THE ROLE OF SERUM LACTATE DEHYDROGENASE AND OTHER BIOCHEMICAL PARAMETERS IN ASSESSING THE SEVERITY OF PREECLAMPSIA

DISSERTATION SUBMITTED FOR

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MADURAI MEDICAL COLLEGE,

MADURAI

CERTIFICATE

This is to certify that the dissertation entitled **THE ROLE OF SERUM LACTATE DEHYDROGENASE AND OTHER BIOCHEMICAL PARAMETERS IN ASSESSING THE SEVERITY OF PREECLAMPSIA** submitted by **Dr.V.SWETHA,** to the Tamil Nadu Dr. M.G.R. Medical University in partial fulfilment of the requirement for the award of M.S.(Obstetrics & Gynaecology) Branch II is a Bonafide research work carried out by her under our direct supervision and guidance.

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This is to certify that the dissertation entitled **"THE ROLE OF SERUM LACTATE DEHYDROGENASE AND OTHER BIOCHEMICAL PARAMETERS IN ASSESSING THE SEVERITY OF PREECLAMPSIA"** is a bonafide and genuine research work done by **Dr.V.SWETHA,** in partial fulfilment of the requirement for the degree in M.S.(Obstetrics & Gynaecology), under guidance of **Prof. Dr. N.MAHALAKSHMI, MD., DGO.,** Additional Professor, Department of Obstetrics & Gynaecology.

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DECLARATION

I, **Dr.V.SWETHA,** hereby declare that, I carried out this work on **"THE ROLE OF SERUM LACTATE DEHYDROGENASE AND OTHER BIOCHEMICAL PARAMETERS IN ASSESSING THE SEVERITY OF PREECLAMPSIA"** in GOVT RAJAJI HOSPITAL at the Department of Obstetrics & Gynaecology, Madurai under the guidance of **Prof. Dr. N.MAHALAKSHMI MD,DGO** Professor of Obstetrics & Gynaecology during the period of September 2015 to September 2016. I also declare that this bonafide work has not been submitted in part or full by me or and others for any award, degree or diploma to any other University or Board either in India or abroad.

This is submitted to The Tamilnadu Dr. M. G. R. Medical University, Chennai in partial fulfilment of the rules and regulation for the award of M.S degree Branch – II (Obstetrics & Gynecology) to be held in April 2017.

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INTRODUCTION

Maternal mortality, remains as a cause for serious concern in India. Preeclampsia can be detected and managed accordingly with the help of blood test during pregnancy.

Hypertensive disorders complicate 5-10% of all pregnancies and together they are one member of the deadly triad along with hemorrhage and infection that contributes greatly to maternal morbidity and mortality. These hypertensive disorders of pregnancies is also one of the main reasons of fetal and neonatal morbidity and mortality.

The Incidence of hypertensive disorders in pregnancy varies between 5-10% and it is rising as women are postponing their first pregnancy to a later age and increased pre pregnancy weight. On the other hand, the incidence of eclampsia is declining in the industrialized and affluent society due to better antenatal care and management of Preeclampsia.

High blood pressure is a sign, not a disease, reflecting an increase in cardiac output or more commonly increased peripheral resistance. These vascular changes can arise in a number of disorders that may have different impact on the course of pregnancy and its outcome.

At the severe end, preeclampsia can result in pulmonary edema, cerebral hemorrhage, hepatic failure, renal failure, eclampsia, DIC, maternal death. Fetal complications include IUGR, still birth, preterm birth and admission to a neonatal intensive care unit.

Data collected by FOGSI shown the pregnancy induced high BP (Preeclampsia- eclampsia – responsible for 12% of maternal death in India is the leading cause of maternal mortality that can be prevented by sample blood tests.

Hypertension in pregnancy is associated with increased maternal as well as perinatal morbidity and mortality. Early recognition of women at risk of Preeclampsia will enable the identification of high risk women who may benefit from enhanced surveillance and prophylaxis. Timely diagnosis and intervention may prevent complications and improve the pregnancy outcome.

Hypertensive disorders continue to occur globally. They are common cause for interventions/operative procedures, and cause around 15-20% maternal mortality and 20-25% of perinatal mortality.

. How pregnancy incites or aggregates hypertension remains unsolved despite decades of intensive research.

The combination of proteinuria and hypertension during pregnancy markedly increases the risk of perinatal mortality and morbidity mainly

due to its effect on the fetus like intrauterine growth restriction, prematurity, and its associated complications.

A method of protecting women from preeclampsia based on insight into the pathogenesis would thus be of great benefit to both mother and baby.

Since the exact etiology is still unknown, not much can be done for prevention even though prophylactic calcium, aspirin, magnesium, zinc, anti oxidants with lifestyle change are being advocated.

Quality antenatal care, early diagnosis of hypertensive disorders, and a high index of suspicion is the key to early diagnosis of complications and timely intervention. Search to identify those at risk, who should undergo extra evaluation and monitoring for prophylactic therapies by accurate, sensitive clinically acceptable screening tests, prior to or during pregnancy before the pathology sets in is the need of the day.

All pregnant women should have their blood pressure regularly monitored as early as possible during gestation. If blood pressure is found to be elevated, the women should be allowed to rest for about 30 minutes and the blood pressure will be measured again.

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AIMS AND OBJECTIVES

To correlate the severity of the disease, maternal and perinatal outcome with serum Lactate dehydrogenase and other biochemical parameters in patients with preeclampsia.

REVIEW OF LITERATURE

1. As per the study on 2011 by S.P.Jaiswar, Amrit Gupta , Rekha sachan in Dept of Obst and gynaec, Queen mary hospital CSMMU, Lucknow, Lactate dehydrogenase levels have significant association with various maternal and fetal outcomes in patients with preeclampsia and eclampsia.

2. As per the study on 1995 by He S, Brennek, Kallnes A, Blomback M, Increased concentrations of lactic dehydrogenase in pregnancy with preeclampsia is found to be a predictor for the birth of small for Gestational age infants.

3. As per the research study 2013, conducted by purnima daysarkar,sonal sogani, Elevated levels of serum LDH indicate the tissue damage related to endothelial vascular damage and is the main cause of occurrence of preeclampsia

4. As per the study conducted byAmrit D.Sonagra,Dattatreya.k in 2012, regular estimation of LDH, ALP and uric acidisadvisable for pregnancy diagnosed with hypertensive disorders in ordrto detect and prevent the morbidity and mortality in mother as well as in fetus. It may also give an idea regarding the disease severity and functioning of liver and kidne in these patients.progreesive increase in their levels should be considered as a signal for prompt intervention to improve the pregnancy outcome.

5. As per the study of international journal of Biomedical research conducted by Mansi Gandhi in 2015 it was found that Serum LDH and serum uric acid are reliable and inexpensive markers to predict the severity and outcome of hypertensive disorders of pregnancy.

6. As per the study conducted on 2015 b,.y sreelatha s, Bharathi A, Ramya s, shwetha sarau, It was found that increased LDH levels correlate with severity of PIH and has got poor perinatal outcome.So it can be considered as one of the biochemical marker.

7. As per the study conducted on 2014 by IOSR, Journal of dental and medical sciences by Andrews L ,Mehta L,Sharma A, it has been found that there exist a statistically significant difference between S.Uric acid, LDH,AST,ALP with maternal death in PIH cases. So higher levels of these parameters can be a very useful marker to identify the occurrence of the complications of preeclampsia in early pregnancy and in the management of women with preeclampsia.

HISTORICAL REVIEW

- Preeclampsia / Eclampsia have been recognized as a clinical entity since the times of Hippocrates.
- The fact that delivery of placenta ameliorates the disease was known 200 years ago and was advocated since then.
- With the invention of sphygmomanometer, a practical method of measuring blood pressure is discovered in 1896. It was known that arterial hypertension was a major sign and precedes the occurrence of convulsion.
- Zweifel in 1916 first termed Toxemia as the disease of theories
- Tehoblad in 1930 consistently held that dietic deficiencies particularly calcium and vitamins B and D together with intra abdominal mechanical factors the cause of eclampsia.
- JCM Browne and Veale in 1953, showed the presence of placental infarcts in PIH.
- Dixon and Robertson in 1958, showed lumen diminishing vascular lesions in patients who had PIH.
- Young in 1974, attributed pregnancy toxemia to placental toxin elaborated in areas of acute red infarction in placenta.

The classification of hypertensive disorders complicating pregnancy

The classification of hypertensive disorders complicating pregnancy by the working group of the NHBPEP (2000). There are five types of hypertensive disease (Williams Twenty fourth Edition)

1. Gestational hypertension (formerly pregnancy-induced hypertension that included transient hypertension)
2. Preeclampsia.
3. Eclampsia.
4. Preeclampsia superimposed on chronic hypertension.
5. Chronic hypertension.

Gestational hypertension

BP ≥ 140/90 mm Hg for first time during pregnancy , No proteinuria , BP returns to normal < 12 weeks postpartum and final diagnosis is made only in the postpartum period.

Preeclampsia

Minimum criteria :

BP ≥ 140/90 mm Hg after 20 weeks gestation and resolves by 12 weeks post partum. Proteinuria ≥300mg/24hours or ≥1+dipstick.

Increased certainty of preeclampsia :

BP ≥ 160/110 mg Hg,

Proteinuria 2.0g/24hours or ≥ 2+ dipstick

Serum Creatinine >1.2mg/dl unless known to be previously elevated

Platelets < 100,000/mm^3

Microangiopathic hemolysis (increased LDH)

Elevated ALT or AST

Persistent headache or other cerebral or visual disturbance

Persistent epigastric pain

Eclampsia

Seizures that cannot be attributed to other causes in a women with preeclampsia

Superimposed Preeclampsia (on chronic hypertension)

New-onset proteinuria ≥ 300 mg/24 hours in a hypertensive women after 20 weeks gestation.

A sudden increase in proteinuria or blood pressure or platelet count <100,000/mm^3 in women with hypertension and proteinuria before 20 weeks gestation.

Chronic Hypertension

BP ≥ 140/90mm Hg before pregnancy or diagnosed before 20 weeks gestation not attributable to gestational trophoblastic disease

or

Hypertension first diagnosed after 20 weeks gestation and persistent after 12 weeks postpartum

DEFINITION:

Hypertension is diagnosed when the resting blood pressure is 140/90 mmHg or greater, Korotkoff phase V is used to define diastolic pressure.

Measurement of Hypertension

Hypertension is diagnosed when two BP readings of 140/90 mm Hg or greater are noted 4 hours apart within a 1-week period. Measuring BP with an appropriate-sized cuff placed on the right arm at the same level as the heart is important. The patient must be sitting and, ideally, have had a chance to rest for at least 10 minutes before the BP measurement. She should not be lying down in a lateral decubitus position, as the arm often used to measure the pressure in this position will be above the right atrium.

The Korotkoff V sound should be used for the diastolic pressure. In cases in which the Korotkoff V sound is not present, the Korotkoff IV sound may be used, but it should be noted as such. The difference between the Korotkoff IV and V sounds may be as much as 10 mm Hg. When an automated cuff is used, it must be able to record the Korotkoff V sound. When serial readings are obtained during an observational period, the higher values should be used to make the diagnosis.

PRE ECLAMPSIA:

This condition is best described as a pregnancy-specific syndrome of reduced organ perfusion secondary to vasospasm and endothelial activation.

Risk factors for preeclampsia:

- Age > 35 years
- Parity – Primi gravida
- Interval from last pregnancy > 10 years
- Obstetric factors

  Preeclampsia or gestational hypertension in prior pregnancy

  Multiple gestation

  Hydatiform mole

  Hydrops fetalis

Abnormal uterine artery Doppler at 18-24 weeks

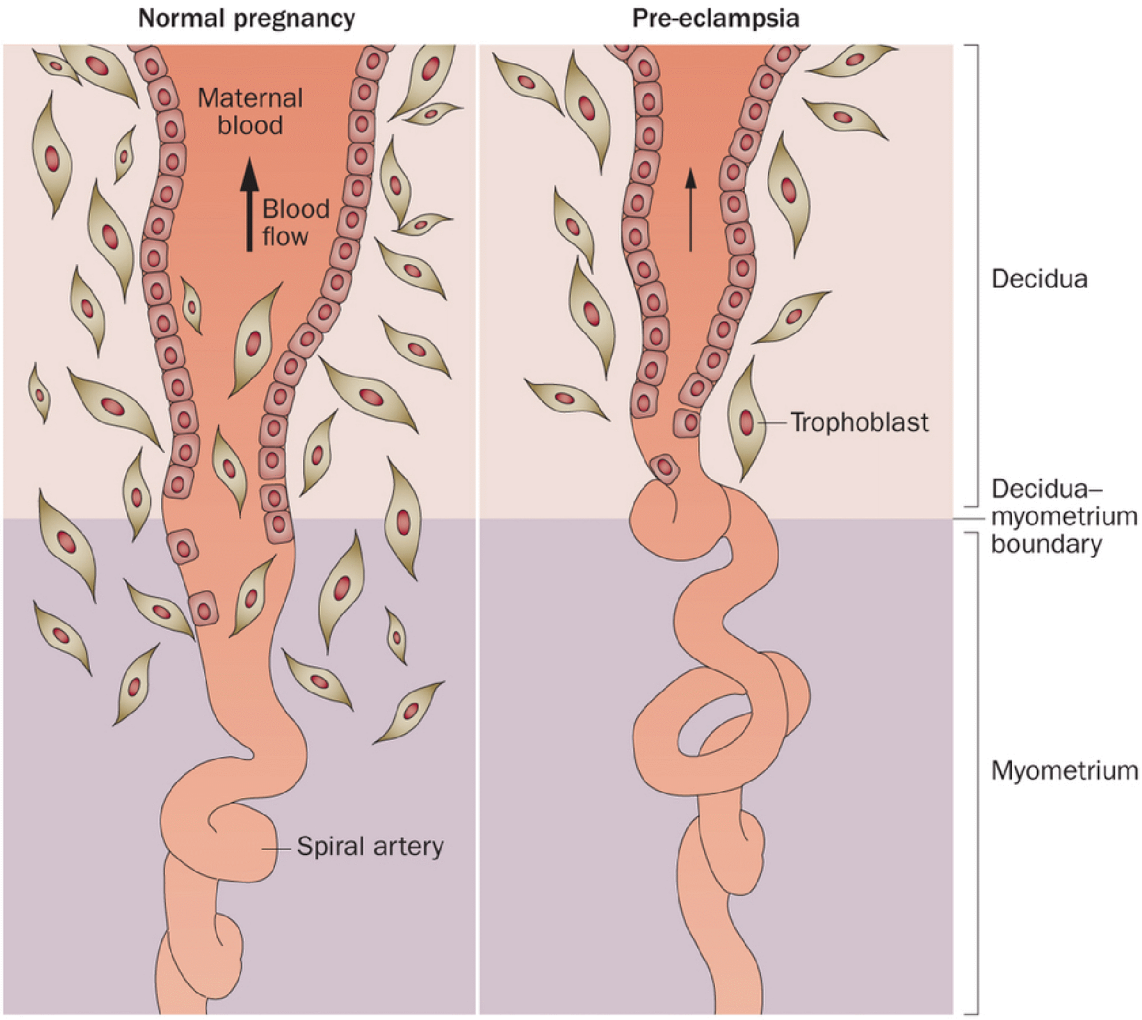
- Family History of preeclampsia
- Pre existing medical disorders

  Hypertension, Diabetes mellitus, Obesity (BMI) of 35kg/m2or more), Renal diseases ,Vascular diseases ,Autoimmune disease Thrombophilias.

ETIOPATHOGENESIS

Currently plausible potential causes include the following.

1. Abnormal trophoblastic invasion of uterine vessels.
2. Endothelial dysfunction and vasospasm
3. Immunological intolerance between maternal and fetoplacental tissues.
4. Proangiogenic and antiangiogenic factors
5. Dietary deficiencies.
6. Genetic influences.



Spiral arterioles at the placental site in normal and pre-eclamptic pregnancies

Using electron microscopy, De wolf and co-workers examined arteries taken from the uteroplacental implantation site. They observed that early preeclamptic changes included endothelial damage, insudation of plasma constituents into vessel walls, proliferation of myointimal cells, and medial necrosis. They found that lipid accumulates first in myointimal cells and then in macrophages. Typically, the vessels affected by atherosis develop

aneurysmal dilatation and are frequently found in association with spiral arterioles that have failed to undergo normal adaptation. Obstruction of the spiral arteriolar lumen by atherosis may impair placental blood flow.

It is thought that these changes cause placental perfusion to be pathologically diminished, which eventually leads to the preeclampsia syndrome.

IMMUNOLOGICAL FACTORS :

The pathological lesions found in the placenta in cases of preeclampsia bear some similarity to those found in kidneys rejected after transplantation suggestive of acute graft rejection There is circumstantial evidence to support the theory that preeclampsia is immune mediated. It has been suggested that loss of maternal tolerance to that paternally derived placental and fetal antigen .The risk of preeclampsia is appreciably enhanced in circumstances where formation of blocking antibodies to placental antigenic sites might be impaired. This may arise in situations in which effective immunization by a previous pregnancy is lacking, as in first pregnancies; or in which the number of antigenic sites provided by the placenta are unusually great compared with the amount of antibody, as with multiple fetuses.

Beginning in the early second trimester, women destined to develop preeclampsia have a significantly lower proportion of helper T cells (Th1) compared with that of women who remain normotensive. This Th1/Th2 imbalance, with Th2 dominance, may be mediated by adenosine, which is found in higher levels in Preeclamptic compared with normotensive women.

These helper T lymphocytes secrete specific cytokines that promote implantation, and their dysfunction, may favour preeclampsia

In women with anticardiolipin antibodies, placental abnormalities and preeclampsia develop more commonly. Antibodies associated with β2 glycoprotein I appear most relevant. Immune complexes and antiendothelial cell antibodies may also be involved.

ENDOTHELIAL DYSFUNCTION

Data show that an imbalance of proangiogenic and antiangiogenic factors produced by the placenta may play a major role in mediating endothelial dysfunction. Angiogenesis is critical for successful placentation and the normal interaction between trophoblasts and endothelium.

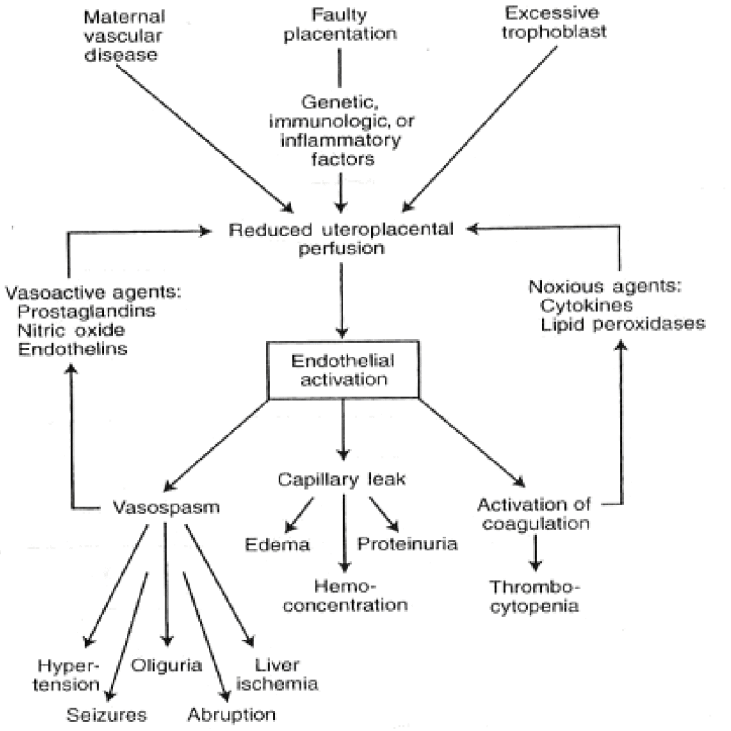
Several circulating markers of endothelial cell injury have been shown to be elevated in women who develop preeclampsia before they became symptomatic. These include endothelin, cellular fibronectin, and plasminogen activator inhibitor-1, with an altered prostacyclin/thromboxane profile also present.

Evidence also suggests that oxidative stress, circulatory maladaptation, inflammation, and humoral, mineral, and metabolic abnormalities contribute to the endothelial dysfunction and pathogenesis of preeclampsia.

The endothelial cell dysfunction associated with preeclampsia can result from a generalized perturbation of the normal, generalized maternal intravascular inflammatory adaptation to pregnancy. In this hypothesis, preeclampsia is considered a disease due to an extreme state of activated leukocytes in the maternal circulation. Briefly, cytokines such as tumor necrosis factor -α (TNF-α) and the interleukins may contribute to the oxidative stress associated with preeclampsia. Oxidative stress is characterized by reactive oxygen species and free radicals that lead to formation of self propagating lipid peroxides. These in turn generate highly toxic

radicals that injure endothelial cells, modify their nitric oxide production, and interfere with prostaglandin balance.

The effect of oxidative stress in preeclampsia have given rise to increased interest in the potential benefit of anti oxidants to prevent preeclampsia.



PROANGIOGENIC AND ANTIANGIOGENIC

Soluble endoglin

Several observations support the role of sEng in the pathogenesis of preeclampsia. It is found in the blood of women with preeclampsia up to 3 months before the clinical signs of the condition, its level in maternal blood correlates with disease severity, and the level of sEng in the blood drops after delivery.

In studies on pregnant rats, administration of sEng results in vascular permeability and causes hypertension. There is also evidence that it has a synergistic relationship with sFlt-1, because it increases the effects of sFlt-1 in pregnant rats; this results in HELLP syndrome, as evidenced by hepatic necrosis, hemolysis, and placental infarction. Moreover, sEng inhibits TGF-beta in endothelial cells and also inhibits TGF-beta-1 activation of nitric oxide mediated vasodilatation.

OXIDATIVE STRESS

Anti oxidants are a diverse family of compounds that function to prevent over production of and damage caused by noxious free radicals. Examples of antioxidants include vitamin E or α tocopherol, vitamin C, and β –Carotene.

NUTRITIONAL FACTORS:

A number of dietary deficiencies or excesses over the centuries have been blamed as the cause of eclampsia. Blood pressure in non pregnant individuals is affected by a number of dietary influences including minerals and vitamins. Some studies have shown a relationship between dietary deficiencies and the incidence of preeclampsia. This was followed by studies of supplementation with various elements such as zinc, calcium, and magnesium to prevent preeclampsia. Other studies showed that in the general population diet high in fruits and vegetables that have antioxidant activity is associated with decreased blood pressure. In one case control study the incidence of preeclampsia was doubled in women whose daily intake of ascorbic acid was less than 85mg.

Obesity is a potent risk factor for preeclampsia. Evidence has accrued that obesity in non pregnant individuals causes endothelial activation and a systemic inflammatory response associated with atherosclerosis. In the study of pregnant women by wolf, C-reactive protein, an inflammatory marker, was shown to be increased in obesity, which in turn was associated with preeclampsia.

GENETIC FACTORS:

Familial predisposition for preeclampsia has been recognized single gene model and polygenic inheritance has been suggested 60% concordance in monozygotic female twin pairs has been reported by a Swedish study. Some have reported a HLA-DR4 association with preeclampsia. A number of single gene mutations and inherited thrombophilias may predispose to preeclampsia. Polymorphisms of the genes for TNF, Lymphotoxin – α and interleukin -1β and have been studied with varying results.

GENERALISED AND INTENSE VASOSPASM

Vascular constriction causes resistance and subsequent hypertension, associated endothelial damage cause interstitial leakage through which blood constituents, including platelets and fibrinogen are deposited subendothelial with diminished blood flow because of maldistribution, and ischemia of surrounding tissues would lead to necrosis, hemorrhage and other end organ disturbances characteristic of the syndrome.

ENDOTHELIAL CELL ACTIVATION:

Various noxious placental factors released by ischemic changes and toxic radicals generated by oxidative stress cause activation and dysfunction of vascular endothelium. Intact endothelium decreases

responsiveness of vascular smooth muscles to agonists by release of Nitric oxide and it also has anticoagulant properties. Damaged or activated endothelium secretes substances that promote coagulation and increased sensitivity to vasopressors. Increased circulating fibronectin is marker of endothelial dysfunction.

ENHANCED PRESSOR RESPONSE:

Normal pregnant women are refractory to infused vasopressors like angiotensin II, However women who are destined to develop preeclampsia have increased vascular reactivity to angiotensin II and norepinephrine. This increased sensitivity precedes the onset of gestational hypertension. Normotensive nulliparous remained refractory to infused angiotensin II, but those who subsequently became hypertensive lost this refractoriness several weeks before the onset of hypertension. Women with underlying chronic hypertension have almost identical responses.

PROSTAGLANDINS:

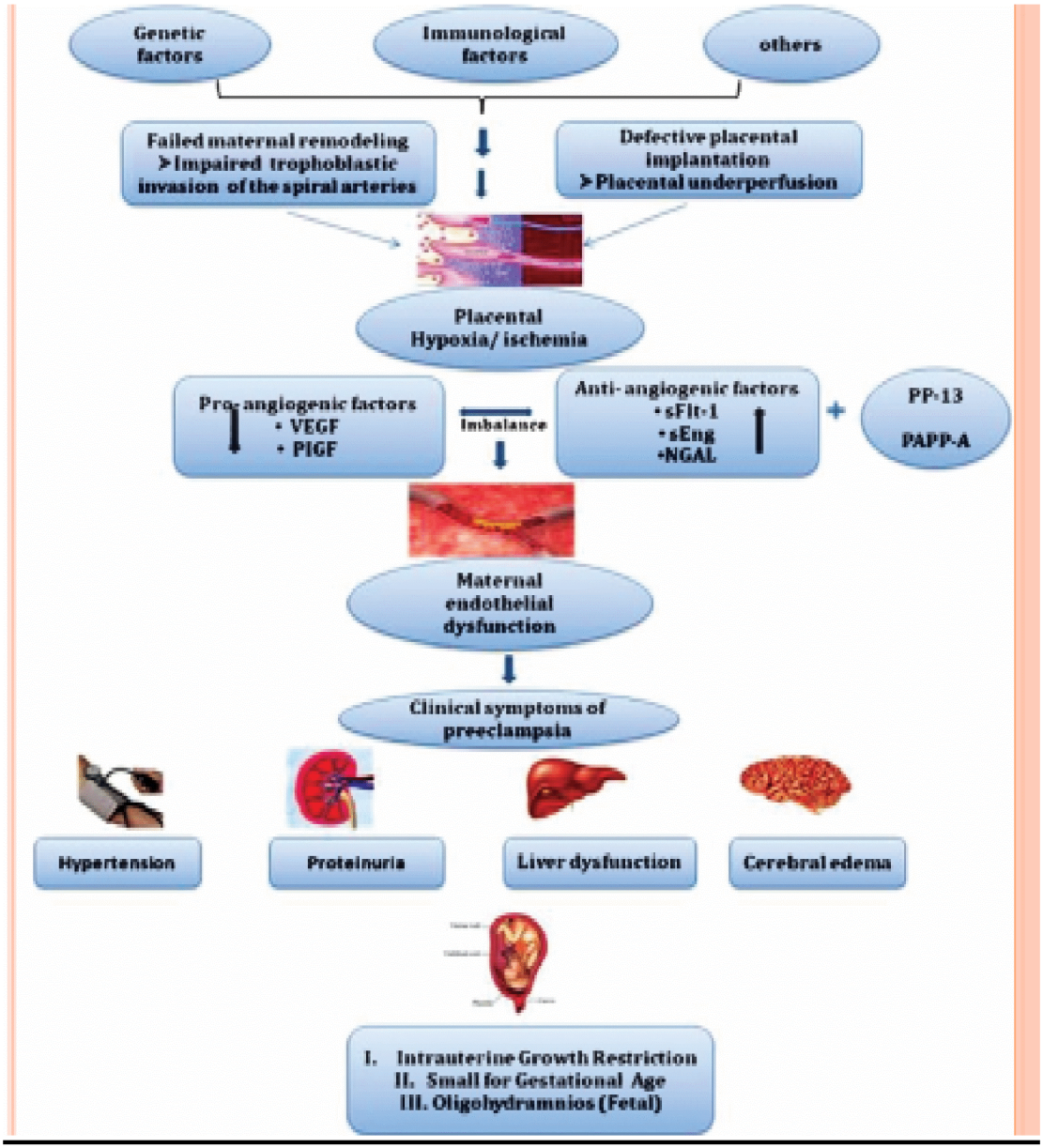
Endothelial Prostacyclin (PGI_2) a vasodilator, production is decreased in preeclampsia mediated by phospholipase A2. Thromboxane A2 (Vasoconstrictor and platelet aggregator) levels are increased. The Prostacyclin I_2: Thromboxane A2 ratio decreases, these changes result in vasoconstriction.

NITRIC OXIDE:

Nitric oxide is a potent vasodilator, synthesized from L-arginine by endothelial cells. Nitric oxide maintains the normal low pressure vasodilated state of fetoplacental circulation. Preeclampsia is associated with decreased endothelial nitric oxide synthesis which increases the cell permeability.

ENDOTHELINS:

Endothelin-1 is the primary isoform produced by human endothelium. These 21–amino acid peptides are potent vasoconstrictors. Levels in preeclampsia are higher when compared to normotensive pregnancies in response to endothelial activation.


Genetic factors
Immunological factors
others
Failed maternal remodeling
➢Impaired trophoblastic invasion of the spiral arteries
Defective placental implantation
➢Placental underperfusion
Placental Hypoxia/ ischemia
Pro- angiogenic factors
• VEGF
• PIGF
Imbalance
Anti- angiogenic factors
• sFlt-1
• sEng
•NGAL
+
PP-13
PAPP-A
Maternal endothelial dysfunction
Clinical symptoms of preeclampsia
Hypertension
Proteinuria
Liver dysfunction
Cerebral edema
I. Intrauterine Growth Restriction
II. Small for Gestational Age
III. Oligohydramnios (Fetal)

PATHOPHYSIOLOGY

PLACENTA

- fibrinoid necrosis,macrophages & mononuclear cell infiltration spiral artery narrowing acute atherosis & infarcts iugr,oligohydramnios,placental abruption & fetal demise

KIDNEY

- Glomerular & tubular dysfunction & glomerular endotheliosis lumen narrowed, reduced GFR & CREATININE CLEARANCE increased urea & creatinine
- ARF is a rare complication of preeclampsia but preeclampsia is a major cause of obstetric ARF
- Tubular dysfunction manifested as hyperuricemia & hypocalciuria proteinuria is due to increased capillary permeability

LIVER

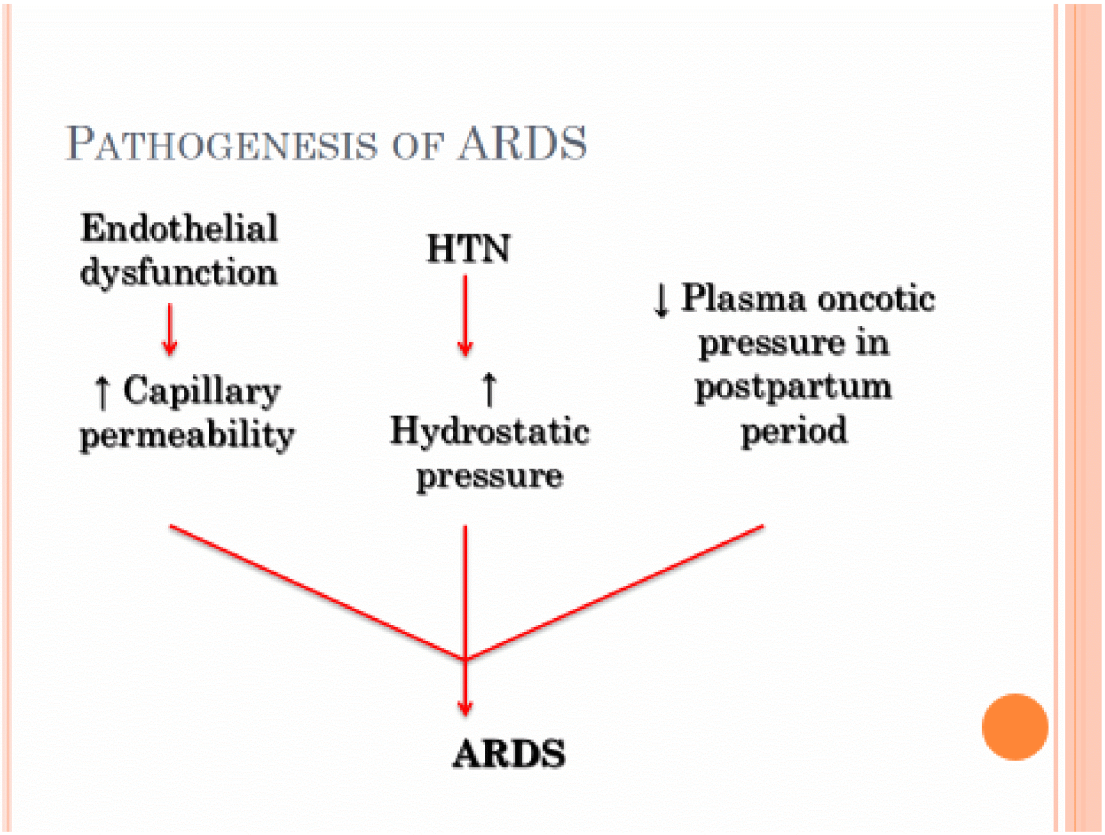
- Periportal thrombosis, fibrin deposition ,hemorrhage & necrosis
- Raised SGOT & SGPT, clinical jaundice, nausea vomiting
- Small hemorrhages coalesce to form subcapsular hematoma, stretching of glissons capsule & epigastric pain
- Rarely liver rupture

CVS

Severe disturbances of normal cardiovascular function are commonly seen in preeclampsia eclampsia. These are related to increased cardiac after load caused by hypertension. Cardiac preload is affected by pathologically diminished hypervolemia of pregnancy or iatrogenically increased by intravenous crystalloid solutions and endothelial activation with extravasation into the extra cellular spaces. Compared with normotensive women, the women who developed preeclampsia had significantly elevated cardiac output before hypertension developed with clinical onset of preeclampsia. Marked reduction in cardiac output and increased peripheral resistance was observed in preeclampsia.

- Increased cardiac afterload due to hypertension
- Diminished preload due to reduced hypervolemia of pregnancy in preeclampsia
- Fluid extravasation due to endothelial cell dysfunction ,pulmonary edema
- Haemoconcentration –hallmark of preeclampsia

ARDS



BRAIN

Cerebral vasospasm, small haemorrhages, thrombosis, fibrinoid necrosis. cerebral edema motor cortex-convulsions, frontal-throbbing headache unrelieved by analgesics, occipital-visual disturbance & cortical blindness

- in CT images localised hyperintense lesions seen in gray white matter in occipital lobe-PRES

EYES

Localised retinal vasospasm (commonest), haemorrhages & papilledema (severe hypertension), ischemia of lateral geniculate nuclei / retinal ischemia, infarction, detachment and blindness

HEMATOPOIETIC SYSTEM

Consumption coagulopathy, microangiopathic hemolysis with platelet adherence & fibrin deposition, schistocytes, burr cells & fragmented cells seen in peripheral smear.

HEMOCONCENTRATION:

Normally expected hypervolemia of pregnancy is severally curtailed in preeclamptic women. It is a likely consequence of generalized vasoconstriction and endothelial dysfunction with increased vascular permeability. Theses changes persists until a variable time after delivery when the vascular endothelium is repaired.

PREDICTION

Measurement in early pregnancy of a variety of biological, biochemical, and biophysical markers implicated in the pathophysiology of preeclampsia has been proposed to predict its development. Investigators have attempted to identify early markers

of faulty placentation, reduced placental perfusion, endothelial cell activation and dysfunction, and activation of coagulation.

Liver :

The pathophysiological changes in liver include infarction & hemorrhage resulting in symptoms like right upper or epigastric pain and tenderness / elevation in serum liver transaminase levels usually seen is severe disease. The characteristic periportal hemorrhage form areas of infection may extend to form a Subcapsular hematoma.

Women have elevated level of AST / ALT in Preeclampsia

Alkaline phosphatase level normally reach upto 3 times normal in pregnancy due to placental phosphatase.

Uric Acid:

Elevated serum uric acid levels due to decreased renal urate excretion are frequently found in women with preeclampsia. Plasma uric acid values exceeding 5.9mg/dL at 24 weeks had a positive predictive value for preeclampsia of 33 percent.

Fibronectin :

Endothelial cell activation is likely the cause of elevated serum cellular fibronectin levels in some women with preeclampsia. The women who subsequently developed preeclampsia had significantly

higher levels of fibronectin by 12 weeks but the positive predictive value was only 29 percent.

Coagulation Activation:

a) Thrombocytopenia and platelet dysfunction are integral features of preeclampsia, Increased destruction causes platelet volumes to increase because of relatively younger, and therefore larger, platelets are found. High platelet volumes are thought to be a marker of impending preeclampsia.

b) Fibrinolytic activity is normally decreased in pregnancy due to increased plasminogen activator inhibitors (PAI) 1 and 2. In preeclampsia PAI-1 is increased relative to PAI 2 because of endothelial cell dysfunction. The PAI-1: PAI-2 ratio to be predictive of preeclampsia in high –risk women.

Oxidative Stress:

Increased levels of lipid peroxides, coupled with decreased activity of antioxidants in women with preeclampsia, have raised the possibility that markers of oxidative stress might predict preeclampsia. For example, malondialdehyde is a marker of lipid peroxidation. Other markers are a variety of pro-oxidants or potentiators of pro-oxidants, including iron, transferrin, and ferritin

blood lipids, including triglycerides, free fatty acids, and lipoproteins; and antioxidants, including ascorbic acid and vitamin E.

Hyperhomocysteinemia is an independent risk factor for atherosclerosis in women who are not pregnant. Women with elevated serum homocysteine levels around midpregnancy had a three to fourfold risk of preeclampsia. Homocysteine levels are influenced by folic acid supplementation.

Cytokines:

These protein messengers are released by vascular endothelium and leukocytes as well as by macrophages and lymphocytes at the trophoblast-decidua interface. There are over 50 cytokines, and a number of these are elevated in preeclampsia. These include some interleukins and TNF-α.. A cascade of markers (e.g. C-reactive protein) arise from these reactions, and elevations of their levels have been suggested as possibly predictive of preeclampsia.

Placental Peptides:

As a result of the inflammatory cascade, a number of peptides are produced by the placenta, and some may prove to be markers for prediction of preeclampsia. Those studied include corticotropin releasing hormone, chorionic gonadotropin, activin A, and inhibin A. The problems with these are similar to those with other markers-

namely, they are variably elevated depending on the duration and severity of preeclampsia. Moreover, there usually is substantial overlap with normal pregnant women. Activin A and inhibin A were increased markedly in women who developed preeclampsia. Conversely, the other investigators have reported significant overlap of activin A and inhibin A levels in normotensive and preeclamptic pregnancies.

Combined first trimester serum levels of PLGF and sFlt1 were highly predictive of subsequent preeclampsia.

Fetal DNA:

Identification of fetal DNA in maternal serum may be predictive of preeclampsia. At same time that endothelial activation and inflammation occur, fetal cells and cellular material are released in to the maternal circulation.

Uterine Artery Doppler Velocimetry:

Measurement uteroplacental vascular resistance during Doppler ultrasound evaluation of uterine artery impedance in the second trimester has been used as an early screening test for preeclampsia. The rationale for this is based on the presumption that the pathophysiology of preeclampsia includes impaired trophoblastic invasion of the spiral arteries leading to reduction in uteroplacental

blood flow. Florio and associates (2003) combined inhibin A and activin A measurements in women with abnormal Doppler findings and reported that the probability of preeclampsia was 86 percent if both hormone markers were elevated and 17 percent if they were both unaltered.

Roll - over test :

A hypertensive response induced by having women at 28 to 32 weeks assume the supine position after lying laterally recumbent predicted gestational hypertension. Women who demonstrated a positive roll-over test were also found to be abnormally sensitive to infused angiotensin II. With preeclampsia, rather than gestational hypertension, as the end point, the positive predictive value was only 33 percent.

1. Uterine artery Doppler

Normally in non pregnant state, there is reduced diastolic flow and notching of the uterine arteries. But in pregnancy, due to trophoblastic invasion, the notching disappears. If there is persistence of uterine artery notching even in pregnancy, then it is highly suggestive of

1. preeclampsia 2. fetal growth retardation.

OXIDATIVE STRESS RELATED TESTS

a. fms like tyrosine kinase 1: VEGF, PEGF neutralises both the angiogenic peptides the serum maternal sFlt -1 levels are highly upregulated in preeclamsia

b. soluble endoglin.

It is a coreceptor for TGF proteins and is increasingly expressed in endothelium and syncytiotrophoblastic cells. Its mutations leads to telengiectasias. In pregnancy normally its level decreases but in preeclampsia, its level, depending on the severity rises several folds.

TESTS TO ASSESS FETOPLACENTAL UNIT ENDOCRINE DYSFUNCTION .

a. pregnancy associated plasma protein

It is secreted by the developing trophoblastic cells. Its level decreases greatly in preeclampsia. More reliable in FGR. Also a good marker for downs syndrome

In preeclampsia and FGR , there is a great fall in PAPP.

b. placental protein 13:

Highly expressed in placenta, helping in its implantation and remodelling of maternal vasculature.

a great fall in PP13 levels are encountered in preeclampsia and FGR around 11-13 weeks.

c. inhibin A, activin A

d. elevated AFP

e. elevated beta HCG.

MEAN ARTERIAL PRESSURE:

MAP= diastolic blood pressure+ 1/3 pulse pressure

MAP of more than 90 mm hg ,lacking the mid trimester fall is indicative of preeclampsia.

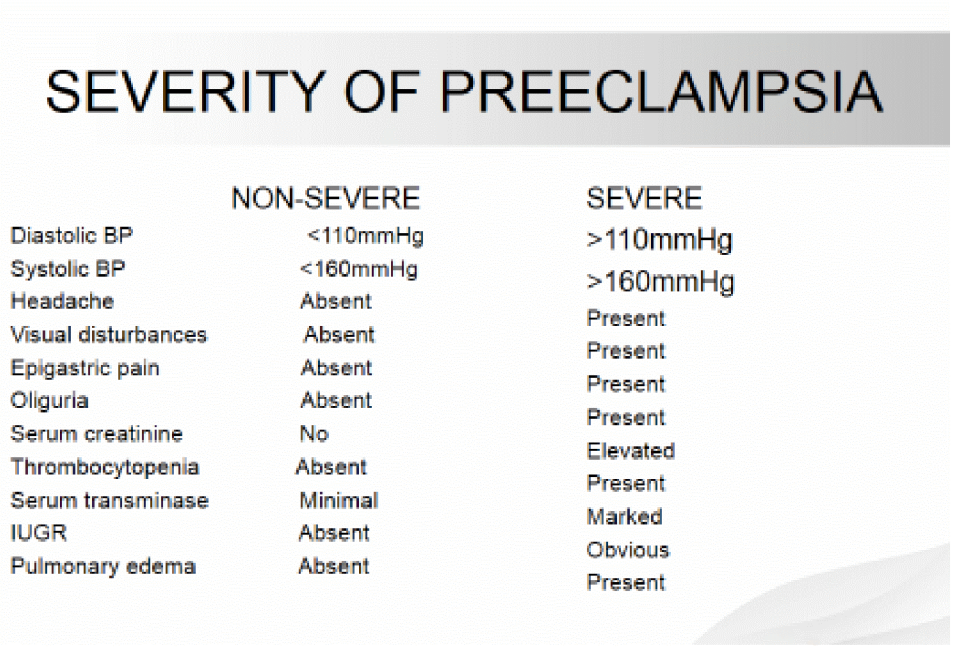
CLINICAL PREDICTORS OF PREECLAMPSIA

1. Previous history of preeclampsia
2. Previous history of gestational hypertension
3. Previous history of anti phospholipid syndrome
4. Previous history of thrombophilias
5. Previous history of chronic kidney disease

DANGER SIGNS

- Persistent maternal headache
- Visual disturbance
- Nausea or vomiting
- Epigastric pain
- Diminished sleep

- Decreased urinary output
- Thrombocytopenia
- Pulmonary edema
- Impaired liver function test findings
- Oligohydraminos
- IUGR
- Microangiopathic hemolysis



LABORATORY DIAGNOSIS

URINE EXAMINATION

- 24-hour urinary protein
- 1+= 300mg/L
- 2+ = 1g/L
- 3+ = 3g/L

- 4+ =4g/L
- Sugar,pus cells and deposits Culture and sensitivity
- Proteinuria is the hallmark of Preeclampsia alone and the quantity of protein excretion has been suggested as an indicator of severity of Preeclampsia.
- Urine albumin excretion rate has the best predictive value for Preeclampsia in hypertensive patients.
- According to ACOG, proteinuria is no longer considered as criteria to diagnosis severity of preeclampsia

BLOOD AND COAGULATION:

HAEMOGLOBIN

The iron-containing protein, which transports oxygen within the red blood cells

- In normal pregnancy, there is a natural decrease in Hb, due to haemodilution
- In preeclampsia, expected plasma volume increase is impaired, affecting Hb estimation As the pregnancy progresses, and capillaries become more damaged, they begin to leak, causing fluid to shift from the blood vessels to extravascular spaces

- Blood therefore becomes more concentrated, and a raised Hb may indicate reduced plasma (hemoconcentration)
- Plasma volume normal with mild disease, but reduced with severe preeclampsia

PLATELETS:

PLATELETS: 1.5 TO 5 LAKHS/MM3

Total number of thrombocytes, which play an integral role in haemostasis

- Platelet levels decrease as they aggregate following damage to the endothelial cells of the capillaries
- Day-to day variations common so serial measurements are necessary and more informative

Thrombocytopenia: Frequency and intensity depends on the intensity of disease process and duration of preeclampsia. Thrombocytopenia < 100,000/cumm indicates severe disease and delivery is indicated, platelet count increases progressively to reach normal level within 3 to 5 days after delivery. Thrombocytopenia results from platelet activation, aggregation and decreased life span. Levels of platelet activating factor are increased, thrombopoietin, platelet bound and circulating platelet binding immunoglobulins are increased.

MEAN PLATELET VOLUME [MPV]: 6.4–9.7 fl

- The average lifespan of platelets is 5 – 9 days, and immature platelets are larger than mature ones

RENAL FUNCTION TESTS

Proteinuria:

Renal perfusion and glomerular filtration are reduced in preeclampsia. Reduced plasma volume may result in mild to moderately diminished glomerular filtration rate. Severe intra renal vasospasm may cause plasma creatinine to elevate several times the non-pregnant value.

Plasma uric acid may be elevated especially in severe disease. Acute renal failure due to acute tubular necrosis may occur in neglected cases. Rarely irreversible renal cortical necrosis can occur.

- Recovery is complete after delivery
- In severe cases, intense anoxia may produce extensive arterial thrombosis leading to bilateral renal cortical necrosis (Incidence 4%)
- Diagnostic criteria
  - Creatinine clearance by measuring 24 hr urine creatinine remains the gold standard of GFR estimation in pregnancy.

ANY SUSTAINED FALL IN OUTPUT < 0.5ML/KG/HR OR RISING SERUM CREATININE SHOULD ALERT THE CLINICIAN OF LIKELIHOOD OF AKI

Endothelial damage results in increased permeability to many large molecular weight proteins. Thus increased albumin excretion is accompanied by other proteins like hemoglobin and globulins and transferrin. On electron microscopy – glomerular capillary endothelial swelling accompanied by subendothelial deposits of protein material known as glomerular capillary endotheliosis is seen.

BLOOD UREA AND SERUM CREATININE

BLOOD UREA: 20-40 mg/dl

An organic chemical compound which essentially is the waste produced when the body metabolizes protein

- A late sign of renal injury as a result of preeclampsia is impairment of glomerular filtration which causes an increase in serum urea
- SERUM CREATININE-0.6-0.9 mg/dl

  A breakdown product of creatinine (muscle waste material), which is constantly produced and filtered by the kidneys
- Creatinine is removed from the body entirely by the kidneys

- If kidney function is abnormal, creatinine levels will increase in the blood

  BOTH BLOOD UREA AND SERUM CREATININE are lower in normal pregnancy as a consequence of increased GFR and hemodilution. However in preeclampsia these levels are higher than in normal pregnancy although usually within the normal non pregnant stage. Levels above the non pregnant range suggest underlying chronic renal impairment, either as a cause or result of hypertension.an acute rise in blood urea may occur in acute renal failure which may rarely complicate severe preeclampsia or may sometimes accompany eclampsia.

URATE (URIC ACID

Normal level- 4-6 mg/dl

End product of protein metabolism

- Uric acid an end product of protein metabolism excreted by renal tubule, in Preeclampsia this function is impaired by damage to kidney and blood levels rise.

- Hyperuricemia an early sign of renal involvement in Pre-eclampsia, is the result of reduced renal clearance due to altered

tubular processing of uric acid preceding glomerular filtration, which will cause albuminuria.

- Hyperuricemia is one of the most consistent & earliest detectable changes in Preeclampsia and is associated with poor fetal outcome.

- Excreted by renal tubule, in preeclampsia this function is impaired by damage to kidney and blood levels rise
- High levels associated with poor fetal outcome
- Useful diagnostic feature of early preeclampsia
- Diet may affect level

PROTEIN-CREATININE RATIO: 0 – 30 mg/mmol

- Random (spot) urine protein-creatinine ratio collected during normal daytime activity
- Provides an accurate and rapid quantitation of proteinuria in pregnant women with systemic arterial hypertension and increased risk of preeclampsia

CREATININE CLEARANCE: 120–160ml/min

The volume of plasma completely cleared of creatinine per unit of time

- Assesses the glomerular filtration rate

- Gives an indication of renal function
- Creatinine clearance may be reduced in preeclampsia as a result of decreased GFR
- Assessed via 24 hour specimen

LIVER FUNCTION TESTS:

Anatomical changes like periportal hemorrhage and infarction are seen in fatal cases. Bleeding from these regions may cause hepatic rupture of subcapsular hematoma. They are more likely in women with HELLP syndrome. HELLP Syndrome characterized by hemolysis, elevated liver enzymes and low platelets. This disorder is characterize by hepatic endothelial dysfunction followed by platelet aggregation, activation and consumption resulting in ischemia and hepatocyte death, this vasculopathy can be limited to a hepatic segment or involve entire liver. The classic hepatic lesion associated with HELLP syndrome is periportal or focal parenchymal necrosis with hyaline deposits in sinusoids. Less frequently occurring larger vessel disease can affect wider vascular distributions in the liver and cause more catastrophic outcomes such as hepatic infarction or subcapsular hematoma.

Hepatic complications

- Transaminases frequently elevated
- Epigastric/ Subcostal pain (distension of liver capsule by edema or subcapsular bleeding)
- Coagulopathy (high INR)
- Acute fatty liver

Serum bilirubin - important factor in predicting Maternal mortality

ASPARTATE TRANSAMINASE [AST] :< 45 U/L

An enzyme, involved in cellular metabolism

that has raised levels in acute liver damage

- Also found in high concentrations in heart, muscle, kidney, pancreas and red blood cells
- If any of these areas are damaged the blood levels of the enzyme will increase
- Not specific for liver function

ALKALINE PHOSPHATASE [ALP]: 90 - 250 U/L

An enzyme made in the liver, bone, and the placenta, released into the blood during injury and during such normal activities as bone growth and pregnancy

- Involved in cell metabolism and present in nearly all tissues

- Levels reach up to 3 times normal in pregnancy due to placental phosphatase

ALANINE TRANSAMINASE [ALT]: 7 - 45 U/L

An enzyme involved in cellular respiration, found in highest amounts in the liver. It is released into the blood through liver injury.

- Found in low levels in other tissues
- High levels are specific for hepatic damage

In normal pregnancy AST and ALT usually remain unchanged.

In severe preeclampsia they may be elevated

<u>BILIRUBIN:</u>

Bilirubin is a product that results from the breakdown of hemoglobin

- Serum bilirubin levels do not usually rise in pre-eclampsia, unless complicated by HELLP syndrome

ACTIVATED PARTIAL THROMBOPLASTIN TIME [APTT]: 24-35 sec When you bleed, the body launches the coagulation cascade. There are three pathways to this event. The APTT test looks at special proteins, called factors, found in two of these pathways (intrinsic).

THROMBIN CLOTTING TIME: 10-18 sec

A test which measures time required for plasma fibrinogen to form thrombin. Exogenous thrombin is added to citrated plasma and the time to clot formation is measured.

- TCT is prolonged with abnormalities of fibrinogen and in the presence of heparin or of fibrin/fibrinogen degradation products
- Prolonged in DIC as clotting mechanism fails
- A blood test that looks at how long it takes for blood to clot
- It can help tell if there are bleeding or clotting problems
- A prolonged APTT time can be indicative of disorders such as DIC, haemophilia, lupus, etc.
- Exaggerated increases may point to placental and hepatic damage in preeclampsia

Coagulation:

Abnormalities of coagulation and erythrocyte destruction is commonly seen in preeclampsia eclampsia. The changes are mild except for thrombocytopenia. Elevation of fibrin degradation products are occasionally seen. Thrombin time may be prolonged even with normal Fibrin Degradation Products. Thrombophilias are clotting factor deficiencies that lead to hypercoagulability and may be associated with early onset preeclampsia. Antithrombin III levels are

decreased in preeclampsia, fibronectin is elevated consistent with the view that preeclampsia cause vascular endothelial injury.

DIC

Bleeding as the incision is being closed & Incision site bleeding

(In preeclampsia there is vasoconstriction which affects blood flow to the liver. Liver releases coagulation factors.)

LACTATE DEHYDROGENASE IN PREECLAMPSIA

Preeclampsia and eclampsia complicate6 to 8% of all pregnancies and lead to various maternal and fetal complications.PIH is an multisystem disorder and lead to a lot of cellular death.Lactate dehydrogenase is an intracellular enzyme which converts pyruvic acid to lactic acid during the process of glycolysis.glycolysis is the major energy pathway in the placenta.hypoxia in preeclampsia further enhances glycolysis and increases LDH activity. Studies have shown that LDH activity and gene expression are higher in placenta of preeclampsia than normal pregnancy.Hypoxia induces LDH enzyme activity in trophoblast resulting in higher lactate production.LDH has five isoforms and among all LDH A4 seen in placenta with preeclampsia is most responsive to hypoxia.Elevated levels of LDH

indicate cellular damage and dysfunction,so it can be used as a biochemical marker because it reflects the severity of disease, occurrence of complications and fetal outcome.its estimation would prove useful because these complications are preventable

Elevated level of LDH also seen in cases of HELLP Syndrome.Many authors have used elevated total LDH (>600IU/L) as a diagnostic criteria for hemolysis.Among all five isoforms, only two of them (LDH1 and LDH2) are released from ruptured red blood cells

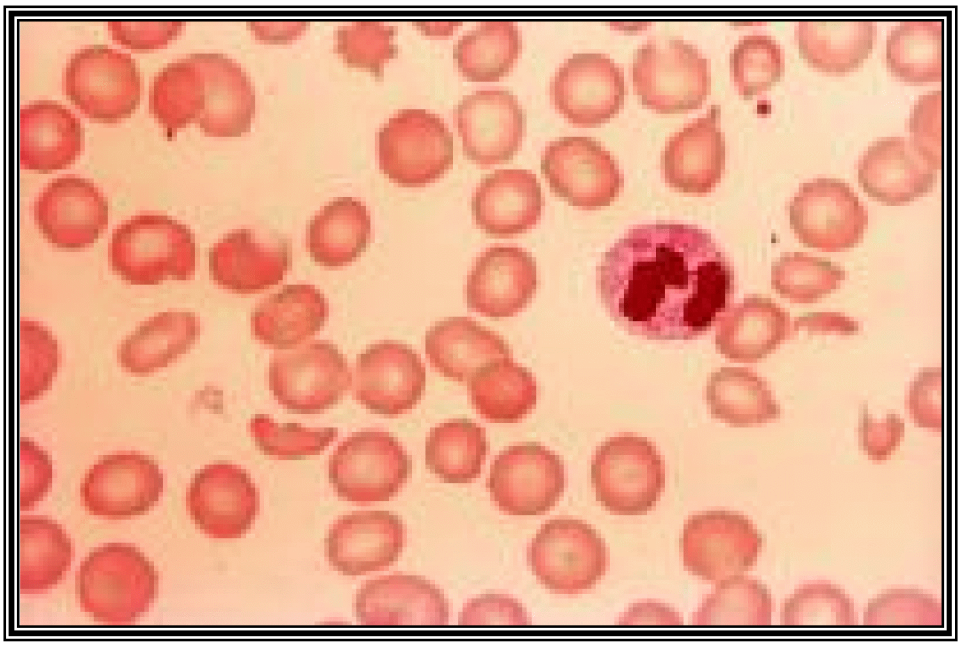
Lactate dehydrogenase :

- LDH is an intracellular enzyme that catalyses the conversion of lactate to pyruvate.
- Cell damage results in leakage of the enzyme into the circulation causing high serum levels of LDH.
- High levels of LDH correlate with the severity of Pre-eclampsia and associated maternal and fetal mortality.
- Complication of Preeclampsia & perinatal mortality have been found to be significantly increased when LDH levels are > 800 IU/l

Since LDH levels are increased in these women due to cellular death, its levels are used to assess the extent of cellular

death and thereby severity of disease in patient with Preeclampsia. This can be further used in making decision, regarding management strategies to improve maternal and fetal outcome.

Fragmentation Hemolysis is frequently seen in severe preeclampsia as indicated by elevated LDH levels and peripheral changes like schisocytosis, spherocytosis and reticulocytosis. These changes are due to microangiopathic hemolysis caused by endothelial disruption with platelet aggregation and fibrin deposition. Increased erythrocyte membrane fluidity associated with HELLP syndrome may predispose to hemolysis. Erythrocyte membrane changes like increased adhesiveness, aggregation may also facilitate a hypercoagulable state.



Hemolysis / Anemia
Target cells, schistocytes & paucity of platelets
Associated with increased LDH
Extremely rare complication is HEPATIC RUPTURE.

BRAIN:

Headaches and visual symptoms are common in severe preeclampsia. Convulsions if associated define eclampsia. Cerebral pathology is either gross hemorrhage due to rupture arteries caused by severe hypertension or cerebral edema, hyperemia, thrombosis, ischemia and hemorrhage, these changes being wide spread. Cerebral blood flow studies have shown that in preeclampsia increased cerebral perfusion is counter balanced by increased cerebrovascular resistance with no net change in the cerebral blood flow. In eclampsia the auto regulation is disrupted, hyperperfusion is seen. Visual disturbances and blindness are due to occipital lobe vasogenic edema resolves completely following delivery, cerebral edema when associated with symptoms like lethargy, confusion, blurred vision due to obtundation and coma, are very susceptible to sudden and severe blood pressure elevation which actually worsen the vasogenic edema.

PRES (Posterior Reversible Encephalopathy Syndrome)

- **Clinical manifestations :**

  head ache, blindness, scotoma convulsions

- **Posterior circulation**

  - more susceptible
  - less sympathetic innervation of the vertebro-basilar vasculature to protect the parenchyma from rapid increases in arterial blood pressure

ACUTE RESPIRATORY FAILURE

- Acute pulmonary edema may complicate severe preeclampsia and is a frequent reason for critical care unit admission associated with high maternal and perinatal mortality and morbidity

CVS complications :

- **Cardiac Failure**

  Low filling pressures and a hyperdynamic circulation

- **Cardiomyopathy**

  Rare complication

  Treatment similar to other types of CCF

Acute MI Enhanced vascular reactivity to angiotensin II & nor epinephrine.

UTEROPLACENTAL PERFUSION :

Compromised uteroplacental perfusion from vasospasm is almost certainly a major culprit in the genesis of increased perinatal morbidity and mortality associated with preeclampsia. Brosens and associates (1972) reported that the mean diameter of myometrial spiral arterioles of 50 normal pregnant women was 500 μm. The same measurement in 36 women with preeclampsia was 200μm. Attempts to assess human maternal and placental blood flow have been hampered by several obstacles, including inaccessibility of the placenta, the complexity of its venous effluent, and the unsuitability of certain investigative techniques for humans.

UTERO PLACENTAL COMPLICATIONS :

Placental abruption :

Placental abruption is defined as premature separation of a normally situated placenta before delivery. Preeclampsia is by far the most important predisposing factor in the etiology of abruption.

IUGR:

IUGR fetus fails to achieve its genetic growth potential and consequently is at risk of increased perinatal morbidity and mortality. One of the most important causes for IUGR is preeclampsia.

Preterm delivery:

Preterm labour is defined by the WHO as the onset of labour prior to the completion of 37 weeks of gestation ion a pregnancy beyond 20 weeks of gestation. In severe preeclampsia in order to reduce maternal complications pregnancy is terminated and hence preterm delivery is iatrogenic.

FETAL OUTCOME:

Preeclampsia and eclampsia are associated with intrauterine growth restriction, intrauterine hypoxia and iatrogenic prematurity.

Preventions :

Numerous intervention have been tried to reduce the incidence and severity of preeclampsia

Various treatment options include pharmacological agents, dietary supplementation and life style modification.

Pharmacological agents :

Low molecular weight heparin, progesterone, nitric oxide donors, antihypertensive medication and diuretics are not effective in preventing preeclampsia. The only drug for the prevention of preeclampsia is low dose aspirin in women at high risk for developing the disease.

Diet and supplementation:

No reliable evidence in diet or dietary supplements on the occurrence of preeclampsia

Calcium supplementation with 1.5 – 2g calcium has shown to reduce the incidence of preeclampsia by half.

Zinc

It has been suggested that low serum zinc levels may be associated with suboptimal outcomes of pregnancy including preeclampsia. However cochrane review (2012) did not reveal any evidence of improved pregnancy outcome.

Nitric oxide :

Nitric oxide a potent vasodilator is synthesized from L arginine by endothelial cells. The dietary supplementation of L arginine is expected to prevent or decrease the severity of preeclampsia. However existing evidence shows no reduction in hypertensive disorders following the use of nitric oxide donors.

Rest :

Cochrane review (2005) included two small trials which investigated the effect of rest or reduced physical activity in normotensive women at risk of developing preeclampsia, from 29-32 weeks. There was a significant reduction in the relative risk of pre-

eclampsia. This data is however, insufficient to recommend a policy of rest as prophylactic intervention to prevent preeclampsia.

Exercise and physical activity:

Exercise and physical activity have been found to be useful in preventing hypertension in non pregnant women. However a prospective population based study failed to show any protective effect of physical activity on the incidence of preeclampsia.

Reduced dietary salt:

Two trials including 600 women at risk of preeclampsia randomized them to reduced dietary salt or normal dietary intake of salt. No correlation was observed between the dietary salt intake and the risk of preeclampsia.

Aspirin:

Low dose aspirin has been widely used further prevention of preeclampsia.

Exaggerated increase may point to placental and hepatic damage in Preeclampsia.

The platelet counts and the hemoglobin measurements will provide the disease severity and further management of labour.

The fetal outcome like growth restriction and other adverse outcomes will be monitored and measured by APGAR score and the outcome with elevated level of these parameters will be correlated.

MANAGEMENT OF MILD PRE-ECLAMPSIA

- **Monitoring of mother**
  - Blood pressure twice daily
  - urine examination – determine the degree of proteinuria
- **Monitoring of foetus**
  1. foetal movement
  2. foetal breathing
  3. non stress test
  4. contraction stress test
  5. amniotic fluid volume
- Foetal growth is assessed both by clinically and sonologically.
- patient is allowed to go if
- **Gestation less than 37 weeks**
  1. Diastolic blood pressure is normal.
  2. Proteinuria is insignificant

- If patient develops significant symptoms of severe pre-eclampsia IMMEDIATELY report to hospital.

- **Gestation more than 37 weeks**

Signs of foetal compromise – LABOUR IS INDUCED.

NO FOETAL COMPROMISE –CONTINUE FOR ANOTHER ONE WEEK

MANAGEMENT OF SEVERE PRE-ECLAMPSIA

- Hospitalisation
- Anti hypertensive therapy

- main goal- to maintain diastolic BP between 90 and 100 mm hg to prevent maternal complications like cerebral hemorrhages

DRUGS

- Formerly alpha methyl dopa has been used but due to delayed action and post partum depression it has been stopped
- *LABETALOL*

- starting dose - 100mg bd max dose – 2.4 g / day

- In case of emergency , intravenous labetalol 10 -20 mg iv is given.

- NIFEDIPINE

starting dose – 10 mg oral (titrated according to bp)

should never be given sublingually.

- Prophylactic magnesium sulphate should be given in case of severe preeclampsia.
- Mag Sulphate and Calcium channel blockers should not be combined

- MANAGEMENT PRINCIPLES OF SEVERE PREECLAMPSIA
- Admission in the labour room.
- Maternal and foetal evaluation for 24 hours.
- Administration of *magnesium sulphate* for 24 hours.
- If gestation
- less than 24 Weeks – STABLIZE THE PATIENT, magnesium sulphate administration and TERMINATION.
- greater than 34 Weeks – stabilise the patient magnesium sulphate and DELIVERY.
- 24 -34 weeks- Expectant management CORTICOSTEROID therapy Inj Betamethasone 12 mg *2 dose IM at 24 hrs interval.

Delivery within 24 hours is indicated in case of following conditions:

- Uncontrolled severe hypertension
- Eclampsia
- Pulmonary odema
- Abruptio placenta
- Signs of imminent eclampsia
- Deterioration of renal function
- Appearance of HELLP syndrome

- Platelet count less than 100000
- Oligohydramnios
- Oliguria
- *Use of an anti-hypertensive* in case of systolic BP greater than 160 and diastolic BP greater than 110.

ECLAMPSIA

- -Convulsive disorder characterized by GHT, Proteinuria, convulsion occurring during pregnancy non attributable to any organic causes.

  Occurrence: It may be antepartum, intrapartum or postpartum

Clinical Stages of Eclampsia:

1. Premonitory Stage:

- Characterized by facial twitching lasting for few sec to 30 sec

2.Tonic Stage:

- Characterized by generalised muscular contraction. with arms flexed & hands clenched and body in tonic spasm. Lasting for 15 to 20secs.

3.Clonic Stage:

- characterized by alternate contraction & relaxation of muscles, tongue may be bitten by violent action of jaws, respiratory muscles are halted & diaphragm is fixed, froth in the mouth.

- last about a min.

4.Coma:

- seizure ceases , breathes stertorously & respiration gradually becomes quieter.

COMPLICATION

- Injuries, Hyperpyrexia, Cerebral haemorrhage, Renal failure, Pulmonary edema

MATERIALS AND METHODS

DETAILED STUDY PROPOSAL

To compare the serum LDH and other biochemical parameters in normal pregnant women and in women with Preeclampsia in Ante partum period admitted in Govt Rajaji Hospital, Madurai from Sep 2015 to Aug 2016.

To study the correlation of maternal and perinatal outcomes with serum LDH level and other biochemical parameters like Blood urea, Serum creatinine, total bilirubin, SGOT, SGPT, Serum Uric acid, Hb and platelets.

STUDY DESIGN

Comparative Study.

STUDY TYPE

Prospective case control study

PERIOD OF STUDY:

12 Months

SAMPLE SIZE:

150 Women

75 Controls.

75 cases of Preeclampsia and eclampsia

PARTICIPANTS:

Patients with Preeclampsia and eclampsia admitted in labour room and normal healthy antenatal pregnant women of third trimester.

INCLUSION CRITERIA:

Pregnant women enclosed in this study in third trimester of pregnancy are divided into following groups,

Group 1 : healthy normal pregnant women in third trimester of pregnancy (Patients with normal blood pressure and with no significant comorbidities as mentioned in exclusion criteria).

Group 2 : Patients with preeclampsia and eclampsia (subjects). This was further subdivided into following subgroups

1. Non severe preeclampsia
2. Severe preeclampsia
3. Eclampsia

Patient was grouped under non severe preeclampsia if the measured blood pressure was more than 140/90 mmhg measured on two occasions atleast 6 hours apart

Patient was grouped under severe preeclampsia if any of the following characteristics features present

- ✓ systolic BP ≥ 160 mmhg , Diastolic BP≥ 110 mmhg, presence of headache , visual disturbances, Epigastric pain, Thrombocytopenia, impaired LFT , oligohydramnios. Fetal growth restriction, pulmonary edema

Patient was grouped under eclampsia if there is sudden onset of tonic clonic convulsions unrelated to other cerebral conditions.

Patient was also grouped under three groups according to serum LDH levels

1. LDH - < 600IU/L
2. LDH - 600- 800 IU/L
3. LDH > 800IU/L

Data collection :

Informed consent and patient history will be obtained from all participants.

Blood samples of all participants are taken from right or left cubital vein and sent for lab analysis.

All women will be followed up for complications until delivery and early postpartum period and babies till early Neonatal period.

EXCLUSION CRITERIA

Mothers with hypertension at or < 20 weeks gestation.

1. preexisting DM
2. Preexisting Renal disease.
3. Pre existing liver disease.
4. Thyroid disorders.
5. Epileptic disorder
6. History of hyperuricemia

Complete obstetric history, general physical examination and systemic examinations were carried out. Patients will be subjected to laboratory investigations such as serum LDH , Hb, platelets, Serum uric acid , Blood urea, S creatinine, total bilirubin, SGOT, SGPT.

Blood urea measured by DAM method. Serum creatinine measured by Jaffes method. Bilirubin measured by Diazo method. SGOT, SGPT and ALP is measured by Modified IFCC method. LDH measured by DGKCH method. Uric acid measured by Uricase - POD method.

For the benefit of the patient and to prevent the adverse fetal outcome in this study we correlate the severity of Preeclampsia with LDH

levels in serum of these patients and also measure the other biochemical parameters as mentioned above.

They will be compared with that of normal healthy antenatal pregnant women without any of the above mentioned symptoms and try to correlate the elevated level of the LDH between the normal and women with Preeclampsia. Thus in this study, LDH will be given more emphasis than rest of these parameters.

RESULTS

Table -1 Distribution of patients with Age

Age in years	Control (75)	Non severe (28)	Severe (27)	Eclampsia (20)
< 20	9	6	3	0
21 - 25	29	13	14	13
26 - 30	28	7	6	6
> 30	9	2	4	1
Total	75	28	27	20
Mean	25.09	24.71	25.7	25.2
SD	4.16	4.07	5.36	2.88

Age in years	Non severe (28)	Severe (27)	Eclampsia (20)	Total
< 25	19	17	13	49
> 25	9	10	7	26

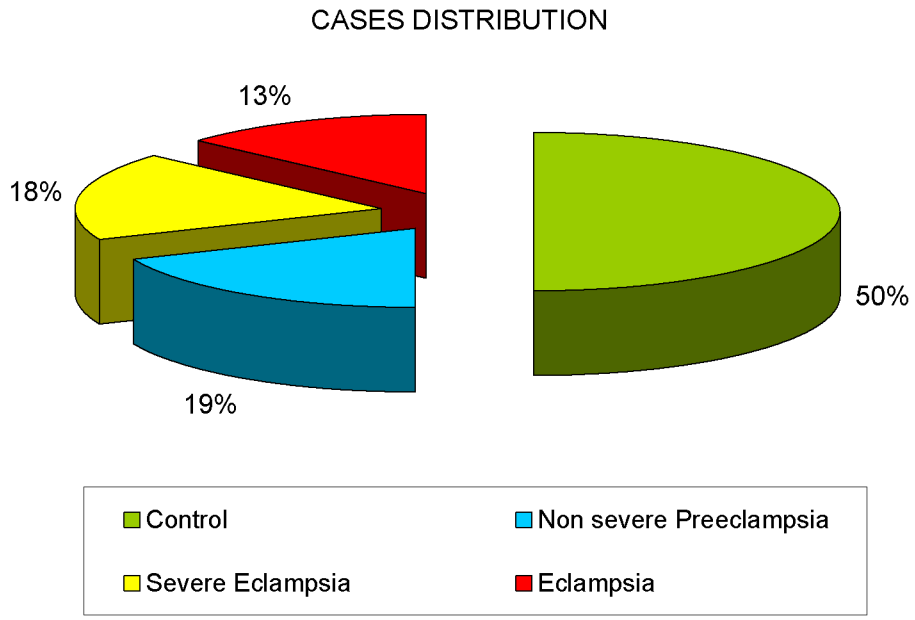
49 / 75 vs 26 / 75

p value is 0.042 Significant

In study of 150 patients, non severe preeclampsia, severe preeclampsia and eclampsia were higher in younger age groups ie. (21-25 years)

The maximum number of patients in control as well as study group belonged to age group of 21-30 years.

Eclampsia were present more in < 25 years age group it is significantly high. P value is 0.042 (significant)


CASES DISTRIBUTION
13%
18%
19%
50%
Control
Non severe Preeclampsia
Severe Eclampsia
Eclampsia

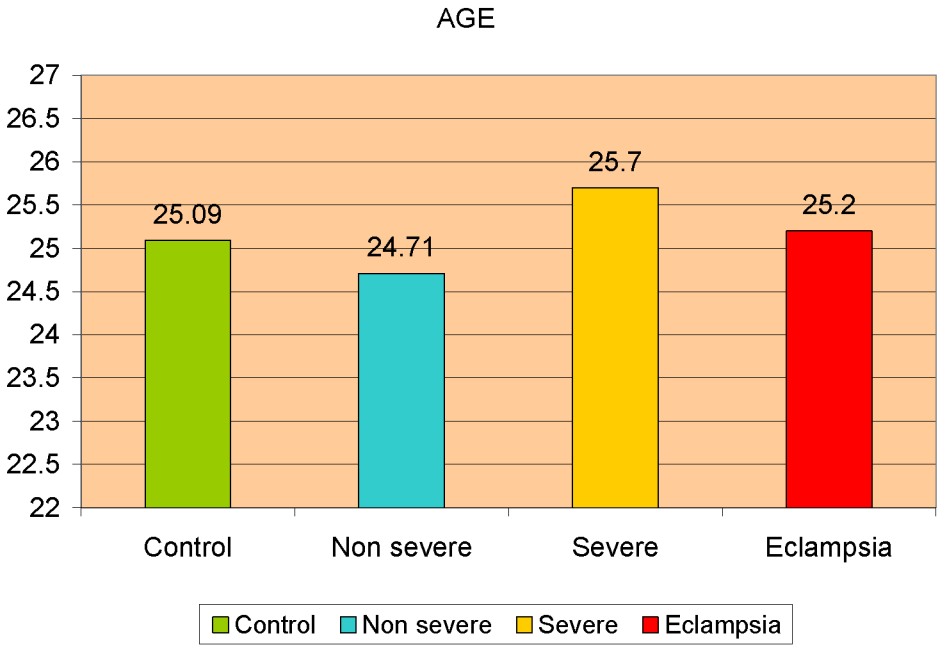

AGE
27
26.5
26
25.5
25
24.5
24
23.5
23
22.5
22
25.09
24.71
25.7
25.2
Control
Non severe
Severe
Eclampsia
Control
Non severe
Severe
Eclampsia

Table -2 Distribution of patients with **Parity**

Parity	Control (75)	Non severe (28)	Severe (27)	Eclampsia (20)
Primi	42	15	19	15
Multi	33	13	8	5
Total	75	28	27	20

Study of 150 patients, NSP, SP, eclampsia were significantly higher in patients with lower parity.

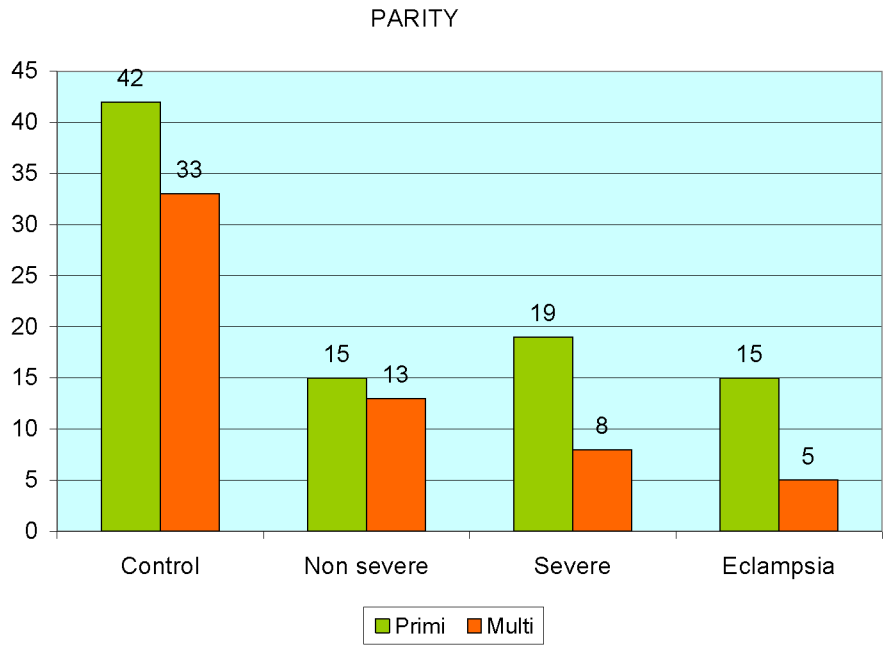


Table -3 Systolic BP

Systolic BP	Control	Non severe	Severe	Eclampsia
<150	75	25	3	2
≥ 150	0	3	24	18
Mean	0	150	155.4	187.8
SD	0	0	6.58	27.6
p value	< 0.001 Sig			

All 75 control had SBP <150 mmHg, 25 patients of NSP had SBP< 150, mean value150 , 3 patients of SP had SBP <150

24 patients of SP had SBP ≥ 150, 3 patients of NSP had SBP ≥150

18 patients of eclampsia is had SBP ≥ 150, only 2 patients of eclampsia had SBP < 150.

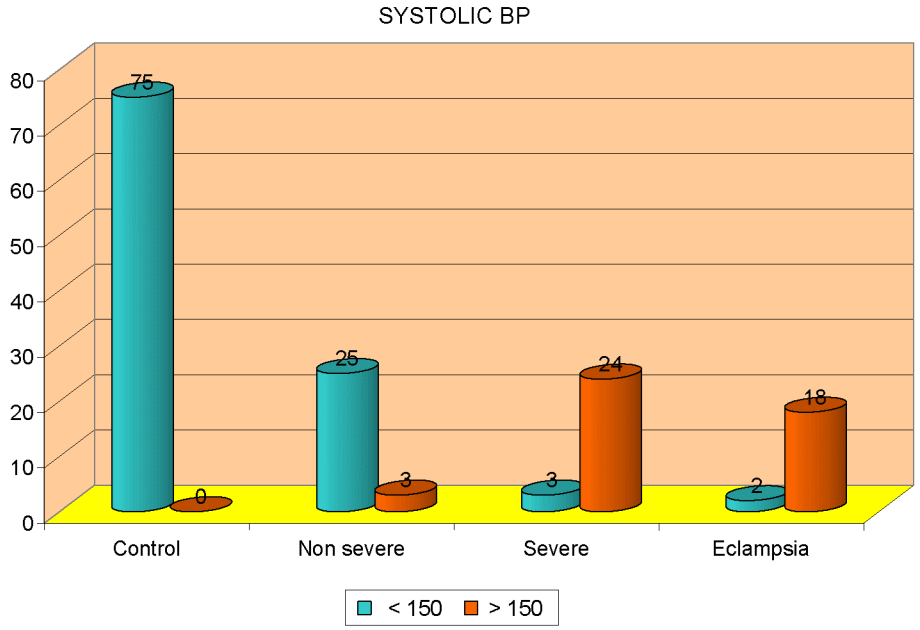
Mean value of SBP is significantly increased and higher in severe preeclampsia group and eclampsia group when compared to other groups. (p value is < 0.001 significant).

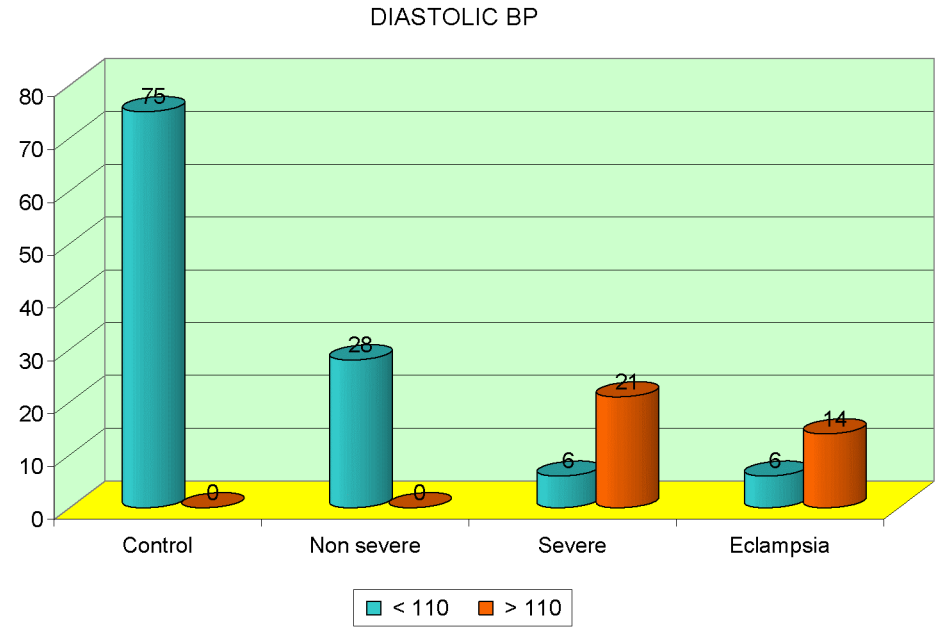
Table -4 Diastolic BP

Diastolic BP	Control	Non severe	Severe	Eclampsia
< 110	75	28	6	6
≥ 110	0	0	21	14
Mean			111.4	112.8
SD			3.58	4.69
p value	< 0.001 Sig			

All cases of control and non severe preeclampsia had diastolic BP less than 110 mmHg. 21 cases of severe preeclampsia and 14 cases of eclampsia had diastolic BP ≥110.

Mean value of DBP is significantly increased and higher in severe preeclampsia group and eclampsia group when compare to other groups. (p value is < 0.001 significant).


SYSTOLIC BP
80
70
60
50
40
30
20
10
0
75
0
25
3
3
24
2
18
Control
Non severe
Severe
Eclampsia
< 150
> 150


DIASTOLIC BP
80
70
60
50
40
30
20
10
0
75
0
28
0
6
21
6
14
Control
Non severe
Severe
Eclampsia
< 110
> 110

LDH LEVEL IN VARIOUS GROUPS

LDH	< 600 (112)	600 - 800 (17)	> 800 (21)
Control (75)	75	0	0
Non severe (28)	25	3	0
Severe (27)	11	12	4
Eclampsia (20)	1	2	17
Total	112	17	21

LDH	Control	Non severe	Severe	Eclampsia
Mean	190.24	392.7	630.44	919.2
SD	72.53	138.8	126.7	369.1
p value	< 0.001 Sig			

LDH	< 600 (112)	600 - 800 (17)	> 800 (21)
Non severe (28)	25 (89%)	3 (11%)	0
Severe (27)	11 (40.7%)	12 (44.4%)	4 (14.9%)
Eclampsia (20)	1 (5%)	2 (10%)	17 (85%)
Normal (75)	75 (100%)		

Control

In the control all 75 had LDH (<600 group)

Mean value of LDH among control is 190.24 $\pm$ 72.53

Non severe preeclampsia

Most of the patients in NSP (89%) had LDH level < 600

Mean LDH - 392.7 + 138.8

Only 3 patients (11%) of NSP had LDH level 600-800 group

Severe preeclampsia

Out of 27 cases of severe PC

12 cases (44.4%) had LDH 600 – 800 group

11 cases (40.7%) had LDH < 600 group

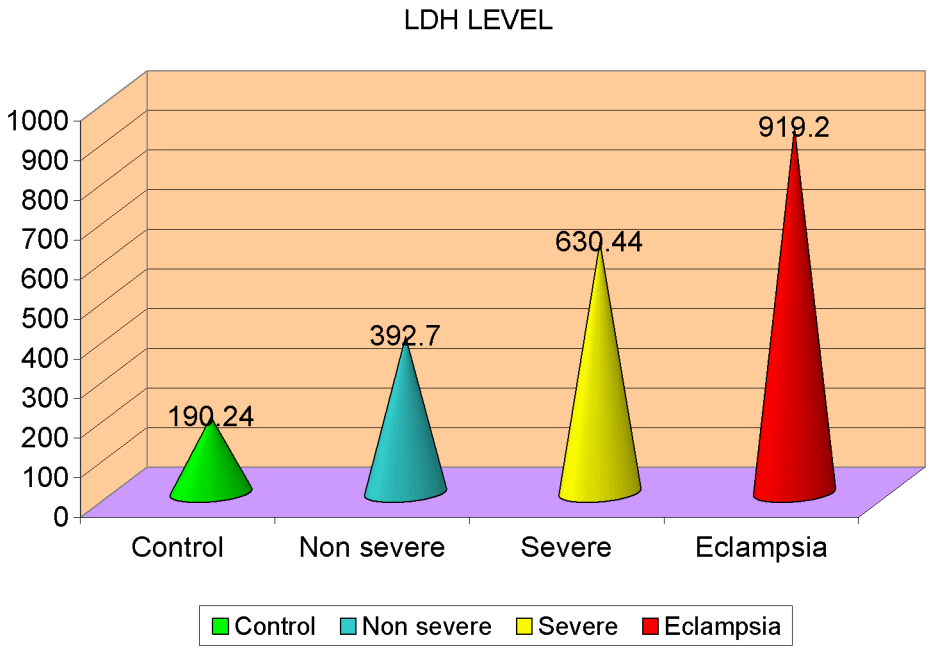
4 cases (14.9%) had LDH > 800 group

Eclampsia

Out of 20 cases of eclampsia

17 cases (85%) had LDH > 800

2 cases (10%) had LDH 600 – 800, 1case(5%) had LDH < 600



ASSOCIATION OF SYSTOLIC AND DIASTOLIC BP WITH LDH LEVELS IN VARIOUS GROUPS

Systolic BP	< 600 (112)	600 - 800 (17)	> 800 (21)
< 150 (105)	101	4	0
≥ 150 (45)	11	13	21

Out of 112 cases with LDH < 600 group

101 had SBP < 150 group and 11 had SBP ≥150 group

Out of 17 patients with LDH 600 – 800 group

4 had SBP < 150 group and 13 had SBP ≥ 150 group

Out of 21 patients with LDH > 800 group

21 (100%) had SBP ≥ 150 group.

Diastolic BP	< 600 (112)	600 - 800 (17)	> 800 (21)
< 110 (116)	106	4	6
≥ 110 (34)	6	13	15

Out of 112 cases with diastolic BP < 600

106 had diastolic BP < 110 and 6 had DBP ≥ 110

Out of 17 cases with DBP 600 – 800

4 had DBP < 110 and 13 had DBP ≥ 110

Out of 21 cases with DBP > 800

6 had DBP < 110 and 15 had DBP ≥ 110

On statistical analysis, it was found that high systolic and diastolic BP were associated with higher level of LDH (p value <0.001 significant)

MATERNAL COMPLICATIONS IN VARIOUS GROUPS:

	NON SEVERE	SEVERE	ECLAMPSIA
HELLP		4(14.8%)	3(15%)
Abruption		2(7.40%)	
DIC			2(10%)
MODS		2(7.40%)	2(10%)
CVA / PRES			6(30%)
Retinopathy	2(7.4%)		2(10%)
AKI			1(5%)
Mild PHT	1(3.57%)		

ASSOCIATION OF LDH AND OCCURRENCE OF MATERNAL COMPLICATIONS

	< 600	**600 - 800**	**> 800**
HELLP		2(11.76%)	5(23.8%)
Abruption / DIC		2(11.76%)	2(9.52%)
MODS		1(5.88%)	3(14.28%)
Macular edema		1(5.88%)	
CVA / PRES			6(28.5%)
Retinopathy	1(0.89%)		2(9.52%)
AKI			1(4.76%)
Mild PHT		1(5.88%)	

LDH> 800

Among 21 patients with LDH > 800 ,19 patients had complications. HELLP was found in 23.8% cases , PRES in about 28.5% cases , MODS in 14.28% cases, DIC and retinopathy contributing 9.52% each and AKI in 4.76 % cases.

LDH 600-800

Among 17 patients with LDH 600-800, 7 patients had complications. HELLP and abruption found in11.76% cases each, MODS , macular edema and mild PHT in 5.88% cases each.

LDH < 600

Among 112 cases with LDH< 600 , only one case had retinopathy contributing to 0.89%

ASSOCIATION BETWEEN LDH AND BIRTH WEIGHT

Birth weight / LDH	< 600 (120)	600 - 800 (10)	> 800 (20)
< 2.5 kg (60)	33 (27%)	8 (80%)	19 (95%)
≥ 2.5 kg (90)	87 (73%)	2 (20%)	1 (5%)

When LDH < 600 → 87 babies had birth weight ≥ 2.5 kgs

33 babies had birth weight < 2.5 kgs

LDH 600 – 800 → 8 babies had birth weight < 2.5 kgs

2 babies had birth weight ≥ 2.5 kgs

when LDH > 800 → 19 babies had birth weight < 2.5 kgs

1 baby had birth weight ≥ 2.5 kgs

ASSOCIATION OF LDH AND PERINATAL OUTCOME

	< 600 (112)	600 - 800 (17)	> 800 (21)
Healthy baby	107	13	4
Sick baby	5	3	12
SVD / IUD	0	1	5

Totally 21 babies were born to mother with LDH> 800 of which 5 babies (23.80%) were IUD, 12 babies(57.14%) expired and only 4 babies (19.04%) were healthy babies

LDH 600 -800 IU/L

Among 17 babies, 13 babies (76.47%) were healthy babies, 3 babies(17.64%) expired and 1 was IUD

LDH <600 IU/L

Among 112 babies , 107(95.53%)were healthy babies, 5 babies(4.46 %) expired and there were no IUD

CORRELATION OF OTHER PARAMETERS

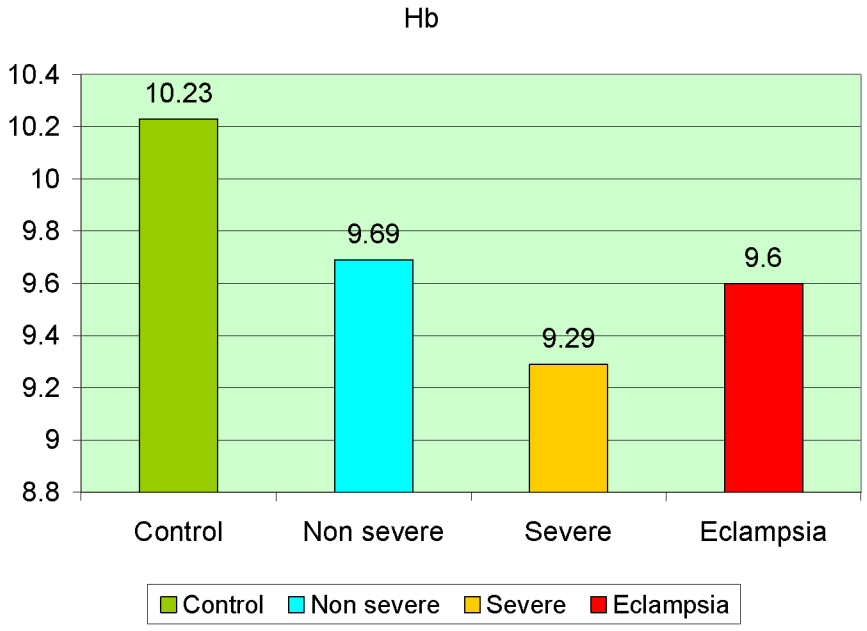
1) HEMOGLOBIN:

Hb	Control	%	Non severe	%	Severe	%	Eclampsia	%	Total	%
< 10	26	34.7	16	57.1	24	88.9	14	70.0	80	53.3
> 10	49	65.3	12	42.9	3	11.1	6	30.0	70	46.7
Total	75	100	28	100	27	100	20	100	150	100

Hb	Control	Non severe	Severe	Eclampsia
Mean	10.23	9.69	9.29	9.6
SD	0.62	0.65	0.65	0.66
p value	< 0.001 Sig			

Hb % was <10g/dl in 34.7% of control 57.1% NSP, 88.9% SP and 70% eclampsia , Hb > 10g/dl in 65.3% control, 42.9% NSP, 11.1% SP and 3% of eclampsia.

P value < 0.001 statistically significant.



2) PLATELET:

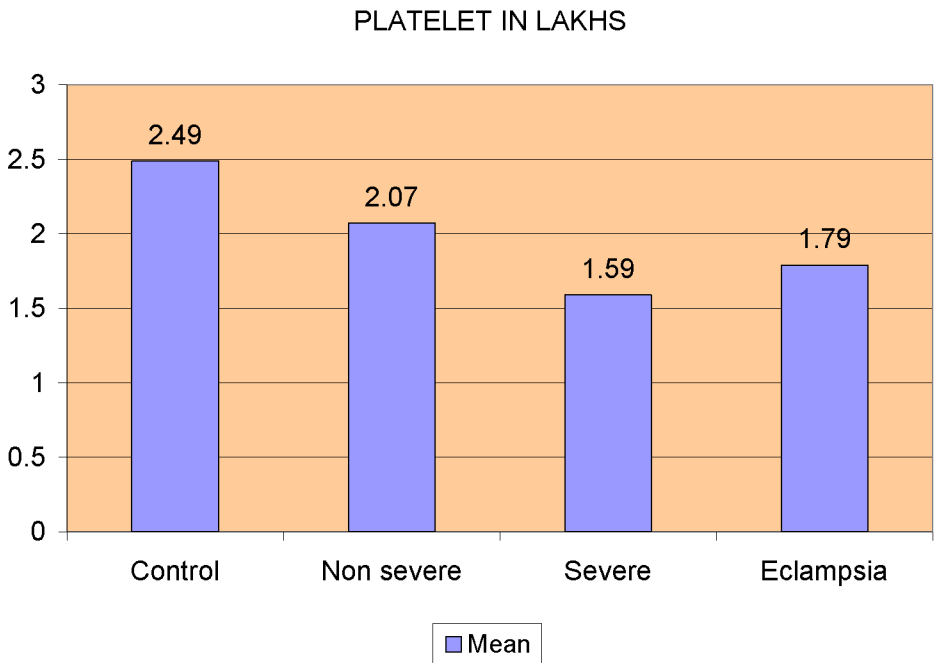
Platelet	Control	%	Non severe	%	Severe	%	Eclampsia	%	Total	%
< 1	0	0.0	0	0.0	11	40.7	11	55.0	22	14.7
> 1	75	100.0	28	100.0	16	59.3	9	45.0	128	85.3
Total	75	100	28	100	27	100	20	100	150	100

Platelet	Control	Non severe	Severe	Eclampsia
Mean	2.49	2.07	1.59	1.79
SD	0.86	0.54	0.88	1.32
p value	< 0.001 Sig			

Platelet < 1 L / mm3 is 40.7% severe preeclampsia and 55% of eclampsia

Platelet > 1 L / mm3 in all cases of control and non severe preeclampsia

P value <0.001 significant



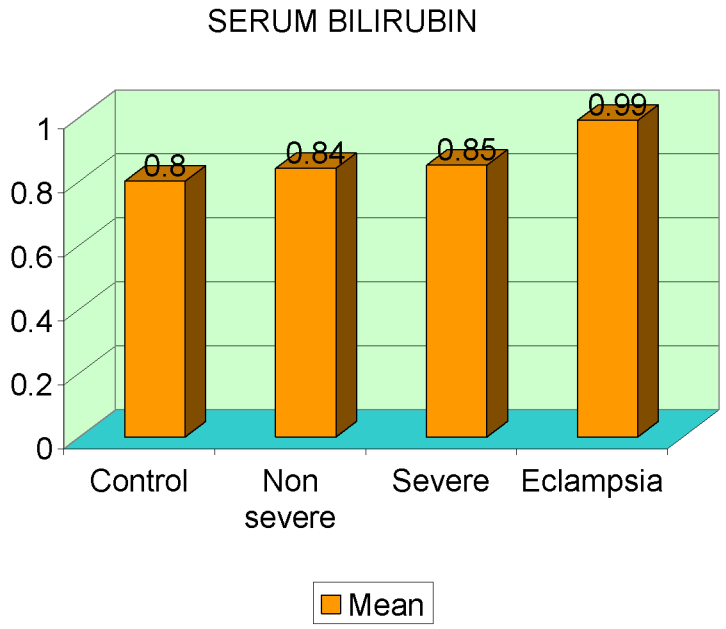
3)SERUM BILIRUBIN

SB	Control	%	Non severe	%	Severe	%	Eclampsia	%	Total	%
< 1	68	90.7	24	85.7	19	70.4	10	50.0	121	80.7
> 1	7	9.3	4	14.3	8	29.6	10	50.0	29	19.3
Total	75	100	28	100	27	100	20	100	150	100

Sr. Bilirubin	Control	Non severe	Severe	Eclampsia
Mean	0.8	0.84	0.85	0.99
SD	0.12	0.55	0.29	0.19
p value	0.071 NS			

Serum bilirubin > 1g/dl is 9.3% in control, 14.3% in NSP, 29.6% in SP and 50% in eclampsia.

P value is 0.071 Not significant.

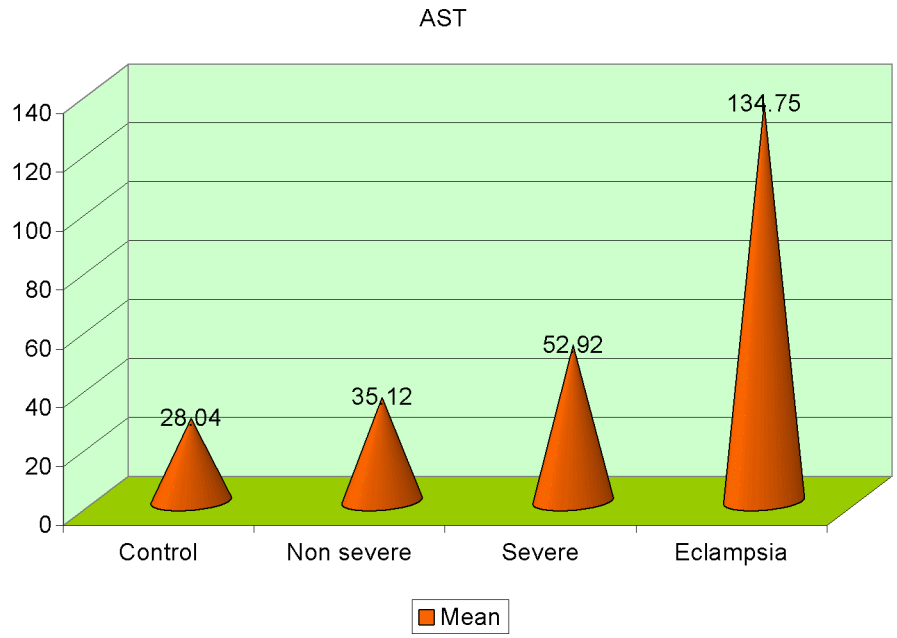


4) AST

AST	Control	%	Non severe	%	Severe	%	Eclampsia	%	Total	%
< 70	75	100.0	28	100.0	24	88.9	15	75.0	142	94.7
> 70	0	0.0	0	0.0	3	11.1	5	25.0	8	5.3
Total	75	100	28	100	27	100	20	100	150	100

AST	Control	Non severe	Severe	Eclampsia
Mean	28.04	35.12	52.92	134.75
SD	4.38	6.29	14.38	229.88
p value	< 0.001 Sig			

AST <70IU/L in all cases of control and non severe preeclampsia AST > 70 IU/L is 14.8% cases of severe preeclampsia and 35% cases of eclampsia. P value is < 0.001 significant.



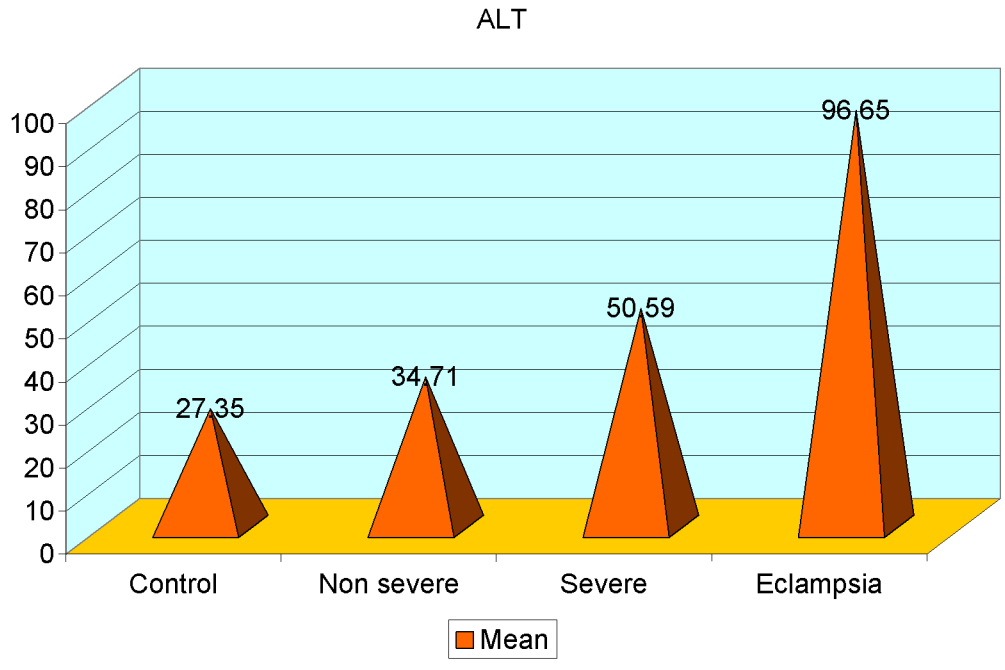
5) ALT

ALT	Control	%	Non severe	%	Severe	%	Eclampsia	%	Total	%
< 70	75	100.0	28	100.0	26	96.3	15	75.0	144	96.0
> 70	0	0.0		0.0	1	3.7	5	25.0	6	4.0
Total	75	100	28	100	27	100	20	100	150	100

ALT	Control	Non severe	Severe	Eclampsia
Mean	27.35	34.71	50.59	96.65
SD	4.77	5.54	13.66	104.4
p value	< 0.001 Sig			

ALT > 70 IU/L is 11.11% of cases of SP and 30% of cases of eclampsia

P value is < 0.001 significant.



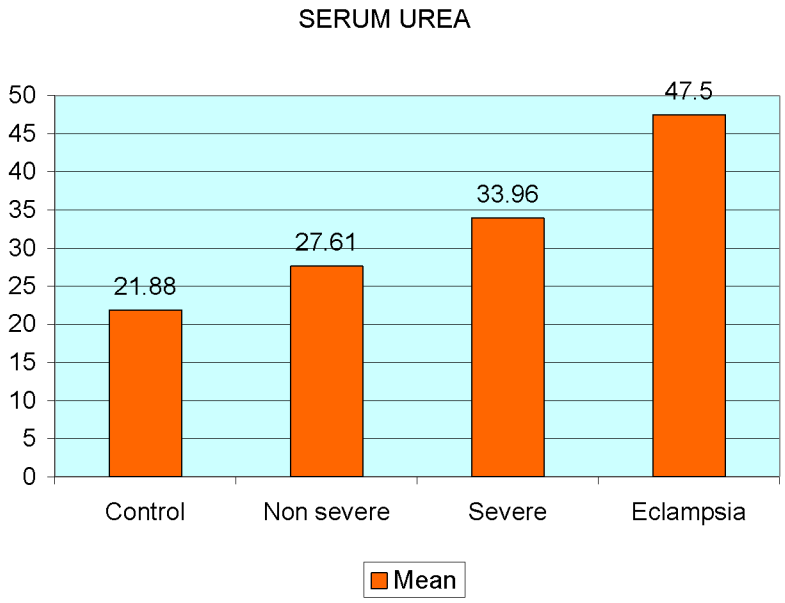
6)SERUM UREA

S Urea	Control	%	Non severe	%	Severe	%	Eclampsia	%	Total	%
< 50	75	100.0	28	100.0	24	88.9	11	55.0	138	92.0
> 50	0	0.0	0	0.0	3	11.1	9	45.0	12	8.0
Total	75	100	28	100	27	100	20	100	150	100

Sr. Urea	Control	Non severe	Severe	Eclampsia
Mean	21.88	27.61	33.96	47.5
SD	4.49	5.04	8.72	10.09
p value	0.002 Sig			

S. Urea >50 mg/dl in 11.1% severe preeclampsia and 45% of eclampsia.

S.Urea < 50 mg/dl in all cases of control and NSP

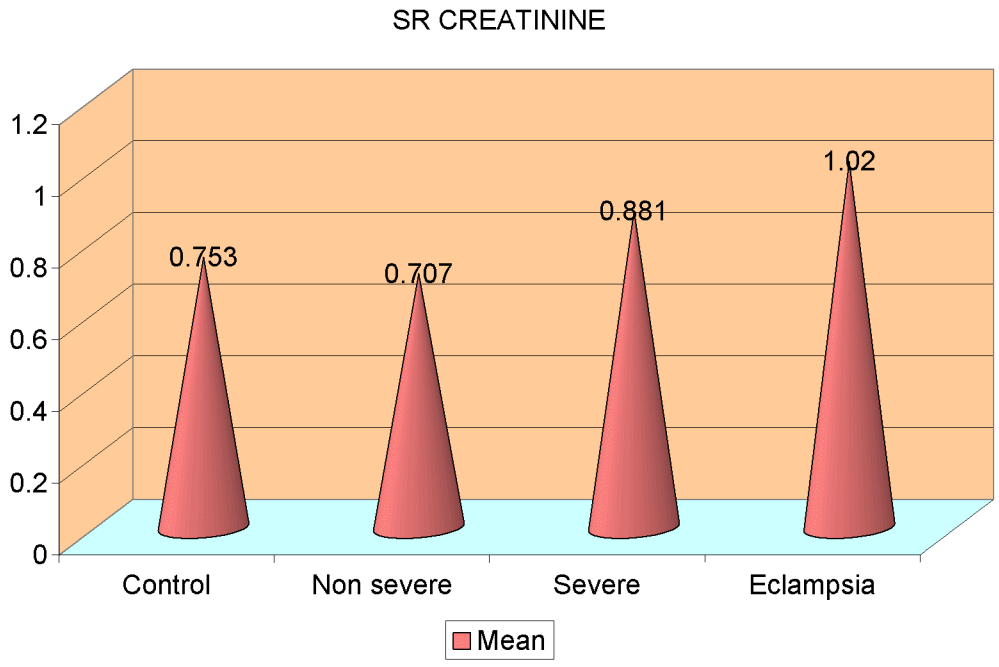


7) SERUM CREATININE

S Creatinine	Control	%	Non severe	%	Severe	%	Eclampsia	%	Total	%
< 1.5	75	100.0	28	100.0	25	92.6	17	85.0	145	96.7
> 1.5	0	0.0	0	0.0	2	7.4	3	15.0	5	3.3
Total	75	100	28	100	27	100	20	100	150	100

Sr. Creatinine	Control	Non severe	Severe	Eclampsia
Mean	0.753	0.707	0.881	1.02
SD	0.18	0.27	0.29	0.33
p value	< 0.001 Sig			

Sr.Creatinine <1.5 mg/dl in all cases of nonsevere preeclampsia Sr. Creatinine > 1.5 mg/dl is 7.4% cases of SP 15% of cases of eclampsia. P value is < 0.001 significant.

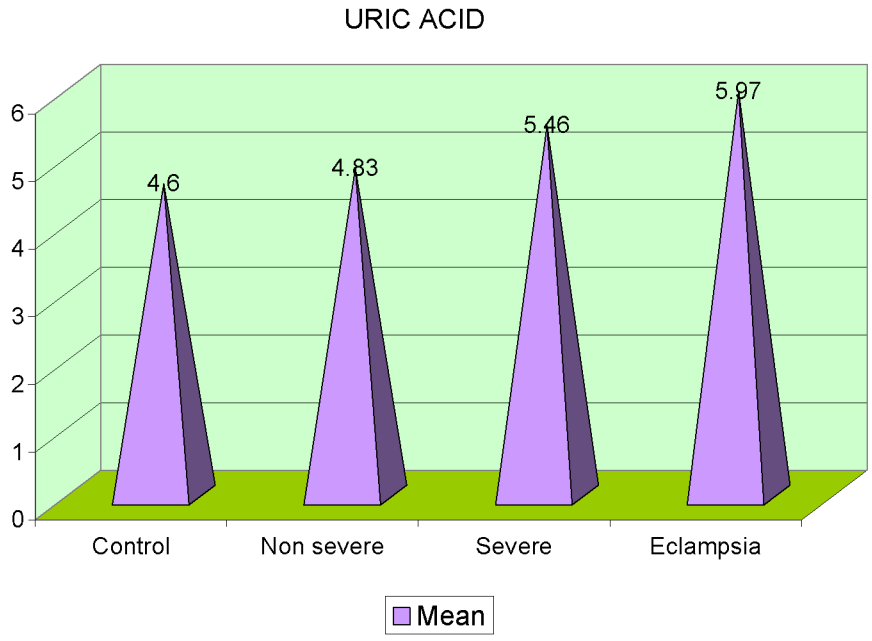


8)S.URIC ACID

Uric Acid	Control	%	Non severe	%	Severe	%	Eclampsia	%	Total	%
< 6	75	100.0	24	85.7	20	74.1	10	50.0	129	86.0
> 6	0	0.0	4	14.3	7	25.9	10	50.0	21	14.0
Total	75	100	28	100	27	100	20	100	150	100

Uric Acid	Control	Non severe	Severe	Eclampsia
Mean	4.6	4.83	5.46	5.97
SD	0.36	0.75	0.73	0.83
p value	< 0.001 Sig			

Uric acid > 6mg/dl is 14.3% in NSP, 25.9% in SP and 50% n eclampsia. P value is <0.001



DISCUSSION

It is well understood that hypertensive disorders of pregnancy are most commonly in association with high risk factors and they are associated with changes in certain hematological parameters.

AGE :

In our study, it was seen that 67.85% cases of non severe preeclampsia , 62.96% cases of severe preeclampsia and 65% cases of eclampsia were < 25 years of age. Queblan et al also reported similar data in their study.

PARITY:

53.5% cases of non severe preeclampsia and 75% eclampsia cases were primiparous. Ali et al, Demir et al and Queblan et al also reported similar data in their study since these are considered as risk factors for preeclampsia..

BIRTH WEIGHT:

In our study it was found that 39.28% cases of NSP, 62.96% cases of SP and 95% cases of eclampsia had birth weight < 2.5kgs. In the same study conducted by Aali et al it was found that 34.1% of preeclampsia and 24.2% cases of eclampsia had low birth weight babies

LDH DISTRIBUTION AMONG PREECLAMPSIA AND ECLAMPSIA:

Main objective of our study was to estimate the levels of serum lactate dehydrogenase.

LDH> 600 IU/L seen in 11% of NSP, 59.3% SP, 95% of eclampsia cases. P value is <0.001. This was found to be statistically significant.

Demir et al had found that in complicated cases of PE and eclampsia, LDH level was significantly higher. Quablan et al in his study reported that LDH is a bio chemical marker predicting adverse pregnancy outcome in severe PE cases. Also in his study it was found that LDH > 600 IU/L was seen in 54.8% cases of severe PE and 12.2% of non severe PE.

In our study cases were divided into three groups on the basis of LDH level.

Group I (LDH < 600 IU/L)

Group II (LDH 600 – 800 IU/L)

Group III (LDH > 800 IU/L)

- 66.96% of control ,22.31% of NSP , 9.82% of SP and 0.89% eclampsia had LDH < 600IU/L

- 70.51% of SP, 17.64% NSP and 11.46% eclampsia had LDH 600-800 IU/L
- 80.95% cases of eclampsia and 19.04% cases of SP had LDH >800 IU/L

These data were statistically significant.

It was found that in all normal or cases taken as control had LDH < 600

Group II with LDH 600-800 had PE and Eclampsia cases and no normal cases.

Group III LDH > 800 had majority of eclampsia cases.

Thus it clearly seen that there is significant rise in LDH levels with increasing severity of disease.

A recent study conducted by Jaiswae et al also reported similar findings.

LDH AND PERINATAL OUTCOME:

If we look at Perinatal outcome according to LDH level **Group I (<600 IU/L)** had 95.53% of healthy baby 4.46% expired after admission into NICU.

27% babies had birth weight < 2.5kg and 73% had birth weight > 2.5kg.

Group II (LDH 600 – 800 IU/L) had 76.47% of healthy babies, 17.64% babies expired, 5.88% of babies were IUD.

80% babies had birth weight < 2.5 kg, 20 % babies had birth weight > 2.5 kg

Group III (>800 IU/L), had 19.04% of healthy babies, 57.14% of babies expired and 23.8% of babies were IUD. Finding was statistically significant.

95% had birth weight < 2.5 kg and 5% had birth weight >2.5kg

Qublan et al in his study also found that 61.5% of perinatal death were found in cases having LDH level > 800 IU/L.

MATERNAL COMPLICATIONS:

It is also important, to analyze the complications as prediction would help in averting them, thereby preventing a lot of associated morbidity and mortality.

HELLP syndrome found in 14.8% cases of severe preeclampsia and 15% cases of eclampsia. Abruption 7.4% cases of severe PE. DIC was found in 10% cases of eclampsia. MODS in 7.4% of SP, 10% of eclampsia. PRES → 30% eclampsia . Retinopathy → 7.14% of NSP and 10% of eclampsia. AKI → 5% of eclampsia and mild PHT → 3.57% cases of NSP.

Ali et al study concluded that 5.4% PE and 24.25% of eclampsia cases had ARF. DIC found in 3% PE and 18.2% eclampsia.

3% of preeclampsia and 27.3% eclampsia had classic HELLP syndrome. 6% of preeclampsia 6% eclampsia had partial HELLP .

4.8% PE and 21.2% eclampsia had ARDS . 9.6% PE 18.2% of eclampsia had abruption.

LDH AND MATERNAL COMPLICATIONS:

On analyzing the complications further according to LDH level

Group 1(LDH< 600) had Retinopathy (0.89%)

Group II (LDH 600-800)

Abruption with DIC present in 11.76% patients, HELLP Syndrome 11.76% of patients, MODS in 5.88% cases, Macular edema in 5.88% cases and mild PHT in 5.88 % cases.

GROUP III (LDH > 800)

HELLP seen in 23.80 % cases, PRES in 28.57%, MODS in 14.28% cases, Retinopathy in 9.52% cases , AKI in 4.76% case, DIC in 9.52 % cases

Quablan et al (16) in his study found eclampsia was a complication in 4.7% of GROUP II patients and 30.8% of GROUP III. Abruption placenta seen in 15.4 . GROUP III patients.

Intracranial hemorrhage seen in 7.7 % GROUP III. HELLP syndrome seen in 15.4% of GROUP III. Acute renal failure seen in 7.7 % of GROUP III . Pulmonary edema seen in 7.7 % cases of GROUP III.

OTHER LAB PARAMETERS:

On further analyzing my study taking into account hematological test Hb% was less than 10 g/dl in 57.1% cases of NSP, 88.9% cases of severe PE and 70% of eclampsia. P value<0.001 statistically significant.

Platelet count was < 1 lakh / cumm in 40.7% of SP, 55% of eclampsia. P value < 0.001 statistically significant

Total bilirubin > 1 mg/dl in 14.3% NSP, 29.6% SP, 50% eclampsia patient. P value 0.071 not significant.

Liu et al, in a study proposed that 20.6% patients with SP had liver function tests which were abnormal. In our study AST was > 70 IU/L in 14.8% of severe preeclampsia and 35% of eclampsia patients P value < 0.001 significant

ALT was > 70 IU/L in 11.1% of severe Preeclampsia and 30% of eclampsia.p value < 0.001 significant

If we look at RFT, Sr.urea > 50mg/dl in 11.1% of severe preeclampsia and 45% cases of eclampsia. P value 0.002 statistically significant

S.creatinine > 1.5mg/dl in 7.4% cases of SP and 1.5% cases of eclampsia P value is < 0.001 which was significant

However still researches are needed in this field .LDH –A (4) isoenzyme activity measurement would be more specific in cases of preeclampsia. Measurement of liver function test in addition would predict cases better. Kozic et al in his study proved that adverse maternal outcomes are common in cases with abnormal LDH, AST, ALT , Total bilirubin, and INR resuts and hence LFT and LDH should be studied in all cases of preeclampsia and eclampsia.

SUMMARY

This study – prospective case control study correlating the severity of disease of the disease, maternal and perinatal outcome with serum LDH and other biochemical parameters in patients with preeclampsia.

- NSP, SP and eclampsia were higher in younger age groups 21-25 yrs and lower parity.
- 66.96% of control ,22.31% of NSP , 9.82% of SP and 0.89% eclampsia had LDH < 600IU/L
- 70.51% of SP, 17.64% NSP and 11.46% eclampsia had LDH 600-800 IU/L
- 80.95% cases of eclampsia and 19.04% cases of SP had LDH >800 IU/L
- Maternal complications were more in LDH> 800IU/L

Among the complications, HELLP in 23.80 % cases, PRES in 28.57%, MODS in 14.28% cases, Retinopathy in 9.52% cases , AKI in 4.76% case, DIC in 9.52 % cases were present when LDH> 800IU/L

Abruption with DIC present in 11.76% patients, HELLP Syndrome 11.76% of patients, MODS in 5.88% cases, Macular

edema in 5.88% cases and mild PHT in 5.88 % cases were present when LDH 600-800IU/L.

Retinopathy contributing to 0.89% present when LDH <600IU/L

- Totally 21 babies were born to mother with LDH >800 of which 5 babies (23.80%) were IUD, 12 babies (57.14%) expired and only 4 babies (19.04%) were healthy babies.

  Among 17 babies born to mother with LDH 600-800 IU/L ,13 babies (76.47%) were healthy babies , 3 babies (17.64%) expired and 1 was IUD

  Among 112 babies born to mothers with LDH < 600IU/L ,107(95.53%) were healthy babies, 5 babies(4.46%) expired and there were no IUD

- . **Group I (<600 IU/L)** 27% babies had birth weight < 2.5kg and 73% had birth weight > 2.5kg.

  Group II (LDH 600 – 800 IU/L)

  80% babies had birth weight < 2.5 kg, 20 % babies had birth weight > 2.5 kg

Group III (>800 IU/L)95% had birth weight < 2.5 kg and 5% had birth weight >2.5kg

Also there is statistically significant association between other parameters like Hb, platelet, AST, ALT, S.Urea, S.Creatinine, S.Uric acid in assessing the severity of preeclampsia except S.bilirubin which is statistically insignificant.

CONCLUSION

Thus serum LDH is the earliest marker in blood in conditions associated with hypoxia & oxidative stress. Thereby it is raised in Preeclampsia & Eclampsia. It predicts the severity & occurrence of complications in Preeclampsia & Eclampsia. The complications can be prevented if it is measured earlier, if adequately managed at a higher center.

Detection of high risk pregnancies with increased level of LDH requires close monitoring in antenatal period & proper management is necessary. This will help to a greater reduction in maternal and fetal morbidity and mortality. Hence we conclude that screening with LDH is essential for all cases of Preeclampsia and Eclampsia for early detection and management of complications.

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CASE PROFORMA

NAME : AGE : IP NO: UNIT :

PARITY : WEIGHT : HEIGHT :

LMP : EDD :

TYPE OF ADMISSION : EMERGENCY OR BOOKED

EDUCATION :

ANTENATAL VISIT :

RESIDENCE :

SOCIO ECONOMIC STATUS :

COMPLAINTS:

REFERRED FROM :

PAST HISTORY :

MENSTRUAL HISTORY :

MARITAL HISTORY :

OBSTETRIC HISTORY :

RECURRENT GHT OR NOT

EXAMINATION :

P/A:
P/V :
USG :
TREATMENT :

INVESTIGATION:

HB :

URINE ALBUMIN SUGAR :

PLATELETS :

HEMATOCRIT :

S.URIC ACID :

RBS :

BLOOD UREA :

S.CREATININE :

LDH :

TOTAL BILIRUBIN :

SGOT :

SGPT :

ALP :

FUNDUS :

DIAGNOSIS :

COMPLICATIONS :

MODE OF DELIVERY :

INDICATIONS:

BABY DETAILS :

PRE TERM OR TERM

ALIVE OR DEATH

BIRTH WEIGHT :

APGAR :

NEONATAL PERIOD :

OTHERS:

LIST OF ABBREVIATIONS

NSP	Non severe preeclampsia
SP	Severe preeclampsia
GHT	Gestational Hypertension
HELLP	Hemolysis elevated liver enzymes and low platelet count
IUFD	Intrauterine fetal death
IUGR	Intra uterine growth restriction
LDH	Lactate dehydrogenase
LMP	Last menstrual period
LSCS	Lower Segment Cesarean Section
MAS	Meconium Aspiration syndrome
NHBPEP	National High Blood pressure education programme
NPV	Negative predictive value
NICU	Neonatal intensive care unit
P	Para
PPV	Positive predictive value
PROM	Premature rupture of membranes
US	Ultra sound
VEGF	Vascular endothelial growth factor

MASTER CHART

S. No		Name	IP No.	Age	Parity	GA	B / UB	SBP	DBP	Mode of delivery	Birth weight	Apgar 1'	Apgar 5'	Hb	Platelet	SB	OT	PT	LDH	Urea	Creatinin
1	NSP	Nivedha	55	20	Primi	36	B	140	90	LSCS	1.25	5 / 10	6 / 10	9.6	1.42	0.9	36	38	360	32	0.9
2	NSP	Mahalakshmi	4241	30	Primi	38	B	150	90	LN	2.1	5 / 10	6 /10	9.8	1.3	1	36	32	482	40	1
3	NSP	Karpagavalli	4000	21	Primi	36	B	150	100	LN	1.1	4 /10	5 /10	8.2	1.1	0.8	48	36	712	22	0.6
4	NSP	Kanimozhi	1109159	24	Primi	38	B	140	100	LN	2.4	5 /10	6 /10	8.2	2.1	0.8	32	38	302	21	0.7
5	NSP	poongodi	1109169	23	G3P1L1	39	B	140	100	OUTLET	2.25	5 /10	6 /10	8.4	1.8	0.8	36	38	208	23	0.7
6	NSP	sudha	1109171	25	Primi	38	B	140	90	LSCS	2.7	7 /10	8 /10	9.6	2	0.6	31	30	516	22	0.3
7	NSP	ponmani	1111370	23	Primi	37	B	140	100	LSCS	2.8	5 /10	6 /10	9.6	2.2	0.7	46	42	363	34	0.8
8	NSP	lakshmi	5340	30	Primi	36	B	140	90	LN	2.8	5 /10	6 /10	10.2	2.3	0.9	29	27	318	25	0.5
9	NSP	swarnalakshmi	1114343	20	Primi	38	B	140	90	LN	3.2	7 /10	8 /10	9.8	2.4	0.8	30	32	418	31	0.6
10	NSP	ajeetha	5946	25	G2P1L1	38	B	140	90	LSCS	2.8	7 /10	8 /10	10.4	2.6	0.7	32	31	212	25	0.9
11	NSP	pancha	7313	22	G3P2L2	40	B	140	100	LN	3.2	7 /10	8 /10	10.2	2.8	0.6	34	36	486	26	0.7
12	NSP	rajeswari	7123	29	G3P1L1A1	38	B	130	90	LN	3.2	7 /10	8 /10	10	3.2	0.8	34	31	382	34	0.8
13	NSP	sakila baanu	6733	32	G2P1L1	36	B	140	90	LN	2.6	7 /10	8 /10	9.6	2.4	0.8	37	36	442	31	0.6
14	NSP	muthupandi	7742	30	G3P2L2	38	B	140	90	LSCS	3.2	7 /10	8 /10	9.4	1.6	0.5	36	34	383	29	0.9
15	NSP	nagajothi	8138	25	G2P1L1	38	B	130	100	LN	2.7	7 /10	8 /10	10.2	1.7	0.6	38	31	362	28	0.8
16	NSP	panchavarnam	1120743	19	Primi	36	B	140	90	LSCS	3.1	7 /10	8 /10	11	1.8	0.9	37	35	312	29	0.9
17	NSP	valavanthal	7371	22	Primi	38	B	130	90	LN	2.8	7 /10	8 /10	9	1.9	0.8	14	15	318	28	0.9
18	NSP	kavitha	1127650	28	G2P1L1	36	B	130	90	LN	2.9	5 /10	6/10	10.1	1.8	0.8	40	38	302	20	0.1
19	NSP	vasanthy	4248	24	Primi	30	B	150	90	LN	2.25	6 /10	8 /10	9.6	1.8	0.9	40	39	318	31	1
20	NSP	shanthi	48881	21	Primi	37	B	130	100	LN	2.5	5 /10	6 /10	9.4	1.8	0.8	38	40	521	24	0.8
21	NSP	kavitha	48846	32	G2P1L1	37	B	130	90	LSCS	2.8	6 /10	8 /10	9.8	2.9	0.3	42	41	346	26	0.2
22	NSP	bhuvaneswari	8297	29	G3P1L1A1	35	B	130	90	LN	2.4	5 /10	6 /10	9.2	2.6	0.2	36	38	382	23	0.9

23	NSP	ramajeyanthi	9856	20	Primi	31	B	130	90	LSCS	2.4	5 10	6/10	9.4	2.6	0.4	40	41	692	34	0.3
24	NSP	sakthiirulayee	10508	23	G2P1LO	35	B	140	100	LSCS	1.5	5/10	6/10	10.1	1.1	1	38	39	683	32	1.1
25	NSP	manimegalai	9196	20	Primi	40	B	130	90	LN	1.9	6 /10	8 /10	10.2	1.6	0.6	32	39	226	28	0.8
26	NSP	vijaya	10436	25	G3P1L1A1	38	B	130	90	LSCS	3.2	6 /10	8 /10	10.2	2.6	0.3	30	32	404	32	0.6
27	NSP	murugeswari	10905	28	G2P1L1	40	B	130	90	LN	2.25	6 /10	8 /10	10.1	2.3	2.3	31	32	384	21	0.5
28	NSP	manimegalai	8720	19	Primi	35	B	140	90	LN	2	6 /10	8 /10	10.1	2.1	2.9	30	31	162	22	0.5
29	SP	durga	3679	25	Primi	34	B	150	110	LSCS	1.5	4 /10	6 /10	9.8	1.2	0.9	46	46	723	40	1
30	SP	dhivya	48247	23	Primi	32	B	140	110	LSCS	1.5	6 /10	6 /10	9.6	1.3	0.8	31	30	628	42	0.9
31	SP	saliya	1108453	26	G2P1L1	36	B	150	100	LN	2.3	4 /10	6 /10	9.8	1.3	0.6	51	45	512	38	0.8
32	SP	poongodi	1109164	23	G2P1L1	35	B	150	110	LSCS	2.1	5 /10	7 /10	9.6	2.1	0.9	54	61	638	30	0.5
33	SP	subalakshmi	1109170	32	Primi	30	B	160	120	LSCS	1.5	3 /10	5 /10	9.6	0.98	1.1	60	58	658	40	1
34	SP	gunasundari	4597	25	Primi	36	B	150	110	LN	2.75	5 /10	7 /10	9.8	3.2	0.6	70	64	456	30	0.6
35	SP	sathya	1109937	20	Primi	32		170	120	LSCS	1.6	4 /10	5 /10	9.4	0.86	1.4	92	90	898	56	1.5
36	SP	aanandhi	4951	30	G3P2L2	30	B	160	110	LSCS	2.1	4 /10	5 /10	10.2	1.01	0.8	51	50	612	34	0.7
37	SP	priyadharshini	5696	20	Primi	36	B	150	110	LSCS	2.8	3 /10	5 /10	8.6	0.96	0.9	42	44	656	30	0.8
38	SP	parameswari	1114329	23	G3P1L1A1	36	B	150	110	LN	2.1	4 /10	6 /10	8.8	3.56	1.1	32	36	486	20	0.7
39	SP	malathy	1117864	25	Primi	30	B	170	110	LN	2.7	4 /10	6 /10	10.2	0.88	1.4	54	37	702	36	1.4
40	SP	rajeswari	7123	29	G3P1L1A1	38	B	150	110	LN	2.9			8.8	3.16	0.5	56	31	612	30	0.7
41	SP	brindha	7614	25	Primi	38	B	140	110	LN	3.3	5 /10	6 /10	9.8	1.56	0.6	67	52	588	31	0.8
42	SP	latha	7814	26	G2P1L1	30	B	160	120	LSCS	1	5 /10	7 /10	9.2	3.56	0.8	80	70	780	32	0.7

43	SP	shanthi	48881	21	Primi	36	B	150	110	LSCS	2.5	5 /10	7 /10	8.8	2.8	0.7	31	30	556	31	0.9
44	SP	tamilmalar	9781	30	Primi	30	B	150	110	LSCS	1.2	5 /10	6 /10	8.8	1.1	0.8	42	51	801	30	0.8
45	SP	nandhinidevi	10251	28	Primi	39	B	150	100	LSCS	2.8	5 /10	6 /10	8.8	1.2	0.4	43	45	516	31	0.9
46	SP	muthurani	1129286	21	Primi	42	B	150	100	LSCS	3.2	5 /10	6 /10	8.6	1.1	0.5	44	57	586	30	0.8
47	SP	bharathi	1127386	32	Primi	38	B	160	100	LSCS	1.8	5 /10	9 /10	9.2	1.3	0.6	49	52	676	34	0.8
48	SP	priyadharshini	11065	23	G2P1L1	38	B	160	110	LSCS	3	3 /10	5 /10	9.4	0.96	1.3	38	39	714	51	1.1
49	SP	palaniammal	1130111	23	Primi	32	B	150	110	LSCS	1.3	4 /10	5 /10	8.8	2.1	0.7	51	40	681	36	0.8
50	SP	shabeeba baanu	112926	23	Primi	36	B	140	100	LSCS	1.75	3 /10	5 /10	8.2	1.2	0.8	56	52	475	24	0.8
51	SP	vanitha	112361	35	Primi	32	B	160	100	LN	1.4	5 /10	6 /10	11.2	1.3	0.7	61	51	498	25	0.6
52	SP	murugeswari	1127010	43	G5P4L4	30	B	160	110	LN	1.2	5 /10	7 /10	8.6	0.84	1.6	72	71	836	54	1.9
53	SP	panchammal	9233	19	Primi	34	B	160	100	LSCS	2.1	6 /10	8 10	9.4	1.1	0.8	54	52	512	26	0.7
54	SP	jeyanthi	11051	23	Primi	34	B	150	110	LN	2.3	5 10	7 10	9	1.2	0.8	52	51	410	27	0.8
55	SP	saranya	1127594	21	Primi	30	B	160	110	LN	0.9			8.8	0.96	0.9	50	61	812	29	0.8
56	E	mareeswari	1043755	26	Primi	34	B	150	100	LSCS	1.25	4 10	6 10	8.2	0.8	1.1	1070	457	2414	29	0.6
57	E	vidya	7383	31	G3A2	31	B	150	100	LSCS	1.15			8.9	1.1	1.2	227	144	821	32	0.8
58	E	abinaya	8750	24	Primi	30	B	160	100	LN	1			9.2	1.2	1.4	88	81	806	52	0.6
59	E	kavitha	10009	23	Primi	37	B	160	100	OUTLET	1.2	2/10	4/10	9.4	0.86	1.4	56	58	928	52	1.2
60	E	tamilmalar	9789	30	Primi	30	B	220	110	LSCS	1.1	2/10	4/10	10.2	1.2	1	287	315	1021	48	0.8
61	E	amsavalli	10867	26	Primi	33	B	180	110	LSCS	1.2			9.6	1.2	1	178	108	901	61	0.9

62	E	muthumari	10468	22	G2P1	30	B	190	120	LSCS	1.4	5 10	6 10	9.8	2.1	0.9	48	46	856	40	0.9
63	E	dhanalakshmi	10301	27	G4P3L3	38	B	150	120	LN	2.8	5/10	7/10	10.1	3.8	0.8	48	48	612	38	0.6
64	E	nagalakshmi	1124408	25	G2P1L1	38	B	140	110	LSCS	2.25	5/10	7/10	9.8	2.7	0.8	50	51	682	42	0.6
65	E	meena	1125583	23	G3P2L2	32	B	190	110	LN	1.1	5/10	7/10	9.6	0.84	0.8	70	66	831	40	1.5
66	E	maarilakshmi	10403	30	Primi	32	B	180	120	LSCS	1	5/10	6/10	9.4	2.1	0.9	48	46	852	44	1.4
67	E	murugeswari	4529	25	Primi	28	B	190	120	LSCS	1.1	5/10	6/10	9.8	0.81	0.9	38	36	832	54	1
68	E	nageswari	1086821	29	Primi	30	B	200	100	LSCS	1.25	4/10	6/10	9.6	0.72	1.1	48	46	901	62	1.6
69	E	raakammal	29269	22	Primi	30	B	200	110	LSCS	0.75			10.2	2	1.2	58	56	902	41	1.2
70	E	abirami	2192	21	Primi	32	B	210	110	LSCS	2	4/10	6/10	8.8	0.92	1.1	61	60	852	62	1.2
71	E	jeyalakshmi	109538	25	Primi	30	B	220	110	LSCS	1.7	4/10	6/10	9.2	2.1	0.9	71	70	902	61	1.1
72	E	kavitha	1124409	23	Primi	38	B	140	110	OUTLET	2	5/10	7/10	10.2	2.1	0.8	52	61	561	58	1
73	E	dhanapriya	11485	24	Primi	32	B	180	110	LN	1	4/10	6/10	8.6	0.6	1	68	60	840	51	1.6
74	E	sakthi	11528	24	Primi	32	B	250	110	LSCS	1.1	4/10	6/10	10.8	1.8	0.9	61	60	882	42	1
75	E	vidya	25874	24	Primi	30	B	200	100	LSCS	1	4/10	6/10	10.6	1.8	0.7	68	64	988	41	0.8
76	C	kandiamma	8808	28	Primi	38	B	110	80	LSCS	2.4	6/ 10	8 /10	10.2	1.2	1	42	41	112	23	0.8
77	C	malliga	8886	24	G2P1L1	39	B	110	80	LN	3.1	6 /10	8 /10	10	1.3	0.9	46	45	118	22	0.8
78	C	arunadevi	1123770	26	G3P2L2	38	B	110	80	LN	2.6	6 /10	8 /10	10.2	2.1	0.8	31	26	118	14	0.7
79	C	selvaraani	1123753	28	Primi	38	B	120	80	LN	2.6	6 /10	8 /10	9.8	1.4	0.8	24	26	177	16	0.6
80	C	sathya	8868	27	Primi	37	B	100	80	LN	2.6	6 /10	8 /10	10.1	2.2	0.8	24	23	176	26	0.4
81	C	indhumathi	8847	30	Primi	38	B	120	80	LSCS	2.4	6 /10	8 /10	10.6	1.36	0.8	35	34	178	18	0.6
82	C	sathya	8869	36	Primi	38	B	120	80	LN	2.8	6 /10	8 /10	9.8	1.26	0.8	34	33	181	17	0.8
83	C	kalaivani	1123802	27	Primi	38	B	100	80	LSCS	2.8	6 /10	8 /10	10.2	2.22	0.6	40	39	118	16	0.7

84	C	manimala	8823	28	G2P1L1	38	B	100	80	LN	2.6	6 /10	8 /10	10.4	2.1	0.6	28	26	131	24	0.6
85	C	arunadevi	1123770	29	G3P2L2	38	B	110	80	LN	2.5	6 /10	8 /10	9.8	2.1	0.8	27	26	231	18	0.8
86	C	maheswari	8769	26	G3P2L2	39	B	120	80	LN	2.8	6 /10	8 /10	10.6	2.9	0.8	24	26	230	21	0.7
87	C	nandhini	8792	28	Primi	40	B	120	80	LSCS	2.4	6 /10	8 /10	9.8	3.2	0.8	30	31	182	18	0.8
88	C	vijayalakshmi	1123762	32	Primi	39	B	110	80	LN	3.2	6 /10	8 /10	10.2	3.1	0.8	20	18	141	18	0.7
89	C	naageswari	8964	31	Primi	38	B	120	80	LN	2.8	6 /10	8 /10	10.6	2.5	0.8	30	32	151	14	0.6
90	C	lakshmi	8834	26	G2P1	39	B	110	80	LSCS	3.6	6 /10	8 /10	10.8	2.3	0.8	30	31	156	24	0.5
91	C	murugeswari	8877	28	Primi	38	B	120	80	LSCS	3.8	6 /10	8 /10	9.8	2.8	0.7	23	21	178	16	0.4
92	C	nagajothi	8865	26	Primi	38	B	120	80	LSCS	2.9	6 /10	8 /10	10.2	2.6	0.6	18	17	231	16	0.8
93	C	renugadevi	8825	21	G3P2L2	39	B	100	80	LN	3.2	6 /10	8 /10	10.1	2.8	0.8	29	17	311	18	0.7
94	C	muthu	8936	22	G3P2L2	39	B	120	80	LN	3.4	6 /10	8 /10	11	3	0.8	31	30	132	20	0.6
95	C	priya	1123921	28	G2P1L1	36	B	130	80	LN	2.5	6 /10	8 /10	9.8	2.1	0.8	27	26	231	18	0.8
96	C	chitirai selvi	1123800	24	Primi	38	B	120	80	LN	2	6 /10	8 /10	10.2	1.8	0.9	32	30	112	22	0.9
97	C	prathiba	1123937	24	Primi	39	B	120	70	LN	2.6	6 /10	8 /10	10.4	1.8	0.8	28	26	132	24	0.8
98	C	nandhini	3014	22	Primi	38	B	110	70	LN	3	5/10	7/10	10.2	2.8	0.8	28	25	311	26	0.8
99	C	meenatchi	8898	22	Primi	39	B	120	80	LN	2.2	6 /10	8 /10	11.2	2.6	0.8	26	24	231	28	0.8
100	C	jeeva	8129	23	Primi	40	B	110	70	LSCS	3.8	6 /10	8 /10	10.6	2.6	0.6	26	25	178	28	0.9
101	C	muthuirulayee	1123981	23	G2P1L1	38	B	120	70	LN	3.1	6 /10	8 /10	9.2	2.89	0.7	26	24	156	28	1
102	C	nandhini	1123980	31	G3P2	39	B	110	80	LN	3	6 /10	8 /10	11.2	2.9	0.8	32	30	151	26	1.1
103	C	dhanalakshmi	1123972	30	G2P1L1	38	B	120	70	LN	2.8	6 /10	8 /10	10.2	3.14	0.8	26	24	141	26	1
104	C	jothi	1123947	31	G2P1	38	B	110	70	LN	2.6	6 /10	8 /10	9.8	3.2	0.8	20	20	182	18	1
105	C	dhilsath	1123056	32	G2P1L1	39	B	120	80	LN	3	6 /10	8 /10	11.2	3.2	0.8	27	26	230	20	0.9
106	C	nandeeswari	8939	20	G2P1L1	38	B	110	70	LSCS	3.2	6 /10	8 /10	9.8	3.4	0.6	24	21	231	21	0.9
107	C	iswarya	1123984	19	Primi	38	B	110	70	LSCS	3.4	6 /10	8 /10	10.2	3.4	0.7	24	32	131	23	0.8
108	C	muthuirulayee	8854	18	Primi	39	B	110	80	LSCS	3.4	6 /10	8 /10	10.4	3.6	0.8	31	24	118	20	0.8

109	C	paandiselvi	1123983	18	G4P3L3	38	B	110	70	LSCS	3.4	5/10	7/10	11.2	2.8	0.6	26	21	181	21	0.8
110	C	vimaladevi	8965	21	Primi	39	B	120	70	LN	3.1	6 /10	8 /10	9.8	3	0.8	30	28	178	22	0.7
111	C	vasanthy	8895	23	G2P1L1	440	B	120	80	LN	2.2	6 /10	8 /10	10.2	3.2	0.8	28	31	118	21	0.7
112	C	thamaraiselvi	8953	24	Primi	38	B	110	70	LN	2.4	6 /10	8 /10	11.2	2.8	0.8	34	30	176	20	0.6
113	C	vanitha	8950	21	Primi	38	B	120	70	LN	2.6	6 /10	8 /10	9.8	2.6	0.8	28	31	174	21	0.7
114	C	dhurga	8975	22	Primi	39	B	120	70	LSCS	2.4	6 /10	8 /10	10.2	3.6	0.6	28	26	172	18	0.6
115	C	raajeswari	8902	24	Primi	40	B	110	70	LN	2.2	6 /10	8 /10	9.6	3.2	0.7	32	24	182	16	0.7
116	C	suleka	8972	28	Primi	38	B	110	80	LN	2.8	6 /10	8 /10	9.2	1.2	0.9	26	22	216	24	0.8
117	C	rakku	8932	29	G2P1L1	39	B	110	80	LN	2.6	6 /10	8 /10	9.8	1.4	0.8	26	21	500	22	0.9
118	C	kalaivaani	1123794	29	G2P2	39	B	120	70	LN	2.8	6 /10	8 /10	10.2	2.3	1.1	26	24	216	24	0.8
119	C	muthurakku	1124095	24	G2P1L1	38	B	110	80	LN	2.8	5/10	7/10	9.8	2.36	1.2	25	20	208	28	0.1
120	C	karupayee	112496	18	Primi	38	B	120	80	LN	3	6 /10	8 /10	10.6	1.8	0.8	28	21	114	26	1
121	C	sudharani	8784	18	Primi	38	B	110	70	LN	2.8	6 /10	8 /10	9.6	2.46	0.8	25	26	126	28	1.2
122	C	kaveri	8986	19	Primi	38	B	120	80	LN	3.2	6 /10	8 /10	10.2	2.6	0.8	28	21	118	28	0.9
123	C	durgadevi	1123950	32	G3P2L2	39	B	110	80	LSCS	3.5	6 /10	8 /10	12	2.8	0.8	25	21	201	29	0.8
124	C	pothumani	48841	21	Primi	38	B	120	80	LN	3.4	6 /10	8 /10	10.4	3.2	0.6	30	20	214	30	0.6
125	C	kavitha	48846	22	Primi	37	B	110	80	LN	2.6	6 /10	8 /10	11.2	2.4	0.6	30	20	216	31	0.4
126	C	kalivaani	1124112	22	Primi	38	B	120	70	LSCS	4.1	6 /10	8 /10	11.4	2.36	0.8	29	21	218	29	0.6
127	C	yogeswari	48840	23	Primi	38	B	110	80	LN	3.3	6 /10	8 /10	10.2	3.3	0.6	31	30	516	28	0.8
128	C	jegadeeswari	8973	23	Primi	38	B	120	80	LN	2.4	4/10	5/10	10.6	1.86	0.6	29	28	216	27	0.6
129	C	saranya	1124119	23	Primi	38	B	110	80	LN	2.5	6 /10	8 /10	11	1.8	0.8	29	21	234	26	0.4
130	C	veerayee	1124111	31	G2P1L1	38	B	120	70	LSCS	2.6	6 /10	8 /10	11.2	1.92	0.8	28	26	302	24	0.5
131	C	lakshmi	8984	30	G2P1L1	38	B	110	80	LN	3.2	6 /10	8 /10	10.8	2.06	0.8	26	25	184	21	0.8
132	C	selvi	1124117	29	G2P1L1	38	B	120	80	LN	3.1	6 /10	8 /10	10.2	1.9	0.9	27	26	208	19	0.9

133	C	vennila	2647	28	G2P1L1	37	B	110	70	LN	3.2	6 /10	8 /10	9.8	3.02	0.8	26	25	141	18	0.8
134	C	rajeswari	8938	28	G2P1L1	38	B	130	80	LSCS	3.2	6 /10	8 /10	11.2	2.01	0.9	27	26	142	20	0.8
135	C	jammeema	8990	21	G2P1L1	38	B	110	80	LN	3	6 /10	8 /10	10.2	2.2	1	21	20	184	16	1
136	C	chitra	1124409	26	Primi	38	B	110	80	LN	2.5	6 /10	8 /10	9.6	1.24	1	30	28	112	32	0.9
137	C	krishnaveni	1114384	28	Primi	37	B	110	80	LN	3.2	6 /10	8 /10	9.8	1.3	1	29	27	114	30	0.8
138	C	kowsalya	9036	26	G2P1L1	38	B	110	70	LN	3.4	6 /10	8 /10	10.2	1.2	0.9	26	20	116	18	0.9
139	C	nithya	9075	27	Primi	39	B	110	80	LN	3.6	6 /10	8 /10	10	2.2	0.8	29	20	118	18	0.8
140	C	hemalatha	8901	28	Primi	39	B	120	80	LSCS	3.8	6 /10	8 /10	10.4	2.46	0.9	26	18	227	20	0.8
141	C	balalakshmi	8541	31	Primi	38	B	110	80	LN	3.4	6 /10	8 /10	10.6	2.68	0.8	28	17	210	22	0.8
142	C	chitradevi	8571	24	G3P1L1A1	38	B	130	80	LN	3.2	6 /10	8 /10	10.7	3.02	0.8	28	16	238	24	0.9
143	C	jeyalakshmi	1124423	22	G2P1L1	39	B	110	70	LSCS	4.2	6 /10	8 /10	10.2	2.6	0.9	28	21	301	21	0.8
144	C	thayammal	1124428	20	Primi	40	B	120	70	LN	2.8	5 /10	7 /10	8.5	3.04	0.8	28	27	239	20	0.8
145	C	suganya	9039	21	Primi	41	B	120	70	LN	3	5 /10	7 /10	9.8	2.8	0.7	21	20	218	22	0.6
146	C	nisha	9084	28	G2P1L1	40	B	120	70	LN	3.2	6 /10	8 /10	9.2	3.8	0.8	27	20	180	18	0.8
147	C	priya	9076	24	G2P1L1	40	B	110	80	LN	3.4	6 /10	8 /10	9.6	2.6	0.8	30	22	180	18	0.8
148	C	muniyammal	8911	23	Primi	38	B	120	70	LN	3.6	6 /10	8 /10	9.2	3.2	0.9	26	21	184	17	0.8
149	C	pandiselvi	8912	22	G2P1L1	39	B	110	70	LSCS	3.1	5 /10	7 /10	9.6	2.8	1.1	28	21	178	16	0.6
150	C	sivapriya	1124877	18	Primi	38	B	120	70	LN	2.6	6 /10	8 /10	9.4	4	0.8	28	27	211	21	0.8

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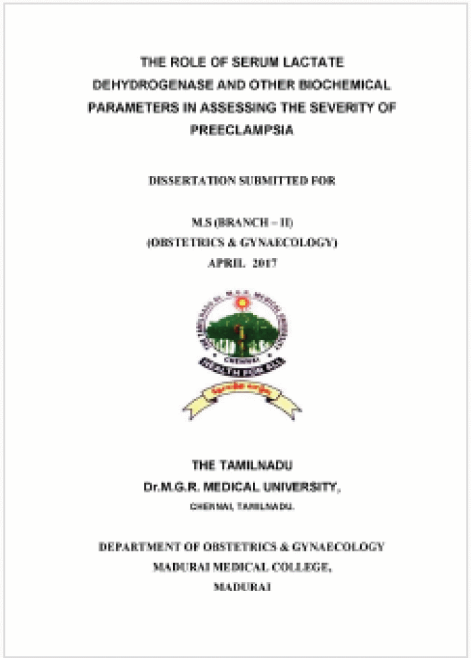

THE ROLE OF SERUM LACTATE DEHYDROGENASE AND OTHER BIOCHEMICAL PARAMETERS IN ASSESSING THE SEVERITY OF PREECLAMPSIA

DISSERTATION SUBMITTED FOR

M.S (BRANCH – II)
(OBSTETRICS & GYNAECOLOGY)
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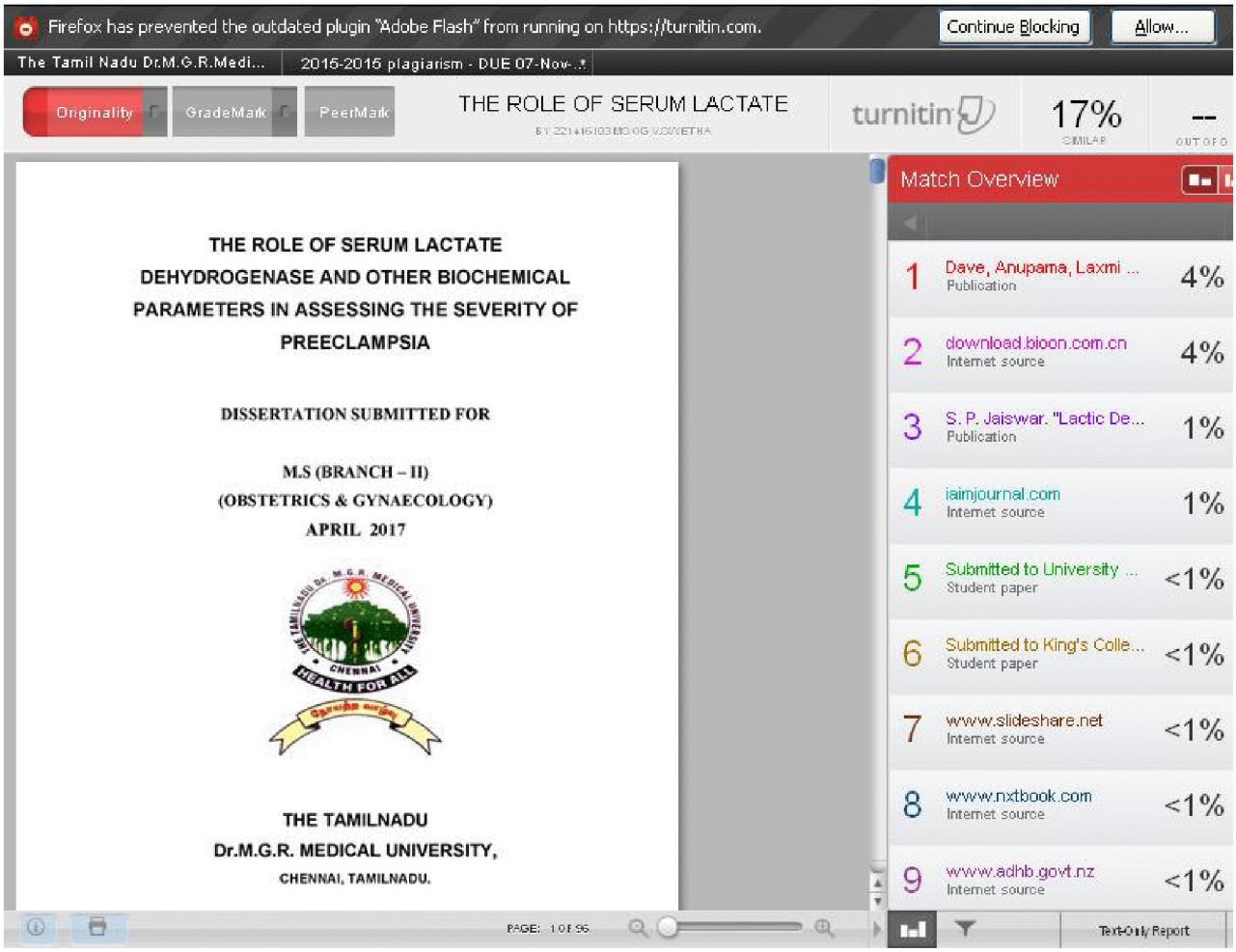
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