LACTATE DEHYDROGENASE – A BIOCHEMICAL MARKER OF PRE-ECLAMPSIA AND ECLAMPSIA

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BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled "**LACTATE DEHYDROGENASE – A BIOCHEMICAL MARKER OF PRE-ECLAMPSIA AND ECLAMPSIA**" submitted by **Dr.M.Vidhya**, appearing for **M.S. (Obstetrics and Gynaecology)** degree examination in April 2015 is a original bonafide record of work done from November 2013 to August 2014 by her under my guidance and supervision in partial fulfillment of requirement of the Tamil Nadu Dr.M.G.R. Medical University, Chennai. I forward this to the Tamil Nadu Dr.M.G.R. Medical University, Chennai, Tamil Nadu, India.

Dr.G.RAJA BILLY GRAHAM, M.S
The Dean,
Chengalpattu Medical College and Hospital,
Chengalpattu -600301.

Dr. K. VANI, DCH, M.D, O & G.,
Professor and Guide,
Head Of The Department,
Department of Obstetrics and Gynaecology,
Chengalpattu Medical College and Hospital,
Chengalpattu - 600 301.

DECLARATION

I, Dr. M. VIDHYA solemnly declare that the dissertation titled "**LACTATE DEHYDROGENASE – A BIOCHEMICAL MARKER OF PREECLAMPSIA AND ECLAMPSIA**" has been prepared by me. I also declare that this bonafide work or a part of this work was not submitted by me for any other award, degree, diploma to any other university board either in India or abroad.

This is submitted to The Tamilnadu Dr. M. G. R. Medical University, Chennai in partial fulfillment of the rules and regulations for the award of M.S degree Branch- II (Obstetrics & Gynaecology) to be held in April 2015.

Place: Chengalpattu **Dr. M. VIDHYA**

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ABBREVIATIONS

LDH	Lactate DeHydrogenase
S. LDH	Serum Lactate DeHydrogenase
CT	Computed Tomography
SBP	Systolic Blood Pressure
DBP	Diastolic Blood Pressure
GA	Gestational age
ISSHP	International Society for the study of hypertension in pregnancy
NIH	National Institute of Health
NHBPEP	National High Blood Pressure Education Program
BP	Blood Pressure
MTHFR	Methylene Tetrahydrofolate Reductase
AGT	Angiotensinogen
HLA	Human Leucocyte Antigen
NOS	Nitric oxide synthase
ACE	Angiotensin converting enzyme
SFlt	Soluble fms- like tyrosine kinase
PGF	Placental growth factor
VEGF	Vascular endothelium derived growth factor
PIGF	Platelet inhibitory growth factor
RNA	Ribonucleicacid
SGOT	Serum glutamate oxaloacetate transaminase
SGPT	Serum glutamate pyruvate transaminase
DIC	Disseminated intravascular coagulation
IUGR	Intrauterine growth restriction

ß-HCG	Beta Human chorionic gonadotrophin
CTG	Cardiotocogram
AST	Aspartate transaminase
RBC	Red blood cells
GTCS	Generalised tonic clonic seizures
OPD	Out patient department
IUD	Intra uterine demise
LSCS	Lower segment cesarean section
LN	Labour naturale
U/A	Urine Albumin

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INTRODUCTION

Hypertensive disorders of pregnancy are the most common medical disorders complicating pregnancy. The incidence is continuously increasing due to unknown reasons and the overall incidence is **7 to 10%** worldwide. One in every **100 to every 2000** pregnancy is complicated by Eclampsia. Pre-eclampsia and Eclampsia as a cause of maternal mortality and morbidity is increasing worldwide both in developing and developed nations currently the contribution being **15 to 20%**[1].

Hypertensive disorders of pregnancy is a spectrum of disorder which include chronic hypertension that antedates pregnancy and gestational hypertension or pre-eclampsia that is unique to human pregnancy. It is still a poorly understood condition. The clinical course is progressive and characterized by continuous deterioration that is arrested only by termination of pregnancy. Hence the disease must be detected in early stage and managed appropriately for improved maternal and fetal outcome [1].

Biochemical markers will enable prompt detection of high risk pregnancies and those who will develop clinically significant disease and hence the maternal and fetal outcome can be improved by enhancing the antenatal care to those target women.

Lactate dehydrogenase (LDH) is an intracellular enzyme which converts lactate to pyruvate and its elevated level indicates cellular death and leakage of enzyme from the cell.

Increased levels of S.LDH is a marker of increased cellular death and is found in association with pre-eclampsia and Eclampsia [2, 3, 4].

Estimation of S.LDH is a simple, minimally invasive and cheap biochemical test which though nonspecific is found to be highly sensitive and a reliable parameter in estimating the severity of the disease in in some studies[2, 3, 4].

This study aims at quantitating the extent of cellular death in terms of S.LDH in patients with clinical profile of pre-eclampsia and eclampsia and thereby using S.LDH as a marker of severity of the disease.

AIM & OBJECTIVES

1. To compare serum LDH levels in the normal pregnant women and in women with Pre-eclampsia and Eclampsia in the antepartum period.
2. To correlate the severity of the disease, with lactate dehydrogenase levels in serum of patients of Pre-eclampsia and Eclampsia
3. To correlate the maternal and perinatal outcome with lactate dehydrogenase levels in serum of patients of Pre-eclampsia and Eclampsia

REVIEW OF LITERATURE

Pre-eclampsia is a multisystem disorder affecting every organ in the body.

DEFINITION

According to the International Society for the study of hypertension in pregnancy (**ISSHP**), Hypertension is defined as a systolic pressure of **≥140mm Hg** and diastolic pressure of **≥ 90 mm Hg** measured on 2 occasions at least 6 hours apart within 7 days [5].

A single Diastolic reading of >110 mm Hg is also considered as Hypertension [6]

A rise in blood pressure of 30 mm Hg in Systolic or 15 mm Hg in Diastolic pressure from mid pregnancy values with absolute values <140/90 is no longer considered as diagnostic criteria for gestational hypertension or Pre-eclampsia as evidence does not show an adverse outcome for these pregnancies [7] [8]

Edema is no longer used as diagnostic criteria because it is a common accompaniment of normal pregnancy [6].

Proteinuria is absent in gestational hypertension and it is an essential criteria in the diagnosis of pre-eclampsia. **Proteinuria of 1g/L**

or more (2+ or more by dipstick) in at least 2 random urine specimen at least 6 hours apart or **>0.3 g** in 24 hours sample is considered as significant pre-eclamptic level.[9]

CLASSIFICATION

The National Institute of Health (NIH) working group of the NHBPEP – **National High Blood Pressure Education Program (2000)** categorized hypertensive disorders of pregnancy into four types [9].

1. Gestational hypertension
2. Pre-eclampsia & eclampsia
3. Pre-eclampsia superimposed on chronic hypertension
4. Chronic hypertension

GESTATIONAL HYPERTENSION

Gestational hypertension is defined as hypertension (systolic pressure of ≥ 140 mm Hg and diastolic pressure of ≥ 90 mm Hg measured on 2 occasions at least 6 hours apart within 7 days) diagnosed for the first time during pregnancy **after 20 weeks** of gestation without associated proteinuria and BP returns to normal **within 12 weeks** postpartum[9]

PRE-ECLAMPSIA & ECLAMPSIA

Pre-eclampsia is defined as Hypertension (systolic pressure of ≥ 140 mm Hg and diastolic pressure of ≥ 90 mm Hg measured on 2 occasions at least 6 hours apart within 7 days) diagnosed after 20 weeks of gestation with **Proteinuria of 1g/L or more (2+ or more by dipstick**) in at least 2 random urine specimen at least 6 hours apart or **>0.3 g** in 24 hours sample.

Eclampsia is the occurrence of **generalized tonic clonic seizures** in a pre-eclamptic woman that cannot be attributed to other causes. [9]

PRE-ECLAMPSIA SUPERIMPOSED ON CHRONIC HYPERTENSION

Pre-eclampsia superimposed on chronic hypertension is a condition defined by

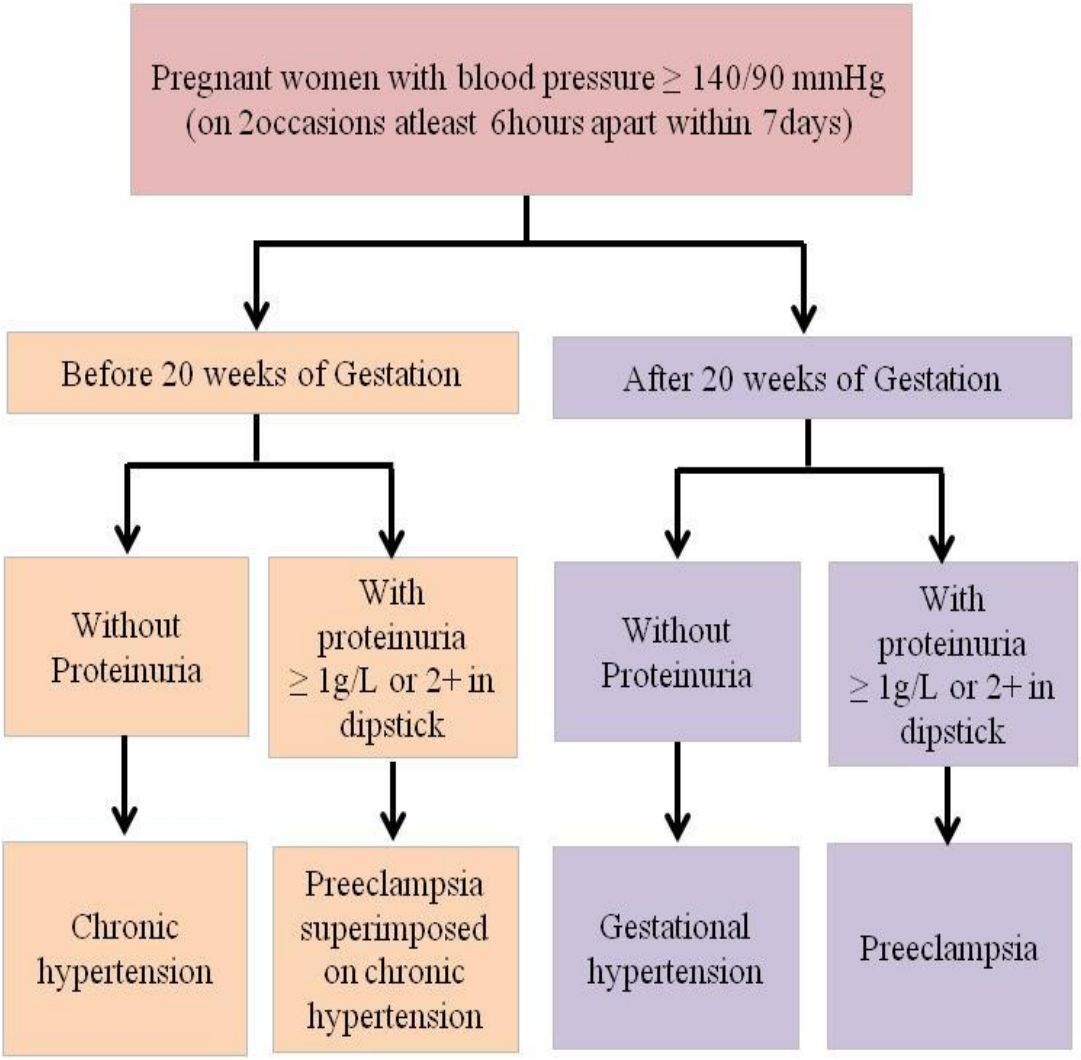
1. **New onset proteinuria** in hypertensive women after 20 weeks of gestation
2. Sudden increase in Blood Pressure or proteinuria or thrombocytopenia in women with hypertension and proteinuria before 20 weeks of gestation.[9]

CHRONIC HYPERTENSION

Chronic hypertension can be

1. Hypertension diagnosed before pregnancy,
2. Hypertension diagnosed **before 20 weeks** gestation, not attributable to Gestational Trophoblastic Disease or Multiple Pregnancy,
3. Hypertension first diagnosed after 20 weeks gestation and persistent **beyond 12 weeks** postpartum.[9]

Fig-1. NHBPEP- Classification of hypertensive disorder in pregnancy



RISK FACTORS [10]

- Age > 35 years
- Primigravida
- Primipaternity
- Interval from last pregnancy > 10 years
- Limited sperm exposure. e.g., Oocyte donation, Donor Insemination.
- **Obstetric Factors**

  Pre-eclampsia or Gestational Hypertension in prior pregnancy

  Multiple Gestations

  Hydatidiform mole

  Hydrops fetalis

  Abnormal uterine artery Doppler at 18-24 weeks

  Chromosomal anomaly (Trisomy 13, Triploidy)

- Family history of Preeclampsia

- **Pre-existing medical disorders**

  Hypertension

  Diabetes mellitus

  Obesity: BMI of 35 kg/m2 or more

  Renal Disease

  Vascular Disease

  Auto Immune Disease

  Thrombophilias

  Anti-phospholipid Antibody Syndrome

  Hyper homocystinaemia

  Sickle Cell Disease/ Sickle Cell Trait

ETIOPATHOGENESIS

The etiology is still largely unknown. The following mechanisms contribute to the pathogenesis.

1. ABNORMAL PLACENTATION [11] [12] [13] [14]

Pre-eclampsia is a disease of the placenta and the fetus is required for the development of the disease.

Optimal placental development in normal pregnancy involves a process of controlled trophoblastic invasion extending from the decidua upto inner third of myometrium by extravilllous trophoblast.

The endothelial lining and the muscular layer of the spiral arteries are disrupted converting the small caliber muscular arteries into large capacity low resistance vascular spaces

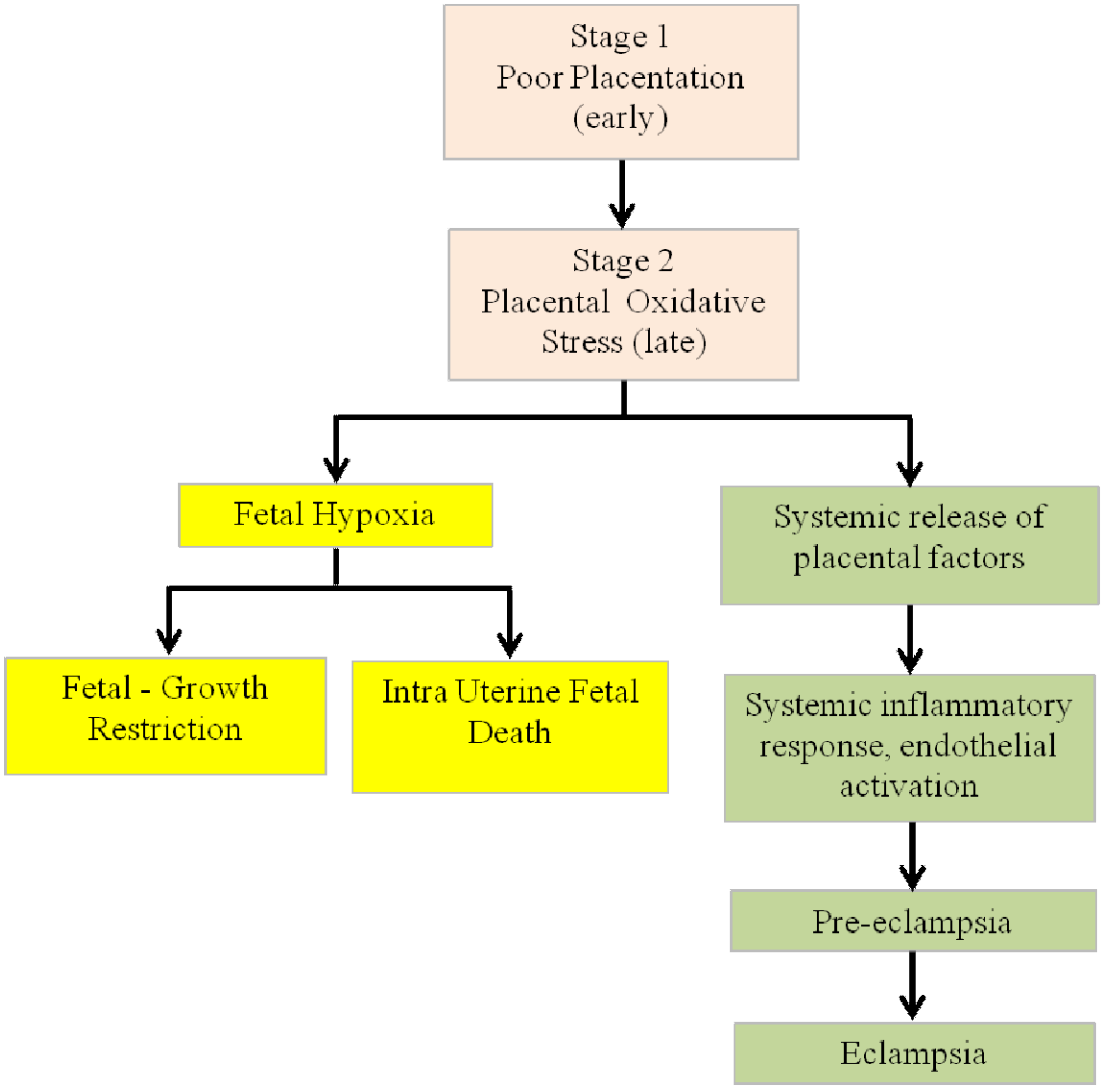
The development of these uteroplacental vessels proceed in two stages:

Stage 1 : Occurs **before 12 weeks** post fertilization upto the interface between decidua and myometrium

Stage 2 : Occurs **between 12 and 16 weeks** and involves invasion of intra-myometrial segments of spiral arteries.

In women destined to develop Preeclampsia, the trophoblastic invasion is incomplete and shallow, the deeper myometrial arterioles do not lose their endothelial lining and musculo-elastic tissue and their mean diameter is half that of vessel in normal placenta resulting in reduced uteroplacental blood flow.

Fig-2. Two stage theory of preeclampsia



2. ENDOTHELIAL DYSFUNCTION & VASOSPASM [15] [16]

This is caused by

a) Decreased formation of endogenous vasodilators such as Nitric Oxide and Prostacyclin A.

b) Increased generation of vasoconstrictors such as Endothelin and Thromboxane.

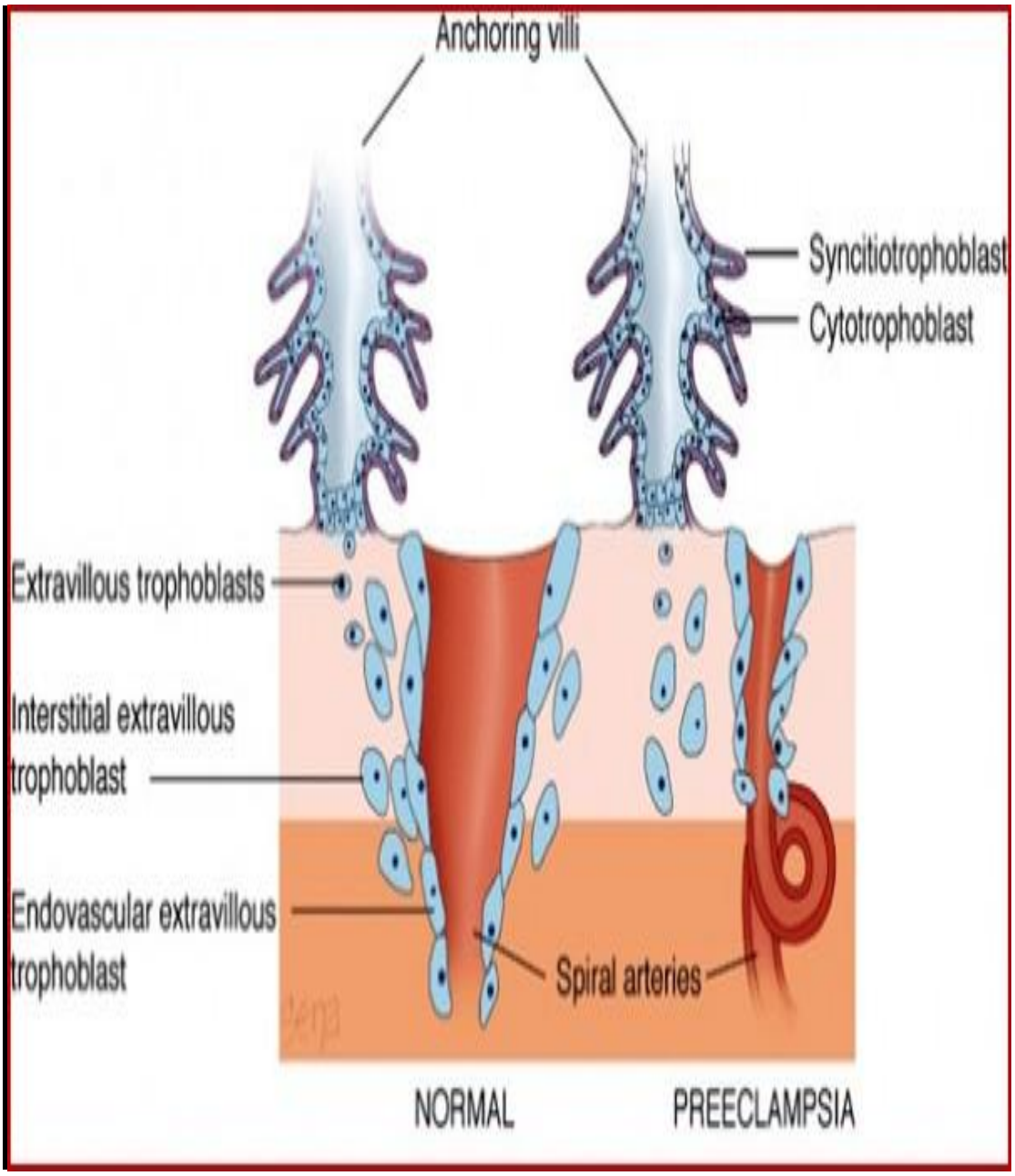
c) Increased sensitivity to angiotension II that alters the vascular tone. Biomarkers of endothelial dysfunction such as plasma fibronectin, thrombomodulin and factor 8 antigen are increased in pre-eclamptic pregnancies.

3. GENETIC FACTORS [19]

Pre-eclampsia is a multifactorial polygenic syndrome. These include

- MTHFR gene affecting methylene tetrahydrofolate reductase
- Factor V (Leiden) gene
- AGT (Angiotensinogen) gene
- HLA genes causing Immunological intolerance
- NOS 3 gene affecting endothelial NO production
- F2 (Prothrombin – Factor II) gene
- ACE (Angiotensin Converting Enzyme) gene

Fig-3. Normal & abnormal placentation



4. PROANGIOGENIC & ANTIANGIOGENIC PROTEINS

Pro-angiogenic factors are vascular endothelial growth factor and placental growth factor. Anti-angiogenic factors are soluble fms like tyrosine kinase 1 and soluble endoglin.

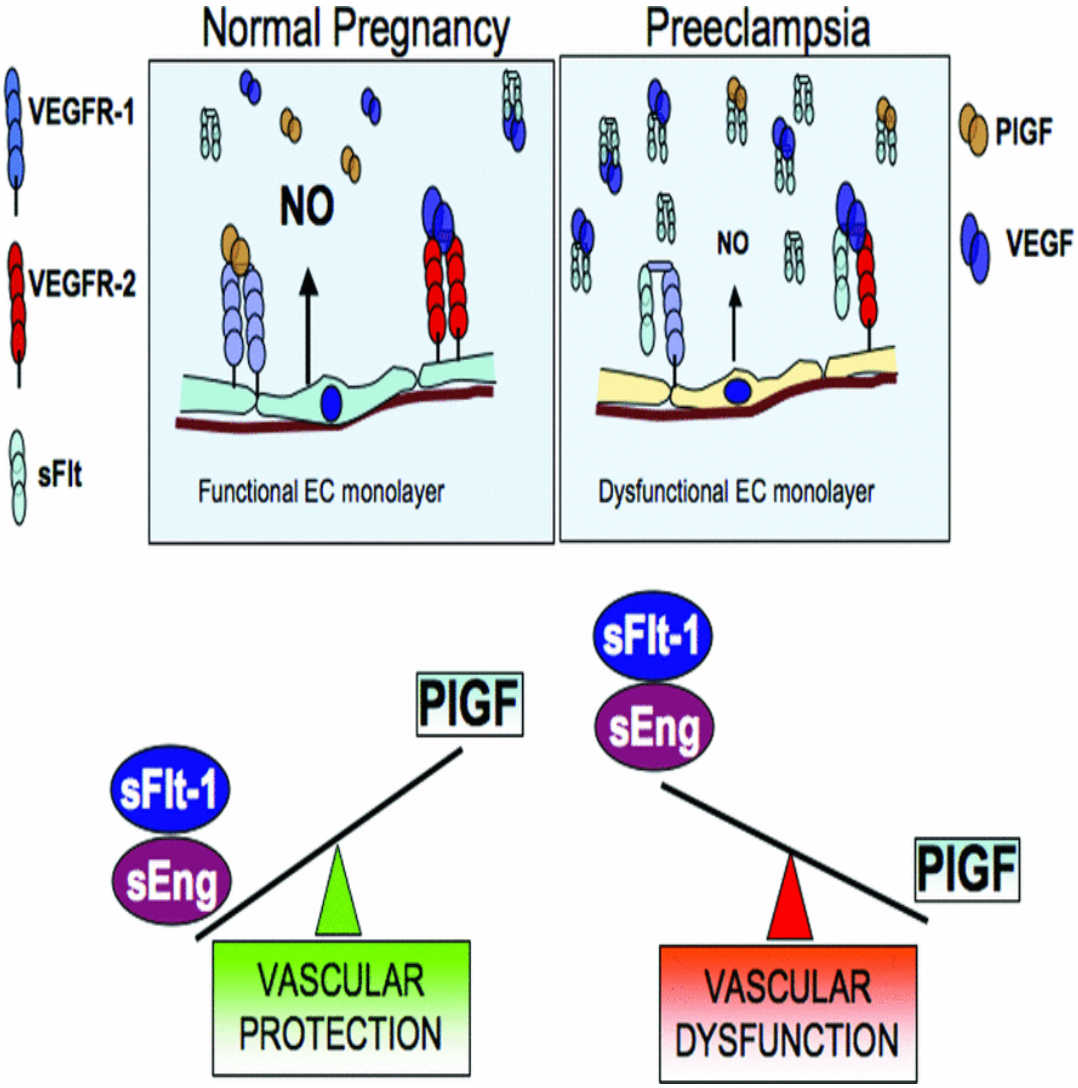
Soluble FMS like tyrosine kinase

SFlt-1 (Soluble FMS like tyrosine kinase) is a variant of receptor for placental growth factor (**PGF**) and vascular endothelial growth factor (**VEGF**). Increased levels of SFlt-1 decreases the activity of Proangiogenic factors like PGF & VEGF. But SFlt-1 levels were increased only within 5 weeks before the onset of hypertension and proteinuria. [20].

Soluble Endoglin

Soluble Endoglin is a placenta derived molecule which causes decrease in endothelial NO dependent vasodilatation and it begins to rise **after 20 weeks** of gestation and rises more steeply in pre-eclamptic patients [19]

Fig-4. Role of antiangiogenic factors in the pathogenesis of pre-eclampsia



5. RENIN-ANGIOTENSION-ALDOSTERONE SYSTEM

The refractoriness to Angiotensin II is lost as early as **18-22 weeks**, several weeks before the onset of hypertension in women who are destined to develop preeclampsia later in gestation [20]

6. IMMUNOLOGICAL INTOLERANCE

Extravillous trophoblast express reduced amount of HLA-G in early pregnancy destined to be pre-eclamptic. A lower level of messenger RNA for HLA-G has been noted in trophoblast from preeclampsia than from normal pregnant patients [21]

7. OXIDATIVE STRESS

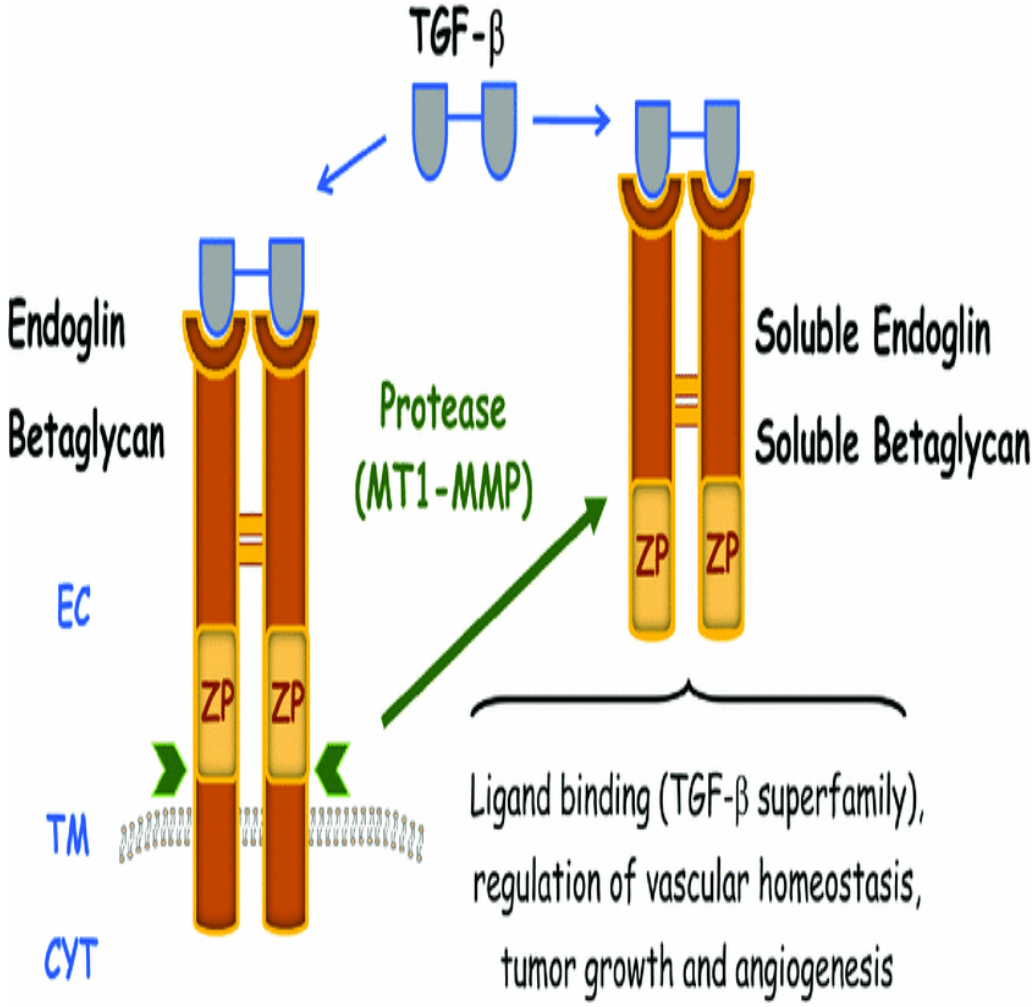
It causes endothelial damage and interferes with nitric oxide production and prostaglandin balance. [22]

Heme oxygenase is a negative regulator of SFlt-1 production and is found elevated in smokers which reduces the risk of preeclampsia in smokers.

PATHOPHYSIOLOGY

Preeclampsia is a multi organ disorder and the following systems are commonly involved in its pathophysiology

Fig-5. Role of soluble endoglin in the pathogenesis of pre-eclampsia



1. KIDNEYS

Kidneys are the most commonly involved organ. **Glomerular capillary endotheliosis** is the characteristic change in the kidney. Early focal segmental glomerular sclerosis is also seen in some of the pre-eclamptic patients.

Proteinuria is the hallmark and its quantity indicates the severity of the disease.

Acute Renal failure due to acute tubular necrosis is rare in preeclampsia.

Serum Uric Acid is elevated as a result of reduced GFR and increased trophoblast turnover and production of purines due to placental Ischaemia.

Urinary Sodium concentration is elevated and urinary excretion of calcium is diminished because of increased tubular reabsorption. [6]

2. PLACENTA

Increased incidence of infarct, hematoma, congested chorionic villi, proliferative end arteritis and degeneration are seen in placenta of preeclamptic women. Microscopic examination reveal increased syncytial

knots, cytotrophoblastic cellular proliferation, fibrinoid necrosis, endothelial proliferation, calcified and hyalinised villous spots. [23]

3. LIVER

Characteristic features are **Periportal hemorrhage** in the periphery of the liver. Increase in liver enzymes SGOT, SGPT and clinical jaundice may occur. The small hemorrhages combine to form subcapsular hematoma which stretches Glisson's Capsule causing epigastric pain which is a sign of impending eclampsia.[5]

4. COAGULATON

These include thrombocytopenia and reduced levels of clotting factors. Thrombocytopenia < 1 lakh cells/mm3 indicate severe disease. Evidence of hemolysis in the form of elevated S. LDH, bizarre shaped erythrocytes, Schistocytes, Spherocytes and reticulocytes are seen in severe disease. [24] [25]

5. CARDIOVASCULAR SYSTEM

Cardiac changes in Pre-eclamptic patients are increased afterload and hemoconcentration. Hemoconcentration is due to generalized vasoconstriction that follows endothelial activation and leakage of plasma into interstitial space. [6]

6. BRAIN

The most common finding in brain in preeclampsia is edema. Concurrent foci of infarct may also be present. Cerebral hemorrhage should be suspected in older gravida with chronic hypertension who present with hemiplegia, focal deficits or coma. [26]

Visual disturbances like scotoma, blurring of vision or diplopia arise from three potential areas

- ❖ Visual cortex of occipital lobe
- ❖ Lateral geniculate nuclei
- ❖ Retina.

MATERNAL COMPLICATIONS WITH UNTREATED SEVERE PRE-ECAMPSIA[27]

- ❖ HELLP Syndrome (13.3%)
- ❖ Abruptio Placenta (11.7%)
- ❖ Pulmonary Edema (3.1%)
- ❖ Thrombocytopenia / DIC (1.4%)
- ❖ Acute Renal Failure (1.4%)
- ❖ Adult Respiratory Distress Syndrome (1%)

- ❖ Hepatic rupture (1%)
- ❖ Eclampsia (1%)
- ❖ Others
    a. Cerebral Hemorrhage
    b. Cortical Blindness
    c. Sudden postpartum collapse

FETAL COMPLICATIONS WITH UNTREATED SEVERE PRE-ECLAMPSIA[27]

- ❖ Fetal growth restriction (43%)
- ❖ Intrauterine death (8.2%)
- ❖ Preterm delivery and cardiovascular morbidity associated with low birth weight

PREDICTORS & MARKERS OF PRE-ECLAMPSIA

Many markers and predictors of pre-eclampsia have been proposed for early detection of high risk pregnancies to improve perinatal outcome. They can be

- Clinical test
- Biophysical test
- Biochemical test

CLINICAL TEST

GANTS ROLL-OVER TEST.

This test is otherwise called **Supine Pressor test**. Elevation of diastolic blood pressure by atleast 20 mm Hg induced by having women at **28-32 weeks** of gestation assume the supine position after lying laterally recumbent predicts gestational hypertension. [28].

MEAN ARTERIAL BLOOD PRESSURE

Mean Arterial Blood Pressure >90 mm HG between **18-26 weeks** is a predictor of preeclampsia.[27][29]

ANGIOTENSIN SENSITIVITY TEST

A women destined to get preeclampsia will respond to less than 8ng/kg/min of an angiotension infusion due to an alteration in vessel wall refractoriness. This test is done between **26-30 weeks.** [28]

PULSE WAVE ANALYSIS

Stiffness in the finger arterial pulse acts as a predictor [27]

AMBULATORY BP MONITORING

Alterations in circadian BP variability in the first trimester predicts pre-eclampsia

BIOCHEMICAL TEST

PLATELET VOLUME

A mean platelet volume of more than 11 Fl at **28 weeks** of gestation was found to be associated with subsequent pre-eclampsia and more over increased platelet turnover may be an early marker of pre-eclampsia.

SERUM FIBRONECTIN

The level rises **within 12 weeks** in those women destined to get preeclampsia with a positive predictive value of 29% and negative predictive value of 98%. [30]

URINARY KALLIKREIN EXCRETION

Inactive urinary kallikrein-creatinine ratio of < 170 between **16 and 20 weeks** of pregnancy predicts future development of preeclampsia.[27][31]

Protein C inhibitor is decreased by kallikrein, which is elevated due to activation of intrinsic coagulation pathway.

URINARY CALCIUM EXCRETION

Preeclampsia is characterised by marked reduction in the fractional excretion of calcium leading to striking hypocalciuria due to increased

tubular reabsorption of calcium. Urinary calcium level of < 12 mg/dL is associated with a sensitivity of 91 % [27]

RAISED SERUM β-hCG

B-hCG is increased by **14-20 weeks** of gestation due to abnormal trophoblastic invasion and increased production by hypoxic trophoblast.[27]

DYSLIPIDEMIA

Elevated levels of triglycerides, free fatty acids and lipoproteins are predictors of preeclampsia. [27]

SERUM URIC ACID

Elevated serum uric acid is seen in the first trimester in women destined to develop preeclampsia.[27]

BIOPHYSICAL TEST

UTERINE ARTERY DOPPLER

Uterine artery impedance between **18 and 26 weeks** has been used as an early morning screening test for women at risk of preeclampsia. The presence of high systolic/diastolic ratio and persistence of diastolic notch may predict preeclampsia or IUGR. [27] [29] [32]

Fig-6. Normal uterine artery doppler

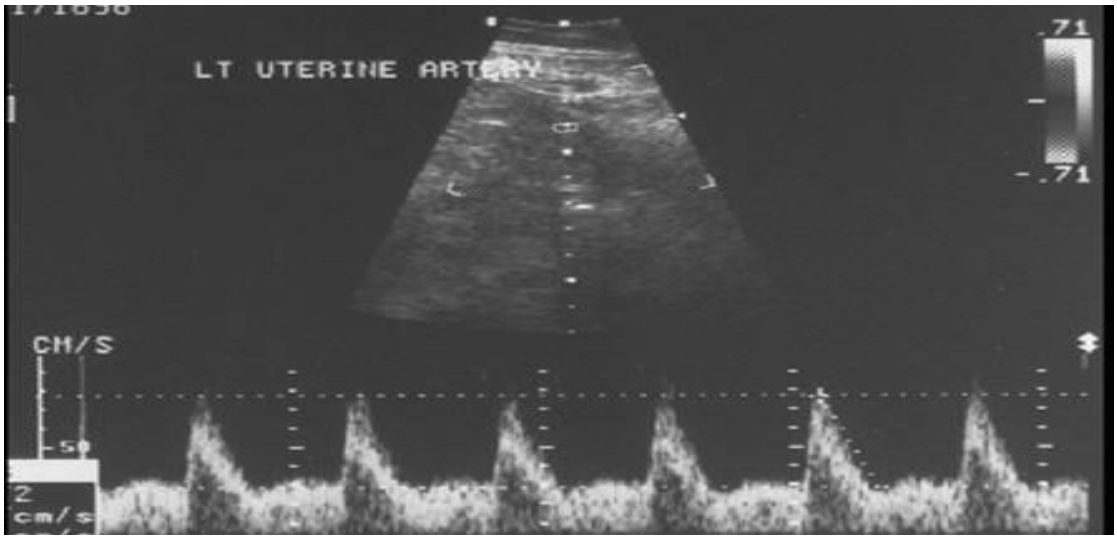


Fig-7. Abnormal uterine artery doppler

TEST RELATED TO FETOPLACENTAL ENDOCRINE DYSFUNCTION[27]

- ❖ Alpha feto protein
- ❖ Estriol
- ❖ Inhibin – A
- ❖ Activin – A
- ❖ Pregnancy associated plasma protein – A
- ❖ Placental protein 13
- ❖ Corticotropin releasing hormone

OTHER MARKERS [27]

- ❖ Platelet Count
- ❖ P-Selectin
- ❖ Neurokinin B
- ❖ Anti-thrombin – III
- ❖ 24 hours ambulatory BP monitoring
- ❖ Soluble fms like tyrosine kinase
- ❖ Endoglin
- ❖ Leptin
- ❖ D-Dimer

MANAGEMENT OF PRE-ECLAMPSIA

Preeclampsia is an unpredictable disorder whose management depends on the gestational age and severity of the disease. The only definitive management is termination of pregnancy. Based on various parameters pre-eclampsia is classified into mild and severe as below.

Table 1- Classification of Pre-eclampsia

Parameter	**Mild Pre-eclampsia**	**Severe Pre-eclampsia**
SBP (mmHg)	140 to 159	160 & above
DBP (mmHg)	90 to 109	110 & above
Proteinuria	>1g/L to <3g/L 2+ in dipstick >0.3g to <5g in 24hours	3g/L & above 3+ & above in dipstick 5g & above in 24hours
Headache	No	Yes
Oliguria	No	Yes
Pulmonary Edema	No	Yes
Thrombocytopenia	No	Yes
Elevated S.Creatinine (>1.2mg/dL)	No	Yes
Elevated S. Transaminase	No	Yes
Severe IUGR	No	Yes

MANAGEMENT OF MILD PRE-ECLAMPSIA[6]

Mild Pre-eclampsia can be managed on **outpatient basis** with rest and blood pressure monitoring at home and more **frequent antenatal visits** weekly or two weekly to detect any signs or symptoms of worsening at the earliest.

The use of **anti-hypertensive drugs** for mild pre-eclampsia is questionable. The main objective of treatment with anti-hypertensives is to reduce the risk of severe hypertension, Eclampsia and cerebrovascular hemorrhage.

There is loss of cerebral auto regulation and risk of cerebral hemorrhage once the mean arterial blood pressure reaches 150 mm Hg.

The first line drugs are **Labetolol, Nifedipine and alpha methyl dopa**. Patients with mild Preeclampsia can be induced at 38 weeks of gestation.

Pregnancy should be terminated earlier if there is progression to severe preeclampsia and Eclampsia and for other obstetric indications like IUGR.

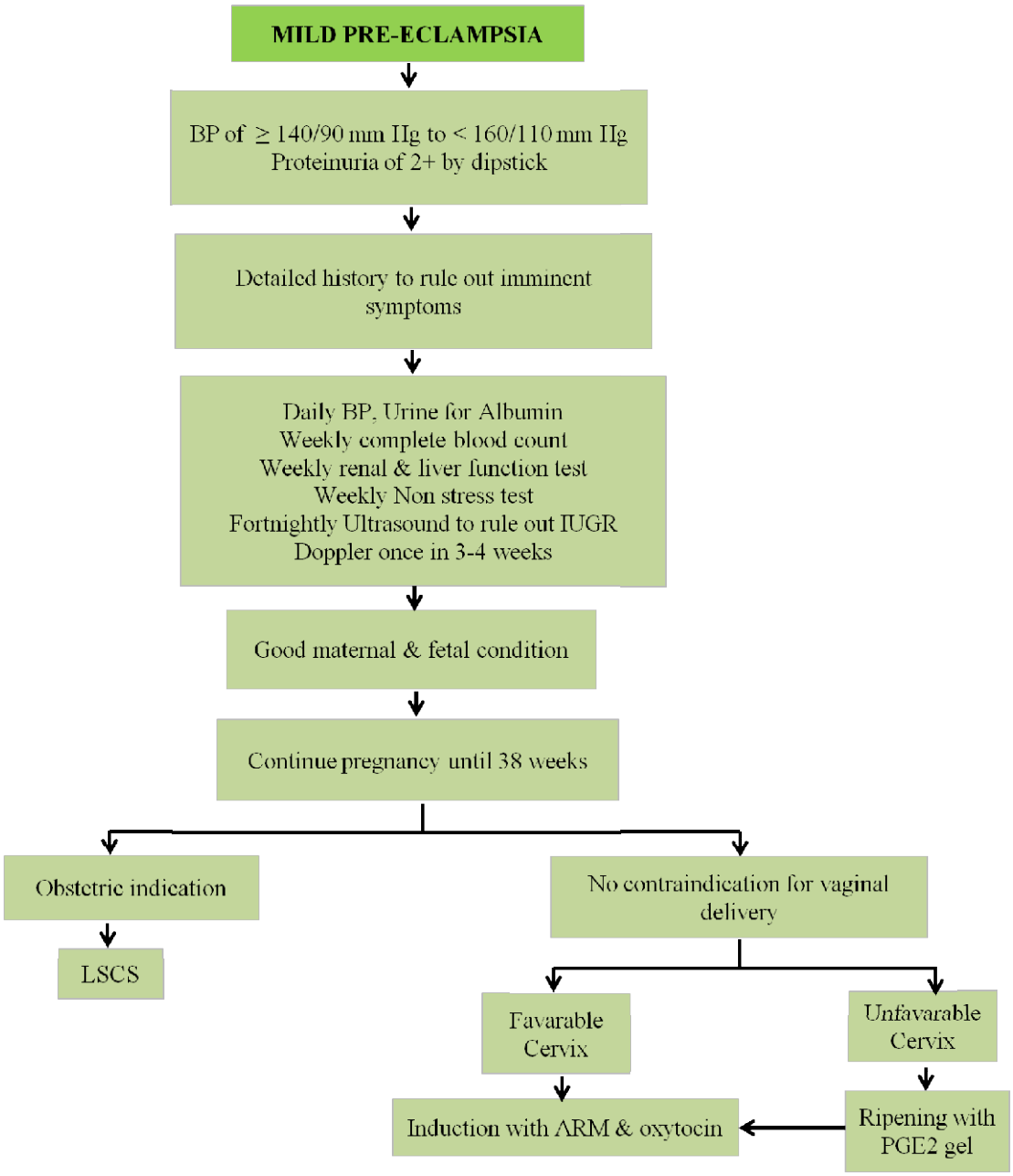
If pregnancy is terminated before 34 weeks, **steroids** are given for lung maturity. Labour can be induced vaginally if there are no obstetric

indications for caesarian section. If the cervix is unfavourable, induction can be done with Dinoprostone Gel. Artificial rupture of membranes and oxytocin acceleration can be done if the cervix is favourable.

During labour continuous blood pressure monitoring, CTG monitoring and active management of third stage of Labour is followed.

Mild pre-eclampsia if untreated can progress to severe pre-eclampsia at any time and therefore watchful expectancy should be the rule in all cases of mild pre-eclampsia.

Fig-8. Management algorithm of mild preeclampsia



MANAGEMENT OF SEVERE PREECLAMPSIA [33] [34]

The management of Severe Preeclampsia according to gestational age is

- < 24 weeks - Stabilize the patient and terminate pregnancy
- 25-33 weeks - Expectant management with intensive maternal and fetal surveillance, steroid therapy
- > 34 weeks - Stabilize the patient with strict fetal surveillance and deliver

Maternal Surveillance in Expectant Management of Severe Pre-eclampsia [6]

- Blood pressure daily
- Urine albumin daily
- Weight daily
- Platelet count, Renal function and Liver Function twice weekly
- History of imminent symptoms
- Coagulation profile

Fetal Surveillance in Expectant Management of Severe Pre-eclampsia [6]

- ❖ Fetal Kick count daily
- ❖ Non stress test – twice weekly
- ❖ Amniotic fluid index twice weekly
- ❖ Ultrasound to assess gestational age and growth
- ❖ Umbilical artery Doppler

Indications For Termination in a Case of Severe Pre-eclampsia Under Expectant Management [33]

- ➢ Severe Intrauterine Growth Restriction
- ➢ Non reassuring fetal heart rate
- ➢ Oligohydramnios with AFI < 5 cm
- ➢ Gestational Age of 38 weeks or greater
- ➢ Platelet count < 1 lakh per mm^3
- ➢ Progressive deterioration of hepatic function
- ➢ Progressive deterioration of Renal function
- ➢ Suspected placental abruption
- ➢ Imminent symptoms like headache, visual changes, vomiting
- ➢ Eclampsia

In case of severe preeclampsia,

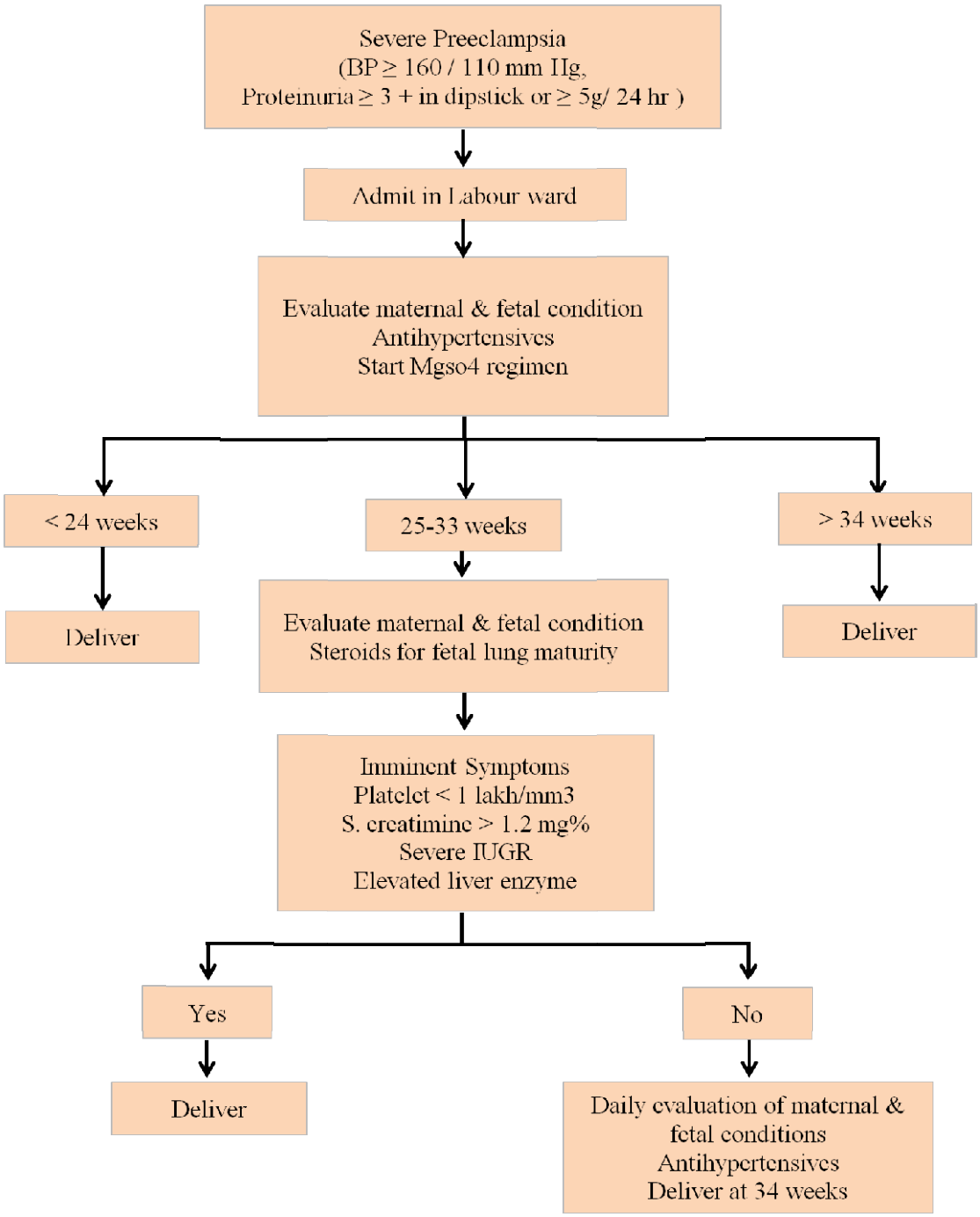
- ✓ Admit to the labour ward
- ✓ Complete fetal and maternal evaluation within 24 hours
- ✓ Start anti-hypertensives if systolic BP ≥ 160 mm Hg or Diastolic BP ≥ 110 mm Hg
- ✓ Use prophylactic Magnesium Sulphate to prevent or reduce the rate of seizures
- ✓ Inject steroids if less than 34 weeks to help speed the Infant's Lung maturity
- ✓ Terminate pregnancy immediately if there is deterioration of maternal or fetal condition

INTRAPARTUM MANAGEMENT [6]

- ✓ Hourly Blood pressure monitoring
- ✓ Urine output and signs of impending eclampsia should be looked for
- ✓ Continuous fetal heart monitoring
- ✓ Adequate pain relief by epidural analgesia to cut down catecholamine release and hypertensive response.

- ✓ Continue magnesium Sulphate in cases of severe preeclampsia
- ✓ Indicate Caesarean Section if there is worsening maternal condition, non-reassuring fetal pattern, failed induction or other obstetric indications.
- ✓ Active management of third stage of labour by giving oxytocin 5 units IV or 10 units IM to avoid post partum hemorrhage

Fig-9. Management algorithm of severe pre-eclampsia



ANTIHYPERTENSIVE THERAPY

Antihypertensive therapy during pregnancy is a double edged sword. Uncontrolled blood pressure during pregnancy will lead to increased maternal morbidity & mortality at one end. On the other end liberal use of antihypertensives will lead to in utero exposure of the fetus to these drugs and reduced fetal perfusion due to decrease in the pressure at which maternal blood perfuses the placental villous spaces. This may adversely affect the fetal growth. Hence antihypertensive therapy should be initiated based on benefit risk ratio.[35]

There are little controversies regarding the ideal blood pressure at which antihypertensive therapy should be initiated. Some studies support the start of antihypertensive at a diastolic pressure of 105 mm Hg and some at 100 mm Hg (5). Till date starting antihypertensive drugs at a systolic blood pressure of greater than or equal to 160 mm Hg or a diastolic pressure of greater than or equal to 110 mm Hg is universally accepted.

According to the current guidelines, antihypertensive treatment for blood pressure ranging from systolic pressure of 140 to 159mm Hg and a diastolic pressure of 90 to 109 mm Hg is to maintain a **target systolic blood pressure of 130 to 155 mm Hg** and a **target diastolic blood**

pressure 80 to 105 mm Hg for women without any comorbid illness. Similarly target systolic blood pressure of 130 to 139 mm Hg and a diastolic blood pressure of 80 to 89 mm Hg diastolic is to be achieved for those women with co-morbidities [36]

Drugs that can used with little side effects and higher benefit risk ratio are

1. Centrally acting adrenergic agonist (Alpha Methyldopa)
2. Beta blockers
3. Calcium channel blockers
4. Hydralazine
5. Alpha blockers
6. Clonidine

Drugs that are absolutely contraindicated for its teratogenicity are angiotensin converting enzyme inhibitors (ACE inhibitors). In utero exposure of the fetus to ACE inhibitors like **enalapril** will lead to growth restriction, oligohydraminos, prolonged hypotension, anuria and limb contractures.

ORAL PREPARATIONS

ALPHA- METHYLDOPA

This is the most commonly used drug. It is a centrally acting **alpha adrenergic agonist**. Predominantly it acts on the central nervous system with little peripheral action. It decreases the sympathetic tone and arterial blood pressure by stimulating alpha 2 receptors.

It can be started with **250 to 500 mg orally two to three times** a day upto a maximum dose of 2g. The drug effect is maximum at 4 to 6 hours and the duration of action is 10 to 12 hours.

Side effects are headache, dryness of mouth, swelling of feet, depression, postural hypotension. Rare incidences of hemolytic anemia and hepatitis have been reported,

The drug crosses the placenta, reaches the fetus and is also secreted in breast milk. But long term follow up did not show teratogenic effect with these drugs and hence considered safe during pregnancy.

BETA BLOCKERS

Labetalol which is a **combined alpha and beta blocker** is now becoming the first line therapy because of its higher efficacy and safety. Dosage is **100 to 400 mg twice daily**.

Side effects are bradycardia, dizziness, nausea, vomiting, fatigue and depression. Care should be taken when used in patients with diabetes on insulin because beta blockers mask the warning signs of hypoglycemia in them.

Other beta blockers that can be used in pregnancy are metaprolol, acebutolol and propanolol. The beta blocker that is contraindicated during pregnancy is atenolol because it causes intra uterine growth restriction, hypoglycemia and hyperbilirubinemia in the fetus.

CALCIUM CHANNEL BLOCKERS

Nifidipine is the commonly used drug and it is available as 5-10 mg capsules or tablets. Dosage can be from **10 to 20 mg three to four times a day**. Extended release tablets have been formulated to avoid the more frequent dosing and they have slower onset and a more prolonged action. Maximum dose of Nifidipine is 120 mg per day. Action starts in 10 to 15 minutes. Oral dose of 10 mg can be repeated every 30 to 60 minutes to achieve the desired range of blood pressure. Nifidipine can also be administered sublingually but oral route is preferred because of the precipitous fall in blood pressure with sublingual route.

Nifidipine when used along with magnesium sulphate will result in exaggerated hypotension because of their synergistic action in blocking

calcium channels. Also there are high chances of postpartum hemorrhage with the combined use of both due to the same reason.

Side effects of nifidipine are flushing, headache, ankle edema, nasal congestion, heart burns, nausea, hypotension and palpitations.

DIURETICS

Diuretics cause depletion of intravascular volume. Since the placenta does not have an autoregulatory mechanism, placental perfusion is dependent on the maternal plasma volume and systemic pressure. Intra vascular volume depletion can severely compromise placental perfusion which is already compromised in pre-eclampsia. Hence diuretics are contraindicated in pre-eclampsia and intra uterine growth restriction with reduced placental perfusion proved by Doppler.

Indications for diuretics in pregnancy are

1. Congestive cardiac failure
2. Acute pulmonary edema
3. Cerebral/ Intracranial tension
4. Renal failure

Fig-10: DRUGS USED IN PRE-ECLAMPSIA AND ECLAMPSIA

(a). Tablet Nifedipine:

(b). Injection Labetalol:

(c). Injection Magnesium Sulphate:

(d). Injection Hydralazine:

INTRAVENOUS PREPARATIONS

Intravenous Preparations are used in hypertensive emergencies with a high blood pressure (systolic blood pressure greater than 160 mm Hg and diastolic blood pressure greater than 110 mm Hg) to avoid complications like cerebrovascular hemorrhage, hypertensive encephalopathy, eclampsia, congestive cardiac failure, placental abruption etc.

LABETOLOL

This combined alpha and beta blocker lowers the blood pressure smoothly and rapidly. Dosage is **10 to 20 mg bolus IV**. This can be repeated every 30 minutes upto 80 mg till a maximum dose of 300 mg is reached. This can also be given in a continuous infusion form of 1 to 2 mg/ minute.

It takes five minutes for the onset of action and the peak action is obtained within 10 to 20 minutes of its administration.

Side effects are maternal and neonatal hypotension and bradycardia.

HYDRALAZINE

Hydralazine can also be administered as a bolus intravenous drug at **5mg every 20 to 30 minutes** and infusion at the rate of 0.5 to 10 mg per hour upto a maximum of 30 mg. It takes only 10 minutes to act.

Side effects are headache, anxiety, restlessness and hyperreflexia. It causes tachycardia in contrast to labetolol which causes bradycardia

Magee LA etal 2003 compared hydralazine with nifidipine for the treatment of severe hypertension. They reported that hydralazine was found to have higher incidences of hypotension, placental abruption, oliguria, low apgar scores and adverse fetal outcome.

Low apgar scores and adverse fetal outcomes associated with hydralazine are due to the release of noradrenaline by the drug which is a potent vasoconstrictor of the uteroplacental circulation. This can be overcome by correction of hypovolemia & administration of intermittent small doses.

NITROPRUSSIDE

Nitroprusside, a potent vasodilator of arterial and venous smooth muscle is short acting and highly effective, given as intra venous infusion at 0.25 μg/ kg/ minute to start with and can be increased to a maximum of 8 μg/ kg/ minute.

It takes less than 1 minute for the onset of action and acts only for 1 to 3 minutes. The main adverse effect seen with nitroprusside is cyanide toxicity to the fetus.

ECLAMPSIA

Eclampsia is defined as the development of Generalised Tonic Clonic Seizures that cannot be attributed to other causes and unexplained coma during pregnancy or puerperium in a woman with preeclampsia. It can be classified into

- Antepartum
- Intrapartum
- Postpartum
- Late Postpartum

COMPLICATIONS [6]

The complications of Eclampsia could be maternal and fetal as follows

MATERNAL

- Maternal injury
- Abruptio placenta
- Aspiration pneumonia
- Status eclampticus
- Pulmonary edema
- Cardiopulmonary arrest
- Acute Renal failure
- Disseminated Intravascular Coagulation
- Coma
- Maternal death

FETAL

- Fetal bradycardia
- Hypoxic Ischaemic Encephalopathy
- Intrauterine death

MANAGEMENT OF ECLAMPSIA

Eclampsia is a life threatening complication of pre-eclampsia which has to be addressed with zero delay.

Management of Eclampsia is a team work involving an Obstetrician, Anesthetist, Physician and a Neurologist.

The four main key components of management of Eclampsia are

- Immediate resuscitative measures
- Antihypertensives
- Anticonvulsants
- Obstetric management

Clearing the airways – With the patient in left lateral position, airway should be cleared of any secretions and vomitus to reduce the risk of aspiration.[6]

Control of seizures – The protocol for magnesium sulphate administration is as follows: [37]

LOADING DOSE

4g of 20% magnesium Sulphate is given IV slowly over 5 minutes followed by intravenous infusion of 1g hourly (or) **10 g of 50%**

solution intramuscularly (5g in each buttock) with 1 mL of 2% Lignocaine in the same syringe.

MAINTANENCE DOSE

5g of Magnesium Sulphate (50% solution) with 1 ml of Lignocaine 2% in the same syringe every 4 hours into alternate buttock.

Continue the treatment with Magnesium Sulphate for 24 hours after delivery or after the last fit whichever is later.

Magnesium Sulphate Toxicity is monitored with respiratory rate, patellar reflexes and urine output.

Repeat doses of Magnesium Sulphate must be withheld or delayed if

- Respiratory rate is < 16 per minute
- Patellar reflexe is absent
- Urine output is < 30 mL/hr over the preceding 24 hours.

In case of respiratory arrest

- Give Calcium Gluconate 1g (10 mL of 10% solution) slow IV until respiration is satisfactory
- Assist ventilation using bag and mask, anesthetic apparatus or intubation

Control of Blood Pressure :

Parenteral anti-hypertensives such as IV labetalol can be administered in uncontrolled hypertension.[6][27]

General Care and Fluid Care :

Ringer Lactate solution is administered at the rate of 60-125 ml/hr after calculating the fluid loss. Diuretics should be avoided unless there is evidence of pulmonary edema.[27]

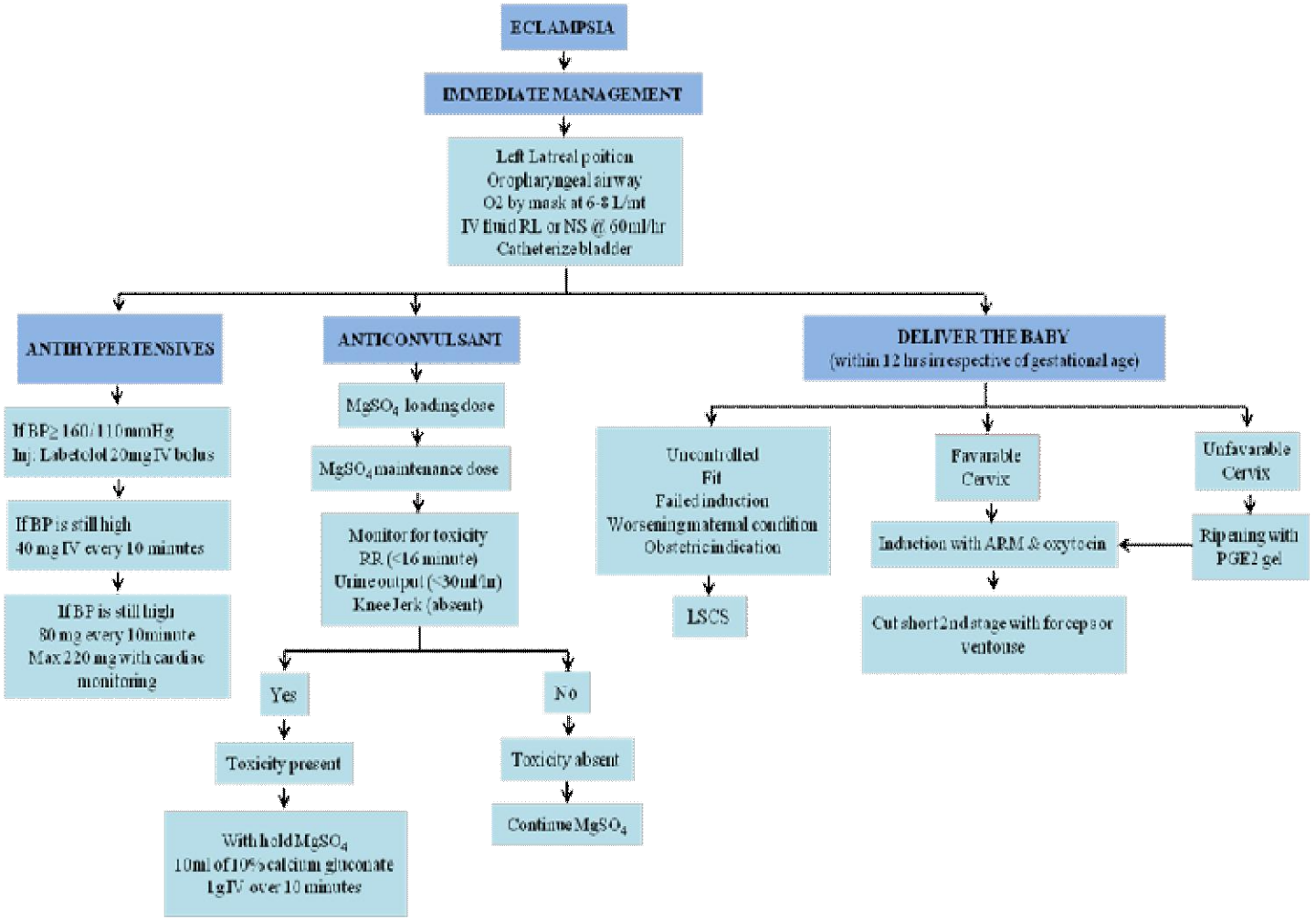
Termination of pregnancy :

The definitive management of Eclampsia is termination of pregnancy irrespective of the gestational age.

Patient must be delivered within 24 hours in case of Severe Preeclampsia and within 12 hours in case of Eclampsia.

Mode of delivery is planned based on the obstetric indication.[6]

Fig-11. MANAGEMENT ALGORITHM OF ECLAMPSIA



HELLP SYNDROME

HELLP Syndrome is a life threatening complication of severe preeclampsia usually occurring during the later stages of pregnancy or sometimes after childbirth characterized by[38]

- ✓ **Hemolysis**
- ✓ **Elevated Liver Enzymes**
- ✓ **Low Platelet count**.

The diagnosis of HELLP Syndrome is based on either of the two criterias namely **Mississippi and Tennessee criterias**. A set of blood investigations is required to diagnose HELLP syndrome based on the above criteria and it includes complete blood count, coagulation profile, liver enzymes, renal function test, serum electrolytes and serum LDH. A positive **D-Dimer test** in the presence of pre-eclampsia was reported to predict HELLP syndrome.[39]

PATHOPHYSIOLOGY

The inciting factor is abnormal vascular tone, vasospasm and microvascular endothelial damage leading to intravascular platelet activation and coagulation defects. Activation of platelets result in the release of thromboxane A2 and serotonin, causing further vasospasm,

platelet agglutination and aggregation and endothelial damage. This cascade is only terminated with delivery.[40]

MISSISSIPPI DIAGNOSTIC CRITERIA

This diagnostic criteria was proposed by The University of Mississippi in 1999 & alternative classification was introduced in the year 2006.

Table 2- Mississippi Diagnostic Criteria

Parameters	**Criteria**
Hemolysis	❖ Serum Bilirubin >1.2 mg/dl ❖ Lactate Dehydrogenase >600 IU/L ❖ Progressive Anemia
Elevated liver enzymes	❖ AST ≥ 40 IU/L ❖ ALT ≥ 40 IU/L
Low Platelet Count	❖ Platelet Count < 1,50,000/mm3 ❖ Alternative Classification • Class 3: 100,000 to 150,000/mm3 • Class 2: 50,000 to 100,000/mm3 • Class 1: <50,000/mm3

Prothrombin time, partial thromboplastin time and fibrinogen level are usually normal in HELLP syndrome[41]

Table 3- Tennessee Diagnostic Criteria

Parameters	Criteria
Hemolysis	❖ Serum Bilirubin >1.2 mg/dl ❖ Lactate Dehydrogenase >600 IU/L ❖ Peripheral Smear • Schistocytes • Burr Cells • Helmet Cells
Elevated liver enzymes	❖ AST ≥ 70 IU/L
Low Platelet Count	❖ Platelet Count < 100,000/mm3

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of HELLP syndrome include acute fatty liver of pregnancy, thrombotic thrombocytopenic purpura, hemolytic uremic syndrome, viral hepatitis, gall bladder diseases, gastroenteritis, kidney stones, pyelonephritis, encephalopathy and hyperemesis gravidarum.

SYMPTOMS

- ❖ Epigastric / Right upper quadrant pain
- ❖ Nausea/Vomiting
- ❖ Headache / Malaise
- ❖ Visual changes
- ❖ Jaundice

COMPLICATIONS OF HELLP

- ✓ Placental abruption
- ✓ DIC
- ✓ Acute Renal Failure
- ✓ Severe Ascites
- ✓ Cerebral Edema
- ✓ Pulmonary Edema
- ✓ Wound Hematoma
- ✓ Subcapsular Liver Hematoma
- ✓ Liver rupture

Women with history of HELLP syndrome in the previous pregnancy is at a high risk of developing pre-eclampsia in the present pregnancy and the risk is estimated to be about 20%.[42]

MANAGEMENT OF HELLP

- Deliver rather than expectant management for pregnancies more than 34 weeks
- For pregnancies less than 34 weeks, deliver after a course of steroids
- Other modalities in HELLP syndrome are antithrombotics, Dialysis, Plasma exchange with FFP & Plasmapheresis[6]

Fig-12. Abnormal CT showing a subcapsular hematoma in a woman with HELLP syndrome

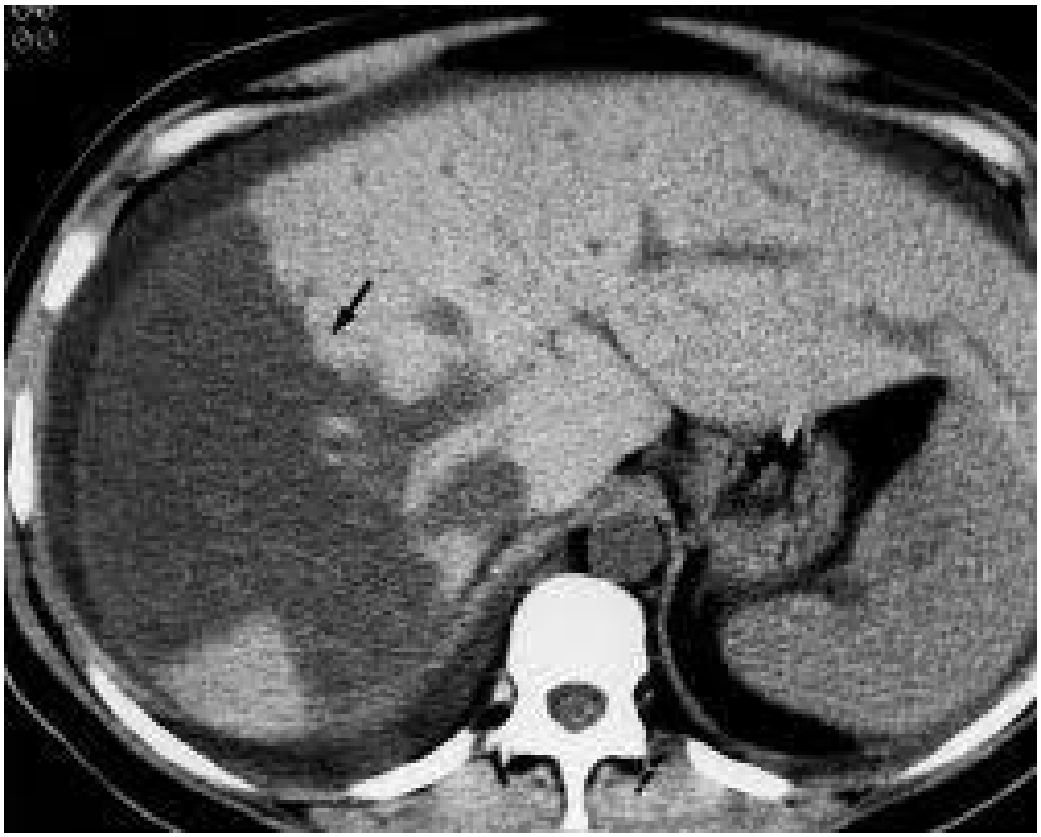
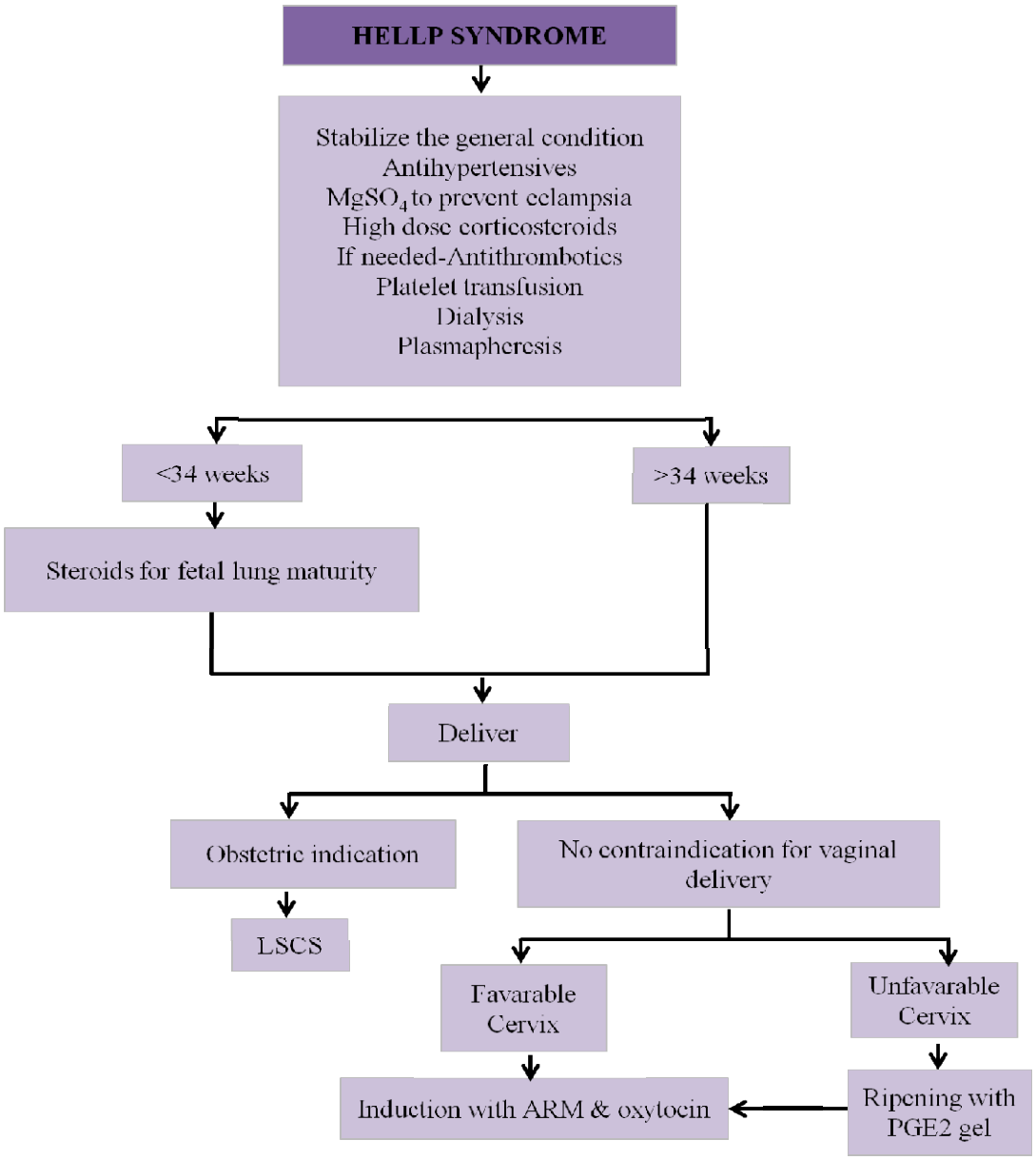


Fig-13. Management algorithm of HELLP syndrome



PREVENTIVE MEASURES OF PRE-ECLAMPSIA

- **Collaborative Low Dose Aspirin Study in pregnancy (CLASP)** studied the role of low dose aspirin in pregnancy for the prevention of pre-eclampsia. .

The study showed a non-significant reduction of 12% in pre-eclampsia but there was a significant reduction of proteinuric pre-eclampsia in women prone to develop early onset pre-eclampsia .The only drug recommended for prevention of pre-eclampsia is low dose aspirin in some women at high risk of developing the disease [43]

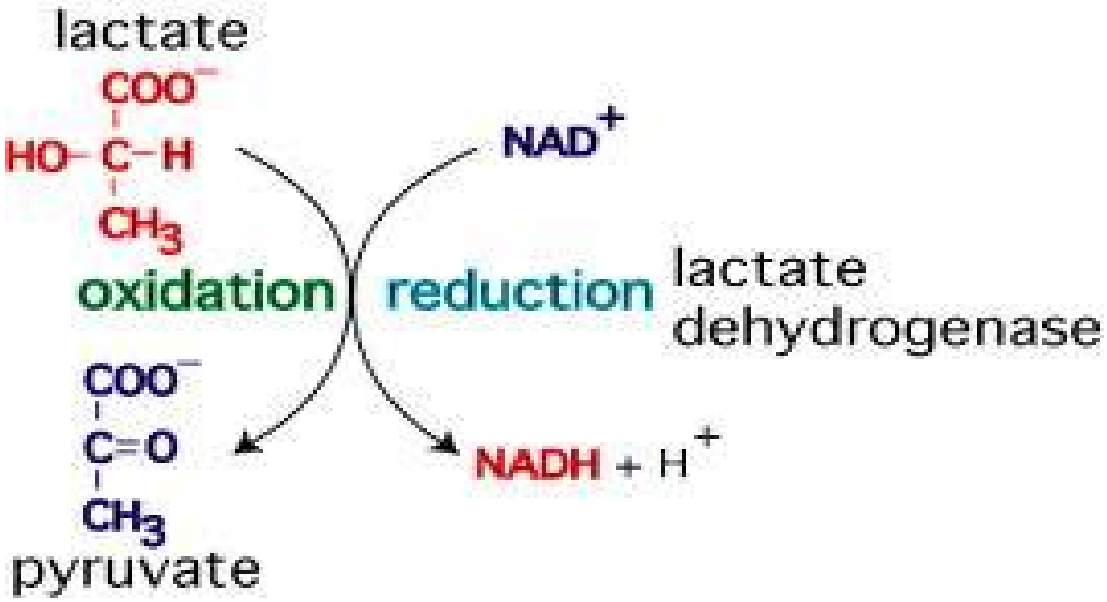
- **Calcium supplementation** with 1.5 to 2g of Calcium carbonate or elemental calcium from various preparations has shown to reduce the incidence of pre-eclampsia almost by half.[44]
- Pharmacological agents such as **Low molecular weight Heparin**, Progesterone, nitric Oxide donors, anti-hypertensive medication and diuretics are not effective in preventing pre-eclampsia[6]
- No correlation was observed between **dietary salt intake** and risk of pre-eclampsia[45]
- The effects of **rest, physical activity or exercise** in the development of pre-eclampsia are still not clearly understood.[6]
- The effect of Zinc and antioxidants supplementation are controversial.[6]

LACTATE DEHYDROGENASE

Systematic Name: L-Lactate NAD^+ Oxidoreductase[46]

It catalyses the interconversion of Lactate and pyruvate. LDH is 100 times more in RBC than in plasma. [46]

Fig-14. Reaction catalysed by LDH



LDH has 5 distinct isoenzymes (LDH 1, 2, 3, 4, 5). They can be separated by electrophoresis (cellulose starch gel, agarose gel). LDH_1 has more negative charge and fastest in electrophoretic mobility while LDH_5 is the slowest. [47]

STRUCTURE OF LDH ISO-ENZYME

LDH is an oligomeric (tetrameric) enzyme made up of four polypeptide subunits. Two types of subunits namely M (muscle) and H (Heart) are produced by different genes. M Subunit is basic while H subunit is acidic. The isoenzymes contain either 1 or both subunits giving LDH1 to LDH5. [46]

M Subunit is encoded by LDH-A located on chromosome 11p15.4.

H subunit is encoded by LDH-B located on chromosome 12p12.2.

A third isoform named LDH-C or LDH-X is expressed only in testis encoded by chromosome 11p15.5.

Preeclampsia causes increase in total LDH.

LDH-1 (H_4) is predominantly found in heart muscle and is inhibited by pyruvate – the substrate. Hence pyruvate is not converted to lactate in cardiac muscle but is converted to acetyl CoA which enters Citric Acid Cycle. It has high K_m (low affinity) for pyruvate and acts well in aerobic conditions

LDH-5 (M_4) is mostly present in skeletal muscle and inhibition of this enzyme by pyruvate is minimal and hence pyruvate is converted to lactate. It has low K_m (high affinity) for pyruvate and acts well in anaerobic conditions.

Fig-15. Structure of LDH iso-enzyme

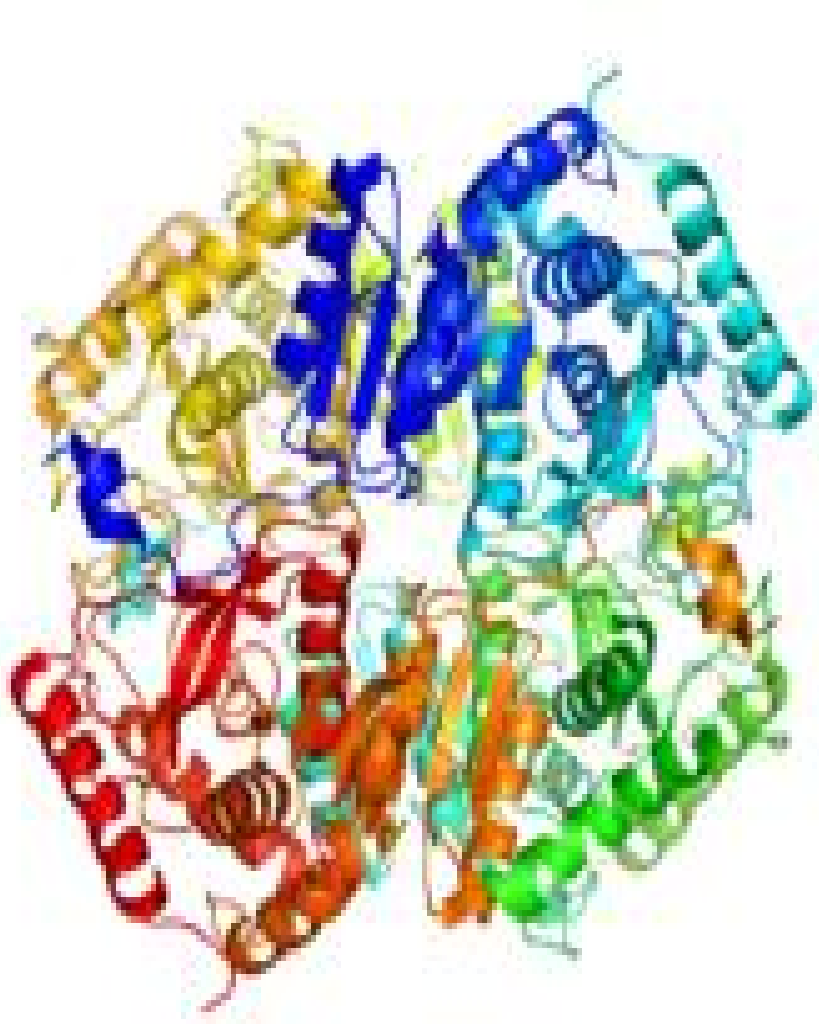


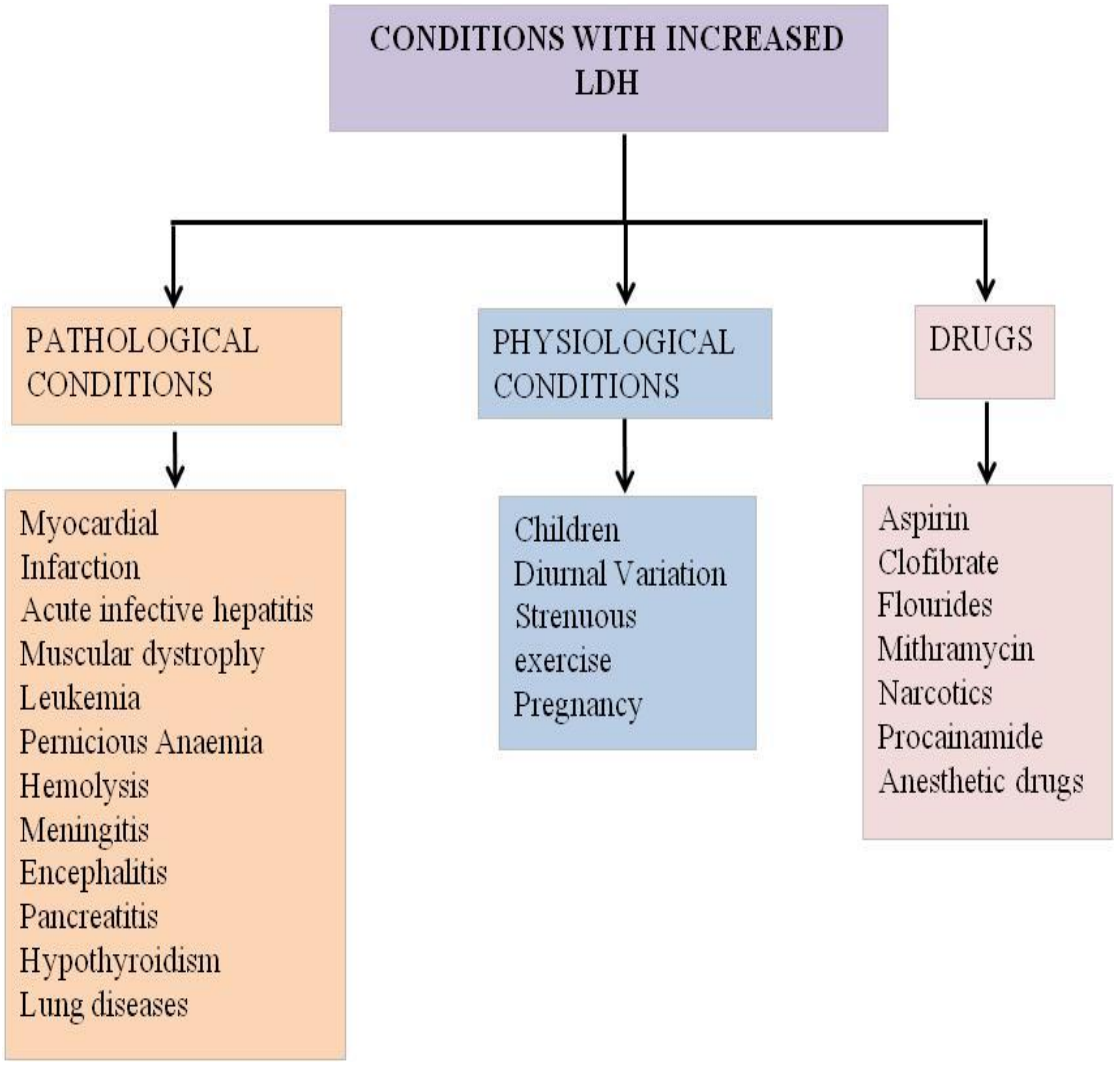
Table 4 - Sub Types of S.LDH

Type	Subunit	Electrophoretic mobility	Activity at 60^0 for 30 mins	Tissue	% in serum
LDH_1	H_4	Fastest	Not destroyed	Heart	30%
LDH_2	H_3M	Faster	Not destroyed	RBC	35%
LDH_3	H_2M_2	Fast	Partially destroyed	Brain	20%
LDH_4	H_1M_3	Slower	Destroyed	Liver	10%
LDH_5	M_4	Slowest	Destroyed	Skeletal muscle	5%

Normal values of LDH

- Non pregnant adult - 115 to 211 IU/L
- First trimester – 78 to 433 IU/L
- Second trimester -80 to 447 IU/L
- Third trimester – 82 to 524 IU/L

Fig-16. Conditions with increased LDH



LDH is decreased with high doses of Vitamin-C

STUDIES ON LDH IN PREGNANCY

- **S.P Jaiswar, Amrit Gupta and Mohan Shaili at CSSM, Lucknow** studied S.LDH levels in 146 women with 39 normotensives, 35 mild pre-eclamptics, 36 severe pre-eclamptics and 36 Eclamptics and stated that LDH levels were significantly elevated in women with pre-eclampsia and Eclampsia ($p<0.001$), high blood pressure ($p<0.10$) as well poor maternal and perinatal outcome.[48]

- Similar studies were done by S**.M. Munde, N.R. Hazari, A.P. Thorat, S.B. Gaikwod & V.S. Halolkar** to examine the possible role of S.GGT & S.LDH in the prediction of severity of pre-eclampsia. The study comprised of 40 pre-eclampsia and 40 normotensive pregnant controls. S.LDH levels were found to be significantly elevated in mild pre-eclamptic women ($p<0.05$) and severe pre-eclamptic women ($P<0.001$)[3]

- **He S, Bremme K, Kallner A and Blomback M** studied Lactate Dehydrogenase as a predictor for the birth of small for gestational age infants. The study included 26 normotensives and 51 pre-eclamptic women. They stated that pre-eclamptic women with

SGA infants had significantly higher LDH concentrations than those in the appropriate for gestational age group[2].

- **Qublan et al** studied S. LDH levels of 171 women with 49 mild preeclamptic, 62 severe preeclamptic & 60 nomotensive women. He compared S. LDH levels with systolic blood pressure, diastolic blood pressure, uric acid, uric albumin & liver enzymes and found a significant correlation between them.[4]

- **Bakskandeh et al** studied S. LDH in 50 preeclampsia & 50 normotensive women & stated that LDH level was not statistically different between healthy and preeclamptic women.[49]

- A study on LDH profile as a retrospective indicator of uterine preparedness for labour were done by **Jeremy et el**. They stated that more efficient cervical dilatation following labour admission is associated with a more anaerobic maternal S.LDH profile in post delivery period.[50]

STUDY DESIGN

Observational Study

METHOD OF STUDY

This study involves the measurement of S.LDH in a total of 173 antenatal patients of gestational age 28weeks and above.

INCLUSION CRITERIA

- ❖ Group 1 = 50 Normotensives (Controls)
- ❖ Group 2 = 123 Hypertensives (Cases) Including
  - Mild Pre-eclampsia –BP of 140/90 to < 160/ 110 mmHg
  - Severe Pre-eclampsia –BP ≥ 160/ 110 mmHg
  - Eclampsia – One or more episode of GTCS

attending antenatal OPD/ Labour ward in Chengalpattu Medical College Hospital during the period of November 2013 ~ August 2014. Patients were selected based on inclusion and exclusion criteria after obtaining their consent **(Annexure II).** A detailed history, clinical examination and all necessary investigations were done. **(Annexure IV)**

EXCLUSION CRITERIA

1. Mothers with hypertension at < 20 weeks gestation,
2. Preexisting diabetes mellitus,
3. Renal disease,
4. Liver disorder
5. Thyroid disorder
6. Epilepsy
7. Heart disease
8. Muscular dystrophy
9. Leukemia
10. Pernicious Anemia
11. Hemolysis and other causes of increased LDH
12. HIV reactive women
13. Meningitis

METHOD OF LDH ESTIMATION

1 ml of venous blood sample was taken under all aseptic precautions. It was then allowed to clot and then centrifuged for serum

separation. Estimation of S. LDH is done by **International Federation of Clinical Chemistry** (IFCC) method.

Assay: Crest Biosystems

Principle:

Lactate dehydrogenase catalyses the reduction of pyruvate with NADH to form NAD.

Total LDH is tested by reaction of serum sample with lactate and NAD.

$$\mathbf{L + Lactate + NAD^{+} \rightarrow Pyruvate + H^{+} + NADH}$$

NADH has absorbance maximum at **340 nm**. The optimal conditions for the reaction are temperature of **30 ± 0.05°C** and **pH of 9.40 ± 0.05**. After a definite time NADH is used up by the reaction and the decrease in absorbance will be proportional to enzyme activity detected by **spectrophotometer.**

Sample material:

Serum free from hemolysis. Total LDH is reported to be stable in serum for 1-3 days at 2-8°C. Freezing inactivates the liver isoenzyme.

Procedure:

Wavelength/ filter : 340nm

Temperature : 30°C

Light path : 1cm

Substrate Start Assay:

Pipette into a clean dry test tube labeled as Test (T):

Addition Sequence	Temp= 30°C
Buffer Reagent Sample	0.8ml 0.05ml
Incubate at the assay temperature for 1minute and add	
Starter Reagent	0.2ml

Mix well and read the initial absorbance A0 & repeat the absorbance reading after every 1, 2 & 3 minutes. Calculate the mean absorbance change per minute (ΔA/min.)

Sample Start Assay:

Pipette into a clean dry test tube labeled as Test (T):

Addition Sequence	Temp=30°C
Working Reagent	1.0 ml
Incubate at the assay temperature for 1 minute and add	
Sample	0.05ml

Mix well and read the initial absorbance A0 & repeat the absorbance reading after every 1, 2 & 3 minutes. Calculate the mean absorbance change per min (ΔA/min.)

Calculations:

Substrate / Sample Start

LDH Activity in U/L ($30^{o}C$) = ΔA/min X 3333

Patients were also divided according to the serum LDH levels into

1. <600 IU/l
2. 600–800 IU/l
3. >800 IU/l

- ❖ All women were followed until delivery and early postpartum period.
- ❖ S.LDH values were correlated with the severity of the disease in terms of maternal complications like HELLP, Eclampsia, Abruptio Placenta, maternal death & fetal complications like IUGR, still birth, late IUD.
- ❖ Factors taken for analysis were age, parity and gestational age in weeks and mode of Delivery.

- Definitions used for defining pre-eclampsia were according to International Society for the Study of Hypertension in Pregnancy **(ISSHP).** Classification into mild and severe pre-eclampsia were based on **NHBPEP** classification.

- **HELLP syndrome** was diagnosed based on **Mississippi criteria**.

- **IUGR**

Fetuses with a birth weight less than 10^{th} percentile of those born at the same gestational age or two standard deviation below the population mean were considered IUGR. (5).

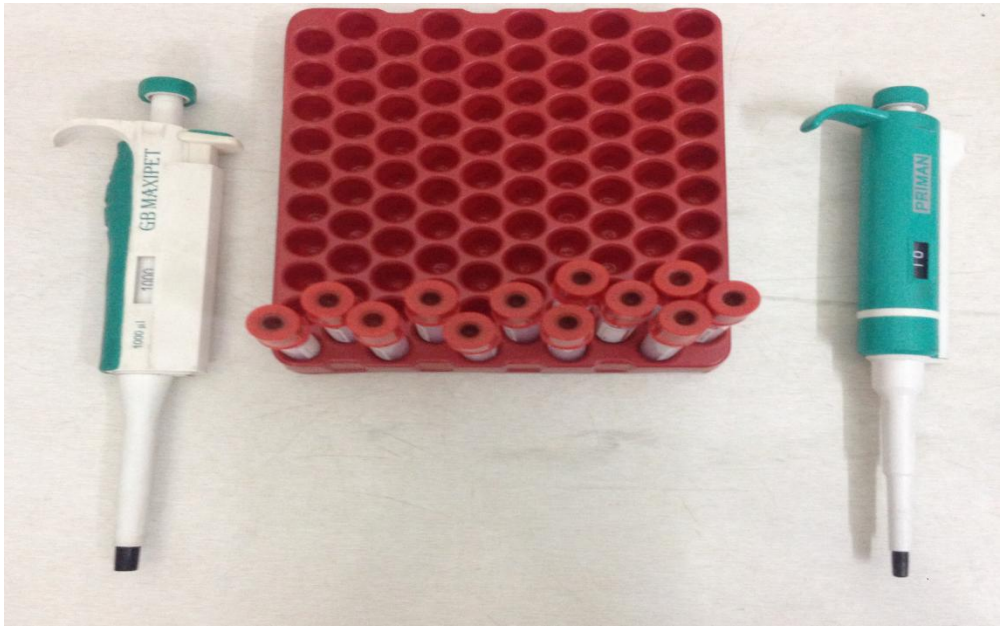
- **IUD**

Death prior to complete expulsion or extractions from its mother of a product of human conception **after 28 weeks** of gestations is considered as late intra uterine fetal death. (31).

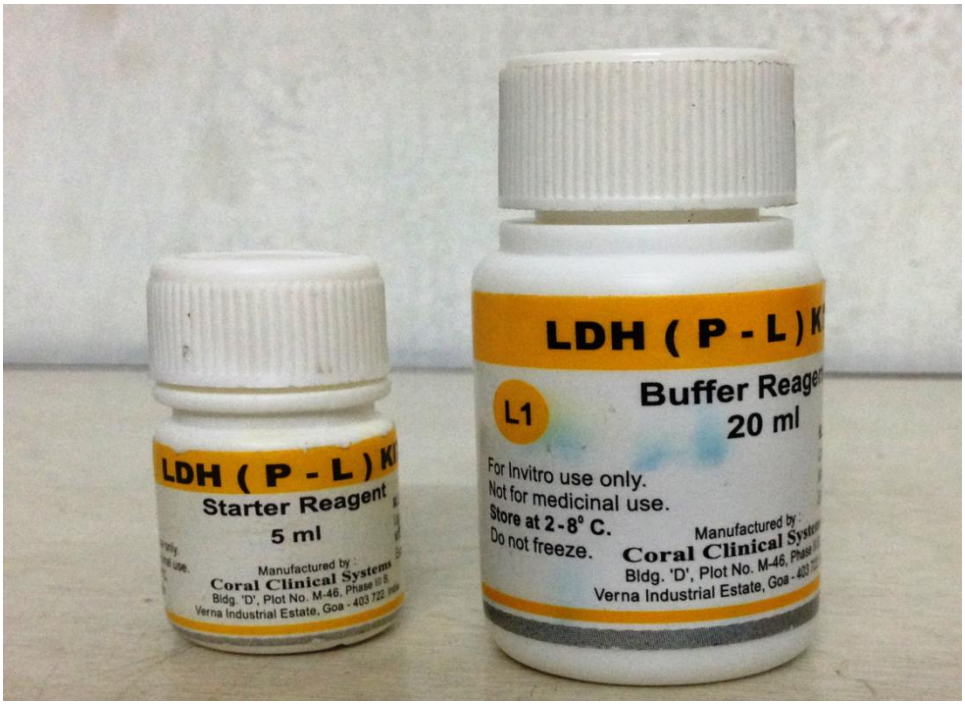
Fig-17. Materials Used in LDH estimation

(a). Centrifuge

(b). Pipette



(c). Reagent



(d). Instruction Sheet

LDH (P - L) KIT

(Mod. IFCC method)

For the determination of Lactate Dehydrogenase activity in serum.
(For Invitro Diagnostic Use Only)

Summary

LDH is found in many body tissues particularly heart, liver, skeletal muscle, kidney and RBC's. LDH is found in the form of isoenzymes based on their electophoretic mobility with each isoenzyme being primarily from different organs. Increased levels are found in myocardial infarction, pulmonary diseases, hepatic diseases, hemolytic anemias, renal diseases and muscular dystrophy.

Principle

Lactate dehydrogenase catalyzes the reduction of pyruvate with NADH to form NAD. The rate of oxidation of NADH to NAD is measured as a decrease in absorbance which is proportional to the LDH activity in the sample.

$$\text{Pyruvate} + \text{NADH} + H^+ \xrightarrow{\text{LDH}} \text{Lactate} + \text{NAD}^+$$

Normal reference values

Serum : 230 - 460 U/L at 37°C

It is recommended that each laboratory establish its own normal range representing its patient population.

Contents	25 ml	2 x 75 ml
L1 : Buffer Reagent	20 ml	2 x 60 ml
L2 : Starter Reagent	5 ml	2 x 15 ml

Storage / Stability

Contents are stable at 2-8°C till the expiry mentioned.

Reagent Preparation

Reagents are ready to use.

Working reagent : For sample start assays a single reagent is required. Pour the contents of 1 bottle of L2 (Starter Reagent) into 1 bottle of L1 (Buffer Reagent). This working reagent is stable for at least 1 week when stored at 2-8°C.
Alternatively for flexibility as much of working reagent may be made as and when desired by mixing together 4 parts of L1 (Buffer Reagent) and 1 part of L2 (Starter Reagent). Alternatively 0.8ml of L1 and 0.2 ml of L2 may also be used instead of 1 ml of the working reagent directly during the assay.

Sample material

Serum. Free from hemolysis. Total LDH is reported to be stable in serum for 1 - 3 days at 2-8°C. **Freezing inactivates the liver isoenzyme.**

Procedure

Wavelength / filter : 340 nm
Temperature : 37°C / 30°C / 25°C
Light path : 1 cm

Substrate Start Assay :

Pipette into a clean dry test tube labeled as Test (T) :

Addition Sequence	(T) 25°C / 30°C	(T) 37°C
Buffer Reagent	0.8 ml	0.8
Sample	0.05ml	0.02
Incubate at the assay temperature for 1 minute and add		
Starter Reagent	0.2 ml	0.2 ml

Mix well and read the initial absorbance A_0 & repeat the absorbance reading after every 1, 2, & 3 minutes. Calculate the mean absorbance change per minute (ΔA / min.)

OBSERVATION AND RESULTS

A total of 173 antenatal women were recruited from Outpatient department/ Labour Ward at Chengalpattu Medical College & Hospital from November 2013 ~ August 2014

All patients were of gestational age 28 weeks and above. Patients were selected irrespective of the age and parity and they were divided into three groups based on NHBPEP classification as 50 normotensives, 50 mild pre-eclamptics, 50 severe pre-eclamptics & 23 eclamptics. Patients were also divided into three groups based on their S. LDH (less than 600, 600 to 800, and more than 800 IU/l).

Influences of age and parity on S. LDH were analyzed using appropriate statistical test.

correlation between S. LDH and maternal and fetal complications were studied using appropriate statistical test.

One-way analysis of variance (ANOVA) and the chi-square test were used to compare the results. Differences were considered significant when $p<0.05$.

The following observation were made and results derived from this study.

Table 5- Distribution of Cases based on Age

Age Group	Controls		Mild pre-eclampsia		Severe pre-eclampsia		Eclampsia	
	n	%	n	%	n	%	n	%
19 & Below	1	2	1	2	1	2	0	0
20 to 23	26	52	20	40	27	54	14	60.87
24 to 28	17	34	24	48	16	32	6	26.09
29 & Above	6	12	5	10	6	12	3	13.04
Total	50	100	50	100	50	100	23	100

Distribution of age in between groups were analysed using ANOVA test. The mean age of patients in Controls, mild pre-eclampsia, severe pre-eclampsia & eclampsia are 23.64, 24.46, 24.08 & 23.95 and their standard deviations are 3.08, 3.69, 3.41 & 3.92 respectively. F Value is 0.47; **P Value is 0.70.**

Inference:

There is no statistically significant difference in the distribution of age between groups.

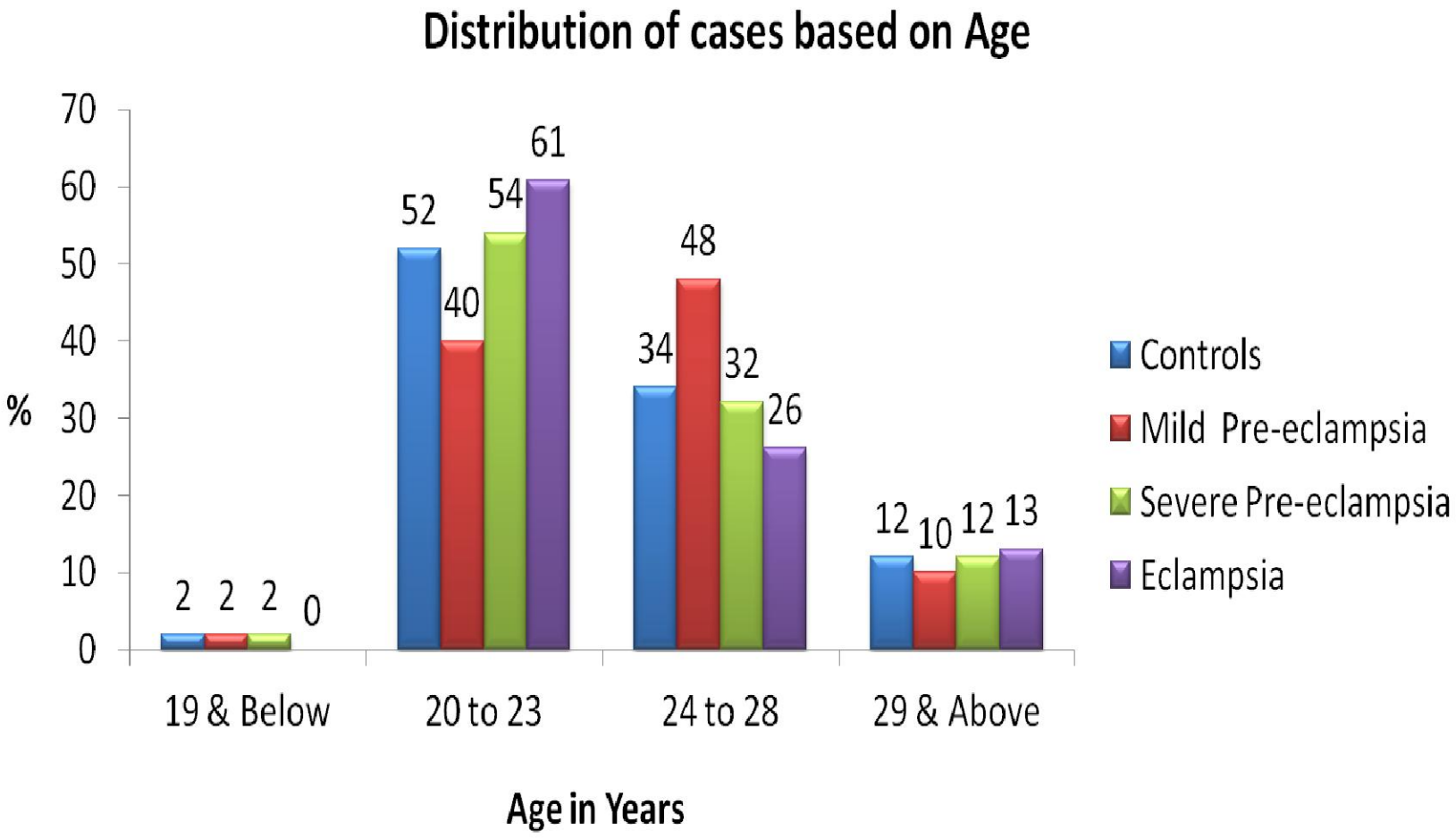

Distribution of cases based on Age
70
60
50
40
30
20
10
0
%
2 2 2 0
52 40 54 61
34 48 32 26
12 10 12 13
19 & Below
20 to 23
24 to 28
29 & Above
Age in Years
Controls
Mild Pre-eclampsia
Severe Pre-eclampsia
Eclampsia

Table 6- Correlation between S.LDH & Age

S.LDH	No	Mean Age ± Std. Deviation
<600	121	23.95 ± 3.48
600 to 800	20	24.10 ± 2.75
> 800	32	24.34 ± 3.82

Out of the total 173 women 121 patients had S. LDH less than 600, 20 had values between 600 and 800 and 32 had values more than 800.

The Mean age of the patients with S.LDH less than 600, 600 to 800 and more than 800 are 23.95, 24.10 and 24.34 and their standard deviations are 3.48, 2.75 and 3.82 respectively.

2-Tailed test was applied and **P value of 0.21** was obtained

Inference:

There is no statistically significant correlation between age and S.LDH.

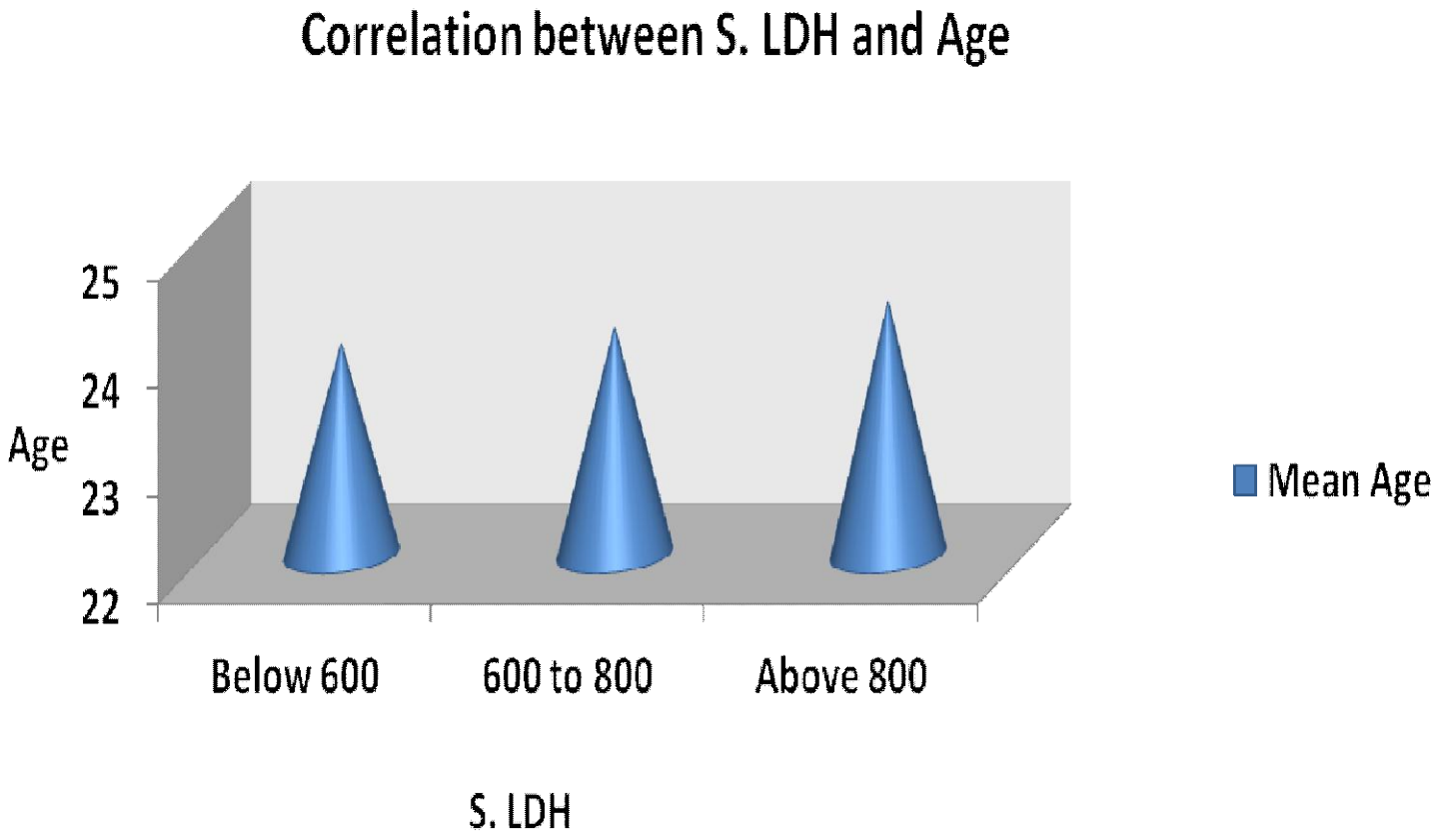

Correlation between S. LDH and Age
25
24
23
22
Age
Below 600
600 to 800
Above 800
S. LDH
Mean Age

Table 7- Distributions of Cases Based on Parity

Parity	Total	Controls		Mild pre-eclampsia		Severe pre-eclampsia		Eclampsia	
		n	%	n	%	n	%	n	%
G1	104	25	50	29	58	32	64	18	78
G2	49	21	42	14	28	12	24	2	9
G3	15	3	6	6	12	3	6	3	13
G4 & Above	5	1	2	1	2	3	6	0	0
Total	173	50	100	50	100	50	100	23	100

The study included 104 primigravida and 69 women with gravida 2 and above irrespective of the outcome of previous pregnancies. Distribution of parity in between groups were analysed using chisquare test, chisquare value is 13.62 & **P value is 0.14.**

Inference:

There is no statistically significant difference in the distribution of parity between groups.

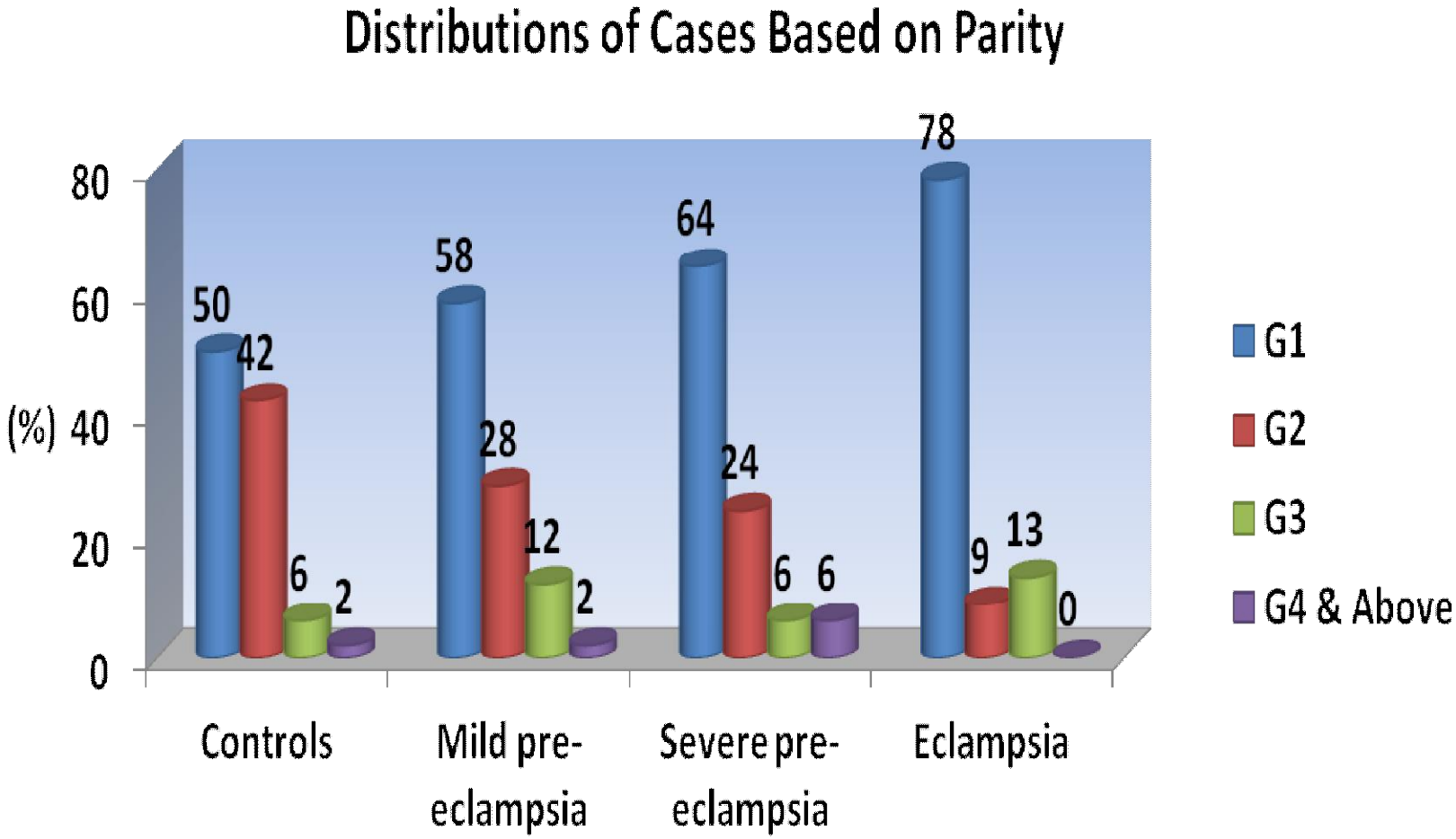

Distributions of Cases Based on Parity
80
60
40
20
0
(%)
50
42
6
2
58
28
12
2
64
24
6
6
78
9
13
0
Controls
Mild pre-eclampsia
Severe pre-eclampsia
Eclampsia
G1
G2
G3
G4 & Above

Table 8 - Correlation between S. LDH & Parity

S.LDH	Parity							
	G1		G2		G3		G4 & Above	
	n	%	n	%	n	%	n	%
<600	71	58.68	40	33.06	8	6.61	2	1.65
600 to 800	11	55.00	5	25.00	4	20.00	0	0.00
>800	22	68.75	4	12.50	3	9.38	3	9.38

Out of the 121 women with S. LDH less than 600, 58.68% women were primigravida and 41.32 % were gravida 2 and above

Out of the 20 women with LDH values between 600 and 800, 55.00% were primigravida and 45.00% were gravida 2 and above

Out of the 32 women with LDH more than 800, 68.75% were primigravida and 31.25% were gravida 2 and above

Influence of parity on S.LDH was studied using TUKEY HSD test.

Table 8.1- Influence of Parity on S. LDH

Parity	Parity	Mean Difference of LDH	Std. Error	Sig.
G1	G2	170.358	85.325	0.193
	G3	-115.446	136.006	0.831
	G4 & Above	-407.646	225.454	0.273
G2	G1	-170.358	85.325	0.193
	G3	-285.804	145.309	0.205
	G4 & Above	-578.004	231.186	0.063
G3	G1	115.446	136.006	0.831
	G2	285.804	145.309	0.205
	G4 & Above	-292.2	254.291	0.66
G4 & Above	G1	407.646	225.454	0.273
	G2	578.004	231.186	0.063
	G3	292.2	254.291	0.66

Inference:

Parity is not statistically related to the changes in S.LDH

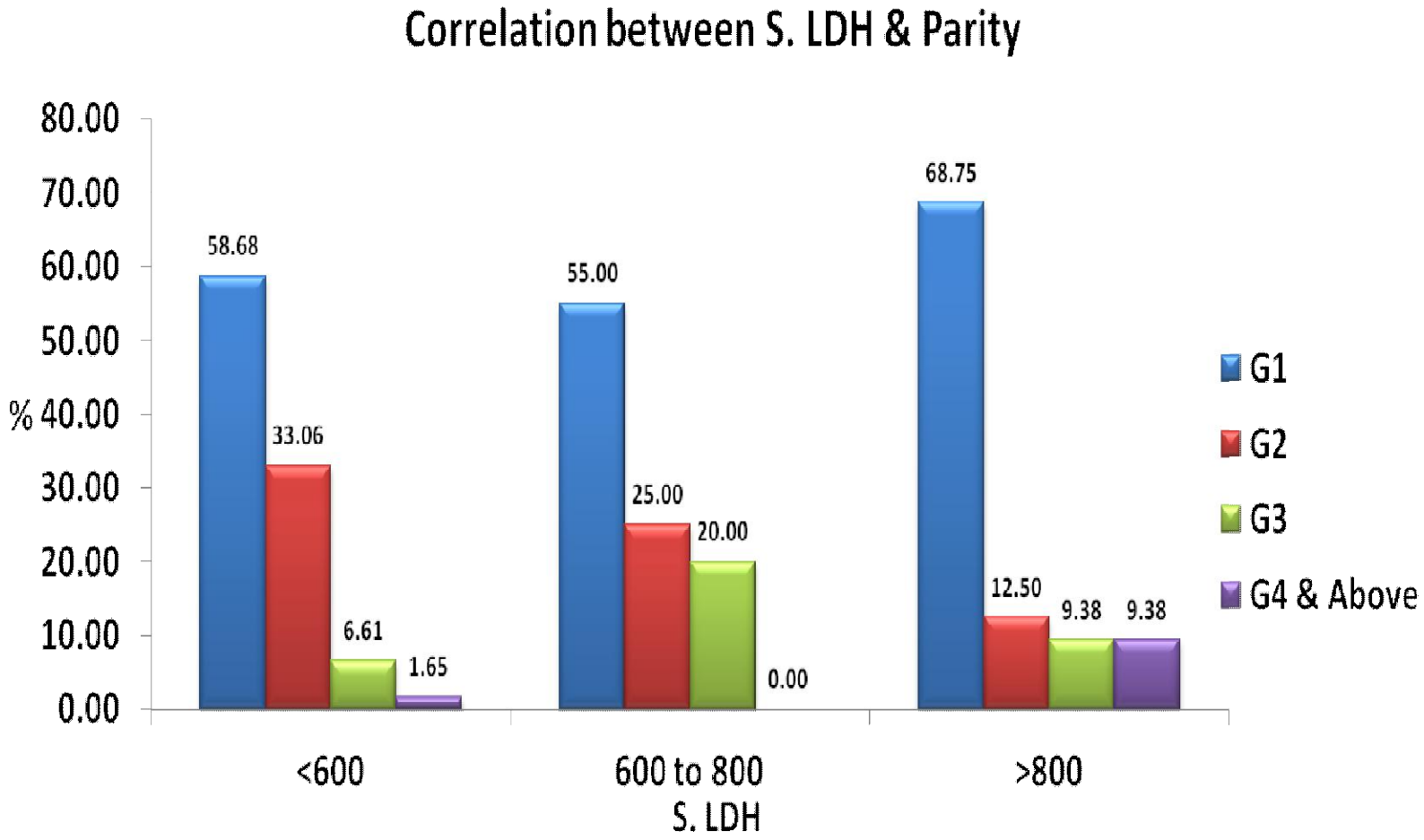

Correlation between S. LDH & Parity
80.00
70.00
60.00
50.00
% 40.00
30.00
20.00
10.00
0.00
58.68
33.06
6.61
1.65
55.00
25.00
20.00
0.00
68.75
12.50
9.38
9.38
<600
600 to 800
>800
S. LDH
G1
G2
G3
G4 & Above

Table 9- Comparison of S. LDH in Cases & Controls

S.LDH	Controls	Mild pre-eclampsia	Severe pre-eclampsia	Eclampsia
Mean ± Std Deviation	275.4 ± 108.36	381.42 ± 178.93	660.84 ± 456.08	1360.43 ± 677.13
Max	494	793	1600	3200
Min	101	109	104	340
Range	393	684	1496	2860

S.LDH values between groups were analysed using ANOVA test.

The mean S.LDH value of controls, mild pre-eclampsia, severe pre-eclampsia and eclampsia are 275.4, 381.42, 660.84 & 1360.43 and their standard deviations are 108.36, 178.93, 456.08 & 677.13 respectively.

TUKEY HSD test was applied to study the difference in S. LDH between groups

Table 9.1- Difference in S. LDH between Groups

Group	Group	Mean Difference of S. LDH	Std. Error	Sig.
Mild pre-eclampsia	Controls	106.02	72.853	0.47
Severe pre-eclampsia	Controls	385.440	72.853	0.00
Severe pre-eclampsia	Mild pre-eclampsia	279.420	72.853	0.00
Eclampsia	Controls	1085.035	91.776	0.00
Eclampsia	Mild pre-eclampsia	979.015	91.776	0.00
Eclampsia	Severe pre-eclampsia	699.595	91.776	0.00

Inference:

There is no statistically significant difference in the S. LDH levels between controls and mild pre-eclampsia with P value of 0.47 but as the severity of the disease goes up there is a significant increase in S.LDH as shown in the above table.(p iess than 0.00)

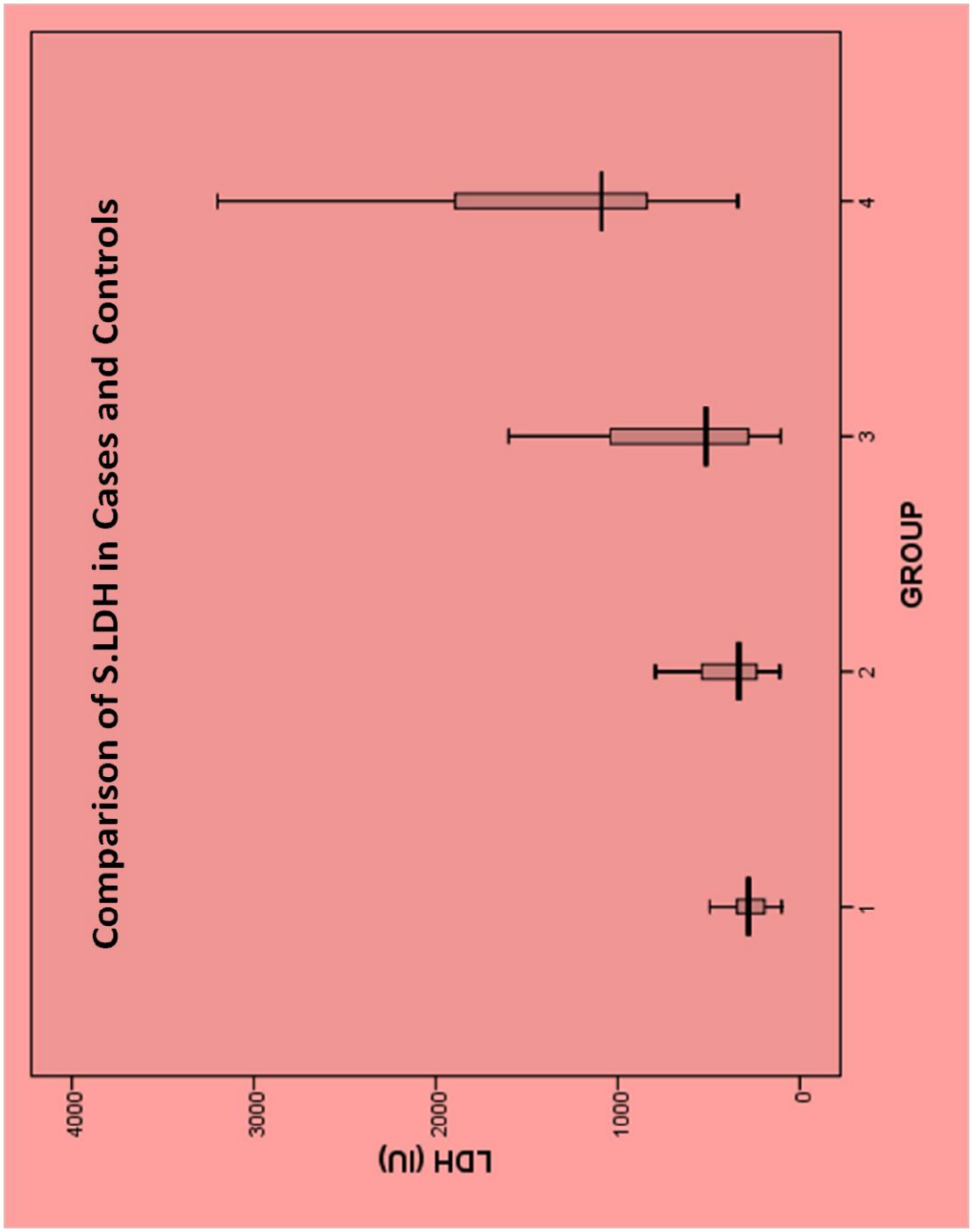

Comparison of S.LDH in Cases and Controls
LDH (IU)
4000
3000
2000
1000
0
1
2
3
4
GROUP

Table 10- Correlation between S. LDH & SBP

SBP (mmHg)	S.LDH			
	Below 600	**600 to 800**	**Above 800**	**Total**
90 to 139	50(41.32%)	0 (0.00%)	0 (0.00%)	50
140 to 159	47 (38.84%)	12 (60.00%)	7 (21.87%)	66
160 & Above	24 (19.83%)	8 (40.00%)	25 (78.12%)	57
Total	121	20	32	173

Out of total 121 patients with LDH levels <600 IU/l, 50 (41.32%) cases had normal SBP, 47 (38.84%) had SBP in the range of 140 to 159 mm of Hg and 24 (19.83%) had SBP 160 and above. Out of 20 patients with LDH levels between 600 and 800 IU/l, none had normal SBP, 12 (60%) had SBP in the range of 140 to 159 mm of Hg and 8 (40%) had SBP 160 and above. In the remaining 32 patients with LDH levels above 800 IU/l, none had normal SBP, 7 (21.87%) had SBP in the range of 140 to 159 mm of Hg and 25 (78.12%) had SBP 160 and above.

Chi-square test was applied to study the correlation between SBP and S.LDH. Correlation coefficient is 0.369, chi-square value is 52.67 and **P Value is 0.00;**

Inference: There was a significant rise in S.LDH with Systolic Blood Pressure.

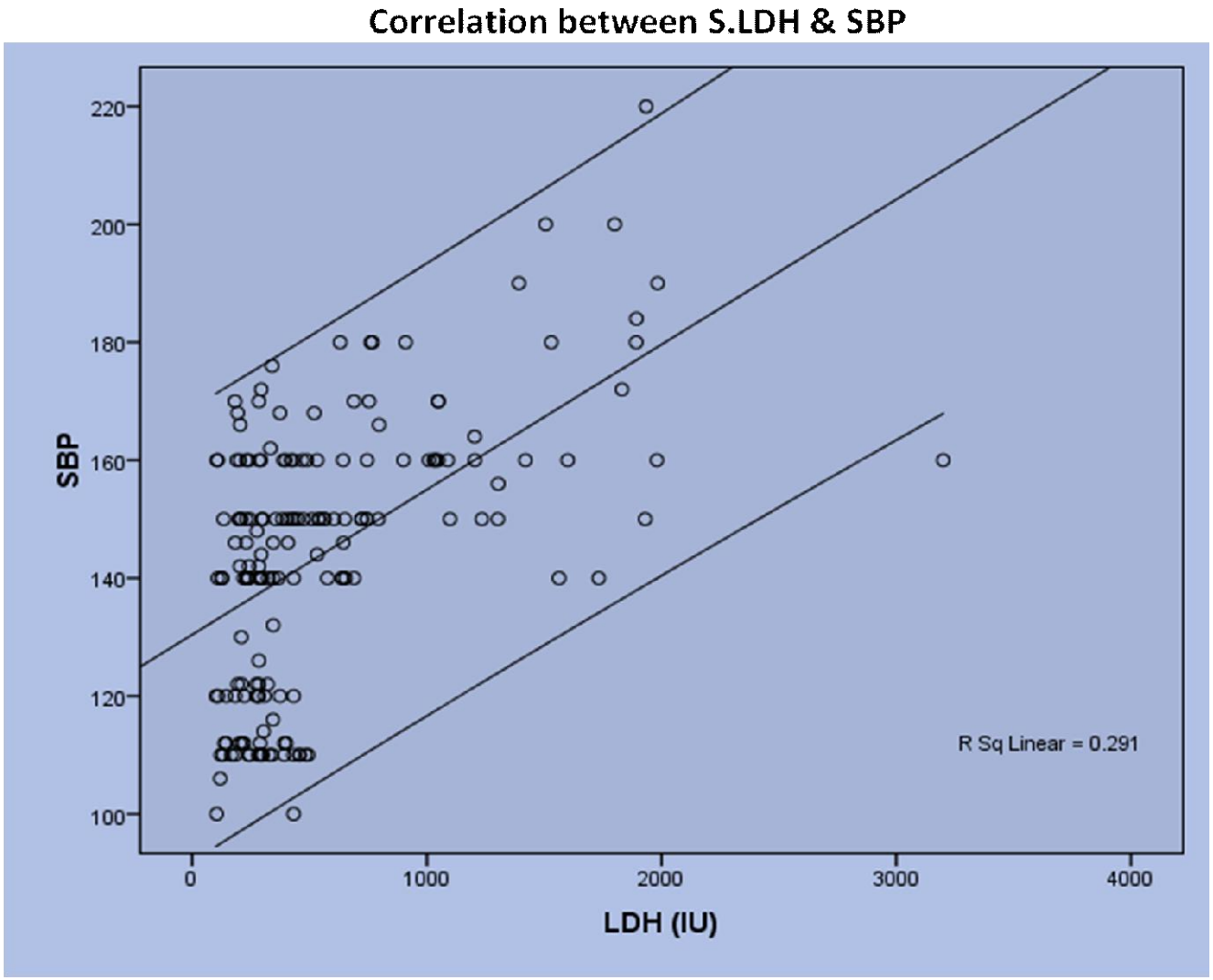

Correlation between S.LDH & SBP
SBP
220
200
180
160
140
120
100
0
1000
2000
3000
4000
LDH (IU)
R Sq Linear = 0.291

Table 11- Correlation between S. LDH & DBP

DBP (mmHg)	S.LDH			
	Below 600	**600 to 800**	**Above 800**	**Total**
60 to 89	56 (46.28%)	1 (5.00%)	0 (0.00%)	57
90 to 109	51 (42.14%)	10 (55.00%)	5 (15.6%)	66
110 & Above	14 (11.57%)	9 (45.00%)	27 (84.37%)	50
Total	121	20	32	173

Out of total 121 patients with LDH levels <600 IU/l, 56(46.28%) had normal DBP, 51 (42.14%) had DBP in the range of 90 to 109 mm of Hg and 14 (11.57%) had DBP 110 and above. Out of 20 patients with LDH levels between 600 and 800 IU/l, one (5%) had normal DBP, 10 (55%) had DBP in the range of 90 to 109 mm of Hg and 9 (45%) had DBP 110 and above. In the remaining 32 patients with LDH levels above 800 IU/l, none had normal DBP, 5 (15.6%) had DBP in the range of 90 to109 mm of Hg and 27 (84.37%) had DBP 110 and above.

Chi-square test was applied to study the correlation between DBP and S.LDH. Correlation coefficient is 0.393, chi-square value is 75.75 and **P Value is 0.00**;

Inference: There was a **significant rise in S.LDH with Diastolic Blood Pressure**.

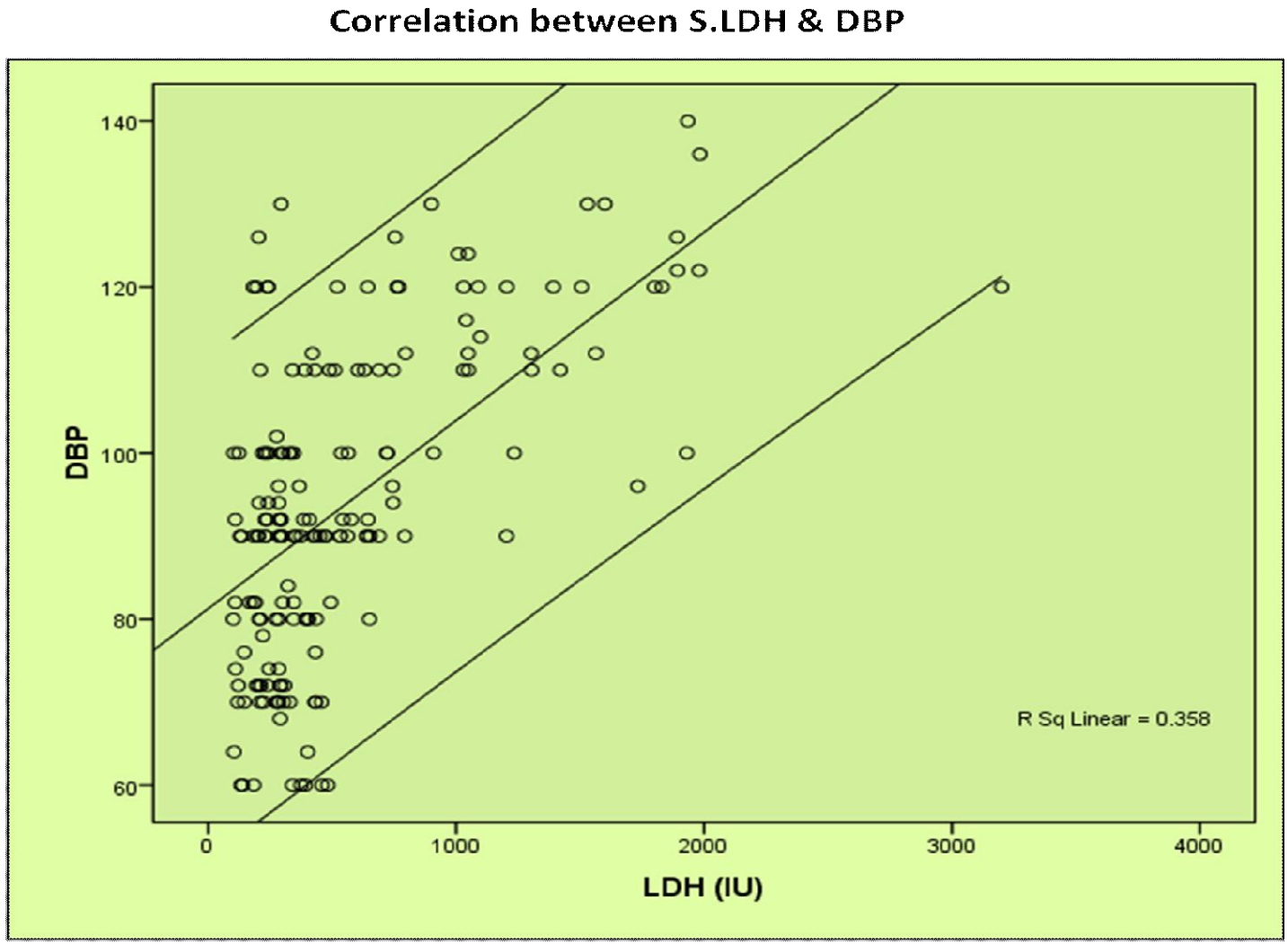

Correlation between S.LDH & DBP
DBP
140
120
100
80
60
0
1000
2000
3000
4000
LDH (IU)
R Sq Linear = 0.358

Table 12- Correlation between S. LDH & Severity of Proteinuria

U/A	S. LDH			
	Below 600	**600 to 800**	**Above 800**	**Total**
1+ (0.3 g/L)	3	0	0	3
2+ (1.0 g/L)	56	10	3	69
3+ (3.0 g/L)	14	6	11	31
4+ (10.0 g/L)	1	4	18	23

Out of the total 173 patients 41 did not have proteinuria, 6 had proteinuria in the range of 0.1 g/L and 3 had proteinuria in the range of 0.3 g/L, whose LDH levels were <600.

69 patients had proteinuria in the range of 1.0g/L, out of which 56 (81.16%) had LDH levels <600, 10 (14.49%) had LDH levels between 600 and 800 and 3 (4.35%) had LDH levels more than 800.

31 patients had proteinuria in the range of 3.0g/L, out of which 14 (45.16%) had LDH levels <600, 6 (19.35%) had LDH levels between 600 and 800 and 11(35.48%) had LDH levels more than 800.

23 patients had proteinuria in the range of 10.0g/L, out of which 1 (4.35%) had LDH levels <600, 4 (17.39%) had LDH levels between 600 and 800 and 18 (78.26%) had LDH levels more than 800.

Chi-square test was applied to study the correlation between S. LDH and severity of proteinuria. Correlation coefficient is 0.567, chi-square value is 58.35 & **P Value is 0.00;**

Inference: There was a significant rise in S.LDH with increasing proteinuria.

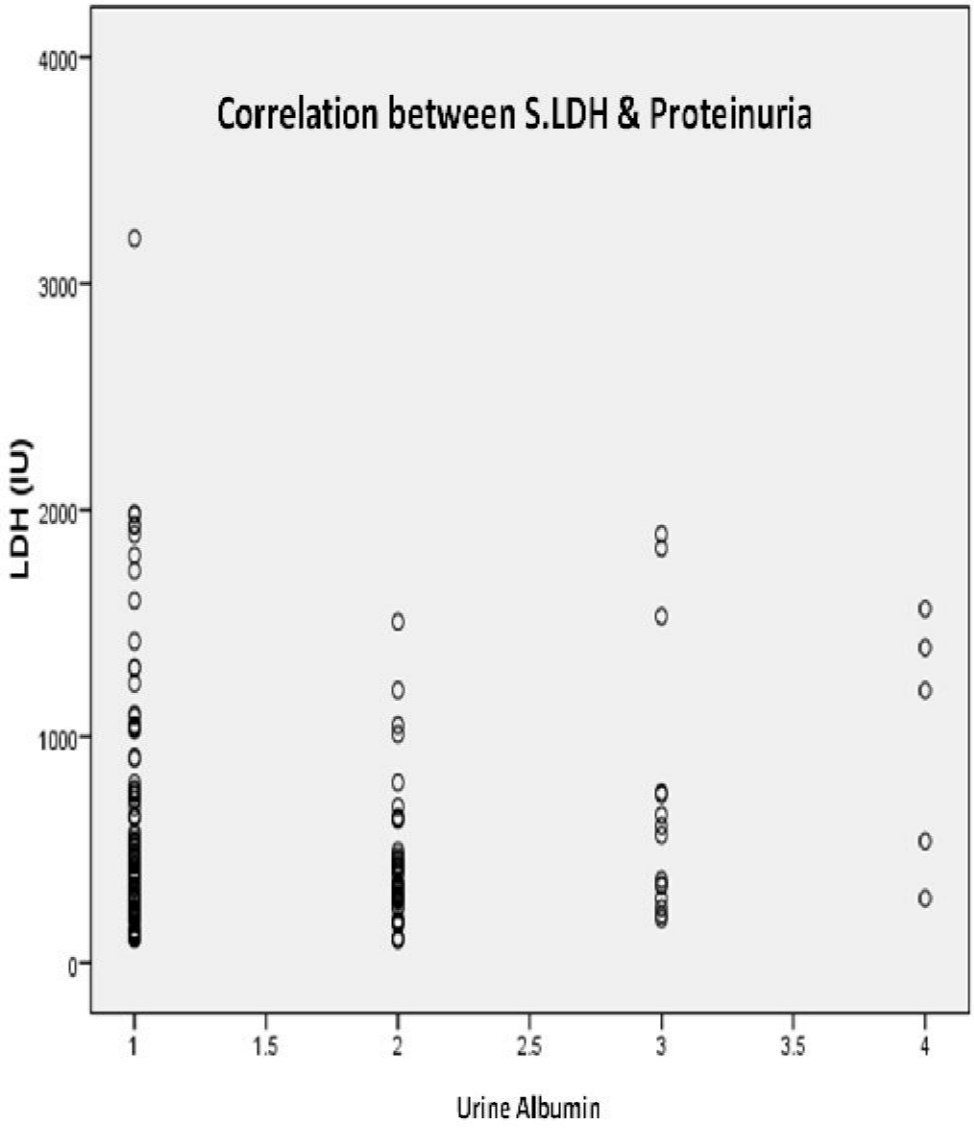


Table 13-Correlation between S. LDH & Abruptio Placenta

S.LDH	Below 600	600 to 800	800 & Above	Correlation Coefficient	Sig. (2-tailed)
Abruptio Placenta	10	9	13	0.271	0.00

Out of the total 173 women 32 had abruptio placenta out of which 14 women had mild pre-eclampsia. Of these women 10 women had LDH values less than 600, 4 had LDH values between 600 and 800 and none had LDH values above 800. 18 had severe pre-eclampsia out of which none had LDH values less than 600, 5 had LDH values between 600 and 800 and 13 others had LDH more than 800.

Chi-square test was applied to study the correlation between S. LDH and abruptio placenta.

Correlation coefficient is 0.271.

chi-square value is 28.12.

P Value is 0.00.

Inference: There was a significant rise in S.LDH in patients with abruptio placenta

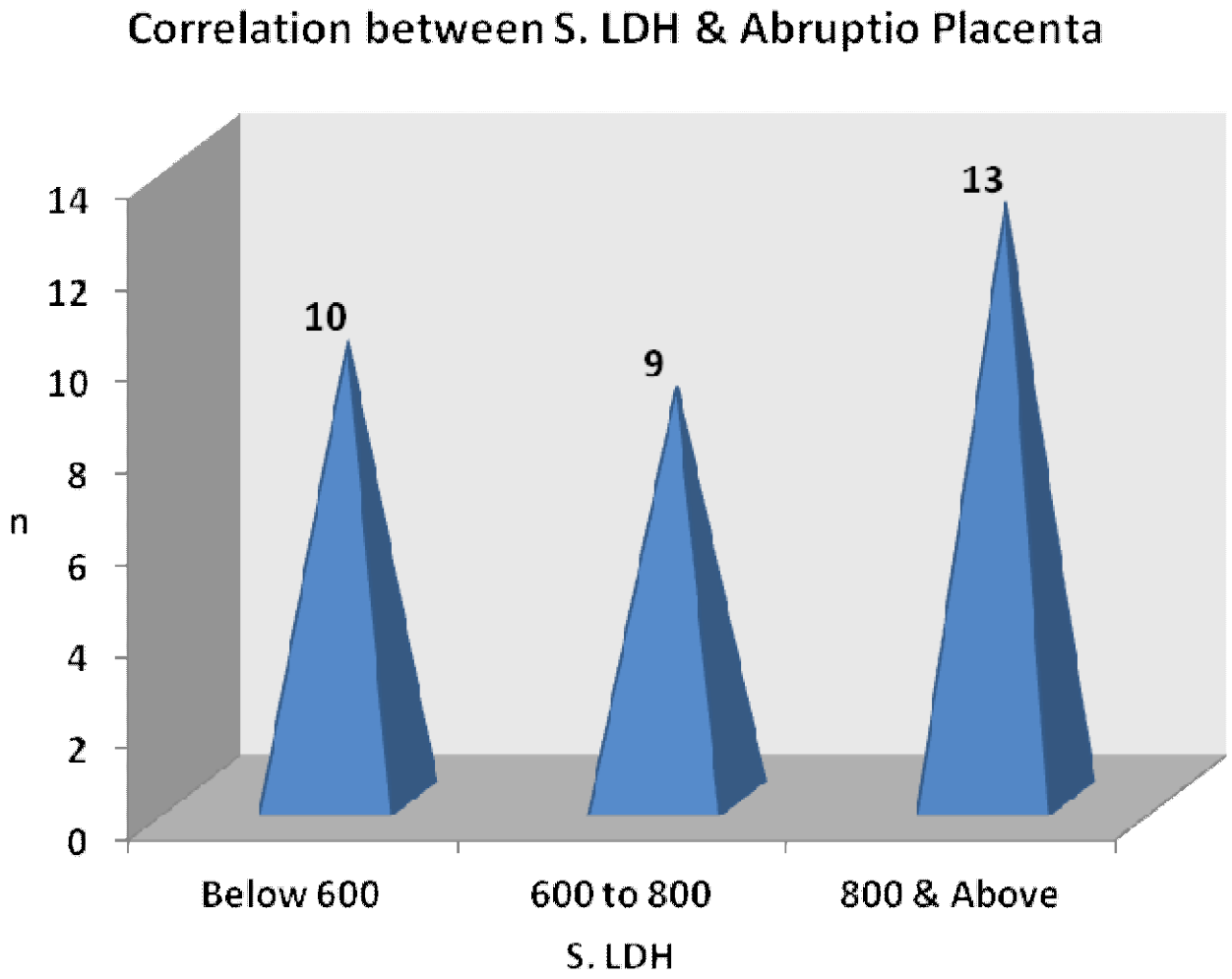

Correlation between S. LDH & Abruptio Placenta
14
12
10
8
6
4
2
0
n
10
9
13
Below 600
600 to 800
800 & Above
S. LDH

Table 14- Correlation between S. LDH & Maternal Mortality

S.LDH	Below 600	600 to 800	800 & Above	Correlation Coefficient	Sig. (2-tailed)
Maternal Mortality	0	0	3	0.160	0.01

Totally 3 maternal deaths were reported, 1 from severe pre-eclampsia due to HELLP with DIC and 2 from Eclampsia, both due to pulmonary edema. No maternal deaths occurred in the normotensives and mild pre-eclampsia groups. All 3 women had very high LDH levels with a mean value of 1943.33 and standard deviation of 499.78 compared to the mean value of 275.4 and standard deviation of 108.36 in normotensive women.

Chi-square test was applied to study the correlation between S. LDH and maternal mortality.

Correlation coefficient is 0.160. Chi-square value is 13.45 and **P Value is 0.01**.

Inference: There was a significant rise in S.LDH in patients with maternl mortality

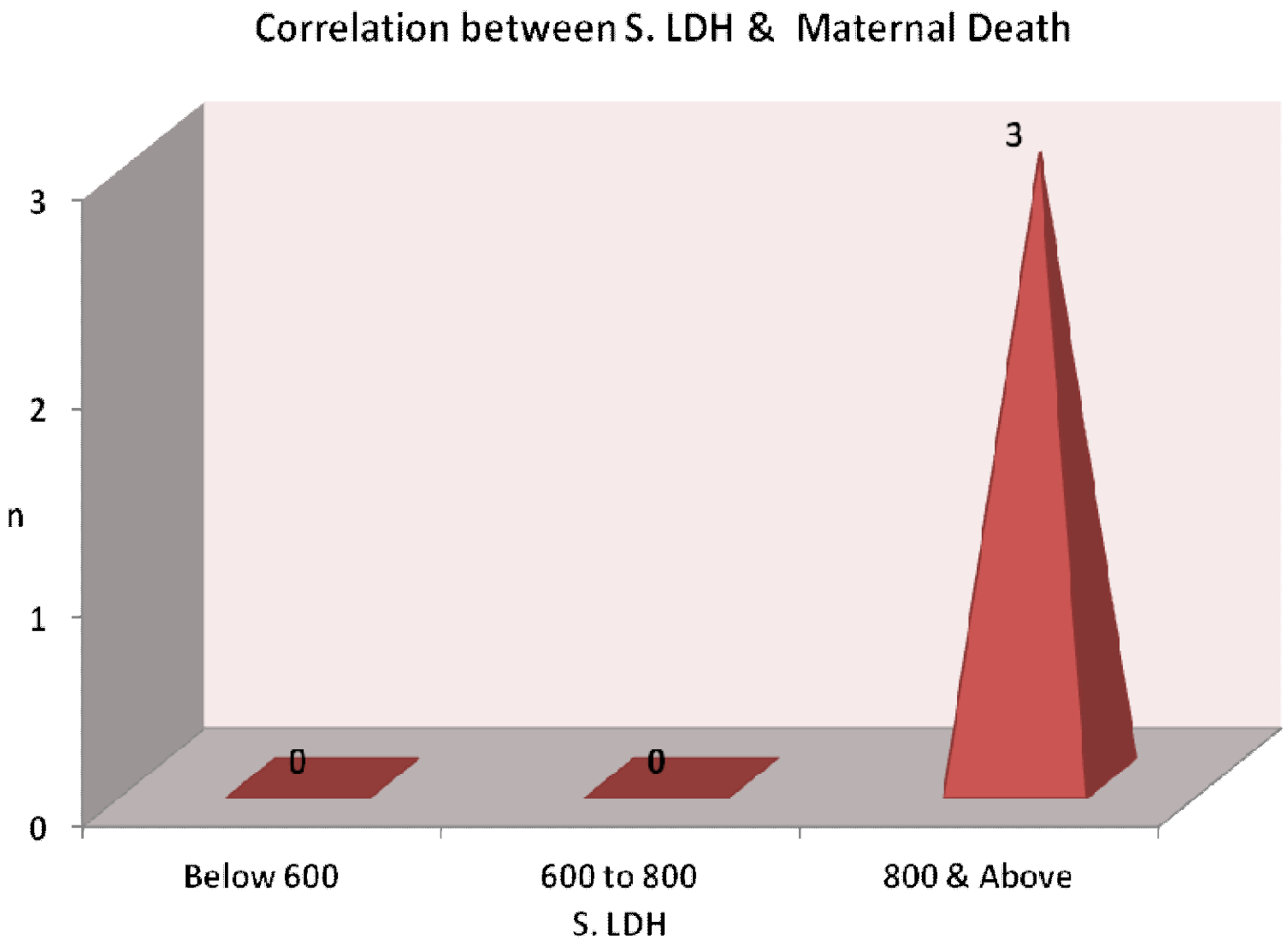

Correlation between S. LDH & Maternal Death
n
3
2
1
0
0
0
3
Below 600
600 to 800
800 & Above
S. LDH

Table 15- Correlation between S. LDH and Operative Delivery

Mode of Delivery	S. LDH					
	<600		600 to 800		>800	
	n	%	n	%	n	%
LSCS	60	49.59	18	90.00	26	81.25
LN	61	50.41	1	5.00	5	15.63
Asst. Breech	0	0.00	1	5.00	1	3.13

Out of the total 173 patients 104 women delivered by LSCS, 67 by normal vaginal delivery and 2 by assisted breech delivery and none by instrumental delivery. Among the women who delivered by cesarean 60 (57.69%) had S.LDH less than 600, 18 (17.30%) had S. LDH between 600 and 800 and 26(25.00%) had S.LDH above 800.

Among the women who delivered vaginally 61 (91.04%) had S.LDH less than 600, 1 (1.49%) had S. LDH between 600 and 800 and 5 (7.46%) had S.LDH above 800. Only 2 women delivered by assisted breech delivery one with S. LDH between 600 and 800 and another with S.LDH above 800.

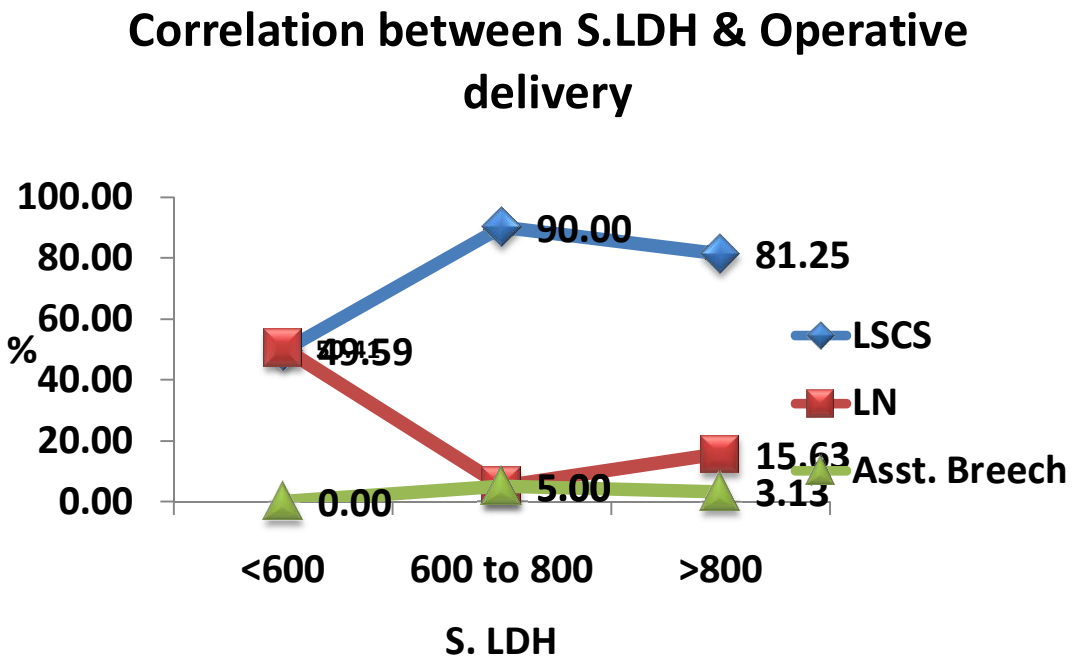
Mode of Delivery	S. LDH			
	<600		>600	
	n	%	n	%
LSCS	60	49.59	44	84.62
LN	61	50.41	6	11.54
Asst. Breech	0	0.00	2	3.85

The incidence of operative delivery significantly increased with S. LDH levels upto 800 and then it declined probably because of the early onset of spontaneous labour in eclamptic women. When the parameters were taken as S. LDH levels less than 600 and more than 600 there was as significant increase in the incidence of operative delivery.

Correlation coefficient value is 0.207, **P value is 0.00**

Inference:

There was a significant rise in S.LDH in patients with operative delivery Correlation between S.LDH and operative delivery explains the severity of the disease with increased LDH and the need for immediate delivery in them.


Correlation between S.LDH & Operative delivery
100.00
80.00
60.00
40.00
20.00
0.00
%
90.00
81.25
49.59
15.63
5.00
0.00
3.13
LSCS
LN
Asst. Breech
<600
600 to 800
>800
S. LDH

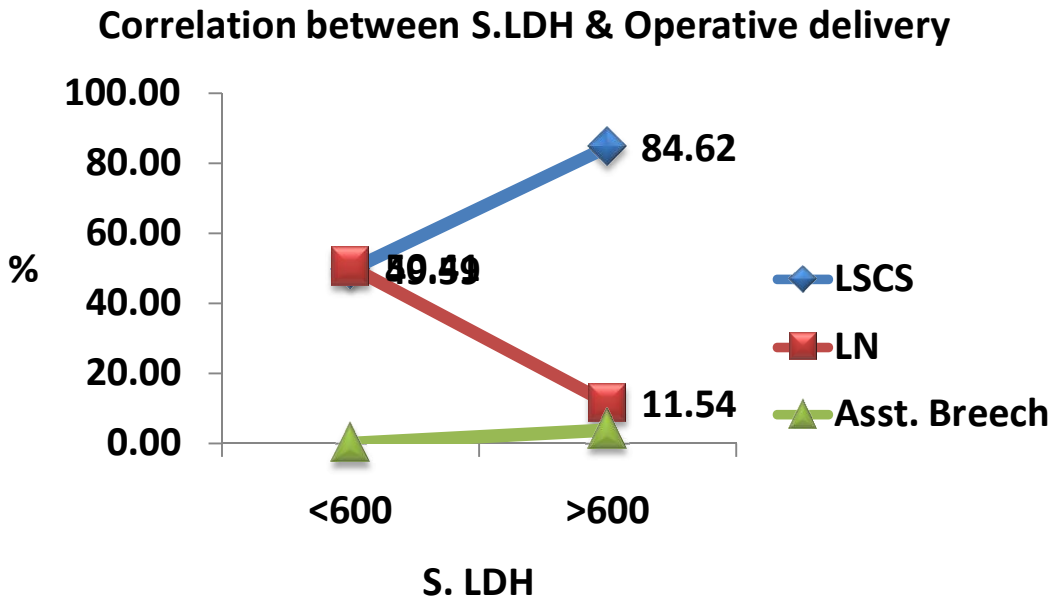

Correlation between S.LDH & Operative delivery
100.00
80.00
60.00
40.00
20.00
0.00
%
84.62
11.54
LSCS
LN
Asst. Breech
<600
>600
S. LDH

Table 16 - Correlation between S. LDH & Mean GA

S.LDH	Below 600	600 to 800	Above 800	Correlation Coefficient	Sig. (2-tailed)
GA (weeks) Mean ± Std. Deviation	38.198 ± 2.16	37.05 ± 2.89	34.46 ± 3.45	-0.276	0.00

The mean gestational age at the time of delivery in patients with S. LDH less than 600, 600 to 800 and more than 800 are 38.19, 37.05 & 34.46 and their standard deviations are 2.16, 2.89 & 3.45 respectively.

Correlation coefficient is -0.276, P value is **0.00**

Inference:

There is reduction in the mean GA with higher level of LDH (*P* = 0.00). This fact could be explained by the increased preterm deliveries associated with pre-eclampsia.

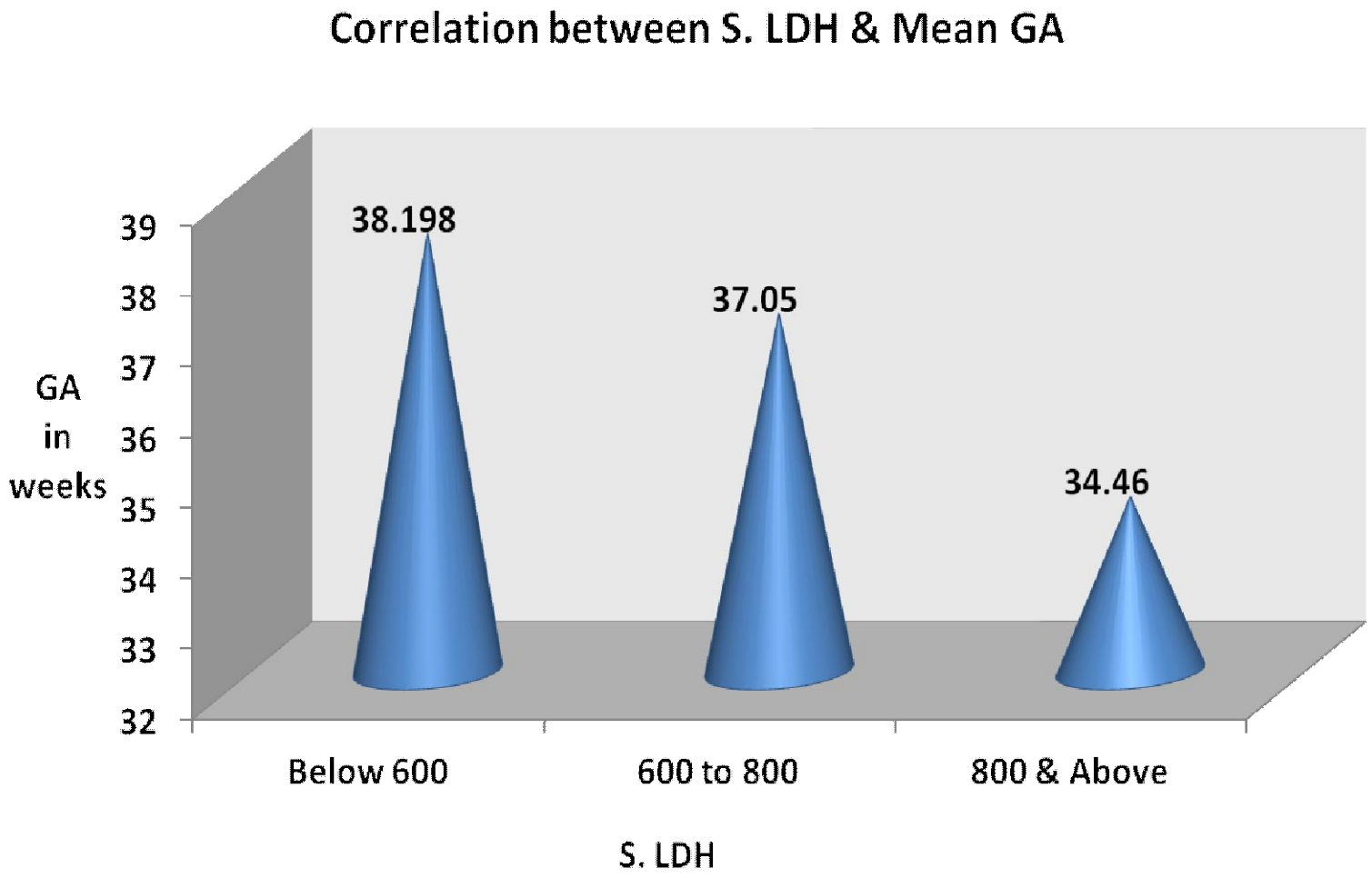

Correlation between S. LDH & Mean GA
GA in weeks
39
38
37
36
35
34
33
32
38.198
37.05
34.46
Below 600
600 to 800
800 & Above
S. LDH

Table 17- Correlation between S. LDH & Mean Baby Weight

S.LDH	Below 600	600 to 800	Above 800	Correlation Coefficient	Sig. (2-tailed)
Mean Baby Weight (kg) ± Std. Deviation	2.76± 0.54	2.377 ± 0.59	1.83 ± 0.59	-0.274	0.00

The mean baby weight in patients with S. LDH less than 600, 600 to 800 and more than 800 are 2.76, 2.37 & 1.83 kg and their standard deviations are 0.54, 0.59 & 0.59 kg respectively.

Correlation coefficient is -0.274, P value is **0.00**

Inference:

This proves that there is reduction in the average weight of babies with higher level of LDH (*P* = 0.00) with a perfect negative correlation of 0.274 probably because of the higher incidence of growth restriction and preterm deliveries among preeclamptic women with higher S. LDH levels.

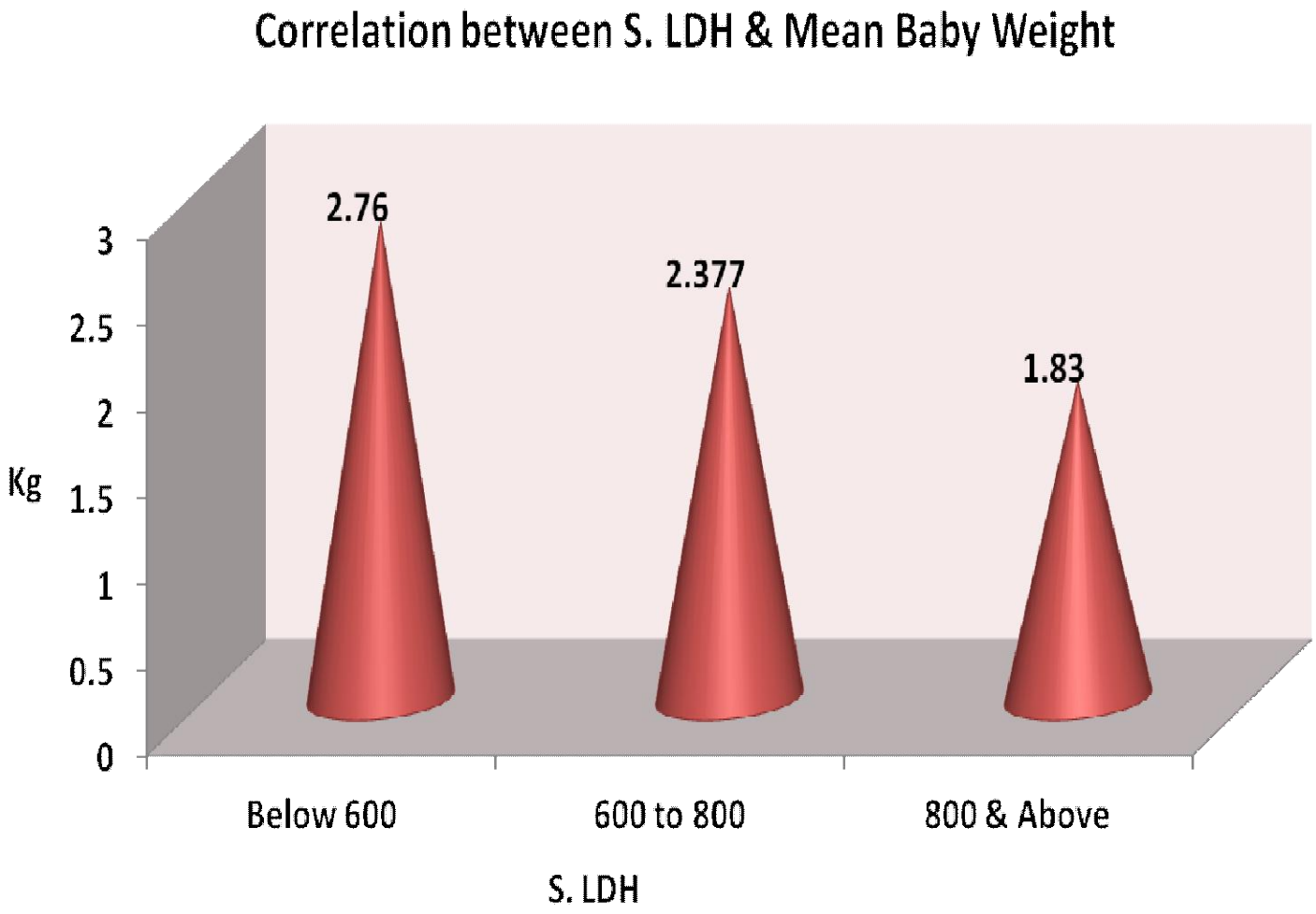

Correlation between S. LDH & Mean Baby Weight
2.76
2.377
1.83
3
2.5
2
1.5
1
0.5
0
Kg
Below 600
600 to 800
800 & Above
S. LDH

Table 18- Correlation between S. LDH & Fetal Growth Restrictions

S.LDH	Below 600	600 to 800	800 & Above	Correlation Coefficient	Sig. (2-tailed)
IUGR	0	3	4	0.205	0.00

Out of the total 173 women 7 had growth restricted babies out of which 4 women had severe pre-eclampsia, 3 0f the 4 women had LDH values between 600 and 800 and 1 had LDH values above 800. 3 out of the 7 women with growth restricted babies had Eclampsia and all three had S.LDH more than 800.

Correlation between S.LDH and intra uterine growth restriction is studied using chi-square test.

Correlation coefficient is 0.205, Chi-square value is 17.17,

P Value is **0.00;**

Inference:

There was a significant rise in S.LDH with incidence of Intra Uterine Growth Restriction with a positive correlation of 0.205.

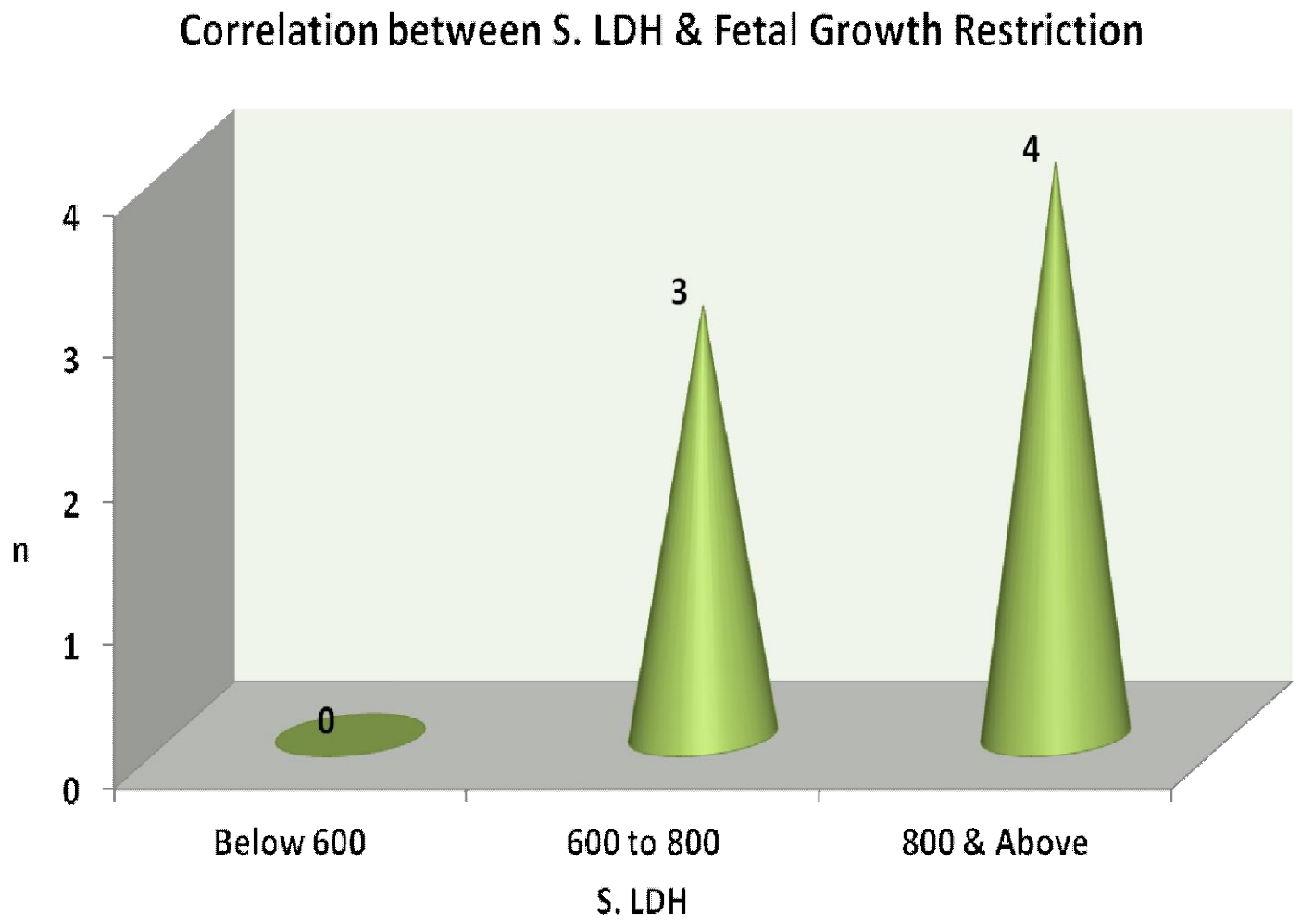

Correlation between S. LDH & Fetal Growth Restriction
n
4
3
2
1
0
0
3
4
Below 600
600 to 800
800 & Above
S. LDH

Table 19- Correlation between S. LDH & Late Intra Uterine Fetal Death

S.LDH	Below 600	600 to 800	800 & Above	Correlation Coefficient	Sig. (2-tailed)
LATE IUD	5	5	10	0.265	0.00

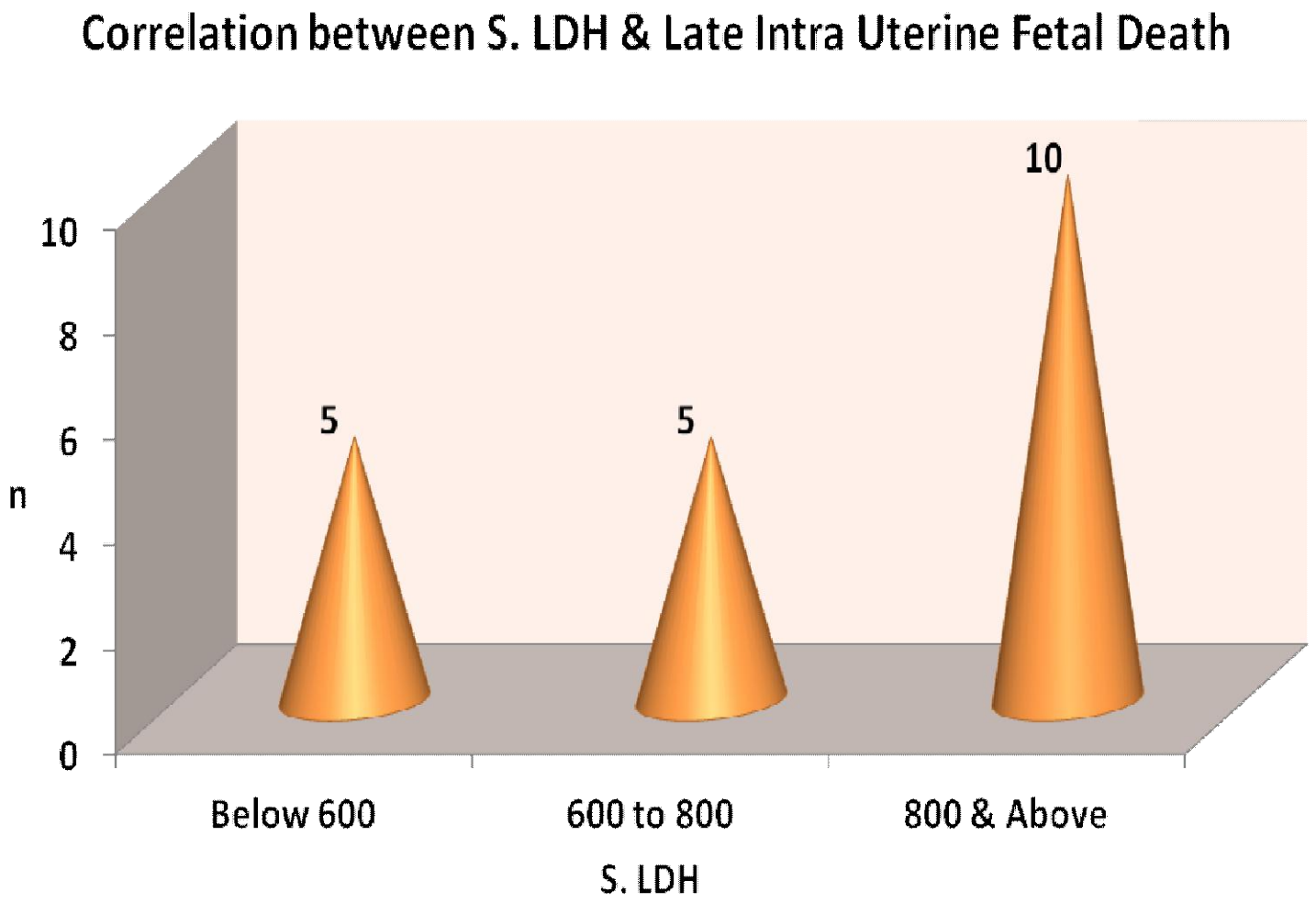
Out of the total 173 women 20 had late intra uterine fetal death out of which 9 women had mild pre-eclampsia, 5 0f the 9 women had LDH values less than 600, 4 had LDH values between 600 and 800 and none had LDH values above 800. 10 out of the 20 women with late intra uterine fetal death had severe pre-eclampsia, only 1 had LDH values between 600 and 800 and all others had S.LDH more than 800. Only 1 woman in the Eclampsia group had late intra uterine fetal death with S.LDH more than 800.

Correlation between S.LDH and IUD is studied using chi-square test.

Correlation coefficient is 0.265, Chi-square value is 22.19.

P Value is **0.00.**

Inference: There was a significant rise in S.LDH with IUD


Correlation between S. LDH & Late Intra Uterine Fetal Death
10
8
6
4
2
0
n
5
5
10
Below 600
600 to 800
800 & Above
S. LDH

DISCUSSION

This study is conducted in the Department of Obstetrics and Gynecology, Chengalpattu Medical College**.**

A total of 173 antenatal women were recruited from Outpatient department/ Labour Ward at Chengalpattu Medical College & Hospital from November 2013 ~ August 2014

All patients were of gestational age 28 weeks and above. Patients were selected based on the inclusion and exclusion criteria irrespective of the age and parity and they were divided into three groups based on NHBPEP classification as 50 normotensives, 50 mild pre-eclamptics, 50 severe pre-eclamptics & 23 eclamptics. Patients were also divided into three groups based on their S. LDH (less than 600, 600 to 800, and more than 800 IU/l).

All the diagnostic components and the possible maternal and fetal complications of pre-eclampsia were correlated with their S.LDH levels.

This study is similar to the study done by **S.P Jaiswar, Amrit Gupta and Mohan Shaili at CSSM, Lucknow**.

The distribution of age & mean age between groups were almost similar. No significant difference was observed in terms of **age and**

parity between groups and moreover they did not influence S.LDH in contrast to **Qublan et al and Jaiswar et al**. Qublan et al stated that the mean age of patients in preeclampsia was significantly less compared to the normotensives and majority were young primigravida in the affected population.

S. LDH levels consistently increased with increasing **systolic & diastolic blood pressure**, more so with diastolic blood pressure with a **P value of <0.001** similar to **Jaiswar & Amrit et al**.

Jaiswar & Amrit et al in their study observed a **mean S. LDH** of 278.33 ± 119.25 in normotensives,400.45 ± 14.21 in mild pre-eclampsia,646.95 ± 49.64 in severe pre-eclampsia,1648.10 ± 1992.29 in eclampsia with a **P value <0.001**. Similar results were observed in this study with mean S. LDH of 275.4 ± 108.38 in normotensives, 381.42 ± 178.93 in mild pre-eclampsia, 660.84 ± 456.08 in severe pre-eclampsia and 1648 ± 677.13 in eclampsia with a **P value of <0.001.**

Most of the women with severe preeclampsia & eclampsia had severe proteinuria and S. LDH significantly increased with the severity of **proteinuria (P<0.00).** Results were comparable to **Qublan et al** who showed a significant increase in S. LDH with severity of proteinuria**(P value of <0.05).**

In the study done by **S.P.Jaiswar et al,** only 2 cases had **abruptio placenta** but the incidence was tremendously high with 32 cases in this group. Significantly high LDH values were observed than controls **P (0.00)** among them.

Only 1 case of **HELLP syndrome** was present in the study. Totally **3 maternal death** occurred in the study population and all of them had very high LDH levels. 2 deaths were due to **pulmonary edema** and 1 due to **DIC**. All the three patients needed **ventilatory support**.

Other maternal complications like CVA, Postpartum hemorrhage, renal failure were not present in the study population. Still births were not present in the study population probably because of the close fetal surveillance, early decision and increased operative delivery among pre-eclamptic women.

The incidence of **operative delivery** moderately elevated with increased LDH levels and this explains the severity of the disease with increased LDH and the need for immediate delivery in them.

The **mean gestational age** at the time of delivery in patients with S. LDH less than 600, 600 to 800 and more than 800 are 38.19, 37.05 & 34.46 and their standard deviations are 2.16, 2.89 & 3.45 respectively.

The mean baby weight in patients with S. LDH less than 600, 600 to 800 and more than 800 are 2.76, 2.37 & 1.83 kg and their standard deviations are 0.54, 0.59 & 0.59 kg respectively

Mean GA and mean baby weight had negative correlation with S. LDH levels with **P value < 0.00** similar to **Jaiswar et al** whose mean GA at the time of delivery was significantly less in patients with increasing LDH levels **(P value = 0.025).** This fact could be explained by the increased preterm deliveries and the need for early termination of pregnancy to improve the maternal outcome in view of severity of disease.

Complications like **growth restriction & late intrauterine fetal demise** are well known complication of pre-eclampsia. **He S , Bremme K, Kallner et al** studied the S. LDH levels in pre-eclamptic women with small for gestational age infants and found a significant correlation between both. Incidence of IUGR and IUD were significantly higher in pre-eclamptic women and their S. LDH levels compared to the controls were abnormally high (**P value < 0.00)** similar to He. S et al.

SUMMARY

This was a comparative observational study done on 173 patients attending Outpatient department/ Labour Ward at Chengalpattu Medical College & Hospital, Chengalpattu from November 2013 ~ August 2014

All patients were of gestational age 28 weeks and above. Patients were selected irrespective of the age and parity and they were divided into three groups based on NHBPEP classification as 50 normotensives, 50 mild pre-eclamptics, 50 severe pre-eclamptics & 23 eclamptics. Patients were also divided into three groups based on their S. LDH (less than 600, 600 to 800, and more than 800 IU/l). All the diagnostic components and the possible maternal and fetal complications of pre-eclampsia were correlated with their S.LDH levels.

The study was done in search of a valuable marker for preeclampsia and Eclampsia which would reflect the severity of the disease and would predict the maternal and fetal outcome. Such markers can help in decision making and can influence the current management protocols in order to achieve a better maternal and perinatal outcome.

Lactate dehydrogenase has been suggested by various authors as a promising marker and the inferences made out of this study are as follows.

1. Age & Parity did not have any relation with S. LDH levels of **P(0.21)**
2. Systolic & Diastolic blood pressure had significant correlation with S. LDH levels with a **P value of 0.00**
3. S. LDH levels did not vary much between normotensives and mid preeclampsia (**P =0.47**) but significant increase was observed between normotenisves, severe pre-eclampsia & eclampsia (p<**0.00**)
4. Proteinuria by itself is a marker of severity of the disease and correlating LDH values **P (0.00)** proves S. LDH to be a similar marker with high significance and hence the need for management strategies based on S. LDH.

Among the 121cases with normal LDH levels (<600 IU/L),

109 cases (90.08%) had uneventful maternal outcome.

12 cases (9.91%) had maternal complications which include

10 cases (8.26%) with abruptio placenta,

2 cases (1.65%) with Eclampsia.

No IUGR cases were observed.

5 cases (4.14%) of late fetal deaths were reported.

Among the 20 cases with LDH levels between 600-800 IU/L,

7 cases (35%) had uneventful maternal outcome.

13 cases (65%) had maternal complications which include

9 cases (45%) with abruptio placenta,

4 cases (20%) with Eclampsia.

3 cases (15%) had IUGR.

5 cases (25%) had late fetal death.

Among the 32 cases with LDH levels more than 800 IU/L,

Only one case (3.12%) had uneventful maternal outcome.

31 cases (96.88%) had maternal complication which include

13 cases (40.62%) with abruptio placenta,

1 case (3.12%) with HELLP & DIC leading to maternal death,

17 cases (53.12%) with Eclampsia including 2 maternal deaths due to pulmonary edema.

4 cases (12.5%) had IUGR.

10 cases (31.25%) had late fetal death..

Overall incidence of maternal complications (Abruption, Pulmonary Edema, DIC, HELLP) were **50%** with increased LDH values **(>600 IU/L)** when compared to **8.26%** with normal LDH values **(<600 IU/L).** Significantly high maternal morbidity and mortality were observed with high LDH levels **(P= 0.00).**

There was an overall incidence of fetal complication (IUGR + IUD) of **42.3 %** with increased LDH values **(>600 IU/L)** when compared to **4.13 %** with normal LDH values **(<600 IU/L).** Significantly high fetal complications were observed with high LDH levels **(P=0.00).**

CONCLUSION

After analyzing the data and comparing the results following conclusion have been drawn from the study.

S. LDH values were significantly high in pre-eclamptic patients depending on the severity of the disease indicating the increased cellular turnover in them.

S. LDH levels had a good correlation with all the diagnostic components of preeclampsia like SBP, DBP & Proteinuria, similarly with maternal and fetal morbidity and mortality

Hence diagnostic and management strategies may be considered based on S.LDH levels and further studies on a larger sample can be done to substantiate our observations on the utility of this parameter as a diagnostic and prognostic component of preeclampsia. Development of new management strategies based on S. LDH levels may help in appropriate decision making thereby avoiding unwanted maternal & fetal deaths.

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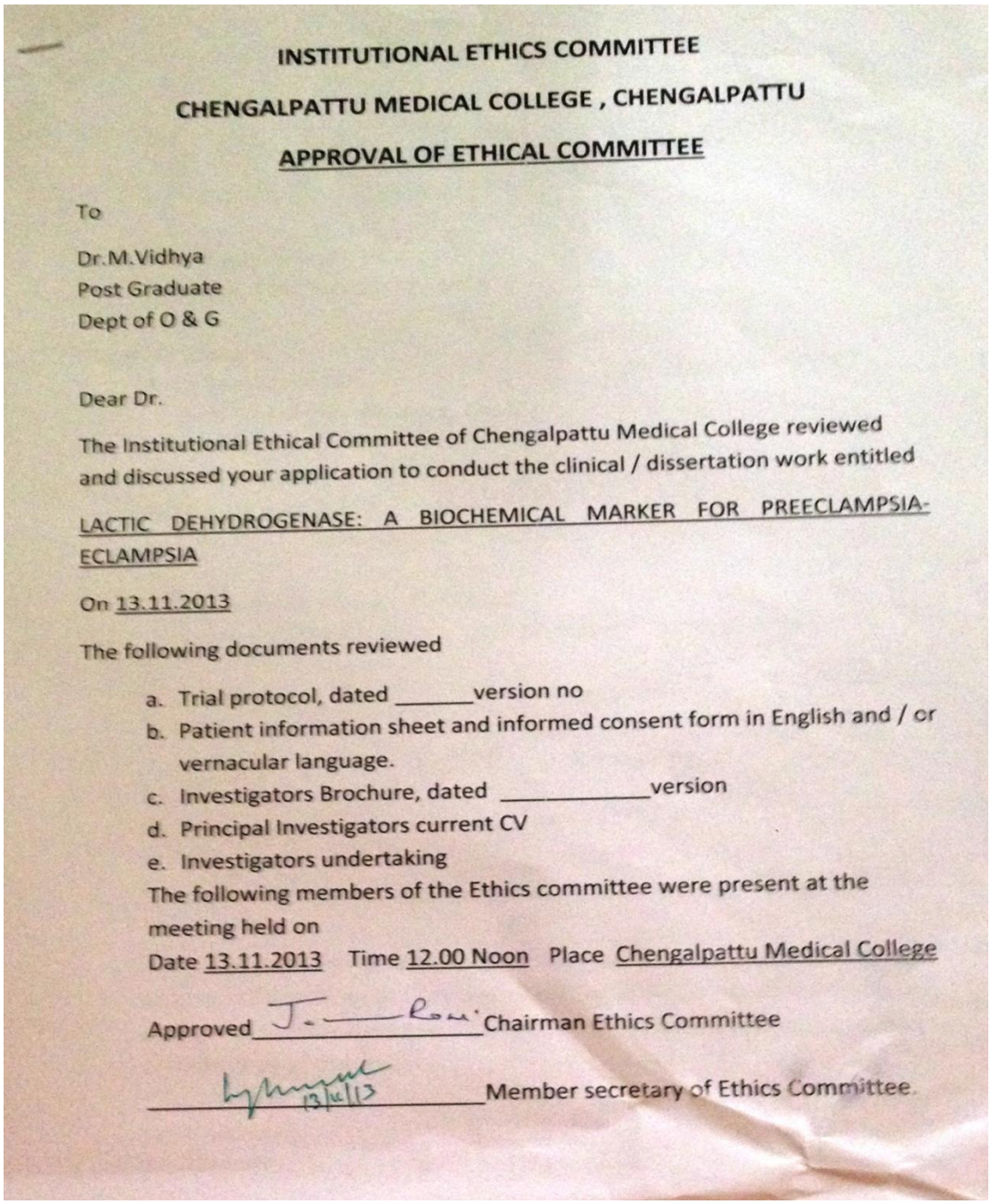
ANNEXURE - I

ETHICAL COMMITTEE APPROVAL



INSTITUTIONAL ETHICS COMMITTEE

CHENGALPATTU MEDICAL COLLEGE , CHENGALPATTU

APPROVAL OF ETHICAL COMMITTEE

To

Dr.M.Vidhya
Post Graduate
Dept of O & G

Dear Dr.

The Institutional Ethical Committee of Chengalpattu Medical College reviewed and discussed your application to conduct the clinical / dissertation work entitled

LACTIC DEHYDROGENASE: A BIOCHEMICAL MARKER FOR PREECLAMPSIA-ECLAMPSIA

On 13.11.2013

The following documents reviewed

a. Trial protocol, dated _____version no
b. Patient information sheet and informed consent form in English and / or vernacular language.
c. Investigators Brochure, dated _____version
d. Principal Investigators current CV
e. Investigators undertaking

The following members of the Ethics committee were present at the meeting held on

Date 13.11.2013 Time 12.00 Noon Place Chengalpattu Medical College

Approved _____Chairman Ethics Committee

13/11/13
_____Member secretary of Ethics Committee

ANNEXURE - I

ETHICAL COMMITTEE APPROVAL

Name of each member with designation

Clinical Members

1. Dr.G.Raja Billy Graham MS.,
   Prof & HOD of Surgery, CHMC

2. Dr.K.Srinivasagalu MD.,
   Prof & HOD of Medicine, CHMC

Biological Scientist

3. Dr.K.Baskaran MD.,
   Asso Prof of Pharmacology, CHMC

Non Clinical Members

4. Dr.P.Parasakthi MD
   Prof & HOD of Forensic Medicine,CHMC

5. Member from Nongovernmental
   Voluntary Organisation : Mr.P.Durairaj

6. Philosopher : Mr.K.S.Ramprasad

7. Lawyer : Lr. I. M. Karimala Basha

8. Layperson : Mr.Dilli

We approve the clinical trial to be conducted in its presented form

The Institutional Ethics Committee expects to be informed about the progress of the study and any SAE occurring in the course of the study, any changes in protocol and patient information / informed consent and asks to provide copy of final report.

Yours sincerely

13/4/13

Member secretary, Ethics Committee

ANNEXURE-II

COPY OF INFORMED CONSENT

ஆராய்ச்சி ஒப்புதல் கடிதம்

ஆராய்ச்சி தலைப்பு :

பெயர் : தேதி :

வயது :

வெளி நோயாளி எண் :

பால் :

ஆராய்ச்சி சேர்க்கை எண் :

இந்த ஆராய்ச்சியின் விவரங்களும் அதன் நோக்கங்களும் முழுமையாக எனக்கு தெளிவாக விளக்கப்பட்டது.

எனக்கு விளக்கப்பட்ட விஷயங்களை நான் புரிந்து கொண்டு நான் எனது சம்மதத்தைத் தெரிவிக்கிறேன்.

இந்த ஆராய்ச்சியில் பிறரின் நிர்ப்பந்தமின்றி என் சொந்த விருப்பத்தின் பேரில் தான் பங்கு பெறுகிறேன் மற்றும் நான் இந்த ஆராய்ச்சியிலிருந்து எந்நேரமும் பின்வாங்கலாம் என்பதையும் அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும் நான் புரிந்துகொண்டேன்.

இந்த ஆராய்ச்சியின் விவரங்களைக் கொண்ட தகவல் தாளைப் பெற்றுக்கொண்டேன்.

நான் என்னுடைய சுயநினைவுடன் மற்றும் முழு சுதந்திரத்துடன் இந்த மருத்துவ ஆராய்ச்சியில் என்னையும் சேர்த்துக்கொள்ள சம்மதிக்கிறேன்.

நோயாளி/ உடன் இருப்பவர் கையொப்பம்

ANNEXURE-III

COPY OF PATIENTS INFORMATION SHEET

ஆராய்ச்சி தகவல் தாள்

செங்கல்பட்டு அரசு பொது மருத்துவமணையில் மகப்பேறு நலப்பிரிவில் கர்ப்ப காலத்தில் பெண்களுக்கு ஏற்படும் இரத்த அழுத்தம் மற்றும் அதன் பின்விளைவுகள் பற்றிய ஆராய்ச்சி நடைபெற்று வருகின்றது.

நீங்களும் இந்த ஆராய்ச்சியில் பங்கேற்க நாங்கள் விரும்புகிறோம். இதனால் உங்களுக்கு எந்த பாதிப்பும் ஏற்படாது என்பதை தெரிவித்து கொள்கிறோம்.

முடிவுகளை அல்லது கருத்துகளை வெளியிடும்போது அல்லது ஆராய்ச்சியின்போது உங்கள் பெயரையோ அல்லது அடையாளங்களையோ வெளியிடமாட்டோம் என்பதை தெரிவித்துகொள்கிறோம்.

இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களுடைய விருப்பத்தின்பேரில் தான் இருக்கிறது. மேலும் நீங்கள் எந்நேரமும் இந்த ஆராய்ச்சியிலிருந்து பின்வாங்கலாம் என்பதை தெரிவித்துகொள்கிறோம்.

இந்த சிறப்பு பரிசோதனைகளின் முடிவுகளை ஆராய்ச்சியின்போது அல்லது ஆராய்ச்சியின் முடிவின்போது தங்களுக்கு அறிவிப்போம் என்பதையும் தெரிவித்துகொள்கிறோம்.

ANNEXURE – IV

PROFORMA

Name: Date of Delivery:

Age/ Sex: Date of Admission:

IP. No: Date of Discharge:

Socio-Economic Status:

Obstetric Formula:

LMP: EDD: WEEKS OF GESTATION:

Chief Complaints:

History of Present Illness:

Obstetric History:

Past:

Present:

1st TRIMESTER

2nd TRIMESTER

3rd TRIMESTER

Menstrual h/o: Menarche , Cycles , Flow

Marital h/o: years, Consanguinity:

Previous Medical Illness:

- ❖ Hypertension
- ❖ Diabetes
- ❖ Autoimmune Disease
- ❖ Dilatation & Curettage
- ❖ Tuberculosis
- ❖ Seizure Disorder
- ❖ Bleeding Disorders
- ❖ Bronchial Asthma
- ❖ Thyroid Disorder
- ❖ Heart Disease
- ❖ Immunosupression
- ❖ Renal/ Liver Disease
- ❖ Others

Family H/O:

General Examination:

Consciousness:

Pallor:

Icterus:

Pedal Edema:

CVS:

RS:

Height:

Weight:

BMI:

Temp:

Pr: / min

Bp: / mmHg

Per Abdomen:

Inspection:

Palpation:

Ausculation:

Per Speculum:

Per Vaginum:

Diagnosis:

Investigations:

- Serum LDH
- Blood Grouping & Typing
- Complete Hemogram
- Urine Routine
- Bleeding Time
- Clotting Time
- Renal Function Test

- ❖ Liver Function Test
- ❖ HIV I & II
- ❖ Fundus Evaluation
- ❖ 24hrs Urine Protein

Obstetric Sonogram:

Singleton/ Multiple/ Viability/ Gestational Age/ Placental Position/ Retro Placental Clots/ Presentation/ Liquor/ IUGR/ IUD

Mode of Delivery:

Normal Vaginal Delivery/ Assisted/ Forceps/ LSCS

- ❖ Intra-op Findings:
- ❖ RP Clots: gms
- ❖ Postpartum Haemorrhage Yes/ No ml

Perinatal Complications:

- ❖ Eclampsia Yes/No
- ❖ ICU Care Yes/ No
- ❖ Blood/ Blood Products Yes/No
- ❖ DIC/Shock Yes/No
- ❖ Pulmonary Edema Yes/No
- ❖ Ventilator Care Yes/No
- ❖ HELLP Yes/No
- ❖ Renal Failure Yes/No
- ❖ Abruptio Placenta Yes/No
- ❖ CVA/ Intracranial Hemorrhage Yes/No

- ❖ Hysterectomy Yes/ No
- ❖ Sepsis Yes/ No
- ❖ Maternal Mortality Yes/ No
- ❖ Cortical blindness Yes/No
- ❖ Adult respiratory distress syndrome Yes/No
- ❖ Hepatic rupture Yes/No

Fetal/ Neonatal Outcome:

- ❖ IUGR
- ❖ Live/ Stillbirth/ Neonatal Death
- ❖ Term/ Preterm (BIRTH WT: Kgs)
- ❖ NICU Care

S. No	Name	Age	IP No	OBS Code	Group	GA(Wks)	LDH (IU)	Mode Delivery	Hellp	Eclampsica	Abruption	Maternal Death	Wt of Baby(Kg)	IUGR	IUD	SBP	DBP	U/A
1	Thamaraikanni	20	46090	Primi	Eclampsia	37	768	EM. LSCS		1			2.54			180	120	3+
2	Sarasu	20	47095	G3P1L1A1	Eclampsia	39	340	EM. LSCS		1			3.5			176	110	3+
3	Lalitha	24	48065	Primi	Eclampsia	38	720	EM. LSCS		1			2.8			150	100	2+
4	Selvakumari	30	48396	Primi	Eclampsia	36	745	EM. LSCS		1			2.7			160	94	4+
5	Vidhya	23	46376	G2A1	Severe Pre-eclampsia	30	240	EM. LSCS					1.2			140	120	3+
6	Rajeshwari	30	46798	G2P1L1	Severe Pre-eclampsia	39	190	EM.Rpt LSCS					2.8			160	82	2+
7	Valarmathi	27	48356	Primi	Severe Pre-eclampsia	36	204	EM. LSCS					2.2			150	94	3+
8	Shakila	20	49096	Primi	Severe Pre-eclampsia	41	203	EM. LSCS					2.75			160	126	3+
9	Kaliyammal	27	46846	G5P2L1A1	Severe Pre-eclampsia	36	1563	EM. LSCS			1		2.3			140	112	4+
10	Kala	34	46821	G2P1L1	Mild Pre-eclampsia	38	450	EM. LSCS			1		3			150	90	1+
11	Vidhya	27	48350	Primi	Severe Pre-eclampsia	28	1420	EM. LSCS			1		1		1	160	110	4+
12	Sumathi	36	46426	Primi	Mild Pre-eclampsia	37	562	EM. LSCS					2.3			150	90	1+
13	Kotteswari	21	46089	Primi	Mild Pre-eclampsia	40	230	EM. LSCS					3.25			146	90	1+
14	Arul Mozhi	20	49186	G2P1L1	Controls	39	110	LN					2.75			120	74	NIL
15	Jayanthi Rani	19	49128	Primi	Controls	38	220	LN					2.75			112	78	1+
16	Sathya	24	49100	G2P1L1	Controls	39	300	LN					2.75			110	82	Trace
17	Dhanalakshmi	28	49206	G2P1L1	Controls	38	432	LN					2.2			100	70	NIL
18	Pandiyammal	23	49676	Primi	Eclampsia	36	1983	EM. LSCS		1			1.8	1		190	136	3+
19	Vasanthi	20	49723	G3A2	Eclampsia	28	1831	EM. LSCS		1			1		1	172	120	4+
20	Susheela	22	50944	Primi	Eclampsia	32	1930	EM. LSCS		1			1.5			150	100	2+
21	Gomathi	21	57583	Primi	Eclampsia	34	1030	EM. LSCS		1			1.9			160	110	4+
22	Kalpana	24	51625	G3P2L1	Eclampsia	36	1893	EM. LSCS		1			1.8	1		184	122	4+
23	Kanniga	22	50221	G2A1	Severe Pre-eclampsia	40	104	EM. LSCS					3.25			160	100	1+

S. No	Name	Age	IP No	OBS Code	Group	GA(Wks)	LDH (IU)	Mode Delivery	Hellp	Eclampsica	Abruption	Maternal Death	Wt of Baby(Kg)	IUGR	IUD	SBP	DBP	U/A
24	Chandra	30	50572	Primi	Severe Pre-eclampsia	38	230	EM. LSCS					2.6			160	90	2+
25	Kavitha	26	57362	Primi	Severe Pre-eclampsia	39	405	EM. LSCS					3.4			150	80	3+
26	Poongodi	23	57575	Primi	Severe Pre-eclampsia	37	398	EM. LSCS					2.5			160	80	2+
27	Selvi	31	49696	G2P1L1	Severe Pre-eclampsia	38	1009	EM. LSCS			1		2	1	1	160	124	3+
28	Kannagi	24	49731	G2P1L1	Mild Pre-eclampsia	32	294	EM. LSCS			1		1.6			144	92	1+
29	Rajeshwari	33	57539	G4P1L1A2	Severe Pre-eclampsia	41	1203	EM.Rpt LSCS			1		3			164	90	2+
30	Kamarunisha	23	57680	Primi	Severe Pre-eclampsia	40	1302	EM. LSCS			1		3.25			150	112	4+
31	Poornima	21	49127	Primi	Mild Pre-eclampsia	38	230	LN			1		3.2			140	92	1+
32	Murugavalli	23	49404	Primi	Mild Pre-eclampsia	40	532	EM. LSCS					3.6			144	90	Trace
33	Shantha	27	50033	Primi	Mild Pre-eclampsia	40	435	EM. LSCS					3.25			150	80	Trace
34	Suganthi	21	49101	Primi	Controls	40	456	LN					3.5			110	70	NIL
35	Mohana	29	49134	Primi	Controls	40	210	LN					2.4			122	80	Trace
36	Lakshmi	22	49113	Primi	Controls	40	222	LN					3			120	70	NIL
37	Bargath	23	49142	G2P1L1	Controls	40	340	LN					3			110	60	NIL
38	Revathi	20	2609	Primi	Eclampsia	38	1934	EM. LSCS		1			1.8	1		220	140	3+
39	Arul Jyothi	21	3400	Primi	Eclampsia	34	1732	Ass Breech		1			1.7			140	96	2+
40	Nirmala	26	1457	Primi	Severe Pre-eclampsia	37	1600	LN	1			1	2.35			160	130	4+
41	Nithya	19	3113	Primi	Severe Pre-eclampsia	30	1048	LN			1		1.1		1	170	124	4+
42	Jeyabarathi	22	1128	G2A1	Mild Pre-eclampsia	39	355	LN			1		3.3			150	90	1+
43	Jerina	22	427	G2P1L1	Severe Pre-eclampsia	40	420	EM. LSCS					2.5			160	112	3+
44	Deivanayaki	21	1998	Primi	Severe Pre-eclampsia	40	689	EM. LSCS					2.1	1		170	110	3+
45	Nandhini	20	2120	Primi	Severe Pre-eclampsia	39	643	EM. LSCS					1.75	1		160	120	4+
46	Jayasree	30	369	G2P1L1	Mild Pre-eclampsia	38	294	EM. LSCS					2.5			140	92	2+
47	Shalini	26	1735	Primi	Mild Pre-eclampsia	40	650	EM. LSCS					3.5			150	80	1+

S. No	Name	Age	IP No	OBS Code	Group	GA(Wks)	LDH (IU)	Mode Delivery	Hellp	Eclampsica	Abruption	Maternal Death	Wt of Baby(Kg)	IUGR	IUD	SBP	DBP	U/A
48	Renuka	24	831	G3P2L2	Mild Pre-eclampsia	40	242	EM. LSCS					2.9			142	94	1+
49	Bhuvaneshwari	21	1361	Primi	Mild Pre-eclampsia	30	245	LN					1.2			150	100	2+
50	Amutha	32	1703	G2P1L1	Controls	38	458	LN					2.8			110	60	NIL
51	Mekala	21	2406	Primi	Controls	39	202	LN					2.7			112	72	Trace
52	Meenatchi	26	2777	G2P1L1	Controls	40	392	LN					4.1			112	80	NIL
53	Punithavathi	21	3110	Primi	Controls	40	239	LN					3			110	72	NIL
54	Usha	25	3192	G3P2L2	Controls	38	283	LN					3.2			120	80	NIL
55	Nirmala	24	3279	G2P1L1	Controls	39	494	LN					2.6			110	82	1+
56	Vani	28	5319	Primi	Eclampsia	32	1800	LN		1			1.7			200	120	4+
57	Nandhini	20	5537	Primi	Mild Pre-eclampsia	28	793	Ass Breech			1		1		1	150	90	1+
58	Umavathi	21	4046	Primi	Severe Pre-eclampsia	34	1098	LN			1		1.5		1	150	114	4+
59	Sandhya	23	4278	Primi	Severe Pre-eclampsia	37	520	EM. LSCS					2.25			168	120	3+
60	Padmavathy	24	4665	G2A1	Severe Pre-eclampsia	34	294	EM. LSCS					2			172	130	3+
61	Jaya	20	570	Primi	Severe Pre-eclampsia	40	469	EM. LSCS					2.5			160	90	2+
62	Padmavathy	23	4786	Primi	Severe Pre-eclampsia	35	510	EM. LSCS					2.2			150	110	3+
63	Noorbevi	21	6876	Primi	Severe Pre-eclampsia	38	284	EM. LSCS					2.75			160	90	1+
64	Jayanthi	27	4907	G2A1	Mild Pre-eclampsia	40	637	EM. LSCS					2.75			140	90	1+
65	Regina	24	5035	G2P1L1	Mild Pre-eclampsia	39	474	EM. LSCS					3.45			150	90	1+
66	Porkodi	24	4578	G4P2L2A1	Mild Pre-eclampsia	34	537	LN					1.75			150	100	2+
67	Geetha	25	4788	G3P2L0	Mild Pre-eclampsia	40	653	LN					3.2			140	90	1+
68	Suganthi	29	3657	Primi	Controls	40	432	LN					3.1			120	70	NIL
69	Maheshwari	24	4179	G3P2L1	Controls	40	212	LN					3.1			112	72	NIL
70	Anjali	22	5395	Primi	Controls	36	119	LN					3			106	70	Trace
71	Anandhi	20	5986	Primi	Controls	38	183	LN					2.2			120	82	NIL

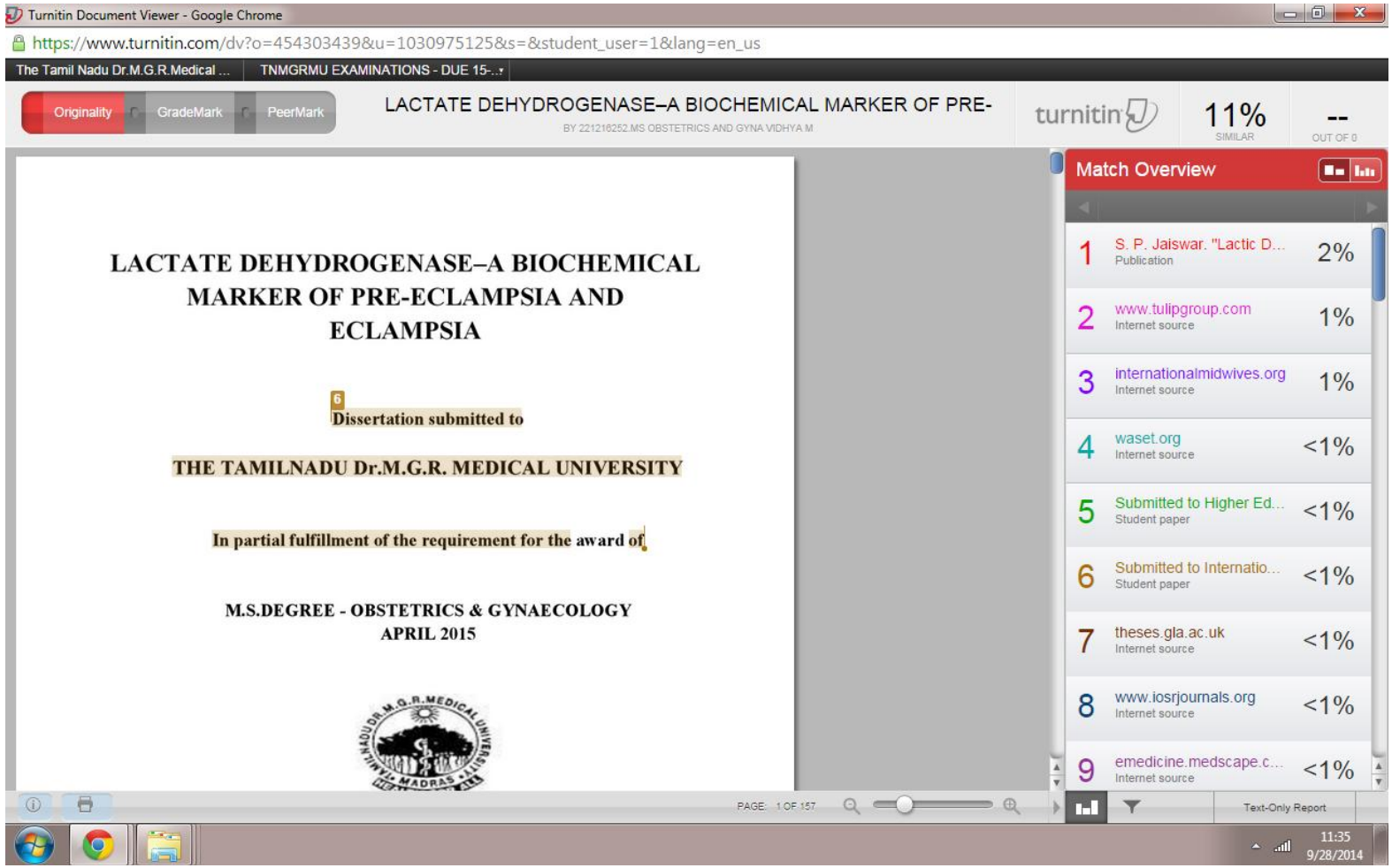
S. No	Name	Age	IP No	OBS Code	Group	GA(Wks)	LDH (IU)	Mode Delivery	Hellp	Eclampsica	Abruption	Maternal Death	Wt of Baby(Kg)	IUGR	IUD	SBP	DBP	U/A
72	Komala	20	6295	Primi	Controls	40	284	LN					2.8			122	74	NIL
73	Anitha	24	6435	G2P1L1	Controls	40	344	LN					2.75			116	80	NIL
74	Gayathri	23	6713	Primi	Controls	39	304	LN					2.9			114	70	Trace
75	Chitra	23	6989	Primi	Controls	37	482	LN					2.5			110	60	NIL
76	Parvathi	24	7064	G3P2L1A1	Mild Pre-eclampsia	40	345	LN					3.5			140	90	2+
77	Jayaseela	26	7128	G2P1L1	Mild Pre-eclampsia	40	423	LN					3.75			150	90	2+
78	Shylabanu	20	8268	Primi	Eclampsia	30	1892	EM. LSCS		1			1.2			180	126	3+
79	Adhilakshmi	23	9286	Primi	Mild Pre-eclampsia	38	234	EM. LSCS			1		2.9			140	100	2+
80	Lakshmi	30	9982	G2P1L1	Severe Pre-eclampsia	37	1204	EM.Rpt LSCS			1		1.6			160	120	3+
81	Sangeetha	28	7608	Primi	Mild Pre-eclampsia	32	123	LN			1		1.5			140	100	2+
82	Kothainayagi	20	7777	Primi	Severe Pre-eclampsia	36	489	EM. LSCS					2.3			160	110	2+
83	Ezhilarasi	22	7926	Primi	Severe Pre-eclampsia	36	194	EM. LSCS					2.2			168	120	3+
84	Indira	27	7232	G2P1L1	Severe Pre-eclampsia	36	182	LN					2.2			170	120	3+
85	Saranya	20	7223	Primi	Severe Pre-eclampsia	38	389	LN					2.25			160	110	3+
86	Prema	26	7229	Primi	Mild Pre-eclampsia	35	642	EM. LSCS					2.2			140	90	1+
87	Sivashankari	21	8830	Primi	Mild Pre-eclampsia	40	203	EM. LSCS					3.25			142	90	1+
88	Sumithra	24	50662	Primi	Mild Pre-eclampsia	34	230	LN					2.6			150	100	2+
89	Deepa	25	14824	G2P1L1	Severe Pre-eclampsia	30	1506	EM. LSCS			1		1.25		1	200	120	3+
90	Sathya	23	15108	G3P1L1A1	Severe Pre-eclampsia	32	1530	EM. LSCS			1		1.5		1	180	130	3+
91	Vinitha	22	15451	Primi	Mild Pre-eclampsia	36	297	EM. LSCS			1		2.14			140	90	2+
92	Lakshmi	22	12004	G2P1L1	Severe Pre-eclampsia	38	293	EM. LSCS					2.75			160	90	2+
93	Alamelu	27	12818	Primi	Severe Pre-eclampsia	39	204	EM. LSCS					2.75			166	80	2+
94	Praveena	22	14618	Primi	Severe Pre-eclampsia	40	109	LN					2.9			160	82	2+
95	Allirani	26	11753	Primi	Severe Pre-eclampsia	40	532	LN					2.8			160	90	1+

S. No	Name	Age	IP No	OBS Code	Group	GA(Wks)	LDH (IU)	Mode Delivery	Hellp	Eclampsica	Abruption	Maternal Death	Wt of Baby(Kg)	IUGR	IUD	SBP	DBP	U/A
96	Kaveri	26	11830	Primi	Mild Pre-eclampsia	40	432	EM. LSCS					3.7			140	90	1+
97	Revathi	22	12460	G2P1L1	Mild Pre-eclampsia	40	345	EM. LSCS					3.45			146	100	1+
98	Guna	24	11539	G3P2L3	Mild Pre-eclampsia	34	564	LN					1.5		1	150	100	2+
99	Ranjani	20	11787	Primi	Mild Pre-eclampsia	40	576	LN					3			140	92	1+
100	Mariammal	22	14178	G2P1L1	Controls	36	273	EM. LSCS					2.3			122	80	NIL
101	Sharmila	24	12350	G2P1L1	Controls	40	282	EM. LSCS					2.7			120	70	NIL
102	Umaudayavani	22	11491	G2P1L1	Controls	37	183	EM. LSCS					2.5			110	60	NIL
103	Maria	25	12465	Primi	Controls	39	138	LN					3.25			112	60	Trace
104	Krishnaveni	21	12730	Primi	Controls	37	209	LN					2.6			130	70	NIL
105	Jayalakshmi	29	13316	G2P1L1	Controls	39	308	LN					3.5			120	72	NIL
106	Sowmya	20	13632	G2A1	Controls	38	401	LN					2.7			112	64	NIL
107	Renuga	22	14897	Primi	Eclampsia	38	723	EM. LSCS		1			2.5			150	100	2+
108	Poongodhai	31	19593	Primi	Eclampsia	30	3200	EM. LSCS		1		1	1.2			160	120	4+
109	Ezhilarasi	23	15137	G2P1L1	Mild Pre-eclampsia	32	689	EM.Rpt LSCS			1		1.5		1	140	90	2+
110	Devaki	22	15408	Primi	Severe Pre-eclampsia	38	240	LN					3			160	120	3+
111	Rekha	22	16084	Primi	Mild Pre-eclampsia	40	408	EM. LSCS					3.2			146	92	1+
112	Kanvizhi	23	15985	Primi	Controls	40	145	EM. LSCS					2.7			120	70	NIL
113	Subadra	20	16439	Primi	Controls	40	132	EM. LSCS					3			110	60	NIL
114	Manjula	23	23162	Primi	Eclampsia	36	909	EM. LSCS		1			2.25			180	100	4+
115	Rasathi	23	20844	Primi	Eclampsia	38	1030	EM. LSCS		1		1	2.2			160	120	3+
116	Sathya	20	23073	Primi	Severe Pre-eclampsia	34	900	EM. LSCS			1		2		1	160	130	4+
117	Nazreen	25	23840	G2P1L1	Mild Pre-eclampsia	38	644	EM. LSCS			1		2.5		1	146	92	1+
118	Lavanya	27	24155	Primi	Severe Pre-eclampsia	38	745	EM. LSCS			1		2.8		1	150	110	4+
119	Radha	23	23331	Primi	Mild Pre-eclampsia	40	302	LN			1		3		1	150	100	2+

S. No	Name	Age	IP No	OBS Code	Group	GA(Wks)	LDH (IU)	Mode Delivery	Hellp	Eclampsica	Abruption	Maternal Death	Wt of Baby(Kg)	IUGR	IUD	SBP	DBP	U/A
120	Pramila	21	23671	Primi	Severe Pre-eclampsia	39	1040	LN			1		2.75		1	160	116	4+
121	Nathiya	21	22094	Primi	Severe Pre-eclampsia	39	209	EM. LSCS					2.2			150	110	3+
122	Prema	22	22389	Primi	Severe Pre-eclampsia	38	1304	EM. LSCS					2.7		1	156	110	3+
123	Premalatha	23	23529	Primi	Mild Pre-eclampsia	40	237	EM. LSCS					3.15			140	92	1+
124	Manju	25	23834	Primi	Mild Pre-eclampsia	40	198	EM. LSCS					3			150	90	1+
125	Rohini	20	22280	Primi	Mild Pre-eclampsia	38	109	EM. LSCS					2.75			140	92	1+
126	Selvi	23	23435	G2P1L1	Mild Pre-eclampsia	39	183	LN					3.25			146	90	2+
127	Tamilselvi	18	22275	Primi	Mild Pre-eclampsia	40	284	LN					3.4			142	94	1+
128	Vinodhini	24	24734	G2P1L0	Controls	40	282	EM. LSCS					3.5			110	70	NIL
129	Varalakshmi	24	24316	Primi	Controls	40	273	EM. LSCS					2.9			120	70	NIL
130	Sangeetha	20	23721	Primi	Controls	39	392	EM. LSCS					3.4			110	60	NIL
131	Geetha	22	24312	Primi	Controls	39	374	LN					3			120	60	NIL
132	Mariyammal	22	24341	Primi	Controls	39	294	LN					3			110	72	NIL
133	Bhavani	24	24124	G2P1L1	Controls	40	290	LN					3.5			110	68	NIL
134	Kalayarsi	26	25935	Primi	Eclampsia	32	1234	EM. LSCS		1			1.4			150	100	3+
135	Jyothi	34	26839	G2P1L1	Eclampsia	39	430	EM.Rpt LSCS		1			3.2			160	110	4+
136	Valli	22	26927	G2A1	Eclampsia	32	1049	EM. LSCS		1			1.2			170	110	4+
137	Priya	26	28019	Primi	Eclampsia	34	1089	EM. LSCS		1			1.6			160	120	4+
138	Saroja	25	25542	G3P2L2	Mild Pre-eclampsia	39	367	EM. LSCS			1		3		1	140	96	2+
139	Dhanya	23	26219	Primi	Severe Pre-eclampsia	36	760	EM. LSCS			1		2.6			180	120	3+
140	Anitha Devi	24	26225	G3P2L2	Mild Pre-eclampsia	38	743	EM. LSCS			1		2.5		1	150	96	2+
141	Selvi	30	26818	G4P1L1A2	Severe Pre-eclampsia	37	1392	EM. LSCS			1		2.8			190	120	3+
142	Deepa	22	27136	Primi	Mild Pre-eclampsia	34	543	EM. LSCS			1		1.7			150	92	2+
143	Vengammal	26	28121	G3P2L2	Severe Pre-eclampsia	36	753	EM. LSCS			1		1.9			170	126	3+

S. No	Name	Age	IP No	OBS Code	Group	GA(Wks)	LDH (IU)	Mode Delivery	Hellp	Eclampsica	Abruption	Maternal Death	Wt of Baby(Kg)	IUGR	IUD	SBP	DBP	U/A
144	Muthalammal	24	28749	G3P2L2	Severe Pre-eclampsia	38	604	EM. LSCS			1		2.75			150	110	3+
145	Aruna	22	29191	G2P1L1	Severe Pre-eclampsia	37	796	EM. LSCS			1		2.25			166	112	4+
146	Pushpalatha	28	24918	Primi	Mild Pre-eclampsia	34	384	LN					1.5		1	150	92	2+
147	Elavarasi	28	24770	G2A1	Mild Pre-eclampsia	34	292	LN					1.5		1	140	100	2+
148	Rajeshwari	27	26404	G2P1L1	Severe Pre-eclampsia	37	630	EM. LSCS					1.7	1		180	110	3+
149	Shanthakumari	20	25668	Primi	Mild Pre-eclampsia	38	128	EM. LSCS					3			140	90	1+
150	Anbarasi	25	25122	Primi	Mild Pre-eclampsia	39	134	LN					2.7			150	90	1+
151	Subha	22	28816	Primi	Controls	38	328	EM. LSCS					2.25			110	70	NIL
152	Shanthi	23	28863	G4A3	Controls	37	284	EM. LSCS					2.5			126	70	NIL
153	Mariya	30	26107	G3A2	Controls	38	194	EM. LSCS					2.75			122	72	NIL
154	Sudha	28	25032	Primi	Controls	39	104	EM. LSCS					2.5			100	64	NIL
155	Thilagavathi	29	24711	Primi	Controls	40	145	EM. LSCS					3			112	76	NIL
156	Jyothi	23	29280	Primi	Eclampsia	36	1980	EM. LSCS		1			2			160	122	4+
157	Kalaimathi	27	29980	Primi	Severe Pre-eclampsia	37	333	EM. LSCS					2.5			162	100	2+
158	Sathya	22	31040	Primi	Severe Pre-eclampsia	37	374	EM. LSCS					2.3			168	90	2+
159	Geethalakshmi	24	30497	Primi	Severe Pre-eclampsia	38	284	LN					3			170	92	2+
160	Amala Mery	29	29274	Primi	Mild Pre-eclampsia	38	219	EM. LSCS					2.8			140	100	1+
161	Suguna	24	29986	Primi	Mild Pre-eclampsia	37	295	EM. LSCS					2.8			150	90	2+
162	Savithri	22	30803	Primi	Mild Pre-eclampsia	39	284	LN					2.5			140	96	1+
163	Kalyani	35	29249	G2P1L1	Mild Pre-eclampsia	40	325	LN					2.7			140	100	1+
164	Tamilselvi	24	29337	G2P1L1	Mild Pre-eclampsia	39	276	LN					2.75			148	102	1+
165	Prema	26	29315	Primi	Controls	36	121	EM. LSCS					2.3			110	72	NIL
166	Amaravathy	20	30032	G2A1	Controls	40	101	LN					3.3			120	80	1+
167	Rajelakshmi	22	30046	G2P1L1	Controls	39	167	LN					3			110	82	NIL

S. No	Name	Age	IP No	OBS Code	Group	GA(Wks)	LDH (IU)	Mode Delivery	Hellp	Eclampsica	Abruption	Maternal Death	Wt of Baby(Kg)	IUGR	IUD	SBP	DBP	U/A
168	Anuradha	27	30007	G2P1L0	Controls	40	289	LN					3.4			112	72	NIL
169	Maheshwari	23	29911	G2P1L1	Controls	36	245	LN					2.4			110	74	NIL
170	Mahalakshmi	25	29861	G2P1L1	Controls	40	321	LN					3.6			122	84	NIL
171	Nalini	24	29560	Primi	Controls	38	345	LN					2.5			132	82	NIL
172	Sathya	21	29721	G2A1	Controls	39	432	LN					3			110	76	NIL
173	Udaya	28	29367	Primi	Eclampsia	34	1048	EM. LSCS		1			1.5			160	112	4+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Dissertation submitted to
THE TAMILNADU Dr.M.G.R. MEDICAL UNIVERSITY
In partial fulfillment of the requirement for the award of
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