THE STUDY OF SERUM URIC ACID AS A BIOCHEMICAL INDICATOR FOR MATERNAL AND FETAL OUTCOME IN GESTATIONAL HYPERTENSION

THESIS

SUBMITTED IN PARTIAL FULFILLMENT OF THE REGULATIONS OF

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M.S. OBSTETRICS AND GYNAECOLOGY

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GOVERNMENT THENI MEDICAL COLLEGE AND HOSPITAL

TAMIL NADU (INDIA)

MAY 2020

CERTIFICATE

This is to certify that this dissertation entitled **"THE STUDY OF SERUM URIC ACID AS A BIOCHEMICAL INDICATOR FOR MATERNAL AND FETAL OUTCOME IN PATIENTS WITH GESTATIONAL HYPERTENSION" is** a bonafide and original work done by **Dr. Kalpana Kumari Meena,** post-graduate student, under my overall supervision in the department of Obstetrics & Gynecology, Government Theni Medical College & Hospital, Theni, in partial fulfillment of the regulations of The Tamil Nadu Dr. M.G.R. Medical University for the award of degree of M.S. Obstetrics & Gynecology May 2020

Dr. B. Shanthi Rani, M.D., DGO

Head of Department

Department of Obstetrics & Gynaecology

GTMCH, Theni

Tamil Nadu

CERTIFICATE II

This is to certify that this dissertation entitled **"THE STUDY OF SERUM URIC ACID AS A BIOCHEMICAL INDICATOR FOR MATERNAL AND FETAL OUTCOME IN PATIENTS WITH GESTATIONAL HYPERTENSION"** is a bonafide work of the candidate **Dr.Kalpana Kumari Meena** with Registration Number 221716653 for the award of degree of M.S. Obstetrics & Gynecology. Government Theni Medical College & Hospital, Theni.

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I**, Dr.Kalpana Kumari Meena**, solemnly declare that this dissertation **"THE STUDY OF SERUM URIC ACID AS A BIOCHEMICAL INDICATOR FOR MATERNAL AND FETAL OUTCOME IN PATIENTS WITH GESTATIONAL HYPERTENSION"** is a bonafide and original work done by me at the Department of Obstetrics & Gynecology, Government Theni Medical College & Hospital, Theni, under the guidance and supervision of **Dr.M.Thangamani, MD, DGO, Dr.K.Kameswari M.D., OG.,** I also declare that this original work or a part of this work was not submitted by me or any other for any award, degree, diploma to any other university board either in India or abroad.

This dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University in partial fulfillment of the University regulations for the award of degree of M.S Obstetrics & Gynecology examinations to be held in.

Place: Theni, Tamil Nadu — Signature of the Candidate

Date: — (Dr.Kalpana Kumari Meena)

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October, 2019 Dr. Kalpana Meena

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ABBREVIATIONS

ACE	Angiotensin Converting Enzyme
ACOG	American College of Obstetricians and Gynecologists
AGT	Angiotensinogen
ALT	Alanine Aminotransferase
API	Association of Physicians of India
AST	Aspartate Aminotransferase
BD	*Bis-in-die* (twice daily)
BMI	Body Mass Index
BP	Blood Pressure
CNS	Central nervous System
CTLA-4	Cytotoxic T-Lymphocyte Associated Protein
CVS	Cardiovascular System
EDD	Estimated Date of Delivery
F2	Prothrombin
F5	Factor V Leiden
GA	Gestational Age
GHT	Gestational Hypertension
GTMCH	Government Theni Medical College and Hospital
HDP	Hypertensive Disorders of Pregnancy
HELLP	Hemolysis, Elevated Liver Enzymes, Low Platelets
HLA	Human Leukocyte Antigen
IBM	International Business Machines

ISSHP	International Society for the Study of Hypertension in Pregnancy
IUD	Intra-Uterine Death
IUGR	Intra-Uterine Growth Retardation
kDa	Kilo Dalton
LBW	Low Birth Weight
LDH	Lactate Dehydrogenase
LFT	Liver Function Test
LMP	Last Menstrual Period
LPL	Lipoprotein Lipase
MTHFR	Methyl Tetra Hydro Folase Reductase
NHBPEP	National High Blood Pressure Education Program
NICE	National Institute of Health and Clinical Excellence
NOS-3	Endothelial Nitric Oxide
OG	Obstetrics & Gynaecology
PIGF	Placental Growth Factor
RAAS	Renin Angiotensin Aldosterone System
RFT	Renal Function Test
SD	Standard Deviation
SERPINE-1	Serine Peptidase Inhibitor
sEng	Soluble Endoglin
sFlt-1	Soluble Fms-like Tyrosine Kinase 1
SPSS	Statistical Package for Social Sciences

TDS	*Ter Die Sumendum* (thrice daily)
TGF-β	Transforming Growth Factor-β
TSH	Thyroid Stimulating Hormone
VEGF	Vascular Endothelial Growth Factor

INTRODUCTION

Hypertensive disorders of pregnancy (HDP) are among the most common medical disorders during pregnancy and are considered to be a major cause of maternal and fetal morbidity and mortality. In developing countries, HDP ranks second only to anemia with approximately 7-10% of all pregnancies complicated by some form of hypertensive disorder and lead to various maternal and fetal complications (1). In India, the incidence of pre-eclampsia, as recorded from hospital statistics, varies widely from 5-15%, while that of eclampsia is about 1.5% (2). Strangely, the exact etiopathogenesis for HDP, including pre-eclampsia and eclampsia, still remains obscured and presents an interesting unsolved mystery in obstetric practice.

The diagnosis of pre-eclampsia is based on the presence of the following clinical features: blood pressure (BP) ≥140/90 mmHg after 20 weeks of gestation AND proteinuria ≥300 mg/24-hr or +1 with dipstick. There may be other associated abnormalities reported which increase the likelihood of occurrence of pre-eclampsia such as elevated serum creatinine (new onset), platelet count <100,000/µL, hepatic enzyme abnormalities, persistent headache with or without cerebral or visual disturbances, and epigastric pain. Pre-eclampsia with presence of seizures is known as eclampsia (2).

Elevated uric acid level in maternal blood, presumably due to decreased renal excretion, is frequently found in women with pre-eclampsia. Of the several hypothesized factors for presence of elevated uric acid in patients with pre-eclampsia, the following appear to be most intriguing: abnormal renal function, increased tissue

breakdown, acidosis, and increased activity of the enzyme xanthine oxidase/dehydrogenase (1).

An association of uric acid elevation in clinically evident pre-eclampsia has been known since 1917. Several studies have correlated the rise in uric acid with the severity of the pre-eclampsia. Although hyperuricemia does correlate with maternal morbidity, there is an even stronger association of increased uric acid with risk for small or low birth weight infants and with overall fetal mortality.

REVIEW OF LITERATURE

According to the National High Blood Pressure Education Program (NHBPEP) (3) and American College of Obstetricians and Gynecologists (ACOG) (4), hypertension is defined as:

1) Systolic BP of ≥140 mmHg and/or

2) Diastolic BP of ≥90 mmHg (Korotkoff V) that is measured on 2 occasions, 4-6 hrs apart, within a 7-day period.

Increase in systolic blood pressure by 30 mmHg or by 15 mmHg diastolic pressure above the patient's baseline was abandoned as the diagnostic criteria in hypertension as it could not prove to be a good prognostic indicator. Pre-eclampsia is a multi-organ disease of unknown etiology that leads to the development of hypertension and proteinuria at or after 20 weeks of gestation. Classically defined as the triad of hypertension, edema and proteinuria, recent definitions exclude edema due to lack of specificity. Appearance of proteinuria remains an important diagnostic criterion in differentiating pre-eclampsia from gestational hypertension**.**

Proteinuria is defined as:

- 24-hr urine protein excretion exceeding 300 mg
- Urine Protein to Creatinine ratio ≥0.3 (30 mg/mmol) or
- Persistent proteinuria ≥30 mg/dL (urinary dipstick of 1+ in random urine samples)

The most up-to-date classification for HDP is provided by the International society for the study of hypertension in pregnancy (ISSHP) 2014 (2) according to which HDP is classified into 4 groups as follows:

1. **Gestational Hypertension (GHT)** – New onset hypertension developing after 20 weeks of gestation, during labor or in the first 24-hr post-partum without proteinuria or any other systemic features of pre-eclampsia in previously normotensive non-proteinuric women and the elevated BP resolves within 3 months post-partum.
2. **Pre-eclampsia and Eclampsia** – Hypertension, associated with proteinuria >300 mg/24-hr urine collection or 1+ by qualitative urine examination, at or after 20 weeks of gestation is known as pre-eclampsia. Presence of convulsions in a woman with pre-eclampsia is called eclampsia.
3. **Chronic Hypertension** – It is defined as hypertension present before 20 weeks of pregnancy or that is diagnosed pre-conceptionally. Blood pressure elevation that persists for >12 weeks post-partum is also, retrospectively, considered as chronic hypertension. Chronic hypertension is of 2 subtypes:
    - **Essential Hypertension -** Diagnosed when there is no apparent underlying cause for chronic hypertension.
    - **Secondary Hypertension** – Usually caused by an underlying disease process, like renal parenchymal disease, reno-vascular disease, endocrine disorders, coarctation of the aorta, etc.
4. **Pre-eclampsia superimposed on Chronic Hypertension** – It is diagnosed when one or more features of pre-eclampsia newly develop during pregnancy, at or after 20 weeks of gestation, in a woman with pre-existing chronic hypertension.

CLASSIFICATION OF HYPERTENSION ACCORDING TO SEVERITY

Hypertension is classified according to severity as per the 2004 NICE guidelines (5) as follows:

- **Mild Hypertension**: Systolic BP 140-149 mmHg, diastolic BP 90-99 mmHg.
- **Moderate Hypertension**: Systolic BP 150-159 mmHg, diastolic BP 100-109 mmHg.
- **Severe Hypertension**: Systolic BP ≥160 mmHg, diastolic BP ≥110 mmHg.

RISK FACTORS FOR PRE-ECLAMPSIA (6)

Not every pregnancy is complicated by the development of hypertension. There are certain risk factors which make certain individuals/pregnancies more prone to developing GHT and complications thereof, such as pre-eclampsia. These risk factors can be couple related, maternal or pregnancy related, or due to pre-existing medical conditions. The risk factors are summarized in the table below.

Couple Related	Maternal or Pregnancy Related	Pre-Existing Medical Conditions
• First paternity • Limited sperm exposure • Pregnancy after donor insemination, donor egg, donor embryo • Dangerous male partner	• Extremes of age (teenage and >35 years) • Parity • Interval from last pregnancy >10 years • Insulin resistance/ Gestational diabetes	• Obesity • Pre-gestational diabetes • Chronic hypertension • Renal disease • Maternal immunologic disease • Thrombophilia

	• Smoking • Multifetal pregnancy • Hydrops fetalis • Hydatiform mole • Pre-eclampsia in previous pregnancy • Family history of pre-eclampsia • Maternal low birth weight • Abnormal uterine artery Doppler at 18-24 weeks	• Antiphospholipid antibody syndrome

ETIOPATHOGENESIS OF PRE-ECLAMPSIA

Though it has been emphasized that the etiopathogenesis of pre-eclampsia remains undetermined, the most suited hypothesis is explained by a six-step process involving multiple organ-systems and is summarized below.

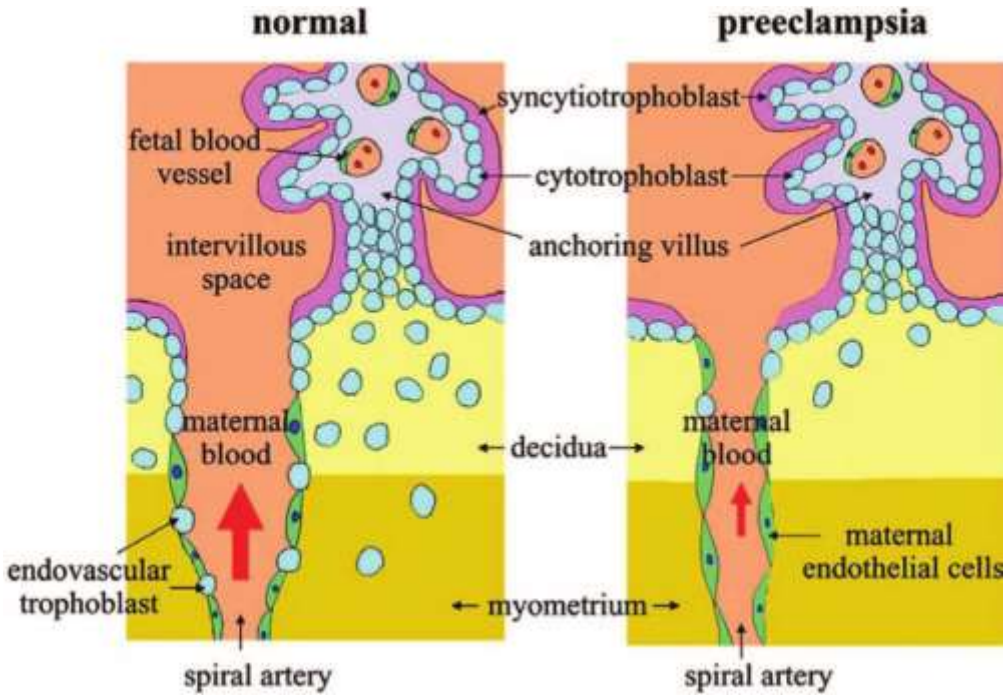
1. **Abnormal Trophoblastic Invasion (7)**

This initial step can be explained by the Two-Stage Disorder Hypothesis (8).

The first stage occurs prior to 12 weeks post-fertilization up to the interface between decidua and myometrium. First stage involves endovascular invasion of spiral arteries

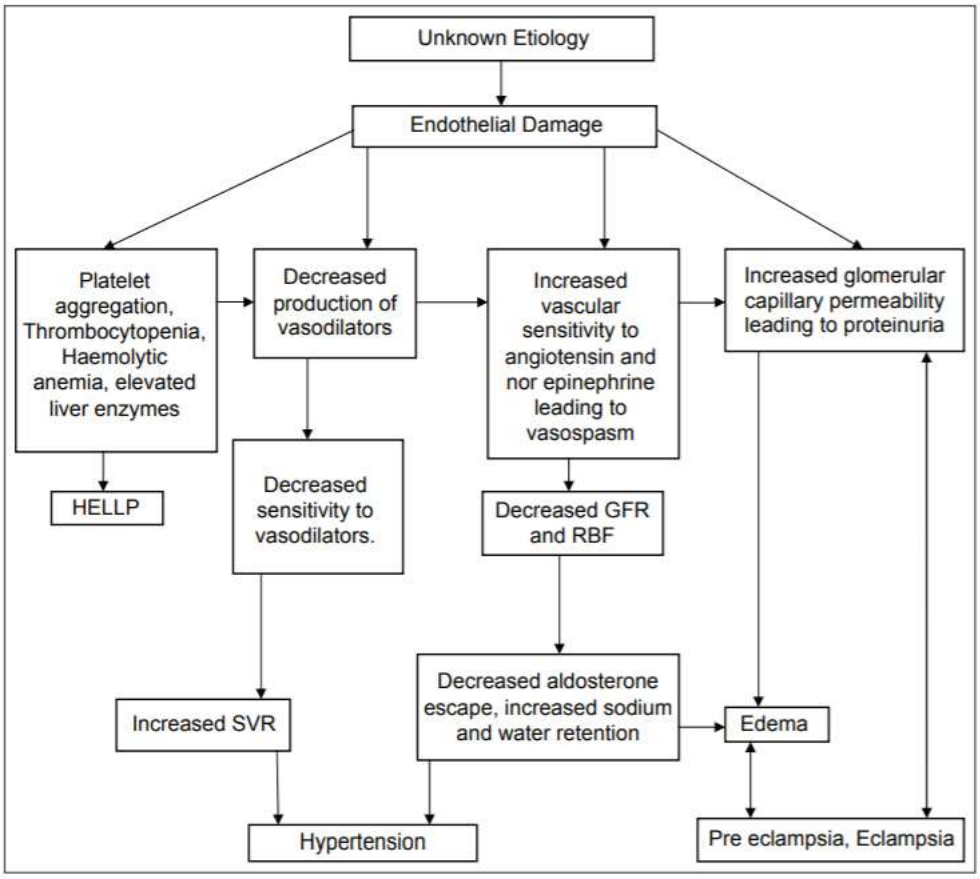
by trophoblastic cells. This migration transforms the small musculo-elastic spiral arteries into large tortuous channels that carry large amount of blood to the inter-villous space and are resistant to the effects of vasomotor agents. These physiologic changes are incomplete in patients of pre-eclampsia and trophoblastic invasion affects only in some of the spiral arteries and does not progress into the myometrial portion of the arteries (Incomplete Trophoblastic Invasion). This deficiency results in decreased utero-placental perfusion.

The second stage normally occurs between 12 and 16 weeks of gestation which involves invasion of intra-myometrial segments of spiral arteries. In pre-eclampsia, second stage involves the conversion of previous utero-placental maladaptation to Maternal Systemic Syndrome of pre-eclampsia which is associated with endothelial cell activation and generalized hyperinflammatory state. This anatomic and physiologic disruption of normal placentation leads to the release of placental debris from the intervillous space into the maternal circulation, thereby inciting a systemic inflammatory response by stimulating the synthesis of inflammatory cytokines, products that affects angiogenesis and abnormal lipid peroxidation. Severity of hypertensive disorder is correlated with the magnitude of abnormal trophoblastic invasion.



2. **Endothelial Dysfunction and Vasospasm**

The figure below summarizes the hypothesized etiopathogenic mechanism for the development of endothelial dysfunction and vasospasm leading to the clinical features seen in GHT.



3. **Immunologic Factors (8)**

In pre-eclamptics, there is loss of maternal tolerance to paternally derived placental and fetal antigens. The placenta being fetal origin has both maternal and paternal haplotypes and genetic determinants. Of the histocompatibility antigens only HLA-G, an immunosuppressive antigen is expressed on the surface of trophoblasts which is a placental cell most intimate to the maternal system. In pre-eclamptics, there is lower level of messenger RNA for HLA-G leading to reduced expression of HLA-G on trophoblasts. This causes immune maladaptation in pre-eclampsia.

4. **Genetic Factors (9)**

The hereditary predisposition to developing pre-eclampsia syndrome is the result of complex interaction of several genes, including both maternal and paternal. Thus, pre-

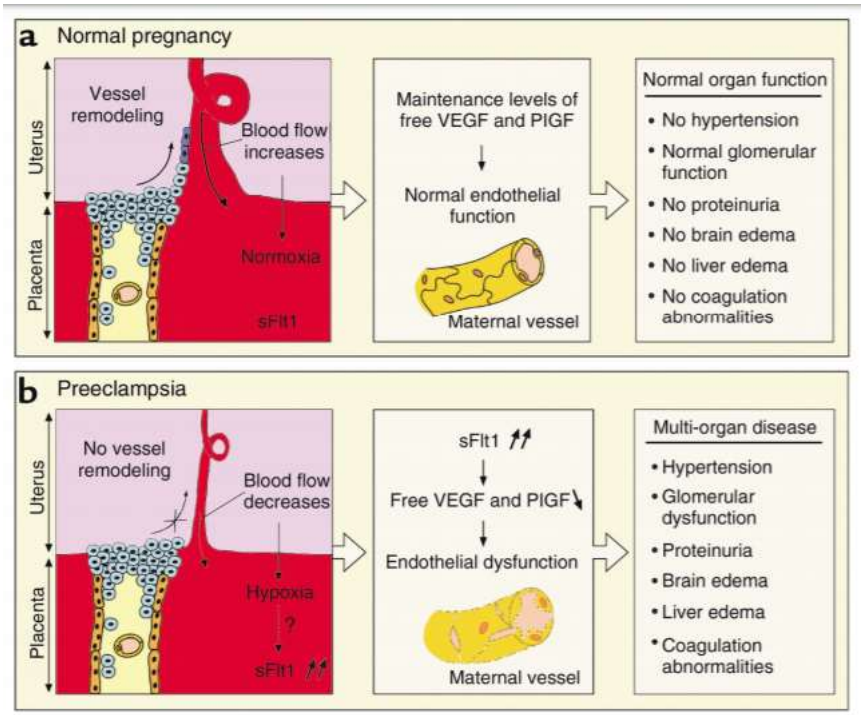
eclampsia is multifactorial and polygenic in origin. Genes that are in positive association with pre-eclampsia syndrome are as follows:

- MTHFR (Gene which affects MethylTetraHydroFolateReductase)
- HLA (Human Leukocyte Antigen)
- F5 (Factor V Leiden)
- F2 (Prothrombin)
- AGT (Angiotensinogen)
- NOS-3 (Endothelial Nitric Oxide)
- ACE (Angiotensin Converting Enzyme)
- CTLA-4 (Cytotoxic T-Lymphocyte Associated Protein)
- LPL (Lipoprotein Lipase)
- SERPINE-1 (Serine Peptidase Inhibitor)

5. **Angiogenic Imbalance**

In pre-eclampsia, due to worsening hypoxia at the utero-placental interface, there is excessive production of anti-angiogenic peptides, especially the following 2 peptides:

- **Soluble Fms-like Tyrosine Kinase 1 (sFlt-1) (10**) – It is a variant of Flt-1 receptor for placental growth factor (PIGF) and vascular endothelial growth factor (VEGF). Increased maternal sFlt-1 levels inactivate and decrease PIGF and VEGF concentrations leading to endothelial dysfunction.
- **Soluble Endoglin (sEng) (11**) – It is a placenta derived 65-kDa molecule that blocks endoglin, which is a surface co-receptor for TGF-β Family. Also called CD-105, soluble form of end isoform binds to endothelial receptors that results in decreased nitric oxide dependent vasodilatation.



6. **Renin-Angiotensin-Aldosterone System (RAAS) (12)**

The RAAS axis maintains sodium concentration, blood volume and BP. In normal pregnancy, renin activity, plasma renin concentration, angiotensin-II levels, and aldosterone levels are increased. However, there is refractoriness to the effects of angiotensin-II. In women who are destined to be pre-eclamptic, there is a loss of refractoriness due to down-regulation of **a**ngiotensin receptors in the presence of persistently elevated levels.

PATHOPHYSIOLOGICAL CHANGES

It involves multiple organ systems as described below.

1. **Cardiovascular System**

In pre-eclampsia, due to increased peripheral resistance (13), there is decreased cardiac output that results in hyperdynamic ventricular function. Pulmonary edema results from alveolar endothelial-epithelial leak. Generalized vasoconstriction causes increased

vascular permeability and leakage of plasma into interstitial space that results in reduced plasma volume and hemoconcentration.

2. **Coagulation Profile**

Coagulation abnormalities seen in severe pre-eclampsia and eclampsia patients include thrombocytopenia, reduced levels of various clotting factors, evidence of platelet activation, intravascular coagulation, and hemolysis. Thrombocytopenia is defined as a platelet count of <100,000/μL and indicates severe disease. Hemolysis is confirmed by increase in lactate dehydrogenase (LDH) levels and peripheral smear showing schistocytosis, spherocytosis and reticulocytosis (14). These abnormalities result from microangiopathic hemolysis. The association of thrombocytopenia, hemolysis along with elevated liver enzymes is referred to as HELLP syndrome.

3. **Renal System**

Due to increased afferent arteriolar resistance, there is reduced renal blood flow and reduced glomerular filtration rate. This results in elevated plasma uric acid. Urine sodium concentration is increased, and urinary excretion of calcium is decreased due to increased tubular reabsorption. Proteinuria is the hallmark of pre-eclampsia. Acute renal failure due to tubular necrosis is rare and is usually induced by hypotension and hypovolemia in severe disease. Pre-eclampsia produces characteristic changes in the kidneys which is known as glomerular capillary endotheliosis, and early focal segmental glomerular sclerosis in some patients (15,16). These lesions usually resolve completely post-pregnancy.

4. **Hepatic System**

Excessive fibrin deposition in hepatic sinusoids obstructs blood flow and hepatic vasoconstriction which causes the release of hepatic enzymes (AST, ALT, LDH) in circulation. Characteristic lesions are peri-portal hemorrhages in the liver periphery. Hepatic vasculature in the subcapsular region is particularly susceptible resulting in sub-capsular hemorrhages which become larger and form sub-capsular hematomas and may even result in hepatic rupture.

5. **Brain**

Intense vasospasm of the cerebral arterioles and over-dilatation of vessels are implicated in the pathogenesis of eclampsia. In hypertension, as a part of auto-regulatory mechanism, cerebral vasoconstriction occurs which leads to ischemia, edema, and infarction. When the regulatory mechanisms fail, dilatation of vessels occur resulting in hyper-perfusion and vasogenic edema (17). The pathognomonic finding in pre-eclampsia is edema with foci of infarction. Cerebral hemorrhage should be suspected in older gravida with chronic hypertension. Such patients present with hemiplegia, focal deficits, and coma, following eclampsia. CT/MRI scans should be done to confirm the diagnosis. Blindness can occur due to severe papilledema, retinal detachment, and occipital lobe lesions (18).

6. **Placenta**

Placenta of hypertensive patients shows increased evidence of infarcts, congested chorionic villi, hematoma, and proliferative endarteritis. Microscopic examination

shows increased syncytial knots, fibrinoid necrosis, cytotrophoblastic cellular proliferation, calcified and hyalinized villous spots, and endothelial proliferation (19).

COMPLICATIONS OF PRE-ECLAMPSIA

The complications are studied under two heads: Maternal and Fetal complications.

Maternal Complications	Fetal Complications
• Abruptio Placentae (most common) • HELLP Syndrome • Pulmonary edema • Thrombocytopenia/Disseminated Intravascular Coagulation • Acute renal failure • Adult respiratory distress syndrome • Eclampsia • Hepatic rupture • Cerebral hemorrhage • Sudden post-partum collapse	• Intra-uterine growth retardation (IUGR) • Pre-maturity • Ante-partum and Intra-partum asphyxia • Intra-uterine death (IUD)

PREDICTIVE TESTS FOR THE DEVELOPMENT OF PRE-ECLAMPSIA

Modality Tested	Tests
Placental Perfusion/Vascular Resistance	Roll-Over Test (12), Isometric Handgrip Test, Pressor Response to Angiotensin-II Infusion (12), Mid-trimester Mean Arterial Blood Pressure (20), 24-Hr

	Ambulatory Blood Pressure Monitoring, Uterine Artery Doppler Velocimetry, Pulse Wave Analysis (20)
Fetoplacental Unit Endocrine dysfunction	α-Fetoprotein, Human Chorionic Gonadotrophin (**Error! Bookmark not defined.**), Estriol, Pregnancy-Associated Plasma Protein-A, Inhibin-A, Activin-A, Placental Protein-13, Corticotropin-Releasing Hormone
Renal Dysfunction	Serum Uric Acid26, Microalbuminuria, Urinary Calcium (**Error! Bookmark not defined.**), Microtransferrinuria, Cystatin-C
Endothelial Dysfunction/Oxidative Stress	Platelet Count, Platelet Activation, Lactate Dehydrogenase, Fibronectin (21), Prostaglandins, Prostacyclins, Thromboxane, C-Reactive Protein, Cytokines, Endothelins, Homocysteine, Anti-Phospholipid Antibody, Placental Growth Factor (PIGF), Vascular Endothelial Growth Factor (VEGF), sFlt-1
Others	Antithrombin-III, Atrial Natriuretic Peptide, β2-Microglobulin, Haptoglobin, Cell Free Fetal DNA, Serum and Urine Proteomics, Hepatic Aminotransferases

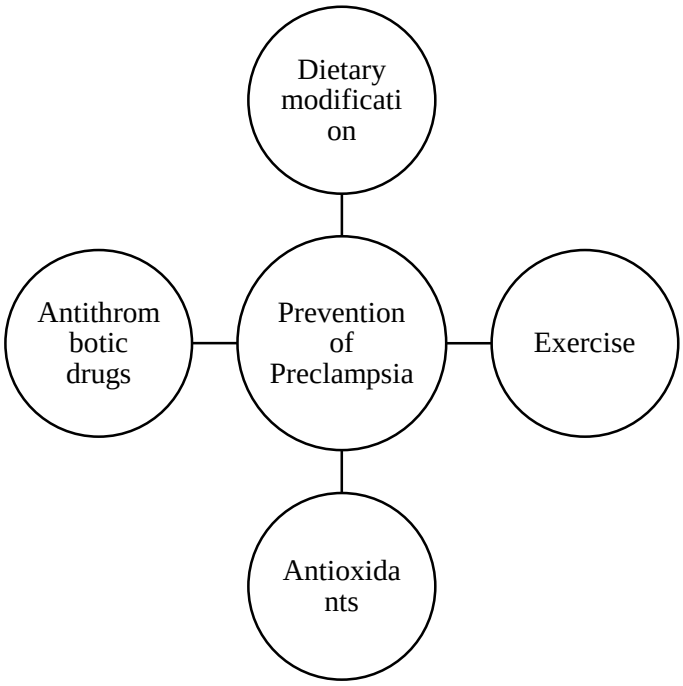
UTERINE ARTERY DOPPLER VELOCIMETRY

In normal pregnancy, impedance to uterine artery blood flow is reduced. In pre-eclampsia, failure of trophoblastic invasion into vessels is reflected by high impedance to uterine blood flow. Increased resistance to flow and presence of diastolic notch is indicative of pre-eclampsia. Whereas in chronic hypertension, it is predictive of superimposed pre-eclampsia. Other indices like pulsatility index, and resistance index are noted**.** Recent studies show combined use of Doppler studies and biochemical

markers in the first trimester of pregnancy have improved prediction rates for pre-eclampsia (20, 22, 23).

PREVENTION OF PRE-ECLAMPSIA

The mainstay for prevention of pre-eclampsia is four-pronged approach as shown in the graph below.



1. **Dietary Modifications**
    - **Low Salt Diet**: Earliest researches have found that salt restriction is useful in preventing pre-eclampsia. However, Knuist et. al. proved that salt restricted diet is ineffective in preventing pre-eclampsia (24).
    - **Calcium Supplementation**: Calcium supplementation of 1.5-2 gm/day reduces the incidence of pre-eclampsia by half (25).
    - **Zinc Supplementation**: Low serum zinc levels associated with suboptimal outcomes of pregnancy including pre-eclampsia. However, Cochrane review

(2012) does not reveal any evidence of improved pregnancy outcome with zinc supplementation (26).

- **Fish oil Supplementation**: Supplementation of common dietary sources such as EPA (eicosapentaenoic acid), ALA (alphalinoleic acid), DHA (docosahexaenoic acid) would prevent inflammatory mediated atherogenesis. However, no trials conducted so far have shown to prevent pre-eclampsia.
- **L-Arginine**: Nitric oxide is a potent vasodilator produced from L-arginine by endothelial cells. Supplementation of L-Arginine has not shown to prevent pre-eclampsia.

2. **Exercise**

Exercise and physical activity have shown to prevent hypertension in non-pregnant women though studies have not shown to be of proven benefit in pre-eclampsia (27).

3. **Anti-Thrombotic Drugs**

Aspirin, being an anti-platelet aggregator, improves the blood flow by preventing the formation of micro-thrombi within the blood vessels. A large RCT '*Collaborative Low Dose Aspirin Study in Pregnancy*' (CLASP trial) (28) shows non-significant reduction of 12% in pre-eclampsia, but there was a significant reduction of proteinuric pre-eclampsia in women prone to develop early onset pre-eclampsia. A recent meta-analysis shows earlier the aspirin started in pregnancy, greater was the benefit in reducing pre-eclampsia. NICE recommends 75 mg of aspirin daily for all women who are at high risk for pre-eclampsia from 12 weeks until birth of the baby (29).

4. **Anti-Oxidants**

A small RCT using vitamin C and E in women at 20 weeks of gestation shows significant decrease in incidence of pre-eclampsia in treated group. However, a second study does not show a significant difference.

INDICATORS OF SEVERITY OF PRE-ECLAMPSIA

Abnormality	**Non-Severe Pre-Eclampsia**	**Severe Pre-Eclampsia (30)**
Systolic BP	140 to <160 mmHg	≥160 mmHg
Diastolic BP	90 to <110 mmHg	≥110 mmHg
Proteinuria	Persistent 1+ to 2+ on dipstick, >300 mg to <5 gm in 24-hr	3+ or more on dipstick, >5 gm in 24-hr
Headache	Absent	Present
Visual Disturbances	Absent	Present
Upper Abdominal Pain	Absent	Present
Convulsions	Absent	Present
Oliguria	Absent	Present
Serum Creatinine	Normal	Elevated (>1.2 mg/dL)
Serum Transaminases	Minimal Elevation	Marked Elevation
Thrombocytopenia (<1 lakh/µL)	Absent	Present
IUGR	Absent	Obvious
Pulmonary edema	Absent	Present

MANAGEMENT OF PRE-ECLAMPSIA

The only effective definitive treatment of pre-eclampsia is delivery**.** Although delivery is always beneficial for the mother, it may not be optimal for the premature fetus. Once the diagnosis of pre-eclampsia is made, the management depends upon the following:

- Severity of pre-eclampsia
- Gestational age of the fetus
- Maternal and fetal status at the time of initial evaluation
- Presence or absence of labor
- Level of specialized neonatal services

MILD PRE-ECLAMPSIA

The recent NICE guidelines (5) suggest that all patients initially diagnosed with pre-eclampsia must be admitted to the hospital for evaluation. The first step in the management is assessment of gestational age. The goal of management in mild pre-eclampsia is early detection of progression to severe pre-eclampsia and organ dysfunction. Use of anti-hypertensive drugs in mild pre-eclampsia is questionable. NICE clinical guidelines suggest treating moderate hypertension (BP 150-159/100-109 mmHg) with anti-hypertensives to keep BP <150/80-100 mmHg range. First-line anti-hypertensive in this situation is labetalol, given orally in doses of 100-400 mg every 8-12 hrs. Alternatives are methyl-dopa or nifedipine.

SEVERE PRE-ECLAMPSIA

The management of severe pre-eclampsia depends upon the gestational age.

- <24 weeks – Seizure prevention, BP control, and immediate delivery.

- 25-33 weeks – Expectant management by maternal and fetal surveillance, ante-natal steroid therapy for fetal lung maturity, deliver if there is maternal and fetal indication.
- >34 weeks – Seizure prevention, BP control, immediate delivery.

MATERNAL AND FETAL SURVEILLANCE

The table below summarizes the maternal and fetal surveillance methodology to be followed.

Maternal Surveillance	Fetal Surveillance
• BP monitoring 4 times/day • Daily input and output • Urine albumin once a day, 24-hr urinary protein • Daily maternal weight • Platelet count, RFT, LFT bi-weekly • Coagulation profile • Anti-hypertensive treatment, steroids • Questioning for imminent symptoms • Fundoscopy	• Daily fetal kick count • Non-stress test bi-weekly • Biophysical profile bi-weekly • Ultrasonography (USG) to assess gestational age and fetal growth every 2 weeks • Umbilical artery and Middle cerebral artery doppler bi-weekly

TERMINATION OF PREGNANCY

The table below summarizes the maternal and fetal indications for termination of pregnancy to deliver the baby.

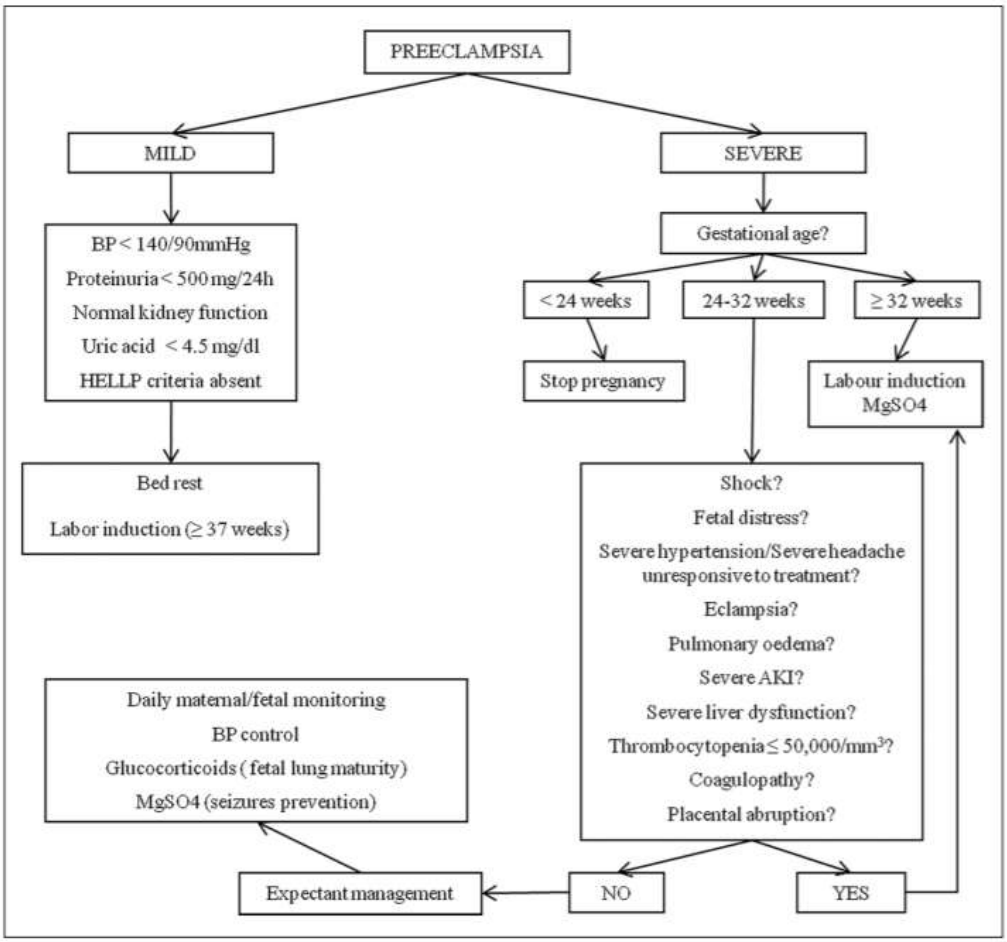
Maternal Indications for Termination	Fetal Indications for Termination
• Persistent severe headache • Visual disturbances • Eclampsia • Shortness of breath with rales, SpO_2 <94% at room air, signs of pulmonary edema • Uncontrolled severe hypertension despite treatment • Persistent platelet count <1 lakh/µL • Oliguria <500 mL in 24-hr • Serum creatinine ≥1.2 mg/dL • Suspected abruption, progressive labor, or ruptured membranes	• Severe Growth Restriction <5 percentile for Gestational Age • Reverse or End Diastolic Flow in Umbilical Artery Doppler • Persistent Severe Oligohydramnios (AFI <5 cm) • Biophysical profile ≤ 4 done 6 hrs apart • Fetal death.

The preferred mode of delivery in pre-eclampsia is vaginal delivery. Caesarean section is indicated in cases of,

- Fetal distress or malpresentation
- Placental abruption or placenta praevia
- Condition of the patient deteriorates

In case of severe pre-eclampsia remote from term, C-section is better than vaginal due to prolonged and unsuccessful induction and fetal compromise.

The diagram below summarizes the plan for management of patients in pre-eclampsia



INTRA-PARTUM MANAGEMENT

- Hourly monitoring of BP to maintain systolic BP <160 mmHg, and diastolic BP <110 mmHg with anti-hypertensives.
- Hourly urine output monitoring
- Monitoring signs & symptoms of impending eclampsia
- Eclampsia prophylaxis for those patients with impending eclampsia
- Continuous fetal heart rate monitoring
- Adequate pain relief during labor which cuts down catecholamine release and hypertensive response. Epidural analgesia is preferred as it is effective in controlling BP and maintaining cerebral blood flow (31).

- If C-section becomes unavoidable, epidural anesthesia avoids the risk of aspiration and difficult intubation due to airway edema
- Do not overload with IV fluids
- Third stage should be managed with oxytocin 10 U IM to prevent post-partum hemorrhage. Ergometrine should not be given as it causes intense vasoconstriction that may lead to hypertensive crisis.

ANTI-HYPERTENSIVE THERAPY

Anti-hypertensive therapy to a pregnant woman results in exposure of fetus to these drugs, hence the potential short-term maternal benefits must be balanced against possible short and long-term benefits and the risks to the fetus. There is lack of agreement regarding the blood pressure levels at which to initiate anti-hypertensive therapy. Initiation of anti-hypertensive therapy for systolic BP ≥160 mmHg or diastolic BP ≥110 mmHg is a universally accepted and followed norm.

According to NICE guidelines (5), anti-hypertensive treatment for non-severe hypertension (140-159/90-109 mmHg) should target maintenance of systolic BP at 130-155 mmHg and diastolic at 80-105 mmHg in a woman without any co-morbid conditions. In those with co-morbidities, anti-hypertensive drug therapy should be used to keep systolic BP at 130-139 mmHg and diastolic BP at 80-89 mmHg (31).

1. **First Line Oral Anti-Hypertensive Drugs**

- Alpha- and beta-blocker – labetalol
- Calcium channel blocker – nifedipine
- Beta-blockers – metoprolol, acebutolol, propranolol

Drugs that are absolutely contraindicated in pregnancy are Angiotensin Converting Enzymes (ACE) inhibitors due to their teratogenicity. In-utero exposure of fetus to these drugs may cause growth restriction, oligohydramnios, hypotension, anuria, and limb contractures.

Oral Preparations

a. **Labetalol**

It has both alpha- and beta-receptor blocking actions (ratio being 1:3 in oral formulation and 1:7 in IV formulation). It is becoming the first-line drug because of its high effectiveness and low incidence of side effects. Beta-blockers are associated with IUGR, hypoglycemia and hyperbilirubinemia in the fetus, and are hence not recommended.

Dosage: 100-400 mg every 8-12 hrs. Maximum dose upto 2400 mg/day

Side Effects:

- Bradycardia
- Hypotension
- Dizziness
- Nausea, Vomiting
- Insomnia
- Fatigue
- Depression
- Masks the symptoms of hypoglycemia in diabetic patients on insulin

It should be cautiously used in asthmatics and patients with heart failure due to its beta-blocking action.

b. **Alpha Methyl-Dopa**

It is a centrally acting alpha-adrenergic agonist which acts primarily on the central nervous system with some peripheral effect to stimulate the alpha-2 receptors. This decreases sympathetic tone and arterial BP.

Dosage: 250-500 mg BD/TDS. Maximum dose upto 2 gm/day.

Onset of action starts within 4-6 hrs and persists for 10-12 hrs.

Side Effects:

- Postural hypotension
- Headache
- Dry mouth
- Peripheral edema
- Depression
- Rarely, Haemolytic anaemia, and
- Drug-induced hepatitis

It crosses placenta and is secreted in breast milk, but no teratogenicity has been reported till date.

c. **Calcium Channel Blockers**

Nifedipine is the most commonly used calcium channel blocker for acute hypertension which is orally/sublingually effective, and easy to administer as well as store. Oral route is preferred because of the precipitous fall in blood pressure with sublingual route.

Dosage: 10-20 mg BD/TDS. Maximum dose upto 180 mg/day

Onset of action within 10-15 mins. BP is monitored every 15 mins and repeat oral dose of 10 mg can be administered every 30-60 mins until adequate response is achieved.

Caution is advised if using nifedipine with magnesium sulphate which will result in exaggerated hypotension because of their synergistic action in blocking calcium channels and there is a high chance of PPH.

Side Effects:

- Headache
- Dizziness
- Flushing
- Palpitation
- Heartburn
- Nasal congestion
- Hypotension
- Ankle edema

d. **Diuretics**

The main indications for diuretic use in pregnancy are as follows:

- Congestive cardiac failure
- Acute pulmonary edema
- Cerebral intra-cranial tension
- Renal failure

Diuretics cause depletion of intravascular volume which can be deleterious in cases of pre-eclampsia which already has a contracted plasma volume. Since the placenta does not have any autoregulatory mechanism, placental perfusion is directly linked to the systemic pressure. So, the reduction of plasma volume will result in reduced utero-placental flow and placental insufficiency. Hence, diuretic use is not recommended in

cases of pre-eclampsia with IUGR or Doppler evidence of reduced utero-placental perfusion.

Intravenous Preparations

These are used in hypertensive emergencies with systolic BP >160 mmHg, and diastolic BP >110 mmHg to avoid complications like cerebro-vascular hemorrhage, cardiac failure, placental abruption, eclampsia, and hypertensive encephalopathy.

a. **Labetalol**

It lowers the BP smoothly and rapidly without tachycardia, which occurs characteristically with hydralazine.

Dosage: Initial dose of 10-20 mg IV followed by 20-30 mg every 30 mins to a maximum dose of 220 mg/day. Alternatively, for continuous IV use, 500 mg of labetalol is added to 400 mL of normal saline and administered at the initial rate of 20 mg/hr. If the BP does not fall into the expected range (systolic <160, diastolic 80-95 mmHg) in 20 mins, the dose is continued to be doubled every 20 mins until expected range is obtained.

Onset of action is within 5 mins and peak effect within 10-20 mins.

Side Effects: Neonatal bradycardia, maternal hypotension and bradycardia.

b. **Hydralazine**

It acts directly on arteriolar smooth muscle to reduce peripheral vascular resistance. It can be given as intermittent bolus of 5 mg IV every 20-30 mins or as an infusion at a rate of 0.5-10 mg/hr upto a maximum dose of 30 mg/day.

Onset of action is within 10 mins. It initially causes increase in intracranial pressure by dilating the capacitance vessels in the cerebral circulation resulting in headache which

mimics impending eclampsia. Subsequently, cerebral resistance vessels dilate and cerebral vascular flow increases. The sympathetic effect of hydralazine causes marked tachycardia due to increased cardiac output.

Side Effects (32):

- Anxiety, restlessness, hyper-reflexia (mimics impending eclampsia)
- Hypotension
- Abruption
- Oliguria
- Adverse effects on fetal heart rate
- Low APGAR at 1 min

Hydralazine releases nor-adrenaline which is a potent vasoconstrictor of utero-placental circulation. Hence, continuous fetal heart rate monitoring is essential. Abnormal fetal heart rate patterns can be prevented by correction of hypovolemia and intermittent use of small doses.

c. **Sodium Nitroprusside**

It is a short acting vasodilator of both arterial and venous smooth muscle. It is given in refractory severe hypertension as an IV infusion, started at the rate of 0.25 mcg/kg/min and increased upto a maximum dose of 8 mcg/kg/min.

Onset of action is immediate (< 1 min) and the duration of action is 1-3 mins.

It causes cyanide toxicity to the fetus.

ECLAMPSIA

Development of seizures that cannot be attributed to other causes or unexplained coma during pregnancy or puerperium in a woman with pre-eclampsia. It can occur during

ante-partum (38-53%), intra-partum (18-36%), post-partum (11-44%), or late post-partum. It occurs more common in teenage primigravida. Eclamptic Seizures are self-limiting and last for 3-4 mins. It includes 4 stages:

- Premonitory stage
- Tonic stage
- Clonic stage
- Coma

PATHOPHYSIOLOGY

Seizures occurs as a result of abnormal autoregulatory response which consists of exaggerated vasoconstriction and ischemic changes with rupture of vascular endothelium and peri-capillary hemorrhages with development of foci of abnormal electrical discharges that generalize and cause convulsions. Autopsy finding shows cerebral edema, cortical and subcortical white matter microinfarcts, peri-capillary parenchymal bleeding and vascular bleeding in predominantly occipital and watershed areas.

MATERNAL AND FETAL COMPLICATIONS

The table below summarizes the maternal and fetal complications as a result of eclampsia.

Maternal Complications	Fetal Complications
• HELLP (9.7-20%) • DIC (7-11%) • Placental abruption (7-10%) • Acute renal failure (5-9%)	• Fetal distress • Hypoxic ischaemic encephalopathy (HIE) • Intrauterine death

• Pulmonary edema (3-5%) • Aspiration pneumonia (2-3%) • Cerebral hemorrhage, cardio-pulmonary arrest (2-5%).	

The most common cause of maternal death is intra-cranial bleeding and acute renal failure secondary to placental abruption. The most common cause of peri-natal death is due to prematurity and fetal asphyxia.

MANAGEMENT OF ECLAMPSIA:

The management of patients with eclampsia includes:

- Control of convulsions
- Control of hypertension
- Delivery of fetus

a. **Control of Convulsions**

- Call for help, insert 2 IV lines
- Put the patient in left lateral position and clearing of airway by suctioning to prevent aspiration
- Elevate the bedside rails to prevent maternal injury
- Give O_2 by mask at 8-10 L/min
- Pulse oximeter to monitor hypoxemia

$MgSO_4$ is the drug of choice in treatment of eclampsia.

MAGNESIUM SULPHATE

It has central anti-convulsant and neuroprotective effect.

PRITCHARD'S REGIMEN (33)

- **Intravenous loading dose:** Give 20 mL of 20% $MgSO_4$ (4 gm) slow IV for 20 mins.
- **Intramuscular loading dose:** Give 10 ml of 50% MgSO4 (5 gm) deep IM in upper outer quadrant of each buttock using 3 inch and 20G needle.
- **Maintenance dose:** Give 5 gm of $MgSO_4$ (10 mL of 50% solution) deep IM in alternate buttock every 4 hrs.

MONITORING FOR MAGNESIUM TOXICITY

Therapeutic range of $MgSO_4$ is 4-7 mEq/L. First sign of toxicity is loss of patellar reflex.

- Urine output should be at least 30 mL/hr or 100 mL/4 hrs
- Deep tendon reflexes should be present
- Respiratory rate should be >14/min
- Pulse oximetry should be ≥96%
- $MgSO_4$ is discontinued 24 hrs post-delivery or after last convulsion

In case of $MgSO_4$ toxicity, calcium gluconate 1 gm (10 mL of 10% solution) slow IV is the recommended treatment.

b. **Control of Hypertension**

Persistent and severe elevation in BP should be treated with parenteral anti-hypertensives to prevent cerebro-vascular accidents, pulmonary edema, and renal failure.

c. **Delivery of Fetus**

The definitive treatment is delivery of the fetus, irrespective of gestational age, in eclampsia. Patient must deliver within 24 hrs in severe pre-eclampsia and within 12 hrs

in eclampsia. If the patient is stable, vaginal examination is done to assess the cervical status. If the cervix is favorable, induction of labor/amniotomy and oxytocin acceleration is done. If unfavorable cervix, LSCS is the only option available.

HELLP SYNDROME

It is an acronym coined by Louis Weinstein denoting

- Hemolysis
- Elevated Liver enzymes
- Low Platelets

It contributes 0.2-0.6 % of all pregnancies and 10-20 % in severe pre-eclampsia.

a. **Hemolysis**

- Abnormal peripheral blood smear (Burr cells, schistocytes)
- Elevated bilirubin >1.2 mg/dL
- Low serum haptoglobin
- Increased LDH (twice the upper limit of normal >600 U/L)

b. **Elevated Liver Enzymes**

Elevated AST and ALT, twice the upper limit of normal (≥72 U/L)

c. **Low Platelet Count**

Platelet count <100,000/µL

MISSISIPPI CLASSIFICATION (34) FOR THROMBOCYTOPENIA

CLASS I (severe thrombocytopenia) – Platelet count <50,000/µL

CLASS II (moderate thrombocytopenia) – Platelet count 50,000/µL to 100,000/µL

CLASS III (mild thrombocytopenia) – Platelet count 100,000/µL to 150,000/µL

TENNESSEE CLASSIFICATION

Complete HELLP – All 3 parameters are abnormal

Incomplete/Partial HELLP – When 1 or 2 parameters are abnormal

Differential Diagnosis:

Differential diagnoses for HELLP syndrome include:

- Acute fatty liver of pregnancy
- Thrombotic Thrombocytopenic Purpura
- Hemolytic Uremic Syndrome

MATERNAL MORBIDITY IN HELLP SYNDROME

- Abruptio Placenta (10-15%)
- DIC (10-15%)
- Subcapsular liver hematoma
- Hepatic rupture (severe complication)
- Pulmonary edema (6-8%)
- Acute renal failure (5-8%)
- ARDS (1-2%)
- Death (~1%)

Women with a history of HELLP syndrome carry an increased risk (approximately 20%) of developing some form of GHT in subsequent pregnancies (35).

MANAGEMENT OF HELLP

Termination of pregnancy is the treatment of choice. When gestational age is >34 weeks, stabilize the condition and deliver the baby. When gestational age is between 27-34 weeks, ante-natal steroids for fetal maturity are administered followed by

delivery of the baby. The potential benefits must be weighed against the risks of expectant management which include abruptio placenta, acute renal failure, pulmonary edema, DIC, perinatal and maternal death. High dose corticosteroids have been proposed to improve maternal prognosis of HELLP. In a recent Cochrane review comparing corticosteroids with placebo, there was no difference in the risk of maternal death. The only clear effect on individual was improved platelet count. Follow-up is done with measurement of platelet count and LDH, which becomes normal within 72 hrs following delivery. Other treatment modalities include platelet transfusion and plasmapheresis.

URIC ACID

Uric acid is the end-product of purine metabolism. Its level is elevated in pre-eclampsia due to its decreased renal clearance or by its increased production by breakdown of purines in placenta. Decreased renal clearance is due to altered renal tubular function.

This uric acid impairs the generation of nitric oxide from endothelial cells causing endothelial cell dysfunction which is the main pathophysiology in pre-eclampsia. Hence it is used as the predictive marker for pre-eclampsia. Various studies have been done on uric acid levels in pre-eclamptic patients and correlation with severity of disease, maternal and fetal complications. Some studies are as follows.

1. A Study was done by Disha et. al. (36) conducted at the Civil Hospital, Ahmedabad in 2012, where 80 Hypertensive women were selected randomly and retrospectively studied. Birth weight, Gestational age at delivery, complications like HELLP, eclampsia, IUD, abnormality in platelet count, creatinine, bilirubin were noted. Groups were divided into 2 based on uric acid levels as <6 mg/dL and >6 mg/dL

and all variables correlated. In this study, increase in maternal and fetal complications were noted in group with uric acid >6mg/dL (p <0.05). Mean gestational age at delivery and mean birth weight were reduced in group with uric acid >6 mg/dL.

2. A prospective study by Sreelatha et. al. (37) conducted in the department of obstetrics and gynecology, ESI Post-Graduate Institute of Medical Science and Research, Bangalore from Jan to Aug 2014, in which 80 pregnant women were included (mild and severe GHT). Uric acid and LDH were estimated. The women were followed up till delivery and early post-partum. Gestational age at delivery, birth weight, mode of delivery, maternal and fetal complications were noted. Significant association was found between LDH and uric acid with severity of pre-eclampsia, maternal morbidity, birth weight of babies. Gestational age at delivery and neonatal outcome was not statistically significant in this study.
3. A prospective study by Patel et. al. (1) conducted in the department of obstetrics and gynecology, BJ medical college and hospital, Ahmedabad, Gujarat, in 2014 on two groups of 50 women each with HDP. The first group comprising of 50 patients with serum uric acid level of ≥6 mg/dL was compared to the second group of 50 patients with serum uric acid level of <6 mg/dL. Maternal and fetal complications like eclampsia, HELLP syndrome, ARF, IUD, low Apgar score, and IUGR were studied. The comparison between the two groups revealed that hyperuricemia in patients with HDP was a strong risk factor for several maternal and fetal complications with an increased risk of an Apgar score of <7 by 6-fold, IUFD by 20-fold, IUGR by 4-

fold, eclampsia by 4.2-fold, and C-section by 3.4-fold in the group with serum uric acid level of ≥6 mg/dL.

4. A prospective clinical observational study by Nair et. al. (38) conducted in 2017 in the department of obstetrics and gynecology, Vydehi institute of medical sciences and research institute, Bangalore, 50 pregnant women with severe pre-eclampsia and 50 normotensive women were included and maternal serum uric acid level was estimated in both groups to evaluate severity of pre-eclampsia with raised uric acid level and perinatal outcome. The author concluded that there was a positive correlation between serum uric acid level and severity of pre-eclampsia, and a significant adverse fetal outcome was observed with raised maternal serum uric acid level in pre-eclamptic patients.

AIM AND OBJECTIVES

AIM:

The study of serum uric acid as a biochemical indicator for maternal and fetal outcome in patients with gestational hypertension.

OBJECTIVES:

1. To study serum uric acid levels in patients presenting with gestational hypertension, pre-eclampsia, or eclampsia.
2. To evaluate the relationship between serum uric acid and fetal outcome.
3. To understand the association between the levels of serum uric acid levels and severity of hypertension.
4. To identify a correlation between serum uric acid level elevation and adverse maternal and fetal outcomes.

MATERIALS AND METHODS

SETTING

Department of Obstetrics and Gynecology, Govt. Theni Medical College and Hospital, Tamil Nadu

STUDY PERIOD

June 2018 to June 2019

STUDY DESIGN

Prospective observational case study

STUDY POPULATION

200 pregnant women with GHT attending GTMCH

INCLUSION CRITERIA

All pregnant women suffering from GHT, pre-eclampsia, and eclampsia >20 weeks of gestation.

EXCLUSION CRITERIA:

1. Normotensive pregnant females
2. Patients with hypertension at <20 weeks of gestation (chronic hypertension)
3. Patients with chronic renal disease
4. Patients suffering from diabetes mellitus
5. Diagnosed patients of hepatic dysfunction
6. Patients suffering from gout
7. Patients with epilepsy
8. Patients with thyroid dysfunction

9. Patients with cardiac disease
10. Patients with leukemias
11. Patients with pancreatitis
12. Patients with hemolysis

METHODOLOGY

After obtaining informed written consent from all the study subjects or their attendants (in case of unconscious patients, or patients unable to provide consent due to their condition), relevant data were documented in a pre-defined datasheet. Maintaining all aseptic precautions, blood samples were collected from all subjects for estimation of serum uric acid concentration and other parameters.

ESTIMATION OF SERUM URIC ACID

Serum uric acid level estimation can be done by 2 methods.

- Colorimetric method (phototungstic method) or
- Enzymatic method (uricase method)

Enzymatic method is the most commonly used method and was also the method used for the present study. It involves oxidation of uric acid by uricase enzyme which convert the substrate to allantoin. It is quantified based on differential absorbance at 293 nm for these substances.

STATISTICAL ANALYSIS

Data were recorded on pre-printed proforma. The data were analysed using the Statistical Package for Social Sciences (IBM SPSS Inc.). Descriptive statistics including frequency, mean, median, minimum, maximum and standard deviation were calculated for the demographic data. Categorical variables were presented as percentages and continuous variables were presented as mean/median. The association between categorical variables was tested by using Chi-Square test. A *p*-value (two-tailed) of <0.05 was considered statistically significant.

OBSERVATIONS AND RESULTS

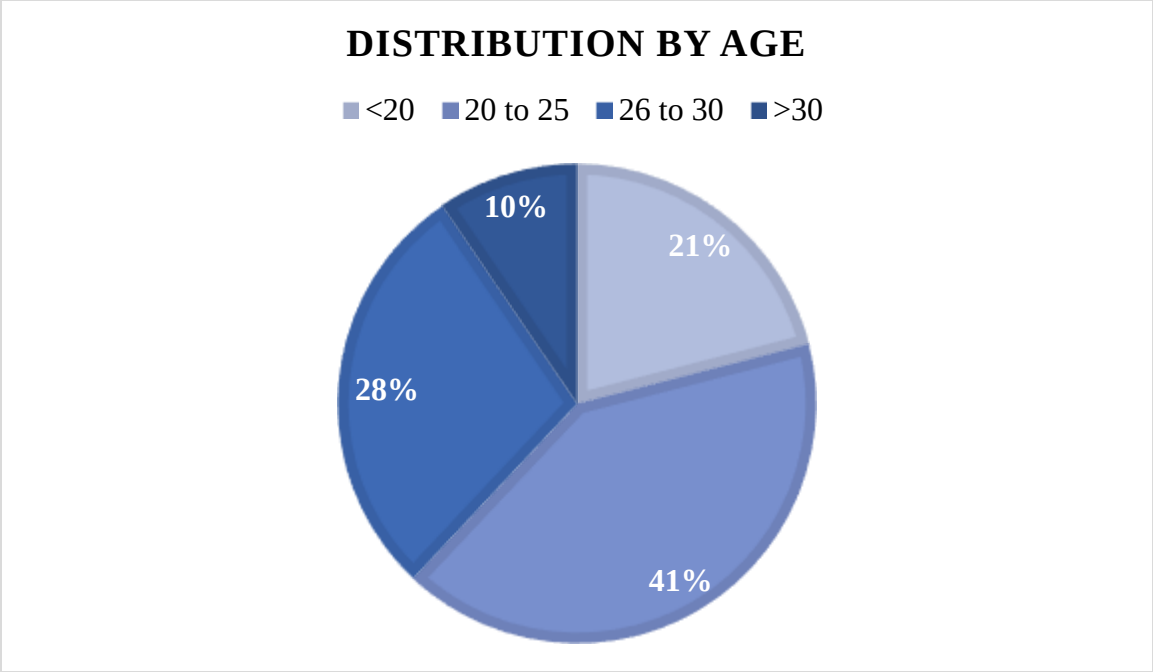
The present study was conducted on 200 pregnant females with clinically proven GHT, pre-eclampsia or eclampsia. The observations from our study were as follows:

General Characteristics of the Study Population

The 200 subjects enrolled to the study were between the age of 18 and 38 years and had GHT, pre-eclampsia, or eclampsia. The subjects were profiled based on their demographics, i.e. age, BMI, obstetric score, gestational age, etc. Details are presented below.

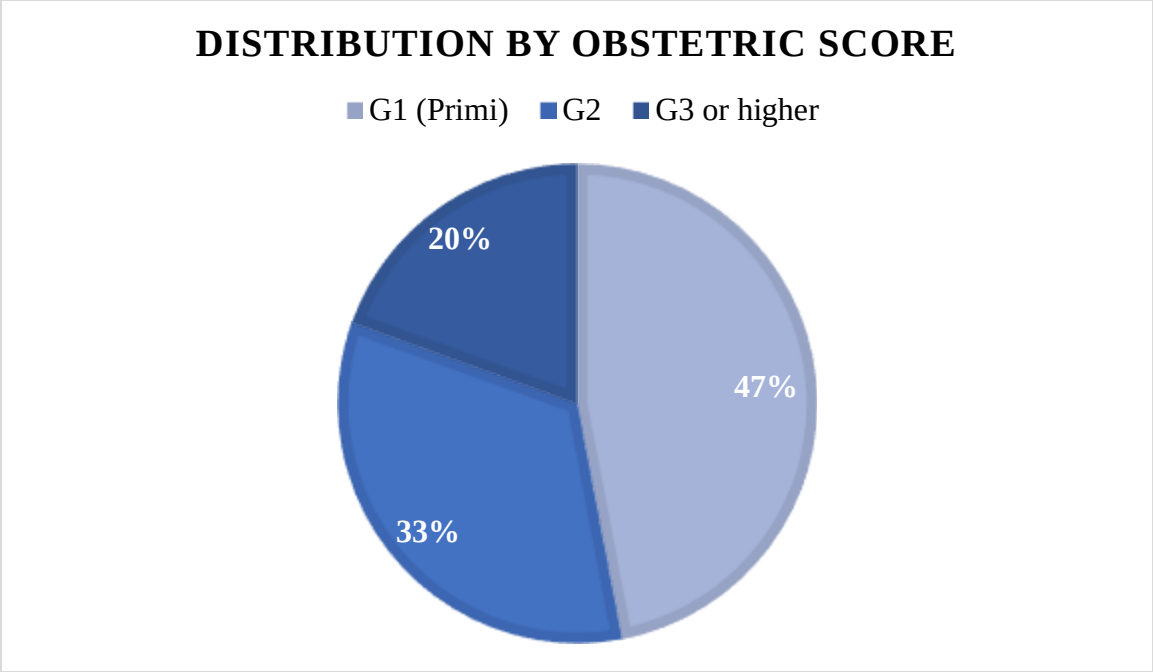
Distribution by Age: All 200 pregnant subjects were adults in the age range of 18 to 38 years with a mean age of 24.2 (SD ± 4.5) years and median of 24 years. The table below shows a frequency distribution of all subjects by age.

Age (Years)	Frequency (%)
<20	42 (21.0%)
20 to 25	82 (41.0%)
26 to 30	57 (28.5%)
>30	19 (9.5%)



Distribution by Parity: Of the 200 pregnant females, 94 (47%) were primigravida, 67 (33.5%) were gravida 2, 30 (15%) were gravida 3, while the remaining 9 (4.5%) were gravida 4 or higher. A frequency distribution is provided in table below.

Obstetric Score	Frequency (%)
G1 (Primi)	94 (47.0%)
G2	67 (33.5%)
G3 or higher	39 (19.5%)

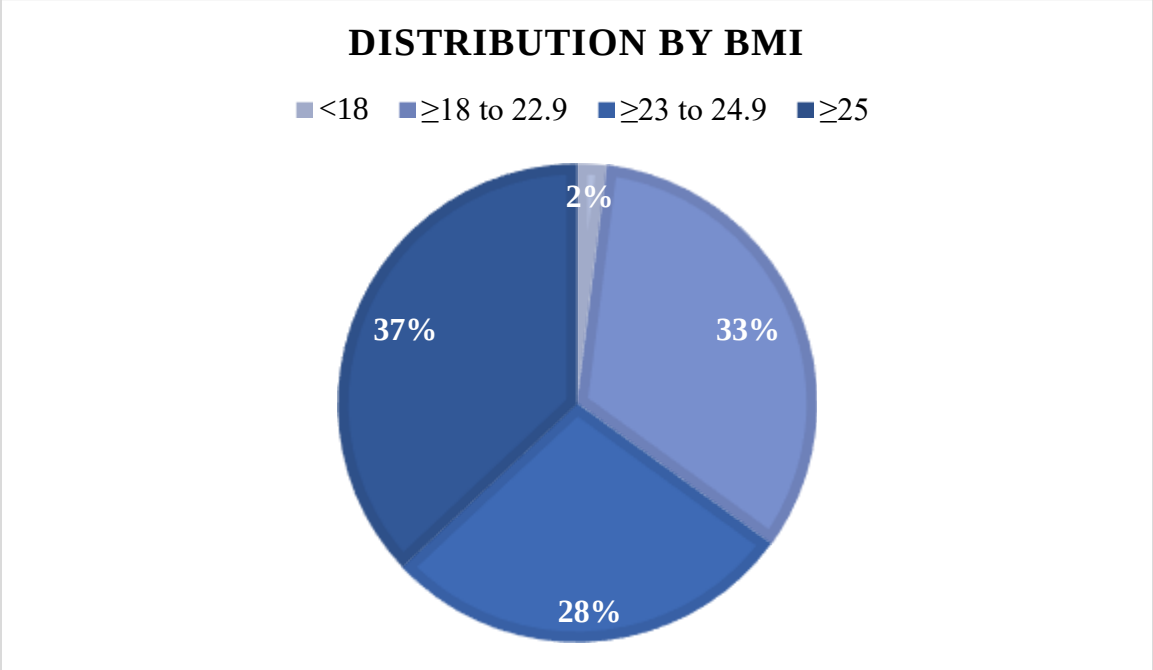


Distribution by Body Mass Index (BMI): The 200 subjects were also profiled on the basis of their height and weight at the time of presentation and diagnosis of GHT. Based on the weight and height measurements, the BMI was calculated, which is the ratio of weight in kilograms and square of the height in meters.

The mean BMI was 24.1 kg/m^2 (SD ± 3.3; BMI range was 16.2 to 43 kg/m^2) and median was 24.2 kg/m^2.

The subjects were categorized as per the Association of Physicians of India (API) classification on obesity, which is a slight modification of the WHO criteria applicable to the Indian population, and the frequency distribution is presented in table below.

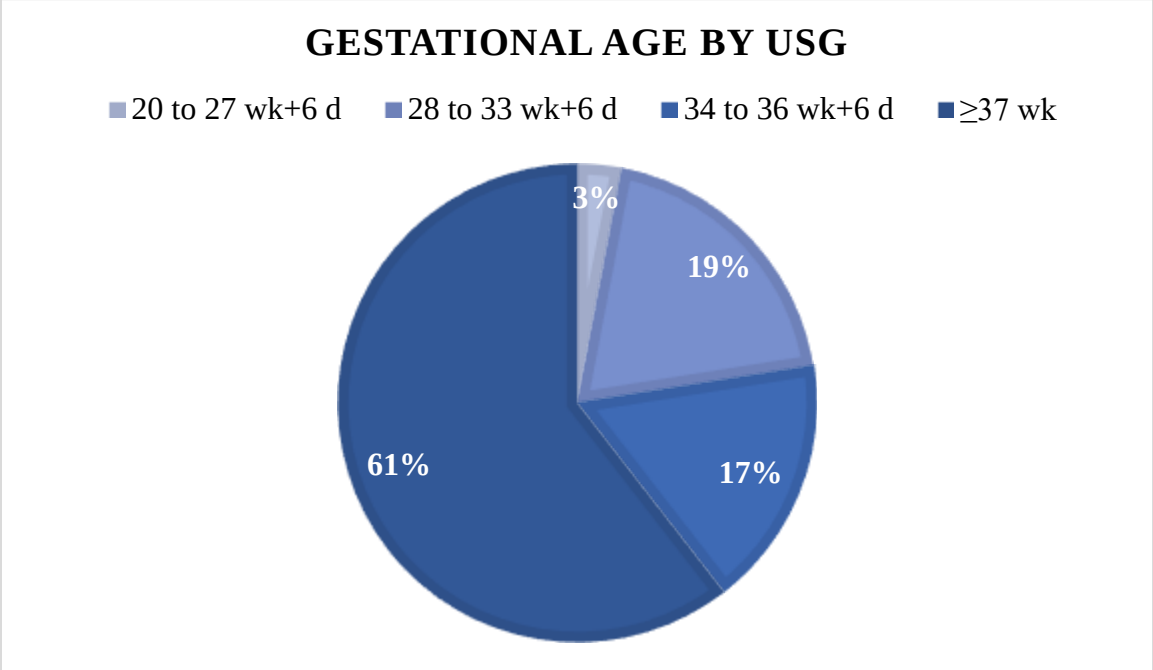
BMI	Frequency (%)
<18	04 (2%)
≥18 to 22.9	66 (33%)
≥23 to 24.9	56 (28%)
≥25	74 (37%)



Gestational Age as Determined by USG

The frequency distribution of the USG determined gestational age for the 200 subjects is presented in table below.

Gestational Age	Frequency (%)
20 to 27 wk+6 d	06 (3.0%)
28 to 33 wk+6 d	39 (19.5%)
34 to 36 wk+6 d	34 (17.0%)
≥37 weeks	121 (60.5%)

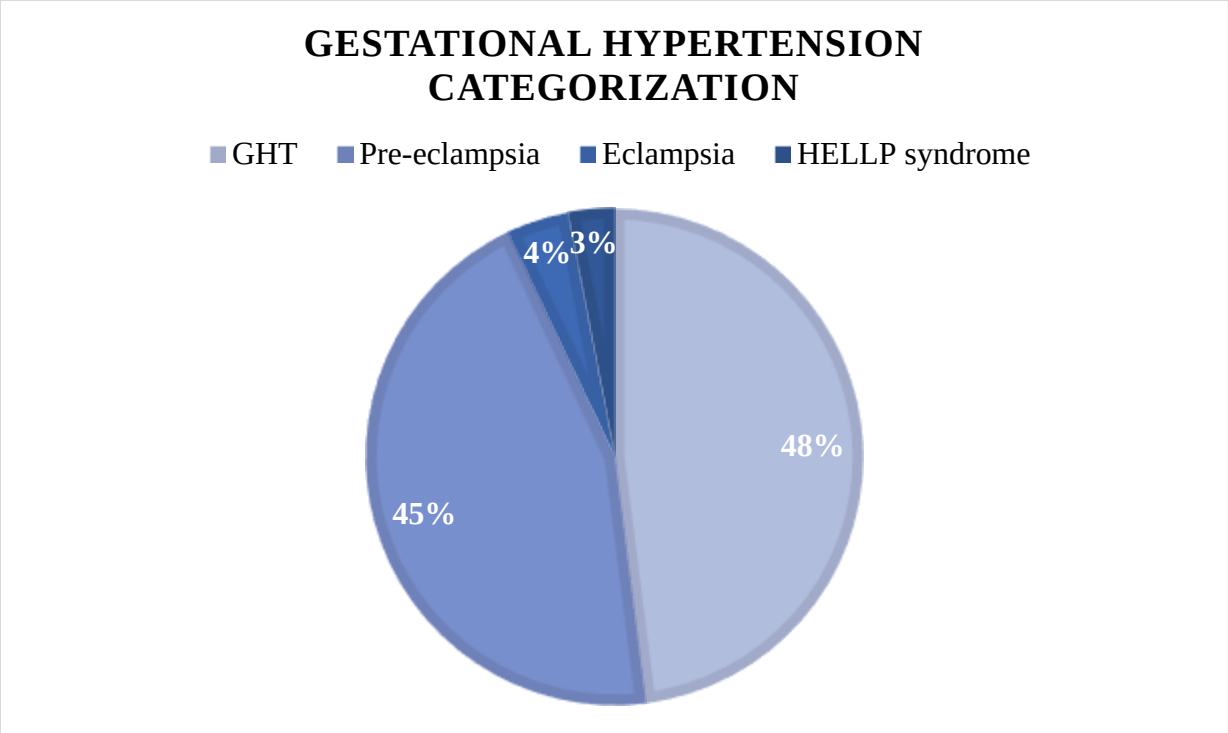


The earliest presentation of GHT in the present study was in the 25th week of gestation (24 week 5 days) while the latest presentation was in the 40th week of gestation (39 week 3 days).

Gestational Hypertension Categorization

The 200 subjects presenting to our hospital had GHT. Based on the diagnoses evoked, the subjects were categorized as per the table below.

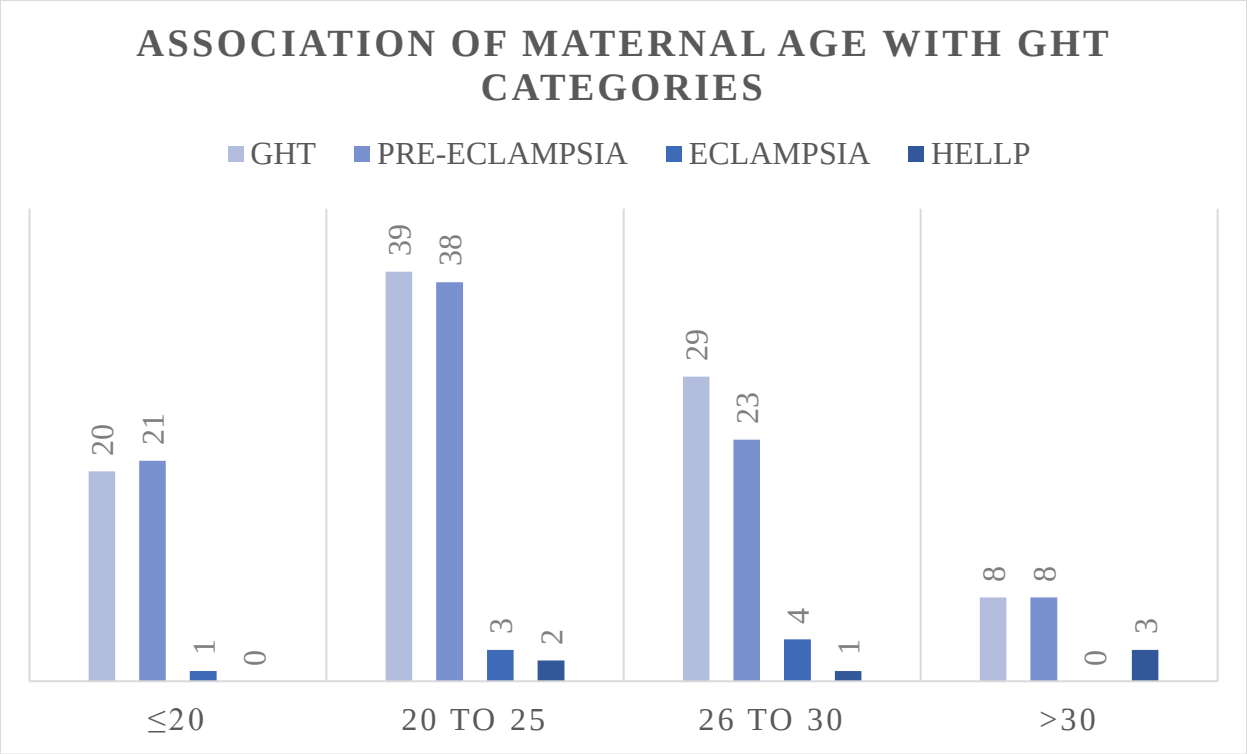
Diagnosis	Frequency (%)
GHT	**96 (48.0%)**
Pre-eclampsia	**90 (45.0%)**
Non-severe pre-eclampsia	47 (23.5%)
Severe pre-eclampsia	43 (21.5%)
Eclampsia	**08 (4.0%)**
HELLP syndrome	**06 (3.0%)**
Partial HELLP	01 (0.5%)
Complete HELLP	05 (2.5%)



The subjects enrolled to the study could be categorized into 4 broad categories: GHT, pre-eclampsia, eclampsia, and HELLP syndrome. Of the 200 subjects, 48% (96/200) had GHT without associated abnormalities. Ninety of the 200 subjects were diagnosed with pre-eclampsia: 47 (23.5%) had non-severe pre-eclampsia while 43 (21.5%) had severe pre-eclampsia. Eight of the 200 subjects (4%) developed eclampsia while the remaining 6 subjects (3%) were diagnosed with HELLP syndrome and were considered to be in imminent risk of eclampsia.

Association of Maternal Age with GHT Categories

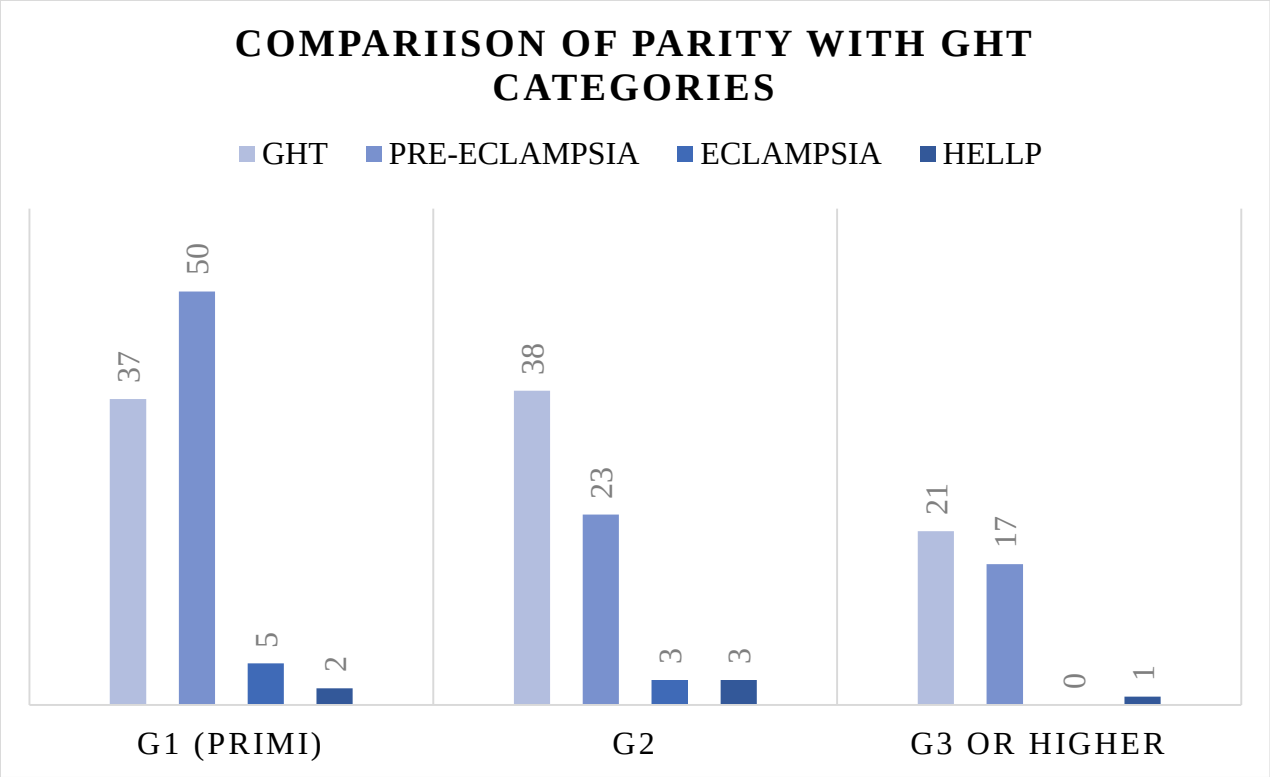
Maternal Age	GHT	Pre-Eclampsia		Eclampsia	HELLP
		Non-Severe	Severe		
≤20	20 (47.6%)	12 (28.6%)	09 (21.4%)	01 (2.4%)	00 (0.0%)
20 to 25	39 (47.6%)	16 (19.5%)	22 (26.8%)	03 (3.7%)	02 (2.4%)
26 to 30	29 (50.9%)	16 (28.1%)	07 (12.3%)	04 (7.0%)	01 (1.8%)
>30	08 (42.1%)	03 (15.8%)	05 (26.3%)	00 (0.0%)	03 (15.8%)
Total	**96**	**47**	**43**	**08**	**06**


ASSOCIATION OF MATERNAL AGE WITH GHT CATEGORIES
GHT
PRE-ECLAMPSIA
ECLAMPSIA
HELLP
20
21
1
0
39
38
3
2
29
23
4
1
8
8
0
3
≤20
20 TO 25
26 TO 30
>30

Of the 42 subjects aged <20 years, 47.6% (20/42) had GHT, 50% (21/42) had pre-eclampsia, 2.4% (1/42) had eclampsia. Of the 82 subjects aged 20 to 25 years, 47.6% (39/82) had GHT, 46.3% (38/82) had pre-eclampsia, 3.7% (3/82) had eclampsia, and 2.4% (2/82) had HELLP syndrome. Of the 57 subjects aged 26 to 30 years, 50.9% (29/57) had GHT, 40.4% (23/57) had pre-eclampsia, 7% (4/57) had eclampsia, and 1.8% (1/57) had HELLP syndrome. Of the subjects aged > 30 years, 42.1% (8/19) had GHT, 42.1% (8/19) had pre-eclampsia, and 15.8% (3/57) had HELLP syndrome. Using the chi-square test, *p*-value for association of maternal age with the GHT categories was 0.064, considered non-significant ($p > 0.05$), indicating no correlation between maternal age and development of GHT.

Association of Parity with GHT Categories

Parity	GHT	Pre-Eclampsia		Eclampsia	HELLP
		Non-Severe	Severe		
G1 (Primi)	37 (39.4%)	29 (30.9%)	21 (22.3%)	05 (5.3%)	02 (2.1%)
G2	38 (56.7%)	11 (16.4%)	12 (17.9%)	03 (4.5%)	03 (4.5%)
G3 or higher	21 (53.9%)	07 (18.0%)	10 (25.6%)	00 (0.0%)	01 (2.6%)
Total	**96**	**47**	**43**	**08**	**06**

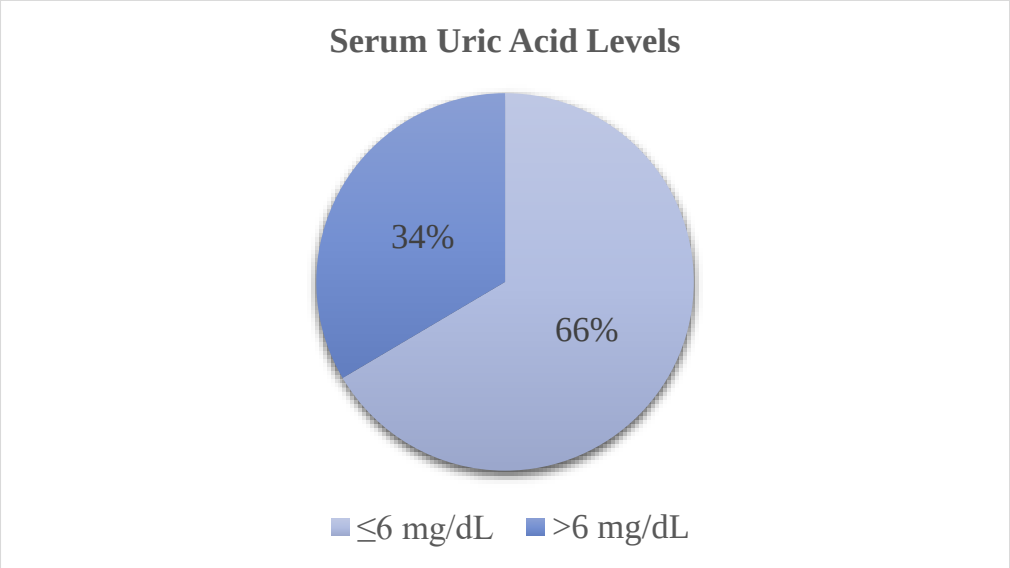

COMPARIISON OF PARITY WITH GHT CATEGORIES
GHT
PRE-ECLAMPSIA
ECLAMPSIA
HELLP
37
50
5
2
38
23
3
3
21
17
0
1
G1 (PRIMI)
G2
G3 OR HIGHER

Of the 200 subjects, 94 (47%) were primigravida, 67 (33.5%) were G2, and 39 (19.5%) were G3 or higher. Of the 94 primigravida subjects, 37 (39.4%) had GHT, 50 (53.2%) had pre-eclampsia, 5 (5.3%) had eclampsia, and 2 (2.1%) had HELLP syndrome. Of the 67 G2 subjects, 38 (56.7%) had GHT, 23 (34.3%) had pre-eclampsia, and 3 each (4.5%) had eclampsia and HELLP syndrome. Of the 39 subjects with G3 or higher parity, 21 (53.9%) had GHT, 17 (43.6%) had pre-eclampsia, none had eclampsia, and 1 (2.6%) had HELLP syndrome. Using the chi-square test, *p*-value for association of parity with the GHT categories was 0.238, considered non-significant ($p > 0.05$).

Serum Uric Acid Levels

Normal serum uric acid levels in females are 2.4 to 6 mg/dL. For the present study, we considered 6 mg/dL as the cut-off for normal versus elevated uric acid levels. The table below shows the frequency distribution of uric acid levels in the 200 study subjects.

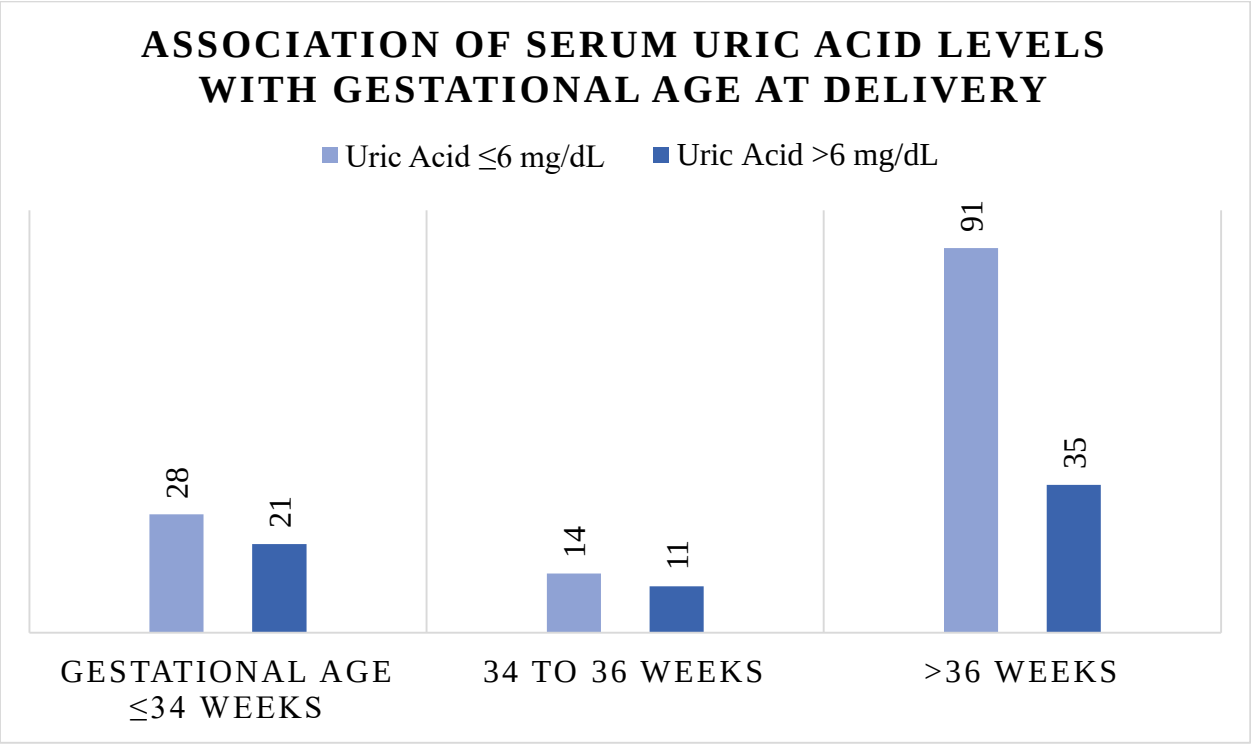
Uric Acid Level (mg/dL)	Frequency (%)
≤6	133 (66.5%)
>6	67 (33.5%)


Serum Uric Acid Levels
34%
66%
≤6 mg/dL
>6 mg/dL

Association of Uric Acid Levels with Gestational Age at Delivery

The table below represents frequency distribution according to gestational age at delivery with the serum uric acid levels (normal or elevated).

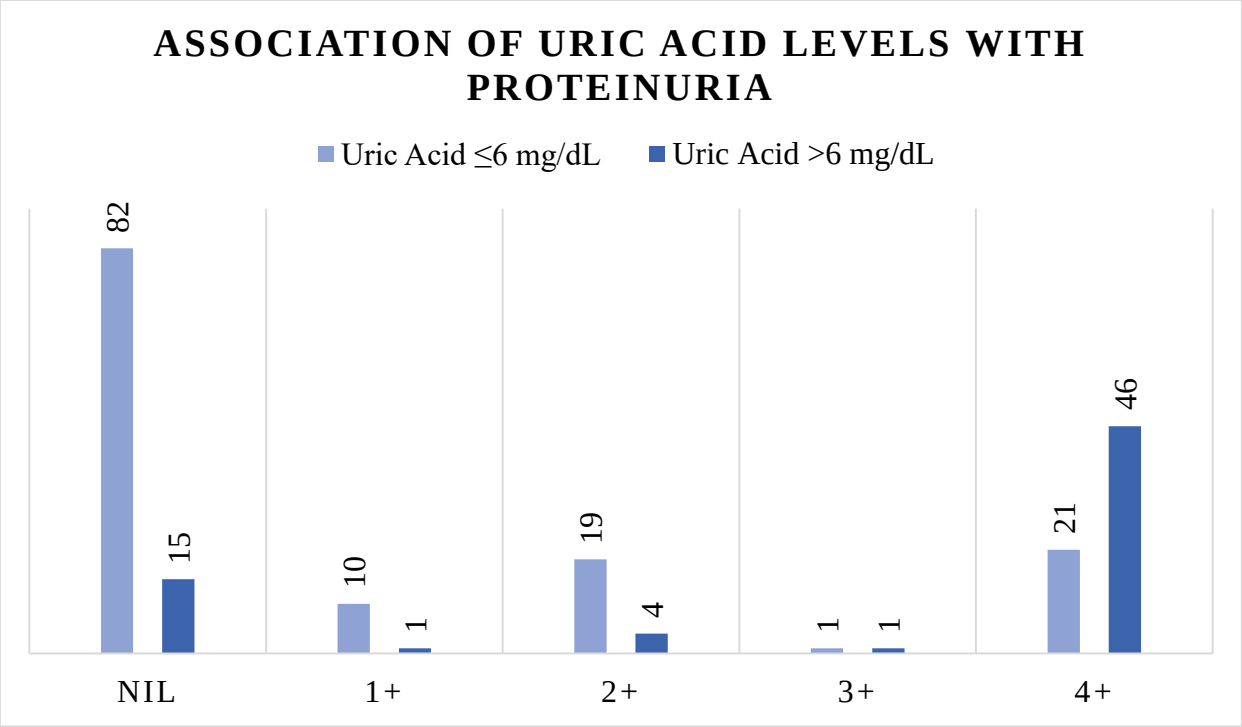
Gestational Age at Delivery (Weeks)	**Frequency (%) of Subjects with Uric Acid ≤6 mg/dL**	**Frequency (%) of Subjects with Uric Acid >6 mg/dL**
≤34	28 (21.1%)	21 (31.3%)
34 to 36	14 (10.5%)	11 (16.4%)
>36	91 (68.4%)	35 (52.2%)
Total	**133**	**67**


ASSOCIATION OF SERUM URIC ACID LEVELS WITH GESTATIONAL AGE AT DELIVERY
Uric Acid ≤6 mg/dL
Uric Acid >6 mg/dL
28
21
14
11
91
35
GESTATIONAL AGE ≤34 WEEKS
34 TO 36 WEEKS
>36 WEEKS

As evident from the table and graph above, pre-term delivery was more commonly seen in women with serum uric acid >6 mg/dL. To summarize the findings above, 31.3% subjects with elevated serum uric acid compared with 21.1% subjects with normal serum uric acid delivered at GA ≤34 weeks; 16.4% subjects with elevated serum uric acid compared with 10.5% subjects with normal serum uric acid delivered at GA 34 to 36 weeks. Using the chi-square test, *p*-value for association of uric acid levels with gestational age was 0.081, considered non-significant ($p > 0.05$).

Association of Uric Acid Levels with Proteinuria

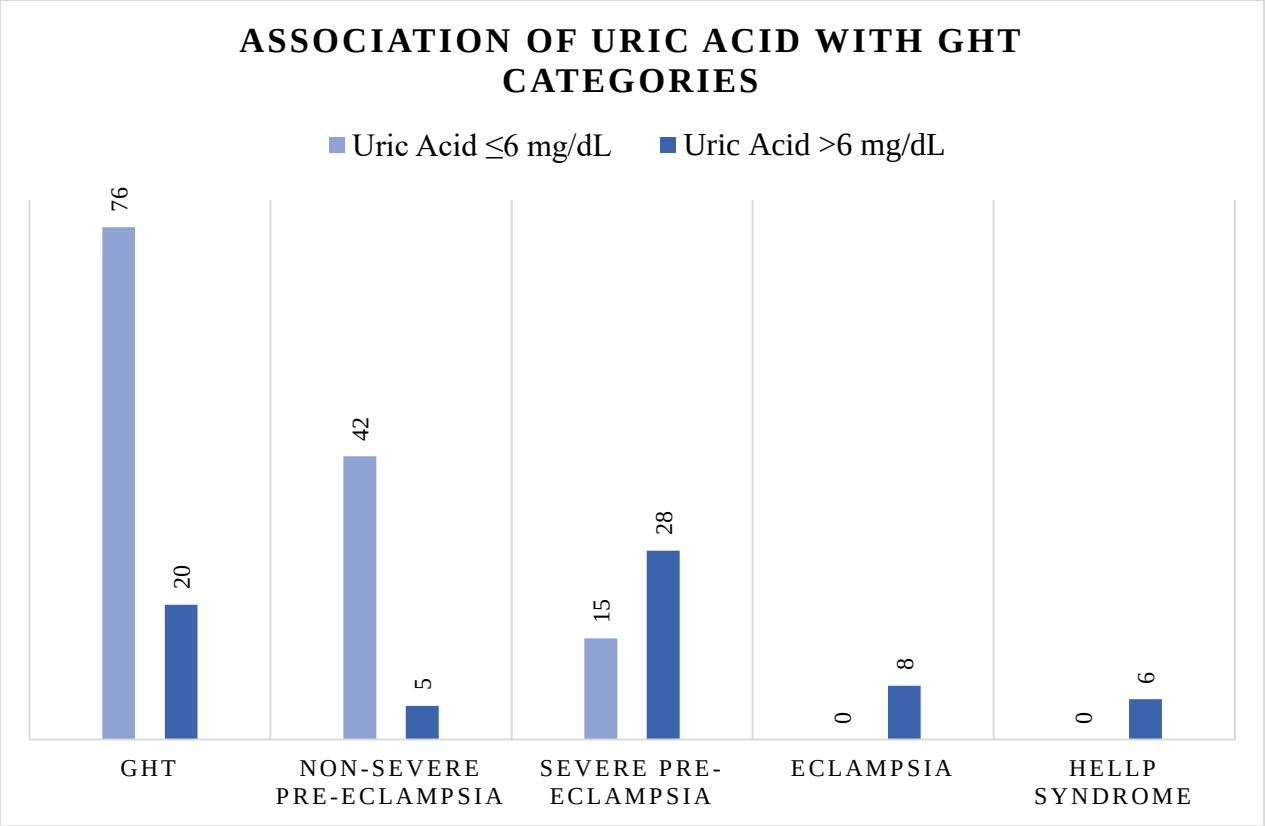
Proteinuria	Number of Subjects with Uric Acid ≤6 mg/dL	Number of Subjects with Uric Acid >6 mg/dL
Nil	82 (61.6%)	15 (22.4%)
1+	10 (7.5%)	01 (1.5%)
2+	19 (14.3%)	04 (6.0%)
3+	01 (0.8%)	01 (1.5%)
4+	21 (15.8%)	46 (68.6%)
Total	**133**	**67**



Using the chi-square test, *p*-value for association of uric acid levels with proteinuria was <0.001. Therefore, the association between uric acid levels with proteinuria was considered statistically significant ($p < 0.05$) indicating that the uric acid levels increase with increasing proteinuria.

Association of Uric Acid with GHT Categories

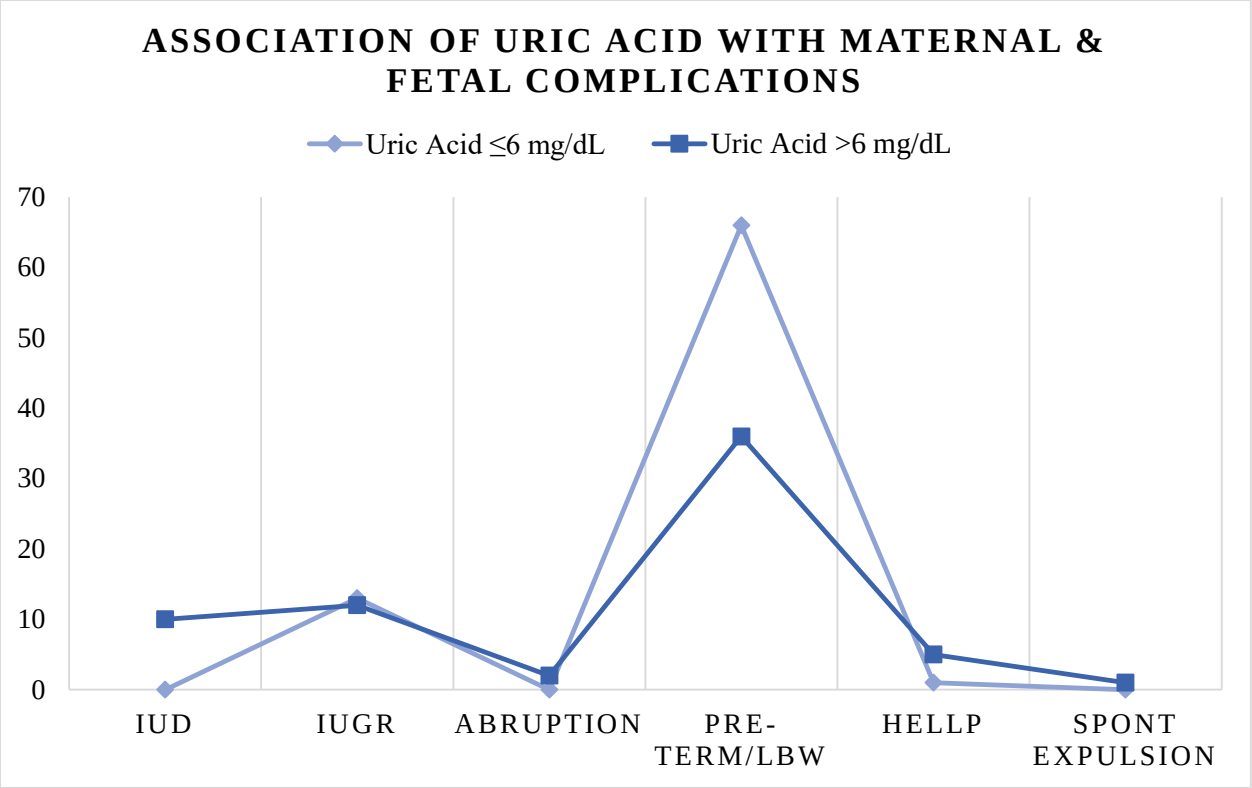
Category	**Frequency (%) of Subjects with Uric Acid ≤6 mg/dL**	**Frequency (%) of Subjects with Uric Acid >6 mg/dL**
GHT	76 (57.1%)	20 (29.9%)
Non-severe Pre-eclampsia	42 (31.6%)	05 (7.5%)
Severe Pre-eclampsia	15 (11.3%)	28 (41.8%)
Eclampsia	00 (0.0%)	08 (11.9%)
HELLP Syndrome	00 (0.0%)	06 (9.0%)
Total	**133**	**67**


ASSOCIATION OF URIC ACID WITH GHT CATEGORIES
Uric Acid ≤6 mg/dL
Uric Acid >6 mg/dL
76
20
42
5
15
28
0
8
0
6
GHT
NON-SEVERE PRE-ECLAMPSIA
SEVERE PRE-ECLAMPSIA
ECLAMPSIA
HELLP SYNDROME

In subjects with uric acid within normal range (≤6 mg/dL), 31.6% and 11.3% subjects had non-severe pre-eclampsia and severe pre-eclampsia, respectively, while none of the subjects had eclampsia or HELLP syndrome. On the contrary, in subjects with elevated uric acid (>6 mg/dL), 7.5% subjects had non-severe pre-eclampsia, 41.8% had severe pre-eclampsia, 11.9% had eclampsia, and 9% had HELLP syndrome. Using the chi-square test, *p*-value for association of uric acid levels with GHT categories was < 0.001, considered statistically significant.

Association of Uric Acid Levels with Maternal and Fetal Complications

Complications	**Number of Subjects with Uric Acid ≤6 mg/dL**	**Number of Subjects with Uric Acid >6 mg/dL**	**P-value**
Intra-Uterine Death	00	10	0.000008
Intra-Uterine Growth Retardation	13	12	0.12
Intra-cranial Hemorrhage	00	00	NA
Pulmonary Edema	00	00	NA
Abruption	00	02	0.046
Pre-Term/Low Birth Weight	66	36	0.70
HELLP Syndrome	01	05	0.0097
Spontaneous Expulsion	00	01	0.16
Maternal Death	00	00	NA
Total	**80**	**66**	


ASSOCIATION OF URIC ACID WITH MATERNAL & FETAL COMPLICATIONS
Uric Acid ≤6 mg/dL
Uric Acid >6 mg/dL
70
60
50
40
30
20
10
0
IUD
IUGR
ABRUPTION
PRE-TERM/LBW
HELLP
SPONT EXPULSION

There was a higher number of IUD in infants born to mothers with uric acid >6 mg/dL (statistically significant, $p < 0.001$). Almost equal number of infants developed IUGR to mothers irrespective of uric acid levels (statistically non-significant, $p > 0.05$). More infants were pre-term or with low birth weight in mothers with uric acid level <6 mg/dL (statistically non-significant, $p > 0.05$). Abruption occurred only in subjects with uric acid >6mg/dL (statistically significant, $p < 0.05$); HELLP syndrome occurred more frequently in mothers with uric acid level >6 mg/dL (statistically significant, $p < 0.05$); spontaneous expulsion occurred only in subject with uric acid >6 mg/dL (statistically non-significant, $p > 0.05$). No maternal mortality was seen in both the groups.

DISCUSSION

The etiopathogenesis of pre-eclampsia remains undetermined to date, and there is no screening test reliable enough to diagnose it. Uric acid is considered one of the most sensitive indicators of disease severity in GHT, and being a potent mediator of inflammation, it was thought to be of great indicative help in monitoring the cause of the underlying disease process. The most plausible explanation for increase in uric acid levels during third trimester of pregnancy was increased reabsorption and decreased excretion of uric acid. Hyperuricemia was a common finding in complicated pregnancies, such as pre-eclampsia and eclampsia. Therefore, our study was aimed at assessing the association of serum uric acid levels as a reliable predictor of maternal and perinatal outcomes in pregnant females with GHT.

The present study was conducted in the department of obstetrics and gynecology, Government Theni Medical College and Hospital. We recruited a total of 200 antenatal women from the outpatient department of the hospital between June 2018 and December 2018. All subjects were of gestational age 24 weeks and greater.

The subjects were all selected based on the inclusion criteria listed previously, irrespective of the maternal age and/or parity and were divided into 4 diagnostic groups: GHT (without pre-eclamptic symptoms), pre-eclampsia (non-severe and severe), eclampsia, and HELLP syndrome. There were no normotensive study subjects (as controls). Patients were also divided into 2 groups based on their serum uric acid levels:

uric acid ≤6 mg/dL and uric acid >6mg/dL. The diagnostic components for all subjects along with all maternal and fetal complications and outcomes were compared with their serum uric acid levels.

In a retrospective cohort study conducted by Wu et. al. in 2012 (39), each SD increase in serum uric acid level at the onset of GHT was associated with 2.3-fold higher odds of progression to pre-eclampsia, and 1.5-fold higher odds of developing adverse maternal/fetal outcomes. Comparing patients who progressed to pre-eclampsia versus those who did not, the proportion of obese women was higher, and the onset of gestational hypertension was earlier. In our study, there were 130 women who were overweight or obese as per BMI. However, no statistical correlation could be established between obesity and the risk of developing pre-eclampsia or other complications.

According to study conducted by Nair et. al. in 2017 (3838), there was a significant increase in LBW babies with increasing serum uric acid levels. In our study, 26.5% (53 subjects) of the pregnancies resulted in LBW neonates, of which approximately 38% (20 subjects) had elevated serum uric acid levels while a figure of 19.2% was observed in the study by Nair et. al. Additionally, in our study, 3% subjects developed HELLP syndrome ($p < 0.05$) and 4% subjects had eclampsia, which is in line with data from the study conducted by Roberts et. al. in 2005 (40) in which 2.8% of the subjects with elevated serum uric acid levels had HELLP syndrome.

According to the study conducted by Williams et. al. in 2002 (41), the time of onset of pre-eclampsia is very important in determining the associated maternal or fetal outcome since early delivery is the only recommended treatment for this disorder. That said, if the onset of pre-eclampsia is between 24 and 30 weeks of gestation, it is likely that the pregnancy may readily progress to either IUD or any other dangerous complication where fetal survival may be at stake. In our study, 10 subjects presented with GHT, severe pre-eclampsia, or eclampsia between 24 and 30 weeks of gestation, of which 4 resulted in IUD ($p < 0.001$). Total number of IUD in our study were 10 (0.5%) while the rate of perinatal mortality in the study conducted by Nair et. al. was 22%.

According to the study conducted by Patel et. al. in 2014 (1), the approximate time at which serum uric acid concentration begins to rise usually coincides with and is a good indicator of the time of onset of pre-eclampsia. Therefore, the significance of measuring serum uric acid in GHT is greatest between 24 and 32 weeks of gestation. Rising or high values obtained during this time indicate high-risk scenarios which are best managed in a hospital setting. Of the 10 subjects that presented between 24 and 30 weeks in our study, 9 (90%) had elevated uric acid levels (>6 mg/dL); whereas in the study conducted by Plouin et. al. in 1986 (42), 59% of women had serum uric acid ≥6 mg/dL in the group with poor perinatal outcome.

In our study, abruptio placenta was the only maternal outcome that could reach a statistical significance in subjects with elevated serum uric acid levels ($p < 0.05$) which is comparable to the rate of 10.8% for abruptio in study by Patel et. al. Also, increased perinatal morbidity (pre-term, LBW, IUGR, IUD) was observed in our study cohort which is in line with studies conducted by Magann et. al. in 1993 (43) (positive correlation with elevated serum uric acid levels), Roberts et. al. in 2005 (40) (23.9% associated with hypertension, proteinuria, and hyperuricemia), and August et. al. in 2004 (44) (17% in pre-eclampsia group with serum uric acid concentration between 3.3 and 5.1 mg/dL). Bed-rest, continued monitoring of fetal well-being in-utero, and anticipation of maternal issue related to pre-eclampsia ensure that there are minimal fetal and maternal complications in emergency delivery.

Limitations of the Present Study

1. Short period of study
2. Lack of normotensive controls
3. Geographical bias

SUMMARY

Ours was a prospective comparative observational study done on 200 patients attending outpatient department/labour ward at GTMCH, Theni, Tamil Nadu, from June 2018 to June 2019. Our study was done in search of a valuable marker for pre-eclampsia and eclampsia which would reflect the severity of the disease and help in predicting the maternal and perinatal outcome to drive decision-making and influence the current management protocols in order to achieve a better maternal and perinatal outcome. Serum uric acid has been suggested as a promising marker by various authors. The inferences made from our study are as follows:

1. Extremes of age was not associated with elevation of serum uric acid levels.
2. Parity of the patient presenting with GHT did not affect the serum uric acid levels.
3. Proteinuria by itself is a marker of severity of the disease and was associated with high serum uric acid levels.
4. Patients with elevated serum uric acid levels had increased numbers of pre-term deliveries, especially between 34-36 weeks.
5. High serum uric acid levels were associated with increased chances of operative deliveries probably to reduce maternal or fetal morbidity and mortality.

6. High serum uric acid levels were associated with low birth weight, probably as a result of increased risk of IUGR and pre-term deliveries.
7. High serum uric acid levels were significantly associated with more number of IUD.
8. High serum uric acid levels were associated with high chances of abruption, HELLP, and eclampsia.
9. There were no maternal mortalities.

CONCLUSION

There has been ongoing debate about the usefulness of uric acid as a significant marker of disease severity and pregnancy outcomes in subjects with varied spectrum of GHT. Our study concludes that the measurement of serum uric acid levels after 20 weeks of gestation is a great diagnostic and prognostic tool to assess fetal outcomes. This study shows that the estimation of serum uric acid levels in pregnancies complicated by hypertension and pre-eclampsia help in assessing the severity of the disease and identifying life-threatening maternal and fetal complications as a result of pre-eclampsia/eclampsia. Since serum uric acid is a very simple analytical tool that can be readily performed at any biochemical laboratory, this is a very cost-effective method to gauge on GHT complications and improve maternal and perinatal outcomes.

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ANNEXURE

PROFORMA

- Name
- Age
- IP No.
- Phone No.
- Address
- Husband’s Name
- Occupation
- Qualification
- Socio-economic Status
- Obstetric History
    - Parity
    - LMP
    - EDD
- GA by LMP
- GA by USG

GENRAL EXAMINATION

- Height
- Weight
- Pulse

- BP
- Pallor
- Pedal Edema
- CVS
- Resp System
- CNS
- Per Abdomen
- Thyroid

BLOOD INVESTIGATIONS

- RFT
- LFT
- TSH
- Urine Albumin
- Uric Acid

SUMMARY:

OUTCOME:

Ref. No. 2544/ME1/18

Government Theni Medical College
Theni Dated: 07.06.2018

Institutional Ethical Committee:

Convenor:

Dr. T. Thirunavukkarasu, M.D., D.A.,
Dean
Govt. Theni Medical College
Theni

Sub: Medical Education – Govt. Theni Medical College,
Theni – Ethical Committee – Minutes – Communicated – Reg.

.....

The Ethical Committee Meeting of the Govt. Theni Medical College, Theni was held at 10.30 A.M. on 07.06.2018 at 150 Lecture Hall, Government Theni Medical College Hospital, Theni.

The following Members of the Committee have attended the Meeting.

1.	**Convener**	:	**Dr. T. Thirunavukkarasu, M.D., D.A., Dean**
2.	**Member Secretary**	:	**Dr. M. Ilangovan, M.S., Deputy Superintendent**
3	**Members**		
	Professor of Medicine	:	**Dr. P. K. Ganesh Babu, M.D.,**
	Professor of Surgery	:	**Dr. R. Murugesan, M.S.,**
	Professor of Obs. & Gynaec.	:	**Dr. Thangamani, M.D., O.G.,**
	Professor of Micro Biology	:	**Dr. K.M. Mythreyee, M.D.,**
4.	**Chairman (Private Consultant)**	:	**Dr. Paulraj, M.D.,** Ramya Clinic, Periyakulam Road, Theni.
5.	**Lawyer**	:	**Thiru.K.Murugesan, B.Com., B.L.,** S/o.Kamaraj, Ambedkar Nagar, Varusanadu, Theni District.
6.	**Sociologist**	:	**Sr. Anaestescia** Director, Jeevan Jothi Hospital Community Care Centre, Periyakulam Road, Kailasapatti, Theni Dist.
7.	**Public**	:	**Mr. P. Deenadhayalan, M.A.,** Land Lord, Koduvilarpatti, Theni District.

The following Project was approved by the Committee:

Name and Designation	Name of the Project	Remarks
Dr. Kalpana Kumari meena First Year MS (OG) Post graduate	**The study of serum uric acid as a biochemical marker for Maternal and Foetal outcome in pregnancy induced hypertension**	**Approved**

Please note that the investigator should adhere the following: He/she should get a detailed informed consent from the Patients/participants and maintain Confidentially.

1. He/she should carry out the work without detrimental to regular activities as well as without extra expenditure to the institution.
2. He/she should inform the Institution Ethical Committee in case of any change of study procedure site and investigation or guide.
3. He/she should not deviate for the area of the work for which applied for Ethical Clearance. He/She should inform the Institution Ethical Committee immediately, in case of any adverse events or any serious adverse reactions.
4. He/she should abide to the rules and regulations of the institution.
5. He/she should complete the work within the specific period and apply for if any extension of time is required. He/she should apply for permission again and do the work.
6. He/she should submit the summary of the research work to the Ethical Committee on completion of the work.
7. He/she should not claim any funds from the institution while doing the work or on completion.
8. He/she should understand that the members of Institutional Ethical Committee have the right to monitor the work with prior intimation.

Chairman

Convenor
DEAN
GOVT. THENI MEDICAL COLLEGE HOSPITAL
THENI-625 512.

To

Dr.M. PALRAJ,M.D.,
Civil Surgeon Retd.,
Chairman
574, Periyakulam Road,
THENI - 625 531.
Regn. No: 28094

The above individual through Head of the Department concerned.

CERTIFICATE – II

This is to certify that this dissertation work titled **THE STUDY OF SERUM URIC ACID AS A BIOCHEMICAL INDICATOR FOR MATERNAL AND FETAL OUTCOME IN PATIENTS WITH GESTATIONAL HYPERTENSION"** of the candidate **Dr. Kalpana Kumari Meena,** with registration Number. **221716653** for the award of M.S., in this branch of Obstetrics and Gynaecology. I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file incudes pages from introduction to conclusion and the result shows 5 percentage of plagiarism in the dissertation.

Signature of the Guide

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Analysed Document:	Dr. Kalpana Meena_third draft.docx (D57193861)
Submitted:	17/10/2019 19:37:00
Submitted By:	kalpana.alax91.km@gmail.com
Significance:	5 %

MASTER CHART

NAME	AGE (years)	OBESTETRIC HISTORY	LMP (DDMMYY)	EDD (DDMMYY)	GA BY LMP	GA BY USG	HEIGHT (m)	WEIGHT (kg)	BMI (kg/m2)	GHT CATEGORY	URINE ALBUMIN	SERUM URIC ACID (mg/dL)	MODE OF DELIVERY	FETAL OUTCOME	IUD	IUGR	HELLP	ABRUPTION	ECLAMPSIA	MATERNAL DEATH	ICH	PULM EDEMA	SPONTANEOUS EXPULSION
Madhumitha	19	Primi	15.10.18	22.7.19	38w 6d	38w	1.5	55	24.4	GHT since 8 month of GA	++++	6.5	LN	LBW	-	-	-	-	-	-	-	-	-
Ramlath Nisha	28	Primi	22.10.18	29.7.19	38w 6d	39w	1.47	93	43.0	GHT since 7 month of GA	Nil	6.6	LN	IUGR	-	1	-	-	-	-	-	-	-
Meghala	29	G5P1 L1A3	9.10.18	16.7.19	38w	37w 6d	1.42	65	32.2	Severe preeclampsia	Nil	5	LN	Mild IUGR	-	1	-	-	-	-	-	-	-
Navanya	23	Primi	8.10.18	15.7.19	39w	39w	1.51	71	31.1	Severe preeclampsia	++++	7	LN	IUGR	-	1	-	-	-	-	-	-	-
Muthulakshmi	23	Primi	10.10.18	17.7.19	39w	39w	1.63	61	23.0	Newly diagnosed GHT	Nil	5.4	Elective LSCS	LBW	-	-	-	-	-	-	-	-	-
Jenifer	22	G3P1 L1A1	20.1.19	27.10.19	24w	24w 5d	1.54	52	21.9	Severe preeclampsia	++++	8.9	Emerg. Rpt. LSCS	IUD	1	-	-	-	-	-	-	-	-
Kousalya	19	Primi	11.10.18	18.7.19	39w 3d	39w 3d	1.43	45	22.0	GHT since 1 month	Nil	5.4	LN	Term baby	-	-	-	-	-	-	-	-	-
Vanitha	32	G2P1 L1	2.10.18	9.7.19	38w	38w 2d	1.5	60	26.7	GHT since 7 month of GA	++++	4.6	Elective LSCS	Mild IUGR	-	1	-	-	-	-	-	-	-

Gopika	23	Primi	8.10.18	15.7.19	39w	39w	1.46	60	28.1	GHT since 6 month of GA	+++	6.7	LN	IUGR	-	1	-	-	-	-	-	-	-
Muthu	26	G2P1 L1	9.10.17	16.7.18	37w	37w 4d	1.5	55	24.4	Non severe preeclampsia	Nil	4.6	LN	LBW	-	-	-	-	-	-	-	-	-
Sangeetha	27	G3P2 L2	9.11.18	16.8.19	34w	34w 2d	1.6	62	24.2	Recurrent GHT	++++	6.7	LN	Severe IUGR	-	1	-	-	-	-	-	-	-
Keerthana	23	Primi	28.12.18	4.10.19	28w	27w 5d	1.55	52	21.6	Severe preeclampsia / APH	++++	10.2	Emerg. LSCS	IUD	1	-	-	1	-	-	-	-	-
Shanthi	32	Primi	8.10.17	15.7.18	32w	32w	1.52	60	26.0	Non severe preeclampsia	Nil	5.6	Emerg. LSCS	Mild IUGR	-	1	-	-	-	-	-	-	-
Subbulakshmi	29	Primi	16.7.17	23.4.18	38w	38w 3d	1.44	58	28.0	GHT since 8 month of GA	+	5.5	Elective LSCS	LBW	-	-	-	-	-	-	-	-	-
Pandimeena	25	G2P1 L1	25.5.18	2.2.19	37w 4d	37w	1.51	53	23.2	Non severe preeclampsia	Nil	6	LN	Term baby	-	-	-	-	-	-	-	-	-
Mutheeshwari	19	Primi	3.8.18	10.5.19	34w 6d	34w 3d	1.38	48	25.2	GHT since 1 week	Nil	6.3	LN	Early Preterm/LBW	-	-	-	-	-	-	-	-	-
Esther	22	Primi	13.6.17	20.3.18	38w 5d	38w 6d	1.49	64	28.8	Severe preeclampsia	++++	6.9	Emerg. LSCS	Severe IUGR	-	1	-	-	-	-	-	-	-
Kaaliyammal	20	G2P1 L1	22.6.17	29.3.18	30w 1d	30w	1.57	59	23.9	Severe preeclampsia	++++	10.4	EMERGENCY LSCS	IUD	1	-	-	-	-	-	-	-	-
Eswari	27	G4P2 L2A1	23.6.17	30.3.18	39w	38w 4d	1.55	57	23.7	Non severe preeclampsia	Nil	4.4	Elective LSCS	Term baby	-	-	-	-	-	-	-	-	-
Krishnaveni	18	Primi	5.7.17	12.4.18	38w 4d	38w 3d	1.44	44	21.2	GHT since 6 month of GA	++	5.1	LN	LBW	-	-	-	-	-	-	-	-	-

Jeyalakshmi	22	Primi	5.7.17	12.4.18	36w 5d	37w	1.53	56	23.9	GHT since 5 days	Nil	6.5	LN	LBW	-	-	-	-	-	-	-	-	-
Saraswathy	29	G2P1 L1	15.7.17	22.4.18	33w	32w 4d	1.42	42	20.8	GHT since 1 month	++	6.6	Emerg. LSCS	Early Preterm	-	-	-	-	-	-	-	-	-
Priya	30	G3P1 L0A1	22.7.17	29.4.18	35w	35w	1.59	53	21.0	GHT since 7 month of GA	+	6	Elective LSCS	Late Preterm	-	-	-	-	-	-	-	-	-
Alagammal	35	G3P2 L2	24.7.17	1.5.18	38w 2d	38w	1.5	58	25.8	GHT since 1 week	Nil	4.7	LN	Term baby	-	-	-	-	-	-	-	-	-
Anushya	23	Primi	20.10.17	27.7.18	37w 6d	37w	1.55	46	19.1	GHT since 1 week	Nil	6.7	LN	Term baby	-	-	-	-	-	-	-	-	-
Vimala Devi	21	Primi	1.11.17	8.8.18	35w	35w 4d	1.56	61	25.1	Non severe preeclampsia	++	5.6	LN	Late Preterm	-	-	-	-	-	-	-	-	-
Pandiyammal	29	Primi	15.11.17	22.8.18	35w 3d	35w 6d	1.52	61	26.4	GHT since 6 month of GA	Nil	4.7	Elective LSCS	Late Preterm/LBW	-	-	-	-	-	-	-	-	-
Thulasi Devi	25	G3P1 L1A1	17.11.17	24.8.18	28w	27w 4d	1.63	58	21.8	GHT since 2 weeks	+++	5.9	Emerg. Rpt. LSCS	Extreme Preterm	-	-	-	-	-	-	-	-	-
Veeru Chinnammal	26	G4P1 L1A2	18.11.17	25.8.18	39w 2d	39w	1.53	61	26.1	GHT since 5 days	Nil	4.4	Emerg. Rpt. LSCS	Term baby	-	-	-	-	-	-	-	-	-
Aarthi	20	G2P1 L1	23.11.17	30.8.18	37w	37w 4d	1.45	41	19.5	New onset GHT/Abruption	++++	9.8	Emerg. LSCS	IUD	1	-	-	1	-	-	-	-	-
Bhavani	25	G3P1 L1A1	18.10.17	25.7.18	35w 5d	35w 6d	1.55	69	28.7	GHT since 8 month of GA	Nil	4.6	Elective Rpt. LSCS	Late Preterm	-	-	-	-	-	-	-	-	-
Ranjitha	26	G2P1 L1	9.11.17	16.8.18	33w 3d	33w	1.57	48	19.5	Newly diagnosed GHT	Nil	5.4	Emerg. LSCS	Early Preterm	-	-	-	-	-	-	-	-	-
Arivuselvi	27	Primi	31.10.17	7.8.18	38w 1d	38w	1.62	71	27.1	GHT since 6 month of GA	++	5.2	Elective LSCS	Term baby	-	-	-	-	-	-	-	-	-

Arunjunai	19	Primi	8.11.17	15.8.18	37w 4d	37w 5d	1.48	48	21.9	Non severe preeclampsia	++++	6	LN	Term baby	-	-	-	-	-	-	-	-	-
Bharathi	23	G3P1 L1A1	16.11.17	23.8.18	32w 6d	324 d	1.55	57	23.7	GHT since 2 weeks	Nil	4.4	Emerg. Rpt. LSCS	Early Preterm/LBW	-	-	-	-	-	-	-	-	-
Kayalvizhi	24	Primi	19.11.17	26.8.18	36w 2d	36w	1.54	66	27.8	GHT since 6 month of GA	++++	5.9	Elective LSCS	IUGR	-	1	-	-	-	-	-	-	-
Vijayalakshmi	23	G3P1 L1A1	20.11.17	27.8.18	37w	37w 2d	1.56	60	24.7	Severe preeclampsia	++++	6.7	Emerg. Rpt. LSCS	LBW	-	-	-	-	-	-	-	-	-
Lakshmipriya	38	Primi	19.11.17	26.8.18	37w	37w 4d	1.5	55	24.4	HELLP Syndrome	++++	7	Emerg. LSCS	Term baby	-	-	1	-	-	-	-	-	-
Neelambari	25	G2P1 L0	30.11.17	7.9.18	33w 5d	33w 6d	1.61	69	26.6	Non severe preeclampsia	Nil	5.2	Elective LSCS	Early Preterm	-	-	-	-	-	-	-	-	-
Sundari	18	Primi	2.12.17	9.9.18	36w	35w 6d	1.5	55	24.4	Non severe preeclampsia	Nil	4.9	LN	Late Preterm	-	-	-	-	-	-	-	-	-
Nagapriya	37	G5P1 L1A3	4.12.17	11.9.18	38w 2d	38w	1.48	50	22.8	Severe preeclampsia	++++	7	Elective LSCS	Term baby	-	-	-	-	-	-	-	-	-
Vinitha	19	Primi	7.12.17	14.9.18	33w 5d	33w 2d	1.45	45	21.4	Newly diagnosed GHT	Nil	6.5	LN	Early Preterm	-	-	-	-	-	-	-	-	-
Anitha	23	G2P1 L1	11.12.17	18.9.18	30w 5d	31w	1.56	56	23.0	GHT since 6 month of GA	Nil	4.4	Elective Rpt. LSCS	Early Preterm	-	-	-	-	-	-	-	-	-
Kavitha	20	Primi	11.12.17	18.9.18	37w 3d	37w 3d	1.56	60	24.7	Non severe preeclampsia	+	6	LN	LBW	-	-	-	-	-	-	-	-	-
Chitra	18	Primi	12.12.17	19.9.18	32w 1d	32w	1.49	48	21.6	Severe preeclampsia	++++	6.2	Emerg. Rpt. LSCS	Early Preterm	-	-	-	-	-	-	-	-	-
Divya Rukmani	32	G3P1 L1A1	18.12.17	25.9.18	38w	38w 2d	1.5	50	22.2	Partial HELLP	++++	7	LN	Term baby	-	-	-	-	-	-	-	-	-

Kannazhagi	29	G2A1	20.12.17	27.9.18	37w 4d	37w 5d	1.6	64	25.0	Non severe preeclampsia	Nil	4.9	Elective LSCS	LBW	-	-	-	-	-	-	-	-	-
Shanthi Devi	27	Primi	23.12.17	30.9.18	32w 2d	32w	1.55	49	20.4	AP Eclampsia	++++	6.8	Emerg. LSCS	Early Preterm/LBW	-	-	-	-	1	-	-	-	-
Nandhini	24	G2P1 L1	30.12.17	7.10.18	38w 5d	39w	1.53	57	24.3	Recurrent GHT	Nil	4.8	Elective Rpt. LSCS	LBW	-	-	-	-	-	-	-	-	-
Kodi	19	Primi	1.1.18	8.10.18	30w 6d	30w 4d	1.48	60	27.4	Severe preeclampsia	++++	6.6	Emerg. LSCS	Early Preterm/LBW	-	-	-	-	-	-	-	-	-
Muthu Meena	18	Primi	2.1.18	9.10.18	37w 4d	37w 3d	1.52	56	24.2	GHT since 8 month of GA	++	4.4	LN	Term baby	-	-	-	-	-	-	-	-	-
Mounam	32	G3P1 A2	4.1.18	11.10.18	32w 1d	32w 3d	1.56	60	24.7	GHT since 2 weeks	++++	5.4	LN	Early Preterm	-	-	-	-	-	-	-	-	-
Geetha	23	G2P1 L1	8.1.18	15.10.18	38w 3d	38w 2d	1.49	53	23.9	Non severe preeclampsia	Nil	5	LN	LBW	-	-	-	-	-	-	-	-	-
Laila	25	Primi	13.1.18	20.10.18	37w 4d	37w 5d	1.54	58	24.5	Non severe preeclampsia	Nil	4.7	LN	LBW	-	-	-	-	-	-	-	-	-
Vinotha	21	Primi	18.1.18	25.10.18	34w 6d	35w	1.55	49	20.4	GHT since 4 days	Nil	5.2	LN	Late Preterm	-	-	-	-	-	-	-	-	-
Haripriya	28	G3P2 L2	19.1.18	26.10.18	34w	34w	1.52	54	23.4	Severe preeclampsia	++++	6.2	Elective Rpt. LSCS	Severe IUGR	-	1	-	-	-	-	-	-	-
Lavanya	34	Primi	19.1.18	26.10.18	38w 5d	39w	1.55	60	25.0	Newly diagnosed GHT	Nil	4.2	Emerg. LSCS	Term baby	-	-	-	-	-	-	-	-	-
Agalya	22	G2P1 L1	25.1.18	2.11.18	30w 3d	30w 1d	1.45	55	26.2	Recurrent GHT	Nil	5.2	Emerg. Rpt. LSCS	Early Preterm	-	-	-	-	-	-	-	-	-
Mallika	26	G2P1 L1	28.1.18	5.11.18	37w 5d	37w 6d	1.5	53	23.6	GHT since 7 month of GA	+	4.4	Elective LSCS	LBW	-	-	-	-	-	-	-	-	-

Kanimozhi	24	G3P1 L1A1	30.1.18	7.11.18	33w 2d	33w	1.53	56	23.9	Severe preeclampsia	++++	5.7	Elective Rpt. LSCS	Early Preterm/LBW	-	-	-	-	-	-	-	-	-
Meenu Kutty	27	Primi	2.2.18	9.11.18	38w	38w	1.6	62	24.2	Non severe preeclampsia	Nil	4.9	LN	Term baby	-	-	-	-	-	-	-	-	-
Ganeshwari	19	Primi	9.2.18	16.11.18	37w 4d	37w 4d	1.47	47	21.8	GHT since 3 weeks	Nil	4.8	LN	LBW	-	-	-	-	-	-	-	-	-
Maariyammal	18	Primi	11.2.18	18.11.18	38w 2d	38w	1.52	48	20.8	Non severe preeclampsia	Nil	6.2	Emerg. LSCS	LBW	-	-	-	-	-	-	-	-	-
Kundhavai	29	G2P1 L1	11.2.18	18.11.18	32w 5d	33w	1.5	53	23.6	GHT since 7 month of GA	++	6	Elective Rpt. LSCS	Early Pretem/LBW	-	-	-	-	-	-	-	-	-
Aavudaiyammal	18	Primi	11.2.18	18.11.18	36w 1d	36w	1.55	62	25.8	Newly diagnosed GHT	++++	5.9	LN	Mild IUGR	-	1	-	-	-	-	-	-	-
Sivagami	20	G2P1 L0	12.2.18	19.11.18	38w 6d	39w	1.54	50	21.1	Newly diagnosed GHT	Nil	4.6	Elective LSCS	Term baby	-	-	-	-	-	-	-	-	-
Seeniyammal	23	Primi	13.2.18	20.11.18	35w	35w 2d	1.48	54	24.7	AP Eclampsia	++++	6.9	Emerg. LSCS	IUGR	-	1	-	-	1	-	-	-	-
Ponnuthai	19	Primi	13.2.18	20.11.18	37w 5d	37w 6d	1.51	67	29.4	Severe preeclampsia	++++	5.8	Emerg. LSCS	Term baby	-	-	-	-	-	-	-	-	-
Anbumani	26	G2P1 L1	15.2.18	22.11.18	37w	37w 2d	1.58	66	26.4	GHT since 1 month	Nil	5.5	LN	LBW	-	-	-	-	-	-	-	-	-
Mullaikodi	33	G3P2 L2	16.2.18	23.11.18	31w 6d	32w	1.49	56	25.2	Non severe preeclampsia	Nil	6	Elective LSCS	Early preterm	-	-	-	-	-	-	-	-	-
Yazhini	26	Primi	16.2.18	23.11.18	37w 5d	37w	1.44	49	23.6	Severe preeclampsia	++++	6.5	Emerg. LSCS	LBW	-	-	-	-	-	-	-	-	-
Pavalam	23	G2P1 L1	20.2.18	27.11.18	37w	37w	1.5	56	24.9	Recurrent GHT	Nil	6.3	Elective Rpt. LSCS	Term baby	-	-	-	-	-	-	-	-	-
Amudha	18	Primi	23.2.18	30.11.18	37w 4d	37w 6d	1.53	48	20.5	GHT since 8	Nil	5.3	LN	Term baby	-	-	-	-	-	-	-	-	-

										month of GA													
Roshini	24	G3P1 L1A1	24.2.18	1.12.18	33w 6d	34w	1.42	52	25.8	severe preeclampsia	++++	6.1	Emerg. Rpt. LSCS	Early preterm	-	-	-	-	-	-	-	-	-
Abhiniya	28	G2P1 L1	26.2.18	3.12.18	28w	28w	1.48	44	20.1	AP Eclampsia	++++	6.8	Spont expul	Spont expul	-	-	-	-	1	-	-	-	1
Dhana	21	Primi	27.2.18	4.12.18	35w 2d	35w	1.5	45	20.0	Newly diagnosed GHT	Nil	5.9	LN	Late Preterm	-	-	-	-	-	-	-	-	-
Arivumathi	19	Primi	1.3.18	8.12.18	38w	38w 2d	1.49	54	24.3	Non severe preeclampsia	Nil	4.4	Emerg. LSCS	LBW	-	-	-	-	-	-	-	-	-
Kavi	32	G4P1 L1A2	2.3.18	9.12.18	36w 4d	36w 3d	1.6	72	28.1	Recurrent GHT	++	6.3	Emerg. Rpt. LSCS	Mild IUGR	-	1	-	-	-	-	-	-	-
Kannamma	25	G2P1 L1	2.3.18	9.12.18	39w	39w	1.56	59	24.2	Newly diagnosed GHT	Nil	6	LN	LBW	-	-	-	-	-	-	-	-	-
Shobika	22	Primi	3.3.18	10.12.18	38w 3d	38w 5d	1.47	48	22.2	Severe preeclampsia	++++	5.9	Emerg. LSCS	Term baby	-	-	-	-	-	-	-	-	-
Shanmugavalli	24	G3P2 L2	5.3.18	12.12.18	34w 3d	34w 2d	1.49	53	23.9	GHT since 6 month of GA	Nil	4.7	Elective Rpt. LSCS	Early preterm	-	-	-	-	-	-	-	-	-
Navaneetha	22	Primi	5.3.18	12.12.18	37w 3d	37w 5d	1.58	61	24.4	Non severe preeclampsia	Nil	5	LN	Term baby	-	-	-	-	-	-	-	-	-
Arul Mari	19	Primi	6.3.18	13.12.18	36w 6d	37w	1.47	59	27.3	GHT since 2 weeks	Nil	5.8	LN	LBW	-	-	-	-	-	-	-	-	-
Koperundhevi	26	G2P1 L1	8.3.18	15.12.18	38w	38w 3d	1.55	53	22.1	GHT since 8 month of GA	+	6.2	Emerg. Rpt. LSCS	Term baby	-	-	-	-	-	-	-	-	-
Chinnaponnu	31	Primi	8.3.18	15.12.18	37w 4d	37w 6d	1.6	75	29.3	Non severe preeclampsia	Nil	5.9	LN	Term baby	-	-	-	-	-	-	-	-	-
Sri Magizh	24	G2P1 L1	10.3.18	17.12.18	38w 6d	39w	1.44	48	23.1	GHT since 1 week	Nil	6	Elective Rpt. LSCS	Term baby	-	-	-	-	-	-	-	-	-

Mughil	29	G2P1 L1	11.3.18	18.12.18	32w 3d	32w	1.52	60	26.0	Severe preeclampsia	++++	5.9	Elective Rpt. LSCS	Early preterm	-	-	-	-	-	-	-	-	-
Ranjitha	18	Primi	14.3.18	21.12.18	36w 6d	37w	1.42	54	26.8	Non severe preeclampsia	Nil	4.3	LN	Term baby	-	-	-	-	-	-	-	-	-
Pounthai	33	G3P1 L1A1	14.3.18	21.12.18	33w 2d	33w	1.6	61	23.8	GHT since 6 month of GA	Nil	5.6	Emerg. LSCS	Early preterm	-	-	-	-	-	-	-	-	-
Valliyammal	23	G2P1 L1	16.3.18	23.12.18	37w	37w 3d	1.57	55	22.3	Recurrent GHT	++	4.6	Emerg. Rpt. LSCS	LBW	-	-	-	-	-	-	-	-	-
Sneha	21	Primi	17.3.18	24.12.18	33w 2d	33w 4d	1.49	56	25.2	AP Eclampsia	++++	6.5	Emerg. LSCS	Early Preterm/LBW	-	-	-	-	1	-	-	-	-
Nivedha	20	Primi	20.3.18	27.12.18	38w 2d	38w	1.44	49	23.6	GHT since 1 month	Nil	5.3	LN	LBW	-	-	-	-	-	-	-	-	-
Jothi Lakshmi	28	G3P2 L2	20.3.18	27.12.18	38w	38w	1.53	47	20.1	Non severe preeclampsia	Nil	4.2	Elective Rpt. LSCS	Term baby	-	-	-	-	-	-	-	-	-
Velammal	25	G2P1 L1	21.3.18	28.12.18	38w 3d	38w 4d	1.61	77	29.7	Newly diagnosed GHT	Nil	4.9	Emerg. Rpt. LSCS	Term baby	-	-	-	-	-	-	-	-	-
Sumathi	32	G2P1 L1	22.3.18	29.12.18	37w 5d	38w	1.49	62	27.9	HELLP Syndrome	++++	7	Emerg. LSCS	LBW	-	-	1	-	-	-	-	-	-
Thanmathi	19	Primi	23.3.18	30.12.18	28w	27w 5d	1.55	51	21.2	Severe preeclampsia	++++	6.2	Emerg. LSCS	Extreme Preterm	-	-	-	-	-	-	-	-	-
Mayilu	18	Primi	23.3.18	30.12.18	34w 5d	35w	1.52	48	20.8	GHT since 8 month of GA	Nil	5.4	LN	Late Preterm	-	-	-	-	-	-	-	-	-
Vaasavi	31	G3P2 L0	26.3.18	3.1.19	37w 4d	37w 4d	1.47	55	25.5	Severe preeclampsia	++++	5.9	Elective LSCS	Term baby	-	-	-	-	-	-	-	-	-
Vimala	20	Primi	28.3.18	5.1.19	37w	37w 1d	1.5	56	24.9	Severe preeclampsia	++++	6.2	Elective LSCS	LBW	-	-	-	-	-	-	-	-	-
Vishali	22	G2P1 L1	31.3.18	7.1.19	38w	38w	1.55	64	26.6	GHT since 2 weeks	Nil	5.4	Emerg. Rpt. LSCS	LBW	-	-	-	-	-	-	-	-	-

Vaidhegi	23	G2P1 L1	1.4.18	8.1.19	37w 5d	37w 2d	1.43	52	25.4	GHT since 7 month of GA	Nil	4.6	Elective Rpt. LSCS	Term baby	-	-	-	-	-	-	-	-	-
Azhagumani	26	Primi	1.4.18	8.1.19	38w 2d	38w 3d	1.56	55	22.6	Non severe preeclampsia	++	4.8	LN	Term baby	-	-	-	-	-	-	-	-	-
Kaaliyammal	29	G3P2 L2	3.4.18	10.1.19	32w 4d	32w 2d	1.58	60	24.0	Recurrent GHT	Nil	5.3	Elective Rpt. LSCS	Early preterm	-	-	-	-	-	-	-	-	-
Vadivu	22	G3P1 L1A1	4.4.18	11.1.19	36w	36w 1d	1.52	49	21.2	Severe preeclampsia	++++	5.9	Emerg. Rpt. LSCS	Severe IUGR	-	1	-	-	-	-	-	-	-
Revathy	18	Primi	6.4.18	13.1.19	33w 5d	34w	1.48	62	28.3	Non severe preeclampsia	Nil	6	Emerg. LSCS	Early Preterm	-	-	-	-	-	-	-	-	-
Nandhini	19	Primi	10.4.18	17.1.19	37w 6d	38w	1.5	59	26.2	GHT since 1 month	Nil	5.4	LN	Term baby	-	-	-	-	-	-	-	-	-
Arasi	24	G2P1 L1	13.4.18	20.1.19	37w 1d	37w	1.43	49	24.0	Severe preeclampsia	++++	6.2	Emerg. LSCS	Term baby	-	-	-	-	-	-	-	-	-
Jothiyammal	26	G2P1 L2	17.4.18	24.1.19	38w 2d	38w 4d	1.57	60	24.3	Newly diagnosed GHT	Nil	5.6	Emerg. LSCS	LBW	-	-	-	-	-	-	-	-	-
Rani	20	Primi	17.4.18	24.1.19	35w 4d	35w 2d	1.63	72	27.1	Severe preeclampsia	++++	6.4	Emerg. LSCS	Late Preterm	-	-	-	-	-	-	-	-	-
Thangammal	30	G2P1 L1	23.4.18	30.1.19	38w 5d	39w	1.53	58	24.8	Non severe preeclampsia	Nil	5.4	LN	LBW	-	-	-	-	-	-	-	-	-
Gandhimathi	21	Primi	26.4.18	3.2.19	37w 3d	37w 2d	1.62	66	25.1	Non severe preeclampsia	+	5	LN	Term baby	-	-	-	-	-	-	-	-	-
Sangavi	18	Primi	28.4.18	5.2.19	36w 3d	36w 3d	1.58	56	22.4	GHT since 7 month of GA	++	5.8	Elective LSCS	Mild IUGR	-	1	-	-	-	-	-	-	-
Tamilarasi	26	G2P1 L0	29.4.18	6.2.19	38w 2d	38w	1.44	45	21.7	Newly diagnosed GHT	Nil	5.3	Elective LSCS	Term baby	-	-	-	-	-	-	-	-	-

Isai Vani	20	G2P1 L1	2.5.18	9.2.19	27w 3d	27w 2d	1.49	56	25.2	Severe preeclampsia	++++	6.5	Emerg. Rpt. LSCS	Extreme Preterm	-	-	-	-	-	-	-	-	-
Kalaivani	28	Primi	3.5.18	10.2.19	32w 3d	32w 1d	1.52	62	26.8	GHT since 1 week	+	6	LN	Early preterm	-	-	-	-	-	-	-	-	-
Gomathi	24	G2P1 L1	7.5.18	14.2.19	39w	39w	1.47	51	23.6	GHT since 8 month of GA	Nil	4.5	Elective Rpt. LSCS	Term baby	-	-	-	-	-	-	-	-	-
Pechiammal	32	G2P1 L1	8.5.18	15.2.19	35w 3d	35w 6d	1.63	63	23.7	Severe preeclampsia	++++	5.9	Emerg. Rpt. LSCS	Mild IUGR	-	1	-	-	-	-	-	-	-
Rekha	23	Primi	12.5.18	19.2.19	37w	37w	1.59	49	19.4	Non severe preeclampsia	Nil	5.6	LN	Mild IUGR	-	1	-	-	-	-	-	-	-
Radha	27	G3P2 L2	15.5.18	22.2.19	36w 6d	37w 2d	1.48	51	23.3	GHT since 2 weeks	Nil	6.2	LN	LBW	-	-	-	-	-	-	-	-	-
Siva Ranjani	23	Primi	15.5.18	22.2.19	38w 5d	38w 3d	1.42	58	28.8	Non severe preeclampsia	++	5.7	LN	LBW	-	-	-	-	-	-	-	-	-
Muthu Nageshwari	29	G2P1 L1	21.5.18	28.2.19	37w 1d	37w 3d	1.53	54	23.1	Recurrent GHT	Nil	4.3	Elective Rpt. LSCS	Term baby	-	-	-	-	-	-	-	-	-
Sri Nandhini	18	Primi	22.5.18	1.3.19	37w 2d	37w	1.6	61	23.8	AP Eclampsia	++++	11	Emerg. LSCS	IUD	1	-	-	-	1	-	-	-	-
Kumudha	26	G2P1 L1	23.5.18	2.3.19	37w 3d	37w 4d	1.58	54	21.6	Severe preeclampsia	++++	5.4	Emerg. Rpt. LSCS	Term baby	-	-	-	-	-	-	-	-	-
Punidha	19	Primi	27.5.18	4.3.19	34w	33w 5d	1.52	65	28.1	GHT since 6 month of GA	++	4.6	Elective LSCS	Early preterm	-	-	-	-	-	-	-	-	-
Parkavi	25	Primi	30.5.18	7.3.19	37w 6d	38w 1d	1.49	57	25.7	Non severe preeclampsia	Nil	5	LN	Term baby	-	-	-	-	-	-	-	-	-
Pavithra	19	Primi	31.5.18	7.3.19	37w 2d	37w	1.58	54	21.6	GHT since 1 week	Nil	4.6	LN	Term baby	-	-	-	-	-	-	-	-	-
Chellammal	28	G2P1 L1	6.6.18	13.3.19	38w	37w 5d	1.56	53	21.8	Recurrent GHT	Nil	4.9	LN	LBW	-	-	-	-	-	-	-	-	-

Sangeetha Priya	23	Primi	7.6.18	14.3.19	34w 5d	35w	1.45	57	27.1	Non severe preeclampsia	++	4.4	LN	Late Preterm	-	-	-	-	-	-	-	-	-
Selva Kumari	20	G2P1 L1	9.6.18	16.3.19	37w	37w 2d	1.48	49	22.4	Severe preeclampsia	++++	6.5	LN	Severe IUGR	-	1	-	-	-	-	-	-	-
Aashish Begham	30	G3P1 L1A1	14.6.18	21.3.19	37w 5d	37w 5d	1.62	73	27.8	GHT since 8 month of GA	+	5.3	LN	Term baby	-	-	-	-	-	-	-	-	-
Jhansi	19	Primi	14.6.18	21.3.19	32w 4d	32w 5d	1.56	63	25.9	Severe preeclampsia	++++	6	LN	Early Preterm	-	-	-	-	-	-	-	-	-
Deepika	25	Primi	17.6.18	24.3.19	36w 6d	37w 1d	1.49	59	26.6	Non severe preeclampsia	Nil	6.2	LN	LBW	-	-	-	-	-	-	-	-	-
Kumudhavalli	26	G2P1 L1	23.6.18	30.3.19	38w 5d	39w	1.41	47	23.6	Non severe preeclampsia	Nil	5.8	LN	Term baby	-	-	-	-	-	-	-	-	-
Thilagavadhi	18	Primi	25.6.18	2.4.19	38w	38w 1d	1.51	56	24.6	GHT since 6 month of GA	Nil	5.7	Elective LSCS	Mild IUGR	-	1	-	-	-	-	-	-	-
Saradha	24	G3P2 L2	26.6.18	3.4.19	39w	38w 4d	1.58	53	21.2	GHT since 4 days	++++	5.9	LN	Term baby	-	-	-	-	-	-	-	-	-
Susheela	30	G2P1 L1	29.6.18	6.4.19	33w 2d	33w 5d	1.53	63	26.9	Non severe preeclampsia	Nil	4.9	Elective Rpt. LSCS	Early preterm	-	-	-	-	-	-	-	-	-
Amirtha	21	Primi	29.6.18	6.4.19	28w	28w 3d	1.5	49	21.8	Severe preeclampsia	++++	6.5	Emerg. LSCS	IUD	1	-	-	-	-	-	-	-	-
Jenifer	29	Primi	1.7.18	8.4.19	35w 4d	36w	1.43	55	26.9	Non severe preeclampsia	Nil	6.2	LN	Late Preterm	-	-	-	-	-	-	-	-	-
Kaamatchi	34	G4P1 L1A2	3.7.18	10.4.19	32w 6d	33w	1.59	65	25.7	GHT since 7 month of GA	Nil	4.7	Elective LSCS	Early preterm	-	-	-	-	-	-	-	-	-

Karpagadevi	27	G2P1 L1	11.7.18	18.4.19	37w	37w	1.62	77	29.3	HELLP Syndrome	++++	7	Emerg. LSCS	LBW	-	-	1	-	-	-	-	-	-
Devika	19	Primi	16.7.18	23.4.19	38w 4d	38w 3d	1.49	56	25.2	Newly diagnosed GHT	Nil	5.9	LN	Term baby	-	-	-	-	-	-	-	-	-
Lakshmi	29	G2P1 L1	17.7.18	24.4.19	37w 6d	38w	1.51	50	21.9	Severe preeclampsia	++++	6	Emerg. Rpt. LSCS	Term baby	-	-	-	-	-	-	-	-	-
Kumari	24	G2P1 L1	19.7.18	26.4.19	30w 2d	30w 1d	1.53	49	20.9	GHT since 1 month	++	5.5	Emerg. Rpt. LSCS	Early preterm	-	-	-	-	-	-	-	-	-
Tharunika	26	Primi	20.7.18	27.4.19	36w 5d	37w 2d	1.59	62	24.5	Severe preeclampsia	++++	6.3	Emerg. LSCS	LBW	-	-	-	-	-	-	-	-	-
Maragatham	23	G2P1 L1	20.7.18	27.4.19	37w 3d	37w 4d	1.46	60	28.1	GHT since 2 weeks	Nil	4.3	Emerg. LSCS	Term baby	-	-	-	-	-	-	-	-	-
Vidhya	20	Primi	23.7.18	30.4.19	36w 5d	37w 1d	1.47	42	19.4	Newly diagnosed GHT	Nil	6.8	LN	LBW	-	-	-	-	-	-	-	-	-
Sowmya	28	G5P1 L1A3	25.7.18	2.5.19	31w 5d	32w 2d	1.57	54	21.9	Non severe preeclampsia	++	5.6	LN	Early preterm	-	-	-	-	-	-	-	-	-
Madhubala	25	G2P1 L1	26.7.18	3.5.19	28w	28w	1.48	59	26.9	AP Eclampsia	++++	6.9	Emerg. Rpt. LSCS	Extreme Preterm	-	-	-	-	1	-	-	-	-
Ilakiya	20	Primi	28.7.18	5.5.19	39w	38w 5d	1.61	64	24.7	Non severe preeclampsia	Nil	6	LN	Term baby	-	-	-	-	-	-	-	-	-
Parvathy	23	Primi	31.7.18	7.5.19	38w 4d	38w 4d	1.45	50	23.8	Severe preeclampsia	++++	5.8	Emerg. LSCS	Term baby	-	-	-	-	-	-	-	-	-
Kalavathy	27	G2P1 L1	2.8.18	9.5.19	38w 5d	38w 3d	1.47	53	24.5	GHT since 6 month of GA	Nil	6	Elective Rpt. LSCS	Term baby	-	-	-	-	-	-	-	-	-
Mangaiyarkarasi	18	Primi	3.8.18	10.5.19	36w 3d	36w 1d	1.45	57	27.1	Severe preeclampsia	++++	6.2	Emerg. LSCS	Severe IUGR	-	1	-	-	-	-	-	-	-
Poongodi	23	G2P1 L1	4.8.18	11.5.19	35w 3d	35w 2d	1.56	58	23.8	Recurrent GHT	Nil	6.4	Elective Rpt. LSCS	Late Preterm	-	-	-	-	-	-	-	-	-
Sakthi Shree	27	G5P1 L1A3	5.8.18	12.5.19	37w 2d	37w	1.52	47	20.3	Non severe	++	5.4	Emerg. Rpt. LSCS	LBW	-	-	-	-	-	-	-	-	-

										preeclampsia													
Krishnaveni	19	Primi	5.8.18	12.5.19	33w 2d	33w 4d	1.63	60	22.6	Severe preeclampsia	++++	4.8	Emerg. LSCS	Early preterm	-	-	-	-	-	-	-	-	-
Parameshwari	21	Primi	9.8.18	16.5.19	38w 1d	38w	1.59	72	28.5	GHT since 6 month of GA	++++	6.4	LN	LBW	-	-	-	-	-	-	-	-	-
Selvammal	20	Primi	10.8.18	17.5.19	36w 6d	37w	1.43	56	27.4	Severe preeclampsia	++++	7.8	Emerg. LSCS	IUD	1	-	-	-	-	-	-	-	-
Lakshmi Devi	29	G3P2 L2	16.8.18	23.5.19	37w 5d	37w 5d	1.49	39	17.6	Non severe preeclampsia	Nil	4.4	Elective Rpt. LSCS	Term baby	-	-	-	-	-	-	-	-	-
Visalatchi	25	G2P1 L1	22.8.18	29.5.19	32w 3d	32w 1d	1.56	43	17.7	HELLP Syndrome	++++	7.2	Emerg. LSCS	Early Preterm	-	-	1	-	-	-	-	-	-
Revathy	18	Primi	25.8.18	2.6.19	32w 1d	32w 3d	1.52	46	19.9	Non severe preeclampsia	+	5.7	LN	Early Preterm	-	-	-	-	-	-	-	-	-
Priya Dharshini	26	G2P1 L1	30.8.18	7.6.19	38w 3d	38w 6d	1.47	42	19.4	GHT since 2 weeks	Nil	5.3	LN	Term baby	-	-	-	-	-	-	-	-	-
Thara	24	G2P1 L1	7.9.18	14.6.19	37w 2d	37w 1d	1.42	39	19.3	GHT since 8 month of GA	Nil	5.8	Emerg. Rpt. LSCS	Term baby	-	-	-	-	-	-	-	-	-
Swarna	18	Primi	9.9.18	16.6.19	38w 1d	38w	1.5	59	26.2	Non severe preeclampsia	Nil	5.2	LN	Mild IUGR	-	1	-	-	-	-	-	-	-
Sakunthala	23	G2P1 L1	9.9.18	16.6.19	31w 3d	31w 2d	1.53	44	18.8	GHT since 1 month	Nil	4.9	LN	Early Preterm	-	-	-	-	-	-	-	-	-
Rani	27	G3P2 L2	13.9.18	20.6.19	37w 6d	37w 5d	1.57	48	19.5	Recurrent GHT	Nil	6.4	Emerg. Rpt. LSCS	LBW	-	-	-	-	-	-	-	-	-
Swarnalatha	32	G3P1 L1A1	15.9.18	22.6.19	35w 2d	35w 2d	1.52	46	19.9	Severe preeclampsia	++++	6.8	Emerg. Rpt. LSCS	Severe IUGR	-	1	-	-	-	-	-	-	-
Padmini Priya	28	Primi	18.9.18	25.6.19	34w	34w 2d	1.51	57	25.0	AP Eclampsia	++++	10	Emerg. LSCS	IUD	1	-	-	-	1	-	-	-	-

Sara	22	G2P1 L1	21.9.18	28.6.19	38w 2d	38w 1d	1.39	48	24.8	newly diagnosed GHT	++	4.7	LN	Term baby	-	-	-	-	-	-	-	-	-
Malavika	18	Primi	23.9.18	30.6.19	37w 34d	37w 2d	1.49	60	27.0	GHT since 1 week	Nil	4.6	LN	Term baby	-	-	-	-	-	-	-	-	-
Usha	26	Primi	27.9.18	4.7.19	37w 3d	37w 3d	1.56	77	31.6	Non severe preeclampsia	Nil	5.6	LN	LBW	-	-	-	-	-	-	-	-	-
Bamini	21	G2P1 L1	27.9.18	4.7.19	34w 2d	33w 5d	1.55	48	20.0	Severe preeclampsia	++++	6.7	Emerg. Rpt. LSCS	Early Preterm	-	-	-	-	-	-	-	-	-
Andrea	24	G3P2 L2	27.9.18	4.7.19	38w 6d	38w 3d	1.59	53	21.0	GHT since 7 month of GA	Nil	5.2	Elective Rpt. LSCS	LBW	-	-	-	-	-	-	-	-	-
Nisha	19	Primi	29.9.18	6.7.19	37w 4d	37w 5d	1.53	55	23.5	Severe preeclampsia	++++	10.2	Emerg. LSCS	IUD	1	-	-	-	-	-	-	-	-
Priya	22	G2P1 L1	30.9.18	7.7.19	37w 6d	38w	1.46	42	19.7	Non severe preeclampsia	++	5.5	Emerg. Rpt. LSCS	Term baby	-	-	-	-	-	-	-	-	-
Banumathi	26	G2P1 L1	1.10.18	8.7.19	32w 3d	32w 3d	1.51	38	16.7	Newly diagnosed GHT	Nil	6	LN	Early Preterm	-	-	-	-	-	-	-	-	-
Shankari	21	G2P1 L1	2.10.18	9.7.19	34w 2d	34w	1.52	44	19.0	Severe preeclampsia	++++	5.8	Emerg. Rpt. LSCS	Mild IUGR	-	1	-	-	-	-	-	-	-
Devayani	18	Primi	2.10.18	9.7.19	38w	38w 1d	1.46	55	25.8	Non severe preeclampsia	++	4.7	LN	Term baby	-	-	-	-	-	-	-	-	-
Radha	22	G2P1 L1	2.10.18	9.7.19	37w 3d	37w 2d	1.42	52	25.8	GHT since 1 week	Nil	5.5	Emerg. Rpt. LSCS	LBW	-	-	-	-	-	-	-	-	-
Kaleeshwari	19	Primi	4.10.18	11.7.19	35w 5d	36w	1.6	56	21.9	Newly diagnosed GHT	Nil	6.2	LN	Late Preterm	-	-	-	-	-	-	-	-	-
Gomathi	27	G2P1 L1	4.10.18	11.7.19	38w 2d	38w 5d	1.56	73	30.0	Non severe preeclampsia	Nil	5.8	Elective Rpt. LSCS	Term baby	-	-	-	-	-	-	-	-	-

Pandiyammal	19	Primi	5.10.18	12.7.19	31w 1d	30w 6d	1.49	56	25.2	Non severe preeclampsia	Nil	6.1	LN	Early Preterm	-	-	-	-	-	-	-	-	-
Pandi Meena	21	Primi	6.10.18	13.7.19	27w 5d	27w 5d	1.55	39	16.2	Severe preeclampsia	++++	6.8	Emerg. LSCS	Extreme Preterm	-	-	-	-	-	-	-	-	-
Yaamini	23	Primi	6.10.18	13.7.19	38w 2d	38w 3d	1.53	45	19.2	GHT since 7 month of GA	Nil	5.2	Elective LSCS	LBW	-	-	-	-	-	-	-	-	-
Karunai Devi	28	G2P1 L1	6.10.18	13.7.19	34w 2d	34w 3d	1.47	47	21.8	AP Eclampsia	++++	9.4	Emerg. Rpt. LSCS	IUD	1	-	-	-	1	-	-	-	-
Julie	29	G2P1 L1	6.10.18	13.7.19	37w	37w	1.62	59	22.5	Newly diagnosed GHT	++	4.7	LN	Term baby	-	-	-	-	-	-	-	-	-
Sneha Rani	25	G2P1 L1	8.10.18	15.7.19	38w 4d	38w 4d	1.57	72	29.2	Non severe preeclampsia	++	5.3	Elective LSCS	Term baby	-	-	-	-	-	-	-	-	-
Ilakiya Priya	24	G3P2 L2	8.10.18	15.7.19	36w 3d	36w 1d	1.59	65	25.7	Recurrent GHT	Nil	5.8	Elective Rpt. LSCS	Mild IUGR	-	1	-	-	-	-	-	-	-
Siva Selvi	25	G2P1 L1	11.10.18	18.7.19	37w 6d	37w 4d	1.47	57	26.4	Severe preeclampsia	++++	5.2	Emerg. Rpt. LSCS	Term baby	-	-	-	-	-	-	-	-	-
Nivedhitha	19	Primi	12.10.18	19.7.19	31w 4d	32w	1.42	38	18.8	Non severe preeclampsia	++	6.2	LN	Early Preterm	-	-	-	-	-	-	-	-	-
Pranitha	33	G3P1 L1A1	12.10.18	19.7.19	38w 2d	38w 2d	1.52	46	19.9	GHT since 1 month	++++	5.8	LN	LBW	-	-	-	-	-	-	-	-	-
Gayathri	24	G2P1 L1	14.10.18	21.7.19	36w 5d	37w 1d	1.64	77	28.6	Newly diagnosed GHT	Nil	5.7	LN	LBW	-	-	-	-	-	-	-	-	-
Ipshitha	19	Primi	14.10.18	21.7.19	35w 4d	35w 6d	1.57	62	25.2	GHT since 6 month of GA	Nil	6	LN	Late Preterm	-	-	-	-	-	-	-	-	-
Mahalakshmi	26	G2P1 L1	14.10.18	21.7.19	38w 4d	38w 5d	1.54	58	24.5	GHT since 1 week	++	6.2	LN	Term baby	-	-	-	-	-	-	-	-	-
Anjana Devi	30	G4P1 L1A2	14.10.18	21.7.19	37w 4d	37w 5d	1.4	43	21.9	Non severe	+	5.7	LN	Term baby	-	-	-	-	-	-	-	-	-

										preeclampsia													
Kousalya	26	G3P1 L1A1	14.10.18	21.7.19	30w 2d	30w 4d	1.53	47	20.1	GHT since 7 month of GA	Nil	5.8	Elective Rpt. LSCS	Early preterm	-	-	-	-	-	-	-	-	-
Kalai Selvi	19	Primi	15.10.18	22.7.19	34w 2d	34w 5d	1.5	52	23.1	Severe preeclampsia	++++	6.7	LN	Severe IUGR	-	1	-	-	-	-	-	-	-
Varalakshmi	24	Primi	15.10.18	22.7.19	38w	37w 6d	1.55	54	22.5	HELLP Syndrome	++++	7.1	Emerg. LSCS	LBW	-	-	1	-	-	-	-	-	-
Bavatharani	27	G2P1 L1	16.10.18	23.7.19	37w 2d	37w 2d	1.55	51	21.2	GHT since 2 weeks	Nil	5.8	LN	Term baby	-	-	-	-	-	-	-	-	-
Priya Latha	32	G2P1 L1	17.10.18	24.7.19	38w	38w	1.52	68	29.4	Severe preeclampsia	++++	6.1	Emerg. Rpt. LSCS	LBW	-	-	-	-	-	-	-	-	-
Devika	18	Primi	17.10.18	24.7.19	37w 3d	37w 4d	1.46	53	24.9	Non severe preeclampsia	+	4.8	LN	Term baby	-	-	-	-	-	-	-	-	-