LIPID PROFILE IN SECOND TRIMESTER OF PREGNANCY AS A PREDICTOR OF PRE ECLAMPSIA IN PATIENTS ATTENDING ANTENATAL CLINIC IN KILPAUK MEDICAL COLLEGE AND HOSPITAL

A PROSPECTIVE COHORT STUDY

Dissertation Submitted To

THE TAMILNADU Dr.M.G.R MEDICAL UNIVERSITY

In partial fulfilment of the requirement

For the award of

M.D.DEGREE – OBSTETRICS & GYNECOLOGYKILPAUK

KILPAUK MEDICAL COLLEGE, CHENNAI.

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CERTIFICATE

This is to certify that this dissertation titled **"LIPID PROFILE IN SECOND TRIMESTER OF PREGNANCY AS A PREDICTOR OF PREECLAMPSIA IN PATIENTS ATTENDING ANTENATAL CLINIC IN KILPAUK MEDICAL COLLEGE AND HOSPITAL"** has been prepared by **Dr. M. PADMAPRIYA,** under my supervision in the Department of Obstetrics and Gynaecology, Government Kilpauk Medical College, Chennai , during the academic period 2010 – 2013 and is being submitted to the **Tamilnadu Dr. M.G.R. Medical University, Chennai** in the partial fulfilment of the University regulation for the award of the M.D (O & G) and her dissertation is a bonafide work.

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DECLARATION

I, **Dr. M. PADMAPRIYA,** solemnly declare that this dissertation **"LIPID PROFILE IN SECOND TRIMESTER OF PREGNANCY AS A PREDICTOR OF PREECLAMPSIA IN PATIENTS ATTENDING ANTENATAL CLINIC IN KILPAUK MEDICAL COLLEGE AND HOSPITAL"** was prepared by me at Government Kilpauk Medical College and Hospital, Chennai, under the guidance and supervision of **Dr.T.K.SHAANTHY GUNASINGH,** M.D.,D.G.O, Professor of Obstetrics and Gynaecology, Govt. Kilpauk Medical College and Hospital, Chennai.

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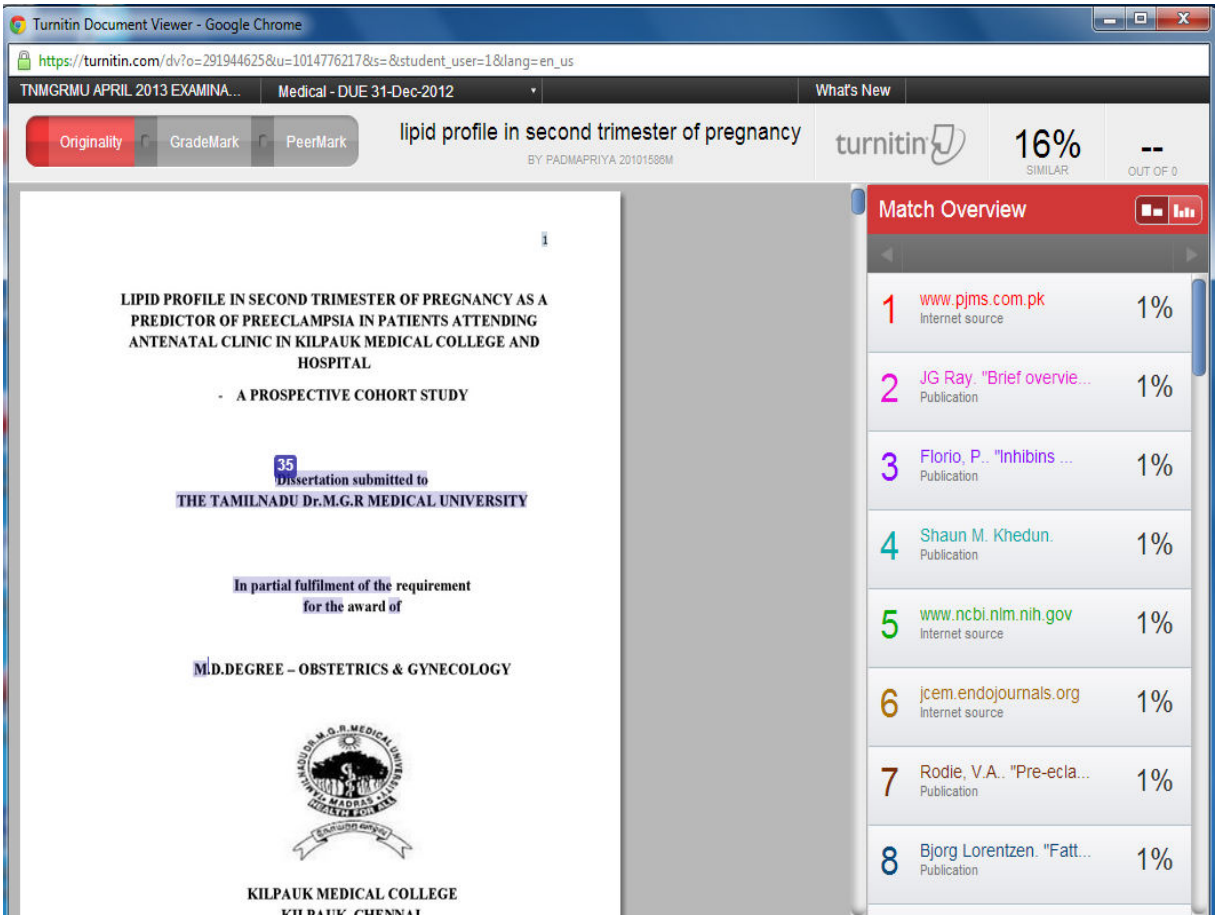
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LIST OF ABBREVIATIONS USED

BMI	-	Body Mass Index
Fig	-	Figure
H/o	-	History of
HDL	-	High Density Lipoproteins
HT	-	Hypertension
LDL	-	Low Density Lipoproteins
Mgs	-	milligrams
S .D.	-	Standard Deviation
S .E	-	Standard Error
TGLS	-	Triglycerides
VLDL	-	Very Low Density Lipoproteins
Yrs	-	Years
%	-	Percentage

INTRODUCTION

Hypertensive disorders of pregnancy complicates around 5-10% of all pregnancies [1]. It is one of the common causes of maternal & perinatal morbidity and mortality.

How pregnancy initiates the rise in blood pressure or aggravates hypertension still remains an enigma inspite of research for many decades and it still remains one among the most significant and unsolved problems in obstetrics.

Studies state that abnormal placentation and endothelial dysfunction are the key factors in the development of preeclampsia. Several markers have been investigated as the predictors of pre-eclampsia. Lipid levels in early pregnancy can be a good predictor of development of pre-eclampsia in patients. Dyslipidemia in early pregnancy leads to more oxidative stress by the formation of lipid peroxides and reactive oxygen species, thus predisposing to the development of pre-eclampsia.

According to WHO even in developed countries, preeclampsia accounts for 16% of maternal mortality (Khan & Colleagues 2009) [27]. For every case of maternal death there are ten near miss cases. Such is the magnitude of this multi system disorder.

Berg & colleagues (2005) stated that around 50% of these preeclampsia related deaths are preventable[6].

Thus early identification of pregnancies at risk may enhance the development of new strategies for antenatal monitoring, to detect disease earlier and intervene appropriately to improve maternal and perinatal outcome.

REVIEW OF LITERATURE

Hypertension is the most common medical problem encountered in pregnancy. It is the second most common cause of maternal mortality in India and a major cause of perinatal mortality and morbidity. Pre-eclampsia is not simply hypertension complicating pregnancy, but a protean disorder affecting virtually every system in the body. Hypertension is only one manifestation.

According to the International Society for the study of Hypertension in pregnancy (ISSHP), hypertension is defined as a systolic blood pressure >140 mm Hg or a diastolic blood pressure >90 mm Hg. A rise in the systolic blood pressure of 30 mm Hg or a rise in the diastolic blood pressure of 15mm Hg, at least 4 hours apart or a single diastolic blood pressure >110mm Hg is also considered as hypertension.

In normal pregnancy, the diastolic blood pressure begins to fall in early pregnancy and continues to fall in the second trimester to reach a nadir at 22-24 weeks.

Then it steadily rises to reach the prepregnant levels by term. This fall is due to the reduced vascular tone, which leads to peripheral vasodilatation.

CLASSIFICATION

According to the latest classification by the working group of the National High Blood Pressure Education Programme (NHBPEP), hypertensive disorders of pregnancy is classified into five types:

1. **Gestational hypertension or pregnancy induced hypertension** is non-proteinuric hypertension arising for the first time after 20 weeks of gestation. The blood pressure returns to normal within 12 weeks postpartum. It is a diagnosis of exclusion.

2. **Pre-eclampsia :**

   New onset proteinuric hypertension after 20 weeks of gestation.

3. Eclampsia :

Eclampsia is defined as seizures that cannot be attributed to any other cause in a woman with pre-eclampsia.

4. Chronic hypertension :

Chronic hypertension is defined as hypertension antedating pregnancy or hypertension diagnosed before 20 weeks of pregnancy, but not attributable to gestational trophoblastic disease. Hypertension

first diagnosed after 20 weeks & persisting 12 weeks postpartum is also considered chronic hypertension.

5. Superimposed Pre-eclampsia :

This is defined as new onset proteinuria or a sudden increase in blood pressure or poteinuria in a woman with chronic hypertension.

ETIOLOGY

The exact etiology of preeclampsia is not known. But there are risk factors predisposing to development of pre-eclampsia. Genetic factors include family history of pre-eclampsia.

Obstetric factors include primiparity, previous history of pre-eclampsia, new paternity, multiple pregnancy, hydrops fetalis with large placenta, hydatidiform mole, triploidy.

Medical factors include chronic hypertension, diabetes, renal disease, antiphospholipid antibody syndrome, inherited thrombophilias, connective tissue disorders, hyperhomocysteinemia.

PATHOGENESIS

The pathology of this multi system disorder should be considered as two staged process.

1. Abnormal placentation

2. Maternal systemic reaction that produces clinical signs / symptoms that characterise this disorder.

Abnormal Placentation :

In normal pregnancy, the spiral arteries of the placenta are invaded by the cytotrophoblast and the elastic & muscular coats are replaced by fibrinoid .Early in second trimester, a second wave of cytotrophoblastic invasion occurs. This transforms the myometrial segments of the spiral arteries into wide mouthed vessels unresponsive to vasomotor stimuli. Thus, the blood supply is transformed from a high-resistance low-flow system to a low resistance high flow system in order to increase the uteroplacental flow to meet the demands of the fetus.

In pre-eclampsia, the primary wave of trophoblastic invasion is partly impaired and the second wave fails to occur. This results in reduced uteroplacental blood flow, which worsens as the pregnancy advances. In addition, the arteries remain very sensitive to vasomotor stimuli. These changes are not specific to pre-eclampsia but also occurs in IUGR without pre-eclampsia.

The myointimal cell proliferation, endothelial damage with medial necrosis forms the early pre-eclamptic changes. These findings with lipid

laden cells infiltration is termed as ‘atherosis’. A similar pathology is seen in atherosclerosis.

Since abnormal lipid profiles are associated with atherosclerosis, the hypothesis of dyslipidemia for development of pre-eclampsia is biologically plausible.

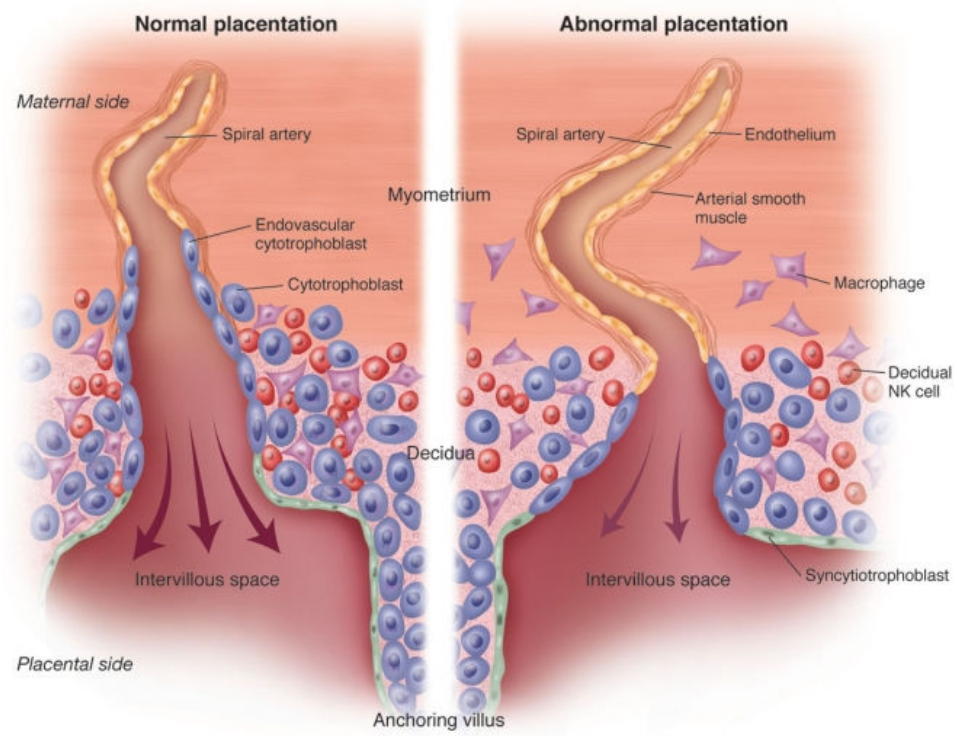


Fig 1: Abnormal Placentation in Preeclampsia

Vasospasm & hypoxia - inducing systemic inflammatory response:

These narrow spiral arteries impair perfusion leading to a hypoxic environment. Hypoxia eventually leads to release of placental debris into maternal circulation inciting a systemic inflammatory response.

Dysregulation or loss of maternal immune tolerance to paternally derived placental debris leads to activation of leucocytes and release of cytokine resulting in endothelial injury.

In pre-eclampsia, due to the endothelial dysfunction, there is a reduction in prostocyclin and nitric oxide, which are vasodilators and an increase in endothelin and thromboxane which are vasoconstrictors. Besides this, loss of vascular insensitivity to pressor agents results in vasospasm and thereby increases vascular resistance and blood pressure.

Endothelial dysfunction leads to release of cytokines such as TNF-alpha and interleukins which may contribute to oxidative stress characterised by increased ROS(Reactive Oxygen Species) & free radicals that leads to formation of self propagating lipid peroxides.

The increased levels of lipid peroxides and decreased antioxidant activity in pre-eclampsia have raised the possibility that markers of oxidative stress may predict pre-eclampsia.

As blood lipids like triglycerides and free fatty acids and lipoprotein belong to the variety of pro-oxidants or potentiators of pro-oxidants, they can be used as markers of pre-eclampsia.

Endothelial dysfunction will lead to activation of platelets & coagulation system by the release of tissue factors from endothelium. This results in widespread DIC ranging from subclinical to frank DIC.

PATHOPHYSIOLOGY

Placenta :

The typical vascular changes in pre-eclampsia is termed ‘atherosis’. This is characterised by fibrinoid necrosis, macrophages and mononuclear cell infiltration.

Kidney :

The main pathology in the kidney is glomerular endotheliosis, which narrows the lumen. This comprises swollen endothelial cells due to fibrin deposition. There is glomerular & tubular dysfunction.

The main pathology is glomerular dysfunction, the manifestation of which is proteinuria. There is also reduction in glomerular filtration rate and creatinine clearance, which in severe cases leads to increase in the blood urea & serum creatinine.Acute renal failure can rarely supervene and is usually due to acute tubular necrosis which is reversible.

Tubular dysfunction is manifested by hyperuricemia. Hyperuricemia is also caused by the placental ischemia leading to increased trophoblastic turnover and increased production of purines.

Liver :

Periportal thrombosis and fibrin deposition, haemorrhages and necrosis are seen in the liver. There is an increase in the liver enzymes SGOT and SGPT and clinical jaundice can occur. The liver changes are responsible for the nausea and vomiting in severe cases. The small haemorrhages may coalesce to form a subcapsular hematoma, which may cause stretching of the Glison's capsule and epigastric pain. This is a very serious sign and seen in impending eclampsia .These changes are responsible for HELLP Syndrome .An extremely rare but catatrophic complication is liver rupture.

Brain:

The main finding in the brain is cerebral vasospasm. Small cerebral haemorrhages, thrombosis and fibrinoid necrosis can occur especially in eclampsia and are secondary to endothelial dysfunction. Cerebral oedema is also usual in eclampsia.

Massive cerebral haemorrhage is a rare complication of severe hypertension. Visual disturbances are common and usually due to edema of

the occipital lobe. Cortical blindness can occur due to occipital edema, which is usually temporary.

Eyes:

Localised retinal vasospasm is the commonest finding. Haemorrhages and papilloedema may be seen rarely seen in severe hypertension. Blindness could rarely due to retinal artery ischemia or infarction.

COMPLICATIONS

Pre-eclampsia can virtually affect any organ in the body. Hence complications can be expected in any organ in this multi-system disorder. Maternal complications include

- Eclampsia
- Cerebral hemorrhage
- Cortical blindness
- Pulmonary edema
- ARDS(Adult Respiratory Distress syndrome)
- HELLP syndrome
- DIC and hemorrhage
- Renal failure
- hepatic rupture,
- abruptio placenta & sudden postpartum collapse.

Fetal complications

- Prematurity
- IUGR
- Intra uterine death.

PREDICTORS OF PRE-ECLAMPSIA:

As early detection of pregnancies at risk may lead to development of new strategies for antenatal monitoring and improve maternal and perinatal outcome, many markers have been proposed as predictors of pre-eclampsia.

These are biological, biochemical, biophysical markers of impaired placental perfusion, vascular resistance, fetal- placental endocrine dysfunction, oxidant stress, endothelial dysfunction, activation of coagulation related to the pathophysiology of pre-eclampsia.

But no single test is economical and sensitive. Some of them are:

1) Mean arterial pressure (MAP) – in the second trimester 90mm Hg or more is a predictor of pre-eclampsia.
2) Gant's roll over test – The woman is turned from the left lateral to the supine position. If there is an increase in the diastolic blood pressure of 20mm Hg or more, the test is considered positive.
3) Angiotensin Sensitivity test – is based on the fact that women destined to develop pre-eclampsia lose their refractoriness between

28-32 weeks of gestation. If a pressor response occurs with <8ng/kg/min of infused angiotensin, 90% are likely to develop pre-eclampsia.But this pressor provocative test is invasive.

4) Uterine artery Doppler – in the non-pregnant state there is decreased diastolic flow and notching of the uterine arteries. In normal pregnancy, due to the trophoblastic invasion, this notch disappears and the flow increases. If there is persistence of a diastolic notch in the uterine artery at 20-22 weeks of gestation, it indicates that the second wave of trophoblastic invasion has not occurred and is predictive of pre-eclampsia.

   The negative predictive value is better than the positive predictive value that is the disappearance of the notch is more likely to predict that the pregnancy is likely to normal.

5) Pulse wave analysis: similar to the uterine artery Doppler velocimetry, 'stiffness' in the finger arterial pulse acts a pre-eclampsia predictor.

6) Raised Uric acid – the placental ischemia leads to increased trophoblastic turnover and increases the production of purines. Uric acid being the metabolite of purine metabolism is increased and is one of the earliest laboratory manifestations of pre-eclampsia.

7) Raised serum beta hCG at 14-20 weeks of gestation – Due to abnormal trophoblastic invasion – beta hCG is produced in increased amounts. This is also due to placental dysmaturity. The hypoxic trophoblast reacts to diminised oxygen by an oversecretory effect thus leading to increased beta hCG production.

8) Tests relating to fetal-placental unit endocrine dysfunction are – alpha fetoprotein (AFP), estriol levels, Pregnancy associated Protein A (PAPP A), inhibin A levels, Activin A, Placental protein 13, corticotrophin releasing hormone.

9) Platelet count, fms – like tyrosine kinase receptor-1(sFlt-1), endoglin plasminogen activitator inhibitor (PAI) , neurokinin B, p-selectin, decreased levels of pro-angiogenic factors that includes vascular endothelial growth factors(VEGF), placental growth factor(PlGF), endothelial adhesion molecules, C- reactive proteins – are all claimed as predictors of pre-eclampsia as they are markers of either endothelial dysfunction or oxidative stress.

10) Free fetal DNA – due to ischemia in placentation – there is accelerated apoptosis of cytotrophoblast in pre-eclampsia. This leads to release of free - fetal DNA in to maternal circulation.

11) Dyslipidemia as a predictor of pre-eclampsia – as blood lipids including triglycerides , free fatty acids and lipoproteins belongs to

the variety of pro-oxidants or potentiators of pro-oxidants they can be used as markers to predict pre-eclampsia.

The correlation between abnormal lipid profiles and development of pre-eclampsia is proposed based on two hypotheses.

a) Lipoproteins and lipids are potentiators of oxidative stress. Hence an abnormally elevated lipid levels leads to more oxidative stress resulting in endothelial dysfunction – which is the key step in the pathogenesis of this common disorder of pregnancy.
b) The second mechanism is through the metabolic syndrome X (or) insulin resistance syndrome. Hyperinsulinemia & hyperuricemia is also present in pre-eclampsia.

Hyperinsulinemia alters the function of lipoprotein lipase in the adipose tissue – an important post hepatic enzyme in the lipid metabolism. The altered lipoprotein lipase eventually leads to abnormally elevated levels of triglycerides and free fatty acids. Increased insulin levels also causes mobilisation of lipids from the visceral fats. It also enhances the production of Very Low Density Lipoprotein in the liver.

This also explains that the genetic & environmental factors leading to metabolic X syndrome is also important in determining the occurrence of pre-eclampsia.

Lipid metabolism in normal pregnancy

The concentrations of lipids, cholesterol, lipoproteins increases during pregnancy. Fat accumulation occurs primarily in the mid pregnancy. The total increase in fat is around 3.5kg, mainly central in its distribution in the abdomen, breasts, hips and thighs rather than in peripheries. This storage occurs to meet the increased demands of third trimester for transfer of essential fatty acids which is required by the fetus during its maximal growth.

These changes are due to the effects of progesterone. Progesterone acts and alters the hypothalamic lipostat. After delivery, the progesterone rests the lipostat to its previous non-pregnant state. Thus, the concentration of lipids decrease and lactation accelerates this decrease.

The most important change in lipid profile during pregnancy is hyperlipidemia. There is a consistent increase in glycerides mainly triglycerides. The other components such as total cholesterol, Low Density Lipoprotein, High Density Lipoprotein, Very Low Density Lipoprotein is also increased during pregnancy.

These are due to the effects of sex hormones especially estrogen & progesterone on the liver.

The lipolytic activity is also increased and the activity of lipoprotein lipase in decreased especially in adipose tissue. Although, there is a physiological hyperlipidemia in normal pregnancy, the rise is even higher in pregnancies complicated by pre-eclampsia and this occurs even earlier.

Management:

Mild preeclampsia:

Patients with mild preeclampsia can be treated on an outpatient basis. Day care units are helpful. Whether admitted to hospital or not, monitoring of maternal and fetal condition is essential.

Sedentary activity throughout the greater part of the day is recommended. There is no place for salt restriction, diuretics or sedatives. Diuretics will further reduce the uteroplacental flow and worsen the IUGR. The only indication for diuretics is pulmonary edema.

Antihypertensives :

The effectiveness of antihypertensives in mild preeclampsia is controversial. The main objective is to reduce the risk of severe hypertension and cerebral haemorrhage. Once the mean arterial pressure (MAP) is more than 150 mm Hg, there is loss of cerebral autoregulation and a high risk of cerebral haemorrhage. Antihypertensives may help in

prolongation of pregnancy, but it is important to remember that the disease process is not modified. Therefore, there is danger that they may mask the detection of severity. Overzealous correction of hypertension may lead to further reduction in uteroplacental flow and IUGR and hence best avoided. The commonly used first line drugs are alpha methyl dopa, nifedipine and labetolol. Labetolol should be avoided in women with known asthma.

Monitoring:

Maternal

- Blood pressure and urine albumin daily.
- Urine output daily
- Alternate day weight
- Watch for imminent symptoms
- Twice weekly peripheral smear,platelet count, coagulation profile,uric acid,renal and liver function tests.

Fetal:

- Daily fetal movement count
- Ultrasound to assess fetal growth and well being
- NST and amniotic fluid volume assessment
- Doppler velocimetry in IUGR

The frequency of monitoring has to be individualised depending upon the severity and presence of IUGR.

Delivery

Delivery is the only definitive treatment for preeclampsia and usually labour is induced at 38 weeks. Early termination may be needed if there is progression to severe preeclampsia or eclampsia with worsening of either the maternal or the fetal condition. Antenatal corticosteroids to accelerate lung maturity should be considered if preterm. If there are no obstetric indications for caesarean section labour can be induced. If the cervix is favourable, ARM and oxytocin infusion is used for induction. If the cervix is unfavourable, PGE_2 gel can be used to ripen cervix .Continuous CTG monitoring is ideal during labour. AMTSL should be followed.

SEVERE PREECLAMPSIA

In severe preeclampsia, there is deterioration of either the maternal and fetal condition or both and again the only definitive treatment is delivery. After 34 weeks, severe preeclampsia is best treated by termination especially if there is worsening of biochemical parameters. Severe preeclampsia developing before 24 weeks is probably best managed by termination of pregnancy. In cases before 34 weeks if the initial condition stabilises there may be a place for expectant management.

EXPECTANT MANAGEMENT

The aim of the expectant management is to protect the mother and the fetus from the consequences of the disease and at the same time, prolong pregnancy if possible to avoid the dangers of prematurity to the fetus. At any time when there is a worsening in the condition, expectant management is abandoned and immediate termination decided.

Antihypertensives are definitely indicated to prevent cerebral haemorrhage. The main danger is that they may give a false sense of security by masking hypertension.

Close monitoring of maternal and fetal condition is performed as in mild preeclampsia but much more frequently. Poor oxygen saturation can occur in pulmonary edema and so measurement of oxygen saturation using pulse oximetry is indicated in such cases. Antenatal corticosteroids to accelerate lung maturity.

INDICATIONS FOR IMMEDIATE TERMINATION OF PREGNANCY

- Uncontrolled hypertension
- Imminent eclampsia or eclampsia
- Abnormal renal or liver function tests or coagulopathy

- HELLP syndrome
- Fetal distress
- Severe IUGR
- Abruption
- Pulmonary edema

INTRAPARTUM MANAGEMENT

Control of blood pressure:

The diastolic blood pressure should not be allowed to cross 110 mm Hg. Labetolol, hydralazine and nife dipine can be used. Whatever the drug used, a rapid fall in blood pressure should be avoided, as it may be deleterious to the fetus. Hence close monitoring of the blood pressure is essential.

Prophylactic magnesium sulphate may be given during labour to prevent eclampsia.

Fluid Management :

Preeclamptic patients have increased extracellular fluid although there is contracted intravascular volume. Excessive parenteral fluids can lead to fluid overload and pulmonary edema, which may be fatal. If there is coagulopathy or HELLP syndrome, blood and blood products like fresh frozen plasma and platelet concentrate may have to be given.

INDICATIONS FOR CESEAREAN SECTION

- Associated obstetric indications
- Failed induction
- Rapid worsening of maternal condition and delivery not imminent
- Fetal distress or severe IUGR

Lewis & Steiner et al (1996) – did a study on several ways by which the vascular function is compromised by elevated triglycerides.

They stated that the lipoprotein that is rich in triglycerides have tendency towards pro-thrombotic activity, thus having a role in pathology of pre-eclampsia. [28]

Plotnick et al and Vogel et al studied the role of lipoprotein that is rich in triglycerides in inhibiting the vasodilation that is endothelium dependent. Triglycerides stimulates NADPH oxidase especially leucocytes NADPH oxidase, and increases peroxidation susceptible LDL particles.

Both of these leads to increased production of Reactive Oxygen Species. Reactive Oxygen Species in turn can either destroy nitric oxide or inhibit nitric oxide synthase leading to decreased bio availability of nitric oxide, a prime factor in endothelium dependant vasodilatation, hence

explaining the role of triglyceride rich lipoprotein in pathogenesis of pre-eclampsia[28.]

R.K. Vidyabati, Hijam Davina et al studied the predictive role of abnormal lipid profile in early pregnancy in development of preeclampsia. This study included 164 pregnant women between 14 to 20 weeks of gestation and their lipid levels checked. 29 cases developed PIH, while 135 cases remained normotensive[2].

Concentration of total cholesterol & very Low Density Lipoprotein in women who subsequently developed PIH were substantially higher than that of normotensive pregnant women ($p < 0.027$).

In this study for each unit increase in total cholesterol there was a 12.6% increase in the risk of occurrence of preeclampsia. And for a unit increase in TG & LDL there was a 0.3%, 7.4% increase in the risk of developing PIH. Thus, this study has shown that the dyslipidemia as a good non-invasive predictor of preeclampsia.

JG Ray, P Diamond b (2006) studied the risk of preeclampsia in the presence of maternal hypertriglyceridemia. Two investigators searched studies from 1980 to 2004 related to dyslipidemia and preeclampsia.

Out of the 22 total studies included; 3 were cohort studies and the rest were case control studies. In 14 studies, there was a higher mean triglyceride concentration among preeclampsia cases than among normotensive controls.

When potential confounders were adjusted in four of the studies, there was a fourfold higher risk of preeclampsia.

The authors concluded that there exists a consistent positive association between elevated maternal triglycerides and risk of preeclampsia. They also concluded that since hypertriglyceridemia is one of the features of insulin resistance syndrome, further additional studies are required to find out whether interventions such as lifestyle modification leading to decreased BMI in prepregnancy can lower the risk of pre-eclampsia.[3]

Lorentzen B, Henriksen T - (1998) did a study to show the role of abnormal lipid profile in pathology of preeclampsia. They suggested that preeclampsia has a similar pathology as that of atherosclerosis. In both these disorders endothelial dysfunction is induced by hyperlipidemia. Hyperlipidemia induces endothelial injury by promoting oxidative stress in the arterial wall.

According to them, the effects of placentally derived endothelial disturbing factors may be enhanced because of hyperlipidemia mediated activation or sensitization of the endothelial cells[4]

Anceschi M M coata G et al in 1992 studied the composition of RBC membrane in preeclampsia. The altered composition acts as an indirect evidence for altered lipid profile.

This study included 30 women with preeclampsia and 26 controls matched for gestational age who were normotensive pregnant women &10 more normotensive nonpregnant nulliparous women. The cholesterol/ phospholipid ratio was significantly higher in women with preeclampsia than pregnant women with normal BP. This represents one factor involved in pathogenesis of preeclampsia and a possible predictive factor for the disease[5].

Van Den Elzen HJ, Wladimroff JW, Cohen Overbeek TE studied the relationship between serum lipid levels in early pregnancy and the occurrence of preeclampsia.

This prospective cohort study included three ninety three pregnant patients. The study told that first trimester serum total cholesterol if higher was significantly associated with the risk of preeclampsia.

The adjusted relative risk exceeds 5 for women with serum total cholesterol Levels more than 6 mmol per litre[12].

Arpita Basu, Peter Alaupovic et al studied the link between maternal abnormal lipid profile and preeclampsia in women with type1 diabetes.

This study included 118 Type1 diabetes pregnant patients. Serum lipid profiles, sub classes of lipoproteins, serum apolipoproteins were measured. Early in pregnancy increased cholesterol rich lipoproteins were associated with subsequent preeclampsia[12].

Lorentzen B Drevon CA et al studied the composition of esterified free fatty acids in the sera of women with preeclampsia and normal pregnancy. The fasting blood samples from 510 healthy nullipara at 17-19 weeks of gestation were taken and analysed. Among the circulating free fatty acids the levels of palmitic, oleic and linoleic acids were significantly higher early in pregnancy in women who later developed proteinuric hypertension.

The same free fatty acids were also increased in women with preeclampsia. Thus the study concluded that the level and the composition of circulating lipids were already altered 10-20 weeks before the clinical diagnosis of preeclampsia [7]

Clausen T, Djurovics , Henriksen TE yet all 2001 investigated whether increased triglyceride is associated with early or late onset preeclampsia. It was a prospective Cohort study. Nested case control study design was also included.

Multiple logistic regressions were used to analyse cohort data and conditional logistic regression for case control data. This study was done on 2157 pregnant volunteers 18 women were diagnosed with early onset preeclampsia. Late onset preeclampsia was diagnosed in 53 women.

They concluded that increased triglyceride in early weeks of pregnancy strongly related with the occurrence of early onset preeclampsia. Late onset preeclampsia did not have any association.

Thus this study supported the hypotheses that there exists pathogenic differences between early and late onset preeclampsia [11].

Satar N, Bendomir A et al studied the relationship of concentration of lipoprotein subfractions in normal pregnant patient and compared it with preeclamptic patients.

This study concluded that there is significant rise of triglyceride rich glycoprotein in patients with proteinuric hypertension compared with normal pregnant patients.

They suggested that abnormal lipid profile will increase oxidative stress through small dense low density lipoprotein formation. Thus eventually leading to endothelial dysfunction. This study also supports the view of pathogenic similarity between atherosclerosis and preeclampsia[14].

Barden AE, Beilin LJ et al(1999) studied the various factors in non-pregnant and pregnant that predisposes to pre-eclampsia. It was a retrospective study that included 62 pre-eclamptic women & 84 normal pregnant women.

Factors such as anthropometry, blood pressure & various laboratory parameters were analysed. In this study, irrespective of the parity, patients with hypertension had abnormally increased BMI and lipid concentration during pre-conceptional, natal and postnatal periods.

The study concluded that abnormal lipid concentrations, increased BMI in the non-pregnant state symbolises insulin resistance syndrome. These factors sensitise the endothelium and predisposes to development of

pre-eclampsia. The same explanation holds good for genetic predisposition of pre-eclampsia.[15.]

Barden A (2006) studied the various maternal constitutional parameters leading to the risk of developing Preeclampsia. He concluded that already existing increased blood pressure, altered glucose tolerance, increased BMI,& dyslipidemia greatly raise the risk of development of preeclampsia. This study also stresses the role of insulin resistance as a risk factor of developing preeclampsia[16]

Daniel et al (2004) studied the role of abnormal lipid profile in early gestation and the occurrence of preeclampsia later in pregnancy. This prospective study included 567 women out of which 57 developed preeclampsia.

The procedure used for calculation is logistic regression. The risk of proteinuric hypertension increased linearly with increasing low density lipoprotein concentrations. Thus they concluded that abnormally raised lipid levels in early gestation can act as a marker and a predictor of preeclampsia[17].

Wolf M, Kettyl et al (2001) studied the relationship of obesity with inflammation in the development of preeclampsia[18].

They showed that the levels of inflammatory mediators are present in increased levels in the serum of patients in early gestation who developed preeclampsia later in gestation.

They stated that obesity through inflammation makes the pregnant women more prone for preeclampsia if she is obese.[18]

Thadhani et al (1999) studied the role of prepregnant obesity and abnormal lipid profile in the development of hypertensive disorders of pregnancy. The study included 15262 women.

They concluded that preconceptional obesity and dyslipidemia may be used as markers of development of preeclampsia during pregnancy.

They found no relationship between dyslipidemia and the occurrence of gestational hypertension. They hypothesised that dyslipidemia promotes the formation of lipid peroxides and reactive oxygen species and makes an imbalance of vasoactive mediators leading to endothelial dysfunction and vasospasm – a protean event in the pathogenesis of preeclampsia[19].

Hubel CA, Lyall et al did a nested case control study in 1998 comparing the levels of SLDL and VCAM-1 in preeclamptic women with gestationally matched normal pregnant women without any complications.

The size and density distribution of LDL is influenced by the serum levels of TGL. These small LDL are promoters of endothelial dysfunction.

Hypertriglyceridemia was observed in preeclamptic patients and the Low density Lipoprotein peak particle diameter had an indirect association with risk of preeclampsia.

They concluded that the rise in concentration of small and denser LDL in directly related to the development of preeclampsia. The other marker of endothelial involvement that was tested was VCAM-1 which was influenced by the amount of lipoproteins and not the particle size[20].

Ware – Jauregui et al (1999) conducted a case control study that included 125 preeclamptic women as cases and 179 pregnant patients without any complications as controls.

The levels of triglycerides were on the higher side in case of patients with proteinuric hypertension. For each quartile increase in the levels of triglycerides there was a consistent rise in the development of preeclampsia.

They concluded that the levels of high density lipoprotein had an inverse relationship with occurrence of preeclampsia[21.]

Vanderjagt DJ, Patel RJ et al (2004) studied the role of homocysteine levels and the development of preeclampsia. They also studied the relationship between homocysteine levels and HDL levels.

They concluded that hyperhomocysteinemia is directly related to preeclampsia and HDL levels are inversely related to preeclamptic risk[22].

O 'Brien et al (2003) investigated the role of prepregnant body weight in the development of preeclampsia. They concluded that increased BMI increases the risk of preeclampsia.

They also highlighted the increasing incidence of preeclampsia in developed countries, where there is an increasing tendency towards obesity, thus insisting on weight reduction during the pre-pregnant state[23].

Gratocos E, casal E et al(2003) did a case control study in 70 patients. LDL oxidation susceptibility and lipids were measured. They found that the mean lipid levels were greatly increased in preeclamptic women when compared with normotensive controls.

The study also showed that the LDL in Preeclampsia patient were more prone to oxidation with a p value <0.01[24.]

Wakatsuki A et al (1996) did a comprehensive study of antioxidant status and oxidative stress in preeclampsia and compared it with normal pregnancy.

This study also supported that the HDL levels were low in patzents with hypertension [25].

Mikhail et al studied the levels of TGL in preecalmptic women and concluded that there exists a positive correlation with severity of preeclampsia.

Bodnar et al (2005)[26] did a study to find out the role of pre-pregnancy obesity in the development of pre-eclampsia in a dose dependant relation. This was a prospective cohort study that included 1179 women in their first pregnancy at 16 weeks of gestation.

They concluded that the risk of pre-eclampsia rises with increasing BMI and the risk decreases with decrease in BMI.

AIM OF THE STUDY

To study whether abnormal lipid profile in the second trimester of pregnancy can be a predictor of pre-eclampsia.

MATERIALS AND METHODS

STUDY DESIGN

Prospective Cohort Study.

STUDY PERIOD

Between Oct 2010 to Mar 2012

PLACE OF STUDY

Antenatal clinic, Department of Obstetrics & Gynaecology Kilpauk Medical College and Hospital.

SAMPLE SIZE

The sample size was calculated using the formula,

$$n = \frac{Z^2 \times P\,(1 - P)}{d^2}$$

Z - Constant (1.96).

P - Prevalence (0.05).

d - Desired precision (0.05).

$$n = \frac{(1.96)^2 \times 0.09 \times 0.91}{0.05 \times 0.05}$$

= 125

My sample size is 129.

INCLUSION CRITERIA

1) Pregnant women with singleton pregnancy
2) With LMP & USG confirmed pregnancy between 17-19weeks of gestation.

EXCLUSION CRITERIA

1) Diabetes mellitus
2) Chronic hypertension
3) Renal disorder
4) Hypothyroidism
5) Family/personal history of dyslipidemia
6) On Any medications except for vitamins & minerals
7) Ultra sound proved congenital anamolies
8) PCOS

METHODOLOGY

A total of 129 antenatal patients who attended the antenatal clinic of the Department of Obstetrics & Gyncecology, Govt Kilpauk Medical College, were selected based on the inclusion & exclusion criteria after obtaining their informed consent.

All selected women were subjected to a detailed history taking comprising of age, parity, prepregnant body weight, medication history, family history, medical history, detailed obstetric history including previous history of preeclampsia. Then they were subjected to clinical examination and routine laboratory investigations were carried out on the first day.

Fasting blood samples (4ml) were collected from these patients on the next day and subjected to lipid profile analysis. Total Cholesterol, HDL, TGL levels were estimated and the levels of LDL and VLDL were calculated indirectly.

These patients were regularly followed up in the antenatal op till their delivery once in every two weeks and a thorough clinical examination was carried with special focus on blood pressure and urine albumin. All the details were entered.

Definitions used for diagnosis of preeclampsia was according to the International Society for the study of Hypertension in pregnancy (ISSHP). Hypertension is defined as a systolic blood pressure ≥140 mm Hg or a diastolic blood pressure ≥90 mm Hg.

A rise in the systolic blood pressure of 30 mm Hg or a rise in the diastolic blood pressure of 15mm Hg, at least 4 hours apart or a single diastolic blood pressure >110mm Hg is also considered as hypertension with proteinuria of at least '1+' or 1 g/L on dipstick.

RESULTS OF THE STUDY

- The patients who developed pre-eclampsia were grouped as preeclampsia cohort.
- And the rest of the patients who remained normotensive till delivery were grouped as normal cohort.

The factors taken for analysis were age distribution, obstetric score, Body Mass Index, history of preeclampsia in previous pregnancy, and the components of lipid profile. Mean $\pm$ SD of all variables of interest were determined for preeclampsia cohort and for normal cohort separately and difference was tested by t test.

The predictive values of the individual components of lipid profile were analysed using ROC curve.

Logistic regression model was used to estimate the causal effect of each predisposing factor on outcome and to find out the most effective predictor.

TABLE – 1 : AGE DISTRIBUTION OF PATIENTS IN THE STUDY

AGE IN YEARS	PREECLAMPSIA COHORT		NORMAL COHORT	
	NO. OF CASES	%	NO. OF CASES	%
<24 YEARS	9	52.9	48	42.9
25-29 YEARS	7	41.2	51	45.5
>30 YEARS	1	5.9	13	11.6
TOTAL	17	100	112	100

Chi square test 0.848

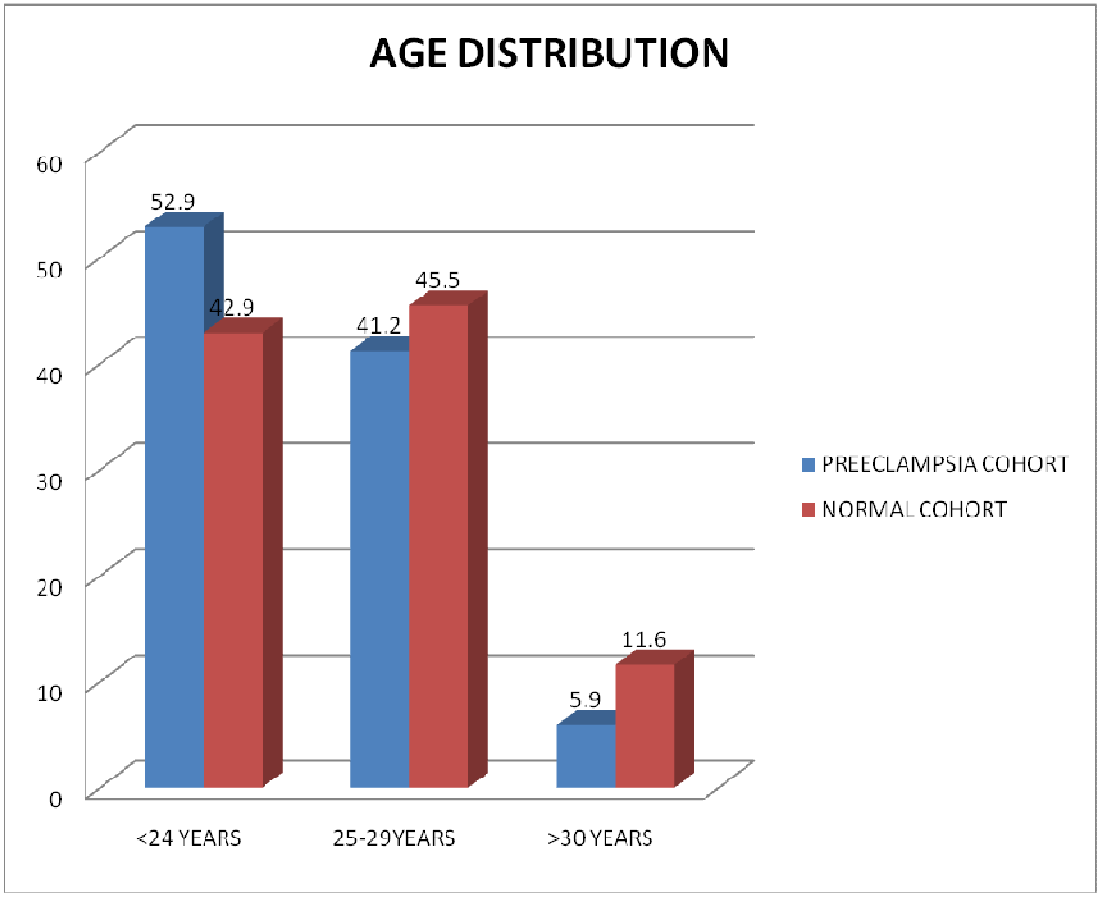
p=0.654

52.9% of the patients in preeclampsia cohort and 42.9% of the patients in the normal cohort were in the age group of < 24 years.

41.2% of the patients in preeclampsia cohort and 45.5% of the patients in the normal cohort were in the age group of 25-29 years.

5.9% of the patients in preeclampsia cohort and 11.6% of the patients in the normal cohort were in the age group of >30 years.

FIGURE 2 : AGE DISTRIBUTION OF PATIENTS IN THE STUDY



INFERENCE

There is no statistical significance between the preeclampsia cohort & normal cohort with respect to age distribution.

TABLE – 2 : OBSTETRIC SCORE OF PATIENTS IN THE STUDY

GRAVIDA	PREECLAMPSIA COHORT		NORMAL COHORT	
	NO. OF CASES	%	NO. OF CASES	%
PRIMI	10	58.8	72	64.3
GRAVIDA2	7	41.2	35	31.3
GRAVIDA3	0	0	5	4.5
TOTAL	17	100	112	100

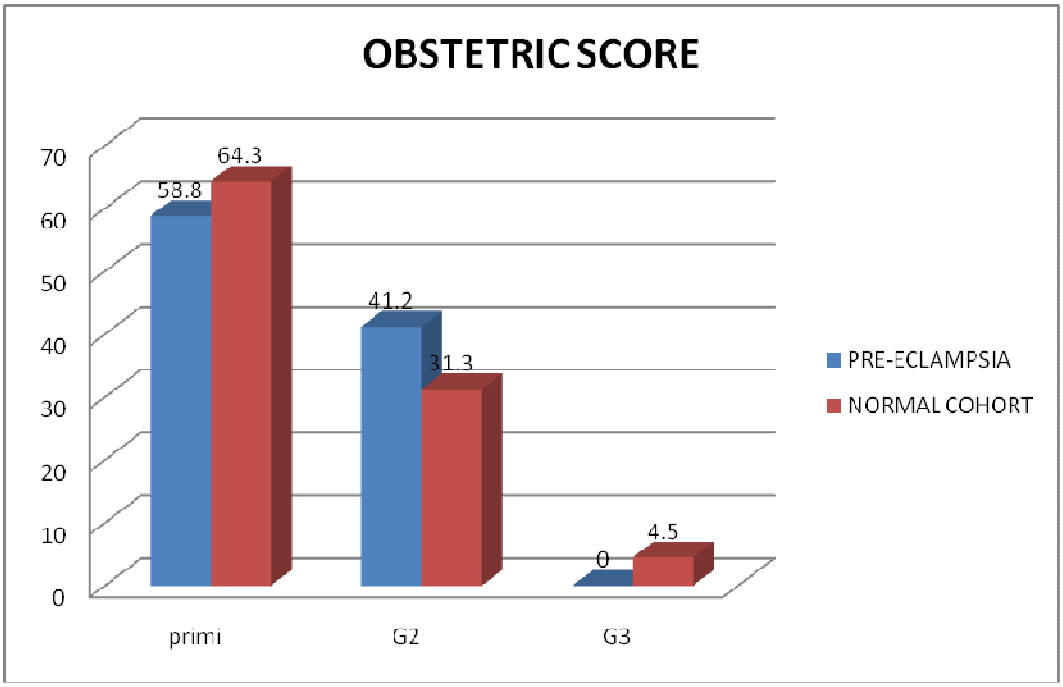
Chi square test = 1.275

p=0.529

58.8% of the patients in preeclampsia cohort and 64.3% of the patients in the normal cohort were primigravida.

No patients in preeclampsia cohort was gravida 3, while 4.5% of patients in normal cohort were gravid 3.

FIGURE 3 : OBSTETRIC SCORE OF PATIENTS IN THE STUDY



INFERENCE

There is no statistical significance between the preeclampsia cohort & normal cohort with respect to obstetric score.

TABLE-3 : SOCIO-ECONOMIC CLASSIFICATION OF PATIENTS IN THE STUDY

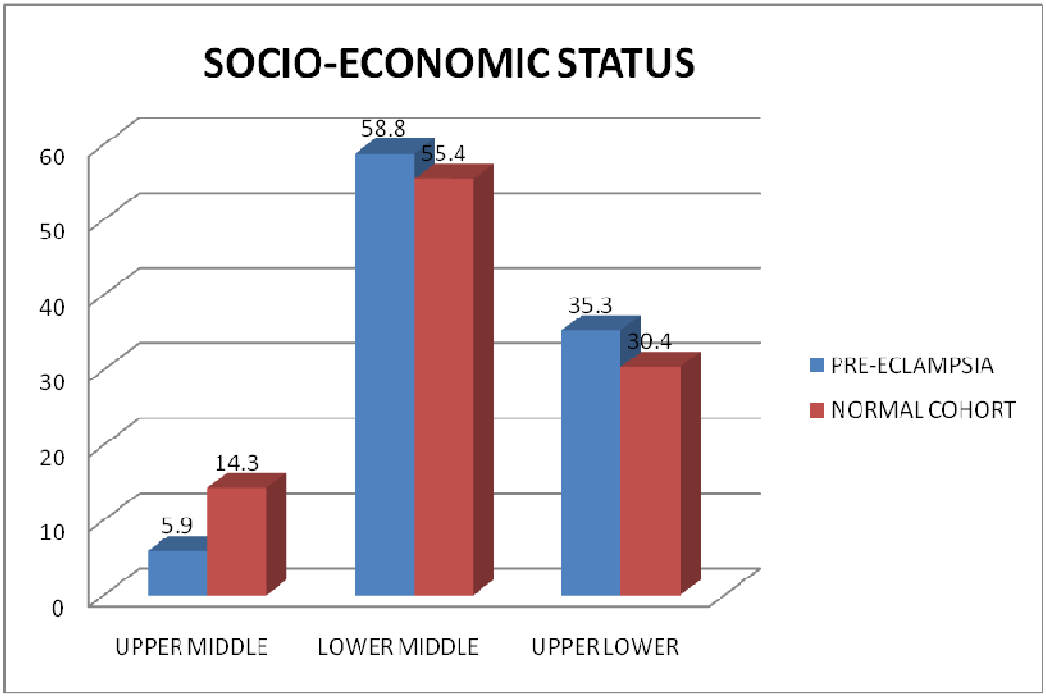
SOCIO-ECONOMIC CLASSIFICATION	PREECLAMPSIA COHORT		NORMAL COHORT	
	NO. OF CASES	%	NO. OF CASES	%
UPPER MIDDLE	1	5.9	16	14.3
LOWER MIDDLE	10	58.8	62	55.4
UPPER LOWER	6	35.3	34	30.4
TOTAL	17	100	112	100

Chi square test = 0.939

p=0.625

58.8% of the patients in preeclampsia cohort and 55.4% of the patients in the normal cohort belongs to lower middle group of socio economic classification according to Kuppuswamy classification.

FIGURE 4 : SOCIO-ECONOMIC CLASSIFICATION OF PATIENTS IN THE STUDY



INFERENCE

There is no statistical significance between the preeclampsia cohort & normal cohort with respect to socio economic classification.

TABLE – 4 : HISTORY OF PREECLAMPSIA IN PREVIOUS PREGNANCY IN PATIENTS IN THE STUDY

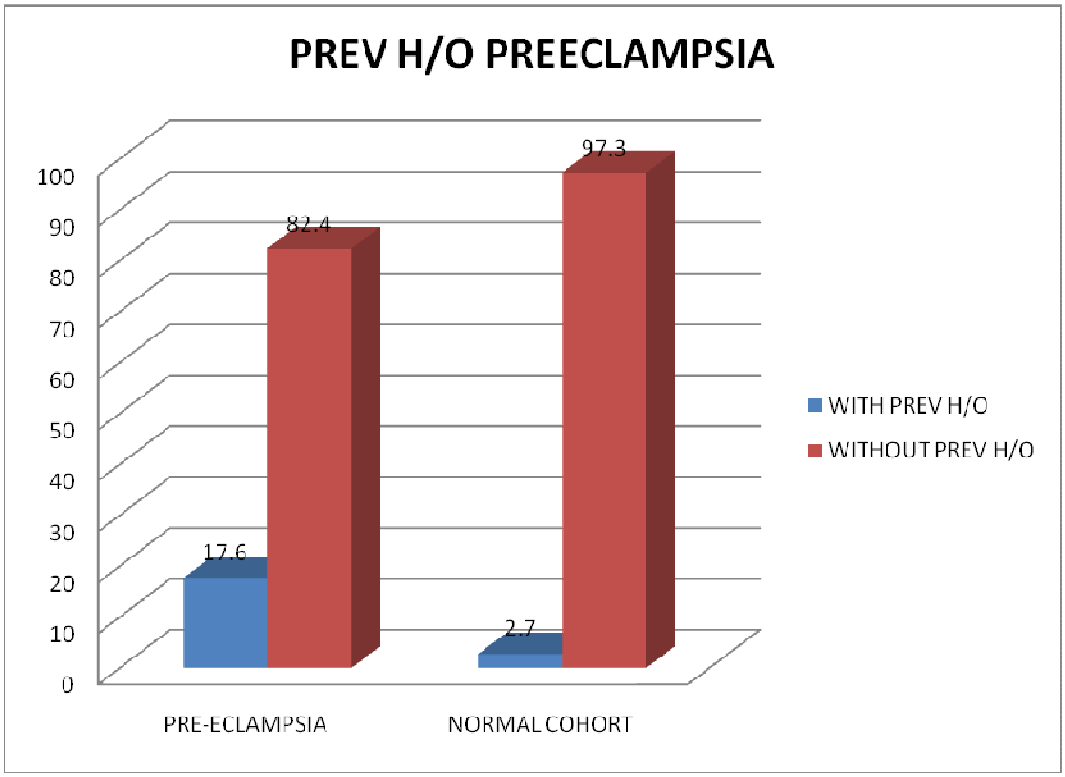
	PREECLAMPSIA COHORT		NORMAL COHORT	
	NO. OF CASES	%	NO. OF CASES	%
With prev H/O preeclampsia	3	17.6	3	2.7
Without prev H/O preeclampsia	14	82.4	109	97.3
TOTAL	17	100	112	100

Chi square test = 7.457

p = 0.06

17.6% of the patients in preeclampsia cohort and 2.7% of the patients in the normal cohort has previous history of preeclampsia .

FIGURE 5 : HISTORY OF PREECLAMPSIA IN PREVIOUS PREGNANCY IN PATIENTS IN THE STUDY



INFERENCE

The p value is 0.06 which shows that there exists a statistical significance between the two groups with respect to previous history of preeclampsia.

TABLE 5 : COMPARISON OF PRE-PREGNANT BMI BETWEEN PREECLAMPTIC & NORMAL COHORT

	NO. OF CASES	MEAN BMI	STANDARD DEVIATION	STANDARD ERROR OF MEAN
Preeclampsia cohort	17	27.32	3.65	0.88
Normal cohort	112	23.08	1.88	0.17

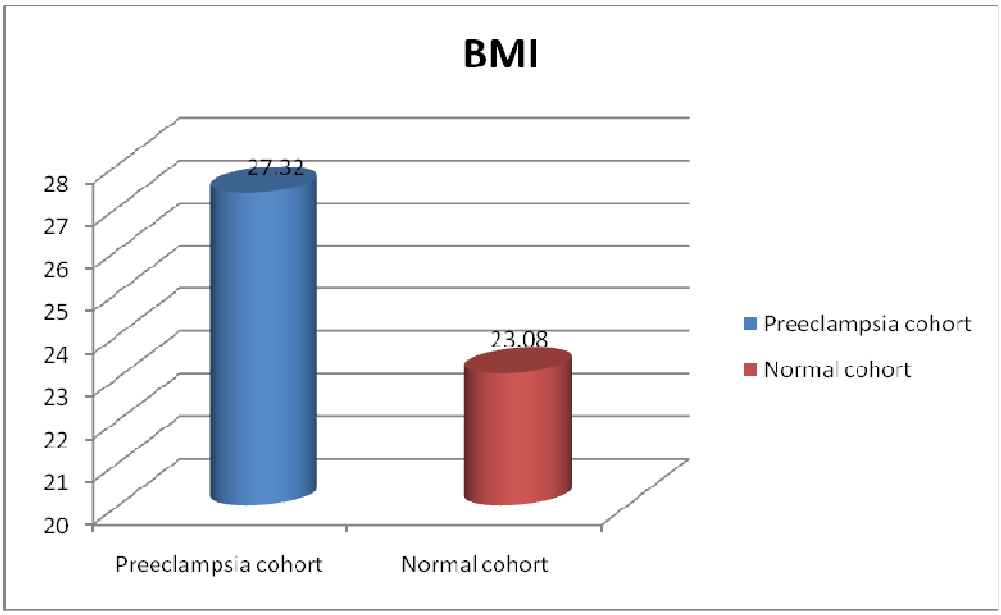
$p = 0.00$ which is < 0.01 therefore significant.

The Mean BMI of Preeclampsia cohort is 27.32kg/m^2.

The Mean BMI of Normal cohort is 23.08kg/m^2.

The Mean BMI of Preeclampsia cohort is 4.24kg/m^2 higher than the Normal cohort.

FIGURE 6: COMPARISON OF PRE-PREGNANT BMI BETWEEN PREECLAMPTIC & NORMAL COHORT



INFERENCE:

It is clear from the above table that the patients who developed preeclampsia had higher Body Mass Index during their prepregnant state than the nomotensive patients. The difference is statistically significant.

TABLE 6 : COMPARISON OF TOTAL CHOLESTEROL BETWEEN PREECLAMPTIC & NORMAL COHORT

	NO. OF CASES	MEAN (mg%)	STANDARD DEVIATION	STANDARD ERROR OF MEAN
Preeclampsia cohort	17	248.24	46.63	11.31
Normal cohort	112	176.10	28.02	2.64

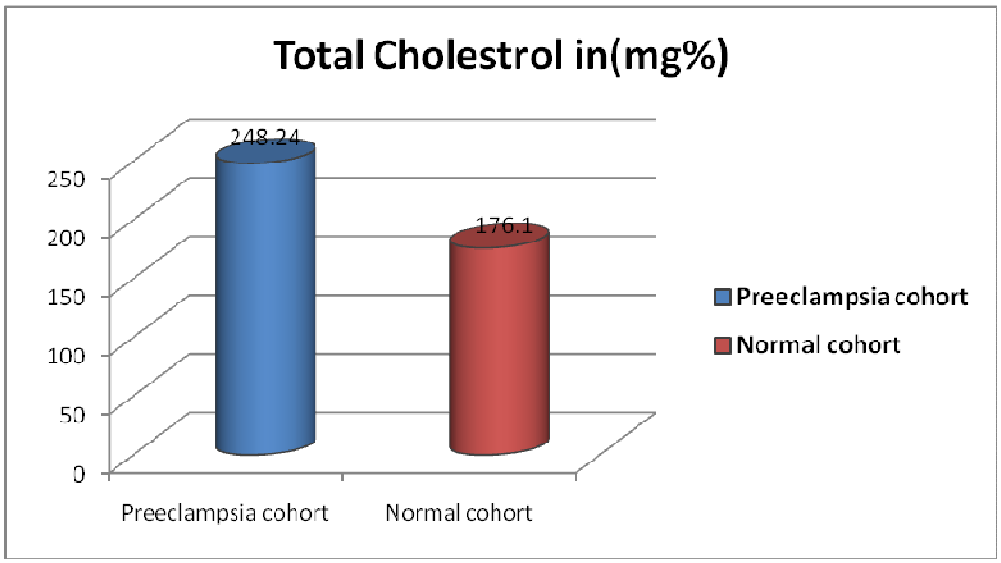
$p = 0.00$ which is < 0.01 therefore significant.

The Mean total Cholesterol of Preeclampsia cohort is 248.24mg%.

The Mean total Cholesterol of Normal cohort is 176.10mg%.

The Mean total Cholesterol of Preeclampsia cohort is 72.14mg% higher than the Normal cohort.

FIGURE 7 : COMPARISON OF TOTAL CHOLESTEROL BETWEEN PREECLAMPTIC & NORMAL COHORT



INFERENCE

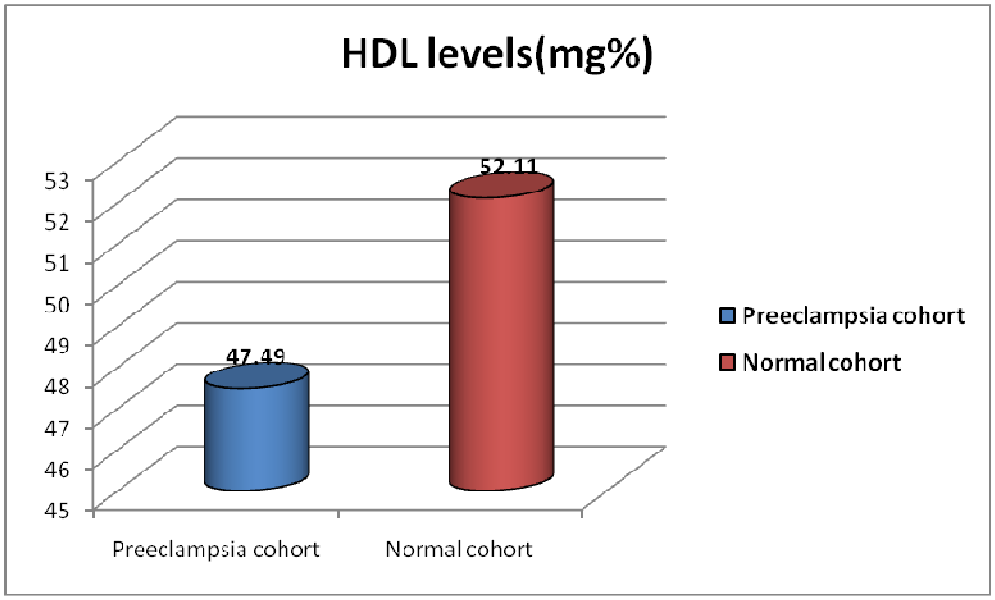
The mean total cholesterol levels were higher in patients who developed preeclampsia.

TABLE 7 : COMPARISON OF HDL LEVELS BETWEEN PREECLAMPTIC & NORMAL COHORT

	NO. OF CASES	MEAN (mg%)	STANDARD DEVIATION	STANDARD ERROR OF MEAN
Preeclampsia cohort	17	47.49	4.40	1.067
Normal cohort	112	52.11	8.918	0.843

p = 0.02 significant.

FIGURE 8 :COMPARISON OF HDL LEVELS BETWEEN PREECLAMPTIC & NORMAL COHORT



INFERENCE

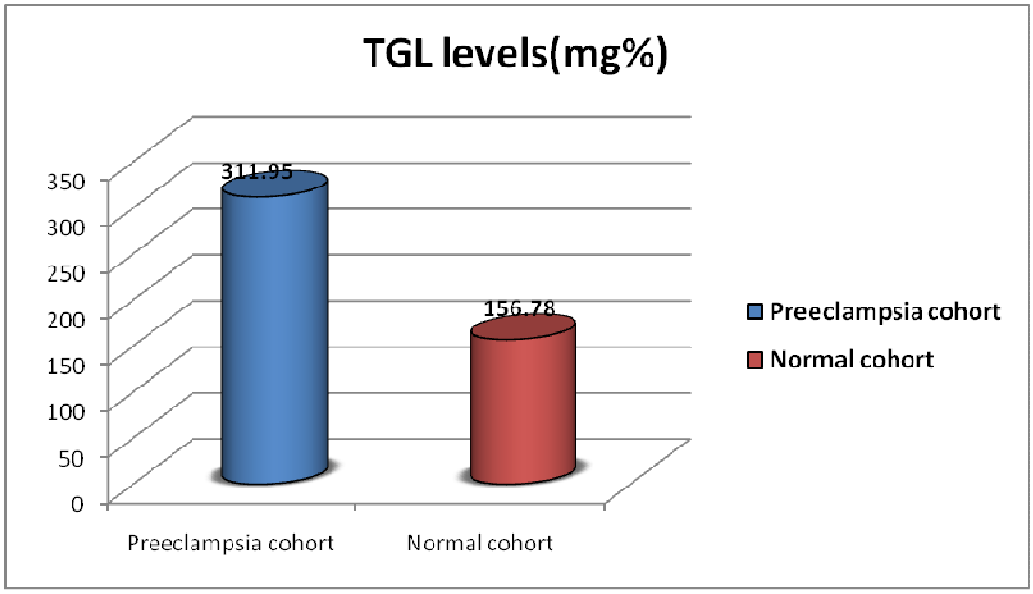
The mean HDL levels were lower in patients who developed preeclampsia.

TABLE 8 : COMPARISON OF TGL LEVELS BETWEEN PREECLAMPTIC & NORMAL COHORT

	NO. OF CASES	MEAN (mg%)	STANDARD DEVIATION	STANDARD ERROR OF MEAN
Preeclampsia cohort	17	311.95	82.334	19.96
Normal cohort	112	156.78	48.830	4.61

p = 0.00 which is < 0.01 therefore significant.

FIGURE 9 : COMPARISON OF TGL LEVELS BETWEEN PREECLAMPTIC & NORMAL COHORT



INFERENCE

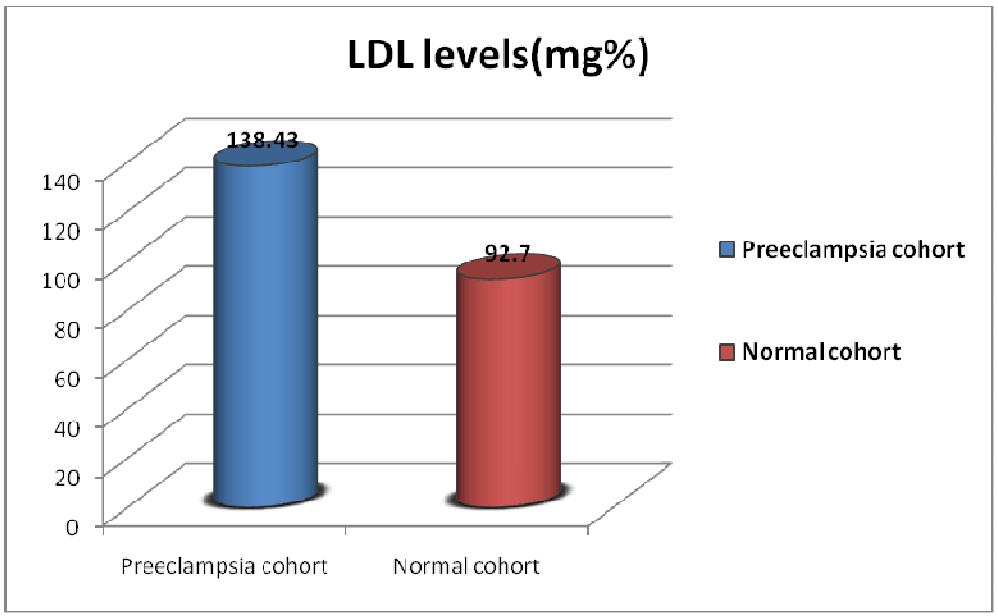
The mean TGL levels were higher in patients who developed preeclampsia.

TABLE 9 : COMPARISON OF LDL LEVELS BETWEEN PREECLAMPTIC & NORMAL COHORT

	NO. OF CASES	MEAN (mg%)	STANDARD DEVIATION	STANDARD ERROR OF MEAN
Preeclampsia cohort	17	138.43	38.92	9.44
Normal cohort	112	92.7	23.5	2.22

p = 0.00 which is < 0.01 therefore significant.

FIGURE 10 : COMPARISON OF LDL LEVELS BETWEEN PREECLAMPTIC & NORMAL COHORT



INFERENCE

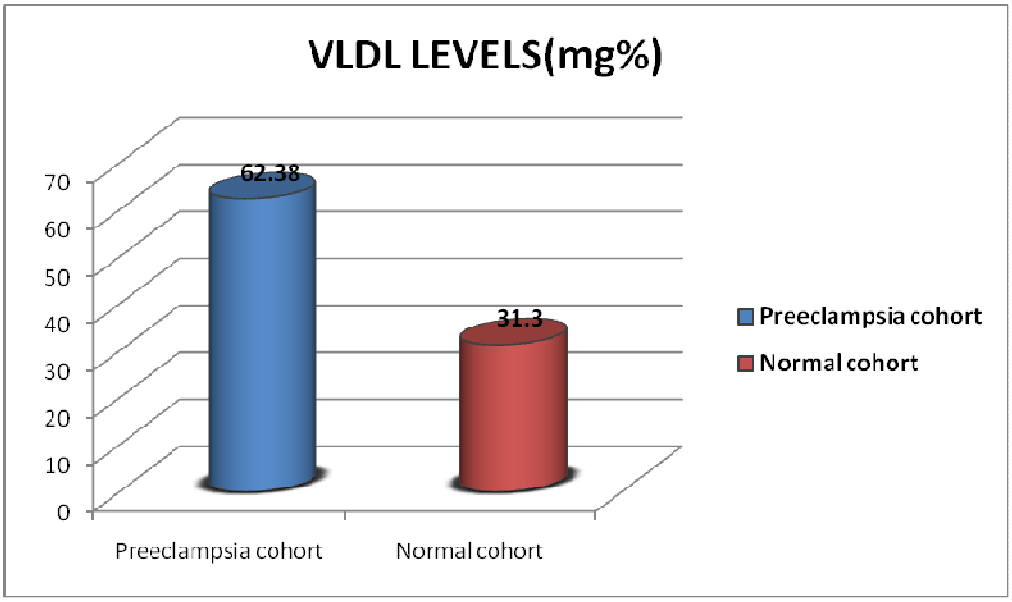
The mean LDL levels were higher in patients who developed preeclampsia.

TABLE 10 : COMPARISON OF VLDL LEVELS BETWEEN PREECLAMPTIC & NORMAL COHORT

	NO. OF CASES	MEAN (mg%)	STANDARD DEVIATION	STANDARD ERROR OF MEAN
Preeclampsia cohort	17	62.38	16.45	3.99
Normal cohort	112	31.3	9.74	0.92

$p = 0.00$ which is < 0.01 therefore significant.

FIGURE 11 : COMPARISON OF VLDL LEVELS BETWEEN PREECLAMPTIC & NORMAL COHORT



INFERENCE

The mean VLDL levels were higher in patients who developed preeclampsia.

TABLE 11 : MEAN VALUES OF BMI & TGL

	Mean	**Std. Deviation**	**N**
BMI	23.6436	2.61297	129
TGL	177.22	75.442	129

TABLE 12 : CORRELATION BETWEEN BMI AND TGL LEVELS

	BMI & TGL
Pearson Correlation	.517
Sig. (2-tailed)	.000

When the correlation co-efficient value is between 0 .*5* to 1.0 it means there is high correlation .The Pearson correlation coefficient for BMI & TGL is 0.517 which means there exists a high positive correlation between BMI & TGL.

TABLE 13 : MEAN VALUES OF BMI & TOTAL CHOLESTROL

	Mean	Std. Deviation	N
BMI	23.6436	2.61297	129
Total CHO	185.61	39.406	129

The p value is 0.000 which means it is statistically significant.

TABLE 14 : CORRELATION BETWEEN BMI AND TOTAL CHOLESTROL VALUES

	BMI & TCHO
Pearson Correlation	**.382**
Sig. (2-tailed)	**.000**

The p value is 0.000 which means it is statistically significant.

The pearson correlation coefficient for BMI & TCHO is 0.382 which means there exists a positive correlation between BMI & TGL, but since the co-efficient value is 0.382 , it is of medium coorelation.

All univariate analysis were done. The following variables were statistically significant.

1. Previous history of preeclampsia
2. Pre-pregnant Body Mass Index
3. Total Cholesterol Levels
4. HDL levels
5. TGL levels
6. VLDL levels
7. LDL levels

The above variables were put in to the Binary Logistic Regression Model taking development of preeclampsia as a dependent variable.

The two most statistically significant variables were

1. TGL levels
2. HDL levels

The outputs are

TABLE 15 : STATISTICAL SIGNIFICANCE OF HDL & TGL

Variable Name	Chi Square (Wald stat)	p
HDL	6.951	0.008
TGL	21.557	0.000

CLASSIFICATION TABLE

	OUTCOME OF THE STUDY		
	PATIENTS WHO REMAINED NORMOTENSIVE	**PATIENTS WHO DEVELOPED PREECLAMPSIA**	**PERCENTAGE CORRECTNESS**
MODEL PREDICTED NORMAL PATIENT	**110**	**2**	**98.2**
MODEL PREDICTED PRECLAMPSIA	**3**	**14**	**82.4**

OVERALL PERCENTAGE CORRECTNESS = 96.1 %

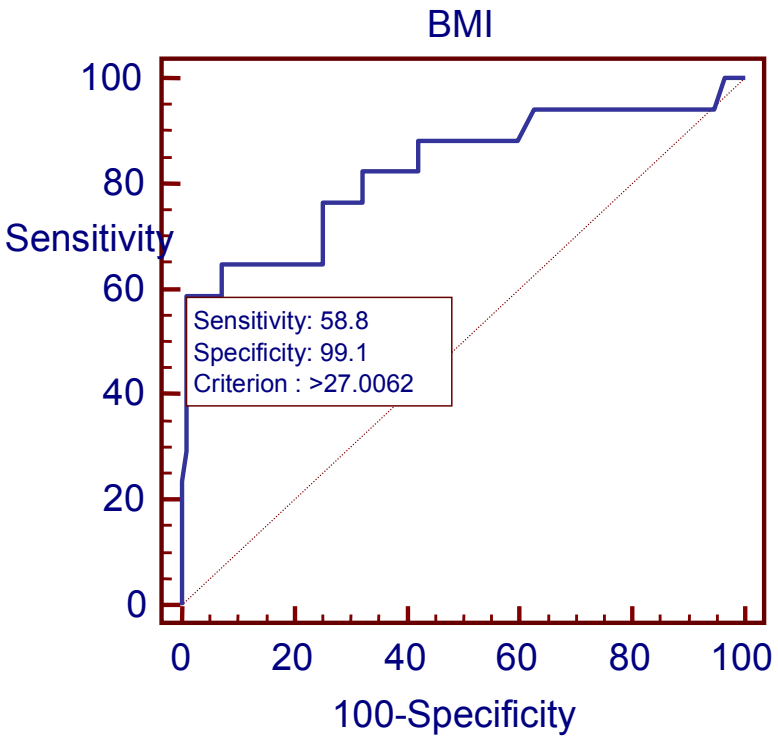
Receiver Operating Characteristic (ROC) Curve

This curve is used for clinical prediction rules. The accuracy of the test is determined from are under the curve.

If area under the curve is 0.90-1 then the accuracy is excellent , and if it is 0.80-0.90 then the accuracy is good .

Testing BMI as a predictor of preeclampsia

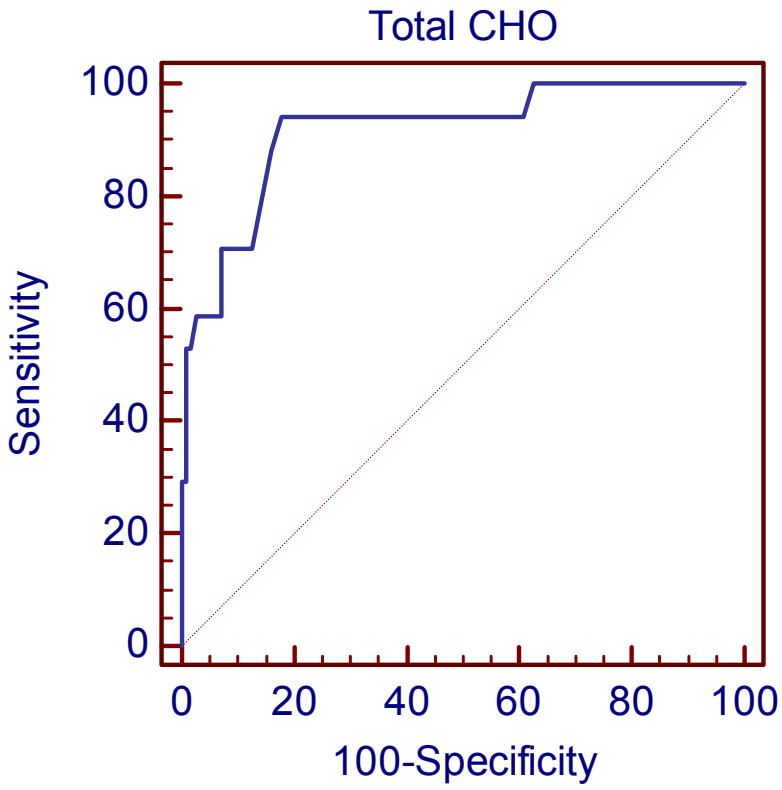
ROC curve for BMI



Area under the ROC curve for BMI is 0.828

Testing Total Cholesterol as a predictor,

The ROC curve for total Cholesterol



Area under the ROC curve for Total CHO is 0.91

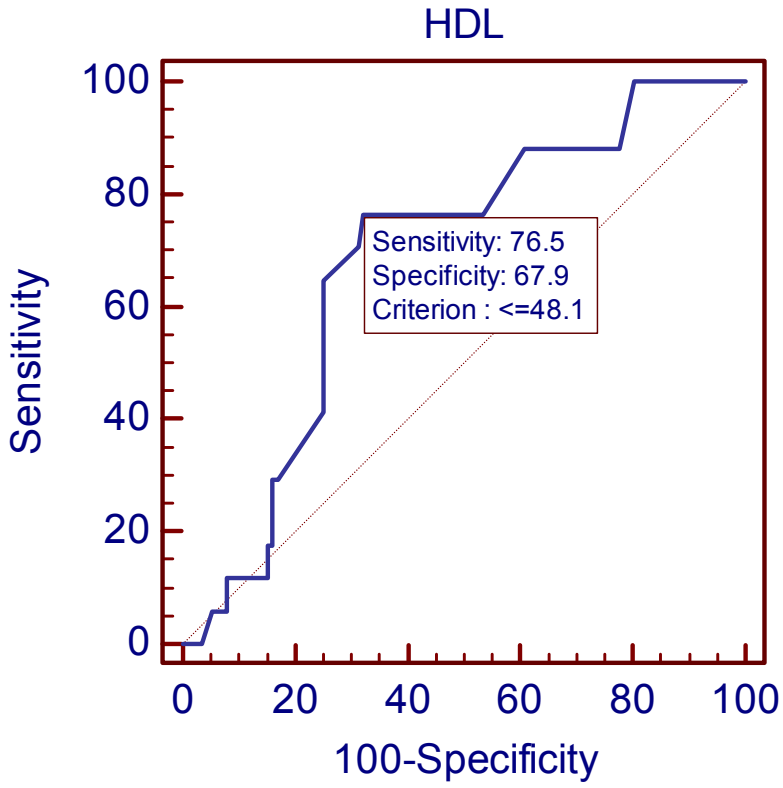
Sensitivity – 94.12 %

Specificity – 82.14%

Criterion >200 mg %

Testing HDL as a predictor,

The ROC curve for HDL



Area under the ROC curve for HDL is 0.68

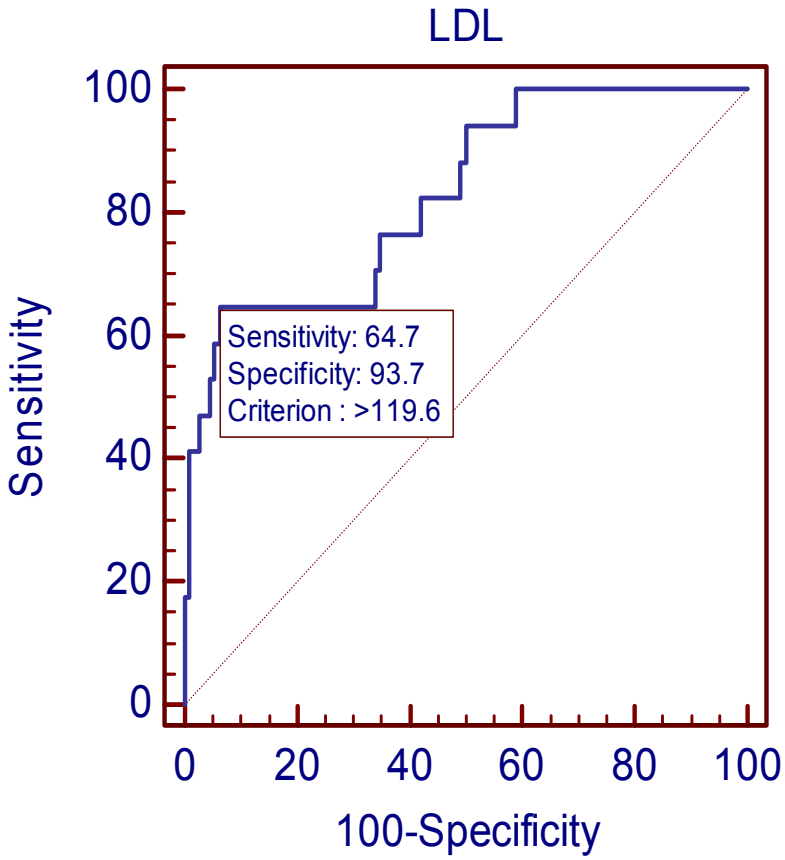
Sensitivity – 76.5%

Specificity – 67.9%

Criterion <48.1mg %

Testing LDL as a predictor ,

The ROC curve for LDL



Area under the ROC curve for LDL is 0.83

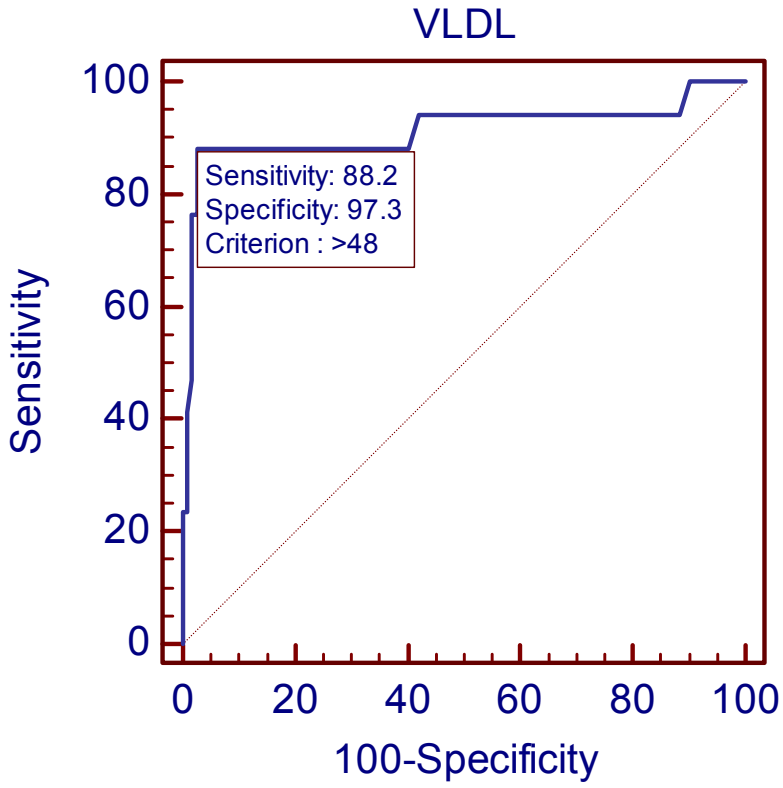
Sensitivity – 64.7%

Specificity 93.7%

Criterion >119.6mg %

Testing VLDL as a predictor ,

The ROC curve for VLDL



Area under the ROC curve for VLDL is 0.91

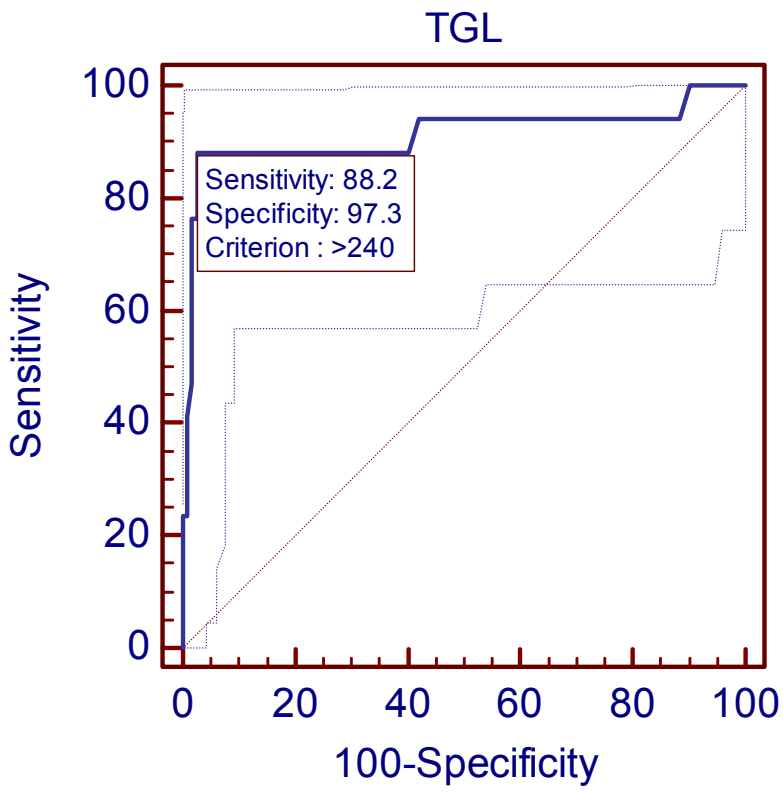
Sensitivity – 88.2%

Specificity – 97.3%

Criterion >48mg %

Testing TGL as a predictor ,

The ROC curve for TGL



Area under the ROC curve for TGL is 0.91

Sensitivity – 88.2%

Specificity – 97.3%

Criterion >240mg %

SUMMARY

There is no statistical significance between the preeclampsia cohort & normal cohort with respect to age distribution. 52.9% of the patients in preeclampsia cohort and 42.9% of the patients in the normal cohort were in the age group of < 24 years .41.2% of the patients in preeclampsia cohort and 45.5% of the patients in the normal cohort were in the age group of 25-29 years . 5.9% of the patients in preeclampsia cohort and 11.6% of the patients in the normal cohort were in the age group of >30 years.

- 58.8% of the patients in preeclampsia cohort and 64.3% of the patients in the normal cohort were primigravida.
- 58.8% of the patients in preeclampsia cohort and 55.4% of the patients in the normal cohort belongs to lower middle group of socio economic classification according to kuppuswamy classification.
- 17.6% of the patients in preeclampsia cohort and 2.7% of the patients in the normal cohort has previous history of preeclampsia .
- The Mean BMI of preeclampsia cohort was 27.32 while that of the normal cohort was 23.08. This clearly shows that the preeclampsia cohort had higher BMI than their normal counterparts.
- The mean total cholesterol levels were higher in patients who developed preeclampsia.

- The mean HDL levels were lower in patients who developed preeclampsia.
- The mean TGL levels were higher in patients who developed preeclampsia.
- The mean LDL levels were higher in patients who developed preeclampsia.
- The mean VLDL levels were higher in patients who developed preeclampsia.
- Area under the ROC curve for BMI is 0.828
- Testing Total Cholesterol as a predictor, the Sensitivity – 94.12 %,Specificity – 82.14%
- Area under the ROC curve for TGL is 0.91
- Area under the ROC curve for VLDL is 0.91 with Sensitivity – 88.2% & Specificity – 97.3%
- Area under the ROC curve for HDL is 0.68

DISCUSSION

Our study included 129 pregnant women; recruited from patients attending antenatal OP department.17 patients developed preeclampsia and were grouped as the preeclampsia cohort and the remaining 112 pregnant women who were normal were taken as controls. Thus the prevalence rate from this study is 13.18%

Our study similar to that by R.K.Vidyabati (2006) , shows that there is hypertriglyceridemia among the patients who subsequentally developed preeclampsia. Their study also showed that total cholesterol,LDL,VLDL levels were also higher in preeclampsia women which was similar to our study.

But the mean value of HDL for both the groups were similar in their study. In our study the level of HDL were lower in preeclampsia group.

Ray et al.(2006) showed that the mean triglyceride concentration were raised among preeclampsia cases than among normotensive pregnant women. Our study also had similar results. Does there exist a causal relationship?

The hypothesis that answers may be that linking insulin resistance and triglyceridemia as risk factors for preeclampsia.

Carl Hubel et al(1995) in their study showed that triglycerides and free fatty acids are raised in preeclampsia. Malondialdehyde, a lipid peroxidation metabolite causes endothelial dysfunction by its interaction. But the interaction or effects of this metabolite is not included in our study.

Ray et al(2006) showed that there exists a positive relation between higher BMI and increased risk of preeclampsia. In our study also the patients who subsequently developed preeclampsia had higher body mass index than the pregnant women who remained normotensive.

Higher body mass index, impaired (or) abnormal glucose tolerance and chronic hypertension the major features of metabolic syndrome are positively correlated with preeclamspsia in many studies.

The study by Barden et al (1999) shows irrespective of the obstetric score women with preeclampsia had higher BMI during pregnancy compared to normotensive pregnancies.

TGL levels were raised in preeclampsia women (than the normal rise during pregnancy) and after six weeks of delivery the TGL levels were decreased ; in Barden et al study.But in our study the concentration of TGL post pregnancy were not included.

Sattar et al (1997) showed that the increased level of cholesterol and TGL in preeclamptic women. In our study women who developed preeclampsia had a higher TGL even before its development.

Kaaja et al(1995) showed raised triglyceride and low HDL in preeclampsia women. In our study also the levels of total cholesterol, triglycerides , LDL,VLDL were raised in preeclamptic women. The levels of HDL were lower.

These abnormal lipid profile lead to endothelial cell dysfunction – a protean step in the pathogenesis of preeclampsia.

Increased F2 isoprostane was found in patients with increased LDL concentration , in a study by The National Heart Foundation of Australia. Our study did not address isoprostane F2. To establish an association between isoprostane F2 and preeclampsia more studies are required.

Similar to theirs our study also found a low HDL concentration in women with preeclampsia.

Bendomir studied the correlation between raised sub fraction of lipoprotein and new onset proteinuric hypertension in pregnancy.

In our study lipoprotein sub fraction were not studied. Further studies are required to determine the effects of lipoprotein sub fraction in pregnancy.

Anceshchi et al (1992) found altered ratio of cholesterol to phospholipids in the membrane of RBC in patients with preeclampsia. The studies of membrane phospholipids were not included in our study.

Our study similar to that by Kokia et al 1999 shows significant raised levels of TGL in preeclamptic women.

When variables with statistical significance were put in to binary logistic regression model, taking development of preeclampsia as dependent variable, TGL was most significant.

The ROC curves for total Cholestrol,HDL,VLDL,TGL,LDL for their role as predictors shows that TGL has maximum area under the curve , revealing that TGL is the most efficient predictor among the components of lipid profile .

Many studies shows that there exists correlation between obesity & TGL levels .Increase in BMI increases TGL levels which in turn puts the patient at risk for developing preeclampsia.

Thus, pre pregnant weight reduction and life style modification may help in reducing the occurrence of preeclampsia.

Further studies are necessary to establish the role of pre pregnant weight reduction and to find out the whether this would help in reducing the incidence of preeclampsia.

CONCLUSION

After analysing and comparing the results between the preeclampsia cohort and normal cohort it was concluded that

- The pre-pregnant BMI was higher among the patients who developed preeclampsia than normotensive counterparts.
- The study cohort had a higher Total cholesterol, Triglyceride, Low density Lipoprotein, Very Low Density Lipoprotein levels.
- The preeclampsia cohort also had a lower HDL level.
- Thus there exists a positive correlation between dyslipidemia and development of preeclampsia.
- Therefore abnormal lipid profile before 20 weeks is a very good predictor of preeclampsia development.

Thus it may be concluded that, detecting dyslipidemia before 20 weeks of gestation would help us to recognise pregnancies at high risk for preeclampsia even before the clinical syndrome.

Early recognition ,would help us in offering better surveillance to detect and treat the disease earlier for a better maternal and perinatal outcome.

Given that the raised triglyceridemia as a feature of the insulin resistance syndrome; interventional studies are required to find out whether pre-pregnancy weight reduction can lower the risk of preeclampsia.

The determination of insulin levels, inflammatory markers in early pregnancy and then followed by a thorough assessment of the outcome through a large cohort study, may help in addressing the role of metabolic syndrome in causation of preeclampsia.

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KEY TO MASTER CHART

S.No	-	Serial Number
SEC	-	Socio Economic Status
Prev H/O pre eclampsia	-	Pre eclampsia in past pregnancy
BMI	-	Body Mass Index
TC	-	Total Cholesterol
HDL	-	High Density Lipoproteins
LDL	-	Low Density Lipoproteins
VLDL	-	Very Low Density Lipoproteins
TGL	-	Triglycerides

PROFORMA

S.no

Name

Age

Obstetric Score

Booking Status

Socio Economic Status

LMP

EDD

Menstrual History

Obstetric History

H/o preeclampsia in Previous Pregnancy

Family History

Any H/o DM/HT/ Bronchial Asthma / Cardiac / Thyriod Disease/Renal disease.

Pre-pregnant weight

General Examination

Height Weight

Anaemia Jaundice Pedal edema JVP

Vitals

PR: BP: RR:

Systemic examination :

Respiratory System.

Cardiovascular System.

CNS :

Obstetric Examination

Investigation :

Albumin

Urine Sugar

Deposits

VDRL

NVP

Blood grouping & typing

CBC

Blood sugar

Sr . Creatinine

Serum Lipid Profile

Obstetric Ultrasound.

MASTER CHART

S.No	Name	Age	Parity	LMP	EDD	SE Status	Prev H/o GHT	Ht	Wt	BMI	Total CHO	HDL	TGL	LDL	VLDL	GHT/preEclampsia
1	Selvi	30	primi	11/5/2010	8/12/2011	4	-	152	68	29.43213296	298	46.2	392.9	173.56	78.44	yes
2	Solaiyammal	26	primi	10/21/2010	7/28/2011	4	-	143	62	30.31933102	278	56	386.2	144.26	77.24	yes
3	sivagami	17	primi	10/20/2010	7/27/2011	4	-	159	76	30.06210197	208	46	303.2	101.4	60.64	yes
4	Esther	21	primi	11/15/2010	8/22/2011	3	-	148	68	31.04455807	211	46.2	314.6	101.88	62.92	yes
5	bindu	28	G2P1L0	11/23/2010	8/30/2011	5	yes	152	65	28.13365651	201	46.2	302.7	94.26	60.54	yes
6	keerthana	23	G2P1L1	11/9/2010	8/16/2011	5	no	145	65	30.91557669	259	47.6	393.9	132.62	78.78	yes
7	valliyammal	21	primi	11/1/2010	8/8/2011	4	-	141	65	32.69453247	302	46	292	197.82	58.4	yes
8	suryakumari	24	G2P1L1	11/7/2010	8/14/2011	5	no	148	52	23.73995617	288	52	386	158	77.2	yes
9	latha	28	G2P1L1	11/14/2010	8/21/2011	4	NO	148	69	31.50109569	276	45.6	394.8	151	78.96	yes
10	nadiya	27	G2P1L1	11/9/2010	8/16/2011	4	no	150	55	24.44444444	202	44.3	299.2	97.85	59.84	yes
11	lakshmi	21	primi	12/7/2010	9/13/2011	4	-	148	55	25.10956903	240	48.1	276.9	138.2	55.38	yes
12	gracy	22	G2P1L1	12/21/2010	9/27/2011	5	yes	160	62	24.21875	278	45.6	395.6	154.12	79.12	yes
13	sarala	19	primi	11/30/2010	9/6/2011	5	No	140	45	22.95918367	165	52	92	94.6	18.4	yes
14	samundeeshwari	27	G2P1L1	12/5/2010	9/11/2011	4	yes	149	44	19.81892708	202	39	172	128.6	34.4	yes
15	kanimozhi	25	primi	11/23/2010	8/30/2011	4	No	155	59	24.55775234	202	56	300.3	85.94	60.06	yes
16	reshmi	26	primi	11/15/2010	8/22/2011	4	No	155	68	28.30385016	298	42.5	302.8	194.94	60.56	yes
17	swapna	21	primi	11/12/2010	8/19/2011	5	No	158	68	27.23922448	312	48	298	204.4	59.6	yes

MASTER CHART

S.No.	Name	Age	Parity	LMP	EDD	SE Status	Prev H/o GHT	Ht	Wt	BMI	Total CHO	HDL	TGL	LDL	VLDL	GHT/pre Eclampsia
1	Sarasvathy	26	G2P1L1	10/21/2010	7/28/2011	4	yes	143	48	24	140	45	88	77.4	17.6	No
2	Kumudha	28	primi	10/30/2010	8/6/2011	3	-	152	56	25	160	49	93	92.5	18.6	No
3	gayathri	28	primi	10/29/2010	8/5/2011	5	-	147	52	24.06404739	175	52	101	105.8	20.5	No
4	sumathi	28	G2P1L1	10/20/2010	7/27/2011	5	-	143	62	30.31933102	136	55	137	53.9	27.4	No
5	Chithra	27	G2P1L1	10/8/2010	7/15/2011	5	No	141	40	20.11971229	152	40	80	94.4	17.6	No
6	venda	21	G2P1L1	10/13/2010	7/20/2011	5	No	147	43	19.89911611	155	34	80	105	16	No
7	Mafagadham	32	G3P2L2	11/4/2010	8/11/2011	5	No	151	42	18.42024473	148	43	130	79	26	No
8	sakunthala	30	G2P1L1	11/2/2010	8/9/2011	4	No	149	54	24.32322868	163	37	142	95.6	28.4	No
9	jayanthi	22	primi	10/14/2010	7/21/2011	5	-	145	52	24.73246136	158	50	189	100.2	37.8	No
10	mahesh	25	primi	10/24/2010	7/31/2011	3	-	159	55	21.75546853	201	53	148	118.4	29.6	No
11	priya	24	G2P1L1	11/4/2010	8/11/2011	3	-	144	56	27.00617284	136	55	136	53.8	27.2	No
12	karthiga	22	G2P1L1	10/24/2010	7/31/2011	5	no	140	44	22.44897959	146	44	148	72.4	29.6	No
13	ammu	30	G2P1L1	11/12/2010	8/19/2011	4	no	144	49	23.63040123	147	49	168	64.4	33.6	No
14	brinda	26	primi	11/6/2010	8/13/2011	5	-	152	58	25.10387812	198	49	203	108.4	40.6	No
15	Devi	27	G2P1L1	10/30/2010	8/6/2011	5	NO	147	50	23.1385071	166	42	158	84.4	31.6	No
16	Poornima	25	primi	10/20/2010	7/27/2011	4	-	151	47	20.613131	185	52	179	97.2	35.8	No
17	nagajothi	22	primi	11/1/2010	8/8/2011	4	-	153	48	20.504934	167	46	94	102	18.8	No
18	chithra	23	primi	11/2/2010	8/9/2011	4	-	141	46	23.13766913	171	50	185	84	37	No
19	darshni	24	G2P1L1	11/5/2010	8/12/2011	3	No	149	44	19.81892708	168	46	185	85	37	No
20	deepa	25	primi	10/27/2010	8/3/2011	5	-	157	62	25.15315023	156	46	192	71.6	38.4	No
21	durga	22	G2P1L1	11/4/2010	8/11/2011	5	no	153	58	24.77679525	203	48	240	107	48	No
22	pavithra	26	primi	11/16/2010	8/23/2011	3	-	148	53	24.19649379	168	46	188	84	37.6	No
23	komala	27	primi	11/5/2010	8/12/2011	4	-	142	47	23.30886729	206	50	195	117	39	No
24	thilaga	23	primi	11/20/2010	8/27/2011	4	-	158	54	21.63114885	184	76	183	71.4	36.6	No
25	nandini	25	G2P1L1	11/27/2010	9/3/2011	4	-	146	51	23.92568962	202	52	200	110	40	No
26	shobana	21	primi	11/24/2010	8/31/2011	5	-	154	50	21.08281329	192	52	165	107	33	No
27	JOTHI	25	primi	11/9/2010	8/16/2011	4	-	158	54	21.63114885	198	50	162	115	32.4	No
28	suganthi	26	primi	11/1/2010	8/8/2011	5	-	140	45	22.95918367	196	48	194	106	38.8	No

29	lavanya	25	G2P1L1	11/18/2010	8/25/2011	4	No	147	43	19.89911611	163	38	142	96.6	28.4	No
30	prabavathy	23	primi	11/13/2010	8/20/2011	5	-	149	54	24.32322868	198	52	154	115.2	30.8	No
31	meena	25	primi	11/6/2010	8/13/2011	5	-	154	50	21.08281329	178	46	169	98.2	33.8	No
32	jenifer	24	G2P1L1	11/25/2010	9/1/2011	4	no	149	54	24.32322868	203	52.3	206	129.56	41.2	No
33	ramajyothi	22	G2P1L1	12/1/2010	9/7/2011	4	no	157	54	21.90758246	200	50.8	152.4	119.2	30.48	No
34	deepa	19	primi	11/18/2010	8/25/2011	4	-	160	55	21.484375	239	62.9	387.6	139.2	77.52	No
35	philomeena	30	G2P1L1	12/4/2010	9/10/2011	3	No	153	58	24.77679525	221	75.3	222.2	101.76	44.44	No
36	mahalaksmi	22	primi	12/8/2010	9/14/2011	4	-	145	48	22.82996433	204	48.1	191.9	118	38.38	No
37	kala	26	G2P1L1	11/19/2010	8/26/2011	5	NO	152	48	20.77562327	181	80.3	181	64.7	36.2	No
38	RAMADEVI	25	primi	11/26/2010	9/2/2011	4	-	159	54	21.35991456	240	65.1	137.1	144.88	27.42	No
39	sharmila	20	primi	12/4/2010	9/10/2011	4	-	149	49	22.07107788	205	66.5	103.9	118.22	20.78	No
40	devi	30	G3P2L2	12/5/2010	9/11/2011	3	no	159	54	21.35991456	284	63.8	165.4	188.2	33.08	No
41	gomathi	33	G3P2L2	11/30/2010	9/6/2011	4	no	156	51	20.9566075	193	60.5	156.8	101	31.36	No
42	jansirani	28	G2P1L1	12/4/2010	9/10/2011	4	no	142	47	23.30886729	231	78.4	297.1	93.65	59.42	No

S.No	Name	Age	Parity	LMP	EDD	SE Status	Prev H/o GHT	Ht	Wt	BMI	Total CHO	HDL	TGL	LDL	VLDL	GHT/pr eEclamp sia
43	backialakshmi	32	primi	11/19/2010	8/26/2011	5	-	149	54	24.32322868	237	49.5	159.3	55.84	31.86	No
44	gomathi	28	primi	11/27/2010	9/3/2011	3	-	146	51	23.92568962	155	75.8	85.5	62.8	17.1	No
45	sabitha	22	primi	11/20/2010	8/27/2011	3	-	141	46	23.13766913	140	52.4	132.9	61.92	26.58	No
46	pushpa	22	G2P1L1	12/10/2010	9/16/2011	4	no	154	50	21.08281329	172	49.9	185.2	85.76	37.04	No
47	muthulakshmi	30	G3P2L2	11/25/2010	9/1/2011	4	No	152	47	20.34279778	145	46	92	80.6	18.4	No
48	sasikala	22	G2P1L1	11/21/2010	8/28/2011	4	yes	157	52	21.09619051	202	55	200	107	40	No
49	nirmala	18	primi	12/9/2010	9/15/2011	5	No	148	53	24.19649379	175	43	101	111.8	20.2	No
50	rukmani	22	primi	11/20/2010	8/27/2011	3	No	141	46	23.13766913	142	52	132	63.6	26.4	No
51	sarojini	30	primi	12/11/2011	9/16/2012	4	No	147	42	19.43634597	156	42	85	97	17	No
52	pushpa	22	primi	11/18/2010	8/25/2011	3	No	151	56	24.5603263	168	46	172	87.6	34.4	No
53	kasiyammal	22	primi	11/21/2010	8/28/2011	4	No	149	54	24.32322868	156	34	72	107.6	14.4	No
54	suseela	32	G2P1L1	12/15/2010	9/21/2011	4	No	159	54	21.35991456	143	46	126	71.8	25.2	No
55	mariyammal	38	G2P1L1	12/9/2011	9/14/2012	5	yes	152	57	24.67105263	166	39	146.5	97.7	29.3	No
56	karpagam	27	primi	11/26/2010	9/2/2011	4	No	143	48	23.47303047	188	45.9	183	105.5	36.6	No
57	shankari	22	primi	11/19/2010	8/26/2011	3	No	154	59	24.87771968	198	55	142.5	144.5	28.5	No
58	sudha	27	primi	11/30/2010	9/6/2011	5	No	140	45	22.95918367	201	56	208.6	103.2	41.72	No
59	pramila	23	G3P2L2	11/27/2010	9/3/2011	4	No	160	55	21.484375	147	48	99.8	79.03	19.96	No
60	manimala	25	G2P1L1	12/8/2010	9/14/2011	4	No	153	48	20.504934	145	58	88.2	69.36	17.64	No
61	bishnu	30	G2P1L1	12/15/2010	9/21/2011	4	No	146	51	23.92568962	196	52	202.3	103.5	40.46	No
62	SELVI	22	primi	12/22/2010	9/28/2011	5	No	142	47	23.30886729	165	48	136	89.8	27.2	No
63	BHUVANA	22	primi	12/10/2010	9/16/2011	5	No	152	57	24.67105263	188	54	184	97.2	36.8	No
64	MANJU	27	primi	11/27/2010	9/3/2011	4	No	148	43	19.6311176	164	48	92	97.6	18.4	No
65	MARAGADHAM	26	primi	12/19/2010	9/25/2011	3	No	153	58	24.77679525	143	56	82.6	70.48	16.52	No
66	SHAKUNTHALA	27	G2P1L1	12/13/2010	9/19/2011	4	No	141	46	23.13766913	172	53	186	81.8	37.2	No
67	SHAKILA	26	primi	11/30/2010	9/6/2011	4	No	159	54	21.35991456	165	44	172	86.6	34.4	No
68	SHAFREEN	28	primi	11/25/2010	9/1/2011	4	No	151	56	24.5603263	152	48	176	68.8	35.2	No
69	JANANI	21	primi	12/5/2010	9/11/2011	4	No	149	54	24.32322868	162	43	108.6	97.28	21.72	No
70	yuvashree	28	primi	12/9/2010	9/15/2011	4	No	144	49	23.63040123	246	49	303.2	136.36	60.64	No
71	jamrath	28	primi	12/15/2010	9/21/2011	4	No	155	50	20.81165453	182	76	182.5	68.5	36.5	No
72	suganya	25	G2P1L1	12/7/2010	9/13/2011	3	No	157	52	21.09619051	185	65	176.3	84.74	35.26	No

73	indira	23	primi	12/10/2010	9/16/2011	5	no	148	43	19.6311176	203	53	182	113.6	36.4	No
74	Kasthuri	22	G2P1L1	12/6/2010	9/12/2011	4	No	160	55	21.484375	172	48	163	91.4	32.6	No
75	Jeyachitra	31	primi	12/30/2010	10/6/2011	5	No	151	56	24.5603263	192	51	163	108.4	32.6	No
76	Rani	28	G2P1L1	12/7/2010	9/13/2011	4	No	156	51	20.9566075	193	48.5	178.5	108.74	35.7	No
77	Ranjani	27	primi	12/8/2010	9/14/2011	4	No	149	44	19.81892708	186.3	52.2	106.8	109.74	21.36	No
78	Singari	22	primi	12/20/2010	9/26/2011	4	No	158	53	21.23057202	188	39	175.6	113.8	35.12	No
79	Nirmala	27	primi	12/12/2010	9/18/2011	4	No	143	48	23.47303047	200	51.3	146.2	119.6	29.24	No
80	Vijayalakshmi	27	primi	12/7/2010	9/13/2011	4	No	151	56	24.5603263	236	60.8	178.9	139.4	35.78	No
81	Gnaneshwari	29	primi	12/15/2010	9/21/2011	3	No	148	53	24.19649379	173	50	186.4	85.72	37.28	No
82	vandana	21	primi	12/5/2010	9/11/2011	4	No	142	47	23.30886729	142	52.6	136.9	62.02	27.38	No
83	madhangi	22	primi	12/7/2010	9/13/2011	5	No	140	45	22.95918367	154	65.1	88.7	71.16	17.74	No
84	Akila	23	primi	12/10/2010	9/16/2011	3	No	142	47	23.30886729	134	50.2	142.6	55.28	28.52	No
85	rajalakshmi	28	G2P1L1	12/5/2010	9/11/2011	4	No	154	59	24.87771968	193	59.6	88.7	115.6	17.74	No
86	Sindu	25	primi	10/22/2010	7/29/2011	4	No	146	54	25.33308313	140	44	96	76.8	19.2	No
87	amudha	24	primi	10/31/2010	8/7/2011	4	No	142	46	22.81293394	166	52	99	94.2	19.8	No

S.No	Name	Age	Parity	LMP	EDD	SE Status	Prev H/o GHT	Ht	Wt	BMI	Total CHO	HDL	TGL	LDL	VLDL	GHT/preEclampsia
88	lakshminarayani	28	primi	11/4/2010	8/11/2011	5	No	156	59	24.24391847	200	59	201	100.8	40.2	No
89	aparna	26	G2P1L1	10/27/2010	8/3/2011	5	No	154	60	25.29937595	176	55	105	100	21	No
90	arthi	23	primi	11/3/2010	8/10/2011	4	No	158	58	23.23345618	202	54	196	108.8	39.2	No
91	nadhiya	24	primi	11/5/2010	8/12/2011	5	No	152	53	22.93975069	136	49	136.6	59.68	27.32	No
92	devaki	20	primi	10/23/2010	7/30/2011	5	No	158	62	24.8357635	143	43	152	69.6	30.4	No
93	sathya	25	primi	11/17/2010	8/24/2011	5	No	160	65	25.390625	146	56	163.5	57.3	32.7	No
94	kavitha	22	primi	11/13/2010	8/20/2011	4	No	162	65	24.76756592	132	55	202.2	36.56	40.44	No
95	ushadevi	23	G2P1L1	10/29/2010	8/5/2011	4	No	157	59	23.93606231	162	46	185	79	37	No
96	Mari	22	primi	11/4/2010	8/11/2011	4	No	153	53	22.64086462	176	58	186	80.8	37.2	No
97	ganga	21	primi	10/26/2010	8/2/2011	5	no	158	62	24.8357635	154.2	48.6	132.2	79.16	26.44	No
98	ponni	25	G2P1L1	11/9/2010	8/16/2011	4	no	156	55	22.60026298	158.6	49.6	202.6	68.48	40.52	No
99	manimegalai	27	primi	11/14/2010	8/21/2011	4	no	157	58	23.53036634	159.6	48.6	182	74.6	36.4	No
##	roopa	28	G2P1L1	11/16/2010	8/23/2011	4	no	160	63	24.609375	184.5	49.6	172.4	100.42	34.48	No
##	Ramya	26	primi	11/19/2010	8/26/2011	4	no	158	58	23.23345618	174	65	146	79.8	29.2	No
##	nisha	21	primi	11/1/2010	8/8/2011	4	no	149	58	26.12494933	172.5	52.5	148	90.3	29.6	No
##	sangeeta	25	G2P1L1	11/8/2010	8/15/2011	4	NO	151	54	23.68317179	146.8	52.4	169.6	60.48	33.92	No
##	viji	24	primi	11/25/2010	9/1/2011	4	NO	153	56	23.922423	184	49	146	105.8	29.2	No
##	sheeli	23	primi	11/3/2010	8/10/2011	4	No	158	60	24.03460984	184.6	49.5	184	98.3	36.8	No
##	sabeena	25	primi	11/13/2010	8/20/2011	5	No	154	56	23.61275089	162	44	126.5	92.7	25.3	No
##	angelin	25	primi	12/4/2010	9/10/2011	5	No	156	62	25.47666009	180	62.5	146	88.7	29.2	No
##	thulasi	22	primi	12/8/2010	9/14/2011	4	No	152	54	23.37257618	202	65	105.6	115.88	21.12	No
##	vallikannu	24	primi	11/18/2010	8/25/2011	4	no	159	54	21.35991456	145	65.2	82.5	63.3	16.5	No
##	sakthi	28	primi	11/22/2010	8/29/2011	4	no	161	65	25.07619305	142.2	52.6	135	62.599	27	No
##	vidya	23	primi	12/12/2010	9/18/2011	5	no	148	54	24.65303141	168.6	52.6	186.4	78.72	37.28	No
##	sivakumari	25	G2P1L1	12/15/2010	9/21/2011	4	no	149	54	24.32322868	186.5	48.6	192	99.5	38.4	No

ETHICAL COMMITTEE CERTIFICATE

ETHICAL COMMITTEE
GOVT. KILPAUK MEDICAL COLLEGE, KILPAUK,
CHENNAI- 10.
Venue: PANAGAL HALL, KMC
Dt: 01.02.2011

CHAIRPERSON
Prof. Dr.V.KANAGASABAI, MD.,
Dean
Govt. Kilpauk Medical College, Chennai-10
Sub: Ethical Committee project work - approved – regarding.
Ref: Lr.No.3944/Audit/E1/09 Dt. 30.11.2010

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With above reference, the Institutional Ethical committee meeting for the following students was conducted at our Institution on 01.02.2011.

S.NO.	Name	Topic
1.	Dr.Navin Kumar, MS(Ortho), PG., Govt. Royapettah Hospital, Chennai.	1.To Identify a Safe Zone to approach proximal Humerus 2.To study Anatomical relations of Axillary nerve, its course & its Variations
2.	Dr.T.Satheesh Kumar, D.Ortho., PG., Govt. Royapettah Hospital, Chennai	Heriditary Multiple Exostosis
3.	Dr.J. Jeya Shambavi, MD(Pathology), PG., Govt. Kilpauk Medical College, Chennai-10	Clinicopathological Histomorphological and Immunohistochemical Study of Neuroendocrine Tumors of GIT
4.	Dr.L. R. Saranya. MD., (Paed.)PG., Govt. Kilpauk Medical College, Chennai-10	Cord Blood Zinc Level in Term-Small for Gestational Age Neonates
5.	Dr.A.Satheesh Kumar, MS(ENT), PG., Kilpauk Medical College, Chennai	Study on Cases of Chronic Suppurative Otitis Media in Tubo Tympanic Type Due to Sinusitis as Focal Sepsis
6.	R.Prathiban,(Msc.,Physiology), PG., Student, The TN. Dr.MGR Medical University,Chennai-32	Prevalence of Cardiac Dysautonomia in Type I Diabetes mellitus
7.	B. Manikandan, (Msc., Physiology), PG., Student, The TN Dr.M.G.R. Medical University, Chennai-32.	A Comparative Study of Left Ventricular Structure and Function in Obese and Non Obese Subjects
8.	G. Selvakumar, (MSc., Physiology), PG., Student, The TN Dr.M.G.R. Medical University, Chennai-32.	A Study of the Intraocular Pressure In Patients with Diabetic Normotensive, Diabetic Hypertensive and Normal Subjects

9.	R. Ragulji, (Msc.,Physiology), PG., The TN Dr.MGR Medical University, Chennai-32.	A Study of Pulmonary function in insulin dependent diabetes mellitus
10.	V.M. Jenila Veny,(MscPhysiology), PG. The TN Dr.MGR Medical University, Chennai-32	Cardiovascular Autonomic Dysfunction in Chronic Kidney Disease
11.	Dr.G. Lakshmi, MD(O&G), PG., Govt. Kilpauk Medical College,Chennai-10	A Study of Association of Thyroid Disorders in Abnormal Uterine Bleeding
12.	Dr.R. Harini, MD(O&G), PG., Kilpauk Medical College, Chennai	Single Dose Antibacterial treatment for Asymptomatic Bacteriuria in Pregnancy
13.	Dr.E.Geetha, MD(O&G), PG., Govt. Kilpauk Medical College, Chennai-10	A Study of the incidence course of Pregnancy and Pregnancy outcome in Obstetric Cholestasis and to evaluate the efficiency of UDCA in relieving the Symptoms and Improving the Perinatal outcome in these Patients
14.	Dr.S. Nithya, MD(O&G), PG., Govt. Kilpauk Medical College, Chennai-10	Prospective Study of Prevalence of diabetes Mellitus, Thyroid Dysfunction and Hyperprolactinemia in Recurrent Pregnancy loss
15.	Dr.Mohideen Fathima, MD(O&G), PG., Govt. Kilpauk Medical College, Chennai-10	A Study of evaluation of multi system changes in Gestational hypertension / severe pre-eclamptic/eclampasia patients
16.	Dr.M.Padma Priya, MD(O&G), PG., Kilpauk Medical College, Chennai	Dyslipidemia as a Predictor of PIH
17.	Mrs.G. Savitha, (Msc.,Medical Bio Chemistry), TN Dr.M.G.R.Medical University, Chennai-32.	Association of subclinical hypothyroidism in metabolic syndrome patients
18.	Dr.K. Bharadhwaj, MD(G.M.), PG., Kilpauk Medical College, Ch-10	A Study on Peripheral Vascular Disease in Type 2 Diabetes Mellitus
19.	Dr.B.Priya, MD(G.M.), PG	Study of Serum Bilirubin Concentration in Established Coronary Artery Disease
20.	Dr.R.Hema, MD(G.M.), PG.,	Study of Troponin I level in Supraventricular Tachycardia in Non Cad Patients
21.	Dr.P.Manoj Kumar, MD(G.M.), PG., Kilpauk Medical College, Ch-10	A Study on Pulmonary Functions in Type 2 Diabetes Mellitus
22.	Dr.M.Dhanasekar, MD(G.M.), PG.,	Prognostic Risk Stratification of Acute Coronary Syndrome – Role of Highly Sencitive – Reactive Protien
23.	Dr.N. Karthik, MD(G.M.), PG., Govt.Kilpauk Medical College, Chennai-10	A Study of Comparison of QT Dispersion in Acute Myocardial Infraction Between Early Reperfusion and Late Reperfusion Therapy

24.	Dr.H. Anuradha, MD(G.M.), PG., Kilpauk Medical College, Ch-10	A Study of Stress Hyperglycemia in Moderate Degree Burns
25	Dr. V. Nandakumar, MD(G.M.), PG.,	A Prospective Study of Clinical Profile of Emphysematous Pyelonephritis in Type Two Diabetes Mellitus
26.	Dr.S.Sasikumar, MS(G.S.), PG., Govt. Royapettah Hospital, Chennai	A Study of Unusual Presentations of Appendicitis.
27.	Dr.S.R.Padmanabhan, MS(GS), PG., Govt. Royapettah Hospital, Chennai	A Comparative Study Between Autologous Platelet Rich Plasma and Saline Dressing for Diabetic Ulcer
28.	Dr.C.Rose, Scientist-G and Head, Biotechnology, Central Leather Institute, Chennai.	Wound healing efficacy of the chitosan – containing collagenous biomaterial, on burn wound
29.	E.K. Lavanya,B.Tech, Biotechnology, PG., Prathyusha Institute of Technology and Management, Tiruvallur.	Isolation and Characterization of Bacterial Pathogens from Eye Infection

We are glad to inform you that at the Ethical Committee meeting, the documents were discussed and the above short term projects are Ethically approved.

4/2/11
CHAIRPERSON
. DEAN
Govt. Kilpauk Medical College,
Chennai-10.

To: The Individuals

சுய ஒப்புதல் படிவம்

ஆய்வு செய்யப்படும் தலைப்பு :

மகளிர் மற்றும் மகப்பேறு மருத்துவத்துறை :

கீழ்ப்பாக்கம் மருத்துவக்கல்லூரி :

பங்கு பெறுபவரின் பெயர் :

பங்கு பெருபவரின் வயது :

பங்கு பெருபவரின் எண் :

பங்கு பெறுபவர் இதனை (√) குறிக்கவும்.

- ❖ மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும் அதற்கான தகுந்த விளக்கற்களை கேட்க வாய்ப்பளிக்கப்பட்டடுள்ளது என அறிந்து கொண்டேன். ☐
- ❖ நான் இவ்வாய்வில் தன்னிச்சையாகத் தான் பங்கேற்கிறேன்.எந்த காரணத்தினாலோ எந்த சட்டசிக்களுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகிக் கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.ஷ ☐
- ❖ இந்த ஆய்வு சம்பந்தமாகவோ அதை சார்ந்து மேலும் ஆய்வு மேற்கொள்ளும் போது இந்த ஆய்வில் பங்கு பெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறோன். ☐
- ❖ இந்த ஆய்வின் மூலம் கிடைக்கும் தகவலையோ முடிவையோ பயன்படுத்திக் கொள்ள மறுக்கமாட்டோன். ☐
- ❖ இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக் கொள்கிறேன். இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உன்மையுடன் இருப்பேன் என்றும் உறுதியளிக்கிறேன். ☐
- ❖ இந்த ஆய்வில் ஒருமுறை 5 மி இரத்த பரிசோதனைக்காக எடுத்தக் கொள்ளப்படும் என்பதை அறிவேன்.

பங்கேற்பவரின் கையொப்பம்_____

இடம்_____ தேதி_____

பங்கேற்பவரின் பெயர் மற்றும் விலாசம்
சாட்சியாளரின் கையொப்பம்

இடம்_____ தேதி_____
சாட்சியாளரின் பெயர் மற்றும் விலாசம்

ஆய்வாளரின் கையொப்பம்
இடம்_____ தேதி_____
ஆய்வாளரின் பெயர்_____