expression.[43]

CHD is linked to elevated plasma amounts of oxidised LDL.[42]

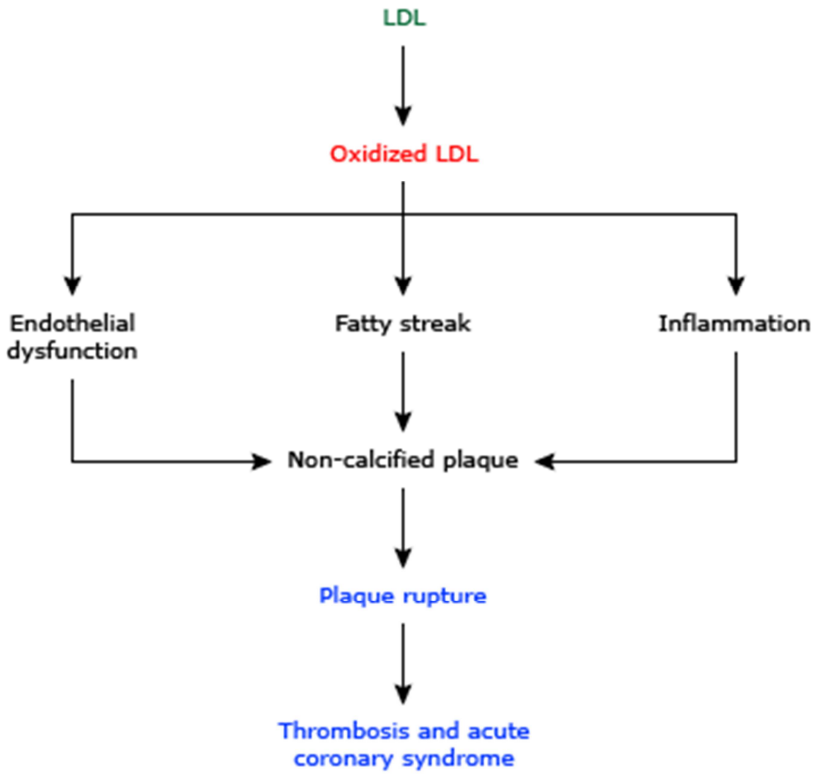


Figure 2: Schematic representation in the pathogenesis of atherosclerosis

Other coronary risk factors, low HDL levels, smoking, hypertension, diabetes mellitus and estrogen deficiency also enhance the oxidation of LDL[25].The endothelial cell surface has been hypothesised to be disrupted by oxidised lipoproteins such as LDL, HDL, remnant lipoproteins and phospholipid which promote inflammation through macrophage cytokine release, impaired cholesterol efflux and the development of atherosclerosis.[26]

Role of oxidized LDL in atherosclerosis[64, 79]

1. Endothelial injury
2. Alteration in vascular tone
3. Monocyte/macrophage recruitment
4. Increased uptake of LDL by macrophages with foam cell formation
5. Induction of growth factors
6. Increased platelet aggregation

Raghuram Pusukuru et al. conducted a study in Navi Mumbai in 2012 on the evaluation of lipid profile in the second and third trimesters of pregnancy, reporting mean cholesterol levels of 214.6±18.16 mg/dl and 242.65±20.44 mg/dl in the second and third trimesters respectively.

In the second and third trimesters, the mean triglyceride levels were 188.68±2 0.88 mg/dl and 216.78±20.09 mg/dl respectively.

In the second and third trimesters, the mean HDL –

Cholesterol levels were 49.13±6.15 mg/dl and 43.07±4.34 mg/dl respectively.

In the second and third trimesters, the mean LDL –

Cholesterol levels were 92.41±18.94 mg/dl and 137.82±13.45 mg/dl respectively.[6]

Tanja M Vrijkotte and colleagues investigated the maternal lipid profile during early pregnancy and pregnancy complications and outcomes published in 2012, every unit rise in triglycerides was linked to an increased risk of PIH, preeclampsia and induced preterm delivery.[7]

Recommended reference levels for blood lipids throughout early and middle p regnancy: a retrospective study in China on 17,610 singleton pregnancies in 2014 was published by Chen Wang et al. From early to middle pregnancy, serum lipid levels in creased considerably, with TG increasing the most. In early pregnancy the values for

TC < 5.64 mmol/L, TG < 1.95 mmol/L, HDL-C > 1.23 mmol/L ,LDL-C < 3.27 mmol/L and in second trimester are TC < 7.50 mmol/L, TG < 3.56 mmol/L, HDL-C > 1.41 mmol/L, and LDL-C < 4.83 mmol/L.

Higher numbers out of the range lipids during early and middle pregnancy were correlated with a higher risk of adverse pregnancy outcomes.[8]

In a study published in 2016, Manish Kumar Mishra compared the lipid profil es of pregnant women to those of non-pregnant women as controls.
TG, total cholesterol and LDL levels were all high in all three trimesters, with LDL le vels being greater in the second and third trimesters.

In a research of non-pregnant and pregnant women, Loke D.F et colleagues found that total cholesterol, triglycerides and LDL levels were higher in pregnant women than non-pregnant women. Because of metabolic changes and diet before and after delivery, lipid levels in the postpartum period of the same women are found to be even higher.[12]

	Non-Pregnant adult	1st trimester	2nd trimester	3rd trimester
Total Cholesterol	<200	141-210	176-299	219-349
HDL	40-60	40-78	52-87	48-87
LDL	<100	60-153	77-184	101-224
VLDL	6-40	10-18	13-23	21-36
Triglycerides	<150	40-159	75-382	131-453
Apolipoprotein A1	119-240	111-150	142-253	145-262
Apolipoprotein B	52-163	58-81	66-188	85-238

Reference: William's Obstetrics, 25th edition

Figure 3: Lipid profile

Sandra G Okala et al studied maternal high lipid levels resulted in low birth weight and small for gestational age which was conducted in 573 pregnant women with term deliveries.[15]

A large study included 5702 pregnant women done by Maria C Adank et al concluded that maternal triglycerides and remnant cholesterol levels were associated with LGA independent of maternal BMI.[16]

Dr. A Sonagra's research found that lipid levels increased during the second an d third trimesters with the exception of HDL, which was decreased.[18]

In a study conducted by Maria, gestational lipid profile as an early marker of metabolic syndrome in later life in 2020, TGs and remnant cholesterol were associated with increased risk of metabolic syndrome in later life.[14]

Two systematic reviews Karalis et al and Kusters et al both found no evidence that statins cause congenital anomalies independent of medical conditions associated

with their use[57, 58]. Recently pravastatin has been used in the midtrimester to successfully reduce preeclampsia in persons with a prior history of preeclampsia.[59]

Role of Lipids in GDM

Placental release of diabetogenic substances such as growth hormone, corticotropin-releasing hormone, placental lactogen (chorionic somatomammotropin), prolactin and progesterone causes insulin resistance throughout pregnancy. These and other metabolic changes, which are most prominent in the third trimester ensure that the fetus gets enough nourishment.

Gestational diabetes mellitus (GDM) develops in pregnant people whose pancreatic function is insufficient to overcome the insulin resistance associated with the pregnant state.

Diabetes is a systemic disease of metabolism that has various complications. This disease can be classified in pregnancy as,

1. Pre-Gestational Diabetes or Overt diabetes
2. Gestational Diabetes Mellitus (GDM)

Untreated hyperglycemia in early pregnancy increases the chance of miscarriage and delivering a child with a congenital defect and mothers may have undiagnosed diabetic consequences (e.g., nephropathy, retinopathy) that put them at risk during pregnancy.[37]

GDM has been associated with increased risks of: [31-34]

1. Hypertensive disorders of pregnancy (Preeclampsia, gestational hypertension)
2. Large for gestational age newborn (LGA) or macrosomia
3. Birth trauma to mother or new born
4. Operative delivery (caesarean, assisted vaginal)

5. Perinatal mortality
6. Fetal/neonatal hypertrophic cardiomyopathy
7. Neonatal respiratory problems and metabolic complications (e.g., hypoglycemia, hyperbilirubinemia, hypercalcemia, polycythemia)
8. Polyhydramnios

In case-control research in Karnataka, Bharathi K. R et colleagues used 50 pregnant women with GDM as the case group and 50 non-GDM patients as the control group. They performed a lipid profile on all 100 women, finding that triglyceride levels were significantly higher and triglyceride levels may be used as a diagnostic for gestational diabetes mellitus.[36]

Many studies indicate that triglyceride levels are raised in all three trimesters but they are especially high in GDM mothers. In GDM mothers, HDL cholesterol is likely to be lower (KK Ryckman et al).[67]

In a systematic review of 292 studies and 97,880 women which showed women with GDM had significantly higher TG which occurred in the first trimester and persist afterwards.[17]

Obese adults with high lipid levels were connected with GDM, according to a study of 2488 pregnant women's early pregnancy maternal lipid profile and the risk of GDM.[19]

Lipids in Preeclampsia

Preeclampsia is a multisystem progressive disorder characterized by the new onset of hypertension (Systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg) and proteinuria or the new onset of hypertension and significant end-organ dysfunction with or without proteinuria in the last half of pregnancy or postpartum

It's caused by placental and maternal vascular malfunction, and it lasts for a long time after delivery.

The four major hypertensive disorders in pregnant women

1. Gestational Hypertension
2. Preeclampsia
3. Chronic hypertension
4. Hypertension superimposed pre-eclampsia
5. Eclampsia

The diagnosis of preeclampsia with severe features (formerly severe preeclampsia) is made in the subset of women with preeclampsia who have severe hypertension and/or specific signs or symptoms of significant end-organ dysfunction that signify the severe end of the preeclampsia spectrum.

The presence of one or more of the following

1. Systolic blood pressure ≥160 mmHg or diastolic blood pressure ≥110 mmHg on 2 occasions at least 4 hours apart while the patient is on bedrest.
2. New-onset cerebral or visual disturbance such as photopsia, scotomata, cortical blindness, retinal vasospasm or severe headache (despite analgesic therapy).
3. Hepatic abnormality: serum transaminase concentration >2 times the upper limit of the normal range or severe persistent right upper quadrant or epigastric pain unresponsive to medication.
4. Thrombocytopenia <1,00,000 platelets/microL
5. Renal abnormality - serum creatinine >1.1 mg/dL
6. Pulmonary edema

Mild to severe microangiopathy of target organs such as the brain, liver, kidney and placenta. Pulmonary edema, cerebral haemorrhage, hepatic failure, renal failure and death are all possible maternal complications. Placental hypoperfusion, placental malfunction and also the necessity for preterm birth are the neonatal complications.

Both maternal and placenta factors are known to play a role in preeclampsia pathogenesis. Early in pregnancy, abnormalities in placental vasculature development may cause relative placental under perfusion/hypoxia/ischemia which causes the release of antiangiogenic factors into the maternal circulation altering maternal systemic endothelial function causing hypertension and other disease manifestations (hematologic, neurologic, cardiac, pulmonary, renal and hepatic dysfunction). The cause of aberrant placental development and the events that follow are unknown.

Pathology of Preeclampsia

1. Abnormal Placentation: Abnormal remodelling of spiral arteries.
2. Deficient trophoblast differentiation — One putative reason for defective trophoblast invasion of the spiral arteries is defective trophoblast differentiation.
3. Hypoperfusion of the placenta, hypoxia and ischemia — Abnormal placental development appears to be both a cause and a result of hypoperfusion.
4. Failure of decidualization in some patients may result in downregulated cytotrophoblast invasion according to some research.
5. Immunological factors: Prior exposure to paternal/fetal antigens seems to protect against preeclampsia[42-49] which led to a focus on immunologic variables as a probable contributor to aberrant placental development. According to some studies nulliparous women, women who change partners between pregnancies, women who have long interpregnancy intervals, women who use barrier

contraception, and women who conceive via intracytoplasmic sperm injection have less exposure to paternal antigens and have a higher risk of developing preeclampsia.

6. Genetic Factor: Primigravida women with a family history of preeclampsia (e.g., a mother or sister who has had the condition) have a two to fivefold increased risk of the disease than primigravida women without a family history. A meta-analysis reported studies of PAI-1 4G/5G polymorphism (recessive model) showed strong consistent evidence for an association with risk for preeclampsia.
7. Environmental risk factors such as low calcium intake, high body mass index, IVF conception has higher risk of preeclampsia.
8. Inflammation: Thromboxane A2 (TxA2) belongs to the eicosanoids family of lipids, which are metabolites of arachidonic acid. TxA2 has prothrombotic characteristics because it increases platelet activation and aggregation. TxA2 is also a recognised vasoconstrictor that is triggered when tissue is injured or inflamed. Increased TxA2 activity could play a role in myocardial infarction, stroke, atherosclerosis and bronchial asthma pathogenesis.[38]
9. Vasospasm: The arteries are typically shown to be prone to vasospasm in many unexplained episodes of PIH and this can be related to local or systemic causes. Endothelial activation in atherosclerotic blood arteries is one of the local factors.

In a study done on Preeclampsia and lipid levels which told preeclampsia occurs in 3-5% of pregnancies and is an important cause of fetal maternal morbidity and mortality worldwide.The mean cholesterol,LDL cholesterol were higher in Preeclampsia group.[21]

A study done by J G Ray et al studied the rise in TG was significantly higher among PE patients which was taken from 19 case controls and 3 prospective cohort

studies.[20] In a study done by S.Niromanesh et al have told preeclampsia, preterm birth, and gestational diabetes have all been linked to hypertriglyceridemia.[22]

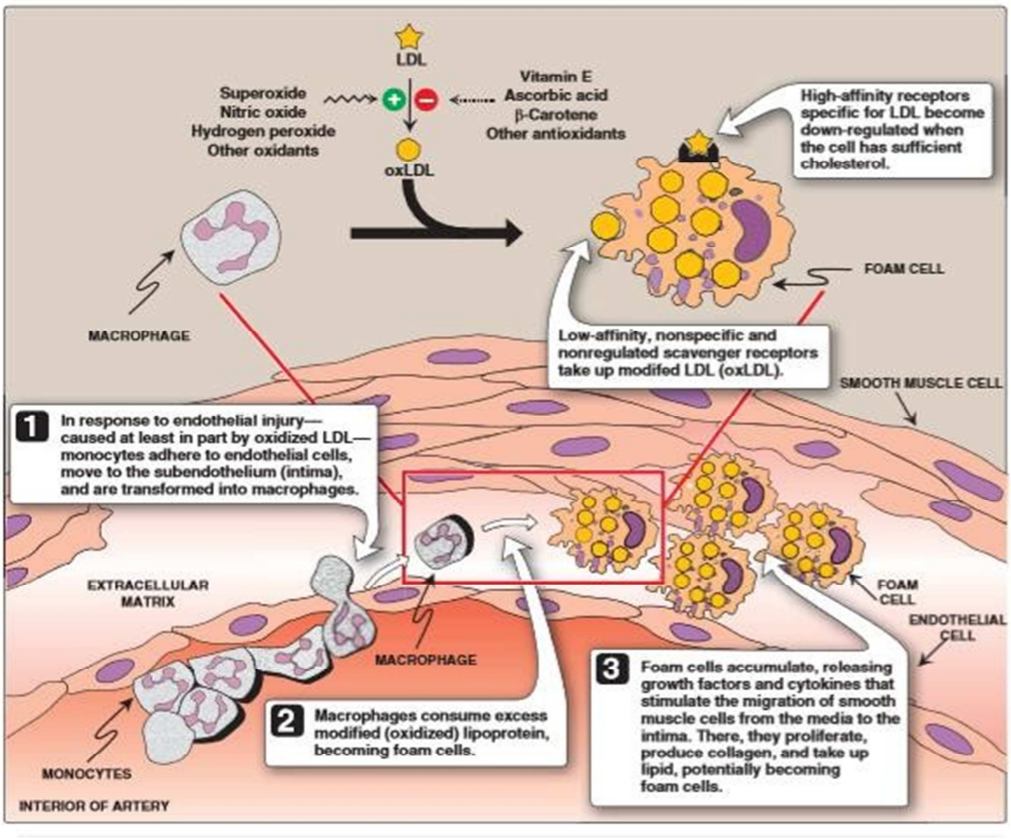


Figure 4: Formation of an atherosclerotic plaque

Abnormal lipid metabolism during pregnancy is the important pathogenesis of atherosclerosis, ischemic heart disease, intrauterine growth restriction and hypertension (Brizzi, P). One of the main factors for perinatal mortality and morbidity must be the maternal atherogenic lipid profile early in pregnancy.[10]

In a study conducted Maria C adnak done in 5690 women studied that triglycerides and remnant cholesterol in early pregnancy was associated with preeclampsia, with hypertension in postpartum and they are at risk of future cardiovascular disease.

Role of lipids in Preterm delivery

Preterm Delivery

Preterm birth (PTB) refers to a birth that occurs from 20+0 to 36+6 weeks of gestation. PTB is relatively common, occurring in 5 to 18 percent of births worldwide. It is divided into[60]

1. Spontaneous Preterm-80%
2. Preterm labor-40-50%
3. Preterm premature rupture of membranes (PPROM)-20-30%.
4. Iatrogenic Fetal or maternal indications-20%

Table 2: Causes of Preterm delivery

Spontaneous	Iatrogenic
Idiopathic Stress	Hypertension
PPROM	Diabetes
Infection	IUGR (Intrauterine growth restriction)
Multiple pregnancies	
Cervical dysfunction	
Antepartum Haemorrhage	

In a study Janet M Catov et al concluded that high cholesterol value in early pregnancy had increased risk for preterm birth.Overweight women who delivered <34 weeks had elevated cholesterol and LDL.[23]

Bacterial infections in the genital tract such as gonococcal and staphylococcal infections have been linked to preterm labour. The presence of phospholipase in the bacterial cells is responsible for this. This enzyme hydrolyses the arachidonic acid in

the cell membrane releasing prostaglandins that cause premature delivery as well as cytokines and inflammatory mediators that weaken membranes and cause early rupture of membranes.[39]

Study by Maryam Moayeri et al suggest that women with high TG levels may be at an increased risk of spontaneous Preterm delivery(sPTD), no link between high- and low-density lipoprotein cholesterol and the risk of sPTD was seen. In the second trimester, high homocysteine levels are linked to sPTD. Triglycerides and homocysteine's role in sPTD should be investigated further.[40]

In a study done by Lanay M Mudd stated that extremely low TC, HDL-C, and LDL-C were linked to a modest increase in the chance of medically induced preterm birth, while high TC, LDL-C and TG were linked to a slight increase in the risk of spontaneous preterm birth.[41]

The relationship between lipid in pregnancy and preterm labor was studied by Zohreh Aghaiem et al in the first trimester of pregnancy, a high maternal TG level was found to be a major risk factor for premature delivery. The probability of spontaneous preterm labour was raised when TC, TG and LDL-cholesterol levels were high.[69]

MATERIALS AND METHODS

STUDY SETTING:

The study was conducted in the department of Obstetrics and Gynecology at "KAHER's Dr. Prabhakar Kore Charitable Hospital and Medical research centre", attached to "Jawaharlal Nehru medical college", Belagavi

STUDY DESIGN:

An observational study

STUDY DURATION:

The study was conducted for a duration of 1 and half year

STUDY PERIOD:

1st January 2020 to 31st June 2021.

SOURCE OF DATA:

The study enrolled all pregnant women seeking prenatal care between 14 and 28 weeks who matched the inclusion criteria and given written informed consent.

SELECTION CRITERIA:

Inclusion criteria-

- Singleton pregnancies
- Period of gestation between 14-28weeks.

- Blood pressure of women less than 140/90mm hg without any endocrine or systemic disorder

Exclusion criteria-

Women with following conditions will be excluded -

- Chronic illness like cardiovascular diseases, thyroid disorder
- Type 2 Diabetes mellitus
- Multiple pregnancy
- Women with treatment on lipid lowering drugs
- Cervical incompetence
- Patients not willing to participate

ETHICAL CLEARANCE:

The study was approved by "Ethical and Research committee, KAHER's Jawaharlal Nehru Medical College" Belagavi, prior to its commencement (Annexure-3).

INFORMED CONSENT:

All participants fulfilling the selection criteria were explained regarding the purpose of the study in their own vernacular language and written informed consent was obtained prior to their enrollment in the study.

METHOD OF DATA COLLECTION

- All antenatal cases from 14- 28 weeks of gestation attending the outpatient department of OBG after obtaining informed consent. Those who were willing to participate and met the selection criteria were included.

- All the women were interrogated about the detailed medical history, obstetric history, menstrual history and past history. General physical examination, abdominal examination was done.

- Gestation age was calculated according to LMP and correlated with USG gestational age. Fasting venous blood samples of the women was sent to biochemistry lab for estimation of lipid profile.

- The parameters estimated in lipid profile include total cholesterol, triglycerides, LDL, VLDL and HDL. The medical records of the patients were studied and detailed data collection was done regarding the blood pressure, DIPSI levels and other relevant data during the second trimester.

- All the patients were followed up at the time of delivery and detailed examination was done and categorized mainly into preeclampsia, gestational diabetes mellitus and preterm delivery.

The Friedewald formula is commonly used to compute LDL-cholesterol in lipid profiles.

Friedewald formula:

LDL cholesterol = Total cholesterol – (very low-density lipoprotein (VLDL) cholesterol - HDL cholesterol)

• The Friedewald formula is used to calculate lipid values in a fasting condition.

• Total and HDL cholesterol levels are directly assessed.

• To calculate VLDL cholesterol, divide the measured total triglyceride level by 5.

Sources of error involved in the estimation of LDL cholesterol using the Friedewald formula include:

- LDL cholesterol levels must be assessed directly (direct LDL) by ultracentrifugal single spin analysis or immunoprecipitation approach in patients with more prono unced hypertriglyceridemia.
- The Friedewald method is only valid if the total triglyceride concentration is less t han 400 mg/dL (4.516 mmol/L).
- LDL cholesterol is underestimated by the method, especially at low levels(25 mg/ dL [0.6 mmol/L])
- The cholesterol content of atherogenic, intermediate-density lipoprotein (IDL) and VLDL remnants is underestimated when VLDL cholesterol is calculated from triglycerides.
- Other lipoproteins, such as lipoprotein (a) and lipoprotein- X are included in the predicted LDL cholesterol concentration.

SAMPLE SIZE:

The minimum sample size formula based on mean and standard deviation is

$$\frac{(Z\alpha + Z\beta)2\ (S_1{}^2 + S_2{}^2)}{(\mathrm{X}_1 - \mathrm{X}_2)^2}$$

Where z_α is related with the level of significance and z_β is related with the power of the test. For 5% level of the significance $z\alpha$ =1.96 and z_β =0.84 for 80% power of the test.

REF:

- X1 is the first group's mean (204.00), and X2 is the second group's mean (214.33).
- S1 is the first group's standard deviation (18.9), and s2 is the second group's standard deviation (18.64).
- The sample size produced with these values is 52.
- Ultimately the collected sample will be compared with respect to three groups as preeclampsia group, gestational diabetes mellitus and preterm deliveries.

  Hence if a single group of 160 cases is taken for the study there will be a sizable number in each group for a meaningful comparison.

RESULTS

The study was conducted at the Department of Obstetrics and Gynecology of KAHER's Dr.Prabhakar Kore Charitable Hospital, Belagavi, Karnataka for a period of one year and six months. This observational study was conducted among antenatal women in the second trimester and followed up till delivery from January 2020 to June 2020, after the study was approved by the Institute Ethics Committee.

Data obtained from structured questionnaires was analysed. Continuous quantitative variables have been given as mean ± SD (minimum, maximum). Categorical data have been expressed in terms of frequencies and percentages and have been compared using Chi-square test. P value<0.05 was considered as significant in all cases.

Recruitment of study participants

The total number of mothers screened and enrolled for this study was 175. Details of inclusion of study participants have been given in Figure 5. The age of the participants ranged from 20 years to 40 years with mean age 25.26 ± 3.77 years.

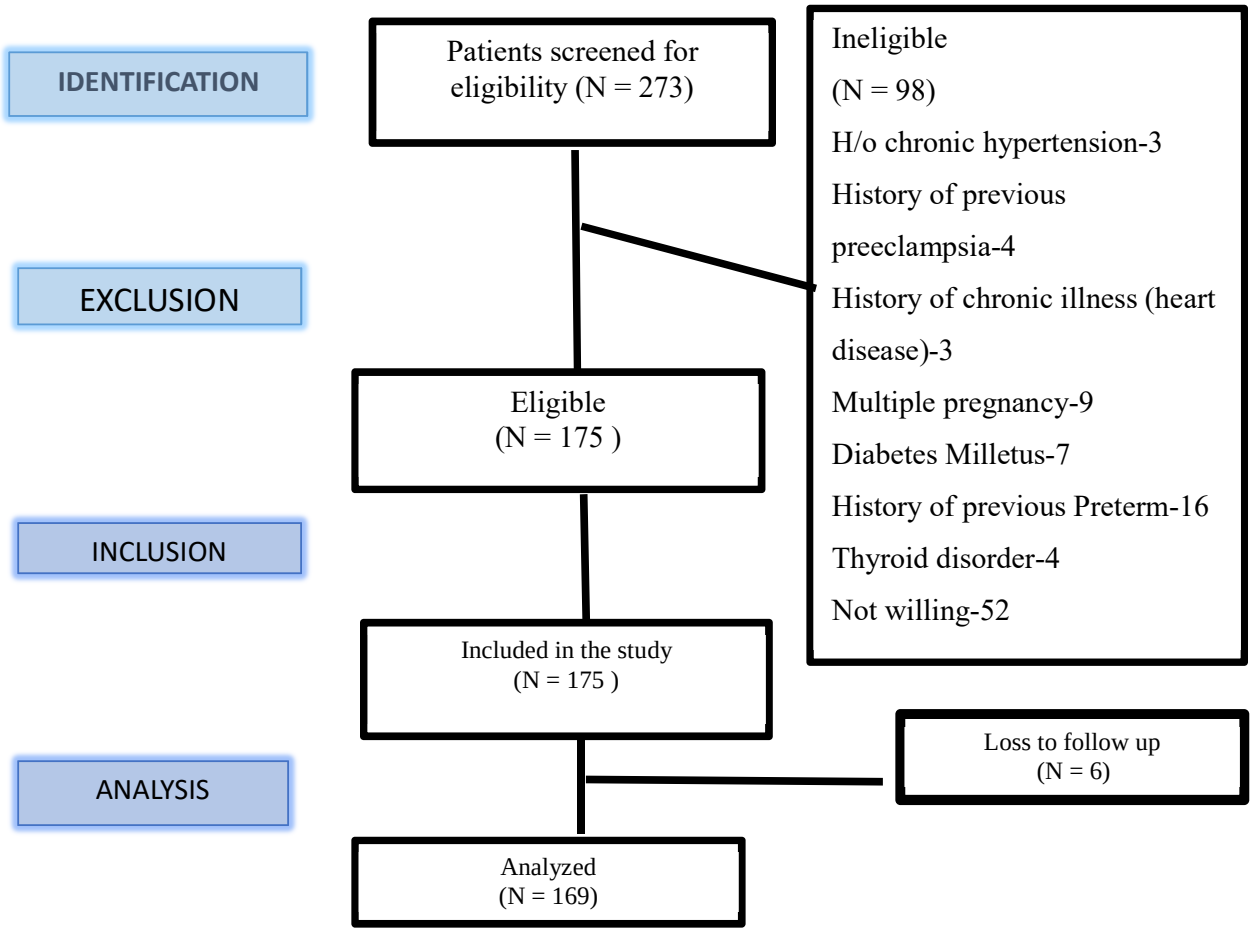


Figure 5: Enrollment of participants for the study

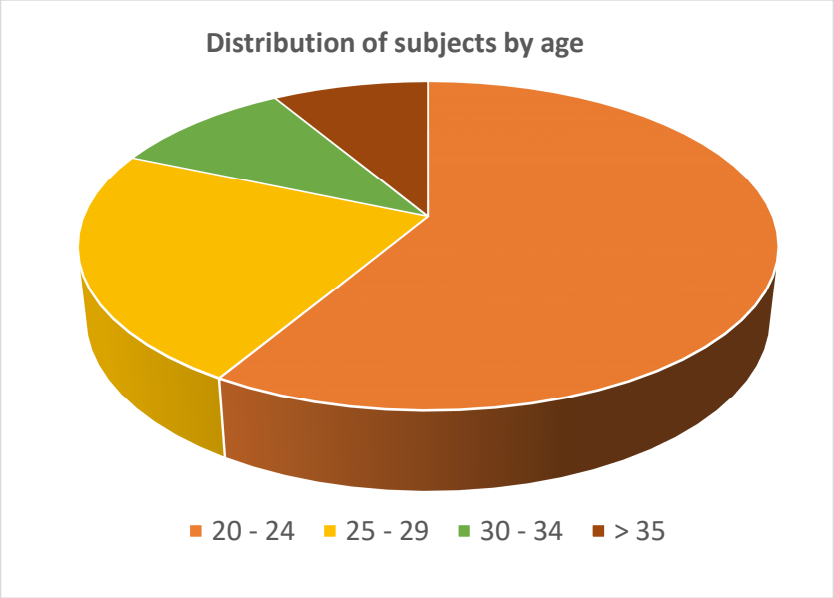
DISTRIBUTION OF PARTICIPANTS BY AGE:

TABLE 3: Distribution of participants according to age

AGE	NUMBER	PERCENTAGE (%)
20 - 24	83	47.42
25 - 29	66	37.73
30 - 34	21	12
> 35	5	2.85
TOTAL	175	100.00

	MEAN	S.D.	MINIMUM	MAXIMUM
AGE	**25.26**	**3.77**	**20**	**40**

FIGURE 6: Distribution of Participants According to Age



In my study, 175 low risk antenatal mother were included out of which 83 patients were under the age of 21-25 years, 66 patients under the age of 26-30 years, 21 patients under the age of 30-34 and more than 35 were 5. The maximum age is 40, while the minimum is 20.

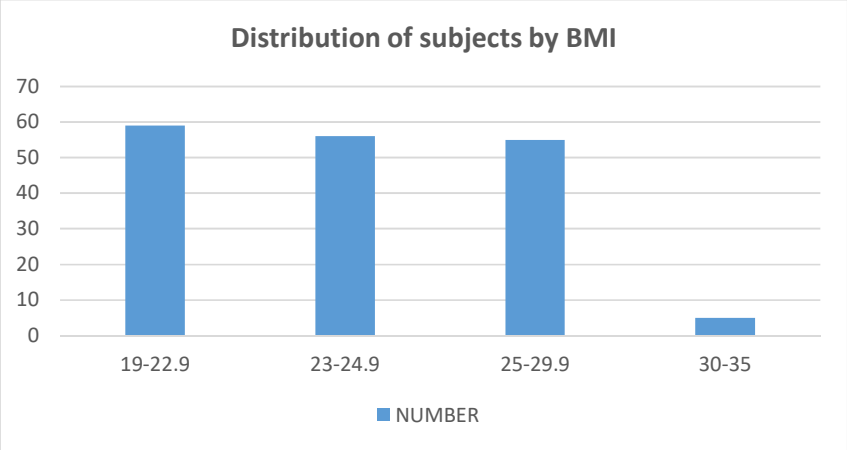
DISTRIBUTION OF SUBJECTS BY BMI:

Table 4: Distribution according to body mass index (BMI)

BMI	NUMBER	PERCENTAGE
19-22.9	59	33.71%
23- 24.9	56	32%
25 -29.9	55	31.44%
30 - 35	5	2.85%
TOTAL	175	100.00%

	MEAN	S.D.	MINIMUM	MAXIMUM
BMI	24.54	2.38	20	33

Table 7: Distribution according to body mass index (BMI)



Overweight is defined as a BMI of 23 to 24.9 kg/m2, while obesity is defined as a BMI of >25 kg/m2, according to WHO and NIH recommendations for Asian people. Underweight is defined as a body mass index of less than 18.5 kg/m2.Normal BMI is defined as a body mass index of 18.5–23 kg/m2.175 women included in this study were classified into underweight, normal, overweight and obese patients.59 people were in normal BMI, 56 people were overweight, and 60 people were obese

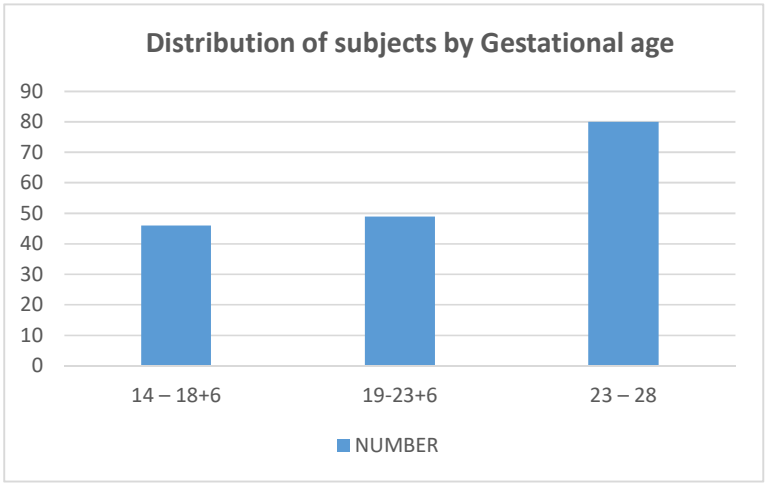
DISTRIBUTION OF SUBJECTS BY GESTATIONAL AGE

TABLE 5: Distribution of Participants According to Gestational Age

GESTATIONAL AGE (weeks)	NUMBER	PERCENTAGE (%)
14-18^{+6}	46	26.2
19-23^{+6}	49	28
24-28	80	45.7
TOTAL	175	100

	MEAN	S.D.	MINIMUM	MAXIMUM
GESTATIONAL AGE	21.50	4.08	14	27

Figure 8: Distribution of participants According to Gestational Age



The above table shows the distribution of participants according to gestational age. Among 175 participants, maximum number of them i.e., 80 participants belonged to the GA between 24 to 27+6weeks amounting upto 45.7%, among the rest, 49 participants belonged to 19 to 23+6weeks accounting to 28% and the rest 46 participants was seen between 14 to 18+6weeks accounting to 26.2%.

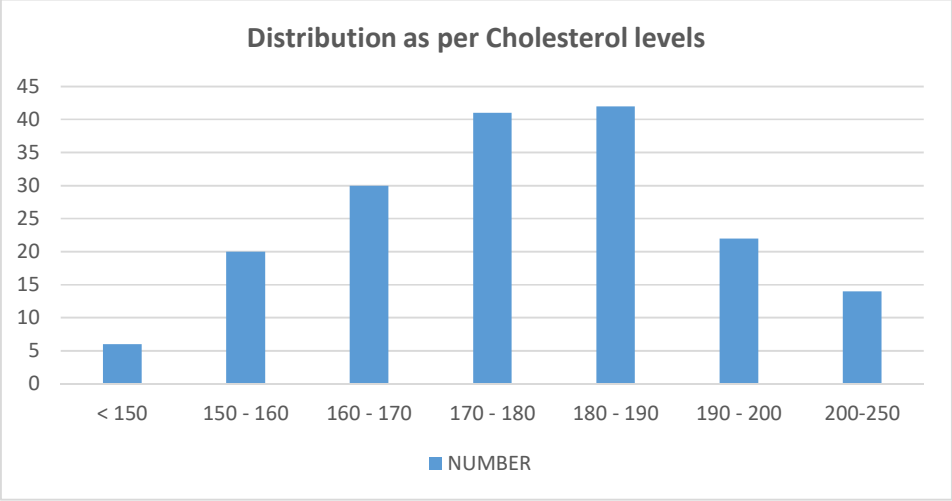
CHOLESTEROL LEVELS

TABLE 6: Distribution as per cholesterol levels

CHOLESTEROL	NUMBER	PERCENTAGE
< 150	6	3.42%
150 - 160	20	11.42%
160 - 170	30	17.14%
170 - 180	41	23.42%
180 - 190	42	24%
190 - 200	22	12.57%
200-250	14	8%
TOTAL	175	100.00%

	MEAN	S.D.	MINIMUM	MAXIMUM
CHOLESTEROL	178.09	17.94	108	221

FIGURE 9: Distribution of Participants as per cholesterol levels



In my study with 175 study population the lipid profile was analysed of which the minimum cholesterol value is 108mg/dl, mean is 178mg/dl and maximum is 221mg/dl. Normal range of cholesterol 150-250mg/dl.41 people had cholesterol levels between 170-180mg/dl which accounts for 23.42%.

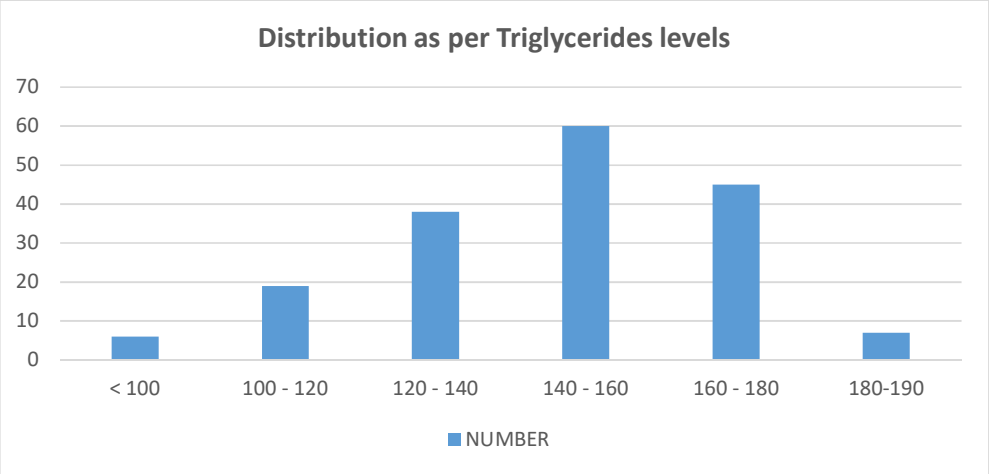
DISTRIBUTION AS PER TRIGLYCERIDES LEVELS

TABLE 7: Distribution according to triglyceride levels

TRIGLYCERIDES	NUMBER	PERCENTAGE
< 100	6	3.42
100 - 120	19	10.85
120 - 140	38	21.71
140 - 160	60	34.28
160 - 180	45	25.71
≥ 180	7	4
TOTAL	175	100.00

	MEAN	S.D.	MINIMUM	MAXIMUM
TRIGLYCERIDES	145.51	22.70	90	189

FIGURE 10: Distribution according to triglyceride levels



In my study with 175 study population the lipid profile was analysed of which the minimum triglycerides value is 90mg/dl, mean is 145.5mg/dl, and maximum is 189mg/dl. Normal range of triglycerides 80-200mg/dl.60people had cholesterol levels between 140-160mg/dl which accounts to 34.28%

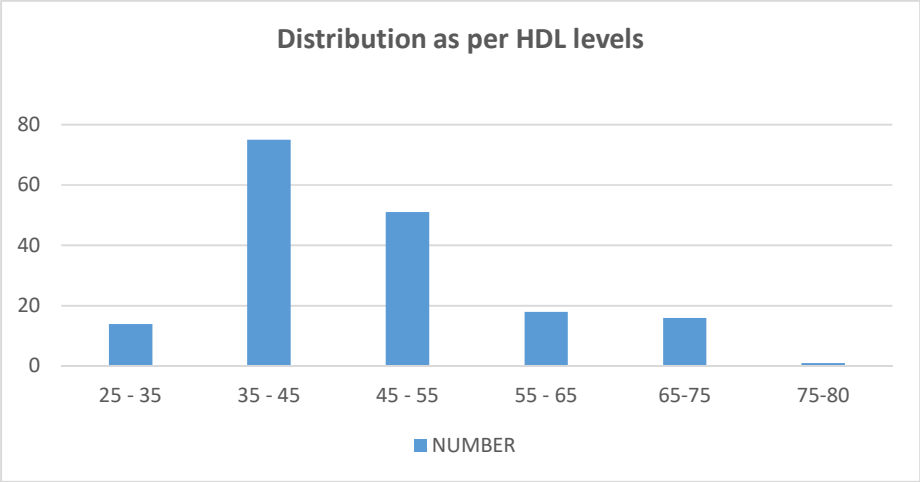
DISTRIBUTION AS PER HDL LEVELS

TABLE 8: Distribution as per HDL levels

HDL	NUMBER	PERCENTAGE
25 – 35	14	8.75
35 – 45	75	25.00
45 – 55	51	38.13
55 – 65	18	15.00
65 -75	16	11.25
<75	1	1.88
TOTAL	160	100.00

	MEAN	S.D.	MINIMUM	MAXIMUM
HDL	45.64	10.57	25	78

FIGURE 11: Distribution as per HDL levels



In my study with 175 study population the lipid profile was analysed of which the minimum HDLvalue is 25mg/dl, mean is 45.64mg/dl and maximum is 78mg/dl. Normal range of HDL 30-75mg/dl.51 people had HDL levels between 45-55mg/dl amounting to 38.13%.

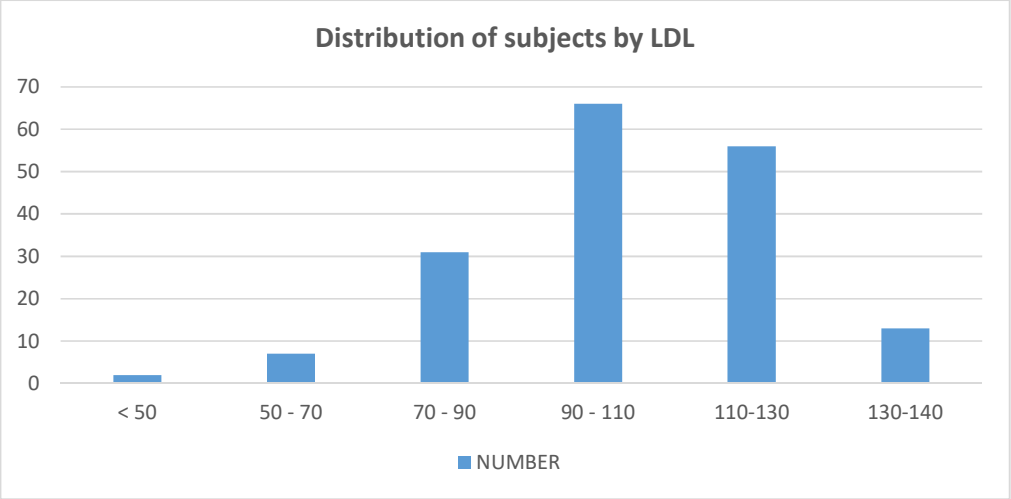
DISTRIBUTION OF SUBJECTS BY LDL

TABLE 9: Distribution according to LDL levels

LDL	NUMBER	PERCENTAGE
< 50	2	1.14%
50 – 70	7	4%
70 – 90	31	17.71%
90 – 110	66	37.71%
110 - 130	56	32%
≥ 130	13	7.42%
TOTAL	160	100.00%

	MEAN	S.D.	MINIMUM	MAXIMUM
LDL	103.35	19.07	39.6	139.4

FIGURE 12: Distribution according to LDL levels



In my study with 175 study population, lipid profile was analysed of which the minimum LDLvalue is 39.6mg/dl, mean is 103.35mg/dl, and maximum is 139.4mg/dl. Normal range of LDL 60-160mg/dl.66 people had LDL levels between 90-110mg/dl which is 37.71%.

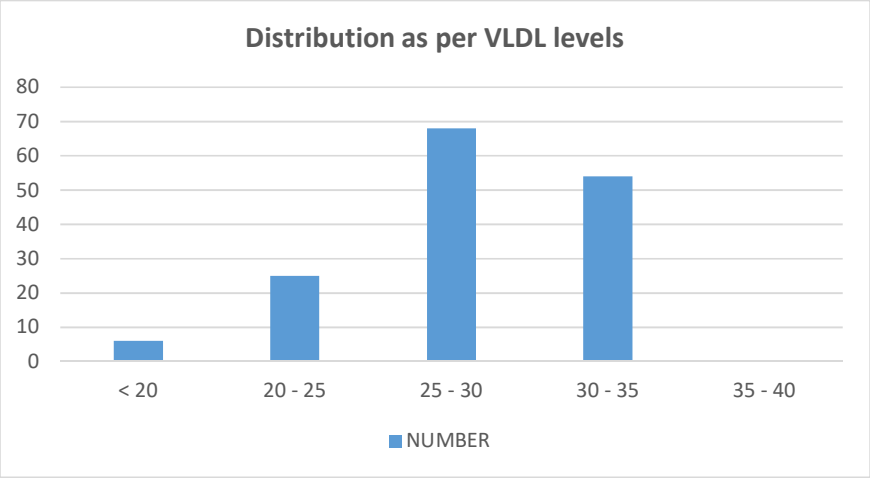
DISTRIBUTION AS PER VLDL LEVELS

TABLE 10: Distribution as per VLDL levels

VLDL	NUMBER	PERCENTAGE
< 20	6	3.42
20 - 25	25	14.28
25 - 30	68	38.85
30 - 35	54	30.85
≥ 35	22	12.57
TOTAL	175	100.00

	MEAN	S.D.	MINIMUM	MAXIMUM
VLDL	29.10	4.54	18	37.8

TABLE 13: Distribution as per VLDL levels



In my study with 175 study population the lipid profile was analysed of which the minimum VLDLvalue is 18mg/dl, mean is 29.10mg/dl and maximum is 37.8mg/dl.VLDL ranges from 0 to 40mg/dl.68 people had VLDL levels between 25-30mg/dl which accounts to 38.85%.

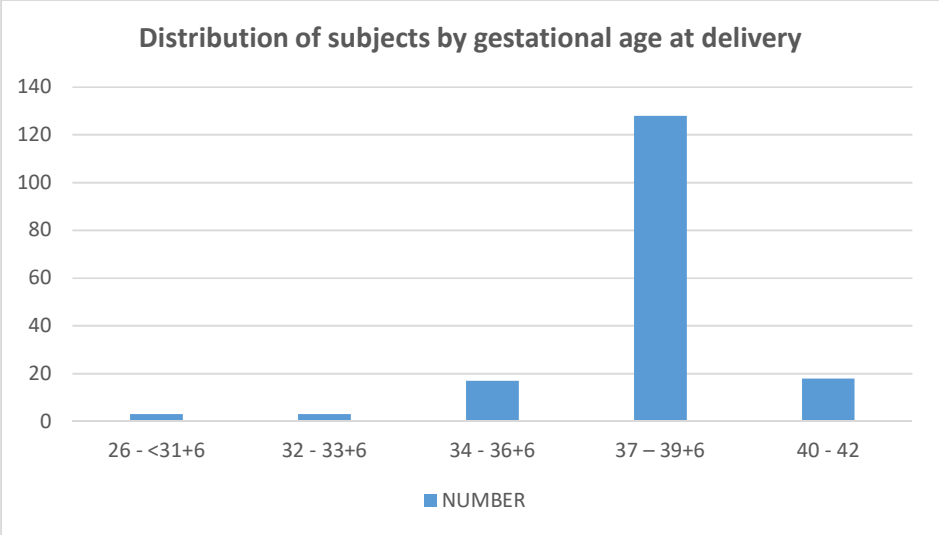
GESTATIONAL AGE AT DELIVERY

TABLE 11: Distribution based on gestational age

DELIVERED AT GESTATIONAL AGE(WEEKS)	NUMBER	PERCENTAGE
26 – 31^{+6}	3	1.77
32 – 33^{+6}	3	1.77
34 – 36^{+6}	17	10.05
37 – 39^{+6}	128	73.14
40 – 42	18	10.65
TOTAL	169	100.00

	MEAN	S.D.	MINIMUM	MAXIMUM
DELIVERED AT GESTATIONAL AGE	38.33	2.11	26	41

FIGURE 14: Distribution based on gestational age



In my study with 175 study population, 128 had a term pregnancy which corresponds to 73.14% and 18 women had a postterm pregnancy.3 participants had a early preterm pregnancy, 17 women had a later preterm pregnancy.

DISTRIBUTION OF SUBJECTS BY BIRTH WEIGHT

Table 12: Classification of study population based on birth weight of neonates

BIRTH WEIGHT	NUMBER	PERCENTAGE (%)
1.5- 2.4	21	12.83
2.5- 3.5	128	75.73
3.6- 4	17	10.05
TOTAL	169	100.00

	MEAN	S.D.	MINIMUM	MAXIMUM
BIRTH WEIGHT	2.76	0.52	1.6	4.1

In my study, 128(75.73%) patients delivered appropriate for gestational age babies (2.5-3.5kgs), 127patients (10.05%) delivered large for gestational age babies (>3.5kgs) and 21 patients (12.3%) delivered small for gestational age babies (<2.5kgs).

PREGNANCY OUTCOMES

Table 13: Maternal complications among study participants

COMPLICATION	NUMBER	PERCENTAGE
GESTATIONAL HYPERTENSION	8	5%
SEVERE PREECLAMPSIA	5	3.1%
PREECLAMPSIA	5	3.1%
GDM	9	5.63%
PRETERM	11	6.88%

Out of 175 participants who were enrolled and met the selection criteria,6 was lost to follow up.5% developed gestational hypertension, 6.1% developed preeclampsia, 9 people developed GDM and 11 people had preterm delivery.

CORRELATION OF LIPID PROFILE WITH PREECLAMPSIA

Table 14: Correlation of lipid profile with preeclampsia

	Preeclampsia									
	NO				YES					
	MEAN	S.D.	MINIMUM	MAXIMUM	MEAN	S.D.	MINIMUM	MAXIMUM	p VALUE	INFERENCE
Cholesterol	177.06	18.29	108	221	181.00	12.24	159	196	0.5044	NS
Triglycerides	145.82	23.27	90	189	143.80	16.14	114	167	0.7877	NS
HDL	46.26	10.72	25	78	40.60	7.56	27	52	0.1039	NS
LDL	101.64	19.20	39.6	139.4	111.64	12.11	93.4	131	0.1072	VS
VLDL	29.16	4.65	18	37.8	28.76	3.23	22.8	33.4	0.7877	NS
Baby Weight (Kg)	2.80	0.48	1.1	4.1	2.24	0.93	0.77	3.4	0.0020	VS
Delivered at Gestational age	38.29	1.62	32	41	35.40	5.08	26	40	< 0.0001	HS

Lipid parameters were compared between Preeclampsia and the control group. There was no difference in Cholesterol, Triglycerides, HDL, LDL and VLDL between the two groups. However, most of the preeclampsia women had a preterm delivery when compared to control group who had a term delivery which was statistically significant.

CORRELATION OF LIPID PROFILE WITH GDM

Table 15: Association of lipid profile with GDM

	GDM									
	NO				YES					
	MEAN	S.D.	MINIMUM	MAXIMUM	MEAN	S.D.	MINIMUM	MAXIMUM	p VALUE	INFERENCE
Cholesterol	177.72	17.80	108	221	185.00	21.47	153	220	0.2416	NS
Triglycerides	144.39	22.13	90	188	170.11	15.73	144	189	0.0008	HS
HDL	45.61	10.64	25	78	49.78	11.42	36	67	0.2577	NS
LDL	103.24	19.19	39.6	139.4	101.20	20.69	62.8	126.8	0.7586	NS
VLDL	28.88	4.43	18	37.6	34.02	3.15	28.8	37.8	0.0008	VS
Baby Weight (Kg)	2.76	0.53	0.77	4.1	2.79	0.32	2.4	3.2	0.8930	NS
Delivered at Gestational age	38.32	2.13	26	41	38.51	1.85	34.57	40.29	0.7916	HS

Out of 169 pregnant women,9 women developed GDM.Triglycerides and VLDL cholesterol in GDM patients were elevated i.e., 170mg/dl and 34.02mg/dl respectively than the control group which had 144.39mg/dl and 28.8mg/dl respectively.However there is no difference in Cholesterol, HDL, LDL between the two groups.

CORRELATION OF FACTORS OF LIPID PROFILE WITH PRETERM

Table 16: Correlation of lipid profile with preterm births

	PRETERM									
	NO				YES					
	MEAN	S.D.	MINIMUM	MAXIMUM	MEAN	S.D.	MINIMUM	MAXIMUM	p VALUE	INFERENCE
Cholesterol	178.43	17.09	116	221	174.45	28.54	108	207	0.4826	NS
Triglycerides	146.01	22.93	90	189	144.45	18.52	112	178	0.8270	NS
HDL	45.78	10.77	25	78	46.82	10.06	32	62	0.7567	NS
LDL	103.46	18.28	43	139.4	98.75	29.72	39.6	136.2	0.4351	NS
VLDL	29.20	4.59	18	37.8	28.89	3.70	22.4	35.6	0.8270	NS
Baby Weight (Kg)	2.79	0.47	1.1	4.1	2.39	0.90	0.77	3.4	-	-

Out of 169 pregnant women, 11 women had a preterm birth. There was no difference in lipid parameters between the two groups. There was no difference in the cholesterol, triglycerides, HDL, LDL and VLDL between the two groups.

KARL PEARSON'S CORRELATION COEFFICIENT BETWEEN BMI AND LIPID PROFILE

Table 17: Correlation coefficient between BMI and lipid profile

	r	p VALUE	INFERENCE
Cholesterol	0.0825	0.3012	NS
Triglycerides	0.0246	0.7583	NS
HDL	0.0272	0.7333	NS
LDL	0.0568	0.4774	NS
VLDL	0.0246	0.7583	NS

The Karl Pearson formula was used to calculate the correlation coefficient between BMI and lipid profile, which was found to be non-significant.

MEAN COMPARISON OF CHOLESTEROL, TRIGLYCERIDES, HDL, LDL AND VLDL WITH BMI CATEGORIES

Table 18: Association between various lipid parameters with BMI.

	MEAN BMI				
	NORMAL	**OVER WEIGHT**	**OBESE**	**p VALUE**	**INFERENCE**
Cholesterol	177.30	177.55	178.76	0.9021	NS
Triglycerides	145.23	146.09	145.01	0.9644	NS
HDL	45.43	46.11	45.50	0.9387	NS
LDL	102.82	102.22	104.25	0.8286	NS
VLDL	29.05	29.22	29.00	0.9642	NS

The lipid parameters of normal, overweight and obese persons were all the same.

DISCUSSION

Hyperlipidemia is more common among pregnant women. If the metabolic alterations are within normal limits, there will be no adverse effects during pregnancy. Problems arises if there is dyslipidemia surpassing the normal levels in pregnancy. Hyperlipidemia causes atherosis in the uteroplacental spiral arteries which results in decreased blood flow to the baby resulting in preeclampsia. Insulin resistance is increased during pregnancy. Insulin resistance will be even higher in dyslipidemia, especially in patients with a high BMI leading to gestational diabetes mellitus. They may develop type 2 diabetes later in life.

Patients with dyslipidemia are known to have preterm birth and perinatal com plications such as small for gestational age newborns, stillbirth.[45] A total of 175 singleton uncomplicated antenatal cases attending the outpatient department of Obstetrics and Gynaecology at "KLE'S Dr. Prabhakar Kore charitable hospital" attached to KAHER'S JNMC, Belagavi were included in the present study after meeting the selection criteria. This study comprised of 175 low-risk antenatal women. In 14 to 28 weeks of pregnancy, a fasting lipid profile was performed following an overnight fasting. These patients were examined for maternal complications at birth.

Maternal diseases such as chronic illness like cardiovascular diseases, hypothyroidism, type 2 Diabetes mellitus, hypertensive disorders, multiple pregnancy, women with treatment on lipid lowering drugs, cervical incompetence, previous history of preterm and patients not willing to participate were excluded.

Dyslipidemia is a major risk factor for coronary heart disease progression. In both men and women, age is a significant predictor of CVD risk. Because postmenopausal women's LDL-C levels were lower after treatment with oral

oestrogen. By acting on LDL receptors, high oestrogen levels in younger women have a favourable effect in decreasing LDL-C.[68]

Deepti G.I. et al discovered that there is increase in total cholesterol, triglyceride, LDL-Cholesterol and decrease in HDL-Cholesterol in perimenopausal women i.e., 35-50 years as compared to women in the younger age group as the estrogen levels decreases.[60]

The mean age of the pregnant women in our study was 25.26 ± 3.77 years. Majority of the subjects i.e., 83 participants were in the age group of 20-24 years which is 47.42% and only 5 patients were above the age group of 35.The mean triglycerides, cholesterol, LDL cholesterol are 170.2,190.8 and 104.96 respectively which is greater than the younger age group.

The metabolic changes that occur during pregnancy are important.The concentrations of lipids, lipoprotein and apolipoproteins in the blood increase during pregnancy. Maternal hormonal alterations (increased insulin, progesterone, 17estradiol, and Human Placental Lactogen) impact lipid levels. Other maternal factors that affect lipid metabolism and plasma levels include BMI (body mass index), maternal weight gain, maternal diet, prepregnancy lipid levels, and numerous medical problems of pregnancy.[46]

33.7 percent of the 175 patients in the present study had a normal BMI. Only 2.85 percent of women had a BMI greater than 30, while 32 percent of women were overweight. A total of 55 persons out of 175 had a BMI of 25 to 29.9.In our study there was no impact of increasing BMI on the lipid parameters which was studied using the Karl Pearson's correlation which was not significant

Lior Shamai conducted studies to determine if there was a link between BMI and lipid profile. Higher BMI was found to be inversely related to HDL and directly related to TG. The relationship between BMI and LDL was not found to be significant. Although insulin resistance may explain the link between BMI and both HDL and TG, the lack of a meaningful association between BMI and LDL is not clear.[47]So this could not explain the association of BMI and lipid parameters.

Amit D. Sonagra et al. in South India studied the lipid profile of pregnant women in each trimester and compared the results to non-pregnant healthy women of the same age group. The above study's mean lipid profile value for 2nd trimester women is similar to our study. TC-184mg/dl, TG-160mg/dl, HDL-41mg/dl and LDL-105mg/dl are the average values from the above study. Their study's mean values are roughly correlated with our study's mean values. Total cholesterol was 178 mg/dl, triglycerides were 145 mg/dl, HDL was 45 mg/d and LDL was 103 mg/dl in our study.[18]

Preeclampsia is caused by high plasma lipid levels, particularly triglyceride levels which promote endothelial dysfunction. As lipid levels rises, lipid peroxidation rises resulting in an increase in oxidative stress. Increased amounts of lipid peroxide damage the endothelium resulting in placental endothelial dysfunction, which is linked to maternal complications like preeclampsia and fetal complications like IUGR.[7]

Enquobahrie DA et al studied in their study that women who subsequently developed preeclampsia had 10.4%, 13.6%, and 15.5% higher concentrations of LDL cholesterol, triglycerides and LDL/HDL ratios respectively than the control subjects.[42]

In our study there was increase in mean cholesterol 181 ± 12.24mg/dl in the preeclampsia compared to 177.06 ± 18.29 mg/dl in the control group which was not

significant. There was a decrease in mean HDL cholesterol 40.60 ± 7.56 mg/dl compared to 46.26 ± 10.72 mg/dl in the control group there is no correlation between triglyceride levels done in the second trimester with the preeclampsia. Many of the preeclampsia patients in our study delivered late preterm with mean age of 35.40 ± 5.08 weeks .However only 9 people developed preeclampsia, so statistical difference could not be made out.

A case control study was undertaken in Iowa City by KK Ryckman and colleagues. They separated the study participants into two groups: pregnant women with GDM and without GDM. They discovered that triglyceride levels were significantly elevated in women with GDM compared with those without GDM (weighted mean difference (WMD) 30.9, 95% confidence interval [95% CI] 25.4-36.4) throughout the pregnancy. HDL-C levels were significantly lower in women with GDM compared with those without GDM in the second (WMD -4.6, 95% CI -6.2 to -3.1) and third (WMD -4.1, 95% CI -6.5 to -1.7) trimesters of pregnancy. There were no differences in aggregate total cholesterol or LDL-C levels throughout pregnancy in pregnant women with GDM and without GDM signalling that dyslipidemia may play a role in the pathogenesis of GDM.[11]

In our study,9 of the 175 pregnant women developed Gestational Diabetes Mellitus. At 14-28 weeks, all of the women in our study were screened for GDM and were excluded from the study. Glucose intolerance diagnosed first time in pregnancy is called as gestational diabetes mellitus.

Our results correlate with the study done by Wiznitzer et al where they have concluded high triglyceride levels were associated with the development of gestational diabetes mellitus[63]. In our study, GDM patients had a higher level of triglycerides and VLDL i.e., 170±15.73 mg/dl and 34.02 ± 3.14 mg/dl respectively

than the control group which had 144.39±22.13mg/dl and 28.8±4.43mg/dl with a p value of 0.0008 which is statistically significant.When compared to control group there was no correlation between cholesterol, HDL, LDL in GDM patients.

To summarise, findings done by Tanja GM imply that high maternal triglyceride levels in the first trimester of pregnancy are a contributor to the expression of PIH, preeclampsia, induced preterm birth and LGA offspring. Although we observed no link between TG levels and total preterm birth, they found a link between induced PTB and the mother's TG levels. In the last trimester of pregnancy, a prominent lipid profile (containing TC and TG levels) is linked to poor pregnancy outcomes. Increased lipid levels assessed in late pregnancy have been shown to increase the risk of PIH (not worsened by proteinuria) and preeclampsia.[7]

According to metanalysis of 292 studies and 97,880 women, an altered lipid profile particularly high triglycerides can lead to premature delivery and an increased risk of fetal compromise. The risk of developing thrombosis, placental infarction and utero placental insufficiency in individuals with abnormal lipid values and superadded hyper coagulable condition of pregnancy will impact the fetus resulting in preterm delivery and small for gestational age newborns.[17]

Out of 175 pregnant women, 6 were lost to follow up, 11 had deliveries before 37 weeks, and 158 had deliveries after 37 weeks in our study. There was no difference in the cholesterol, triglycerides, HDL, LDL and VLDL parameters between the preterm delivered patients and control group.

In a study conducted in Saudi Arabia, IA Siddiqui et al found that while triglycerides are raised in preeclampsia and gestational diabetes mellitus. Total cholesterol, HDL and LDL did not differ significantly between pregnant and non-pregnant women.[49]

Niromanesh, et al. compared the outcomes of 45 pregnant women with high TG levels (> 195 mg/dl) to 135 pregnant women with TG levels less than 195 mg/dl in a study. The incidence of GDM, preterm birth, preeclampsia and uterine artery pulsatility index were all significant outcomes. Eight women had preeclampsia with high TG levels (17.8% vs. 3.7 percent in the control group, p= 0.004), while 11 women had preterm birth (24.4 percent vs. 5.9% in the control group, OR 5.1, 95 percent CI 1.9 – 13.8, p= 0.0001).The risk of GDM was considerably higher in the high triglyceride group than in the control group. They came to the conclusion that hypertriglyceridemia is linked to preeclampsia, premature birth and gestational diabetes mellitus.

From the present study, we cannot predict the occurrence of preeclampsia, GDM and preterm delivery based on cholesterol levels of the second trimester as the lower bound value is within the normal range. However, we can conclude that cholesterol levels of more than 181.0 ±12.24mg/dl and 185.0 ± 21.47mg/dl during the second trimester can lead to complications like preeclampsia and GDM.

From the present study, we cannot predict the occurrence of preeclampsia, GDM and preterm delivery based on LDL-C, VLDL-C, HDL-C levels of the second trimester as the lower bound value and mean are within normal range.

According to Babita G Gidke et al study concluded that the total cholesterol, HDL, LDL and VLDL levels do not indicate the risk of preeclampsia, gestational diabetes or preterm birth.[42]

In our study, only ten patients (out of 175 total) developed preeclampsia, while nine people developed Gestational Diabetes Mellitus and 11 of them had a preterm delivery. To prove the importance of triglycerides in preeclampsia, GDM and preterm birth a large sample size is required.

Limitation of the study includes sample size is limited which was 175 in out study, fasting lipid profile was taken in the second trimester and it was a single measurement throughout the pregnancy .We also lack the diet history of the patient which might affect lipid profile during the course of pregnancy. The fasting hours, lifestyle and clinical characteristics were self-reported which may have led to bias for some measures. It cannot be generalized as it was done in Asian ethnicity and in a tertiary care hospital in Belagavi.

CONCLUSION

In conclusion, serum triglycerides and VLDL were statistically significantly higher in Gestational Diabetes Mellitus women compared to the control group in a study population of 175 pregnant women. However, there was no difference between the GDM and control groups in terms of cholesterol, LDL or HDL. A huge group, however is required.

There was no difference in lipid markers between preterm and full term deliveries. In the present study, preeclampsia patients delivered at a much earlier gestational age than the normotensive patients and there was no difference in the lipid parameters between the two groups. In patients with dyslipidemia, postpartum follow-up is required to prevent future consequences such as atherosclerosis and heart disease.

As a result, recognising and treating dyslipidemia prior to pregnancy might help to avoid adverse pregnancy outcomes. Specific lifestyle changes such as low fat diet, avoidance of simple sugars, avoidance of excessive glucocorticoids or retinoic acid (contraindicated in pregnancy), avoidance of alcohol, increased exercise and weight loss are required in women of reproductive age to lower lipid levels, which can help to prevent hypertensive problems during pregnancy, GDM and preterm birth.

SUMMARY

The current study was an observational study that was conducted for a period of 1 and a half year from January 2020 to June 2021 at "KLE's Dr Prabhakar Kore Charitable Hospital and Medical Research centre" attached to KAHER's JNMC, Belagavi.

The objective of this study was to find out any association of serum lipid levels during the second trimester with the development of pregnancy-associated complications such as preeclampsia, GDM and preterm delivery.

A total of 175 singleton, uncomplicated antenatal women whose pregnancies were dated according to ACOG guidelines from 14- 28 weeks period of gestation, attending the OPD after meeting the selection criteria and obtaining written informed consent were recruited for the study.

All the women were interrogated about the detailed medical history, obstetric history, menstrual history and past history. General physical examination and abdominal examination was done. Gestational age was calculated according to LMP and correlated with USG gestational age. Fasting venous blood samples of the women were sent to biochemistry lab for estimation of lipid profile.

The parameters estimated in lipid profile include total cholesterol, triglycerides, LDL, VLDL and HDL. The medical records of the patients and detailed data collection was done regarding the blood pressure, DIPSI levels and other relevant data was collected during the second trimester.

All the patients were followed up at the time of delivery and detailed examination was done and categorized mainly into preeclampsia, gestational diabetes mellitus and preterm delivery.

The mean age of the pregnant women in our study was 25.26 ± 3.77 years. Majority of the subjects i.e., 80 out of 175, belonged to the gestational age between 24 to 28 weeks accounting to 45.7%, followed by 49 subjects between 19 to 23 weeks accounting to 28%.

There was a significant correlation between birth weights in the preeclampsia and control groups, with preeclampsia patients delivering at a mean gestational age of 35 weeks compared to 38.29 weeks in the control group. When lipid levels rise, oxidative stress rises, damaging the endothelium and leading to placental malfunction and induced premature birth. However, there was no correlation between the lipid parameters and preeclampsia.

In our study, GDM patients had a higher level of triglycerides and VLDL i.e., 170mg/dl and 34.02mg/dl respectively, than the control group which had 144.39mg/dl and 28.8mg/dl with a p value of 0.0008 which was statistically significant.

There was no association seen between lipid profile and preterm delivery.

The correlation coefficient between BMI and lipid profile was calculated by Karl Pearson and found to be non-significant.

There was no difference in lipid parameters between normal, overweight and obese pregnant women.

In the second and third trimesters, maternal metabolic changes occur resulting in glucose sparing and a rise in serum fatty acids which increases the risk of gestational diabetes mellitus, hypertension and preterm delivery.

Because the lipid profile was only performed in the second trimester in our study, the consequences of lipid abnormalities could not be thoroughly assessed. Triglycerides increase in GDM patients, according to our research. To notice the

difference in lipid profile and know the outcome, a large number of people are needed.

Strength of the study:

- Lipid profile was tested in the second trimester of pregnancy. So, dyslipidemia can be a useful early predictor of preeclampsia risk. This may help in developing effective early preventive or therapeutic measures.
- It is a prospective study.

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ANNEXURE I: ETHICAL CLEARANCE

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed - to-be- University)
Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (GoI)
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
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Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/ 214 **Date: 24/12/2019**

To,

REG. NO. BJ0119016

PG student in Obstetrics and Gynaecology,
J.N.Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"MATERNAL LIPID PROFILE DURING SECOND TRIMESTER ON PREGNANCY OUTCOMES AND ITS COMPLICATIONS – A ONE YEAR OBSERVATIONAL STUDY",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Anita Dalal)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Roopa M Bellad)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

ANNEXURE II – INFORMED CONSENT FORM.

Mrs _____ we are requesting you to enroll yourself in study titled "Maternal lipid profile during second trimester on Pregnancy outcomes and its complications -A one-year observational study" Conducted by **REG. NO. BJ0119016**, Post Graduate in M.S. Obstetrics and Gynaecology under the guidance of DR. _____ Department of Obstetrics and Gynaecology, J.N. Medical College, Belgaum under KAHER, Belagavi.

Objectives /purpose of study:

Respected Madam we request you to participate in our study as you are eligible for participating and your participation in this study is important as it helps in assessing the derangement in lipid profile and its association with preeclampsia, gestational diabetes mellitus, preterm delivery and other maternal complications

Your participation in research is voluntary. Your decision whether to participate in the study or not will not change present or future health care services offered to you and will not affect your relationship with J.N. Medical College. If you decide to participate you are free to withdraw at any time. All pregnant women meeting the inclusion criteria will be recruited in our study. The purpose of research study is to assess the lipid profile and to see the outcome of these women. I will be the investigator for our study. This study is not being funded.

Procedure Involved:

If you agree to enroll yourself in my study, you will be interviewed regarding your present, past and family history, then you will be clinically examined in detail and investigated which may or may not cause pain. The procedures don't cause any temporary or lasting problems to you. Your co-operation is necessary as the investigation may be repeated number of times as required.

Risks and Benefits:

There are no potential risks and discomforts associated with any procedure involved in our study. The benefits of taking part in this research is your participation being valuable contribution to medical research to improvise treatment currently practiced.

Alternative:

If I decide not to participate in the study, my health care provider will provide the usual standard care during my pregnancy, delivery and up to through 6 weeks after delivery**.**

Withdrawal from study:

You can withdraw at any time from the study. There will be no penalty for withdrawal. You can be removed from the study if necessary.

Privacy and Confidentiality:

The only people who will know that you are the research subject will be the members of the research team. No information about you or information provided by you during the research will be disclosed to others without your written permission except:

1. In emergency to protect your rights and welfare.
2. If required by law.

Institutional/sponsor's policy:

In the event of any injury related to the study, treatment will be made available through KLE's Hospital & MRC, Belagavi. There is no compensation or payment for such medical treatment by law. If you are injured you may contact **REG. NO. BJ0119016**, Post graduate student, Department of Obstetrics and Gynaecology, KLE's Hospital& MRC or by Ph. No: _____.

Financial Incentives for participation:

No financial incentives are being offered to enrolled patients. It is purely being done with the idea of research and all the cost of the study will be borne by the investigator. You will not be reimbursed for any expenses for participation in this research.

Contact details:

In case you have any questions related to the study, in future or in case of study related injury or illness, you can contact **REG. NO. BJ0119016**, Post graduate student, Department of Obstetrics and Gynaecology, KLE's Hospital and MRC, Ph. No: _____ or Dr. _____, Dept. Of Obstetrics and Gynaecology, KLE's Hospital and MRC, Belgaum, Ph. No: _____.

If you have any queries about your rights as a study participant, you may contact Dr. Roopa M Bellad, Prof. of Paediatrics as Chairman of J. N. Medical College Institutional Ethics Committee on Human Subjects Research, Phone No.0831 2473777 ext-1527 at J. N. Medical College,
Belgaum.

Authorization to Publish Results:

When the results of the research are published or discussed in a conference, no information will be displayed that would disclose your identity. Any information that is obtained in connection with this study and that can be identified with you will remain confidential. Results of the study will be used to improve maternal and perinatal outcome.

Consent statement:

I, _____ voluntarily agree for participating in this study. By signing this consent form, I am not giving up any of my legal rights, I may withdraw from the study anytime. I am signing the consent form after having read or been read form in my own vernacular language, including the risks and the benefits and having all my questions answered.

Participant Name : _____

Signature or the Left Thumb Print of Participant : _____

Signature or the Left Thumb Print of Legally authorized person: _____

Investigators Name: _____ Signature: _____

Witness Name : _____ Signature: _____

Date: _____

ANNEXURE III - SCREENING FORM.

NUMBER: ☐☐☐☐☐☐

First name: _____ Middle name: _____ last name: _____ Age (years): ☐☐

IP number: ☐☐☐☐☐☐

Husband's name: _____

Address: _____ Phone

number: _____

Eligibility:

Yes-1
No-2

i) Hypertension detected before <20 weeks ☐

ii) Any chronic illness ☐

iii) Diabetes mellitus ☐

iv) Multiple pregnancy ☐

v) Women with treatment on lipid lowering drugs ☐

vi) Women who are less than 14 weeks of gestation ☐

vii) Women who are greater than 28weeks of gestation ☐

Is she eligible? If eligible, consent to be taken Enrollment done: ☐

If not enrolled, indicate reasons Patient is not willing: ☐

Patient withdraws from the study: ☐

Other reasons: ☐

ANNEXURE IV– PROFORMA

ENROLLENT NUMBER: ☐☐☐

First name: _____ Middle name: _____ last name: _____ Age (years): ☐☐

IP number: ☐☐☐☐☐☐

Husband's name: _____

Address: _____

Phone number: _____

Data collection instrument

1**. Socio-economic status** (according to modified B.J Prasad classification)

1 =upper class ☐

2=upper middle class ☐

3=middle class ☐

4=Lower middle class ☐

5=lowerclass ☐

Obstetric score: ☐

Gravida: ☐

Para: ☐

Living: ☐

Abortion: ☐

Stillbirth: ☐

Married life –

4. **Menstrual history:**

1. What was the last day of your menstrual cycle? ☐☐ - ☐☐ - ☐☐☐☐

   d d m m y y y y

2. Were your cycles regular? ☐

   1= yes , 2=no, 3=don't know

3. Estimated date of delivery (EDD) by LMP

   ☐☐ ☐☐ ☐☐☐☐

4. Corrected estimated date of delivery (EDD) if any

   ☐☐ - ☐☐ - ☐☐☐☐

5. Gestational age as per corrected EDD- ☐☐ weeks ☐☐ days **Past history:**

Family history:

General physical examination:

Weight (kg):

Pallor: Thyroid:

Edema: Breast:

Icterus: Spine:

Vitals:

Pulse rate (/min):

BP (/mm Hg):

RR (/min):

Systemic Examination:

CVS:

RS:

P/A: Fundal height:

FHR:

Investigations:

Blood group:

Hb:

PCV:

WBC:

RBC-

Platelet count-

Urine routine and microscopy:

HIV:

HbsAg:

VDRL:

TSH:

DIPSI:

Outcomes of Pregnancy:

Chief complaints:

Any maternal complication during pregnancy/delivery:

Result:

General physical examination-

Weight:

Pallor:

Icterus:

Edema:

Pulse rate (/min):

BP (/mm Hg):

RR (/min):

Systemic Examination:

CVS:

RS:

Investigations:

Hb:

PCV:

WBC:

RBC-

Platelet count-

Urine routine and microscopy:

DIPSI:

If preeclampsia:

TB: DB: SGOT: SGPT: TP: ALP: ALB: BIL: AG RATIO

UREA: CREATININE: URIC ACID: IF GDM:

RBS: HBA1C: DIPSI:

At what gestation age-

Any maternal complications such as

Pregnancy induced hypertension: ☐

Intrahepatic cholestasis of pregnancy: ☐

Preeclampsia: ☐

GDM: ☐

Child losses: ☐

Any other maternal complications: ☐

Date, Time & Details of Delivery	**Neonatal Details**
Date: ☐☐☐ Time: ☐ Spont: ☐ Induced: ☐ Term: ☐ Preterm: ☐ 1.Vaginal: Normal: Forceps ☐ Ventouse: Episiotomy: ☐ Others: ☐ 2.VBAC: Successful: Failed ☐ 3.LSCS Emergency ☐ Elective ☐ Indication: ☐ Tubectomy: Placental Wt: gms Blood Loss: ml	Weight: ☐ Kgs. Sex: Male Female ☐ Live Birth ☐ Apgar Score: 1[1] 5[1] ☐ NICU Admission Still Birth: FSB: MSB: ☐ Cong Anomalies ☐

Sl. No.	OPD. No.	Age	BMI	Gestational age	Cholesterol	Triglycerides	HDL	LDL	VLDL	Married life (yrs)	Obstetric Score	Delivered at Gestational age	Preeclampsia	GDM	PRETERM	Complication	Baby Weight (Kg)	FTD/LSCS	Indication
1	5693088	27	26	22^{+6}	174	131	36	111.8	26.2	6	G2P1L1	38^{+1}	No	No	No	NO	2.2	FTD	N/A
2	5691919	22	25	16^{+2}	159	116	30	105.8	23.2	2	PRIMI	40+2	No	No	No	No	Unknown	FTD	N/A
3	4010396	21	22	20^{+2}	164	142	39	96.6	28.4	5	G2P1L1	39+2	No	No	No	No	3	FTEMG LSCS	Unknown
4	5676951	19	23	16	182	135	47	108	27	1	PRIMI	40	Gest HTN	No	No	NO	3	FTD	N/A
5	5694354	28	27	22^{+5}	171	152	43	97.6	30.4	8	G2P1L1	39	No	No	No	No	2.7	FTEMG LSCS	Thick MSL
6	5694346	20	24	20	180	143	50	101.4	28.6	2	PRIMI	39^{+4}	No	No	No	NO	2.4	FTEMG LSCS	Oligo Hydraminos
7	5639015	24	23	21^{+1}	168	141	44	95.8	28.2	8	G3P2L2	39^{+4}	No	No	No	No	2.6	FTD	N/A
8	5694661	21	22	17^{+5}	180	172	39	106.6	34.4	1	PRIMI	39	No	No	No	No	3.2	FTD	N/A
9	5800423	26	24	14	174	114	40	111.2	22.8	10	G2P1L0	29+2	Severe	No	No	NO	800gm	FTEMG LSCS	Severe PE
10	5604392	27	24	21^{+5}	221	163	49	139.4	32.6	12	G3P2L2	38^{+4}	No	No	No	No	2.5	FTEMG LSCS	Previous 2 LSCS
11	5695667	24	24	27^{+5}	174	148	43	101.4	29.6	3	PRIMI	40+1	Severe	No	No	No	3.4	FTEMG LSCS	Thick MSL with Fetal Distress
12	4249936	21	24	20	196	165	32	131	33	1	PRIMI	26	Severe	No	No	No	770gm	PTD (FSP)	N/A
13	5696023	22	26	18^{+2}	208	135	49	132	27	1	G2A1	38+4	Gest HTN	No	No	NO	2.6	FTD	N/A
14	3375939	24	26	23^{+4}	176	116	42	110.8	23.2	4	G2P1L1	38	No	No	No	No	3	FTD	N/A
15	5695713	23	24	23^{+1}	182	142	51	102.6	28.4	3	PRIMI	39^{+4}	No	No	No	No	2.8	FTEMG LSCS	Thick MSL
16	5750376	26	25	22^{+4}	180	136	40	112.8	27.2	5	PRIMI	39^{+3}	No	No	No	No	2.7	FTEMG LSCS	Pathological Trace
17	3618106	23	26	19^{+6}	188	161	38	117.8	32.2	1	PRIMI								
18	5695592	26	22	22^{+2}	174	129	46	102.2	25.8	3	G2A1	38	No	No	No	No	2.7	FTD	
19	5696648	24	25	18	199	139	35	136.2	27.8	5	G2P1L1	37	No	No	No	No	3	FTD	
20	5813103	25	23	20^{+1}	116	114	42	51.2	22.8	1	PRIMI	38^{+3}	No	No	No	HbsAg Positive	2.3	FTEMG LSCS	Breach in Labor
21	5670628	22	28	22^{+4}	181	146	39	112.8	29.2	3	G2P1L1L	38+2	No	No	No	No	3.3	FTD	
22	5696791	20	20	22^{+3}	156	130	37	93	26	1	PRIMI	39	No	No	No	No	3	FTD	N/A
23	5699904	21	24	21^{+6}	172	92	30	123.6	18.4	2	PRIMI	39^{+4}	No	No	No	No	3.5	FTEMG LSCS	Thick MSL
24	5679748	24	22	14	168	107	30	116.6	21.4	7	G2P1L1	39	No	No	No	No	2.5	FTD	
25	3240259	22	26	20^{+5}	149	142	41	79.6	28.4	3	PRIMI	37	No	No	No	No	2.6	FTEMGLSCS	Fetal distress
26	4747754	26	26	28	108	112	46	39.6	22.4	4	G3P1L1A1	34+3	No	No	Yes	No	2.5	PTEMG LSCS	Previous LSCS not W/F webac
27	5699696	20	24	19^{+6}	199	105	39	139	21	1	PRIMI	32	No	No	No	No		IUD	Fever
28	5699868	22	25	20^{+4}	171	154	46	94.2	30.8	1	PRIMI	39^{+3}	No	No	No	No	3	FTEMG LSCS	Breach in Labor
29	5699866	24	24	18^{+3}	151	122	41	85.6	24.4	4	G2P1L1	39	No	No	No	No	3	FTD	N/A
30	5707811	26	21	14	194	157	32	130.6	31.4	2	PRIMI	41	No	No	No	No	2.5	FTD	N/A
31	4058185	28	25	13^{+3}	201	174	39	127.2	34.8	1	PRIMI	32	No	No	No	No	1.5	PTD	Baby Died
32	4798063	23	22	16^{+6}	176	136	41	107.8	27.2	6	G2P1L1	37	No	No	No	No	2.4	FTEMG LSCS	Previous 1 LSCS not W/F webac
33	5707897	34	27	25^{+6}	158	99	29	109.2	19.8	18	G2P1L1	40+3	No	No	No	NO	Unknown	FTEMG LSCS	Oligo Hydraminos
34	5707763	25	26	25^{+5}	161	122	25	111.6	24.4	4	G2P1L1	38	No	No	No	No	3	FTD	
35	5686822	22	26	19^{+5}	178	161	38	107.8	32.2	1	PRIMI	41	No	No	No	No	Unknown	FTEMG LSCS	Unknown

36	570792	23	27	29	151	92	46	86.6	18.4	4	PRIMI							
37	4879240	22	23	23	156	121	42	89.8	24.2	4	G2P1L1	38^{+4}	No	No	No	No	2.7	FTEMG LSCS	Previous 2 LSCS in labor
38	5701956	21	24	27	157	132	37	93.6	26.4	3	G2P1L1	39^{+4}	No	No	No	No	2	FTEMG LSCS	Previous LSCS in Labor
39	5681239	26	22	13^{+1}	182	145	39	114	29	5	G3A1	38+2	No	No	No	No	2.8	FTD	
40	4200436	26	26	22	154	132	45	82.6	26.4	1	PRIMI	39+2	No	No	No	No	3	FTD	
41	5694334	20	23	27^{+2}	164	106	33	109.8	21.2	3	G2P1L1	38+3	No	No	No	NO	2.6	FTD	
42	5683871	21	21	14^{+5}	171	109	39	110.2	21.8	1.5	PRIMI	40	No	No	No	No	2.3	FTEMG LSCS	Oligo Hydraminos
43	5708434	22	21	14^{+2}	180	118	43	113.4	23.6	1	PRIMI	38	No	No	No	No	2.5	FTD	N/A
44	5700175	27	26	15	192	135	48	117	27	1.5	G2P1L1	39	No	No	No	NO	3	FTEMG LSCS	Previous LSCS in Labor
45	5708064	27	24	26	161	102	33	107.6	20.4	5	G2P1L1	40	No	No	No	NO	2.2	FTD	
46	3710161	23	25	24^{+2}	180	169	45	101.2	33.8	3	PRIMI	38	No	No	No	No	2.5	FTD	
47	4500577	22	25	19^{+3}	186	143	41	116.4	28.6	4	G2P1L1	38	No	No	No	No	3	FTEMGLSCS	Fetal distress
48	5622662	27	26	29	209	162	49	127.6	32.4	3	PRIMI	39	No	No	No	No	3.2	FTD	
49	5670519	25	22	16	170	152	37	102.6	30.4	2	PRIMI	40	No	No	No	No	3	FTD	
50	5761224	30	26	22	180	106	36	122.8	21.2	1	PRIMI	39^{+3}	No	No	No	No	2.8	FTEMG LSCS	Thick MSL
51	5721536	30	26	21^{+2}	166	122	36	105.6	24.4	10	G3P2L2	39^{+3}	No	No	No	No	3	FTEMG LSCS	Previous 2 LSCS in labor
52	5588975	22+SD53:U53	23	23^{+3}	166	100	38	108	20	1	PRIMI	38	No	No	No	No	2.8	FTEMG LSCS	Severe Oligohydraminos
53	5665243	26	20.93	20	182	177	38	108.6	35.4	10	G3P2L2	37	No	No	No	No	3	FTD	
54	4115761	27	24	14	199	151	45	123.8	30.2	2	PRIMI	37+5	No	No	No	No	2.3	FTD	
55	5683863	24	22	22	186	149	39	117.2	29.8	3	G2P1L1	37+3	No	No	No	No	2.7	FTD	
56	5687117	26	26	23	192	176	45	111.8	35.2	2	PRIMI	38	No	No	No	No	3	FTD	
57	4511308	32	27	20^{+4}	172	149	51	91.2	29.8	1	PRIMI	38	No	No	No	No	3.4	FTEMGLSCS	Breech
58	5761884	28	25	18	165	109	48	95.2	21.8	6	G4P2L2A1	39	No	No	No	No	3.6	FTEMGLSCS	Fetal distress
59	5762462	30	24	20	186	159	43	111.2	31.8	2	PRIMI	38+4	No	No	No	No	2.5	FTD	
60	5762828	30	26.6	28	170	161	38	99.8	32.2	5	G2P1L1	39	No	No	No	No	3.4	FTEMG LSCS	Previous LSCS not W/F webac
61	5765615	26	27	13	183	165	40	110	33	1	PRIMI	37	No	No	No	No	2.8	FTD	
62	5765261	24	24.8	20	199	181	46	116.8	36.2	2	PRIMI	39+4	No	No	No	No	2.7	FTEMG LSCS	Breach in Labor
63	5765157	25	25	22	174	169	41	99.2	33.8	1	PRIMI	38	No	No	No	No	3.3	FTEMGLSCS	Fetal distress
64	5236784	26	24	21^{+3}	169	152	38	100.6	30.4	1	PRIMI	39	No	No	No	No	3	FTD	
65	2517814	28	26	27	164	132	38	99.6	26.4	7	G2P1L1								
66	5722924	25	27	28	181	141	36	116.8	28.2	6	G2P1L1	39	No	No	No	No	4	FTD	N/A
67	4177186	25	22	16	174	155	41	102	31	8	G4P2L1A1	38	No	No	No	No	2	FT Elective	Precious Pregancy
68	5768339	21	20.4	22	196	142	39	128.6	28.4	1	G2A1								
69	5768671	23	22	26	182	161	42	107.8	32.2	3	PRIMI	39	No	No	No	No	3.6	FTEMGLSCS	Macrosomia With Excess Liquor
70	5611125	30	26	22^{+3}	173	140	35	110	28	1	PRIMI	39	Gest HTN	No	No	No	3	FTEMG LSCS	NPL
71	5814237	27	24	21^{+1}	185	133	32	126.4	26.6	1.5	PRIMI	36	No	No	Yes	Severe IUGR with severe Oligo	1.1	PTD	N/A
72	4936774	24	23	26	207	164	39	135.2	32.8	6	G2P1L1	38	No	No	No	No	3	FTD	
73	5595561	21	23.7	21	158	129	42	90.2	25.8	5	G2P1L1	39^{+3}	No	No	No	No	3	FTD	
74	5769516	27	23	25^{+3}	179	162	36	110.6	32.4	5	G2P1L1	40+5	No	No	No	No	2.5	FTEMG LSCS	Previous LSCS in Labor

75	5701726	28	31	28	166	149	42	94.2	29.8	9	G3P2L2	39^{+4}	No	No	No	No	3	FTD	
76	5769239	22	25	23	129	172	40	54.6	34.4	1	PRIMI	38	No	No	No	No	2.9	FTD	
77	5760594	24	27.5	21^{+6}	181	156	50	99.8	31.2	1.5	PRIMI	37	No	No	No	No	2.6	FTD	
78	5697445	21	22	15^{+6d}	174	146	39	105.8	29.2	4	G2P1L1	38	No	No	No	No	3	FTD	
79	5772612	30	27	22^{+6}	162	108	46	94.4	21.6	15	G4P2L2A1	39^{+4}	No	No	No	No	2.4	FTD	
80	5721727	35	30	14	188	161	41	114.8	32.2	8	G5P1L1A3	40	No	No	No	No	3	FTD	N/A
81	5721639	24	23	26	196	135	42	127	27	3	G2P1L1	39	No	No	No	No	3	FTD	
82	5665240	24		24^{+1}	183	159	36	115.2	31.8										
83	5738735	25	25	21^{+6}	208	179	40	132.2	35.8	8	G2P1L1	41	No	No	No	No	2.5	FTD	N/A
84	5736086	33	26	20^{+2}	193	168	35	124.4	33.6	4	PRIMI	1	No	No	No	No	3	FTD	
85	5775606	26	28	27^{+1}	159	136	27	104.8	27.2	2	PRIMI	39	Yes	No	No	No	3	FTEMG LSCS	MSL
86	5778966	24	23	26^{+2}	178	161	35	110.8	32.2	4	G2P1L1	40	No	No	No	No	3	FTD	
87	5717389	27	25	22^{+1}	182	146	39	113.8	29.2	1	PRIMI	38	No	No	No	No	2.8	FTD	
88	4263655	25	24	26^{+1}	171	159	46	93.2	31.8	1	PRIMI	39	No	No	No	No	2.6	FTEMGLSCS	Fetal distress
89	5961434	24	20	23	166	144	53	84.2	28.8	3	G2P1L1	38^{+1}	No	No	No	No	2.3	FTEMG LSCS	Previous LSCS with OligoHydraminos
90	1023800	20	26	18	190	90	67	105	18	5	G2P1L1	39^{+6}	No	No	No	No	3.5	FTD	N/A
91	5773841	30	23	18	156	135	52	77	27	5	PRIMI	39^{+3}	No	No	No	No	2.8	EMG LSCS	Thick MSL
92	5775442	20	23.6	14^{+3}	188	156	44	112.8	31.2	1	PRIMI	39^{+4}	No	No	No	Macrosomia	3.4	FTD	N/A
93	5336909	27	27.9	24	200	144	35	136.2	28.8	6	PRIMI	36^{+5}	Gest HTN	No	Yes	No	2.8	FTD	N/A
94	5663671	30	25	20	194	133	37	130.4	26.6	8	G4P3L2	31^{+1}	Yes	No	No	No	1,2(MSB)	PTD	N/A
95	5443512	32	23	20	190	189	36	116.2	37.8	5	G3P1L1A1	37	No	Yes-MNT	No	Severe Anima	2	FTD	N/A
96	5313451	29	26	20	200	178	64	100.4	35.6	1	PRIMI	40^{+3}	No	No	No	Meternal Tachycardia	3	FTEMG LSCS	Fetal Distress
97	5844546	25	29	24	198	166	68	96.8	33.2	6	G2P1L1	40^{+4}	No	No	No	No	3.6	FTD	N/A
98	5713734	25	22	18	170	158	57	81.4	31.6	2	PRIMI	39	No	No	No	No	2.4	FTEMG LSCS	Thick MSL
99	1026172	27	23	24	188	145	78	81	29	2	PRIMI	40^{+3}	No	No	No	No	2.8	FTEMG LSCS	Thick MSL
100	1027547	22	23.2	20	153	166	57	62.8	33.2	2	PRIMI	39^{+2}	No	Yes - MNT	No	No	2.9	FTD	N/A
101	1040299	27	23	16	190	158	62	96.4	31.6	3	G2P1L1	37	No	No	No	Thramobocytopinia	3.5	FTEMG LSCS	Previous LSCS in Labor
102	1025564	30	24.8	14	186	145	30	127	29	8	G3P2L2	38^{+1}	No	No	No	No	3.2	FTEMG LSCS	Previous 2 LSCS
103	1028379	24	27.2	26	168	138	47	93.4	27.6	6	G2P1L1	34^{+1}	Yes	No	No	No	1.8	PTD	N/A
104	1028490	35	33	18	190	178	53	101.4	35.6	9	G2P1L1	39^{+1}	No	No	No	No	2.4	FTEMG LSCS	Previous LSCS not W/F webac
105	5906501	21	26	22	207	156	46	129.8	31.2	6	G3P3L2	36^{+4}	No	No	Yes	No	2.6	PTEMG LSCS	Non Reassuring FHR
106	3195030	23	31	24	188	167	52	102.6	33.4	8	G2P1L1	39^{+3}	Yes	No	No	No	2.5	FTEMG LSCS	Severe PE with uncontrol hypertension
107	4035916	21	28	20	206	156	56	118.8	31.2	5	G2P1L1	39^{+1}	No	No	No	Atonic PH	3.4	FTD	N/A
108	1039295	23	24	18	156	178	40	80.4	35.6	3	G2A1	34^{+4}	No	Yes - MNT	Yes	No	2.4	EMG LSCS	NPL
109	1028578	28	22	25^{+6}	178	156	52	94.8	31.2	4	G2E1	40	No	Yes - MNT	No	No	3.2	EMG LSCS	NPL
110	1026004	19	21.6	21	200	177	26	138.6	35.4	2	PRIMI	40	Gest HTN	No	No	Cervical Tear	3	FTD	N/A

111	1025884	26	20	30^{+2}	159	169	68	57.2	33.8	2	PRIMI	39^{+5}	No	No	No	No	3	FTEMG LSCS	CPD with Oligo
112	1039512	25	20	28^{+4}	190	146	70	90.8	29.2	5	G2P1L1	40	No	No	No	No	2.4	FTD	N/A
113	1039556	23	24	30^{+2}	178	148	61	87.4	29.6	3	G2P1L1	36^{+3}	No	No	Yes	No	3.2	PTELECTIV LSCS	Macrosomia With Excess Liquor
114	1037814	30	20	22^{+6}	200	179	60	104.2	35.8	1	PRIMI	38	Gest HTN	GDM	No	No	2.9	FTEMG LSCS	Fetal Distress
115	5800424	23	25	22	190	147	58	102.6	29.4	8	G4P3L3	32^{+6}	No	No	Yes	No	1.5	PTD	N/A
116	1020242	22	26	24	156	138	48	80.4	27.6	3	G3A2	36^{+2}	No	No	Yes	No	2.6	PTD	N/A
117	1020128	27	25	24	200	188	39	123.4	37.6	3	G2P1L1	40	No	Yes - MNT	No	No	2.2	FTD	N/A
118	5367171	25	23	20	201	166	36	131.8	33.2	5	G4P1L1A2	37	No	No	No	No	3	FTEMG LSCS	Previous 1 LSCS not W/F webac
119	1023959	21	25	24	167	144	47	91.2	28.8	5	G2P1L1	36^{+1}	No	No	Yes	No	2.2	FTEMG LSCS	Fetal Tachycardia
120	1025491	22	22	24	188	139	48	112.2	27.8	3	PRIMI	38^{+6}	No	No	No	No	2.2	FTEMG LSCS	Fetal Distress
121	1024766	22	24	24	190	148	46	114.4	29.6	3	G2P1L1	36^{+2}	No	No	No	No	2.2	FTD	
122	1026753	30	20	24	178	155	39	108	31	9	G4P2L2A1	38	No	Yes-MNT	No	No	2.8	FTD	
123	1026782	33	26	16	204	166	40	130.8	33.2	8	G5P2L2	36^{+3}	No	No	Yes	No	2.6	PTD	
124	1026775	23	24	18	178	133	51	100.4	26.6	7	G2P1L1	37^{+6}	No	No	No	No	2.8	FT Elective	Previous LSCS not W/F webac
125	1025155	23	24	16	167	178	59	72.4	35.6	5	G4P2L1A1	39^{+3}	No	No	No	No	3.2	FTD	
126	1024616	36	27	24	166	148	53	83.4	29.6	5	G2P1L1	39^{+1}	No	No	No	No	2.9	FTEMG LSCS	Previous LSCS not W/F webac
127	1024610	29	22	24	178	138	66	84.4	27.6	3	G2P1L1	38^{+4}	No	No	No	No	3.3	FTD	
128	1024655	29	24	18	190	179	69	85.2	35.8	5	G2P1L1	38^{+5}	No	No	No	No	4.1	FTEMG LSCS	Fetal Distress
129	1024963	30	32	18	204	132	48	129.6	26.4	10	G2P1L1	37	Gest HTN	No	No	No	2.3	FTEMG LSCS	Previous LSCS with Gest HTN
130	1023933	29	26	23	178	144	37	112.2	28.8	1.5	PRIMI	39^{+1}	No	No	No	No	3.3	FTEMG LSCS	CDMR
131	1023798	27	26	20	167	185	48	82	37	6	G4P1L1A2	39^{+6}	No	No	No	No	2.7	FTD	
132	1023641	20	23	24	189	155	44	114	31	1.5	PRIMI	40^{+2}	Severe	No	No	No	2.6	FTEMG LSCS	Severe PE with uncontrol hypertension
133	1023193	28	24	24	178	148	37	111.4	29.6	4	G2A1	38^{+2}	Severe	No	No	No	2.5	FTEMG LSCS	Severe PE with uncontrol hypertension
134	1024309	30	28	26	190	144	67	94.2	28.8	1	PRIMI	39^{+3}	No	Yes-MNT	No	No	3	FTEMG LSCS	Pathological Trace
135	1023048	22	23	24	178	165	34	111	33	1	PRIMI	38^{+1}	No	No	No	Birth Axphyxia	2.9	FTD	
136	1023539	33	29	26	180	178	66	78.4	35.6	6	G2P1L1	37^{+6}	No	No	No	No	3.2	FTEMG LSCS	Previous LSCS in Labor
137	1024410	28	26	22	158	136	48	82.8	27.2	7	G3P1L1A1	38	No	No	No	No	2.4	FTD	
138	5713163	24	23	28^{+2}	178	128	44	108.4	25.6	1	PRIMI								
139	1059968	26	25	26	158	118	45	89.4	23.6	5	G2P1L1	38+2	No	No	No	No	2.9	FTEMG LSCS	Previous LSCS not W/F webac
140	1065528	23	24	14	162	156	65	65.8	31.2	8	G3P2L2	39	No	No	No	No	3.2	FTELECTIVE	Previois 2 LSCS
141	5606810	25	20	24	145	112	55	67.6	22.4	3	G2A1	38	No	No	No	No	3.5	FTEMG LSCS	NPL
142	5406870	25	23	22	200	156	67	101.8	31.2	1	PRIMI	40+1	No	No	No	No	2.7	FTEMG LSCS	CDMR
143	1053585	21	24	16	146	165	70	43	33	2	PRIMI	39+6	No	No	No	No	3	FTD	N/A
144	1053208	32	26	26	212	188	66	108.4	37.6	3	G2P1L1	38+4	No	No	No	No	3	FTELECTIVE	Previous LSCS not W/F webac
145	5603069	22	24	18	163	156	46	85.8	31.2	6	G4P3L2	36+5	No	No	No	No	2.5	PTD	
146	5306840	27	24	20	158	116	43	91.8	23.2	3	PRIMI	40+3	No	No	No	No	2.6	FTD	
147	5208690	22	26	16	188	166	67	87.8	33.2	1	PRIMI	40+1	No	No	No	No	3.6	FTELECTIVE	CDMR

148	5403682	22	23	20	204	148	53	121.4	29.6	3	PRIMI	40+1	No	No	No	No	2.6	FTEMG LSCS	Fetal Distress
149	5604062	22	22	16	190	134	47	116.2	26.8	1	PRIMI	38	Yes	No	No	No	2.8	FTD	
150	5604604	25	27	28	178	127	54	98.6	25.4	4	PRIMI	39	No	No	No	No	3	FTEMG LSCS	Pathological Trace
151	1066439	25	26	24	176	176	48	92.8	35.2	4	G3P2L2	39	No	No	No	No	3.1	FTD	
152	1066462	22	23	20	154	155	55	68	31	1	PRIMI	37+1	No	No	No	FGR	2.1	FTEMG LSCS	Fetal Distress
153	1069306	29	26	16	164	94	44	101.2	18.8	4	G3P1L1A1	37	No	No	No	IUGR	1.8	FTEMG LSCS	Fetal Distress
154	1055412	24	25	24	166	110	56	88	22	1	PRIMI	39+4	No	No	No	No	3.1	FTEMG LSCS	CDMR
155	1055392	23	27	22	187	116	57	106.8	23.2	1	PRIMI	39+2	No	No	No	No	2.5	FTEMG LSCS	MSL
156	1055104	27	28	24	188	132	50	111.6	26.4	1	PRIMI	40+2	No	No	No	No	3.3	FTEMG LSCS	Severe Oligohydraminos
157	5663421	40	23	24+2	220	176	58	126.8	35.2	5	G3P2L2	40+2	No	Yes-MNT	No	No	3.2	FTEMG LSCS	Fetal Distress
158	5602346	24	22	18	168	123	62	81.4	24.6	3	G3P2L1	34+3	No	No	Yes	No	1.8	FTEMG LSCS	Previous LSCS not W/F webac
159	5611241	20	23	24	178	156	64	82.8	31.2	1	PRIMI	37	Gest HTN	No	No	No	2.7	FTEMG LSCS	MSL with Fetal Distress
160	1050253	24	26	25	188	174	62	91.2	34.8	4	G2p1L1	38	No	No	No	No	2.9	FTEMG LSCS	Previous LSCS not W/F webac
161	5610206	40	32	24^{+1}	166	100	38	108	20	1	PRIMI	38	No	No	No	No	2.8	FTEMG LSCS	Severe Oligohydraminos
162	5765157	26	28	21^{+6}	182	177	38	108.6	35.4	10	G3P2L2	37	No	No	No	No	3	FTD	
163	5236755	28	26	20^{+2}	199	151	45	123.8	30.2	2	PRIMI	37+5	No	No	No	No	2.3	FTD	
164	2517814	34	28	27^{+1}	186	149	39	117.2	29.8	3	G2P1L1	37+3	No	No	No	No	2.7	FTD	
165	5722924	26	25	26^{+2}	192	176	45	111.8	35.2	2	PRIMI	38	No	No	No	No	3	FTD	
166	4177186	28	25	22^{+1}	172	149	51	91.2	29.8	1	PRIMI	38	No	No	No	No	3.4	FTEMGLSCS	Breech
167	5768339	27	24	26^{+1}	165	109	48	95.2	21.8	6	G4P2L2A1	39	No	No	No	No	3.6	FTEMGLSCS	Fetal distress
168	5768671	28	22	23	186	159	43	111.2	31.8	2	PRIMI	38+4	No	No	No	No	2.5	FTD	
169	5611125	32	25	18	170	161	38	99.8	32.2	5	G2P1L1	39	No	No	No	No	3.4	FTEMG LSCS	Previous LSCS not W/F webac
170	5814237	22	25	18	183	165	40	110	33	1	PRIMI	37	No	No	No	No	2.8	FTD	
171	4936774	29	27	14^{+3}	199	181	46	116.8	36.2	2	PRIMI	39+4	No	No	No	No	2.7	FTEMG LSCS	Breach in Labor
172	5595561	27	28	24	174	169	41	99.2	33.8	1	PRIMI	38	No	No	No	No	3.3	FTEMGLSCS	Fetal distress
173	5769516	27	29	20	169	152	38	100.6	30.4	1	PRIMI	39	No	No	No	No	3	FTD	
174	5546721	26	23	24	198	166	68	96.8	33.2	6	G2P1L1	40^{+4}	No	No	No	No	3.6	FTD	N/A
175	5454670	32	26	26	212	188	66	108.4	37.6	3	G2P1L1	38+4	No	No	No	No	3	FTELECTIVE	Previous LSCS not W/F webac