"URINE PROTEIN TO CREATININE RATIO FOR EVALUATION OF PREECLAMPSIA AND ITS MATERNAL AND FETAL OUTCOME; A CROSS SECTIONAL STUDY"

By

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SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, KOLAR, KARNATAKA
IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF
MASTER OF SURGERY IN
OBSTETRICS AND GYNAECOLOGY

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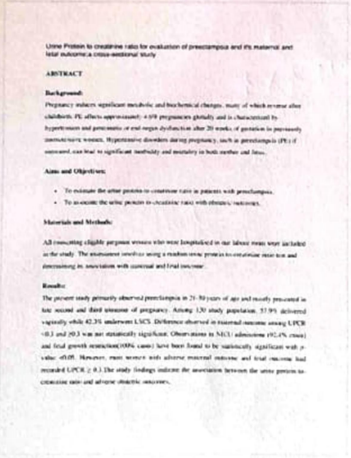

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ABSTRACT

Background:

Aims and Objectives:

- To estimate the urine protein to creatinine ratio in patients with preeclampsia.
- To associate the urine protein to creatinine ratio with obstetric outcomes.

Materials and Methods:

Results:

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Dr. ASHWINI KASHI

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LIST OF ABBREVIATIONS

ACOG	American College of Obstetricians and Gynecology
ALP	Alkaline Phosphatase
ALT	Alanine Aminotransferase
APTT	Activated Partial Thromboplastin Time
AST	Aspartate Aminotransferase
BP	Blood Pressure
CBC	Complete Blood Count
CSF1	Colony Stimulating Factor 1
DB	Direct Bilirubin
DBP	Diastolic Blood Pressure
DNA	Deoxyribonucleic acid
ET	Endothelin
FGR	Fetal Growth Restriction
GGT	Gamma-Glutamyl Transferase
GFR	Glomerular Filtration Rate
GNB3	G-Protein Subunit Beta 3
Hb	Hemoglobin
HELLP	Hemolysis, Elevated Liver enzymes and Low Platelets
HLA	Human leukocyte antigen
ICU	Intensive Care Unit
IGF	Insulin like growth factor
IL	Interleukin
INR	International Normalized Ratio
IUD	Intrauterine fetal demise
LDH	Lactate Dehydrogenase
LSCS	Lower Segment Cesarean Section

NK	Natural killer cells
NICU	Neonatal Intensive Care Unit
NO	Nitric Oxide
PE	Preeclampsia
PGI2	Prostacyclin
PIERS	Preeclampsia Integrated Estimate of Risk
PIH	Pregnancy Induced Hypertension
PIGF	Placental Growth Factor
POG	Period of gestation
PREP	Prediction of Risks in Early onset Pre-eclampsia
PT	Prothrombin Time
RAAS	Renin-Angiotensin-Aldosterone System
RBC	Red Blood Cells
SBP	Systolic Blood Pressure
sEng	Soluble Endoglin
sFlt-1	Soluble fms-like Tyrosine Kinase-1
SGOT	Serum Glutamic-Oxaloacetic Transaminase
SGPT	Serum Glutamic-Pyruvic Transaminase
SNP	Single Nucleotide Polymorphism
TB	Total Bilirubin
TGF-beta	Transforming Growth Factor-beta
THBS4	Thrombospondin 4
TNF-alpha	Tumor Necrosis Factor-alpha
TXA2	Thromboxane A2
UPCR	Urine Protein-to-Creatinine Ratio
VEGF	Vascular Endothelial Growth Factor
WBC	White Blood Cells

ABSTRACT

Background:

Pregnancy induces significant metabolic and biochemical changes, many of which reverse after childbirth. PE affects approximately 4.6% pregnancies globally and is characterized by hypertension and proteinuria or end-organ dysfunction after 20 weeks of gestation in previously normotensive women. Hypertensive disorders during pregnancy, such as preeclampsia (PE) if untreated, can lead to significant morbidity and mortality in both mother and fetus.

Aims and Objectives:

- To estimate the urine protein-to-creatinine ratio in patients with preeclampsia.
- To associate the urine protein-to-creatinine ratio with obstetric outcomes.

Materials and Methods:

All consenting eligible pregnant women who were hospitalised in our labour room were included in the study. The assessment involved a random urine protein-to-creatinine ratio test and determining its association with maternal and fetal outcome.

Results:

The present study primarily observed preeclampsia in 21-30 years of age and mostly presented in late second and third trimester of pregnancy. Among 130 study population, 57.9% delivered vaginally while 42.3% underwent LSCS. Difference observed in maternal outcome among UPCR <0.3 and ≥0.3 was not statistically significant. Observations in NICU admissions (92.4% cases) and fetal growth restriction (100% cases) have been found to be statistically significant with *p*-value <0.05. However, most women with adverse maternal outcome and fetal outcome had recorded UPCR ≥ 0.3. The study findings indicate the association between the urine protein-to-creatinine ratio and adverse obstetric outcomes.

Conclusion:

Monitoring the urine protein-to-creatinine ratio in preeclamptic patients has both diagnostic and prognostic value, aiding in evaluating disease progression and making clinical decisions about delivery timing. This method offers a feasible and faster alternative to the 24-hour urine protein test, improving patient compliance and early intervention.

Keywords:

Preeclampsia, urine protein-to-creatinine ratio, proteinuria, hypertension, obstetric outcomes, maternal-fetal medicine.

INTRODUCTION

Pregnancy induces extensive metabolic and biochemical changes, many of which reverse after childbirth. Pregnancy-related health issues are the main emphasis of maternal-fetal medicine, which also includes postpartum care. Hypertensive disorders during pregnancy, which have been long recognized as major complications, illustrate the challenge of addressing only one aspect of the maternal-fetal unit. These conditions are important contributors to the mortality and morbidity of both fetus and mother.[1-3]

Preeclampsia (PE) is marked by development of hypertension beyond gestational age of 20 weeks in women who were previously normotensive, along with proteinuria or end-organ dysfunction. PE contributes significantly to health issues of mother and fetus, affecting approximately 4.6% of pregnancies globally. Untreated PE has significant contribution to the death of the mother in developing countries, mainly eclampsia.[4] Incidence of PE in 2-8% of pregnancies is linked with high feto-maternal morbidity and mortality, leading to increased healthcare costs.[5] Preeclampsia suggests that individuals with severe proteinuria and ongoing hypertension during pregnancy may have tonic-clonic seizures in the absence of prophylaxis.[6]

PE is a disorder involving multiple systems causing dysfunction in kidneys, liver, lungs, nervous system and hematologic system.[7, 8] It can also lead to fetal complications such as oligohydramnios, fetal growth restriction, prematurity, abruption placenta and perinatal death.[9, 10]

PE is a disorder with two stages. The first stage involves defect in invasion of trophoblast and remodelling of spiral artery, decreasing uteroplacental blood flow and causing hypoxia. Thus resulting in oxidative-stress in placenta, leading to the release of cell-free fetal DNA, anti-angiogenic substances and pro-inflammatory cytokines into the mother's circulation, triggering dysfunctioning of endothelium and increases vascular permeability.[11-13] This pathological cascade has led to use of biomarkers as early predictor of PE.[14] Quick diagnosis of patients with high-risk has allowed for prophylaxis with aspirin and timely interventions.[15]

Proteinuria, defined as urinary-protein excretion of more than 150 mg/day in a 24-hour urine sample, indicates increased glomerular permeability to plasma macromolecules like albumin. Healthy individuals excrete < 150 mg/day of urinary protein. Urine proteins are made up of 40% tubular Tamm-Horsfall protein and 60% plasma proteins, mostly albumin. Proteinuria in pregnancy is urinary protein excretion of >300 mg/day in a 24-hour urine.[16] In PE, proteinuria reflects the severity of the condition, although its absence does not rule out PE.[17]

Proteinuria in patients with PE results due to raised permeability of renal tubules to high molecular- weight proteins like albumin, transferrin, hemoglobin and globulin.[18] Renal tubular damage in PE is a result of both low nitric oxide levels and high levels of circulating sFlt-1. sFlt-1 inhibits VEGF, causing glomerular endothelial injury known as glomerular endotheliosis, a hallmark of PE.[17] Swollen mesangial cells, tubular casts, subendothelial protein deposits from the glomerular filtrate and swollen endothelial cells with vacuoles are the hallmarks of glomerular endotheliosis.[18] Elevated sFlt-1 levels in the blood inhibit VEGF which is podocyte specific, disrupting the barrier for glomerular filtration and forming fenestrae that contribute to proteinuria.[6] Podocyte damage is the reason for proteinuria. Nephrin, podocin, synaptopodin and podocalyxin are slit diaphragm proteins that are essential for preserving the glomerular barrier's integrity.

Proteins are detectable in urine before the clinical symptoms of PE appear. Proteins implicated in the complement pathway, coagulation cascade and RAAS are found in high levels in urine of patients with PE. Frequently, these proteins are misfolded. Podocyte injury results from proangiogenic and antiangiogenic factors imbalance, which raises the risk of end-stage renal disease in PE, persistent proteinuria, ischemic heart disease, hypertension and stroke. After PE, the length of time it takes to resolve proteinuria is positively associated with both the severity of PE and the delivery time.[6]

Kidney dysfunction in PE is “serum creatinine levels above 1.1 mg/dl or a doubling of baseline creatinine”. PE often results in decreased renal perfusion and glomerular filtration rate.[11] Fibrin deposition, enlargement of endothelium, loss of

podocyte and glomerular endotheliosis—the obliteration of capillary spaces—are all seen in renal biopsies of PE patients. Glomerular endotheliosis has dysregulation of glomerular filtration apparatus.[16] Endothelial cells in healthy pregnancies transition to a procoagulant state due to increased tissue factor release from placenta and maternal decidua.[16] Increased proinflammatory cytokines in PE cause leukocytes and endothelial cells to express tissue factors. Renal thrombotic microangiopathy is caused by damaged endothelial cells that cause clot formation and lose their anticoagulant qualities when prostaglandin and nitric oxide levels drop. Elevated levels of inflammatory cytokines are caused by increased expression of toll-like receptor-4 and this worsens renal and placental dysfunction.[10] Because of increased tubular calcium reabsorption, urine calcium decreases which causes electrolyte imbalances.[13] Salt and free-water retention are increased in PE when the intravascular volume is reduced.[13] Patients are more likely to experience acute kidney injury and renal dysfunction as a result of mechanisms involving sFlt-1 and RAAS system that cause hypertension thus, developing chronic kidney disease and subsequent hypertension.[13]

When making decisions for delivery in preeclampsia, obstetricians ought to take proteinuria into account. It's crucial to understand that urine protein excretion rises throughout pregnancy but is abnormal when > 300 mg in a 24-hour urine sample.[10] Proteinuria is a stand-alone risk factor and predictor of end- organ damage in PE. Monitoring protein excretion is useful for prognostication as well as diagnosis, aiding in the assessment of disease progression. Unaffected by hydration status, the UPCR standardizes protein excretion in relation to glomerular-filtration-rate.[17]

Although the 24-hour urine-protein test is the gold standard for measurement of proteinuria, it has limitations such as prolonged detection time, inconvenience, preanalytical errors, sampling errors and poor patient compliance.[18] The random UPCR test, extensively studied and recommended as it is accurate, feasible and quicker results compared to the 24-hour urine-protein test and hence, a simple and effective alternative.[19]

NEED FOR STUDY

According to National Health Portal of India, the incidence of preeclampsia is 8-10 % among pregnant women and contributes to 14-16% of maternal deaths. In our tertiary care center, 40-45% of pregnant women who are referred have preeclampsia in late stage. A non-invasive test like Urine protein-to-creatinine ratio is unaffected by hydration status and standardizes protein excretion in relation to glomerular-filtration-rate.

UPCR has a positive correlation with clinical parameters like blood pressure, thrombocytopenia, abnormal liver function tests, eclampsia, renal dysfunction, thrombocytopenia, fetal growth restriction, prematurity, low birth weight and neonatal death within one week of life. It predicts pregnancies at risk of adverse maternal and fetal outcome and their prognosis and is necessary for timely clinical interventions. Proteinuria alone is recognized as an independent risk factor and a major predictor of end organ damage.

AIMS AND OBJECTIVES

1. To estimate urine protein-to-creatinine ratio in patients with preeclampsia
2. To associate urine protein-to-creatinine ratio with obstetric outcome

REVIEW OF LITERATURE

HISTORICAL ASPECTS:

Hippocrates documented preeclampsia and eclampsia for the first time in 400 B.C.and he associated headaches, heaviness and convulsions during pregnancy with poor outcomes. Ancient remedies included charms, amulets, faith-healing and prayers, reflecting orthodox beliefs. Over time, this shifted to more practical treatments like diet changes, purging and bloodletting.[19]

The understanding of preeclampsia and eclampsia was restricted until the late 20th century when it became a subspecialty of obstetric medicine. Early advances to our knowledge of puerperal convulsions were provided by Francois Mauriceau.[20] In the late 1800s, with the shift towards toxin theory in disease causation, women with headaches and edema were treated with bleeding and purging to prevent convulsions and eliminate toxins. Bossier de Sauvages (1710-1795) first used the term eclampsia, meaning lightning in Greek, possibly referring to the sudden onset of convulsions. By 1739, eclampsia was identified as distinct from epilepsy, with seizures ceasing when the initiating cause was eliminated. Pierre Rayer (1793–1867) was the first to identify proteinuria in eclamptic patients, while Demanet (1797) initially reported severe edema in eclamptic women. Proteinuria in eclampsia was shown by John Lever (1811– 1859) to be exclusive to the condition rather than a symptom of a broader illness.

Simpson validated Lever's results. By the middle of the 1800s, symptoms such as headache, transient blindness, excruciating abdominal pain and upper body edema were identified as preeclamptic indications, a potentially fatal illness. The identification of preeclampsia as a hypertensive illness was facilitated by the invention of Scipione Riva-Rocci's mercury manometer (1896) for measuring blood pressure, with new- onset hypertension and proteinuria serving as crucial classification indicators.[21]

HYPERTENSIVE DISORDERS IN PREGNANCY:

Hypertension during pregnancy is defined as a systolic blood pressure of 140 mmHg or higher and/or a diastolic blood pressure of 90 mmHg or higher, confirmed on

at least two occasions 4 to 6 hours apart within a week.[22] Preeclampsia is a multi-systemic illness unique to pregnancy and the puerperium, which resolves by 12 weeks postpartum and is characterized by hypertension and proteinuria after 20 weeks of gestation. It is a significant contributor to maternal and neonatal morbidity and mortality, affecting 2-8% of pregnancies. Severe preeclampsia is characterized by abrupt onset, excessive weight gain, epigastric pain, headaches, scotoma, blurring of vision or blindness in women with hypertension. Non-dependent edema, in particular, affects the face, hands and ankles.[23]

Preeclampsia is classified as a systemic vascular illness and it is thought that the endothelium is the disease's target because of both hypertension and proteinuria. Peripheral vasoconstriction and reduced arterial compliance are features of preeclamptic hypertension.[24] Research indicates that quite a few of patients with eclampsia before or after 32 weeks of gestation do not experience edema.[25]

DEFINITION:

- **Pregnancy-Induced Hypertension (PIH):** PIH is defined as hypertension (blood pressure ≥ 140/90 mmHg) with or without proteinuria (≥ 300 mg/24 hours) occurring after 20 weeks of gestation and resolving up to 12 weeks postpartum. It also includes new-onset proteinuria (≥ 300 mg/24 hours) in hypertensive women without prior proteinuria before 20 weeks of gestation.[26]
- **Preeclampsia (PE):** A multisystem disorder of unknown etiology characterized by hypertension (blood pressure ≥ 140 / 90 mmHg) with proteinuria exceeding 300 mg/24 hours, emerging after the 20th week of gestation and resolving by 12 weeks postpartum.[26]
- **Eclampsia (E):** Defined by the onset of convulsions in a woman with PIH not attributable to other causes. Seizures may occur before, during, or after labor.[26]

EPIDEMIOLOGY:

Preeclampsia affects 3-5% of pregnant-women globally, while hypertensive-disorders affect 10% of them. Pre-eclampsia, eclampsia, chronic-hypertension and

gestational-hypertension are the four ways in which these conditions present. Pregnancy-related hypertension illnesses are linked to around 10% of maternal fatalities. Preeclampsia has been found to affect 8–10% of pregnant women in India. Research indicates that 7.8% of pregnant women have hypertensive disorders, and 5.4% of those women have preeclampsia.[27]

A 10% prevalence of hypertension was found in all pregnancies, according to community intervention studies conducted by Magee L et al. for Pre-eclampsia (CLIP) trials in 27 geographical clusters from India, Pakistan, Mozambique and Nigeria (2013-2017). About 7–8% of pregnancies with hypertension were "gestational", meaning they had no other complications besides hypertension. Up to one in ten pregnant women may develop hypertensive problems, according to the study. Mozambique had the highest prevalence among the four countries, while Pakistan had the lowest. More than 75% of instances of hypertension in India were not severe, and the condition was identified early through extensive blood pressure monitoring.

About 40% of cases of hypertension were discovered after delivery, with most cases being identified in the prenatal and mid-late third trimester. With a median diagnosis of 10 days postpartum, India had the lowest incidence of postpartum hypertension compared to other countries where the typical diagnosis was 7 days. India and Pakistan had lower rates of chronic hypertension before 20 weeks of pregnancy than Mozambique and Nigeria. Hypertension was mostly diagnosed during the antenatal period in India and Mozambique, while about half were diagnosed in Pakistan and Nigeria. Preeclampsia was observed at almost the same frequency in all four countries: the most prevalent kind of hypertension was chronic (<1.0%), gestational hypertension was most common (6%–12%) and preeclampsia was detected in between (3%–6%).[28]

Genetic and non-genetic variables that increase the risk of gestational hypertension were found by Umesawa M et al. In contrast to non-modifiable risk factors, which include age, primipara, multiple pregnancies, history of hypertension in previous pregnancy, gestational diabetes mellitus, chronic hypertension, preexisting urinary tract infections and a family history of these diseases, modifiable risk factors include body mass index, anemia and illiteracy[27]. Beyond the

postpartum phase, hypertensive problems during pregnancy may result in long-term hazards such as dysrhythmia, stroke, heart failure, coronary heart disease, cardiomyopathy, persistent hypertension, diabetes-mellitus and end-stage renal dysfunction.[27]

RISK FACTORS:[29]

Factors predisposing to Preeclampsia include:

- Primigravida
- Multiple pregnancy
- Insulin resistance
- History of previous preeclampsia
- Inter-pregnancy interval
- Family history
- Race and ethnicity (genetic predisposition)
- Obesity
- Chronic hypertension
- Renal disorders
- Gestational diabetes
- Type 1 Diabetes Mellitus
- Antiphospholipid antibody syndrome
- Molar pregnancy
- Chromosomal anomalies (trisomy 13)
- Donor insemination

ETIOLOGY:

Several mechanisms have been proposed to explain the etiology of preeclampsia, including:

- Abnormal placental implantation and trophoblastic invasion of uterine vessels.
- Immunological maladaptation between maternal, paternal (placental), and fetal tissues.
- Maternal maladaptation to cardiovascular or inflammatory changes of normal pregnancy.
- Genetic factors, including inherited predisposing genes and epigenetic influences.[30]

Abnormal-placentation:

In the pathophysiology of preeclampsia, the placenta is crucial. Research suggests that the condition can be cured by removing the placenta, however the placenta is necessary for embryonic growth. Abnormalities such infarcts, atherosis, thrombosis and chronic inflammation are found in the placentas of women with severe preeclampsia according to pathological tests.[31] The theory that preeclampsia is a systemic illness with extensive maternal endothelial dysfunction that originates in the placenta is supported by the two-stage disorder hypothesis.[32]

Two-stage disorder hypothesis:

Abnormal placentation occurs during the first stage. Normal pregnancy causes the spiral arteries' walls to be invaded by endovascular trophoblastic cells, which then grow into long, winding channels that are resistant to vasomotor drugs. Preeclampsia results in diminished uteroplacental perfusion and a persistent responsiveness to vasomotor effects because this change is incomplete.[33] Lindheimer, Roberts et al., discovered that cytotrophoblasts take on an endothelial phenotype during a process known as pseudo-vasculogenesis, also known as vascular mimicry, during normal placental development. Because cytotrophoblasts do not make this transition, they cannot sufficiently penetrate the spiral arteries of myometrium in preeclampsia.[34]

Pre-existing maternal problems such as diabetes, obesity, heart disease, kidney disease or genetic factors can have an impact on stage 2. This phase is characterized by a wide-spread hyper-inflammatory state and enhanced endothelial cell activation. Placental debris enters the mother's bloodstream as a result of oxidative stress, apoptosis and necrotic disruption of the syncytial architecture brought on by placental hypoxia or reperfusion episodes. This induces aberrant lipid peroxidation, influences angiogenesis and stimulates the manufacture of cytokines, all of which culminate in a systemic inflammatory response. Multiple organ compromise symptoms and indicators are brought on by these alterations.[33] Defective trophoblastic invasion size is correlated with hypertension severity.[34]

Immunological factors:

The dysregulation of maternal immune tolerance to paternal placental and fetal antigens is another theory that explains preeclampsia.[35] NK cell cytotoxicity and cytokine-production are controlled by natural cytotoxicity receptors. Pregnant patients with preeclampsia exhibit aberrant natural cytotoxicity receptor immunology on peripheral blood NK cells. Higher NK1 cytokine production in preeclamptic women may be explained by lower NKp46 (+) NK cells.[36] Endometrial mesenchymal cells, which express and secrete HLA-G, are involved in immunological modulation that is essential for good implantation and normal trophoblast invasion.[37] Extravillous trophoblasts exhibit decreased immunosuppressive non-classic HLA-G early in pregnancy in preeclamptic women, which results in impaired placental vascularization. Stage I preeclampsia was exacerbated by this. Placental debris and adipocytes promote an inflammatory response that is brought on by increased Th1 action and alterations in Th1/Th2 ratio in early stages of second trimester. Preeclampsia results from damage to endothelial cells brought on by this inflammatory cytokine.[33]

Endothelial cell activation:

A series of actions start in reaction to placental factors released because of ischemia alterations or other reasons.[38] It is believed that several inflammatory mediators, antiangiogenic agents and metabolic variables cause endothelial cell damage. Oxidative stress is brought on by cytokines like TNF-alpha and IL- 1, which release free radicals that multiply into self-propagating lipid-peroxides.[39] Lipid peroxides produce harmful radicals damaging endothelial cells, change the amount of prostaglandin produced and reduce the generation of nitric oxide. The lipid-laden macrophages and foam cells that are present in atherosclerotic plaque are also caused by oxidative stress. Edema and proteinuria occur as a result of raised capillary permeability, while microvascular coagulation activation produces thrombocytopenia.

Genetic factors:

Preeclampsia is a complex illness involving multiple genes. Research indicates that girls of preeclamptic mothers have a 20–40% incident risk for preeclampsia, sisters of preeclamptic women have an 11–37% incident risk, and twins have a 22-47%

incident risk.[40] In approximately 350 pairs of PE mothers and kids and 600 control pairs, 775 SNPs in 190 genes were assessed as part of a comprehensive genetic association study conducted by Goddard et al. They found that six genes—IGF1, IL4R, IGF2R, GNB3, CSF1 and THBS4—have a substantial maternal-fetal genotype interaction associated with PE.[41]

PATHOGENESIS:

Vasospasm*:*

Volhard began researching vasospasm in 1918. High vascular resistance from constricted arteries results in hypertension. Endothelial damage results in interstitial leakage, which sub-endothelial deposits blood components including platelets and fibrinogen. Reduced blood flow results in ischemia, leading to the syndrome's hallmark end-organ disruptions, bleeding and tissue necrosis.[30]

Endothelial cell injury:

Grundmann and colleagues reported elevated levels of circulating endothelial cells in preeclampsia.[42] In addition to secreting chemicals that promote coagulation and greater pressor sensitivity, damaged or activated endothelial cells produce less nitric oxide.[43]

Increased pressure response:

Refractoriness to infused vasopressors develops in pregnancy.[44] Increased vascular responsiveness to norepinephrine and angiotensin II infusion is observed in women with early preeclampsia.[45]

Prostaglandins:

The pathophysiology of preeclampsia involves many prostanoids. Reduced vascular reactivity caused by endothelial prostaglandin production is one reason for the muted pressor response in a typical pregnancy.[30] Phospholipase A2 mediates the decrease in endothelial prostacyclin (PGI2) synthesis in preeclampsia when compared to

a normal pregnancy.[38] Vasoconstriction and angiotensin II sensitivity are enhanced by increased thromboxane-A2 secretion and decreased prostacyclin: thromboxane-A2 ratio.[46]

Nitric oxide (NO):

Endothelial cells use L-arginine to generate NO, a powerful vasodilator. The inhibition of NO production reverses pregnancy-induced refractoriness to vasopressors and raises mean arterial pressure while lowering heart rate. In humans, NO preserves the vasodilated condition essential for feto-placental perfusion.[47] Preeclampsia is linked to reduced endothelial NO synthase expression, which increases NO inactivation. The implications of NO synthesis in this disease are not known.[38]

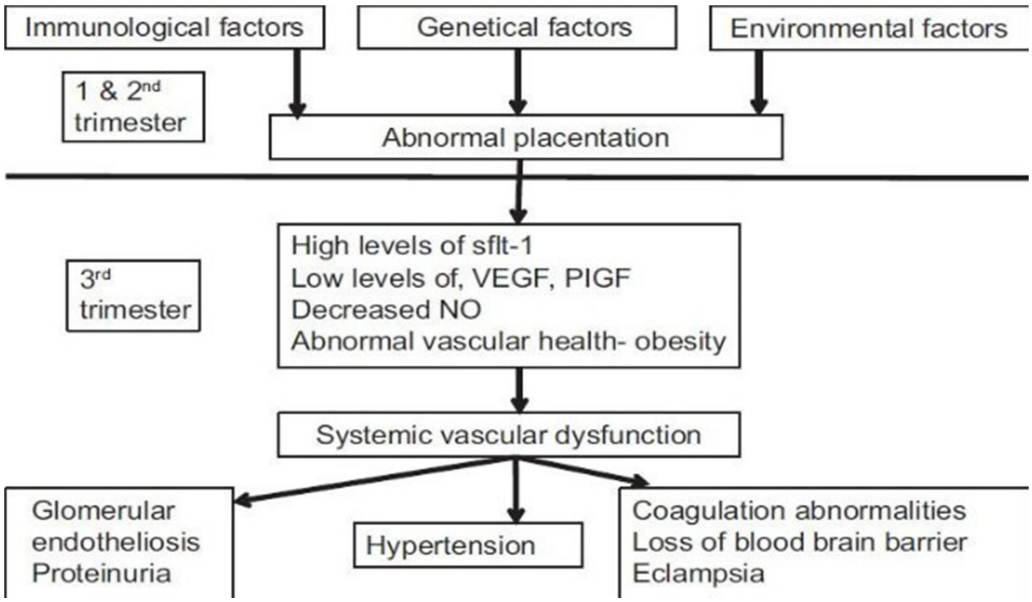
Endothelins (ET):

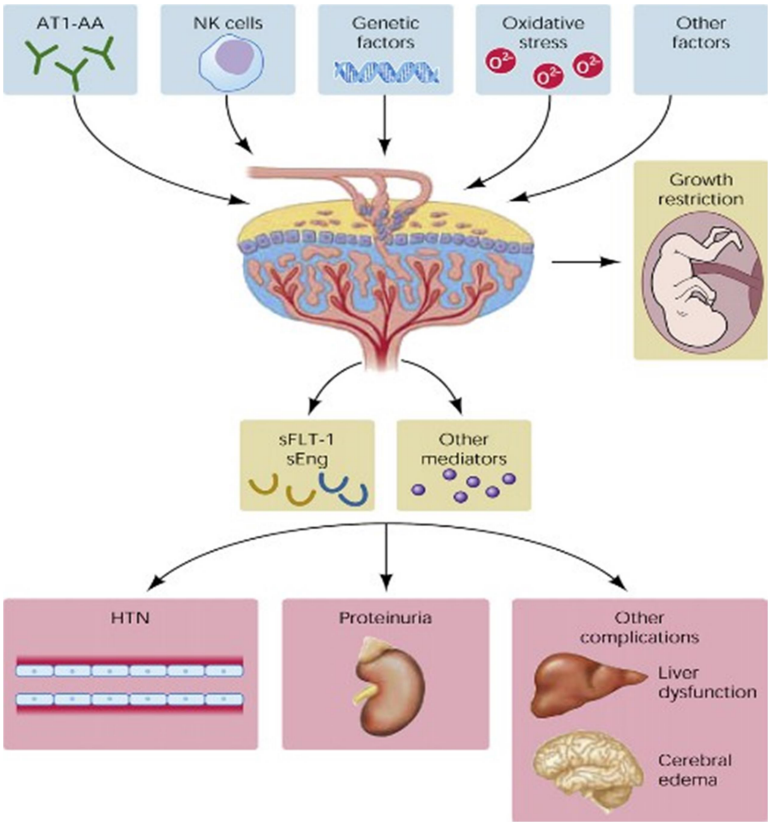
Endothelins are peptides with 21 amino acids that have strong vasoconstriction properties. Pregnant normotensive women had increased plasma ET-1 levels, with greater levels in preeclampsia.[48] Elevated ET-1 results from systemic endothelial activation and not from the placenta.[49]

Angiogenic and anti-angiogenic factors:

"Angiogenic imbalance" is high levels of anti-angiogenic factors at uteroplacental interface that are induced by worsening hypoxia. Women who are at risk of preeclampsia have trophoblasts that overproduce sFlt-1 and sEng, two antiangiogenic peptides. Months before onset of preeclampsia, soluble fms-like tyrosine kinase-1 (sFlt-1) are elevated in the maternal serum. Endothelial dysfunction is caused by increased sFlt-1, which also reduces and inactivates circulating free PIGF and VEGF.[29] Soluble endoglin (sEng) inhibits endoglin-co-receptor for TGF-beta family, hence blocking TGF-beta isotypes from binding to endothelial receptors and decreasing endothelial NO-dependent vasodilation.[50]

FIGURE 1: PATHOGENESIS OF PRE-ECLAMPSIA:[32]





CLINICAL FEATURES OF PRE-ECLAMPSIA:

- **Hypertension**: BP ≥ 140/90 mmHg or increase in systolic BP by 30 mmHg or diastolic BP by 15 mmHg, due to excess pressor substances in circulation. [51]
- **Proteinuria**: >0.3 g protein in 24-hour urine sample or protein (mg/dl)/creatinine (mg/dl) ratio of ≥0.3, or urine dipstick protein of 1+ (>30 mg/dl), due to glomerular afferent arteriole vasospasm causing protein leakage.[51]
- **Edema & Excess weight gain**: >1 lb weight gain in a week or >5 lb in a month in the last trimester, pitting edema over the ankle, due to decreased osmotic pressure and increased capillary permeability.
- **Epigastric pain**: Due to hepatocellular necrosis and stretching of Glisson's capsule.[51]
- **Thrombocytopenia**: Due to severe vasospasm leading to platelet aggregation and microangiopathic hemolysis.[51]
- **Other symptoms**: Headache and visual disturbances (blurring of vision) due to cerebral vasospam, reduced urine output due to decreased renal blood flow and GFR.[51]

SYSTEMIC CHANGES:

Hematological variations:

In severe preeclampsia, blood volume increases less than in normal pregnancy. Vasospasm causes hemoconcentration and elevated hematocrit levels by pushing intravascular fluid into the extravascular location. After eclampsia, hemolysis rises, leading to hemoglobinemia and hemoglobinuria. Preeclampsia is characterized by hypercoagulability; however, thrombocytopenia and coagulopathy develop later as a result of the consumption of platelets and coagulation factors. Preeclampsia is associated with platelet activation, characterized by increased degranulation, thromboxane A2 production, and a reduced RBC lifespan. There are elevated amounts of thrombin, D-dimer and fibronectin.[52–54]

Liver function:

Arteriole thrombosis causes periportal hemorrhagic necrosis and because of the liver's reserve , and hepatic insufficiency is uncommon because of the liver's reserve capacity and regenerating abilities of cases, have abnormal liver function tests. The symptoms of this condition include hematological and biochemical abnormalities, nausea, vomiting and pain in the right upper quadrant or epigastrium.[55–59]

Renal function:

Severe vasospasm damages vascular walls and endothelial integrity as it obstructs circulation in the vasa vasorum. In 10-15% of HELLP syndrome cases and 17% of eclamptic women, proteinuria may not be evident during convulsions, suggesting that the condition may develop later. The impairment of renal function, which affects both tubular and glomerular functions, advances with the severity of the disease.[60] Severe preeclampsia is implied by oliguria.

PREDICTORS FOR PREECLAMPSIA:[61, 62]

Preeclampsia in the early pregnancy can be predicted by multiple biochemical and clinical assays, but no one test is easy to use, valid and credible.

1. **NON-LABORATORY-METHODS.**
2. **LABORATORY-METHODS.**

1) NON-LABORATORY-METHODS:

a) History: Nulliparity, past/family history of hypertensive disorders.
b) Blood Pressure Monitoring.
c) Early pregnancy mean arterial pressure.

PRESSOR TESTS:

- **Roll-over test**: Lateral position followed by supine position measurement of blood pressure. Rise of ≥20 mmHg indicates a positive test. It has Poor sensitivity and specificity.[61, 62]
- **Isometric-exercise test**: Rise in systolic BP of 15 mmHg predicts preeclampsia with 81.8% sensitivity and 68.4% specificity, but poor

reproducibility.[61, 62]

- **Angiotensin-sensitivity test**: Determines vascular responsiveness by estimating amount of angiotensin-II required to elevate blood pressure by 20 mmHg, weeks before disease manifests. Poor specificity and sensitivity has been noted.[61, 62]

d) Doppler velocimetry.

- Uses Doppler imaging of uterine and umbilical arteries to assess reduced placental perfusion and uterine arterial resistance.

2) LABORATORY TESTS:

Micro-albuminuria:

Urinary excretion of > 300 mg of albumin in 24 hours is considered micro-albuminuria, a good predictor in the second trimester.

Urinary-Calcium / Creatinine Ratio:

Because of increased tubular reabsorption and decreased renal filtration, preeclampsia is linked to hypocalciuria, which is seen as a decreased calcium/creatinine ratio with 70% sensitivity and 95% specificity.

Serum uric-acid:

Elevated blood uric acid levels, however not very sensitive or specific, is a result of impaired glomerular filtration, increased tubular reabsorption and decreased tubular secretion in preeclampsia.

Serum fibronectin:

Fibronectin is a glycoprotein involved in cellular adhesion that is secreted after endothelial damage. In the third trimester, its level typically increases by 20%; but, in preeclampsia, the increase can be as high as 30%.

Serum anti-thrombin:

In preeclampsia, there is a reduction in antithrombin levels that fall between normotensive and high blood pressure values.

Free fetal DNA:

Endothelial cell activation, placental site ischemia and inflammation resulting in apoptosis releases free DNA into the bloodstream, are all involved in preeclampsia.

Homocysteine:

Hyperhomocysteinemia in preeclampsia causes damage to endothelium and release of free radicals.

Cytokines:

Increased cytokines are nonspecific since they are also generated by macrophages and vascular endothelium in inflammatory illnesses and infections.

Coagulation-activation:

Platelet dysfunction and thrombocytopenia are caused by endothelial damage, vasospasm and platelet aggregation in pregnancy.

DIAGNOSIS OF PRE-ECLAMPSIA AND ECLAMPSIA:

The American College of Obstetricians and Gynecology (ACOG) has updated the diagnostic criteria for preeclampsia, which no longer solely relies on proteinuria. The new criteria include hypertension after 20 weeks of gestation (≥140/90 mmHg on two occasions 4 hours apart or a single reading of ≥160/110 mmHg) with proteinuria of ≥300 mg/day or other symptoms such as thrombocytopenia, elevated creatinine or liver enzymes.[51]

Preeclampsia with severe features is diagnosed if any of the following are present:

- Systolic BP ≥160 mmHg or diastolic BP ≥110 mmHg on two occasions 4 hours apart.
- Headaches, visual symptoms or other neurological symptoms.
- Pulmonary edema.
- Platelet countn <1lakh cells/μL.
- Right upper quadrant pain not explained by other pathology or elevated AST/ALT.
- Creatinine >1.1 mg/dl or doubling of creatinine.

Maternal complications associated with preeclampsia can be severe and varied. These include accidental hemorrhage, which poses significant risks during pregnancy and delivery and acute renal failure, which can lead to long-term kidney damage. Cerebrovascular accidents, commonly known as stroke and HELLP syndrome are critical conditions that require immediate medical intervention. Microangiopathic hemolytic anemia and acute left ventricular failure with pulmonary edema further complicate the clinical picture. Eclampsia, characterized by seizures, and aspiration pneumonia are life-threatening emergencies. Other complications include preterm labor, sepsis and shock, which significantly increase maternal morbidity and mortality. There is a recurrence risk of preeclampsia in subsequent pregnancies and potential for long-term metabolic syndrome, predisposing affected women to diabetes and hypertension.[51]

Fetal complications arising from preeclampsia are equally concerning. Intrauterine death remains a devastating outcome, while prematurity is a frequent consequence due to the need for early delivery to mitigate maternal risks. Chronic

placental insufficiency often results in intrauterine growth restriction, impacting the baby's development. Neonatal encephalopathy, linked to systemic inflammatory responses and oxidative stress, poses a significant threat to the newborn's neurological development. Antepartum and intrapartum asphyxia as a result of reduced placental flow can lead to chronic hypoxia causing long-term adverse outcomes for infant.[51]

PRE-ECLAMPSIA PREVENTION:

Various methods proposed to prevent preeclampsia includes the use of:

Antioxidants:

There is a correlation between preeclampsia and elevated oxygen free radicals and reduced antioxidant levels. Specifically, vitamins C and E, which help reduce oxidative stress, are found in lower concentrations in at-risk women. Despite this, clinical trials have generally shown no significant decrease in pre-eclampsia incidence with antioxidant supplementation although some studies have indicated potential benefits.[63, 64]

Fish Oil Supplementation:

Fish oil, which contains alpha-linoleic acid and eicosapentaenoic acid, was initially thought to prevent atherogenesis due to its anti-inflammatory properties. However, recent studies have not demonstrated any beneficial effect of fish oil supplementation in preventing preeclampsia. Makrides et al., did not support the role of fish oil in preeclampsia prevention.[65]

Calcium Supplementation:

Research by Villar et al. indicated that women with less intake of calcium are at a higher risk of preeclampsia. Calcium supplementation does not significantly reduce the incidence of preeclampsia unless the woman is calcium deficient. Historical observations have linked calcium deficiency with preeclampsia, and protective effects of calcium supplementation have been noted particularly in women with less calcium intake.[66]

Aspirin (Low-Dose)

Aspirin (low dose) works by inhibiting thromboxane (TXA2) synthesis in platelets while having minimal impact on prostacyclin (PGI2). A meta-analysis of 31 trials revealed that antiplatelet agents significantly lowered preeclampsia risk by 10%.[67]

Heparin

Sergis F et al., evaluated the efficacy of heparin treatment in preeclampsia prevention. Their study found that women who were given a combination of aspirin (low-dose) with low molecular weight heparin had better pregnancy outcomes compared to those who received low-dose aspirin alone.[68]

MANAGEMENT OF PRE-ECLAMPSIA:

TABLE 1: Full PIERS or PREP model.[69]

	Hypertension: Blood pressure of 140/90–159/109mmHg	Severe hypertension: Blood pressure of 160/110 mmHg or more
Admission to Hospital	Admit if any clinical concerns for the wellbeing of the woman or baby or if high risk of adverse events suggested by the full PIERS or PREP-S risk prediction models	Admit, but if BP falls below 160/110 mmHg then manage as for hypertension
Antihypertensive pharmacological treatment	Offer pharmacological treatment if BP remains above 140/90 mmHg	Offer pharmacological treatment to all women
Target blood pressure once on antihypertensive treatment	Aim for BP of 135/85 mmHg or less	Aim for BP of 135/85 mmHg or less
Blood pressure Measurement	At least every 48 hours, and more frequently if the woman is admitted to hospital	Every 15–30 minutes until BP is less than 160/110 mmHg, then at least 4 times daily while the woman is an inpatient, depending on clinical circumstances
Dipstick proteinuria testing	Only repeat if clinically indicated, for example, if new symptoms and signs develop or if there is uncertainty over diagnosis	Only repeat if clinically indicated, for example, if new symptoms and signs develop or if there is uncertainty over diagnosis
Blood tests	Measure full blood count, liver function and renal function twice a week	Measure full blood count, liver function and renal function 3 times a week
Foetal assessment	Offer fetal heart auscultation at every antenatal appointment Carry out ultrasound assessment of the fetus at diagnosis and, if normal, repeat every 2 weeks	Offer fetal heart auscultation at every antenatal appointment Carry out ultrasound assessment of the fetus at diagnosis and, if normal, repeat every 2 weeks

TIMING OF BIRTH:

It is important to document the maternal and fetal thresholds for planned early births before 37 weeks. Thresholds include severe preeclampsia features like uncontrolled BP despite three or more antihypertensive classes, SpO2 < 90%, progressive hepatic or kidney dysfunction, neurological symptoms, abruption placenta, non-reassuring fetal heart rate or stillbirth.[69]

TABLE 2: Adverse impact of preeclampsia on fetus and mother.

On fetus	On mother
• Growth restriction	• Hypertension
• Preterm delivery	• Future HTN, CVD
• Placental abruption	• Kidney injury
• Respiratory distress	• Chronic kidney disease and risk for ESRD
• Cerebral palsy	• Liver failure
• Retinopathy of prematurity	• Cardiomyopathy
• Necrotizing enterocolitis	• CNS damage and stroke
• Sepsis	• Seizure
• Stillbirth	• Diabetes mellitus
	• Coronary artery disease
	• Pulmonary edema
	• Death

PATHOPHYSIOLOGY OF PROTEINURIA:

Glomerular-Tubular Protein Handling:

Healthy adults excrete < 150 mg of protein in 24-hours, and urine in non-pregnant women is almost completely protein-free.[70] Blood is selectively filtered from afferent arteriole to Bowman's space by glomerular filtration barrier, which is made up of the glomerular epithelium, basement membrane, and slit diaphragm.[71] A key factor in preserving membrane integrity and influencing protein filtration are podocytes, whose primary, secondary and tertiary processes comprise the slit diaphragm. Filtrate travels through Bowman's space and enters proximal tubule and Henle loop. Large proteins nearly never pass through an undamaged glomerular filter; proteinuria is the result of either excessive glomerular permeability or compromised tubular reabsorption. When tubular reabsorption capacity is saturated, as is case in pregnant women with underlying glomerular diseases, proteinuria of glomerular origin arises.[72]

Renal Adaptation in Pregnancy:

Urine protein levels double during a healthy pregnancy. In comparison to non-pregnant levels, early renal adaptation is characterized by 75% rise in renal plasma flow by 16 weeks and 50% rise in GFR by 5 to 7 weeks. GFR remains high throughout

pregnancy.[74] Despite being least accurate approach, creatinine clearance rises moderately (110–150 mL/min) during pregnancy. Pregnancy-related increases in GFR from hypervolemia, hemodilution and increased renal blood flow are hypothesized to be the cause of increased protein excretion. But timing of proteinuria during pregnancy raises possibility that changes in tubular reabsorption capacity are also relevant.[75]

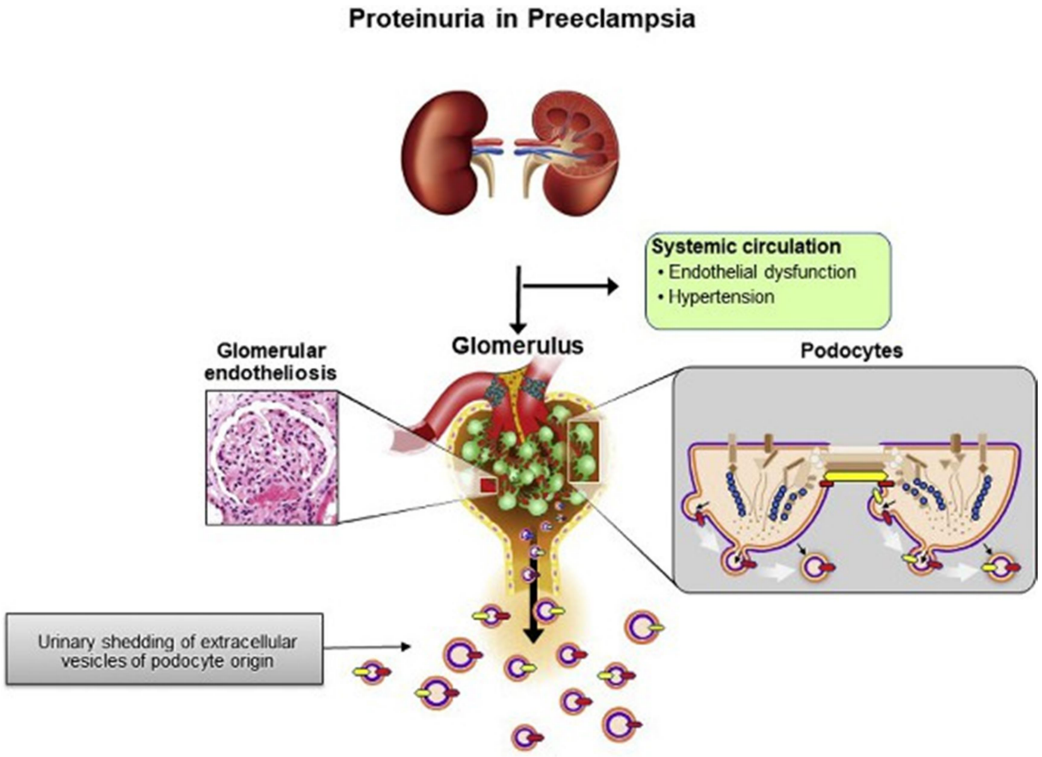
Pathophysiology of proteinuria in preeclampsia:

Rayer originally reported proteinuria in preeclampsia in 1840. Schedoff and Porockjakoff subsequently connected proteinuria to eclampsia and hypertension.[76, 77] Initial theories suggested increased glomerular permeability to proteins. Electron microscopy later revealed distinctive glomerular changes in preeclampsia, such as endothelial vacuolization and cytoplasmic organelle hypertrophy.[78]

Renal Injury in Preeclampsia:

The glomeruli in preeclampsia are enlarged and bloodless, with endothelial and mesangial cell swelling causing capillary lumen occlusion. These lesions, referred to as glomerular capillary endotheliosis, were later found in gestational hypertension and normal pregnancies.[79] Recent studies suggest podocyte damage and slit diaphragm disruption play crucial roles in preeclampsia related proteinuria.[80] Podocyte function depends on VEGF and its antagonist sFlt-1.[81] The glomerular filtration barrier is disrupted in PE due to elevated sFlt-1 and soluble endoglin, which cause damage to podocytes and endothelial cells.[82] Animal studies show VEGF replacement can reverse glomerular lesions and decrease proteinuria.[83]

FIGURE 2: Renal injury in preeclampsia[83]



Definition of pathologic proteinuria during pregnancy:

By the third trimester, normal pregnancy raises urine protein excretion to 200–260 mg/day. The conventional definition of proteinuria during pregnancy is >300 mg/24 hours; there is no clinical outcome correlation for this threshold, therefore it may need to be reviewed.[84]

Urine Protein-to-Creatinine Ratio:

To approximate 24-hour protein loss, albumin content in spot urine tests is adjusted for urinary creatinine. UPCR of >0.3 indicates abnormal proteinuria during pregnancy.[85] Studies suggest spot UPCR is a reasonable "rule-out" test for proteinuria >300 mg/day in hypertensive pregnant women.[86] However, UPCR is less reliable during labor and can be elevated postpartum.[87]

Proteinuria and Preeclampsia:

Compared to gestational hypertension alone, proteinuria with hypertension predicts greater adverse outcomes for the mother and the newborn. Previously,

proteinuria was essential for diagnosing preeclampsia with disease severity.[88] Recent studies suggest severe hypertension poses the highest risk of adverse outcomes, severe preeclampsia associated with higher rates of preterm deliveries and low-birth weight infants.[89]

TABLE 3: Outcomes in gestational hypertension v/s preeclampsia:

	Gestational hypertension	Preeclampsia without severe feature	Severe gestational hypertension
Maternal outcomes			
Elevated liver enzymes	1.1	3.2	6.3
Placental abruption	0.3–1.3	0.5–3.2	3.1–4.2
Disseminated intravascular coagulation	0.1	0.5	3.1
Induction of labor	23.8	41.5	50
Cesarean delivery	29.1	30.9	28.1
Neonatal outcomes			
Preterm delivery at <37 wk gestation	17.8	25.8	54.2
Preterm delivery at <34 wk gestation	1	1.9	3.2
Small for gestational age	6.5–6.9	4.8–9.2	10.2–20.8
Birthweight of <2500 g	7.7	11.1	25.8
Intensive care unit admission	12.5–18.2	24.2–27.3	20.8–29
Respiratory distress syndrome	4.8–5.5	3.2–4.8	6.5–12.5
Perinatal death	0.1–1.7	0.5	0.1–3.1

MATERIALS AND METHODS

Source of data:

All pregnant women more than 20 weeks of gestation getting admitted in the department of Obstetrics and Gynecology at R.L.Jalappa Hospital and Research Centre, Tamaka.

Study design: Cross sectional study

Study period: 15months (SEPT 2022-DEC 2023)

Inclusion criteria:

- Primigravida
- Aged between 21 to 35 years.
- Gestational age more than 20 weeks.
- Two blood pressure readings of more than 140/90mmHg measured 4 hours apart.
- Proteinuria (1+ or above on dipstick).

Exclusion criteria:

Pregnant women with

- Multiple pregnancy
- Chronic hypertension
- Renal disease
- Gestational diabetes mellitus with associated hypertension

Sample size:

Sample size is estimated based on urine protein to creatinine ratio for predicting adverse maternal and fetal outcome in preeclampsia according to study conducted by Arzoo Chadha,Surekha Tayade published in Journal Cureus: A prospective observational study conducted in Jawaharlal Nehru Medical College, Datta Meghe Institute of Medical sciences, Wardha India. Considering alpha error of 1% with Power 90%, the estimated sample size is 130. The above given sample size is calculated using nMaster2.0 software.

- $N=\frac{\left(Z_{\frac{\alpha}{2}}\sqrt{2p(1-p)}+Z_{1-\beta}\sqrt{p_1(1-p_1)p_2(1-p_2)}\right)^2}{(p_1-p_2)^2}$

- Where p1 and p2 are the population of event of interest (outcome) for group I and group II, and p is p=$\frac{(p_1+p_2)}{2}$, $Z_{\frac{\alpha}{2}}$ is normal deviate at a level of significance and $Z_{1-\beta}$ is the normal deviate at 1-β% power with β% of type II error, normally type II error is considered 20% or less.

METHOD OF DATA COLLECTION:

After written informed consent, patients fulfilling the inclusion criteria were included in the study. Totally 130 patients with preeclampsia were included. A detailed clinical history along with antenatal examination was done. Patients with Two BP readings more than or equal to 140/90 mm Hg 4 hours apart and Urinary protein on dipstick (1+ and above) were documented and included. Spot urine sample was collected and estimated for Urinary protein creatinine ratio in all the patients and maternal and fetal outcome were noted under the following parameters:

- Maternal outcome : maternal mortality, ICU admission, HELLP syndrome, abruption, eclampsia; and
- Fetal outcome: Intrauterine death, fetal growth restriction, prematurity, NICU admission, neonatal death <1week of life.

The study population was followed throughout the pregnancy to monitor the maternal and fetal outcome. The outcome of this study was to prove that Urine protein creatinine ratio (UPCR) which is a simple, affordable, accurate and precise tool with more sensitivity and specificity to predict maternal and fetal outcomes in preeclampsia.

STATISTICAL ANALYSIS

The data was collected and compiled in MS Excel. Descriptive statistics has been used to present the data. To analyse the data SPSS (Version 26.0) was used. Significance level was fixed as 5% ($\alpha = 0.05$). Qualitative variables are expressed as frequency and percentages and Quantitative variables are expressed as Mean and Standard Deviation. To compare the proportion between variables, chi-square test was used. To compare the mean values between variables, student t test and ANOVA was used.

Graphical representation of data: MS Excel and MS word was used to obtain various types of graphs such as bar diagram, pie chart.

***p*-value** (Probability that the result is true) of < 0.05 was considered as statistically significant after assuming all the rules of statistical tests.

Statistical software: MS Excel, SPSS version 26.0 was used to analyze data.

RESULTS

TABLE 4: AGE CASE FREQUENCY DISTRIBUTION (n=130)

AGE GROUP	FREQUENCY(N)	PERCENT-AGE (%)
18- 20 years	33	25.4
21- 25 years	43	33.1
26- 30 years	43	33.1
31- 35 years	11	8.5
MEAN±SD	24.47±4.32	

33.1% of study participants was found to be **21-25** years. 33.1% of study participants was found to be **26-30** years. Mean-age of study participants was 24.47±4.32 years.

FIGURE 3: GRAPHICAL REPRESENTATION: PIE CHART-AGE CASE FREQUENCY DISTRIBUTION

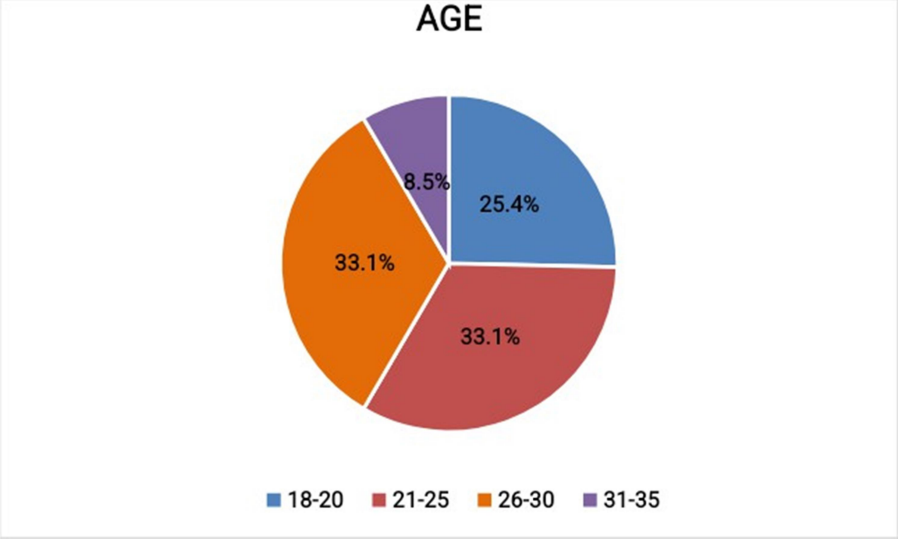


TABLE 5: PERIOD OF GESTATION WEEK WISE DISTRIBUTION (n=130)

PERIOD OF GESTATION	FREQUENCY	PERCENTAGE (%)
<28 weeks	24	18.5
28-32 weeks	10	7.7
32-37 weeks	41	31.5
37-40 weeks	41	31.5
More than 40 weeks	14	10.8

31.5% of the study participants was found to have POG of **32-37 weeks**. 31.5% of the study participants were found to have POG of **37-40 weeks.**

FIGURE 4: GRAPHICAL REPRESENTATION: PIE CHART PERIOD OF GESTATION IN WEEKS.

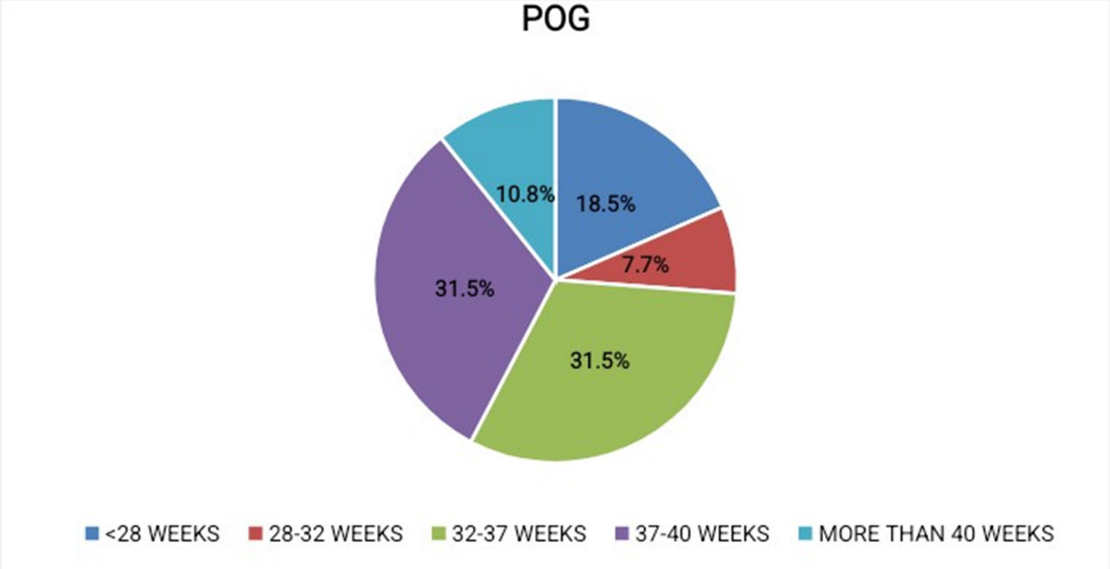


TABLE 6: PALLOR AND EDEMA CASE FREQUENCY DISTRIBUTION

General examination	FREQUENCY	PERCENTAGE (%)
Pallor	18	13.8
Pedal edema	30	23.1

Pallor was present in 13.8% of the study participants and pedal edema was found in 23.1% of the study participants.

TABLE 7: URINE PROTEIN BY DIPSTICK – FREQUENCY DISTRIBUTION

Urine routine	FREQUENCY	PERCENTAGE (%)
1+	8	6.2
2+	72	55.38
3+	50	38.46

On urine routine examination 38.46% had spot urinary protein 3+ and 55.38% had 2+ and 6.2% had 1+.

FIGURE 5: GRAPHICAL REPRESENTATION: PIE CHART URINE PROTEIN BY DIPSTICK

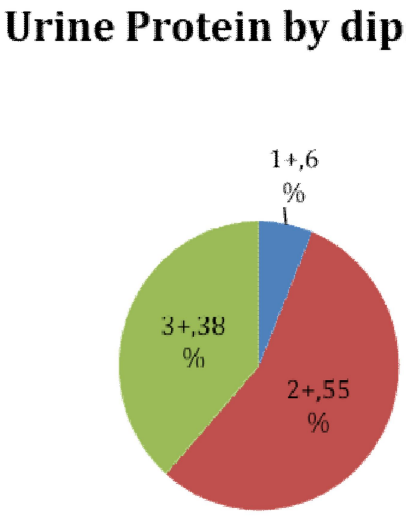


TABLE 8: MEAN±SD OF VITAL PARAMETERS

VITAL PARAMETERS	MEAN±SD
PULSE (bpm)	86.32±6.358
Systolic BP (mmHg)	151.62±14.295
Diastolic BP (mmHg)	97.46±9.907

Mean pulse rate of participants was 86.32± 6.3 bpm. Mean SBP was 151.6±14.2 mmHg and mean DBP was 97.46 ±9.9 mmHg.

FIGURE 6: GRAPHICAL REPRESENTATION: BAR DIAGRAM-VITAL PARAMETERS

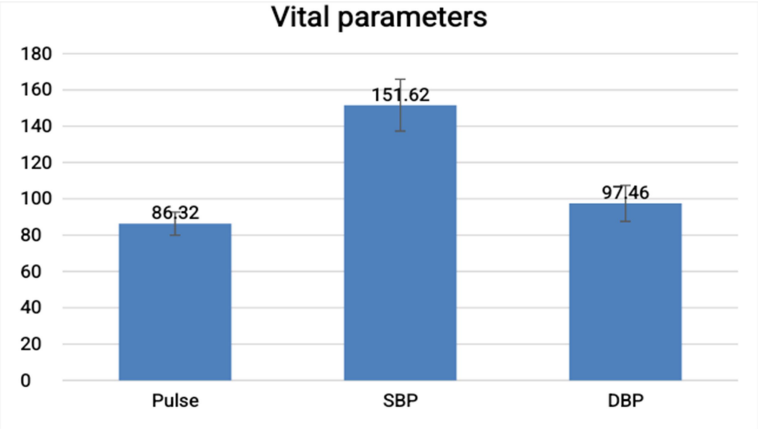
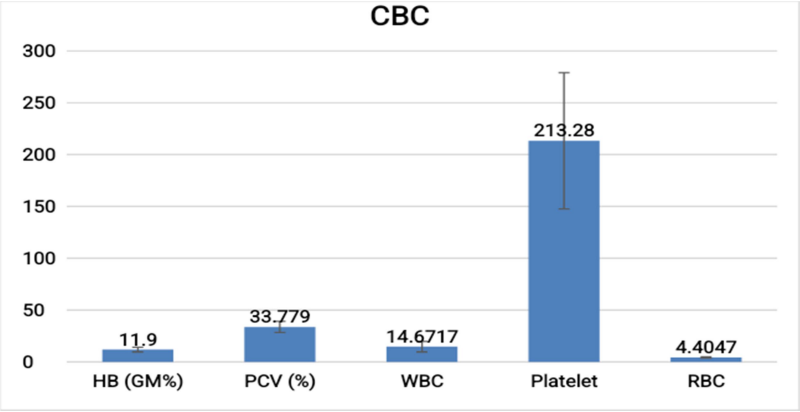


TABLE 9: MEAN±SD OF COMPLETE BLOOD COUNT

CBC	MEAN±SD	Methodology	Biological reference interval	Instrumentation
HB (GM%)	11.9±2.19	Cell counter SLS method	11.5-15.5	SYSMEX XN 1000
PCV (%)	33.779±5.35	Cell counter SLS method	35-45	SYSMEX XN 1000
WBC (th/mm^3)	14.6717±5.016	Cell counter automated flow cytometry	4-11	SYSMEX XN 1000
Platelet (th/mm^3)	213.28±65.751	Electrical impedance	150-450	SYSMEX XN 1000
RBC (mil/mm^3)	4.4047±0.587	Cell counter SLS method	4.5-5.5	SYSMEX XN 1000

FIGURE 7: GRAPHICAL REPRESENTATION: BAR DIAGRAM- PARAMETERS OF COMPLETE BLOOD COUNT



Lab parameter of participants were as follows, Mean Hb was 11.9±2.19gm%, PCV was 33.7±5.3%, WBC was 14.6±5.01 th/mm^3, Platelet count was 213.28±65.7 th/mm^3 and RBC count was 4.404±0.5 mil/mm^3.

TABLE 10: MEAN±SD OF PARAMETERS OF LIVER FUNCTION TEST

Liver function test	MEAN±SD	Methodology	Minimum value-maximum value	Biological reference interval	Instrumentation
PT (sec)	19.84±27.14	Photo-optic clot detection	-	10-14 sec	ACL ELITE PRO
APTT (sec)	24.94±7.16	Photo-optic clot detection	-	27-33 sec	ACL ELITE PRO
INR	1.51±0.324	Calculated	-	1.0-1.5	
TB (mg/dl)	0.67±0.54	Azobilirubin/ dyphyline	0.10-27.0	0.2-1.3	VITROS 5.1 FS
DB (mg/dl)	0.36±0.53	Calculated	0.0-0.4	0.0-0.4	VITROS 5.1 FS
SGOT(U/L)	33.22±25.13	Multipoint rate with p-5-p	3-750	15-46	VITROS 5.1 FS
SGPT(U/L)	21.28±10.04	Multipoint rate/UV with p-5-p	6-1000	13-69	VITROS 5.1 FS
ALP(U/L)	201.79±73.53	Multipoint rate/P-4-Nitrophenyl phosphate	20-1500	38-126	VITROS 5.1 FS
TP(g/dL)	5.980±0.73	Biuret	2.0 –11.0	6.3-8.2	VITROS 5.1 FS
Albumin(g/dL)	2.983±0.46	Bromocresol green	1.0-6.0	3.5-5.0	VITROS 5.1 FS
A: G	0.88±0.34	Calculated		Upto 1.2	
GGT(U/L)	22.21±20.68	L-gamma-glutamyl-4-nitroanilide	10-1400	12-58	VITROS 5.1 FS

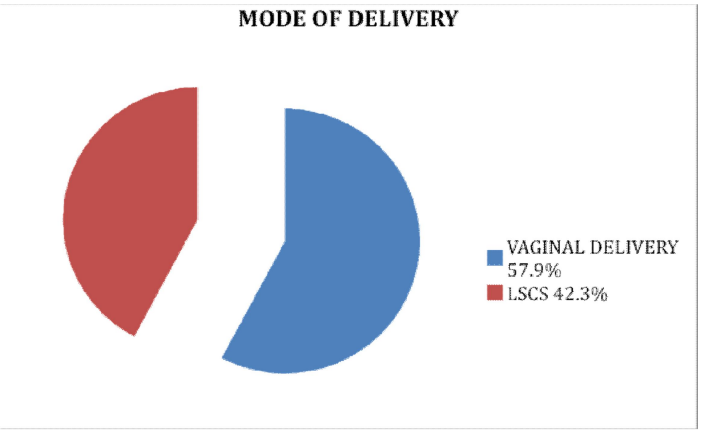
TABLE 11: MEAN±SD OF PARAMETERS OF RENAL FUNCTION TEST

Renal function test	MEAN± SD	Methodology	Minimum value-maximum value	Biological reference interval	Instrumentation
Sodium (mmol/L)	133.10±3.93	Potentiometric	75.0-250.0	137-145	VITROS 5.1 FS
Potassium (mmol/L)	3.785±0.59	Potentiometric	1.0-14.0	3.5-5.1	VITROS 5.1 FS
Blood urea (mg/dl)	16.32±6.22	Urease	2.0-120.0	7.0-17.0	VITROS 5.1 FS
Serum creatinine (mg/dl))	0.64±0.25	Enzymatic-Sarcosine oxidase/ peroxidase	0.05-14.0	0.7-1.2	VITROS 5.1 FS
Serum Uric acid (mg/dl)	5.41±1.59	Enzymatic-Uricase/peroxidase	0.5-17.0	2.5-6.2	VITROS 5.1 FS
Urine protein (mg/dl)	49.39±39.41	Reflectance spectrophotometric method	5.0-200	<12mg/dl	VITROS 5.1 FS
Urine creatinine (mg/dl)	67.21±63.65	Sarcosine oxidase peroxidase	1.05-346.5	20 – 320 mg/dl	VITROS 5.1 FS
UPCR	2.85±2.06	Calculated			

TABLE 12: FREQUENCY DISTRIBUTION OF VAGINAL DELIVERY VERSUS LSCS (n=130)

DELIVERY	FREQUENCY	PERCENTAGE (%)
VAGINAL DELIVERY	75	57.9
LSCS	55	42.3

FIGURE 8: GRAPHICAL REPRESENTATION: PIE CHART VAGINAL DELIVERY VERSUS LSCS



Among 130 participants with PE, 57.9% of them delivered vaginally, while 42.3% underwent LSCS.

TABLE 13: FREQUENCY DISTRIBUTION OF MATERNAL OUTCOME (n=130)

MATERNAL OUTCOME	FREQUENCY	PERCENTAGE (%)
ABORTION	9	6.9
STILL BIRTH	4	3.1
ECLAMPSIA	22	16.9
HELLP SYNDROME	9	6.9
ABRUPTION	5	3.8
ICU ADMISSION	1	0.8

Among 130 cases with PE, eclampsia was an adverse outcome in 16.9% cases, while ICU admission was in 0.8% cases only.

FIGURE 9: GRAPHICAL REPRESENTATION: BAR DIAGRAM PARAMETERS OF MATERNAL OUTCOME

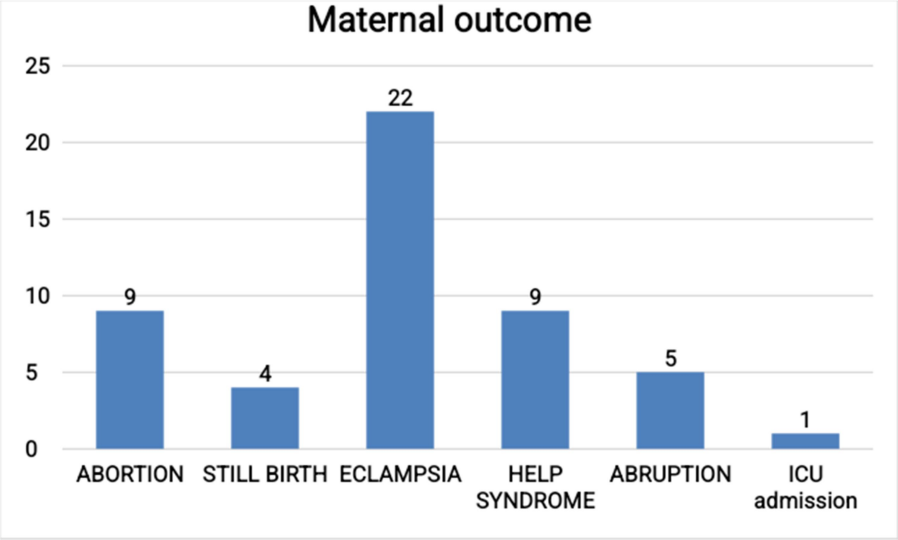


TABLE 14: FREQUENCY DISTRIBUTION OF FETAL OUTCOME (n=130)

FETAL OUTCOME	FREQUENCY	PERCENTAGE (%)
NICU ADMISSION	69	53.1
PREMATURITY	39	30.0
FETAL GROWTH RESTRICTION	24	18.5
INTRAUTERINE FETAL DEMISE	15	11.5
NEONATAL DEATH <1WK	4	3.1

Out of 130 cases fetal outcome was analysed, 53.1% cases required NICU admission and neonatal death was seen among 3.1% of them.

FIGURE 10: GRAPHICAL REPRESENTATION: BAR DIAGRAM- PARAMETERS OF FETAL OUTCOME

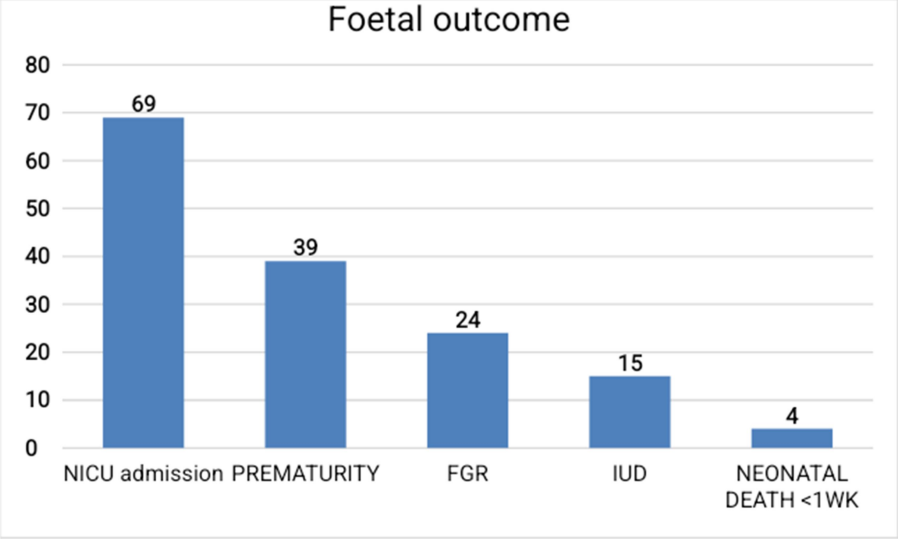


TABLE 15: FREQUENCY DISTRIBUTION OF URINE PROTEIN CREATININE RATIO (UPCR) (n=130)

UPCR	FREQUENCY	PERCENT
<0.3	16	12.3
≥0.3	114	87.7

87.7% of participants had UPCR score ≥0.3 and 12.3% had <0.3 respectively, out of 130 cases of PE.

FIGURE 11: GRAPHICAL REPRESENTATION: PIE CHART UPCR <0.3 AND ≥ 0.3

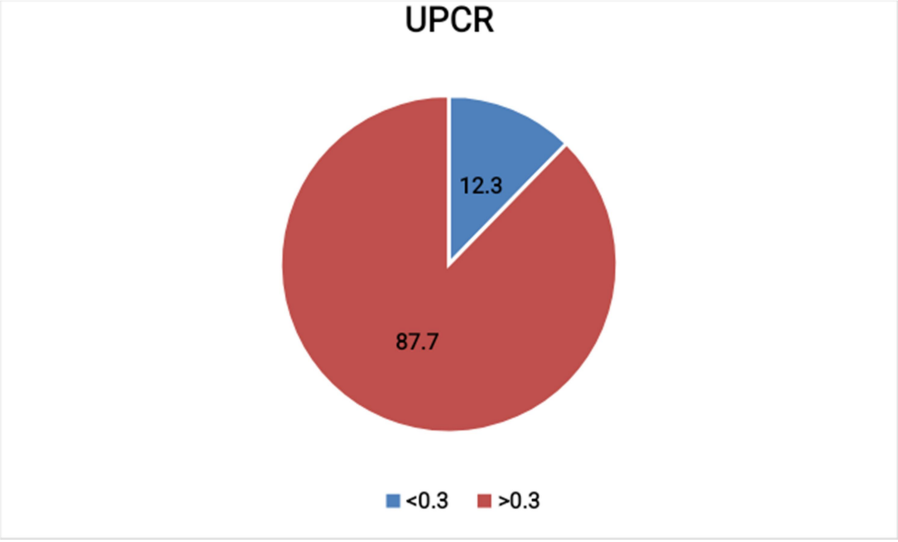
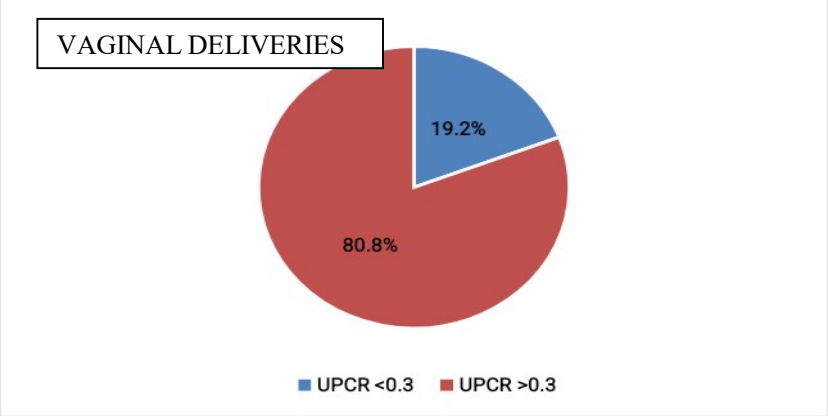


TABLE 16: MATERNAL OUTCOME WITH URINE PROTEIN-TO-CREATININE RATIO

MATERNAL-OUTCOME	UPCR <0.3		UPCR ≥0.3		*p*-value
	n	%	n	%	
VAGINAL DELIVERY	12	16%	63	84%	0.230
LSCS	4	7.3%	51	92.7%	0.135
ECLAMPSIA	5	22.7%	17	77.3%	0.103
HELLP SYNDROME	-	-	9	100.0%	0.244
ABORTION	1	11.1%	8	88.9%	0.910
ABRUPTION	1	20.0%	4	80.0%	0.593
STILL BIRTH	-	-	4	100.0%	0.447
ICU ADMISSION	-	-	1	100.0%	0.707

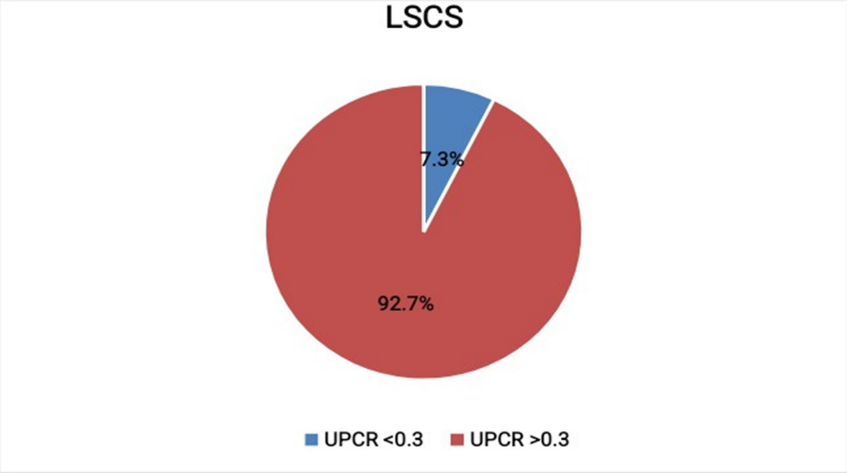
Among study population who delivered vaginally and underwent LSCS and among those who had complications such as eclampsia, HELLP syndrome, abortion, abruption, still birth and ICU admission, difference observed in maternal outcome was not statistically significant.

FIGURE 12: GRAPHICAL REPRESENTATION: PIE CHART OF VAGINAL DELIVERIES IN UPCR<0.3 AND ≥ 0.3



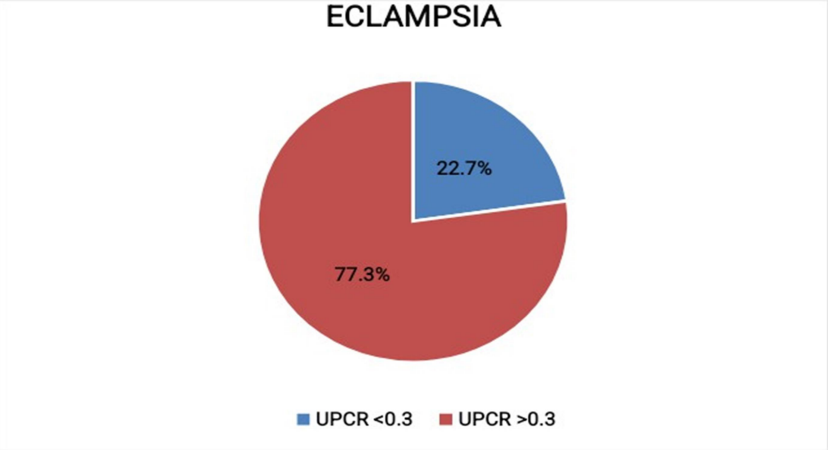
Among 75 cases who delivered vaginally, UPCR was ≥0.3 in 80.8% cases and <0.3 in 19.2% cases.

FIGURE 13: GRAPHICAL REPRESENTATION: PIE CHART OF LSCS IN UPCR <0.3 AND ≥ 0.3



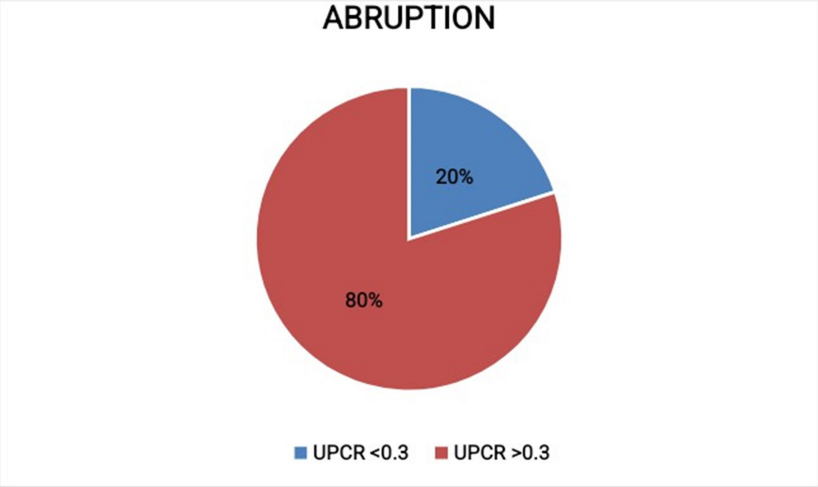
Among 55 cases who underwent LSCS, UPCR was ≥0.3 in 92.7% cases and <0.3 in 7.3% cases.

FIGURE 14: GRAPHICAL REPRESENTATION: PIE CHART OF ECLAMPSIA CASES IN UPCR <0.3 AND ≥ 0.3



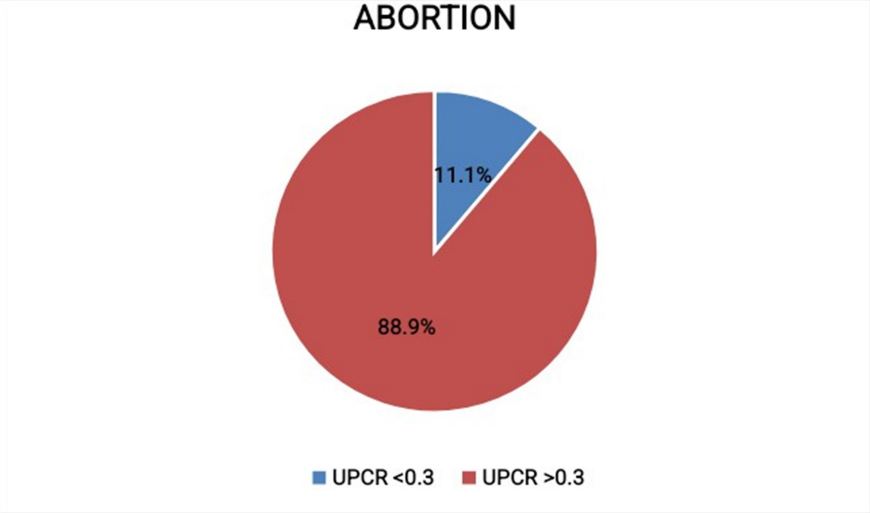
Among cases of preeclampsia leading to eclampsia, 77.3% cases had UPCR ≥0.3.

FIGURE 15: GRAPHICAL REPRESENTATION: PIE CHART OF ABRUPTION IN UPCR<0.3 AND ≥ 0.3



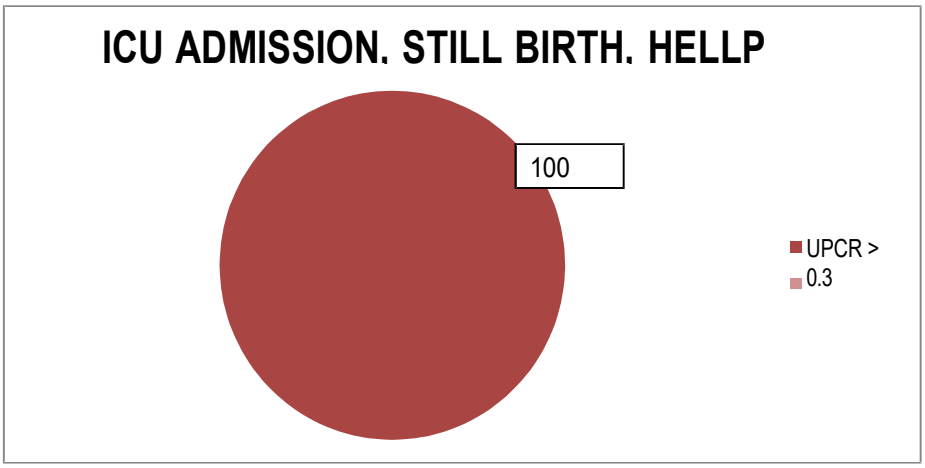
Among cases of preeclampsia leading to placental abruption, 80% cases had UPCR ≥ 0.3.

FIGURE 16: GRAPHICAL REPRESENTATION: PIE CHART OF ABORTION IN UPCR<0.3 AND ≥ 0.3



Among cases of preeclampsia leading to abortion, 88.9% cases had UPCR ≥ 0.3

FIGURE 17: GRAPHICAL REPRESENTATION: PIE CHART OF ICU ADMISSION, STILL BIRTH, HELLP SYNDROME IN UPCR<0.3 AND ≥ 0.3



All cases of preeclampsia with complications like ICU admissions, still birth and HELLP syndrome had UPCR ≥0.3.

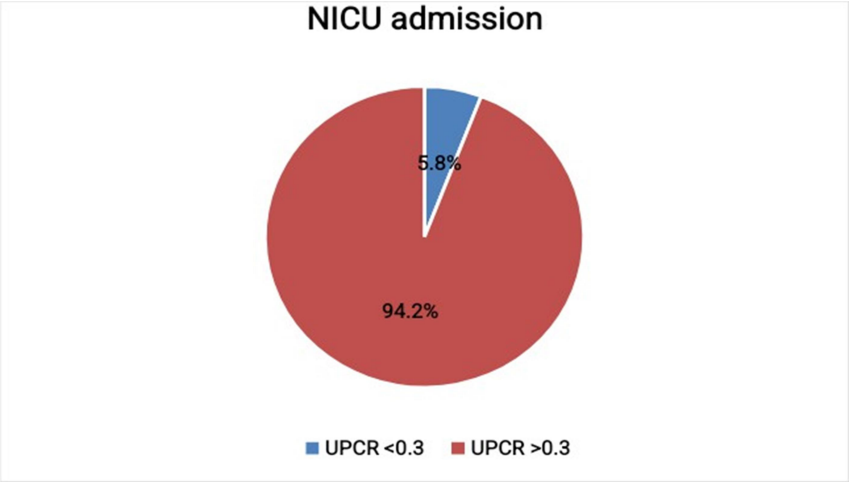
TABLE 17: FETAL OUTCOME WITH URINE PROTEIN-TO-CREATININE RATIO

FETAL-OUTCOME	UPCR <0.3		UPCR ≥0.3		*p*-value
	n	%	N	%	
STILL BIRTH	-	-	4	100.0%	0.447
NICU ADMISSION	4	5.8%	65	94.2%	**0.016***
PREMATURITY	3	7.7%	36	92.3%	0.294
FETAL GROWTH RESTRICTION	-	-	24	100.0%	**0.042***
INTRAUTERINE FETAL DEMISE	2	13.3%	13	86.7%	0.898
NEONATAL DEATH <1WK	-	-	4	100.0%	0.447

p- value <0.05, statistically significant

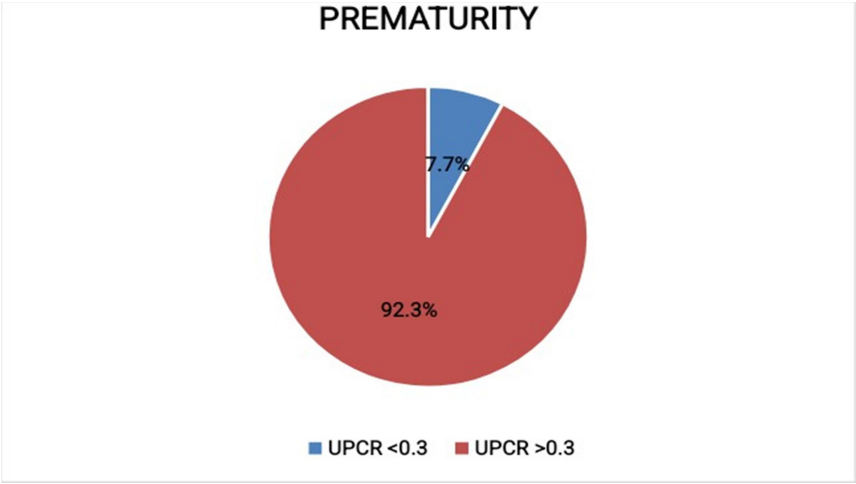
In present study, all neonatal deaths, 86.7% of intrauterine fetal demise cases,94.2% cases with NICU admission and all cases with fetal growth restriction were observed among those had UPCR score ≥0.3.The difference observed in NICU admissions and fetal growth restriction has been found to be statistically significant.

FIGURE 18: GRAPHICAL REPRESENTATION: PIE CHART OF NICU ADMISSIONS IN UPCR <0.3 AND ≥ 0.3



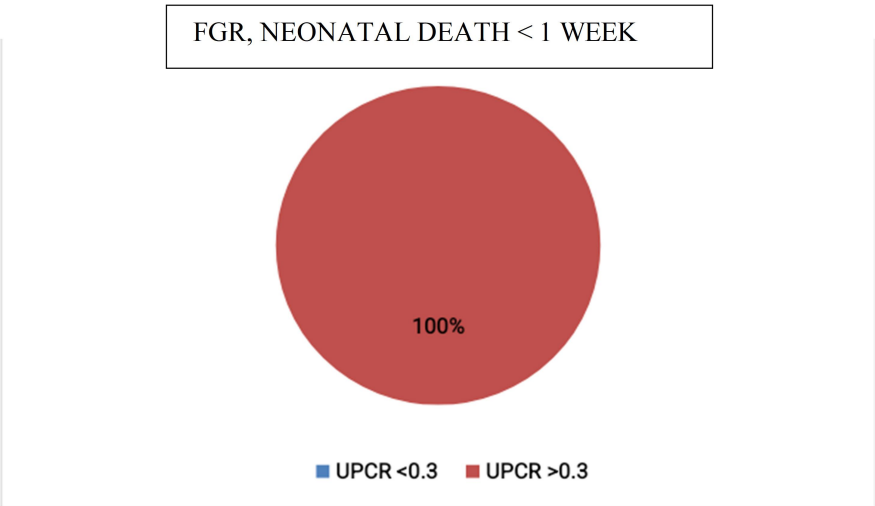
Among 69 cases with NICU admission, 94.2% cases had UPCR score ≥0.3.

FIGURE 19: GRAPHICAL REPRESENTATION: PIE CHART OF CASES WITH PREMATURITY IN UPCR<0.3 AND ≥ 0.3



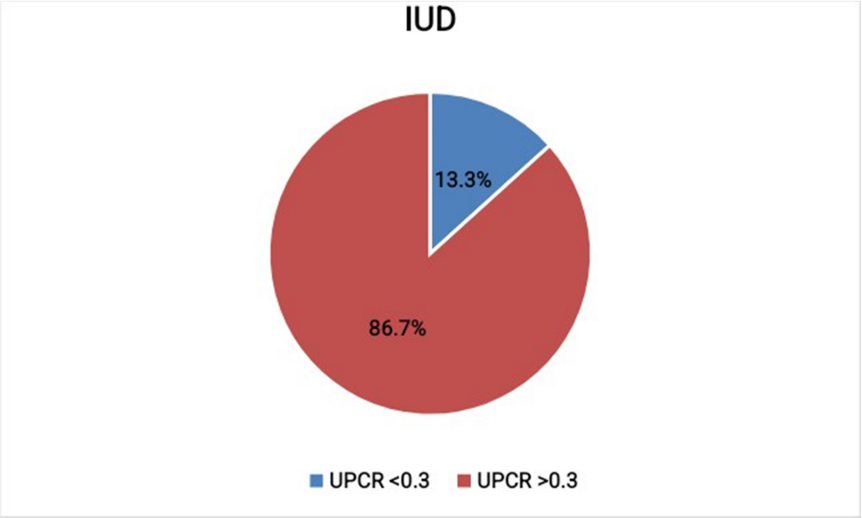
Among 39 cases with prematurity, 92.3% cases had UPCR score ≥ 0.3.

FIGURE 20: GRAPHICAL REPRESENTATION: PIE CHART OF CASES WITH FETAL GROWTH RESTRICTION AND NEONATAL DEATH <1 WEEK IN UPCR<0.3 AND ≥ 0.3



All cases with fetal growth restriction and neonatal death within 1 week had UPCR ≥ 0.3.

FIGURE 21: GRAPHICAL REPRESENTATION: PIE CHART OF CASES WITH INTRAUTERINE FETAL DEMISE IN UPCR<0.3 AND ≥ 0.3



Among 15 cases with intrauterine fetal demise, 86.7% cases had UPCR ≥ 0.3.

DISCUSSION

The pathogenesis of preeclampsia, a hypertensive multisystem illness, is unknown. PE is connected with increased activation of maternal circulating leukocytes, which may be the cause of the vascular dysfunction that results from PE. The study aimed to measure UPCR in women with preeclampsia and to analyse variations in maternal and fetal outcomes in UPCR <0.3 and ≥0.3.

Age:

The present study primarily observed preeclampsia in 21-30 years. The outcome is in line with earlier research by Safirani et al. and Sumampouw et al.[86, 90] This could be as certain age is considered to be the most fit for conception, pregnancy, and delivery.[90]

Similar to the present findings, Jaleel et al. and Kumar et al. discovered that majority of cases occurred in the 21–30 age range.[91, 92] Preeclampsia is more common at younger ages, which is indicative of earlier marriage and childbearing rates in our nation as compared to Western nations.

Period of gestation (POG):

31.5% of the study participants were found to have POG of 32-37 weeks. 31.5% of the study participants were found to have POG of 37-40 weeks. Study done by Khan B et al., shows the period of gestation for majority was between 35-37 weeks.[90] Study by Bindhu et al., shows period of gestation was between 35-37 weeks which was consistent with our study.[94]

Spot urine protein:

In our study, urine routine examination 38.46% had spot urinary protein 3+ and 34.6% had 2+.

According to study by Khan B et al., in 24-hour urine test, 57.8% of patients had protein levels >0.3–3 g/L.[95]

Among our participants, mean UPCR was 2.85±2.06. According to Chauhan et al., the platelet count was 157.18±56.66 x 109 th/mm3 in cases and 222.93±97.94 x 109 th/mm^3 in controls.[96] Platelet counts were 242±62 x109 th/mm^3 in the control group and 160±51 x109 th/mm^3 in the preeclamptic group, according to Meshram et al.[97] These authors discovered considerably lower platelet counts in preeclampsia compared to controls, which is inconsistent with the results of our study. This finding was probably due to early diagnosis before thrombocytopenia set in.

Modalities of delivery:

In our study, 57.9% delivered vaginally while 42.3% underwent LSCS. Study by Bindhu et al. shows that vaginal deliveries accounted for 36.7% of deliveries, cesarean sections accounted for roughly 63.4% of deliveries resulting from preeclampsia-induced pregnancy problems. More than two-thirds of pregnancies ended in cesarean sections, according to additional studies.[98, 99] According to study by Jackson, Lee and colleagues, 41.9% of women experienced spontaneous labor, 48.3% of whom had an induced caesarean section and the remaining women gave birth vaginally. Preeclampsia has no known treatment except for fetal delivery. The general management is to induce labor once the fetus reaches maturity. There are no additional benefits noted for elective cesarean delivery.[99] The stringent antenatal check-ups, dedicated patient care, appropriate interventions and follow up might have resulted in higher chances of vaginal delivery versus LSCS in our study.

Maternal outcome:

According to numerous studies, there is zero to variable rates of maternal mortality associated with severe complications in the mother. Three maternal deaths were recorded in a study on the consequences of eclampsia by Wassie, Anmut, et al.[100] In Enugu, Nigeria, a study by Johnson, Brown et al., found that there were no maternal deaths, very few cases of HELLP syndrome and placental abruption, and a small number of studies indicated that preeclampsia was the cause of 8% and 10% of maternal deaths.[101] We did not find any maternal deaths in our analysis, which is different from these two studies but similar to the Nigerian study. This could be because our cohort received prompt diagnosis and treatment.

Fetal outcome:

In our investigation, fetal growth restriction was determined to be 18.5%. Fetal growth restriction was reported to be 9.67% in Aabidha et al. study and 4.4% were low birth weight. These results are consistent with another study that found 4.45% of low birth weight babies.[102] In present study 53.1% required NICU admission and neonatal death was seen among 3.1% of participants.

Similar to our findings, Green, Patel et al. reported 9.4% of fetal fatalities. Linked to respiratory distress, low-birth weight and other related disorders are 11.1% of neonatal deaths.[103] In line with a study conducted in Alexandria by Davis, Thompson, et al., which discovered that 4.4% of babies had low birth weights, the current study's results, which indicate that 4.45% of the neonates had low birth weights, are consistent. [104] There is evidence that preeclampsia risk factors are connected to maternal and fetal outcomes. Premature birth and the method of delivery are also impacted by elevated systolic and diastolic readings.

UPCR and Study Outcomes:

In our study, high UPCR is more common in those who delivered vaginally due to diligent monitoring and timely intervention and administration of anti-hypertensives, but the proportion does not show significant variation from the expected distribution. High UPCR is prevalent among those undergoing LSCS, suggesting complications as a result of severe preeclampsia may lead to a higher rate of caesarean sections. High UPCR is associated with eclampsia cases, indicating a potential link between severe proteinuria and the occurrence of eclampsia. Every case of HELLP syndrome and ICU admission was associated with a high UPCR, highlighting a strong correlation between severe proteinuria and HELLP syndrome. A high UPCR is seen in the majority of abruption cases, suggesting an association between high proteinuria levels and placental abruption. Stillbirths and abortions were exclusively associated with high UPCR, underscoring the critical risk severe proteinuria poses to pregnancy outcomes.

Similar to maternal outcomes, high UPCR is strongly correlated with stillbirths. Significantly higher proportion of infants admitted to the NICU had high UPCR, indicating severe preeclampsia and associated complications needing intensive neonatal care. High UPCR is noted in cases of prematurity, suggesting that severe proteinuria is a strong predictor of preterm delivery. Every case of FGR was associated with high UPCR, indicating a strong link between severe proteinuria and restricted fetal growth. A high proportion of intrauterine deaths were associated with high UPCR, emphasizing the critical impact of proteinuria on fetal viability. All neonatal deaths within the first week were linked with high UPCR, highlighting the severe fetal outcomes of preeclampsia.

SUMMARY

Preeclampsia (PE) is a multi-systemic hypertensive disorder of pregnancy, marked by hypertension and proteinuria or end-organ dysfunction > 20 weeks of gestation. PE is a significant cause of maternal and fetal morbidity and mortality, particularly in developing countries. This study aims to estimate the urine protein-to-creatinine ratio in preeclamptic patients and examine its association with obstetric outcomes.

A total of 130 primigravida aged between 21 to 35 years with gestational age more than 20 weeks whose two blood pressure readings were more than 140/90mmHg measured 4 hours apart with proteinuria (1+ or above on dipstick) were included in our study. Spot urine sample was collected and estimated for Urinary protein-to-creatinine ratio in all the patients and maternal outcome (maternal mortality, ICU admission, HELLP syndrome, abruption, eclampsia) and fetal outcome (intrauterine fetal death, fetal growth restriction, prematurity, NICU admission, neonatal death <1week of life) were noted among UPCR <0.3 and ≥0.3.

The present study primarily observed preeclampsia in 21-30 years of age and mostly presented in late second and third trimester of pregnancy. On urine routine examination 38.46% had spot urinary protein 3+ and 55.38% had 2+. UPCR score ≥0.3 was noted in 87.7% of participants and 12.3% had <0.3 respectively, out of 130 cases of PE. 57.9% of them delivered vaginally, while 42.3% underwent LSCS**.** Eclampsia was an adverse outcome in 16.9% cases, while ICU admission was in 0.8% cases only.

Among study population who delivered vaginally and underwent LSCS and among those who had complications such as eclampsia, HELLP syndrome, abortion, abruption, still birth and ICU admission, difference observed in maternal outcome was not statistically significant. But among UPCR ≥ 0.3, preeclampsia leading to eclampsia (77.3%), placental abruption (80%) and abortion (88.9%) are noted. All cases of preeclampsia with ICU admissions, still birth and HELLP syndrome had UPCR ≥ 0.3.

Out of 130 cases among which fetal outcome was analysed, 53.1% cases required NICU admission and neonatal death within 1 week was seen among 3.1% of them. In the present study, all neonatal deaths, all cases with fetal growth restriction, 94.2% cases with NICU admission, 92.3% cases with prematurity, 86.7% of intrauterine fetal demise were observed among those had UPCR ≥0.3.Observations in NICU admissions and fetal growth restriction have been found to be statistically significant with *p*-value <0.05.

The random urine protein-to-creatinine ratio test provides a faster and more feasible alternative to the traditional 24-hour urine protein test, offering both diagnostic and prognostic value. Early detection and monitoring of proteinuria through this method can aid in timely interventions and improved clinical decision-making, ultimately enhancing maternal and fetal outcomes. Stringent antenatal care, regular follow up in cases with preeclampsia, apt anti-hypertensive dosing and fetal monitoring play an additional but pivotal role in the early detection and management of preeclampsia and its complications.

CONCLUSION

A high Urine protein-to-creatinine ratio is significantly associated with adverse obstetric outcome in pregnancies complicated by preeclampsia as per our study. There is a rise in rate of LSCS, eclampsia, abruptio placenta, HELLP syndrome, NICU admission, prematurity, fetal growth restriction and neonatal mortality in cases with high UPCR. At risk patients with preeclampsia must undergo routine monitoring of UPCR as timely interventions can be taken to avoid adverse maternal and fetal complications. Thus, UPCR is a reliable, inexpensive and good prognostic tool which can be incorporated in our routine clinical practise.

There is need for future research to diversify in population and develop proper management techniques for high UPCR in preeclampsia to improve clinical outcomes for healthy mother and baby.

Strengths of our study:

- Majority of cases delivered vaginally.
- Stringent measures in patient care, administration of anti-hypertensive agents and follow up of preeclampsia.
- Minimal admissions of mother to Intensive Care Unit.

Limitations:

- Socioeconomic attributes and ignorance play a crucial role in complications at initial presentation.
- Study is conducted in a rural population where primary access to quality health care services might be compromised, and referred to a higher centre at a later stage.
- Illiteracy and poverty could be a barrier in taking timely treatment.
- Agriculture being the major occupation, patients might have been unable to follow up for antenatal care.

Recommendations:

- Regular antenatal visits for early detection and follow up of the clinical condition will reduce the risk of preeclampsia and its complications.
- Medications: Administration of appropriate dose of anti-hypertensives and strict monitoring.
- Lifestyle modification: Healthy diet and prevention of malnutrition add to the better feto-maternal outcomes.
- Prevention and appropriate management of risk factors or associated conditions like diabetes and obesity should be treated.
- Developing targeted interventions to manage high UPCR in pregnant women with preeclampsia could help mitigate the associated risks and improve both maternal and fetal outcomes.

New knowledge:

- Early identification of the causal factors of PE results in better outcome.
- Continuous follow up and monitoring of the vital parameters and implementation of treatment results in uneventful deliveries.
- Proper care of preeclampsia with regular monitoring results in vaginal deliveries versus LSCS in PE.
- Vaginal deliveries can be achieved by adequate care and strict monitoring of blood pressure readings and vital parameters.

REFERENCES

1. Mustafa R, Ahmed S, Gupta A, Venuto RC. A comprehensive review of hypertension in pregnancy. J Pregnancy. 2012;2012:105918. doi:10.1155/2012/105918.

2. Cunningham FG. Hypertensive disorders in pregnancy. Williams Obstetrics. McGraw-Hill, New York, NY; 2005:2:5-10.

3. Garovic VD, Dechend R, Easterling T, et al. Hypertension in pregnancy: diagnosis, blood pressure goals, and pharmacotherapy: a scientific statement from the American Heart Association. Hypertension. 2022;79.doi:10.1161/HYP.0000000000000208.

4. Abalos E, Cuesta C, Grosso AL, Chou D, Say L. Global and regional estimates of preeclampsia and eclampsia: a systematic review. Eur J Obstet Gynecol Reprod Biol. 2013 Sep 1;170(1):1-7. Available from: https://www.ejog.org/article/S0301-2115(13)00196-6/fulltext

5. Khan KS, Wojdyla D, Say L, Gülmezoglu AM, Look PFV. WHO analysis of causes of maternal death: a systematic review. Lancet. 2006 Apr 1;367(9516):1066-74. Available from: https://www.thelancet.com/journals/lancet/article/PIIS0140-6736(06)68397-9/fulltext

6. Steegers EA, von Dadelszen P, Duvekot JJ, Pijnenborg R. Pre-eclampsia. Lancet. 2010 Aug;376(9741):631-44. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0140673610602796

7. Kirbas A. Prediction of preeclampsia by first trimester combined test and simple complete blood count parameters. J Clin Diagn Res. 2015. Available from: http://jcdr.net/article_fulltext.asp?issn=0973 709x&year=2015&volume=9&issue=11&page=QC20&issn=0973-709x&id=6833

8. Yücel B, Ustun B. Neutrophil to lymphocyte ratio, platelet to lymphocyte ratio, mean platelet volume, red cell distribution width and plateletcrit in preeclampsia. Pregnancy

Hypertens. 2017 Jan;7:29-32. Available from: https://linkinghub.elsevier.com/retrieve/pii/S2210778916303002

9. Gestational hypertension and preeclampsia: acog practice bulletin, number 222. Obstet Gynecol. 2020 Jun;135(6) Available from: https://journals.lww.com/greenjournal/Abstract/2020/06000/Gestational_Hypertension_and_Preeclampsia ACOG.46.aspx

10. Tomimatsu T, Mimura K, Matsuzaki S, Endo M, Kumasawa K, Kimura T. Preeclampsia: maternal systemic vascular disorder caused by generalized endothelial dysfunction due to placental antiangiogenic factors. Int J Mol Sci. 2019 Jan;20(17):4246. Available from: https://www.mdpi.com/1422-0067/20/17/4246

11. Oğlak SC, Tunç Ş, Obut M, Şeker E, Behram M, Tahaoğlu AE. Maternal near-miss patients and maternal mortality cases in a Turkish tertiary referral hospital. Ginekol Pol. 2021;92(4):300-5. Available from: https://journals.viamedica.pl/ginekologia_polska/article/view/GP.a2020.0187

12. Magro-Malosso ER, Saccone G, Di Tommaso M, Roman A, Berghella V. Exercise during pregnancy and risk of gestational hypertensive disorders: a systematic review and meta-analysis. Acta Obstet Gynecol Scand. 2017 Aug;96(8):921-31. Available from: https://onlinelibrary.wiley.com/doi/10.1111/aogs.13151

13. Oğlak SC, Bademkıran MH, Obut M. Predictor variables in the success of slow-release dinoprostone used for cervical ripening in intrauterine growth restriction pregnancies. J Gynecol Obstet Hum Reprod. 2020 Jun 1;49(6):101739. Available from: https://www.sciencedirect.com/science/article/pii/S2468784720300738

14. GathiramP, Moodley J. Pre-eclampsia: its pathogenesis and pathophysiology. Cardiovasc J Afr. 2016 May 18;27(2):71-8. Available from: http://cvja.co.za/onlinejournal/vol27/vol27_issue2/#17/z

15. Burton GJ, Redman CW, Roberts JM, Moffett A. Pre-eclampsia: pathophysiology and clinical implications. BMJ. 2019 Jul 15;366Available from: https://www.bmj.com/content/366/bmj.l2381

16. Obut M, Oğlak SC. Expression of CD44 and IL-10 in normotensive and preeclamptic placental tissue. Ginekol Pol. 2020;91(6):334-41. Available from: https://journals.viamedica.pl/ginekologia_polska/article/view/GP.2020.0058

17. Pihl K, Sørensen S, Stener Jørgensen F. Prediction of preeclampsia in nulliparous women according to first trimester maternal factors and serum markers. Fetal Diagn Ther. 2020;47(4):277-83. Available from: https://www.karger.com/Article/FullText/503229

18. Chaiworapongsa T, Chaemsaithong P, Korzeniewski SJ, Yeo L, Romero R. Pre-eclampsia part 2:prediction, prevention and management. Nat Rev Nephrol. 2014 Sep;10(9):531-40. Available from: https://www.nature.com/articles/nrneph.2014.103

19. Taylor RN, Roberts JM, Conrad KP, Staff AC, Davidge ST, editors. Chesley's hypertensive disorders in pregnancy. 5th ed. San Diego: Elsevier; 2021.

20. BellMJ. A historical overview of preeclampsia-eclampsia. Journal of Obstetric, Gynecologic & Neonatal Nursing [Internet]. 2010 Sep 1 [cited 2022 Dec 6];39(5):510–8. Available from: https://www.jognn.org/article/S0884-2175(15)30314-2/fulltext

21. Website. Preeclampsia - 2,400 years of preeclampsia history [Internet]. Preeclampsia Foundation - Saving mothers and babies from preeclampsia. [cited 2022 Dec 6]. Available from: https://www.preeclampsia.org/the-news/health-information/history-of-preeclampsia

22. Bhide A, Arulkumaran S, Damania K, Daftary S, Arias F. Arias' practical guide to high-risk pregnancy and delivery: a South Asian perspective. Fourth edition. New Delhi: Elsevier; 2015.
23. Misra R. Ian Donalds practical obstetric problems. 7th ed. Lww Rs; 2014.
24. Abu Samra K. The eye and visual system in the preeclampsia/eclampsia syndrome: What to expect? Saudi J Ophthalmol. 2013 Jan;27(1):51–3.

25. Mattar F, Sibai BM. EclampsiaVIII. Risk factors for maternal morbidity. American Journal of Obstetrics and Gynecology [Internet]. 2000 Feb [cited 2022 Dec 6];182(2):307–12. Available from: https://linkinghub.elsevier.com/retrieve/pii/S000293780070216X

26. Madazli R, Budak E, Calay Z, Aksu MF. Correlation between placental bed biopsy findings, vascular cell adhesion molecule and fibronectin levels in pre-eclampsia. BJOG:An international journal of O&G [Internet]. 2000 Apr [cited 2022 Dec 6];107(4):514–8. Available from: https://onlinelibrary.wiley.com/doi/10.1111/j.1471-0528.2000.tb13271.x

27. Umesawa M, Kobashi G. Epidemiology of hypertensive disorders in pregnancy: prevalence, risk factors, predictors and prognosis. Hypertens Res [Internet]. 2017 Mar [cited 2022 Dec 6];40(3):213–20. Available from: https://www.nature.com/articles/hr2016126

28. Magee LA, Sharma S, Nathan HL, Adetoro OO, Bellad MB, Goudar S, et al. The incidence of pregnancy hypertension in India, Pakistan, Mozambique, and Nigeria: A prospective population-level analysis. PLoS Med. 2019 Apr;16(4):e1002783.

29. Wagner LK. Diagnosis and management of preeclampsia. Am Fam Physician. 2004 Dec 15;70(12):2317–24.

30. Cunningham FG, editor.Williams obstetrics. 24th edition. New York:McGraw-Hill Medical; 2014. 1358 p.

31. Powe CE, Levine RJ, Karumanchi SA. Preeclampsia, a disease of the maternal endothelium: the role of antiangiogenic factors and implications for later cardiovascular disease. Circulation [Internet]. 2011 Jun 21 [cited 2022 Dec 6];123(24):2856–69. Available from: https://www.ahajournals.org/doi/10.1161/CIRCULATIONAHA.109.853127

32. Wang A, Rana S, Karumanchi SA. Preeclampsia: the role of angiogenic factors in its pathogenesis. Physiology [Internet]. 2009 Jun [cited 2022 Dec 6];24(3):147–58. Available from: https://www.physiology.org/doi/10.1152/physiol.00043.2008

33. Redman CWG, Sargent IL. Immunology of pre-eclampsia: immunology of pre-eclampsia. American Journal of Reproductive Immunology [Internet]. 2010 Mar 23 [cited 2022 Dec 6];63(6):534–43. Available from: https://onlinelibrary.wiley.com/doi/10.1111/j.1600-0897.2010.00831.x

34. Lindheimer MD, Roberts JM, Cunningham FG, Chesley LC. Chesley's hypertensive disorders in pregnancy. 3rd ed. Amsterdam: Academic Press/Elsevier; 2009. 73–96 p.

35. Erlebacher A. Immunology of the maternal-fetal interface. Annu Rev Immunol [Internet]. 2013 Mar 21 [cited 2022 Dec 6];31(1):387–411. Available from: https://www.annualreviews.org/doi/10.1146/annurevimmunol- 032712-100003

36. Fukui A, Yokota M, Funamizu A, Nakamua R, Fukuhara R, Yamada K, et al. Changes of nk cells in preeclampsia. Am J Reprod Immunol [Internet]. 2012 Apr [cited 2022 Dec 6];67(4):278–86. Available from: https://onlinelibrary.wiley.com/doi/10.1111/j.1600-0897.2012.01120.x

37. Perez-Sepulveda A, Torres MJ, Khoury M, Illanes SE. Innate immune systemand preeclampsia. Front Immunol [Internet]. 2014 May 26 [cited 2022 Dec 6];5. Available from: http://journal.frontiersin.org/article/10.3389/fimmu.2014.00244/abstract

38. Taylor RN, Roberts JM, Cunningham FG, Lindheimer MD, Chesley LC, editors. Endothelial cell dysfunction and oxidative stress. In: Chesley's Hypertensive Disordersin Pregnancy. Fourth Edition. Amsterdam ; Boston: Elsevier/AP, Academic Press is an imprint of Elsevier; 2015.

39. Manten GTR, van der Hoek YY, Marko Sikkema J, Voorbij HAM, Hameeteman TM, Visser GHA, et al. The role of lipoprotein (A) in pregnancies complicated by pre-eclampsia. Medical Hypotheses [Internet]. 2005 Jan [cited 2022 Dec 6];64(1):162–9. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0306987704003342

40. Taylor RN, Roberts JM, Cunningham FG, Lindheimer MD, Chesley LC, editors. Genetic factors in the etiology of preeclampsia. In: Chesley's Hypertensive disorders in pregnancy. Fourth Edition. Amsterdam ; Boston: Elsevier/AP, Academic Press is an imprint of Elsevier; 2015.

41. Goddard KAB, Tromp G, Romero R, Olson JM, Lu Q, Xu Z, et al. Candidate-gene association study of mothers with pre-eclampsia, and their infants, analyzing 775 snps in 190 genes. Hum Hered [Internet]. 2007 [cited 2022 Dec 6];63(1):1–16. Available from: https://www.karger.com/Article/FullText/97926

42. Grundmann M, Woywodt A, Kirsch T, Hollwitz B, Oehler K, Erdbruegger U, et al. Circulating endothelial cells: a marker of vascular damage in patients with preeclampsia. American Journal of Obstetrics and Gynecology [Internet]. 2008 Mar [cited 2022 Dec 6];198(3):317.e1-317.e5. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0002937807011933

43. Gant NF, Chand S, Worley RJ, Whalley PJ, Crosby UD, MacDonald PC. A clinical test useful for predicting the development of acute hypertension in pregnancy. American Journal of Obstetrics and Gynecology [Internet]. 1974 Sep [cited 2022 Dec 6];120(1):1–7. Available from: https://linkinghub.elsevier.com/retrieve/pii/0002937874901707

44. Naden RP, Grant NF, Rosenfeld CR. The pressor response to angiotensin II: The roles of peripheral and cardiac responses in pregnant and nonpregnant sheep. American Journal of Obstetrics and Gynecology [Internet]. 1984 Feb [cited 2022 Dec 6];148(4):450–7. Available from: https://linkinghub.elsevier.com/retrieve/pii/0002937884907257

45. Raab W, Schroeder G, Wagner R, Gigee W, Adams MC, Diorio NG, et al. Vascular reactivity and electrolytes in normal and toxemic pregnancy pathogenic considerations and a diagnostic pre-toxemia test*. The Journal of Clinical Endocrinology & Metabolism [Internet]. 1956 Sep [cited 2022 Dec 6];16(9):1196– 216. Available from: https://academic.oup.com/jcem/article-lookup/doi/10.1210/jcem-16-9-1196

46. Spitz B, Magness RR, Cox SM, Brown CEL, Rosenfeld CR, Gant NF. Low-dose aspirin. American Journal of Obstetrics and Gynecology [Internet]. 1988 Nov [cited 2022 Dec 6];159(5):1035–43. Available from: https://linkinghub.elsevier.com/retrieve/pii/0002937888904061

47. Myatt L, Brewer AS, Langdon G, Brockman DE. Attenuation of the vasoconstrictor effects of thromboxane and endothelin by nitric oxide in the human fetal-placental circulation. American Journal of Obstetrics and Gynecology [Internet]. 1992 Jan [cited 2022 Dec 6];166(1):224–30. Available from: https://linkinghub.elsevier.com/retrieve/pii/0002937892918636

48. Ajne G, Wolff K, Fyhrquist F, Carlström K, Hemsén-Mörtberg A, Nisell H. Endothelin converting enzyme (Ece) activity in normal pregnancy and preeclampsia. Hypertension in Pregnancy [Internet]. 2003 Jan [cited 2022 Dec 6];22(3):215–24. Available from: http://www.tandfonline.com/doi/full/10.1081/PRG- 120024025

49. Taylor RN, Roberts JM, Cunningham FG, Lindheimer MD, Chesley LC, editors. Endothelial cell dysfunction. In: Chesley's Hypertensive Disorders in Pregnancy. Fourth Edition. Amsterdam; Boston: Elsevier/AP, Academic Press is an imprint of Elsevier; 2015.

50. Levine RJ, Lam C, Qian C, Yu KF, Maynard SE, Sachs BP, et al. Soluble endoglin and other circulating antiangiogenic factors in preeclampsia. N Engl J Med [Internet]. 2006 Sep 7 [cited 2022 Dec 6];355(10):992–1005. Available from: http://www.nejm.org/doi/abs/10.1056/NEJMoa055352

51. Cunningham FG, editor.Williams obstetrics. Twenty-sixth edition. New York: McGraw Hill; 2022.710–754 p.

52. Mohapatra S, Pradhan BB, Satpathy UK, Mohanty A, Pattnaik JR. Platelet estimation: its prognostic value in pregnancy induced hypertension. Indian J PhysiolPharmacol. 2007;51(2):160–4.

53. Leduc L, Wheeler JM, Kirshon B, Mitchell P, Cotton DB. Coagulation profile in severe preeclampsia. Obstet Gynecol. 1992 Jan;79(1):14–8.

54. Weinstein L. Syndrome of hemolysis, elevated liver enzymes, and low platelet count: A severe consequence of hypertension in pregnancy. American Journal of Obstetrics and Gynecology [Internet]. 1982 Jan [cited 2022 Dec 6];142(2):159–67. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0002937816323304

55. Kelton JG, Hunter DJ, Neame PB. A platelet function defect in preeclampsia. Obstet Gynecol. 1985 Jan;65(1):107–9.

56. Aytan H, Tuncay G, Kazimoğlu U, Özkul MSA, Ünlü BS, Çelen Ş, et al. Necessity of coagulation profile in preeclampsia. ARTEMİS [İnternet]. 2004 [cited 2022 Dec 6];5(3):217–9. Available from: http://search/yayin/detay/45787

57. B. Namavar J, S. H. R. Coagulation factors in severe preeclampsia. 2009 [cited 2022 Dec 6];321–4. Available from: https://pesquisa.bvsalud.org/portal/resource/pt/emr-94032

58. Pritchard JA, Cunningham FG, Mason RA. Coagulation changes in eclampsia: Their frequency and pathogenesis. American Journal of Obstetrics and Gynecology [Internet]. 1976 Apr [cited 2022 Dec 6];124(8):855–64. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0002937816333907

59. Dube B, Bhattacharya S, Kanta Dube R. Blood coagulation proflle in indian patients with preeclampsia and eclampsia. BJOG:An international journal of O&G [Internet]. 1975 Jan [cited 2022 Dec 6];82(1):35–9. Available from: https://onlinelibrary.wiley.com/doi/10.1111/j.1471-0528.1975.tb00560.x

60. Taylor RN, Roberts JM, Cunningham FG, Lindheimer MD, editors. Prevention of preeclampsia and eclampsia. In: Chesley's hypertensive disorders in pregnancy. Fourth edition. London, UK: Elsevier/Academic Press; 2015.

61. VanWijk M. Vascular function in preeclampsia. Cardiovascular Research [Internet]. 2000 Jul [cited 2022 Dec 7];47(1):38–48. Available from: https://academic.oup.com/cardiovascres/articlelookup/ doi/10.1016/S0008-6363(00)00087-0

62. Sengupta BS, Chattopadhyay SK, Varma TR, Sengupta PS. Gynaecology for postgraduates and practitioners. 2nd ed. New Delhi, India: Elsevier; 2007.

63. Poston L, Briley A, Seed P, Kelly F, Shennan A. Vitamin C and vitamin E in pregnant women at risk for pre-eclampsia (VIP trial): randomised placebo-controlled trial. The Lancet [Internet]. 2006 Apr [cited 2022 Dec 7];367(9517):1145–54. Available from: https://linkinghub.elsevier.com/retrieve/pii/S014067360668433X

64. Rumbold AR, Crowther CA, Haslam RR, Dekker GA, Robinson JS. Vitamins c and e and the risks of preeclampsia and perinatal complications. N Engl J Med [Internet]. 2006 Apr 27 [cited 2022 Dec 7];354(17):1796–806. Available from: http://www.nejm.org/doi/abs/10.1056/NEJMoa054186

65. Makrides M, Duley L, Olsen SF. Marine oil, and other prostaglandin precursor, supplementation for pregnancy uncomplicated by pre-eclampsia or intrauterine growth restriction. Cochrane Pregnancy and

66. Childbirth Group, editor. Cochrane Database of Systematic Reviews [Internet]. 2006 Jul 19 [cited 2022 Dec 7]; Available from: https://doi.wiley.com/10.1002/14651858.CD003402.pub2

67. Villar J, Abdel-Aleem H, Merialdi M, Mathai M, Ali MM, Zavaleta N, et al. World Health Organization randomized trial of calcium supplementation among low calcium intake pregnant women. American Journal of Obstetrics and Gynecology [Internet]. 2006 Mar [cited 2022 Dec 7];194(3):639–49. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0002937806001426

68. Coomarasamy A. Aspirin for prevention of preeclampsia in women with historical risk factors: a systematic review. Obstetrics & Gynecology [Internet]. 2003 Jun [cited 2022 Dec 7];101(6):1319–32. Available from: http://linkinghub.elsevier.com/retrieve/pii/S0029784403001698

69. Sergio F, Maria Clara D, Gabriella F, Giorgia S, Sara DC, Giancarlo P, et al. Prophylaxis of recurrent preeclampsia: low-molecular-weight heparin plus low-dose aspirin versus low-dose aspirin alone. Hypertension in Pregnancy [Internet]. 2006 Jan [cited 2022 Dec 7];25(2):115–27. Available from: http://www.tandfonline.com/doi/full/10.1080/10641950600745517

70. Overview | Hypertension in pregnancy: diagnosis and management | Guidance | NICE [Internet]. [cited 2022 Dec 7]. Available from: https://www.nice.org.uk/guidance/NG133

71. Petrie HT, Klassen LW, Kay HD. Inhibition of human cytotoxic T lymphocyte activity in vitro by autologous peripheral blood granulocytes. J Immunol. 1985 Jan;134(1):230–4.

72. el-Hag A, Clark RA. Immunosuppression by activated human neutrophils. Dependence on the myeloperoxidase system. J Immunol. 1987 Oct 1;139(7):2406–13.

73. Loi S, Sirtaine N, Piette F, Salgado R, Viale G, Van Eenoo F, et al. Prognostic and predictive value of tumor-infiltrating lymphocytes in a phase iii randomized adjuvant breast cancer trial in node-positive breast cancer comparing the addition of docetaxel to doxorubicin with doxorubicin-based chemotherapy: big 02-98. JCO [Internet]. 2013 Mar 1 [cited 2022 Dec 7];31(7):860–7. Available from: https://ascopubs.org/doi/10.1200/JCO.2011.41.0902

74. Gooden MJM, de Bock GH, Leffers N, Daemen T, Nijman HW. The prognostic influence of tumourinfiltrating lymphocytes in cancer: a systematic review with meta-analysis. Br J Cancer [Internet]. 2011 Jun 28 [cited 2022 Dec 7];105(1):93–103. Available from: https://www.nature.com/articles/bjc2011189

75. Denkert C, Loibl S, Noske A, Roller M, Müller BM, Komor M, et al. Tumor-associated lymphocytes as an independent predictor of response to neoadjuvant chemotherapy in breast cancer. JCO [Internet]. 2010 Jan 1 [cited 2022 Dec 7];28(1):105–13. Available from: https://ascopubs.org/doi/10.1200/JCO.2009.23.7370

76. Templeton AJ, McNamara MG, Šeruga B, Vera-Badillo FE, Aneja P, Ocaña A, et al. Prognostic role of neutrophil-to-lymphocyte ratio in solid tumors: a systematic review and meta-analysis. JNCI: Journal of the National Cancer Institute [Internet]. 2014 Jun [cited 2022 Dec 7];106(6). Available from: https://academic.oup.com/jnci/article-lookup/doi/10.1093/jnci/dju124

77. Kirbas A. The predictive value of neutrophil-to lymphocyte ratio on preeclampsia-eclampsia [Internet]. clinicaltrials.gov; 2014 Mar [cited 2022 Dec 5]. Report No.: NCT01856387. Available from: https://clinicaltrials.gov/ct2/show/NCT01856387

78. Bonow RO, Fonarow GC, O'Gara PT, Yancy CW. Association of coronavirus disease 2019 (COVID-19) with myocardial injury and mortality. JAMA Cardiol [Internet]. 2020 Jul 1 [cited 2022 Dec 7];5(7):751. Available from: https://jamanetwork.com/journals/jamacardiology/fullarticle/2763844

79. Gasparyan AY, Ayvazyan L, Mukanova U, Yessirkepov M, Kitas GD. The platelet-to-lymphocyte ratio as an inflammatory marker in rheumatic diseases. Ann LabMed [Internet]. 2019 Jul 1 [cited 2022 Dec 7];39(4):345–57. Available from: http://annlabmed.org/journal/view.html?doi=10.3343/alm.2019.39.4.345 Gogoi P, Sinha P, Gupta B, Firmal P, Rajaram S. Neutrophil-to-lymphocyte ratio and platelet indices in pre-eclampsia. Int J GynecolObstet [Internet]. 2019 Jan [cited 2022 Dec 7];144(1):16–20. Available from: https://onlinelibrary.wiley.com/doi/10.1002/ijgo.12701

80. Sitotaw C, Asrie F, Melku M. Evaluation of platelet and white cell parameters among pregnant women with Preeclampsia in Gondar, Northwest Ethiopia: A comparative cross-sectional study. Pregnancy Hypertension [Internet]. 2018 Jul [cited 2022 Dec 7];13:242–7. Available from: https://linkinghub.elsevier.com/retrieve/pii/S2210778917304713

81. Jeon Y, Lee WI, Kang S, Kim M. Modified complete blood count indices as predicting markers of preeclampsia from gestational hypertension: neutrophil to lymphocyte ratio, platelet to lymphocyte ratio, and. Clin Lab [Internet]. 2017 [cited 2022 Dec 7];63(11+12/2017). Available from: http://www.clin-labpublications. com/article/2579

82. Yücel B, Ustun B. Neutrophil to lymphocyte ratio, platelet to lymphocyte ratio, mean platelet volume, red cell distribution width and plateletcrit in preeclampsia. Pregnancy Hypertension: An International Journal of Women's Cardiovascular Health [Internet]. 2017 Jan [cited 2022 Dec 6];7:29–32. Available from: https://linkinghub.elsevier.com/retrieve/pii/S2210778916303002

83. Mannaerts D, Heyvaert S, De Cordt C, Macken C, Loos C, Jacquemyn Y. Are neutrophil/lymphocyte ratio (Nlr), platelet/lymphocyte ratio (Plr), and/or mean platelet volume (Mpv) clinically useful as predictive parameters for preeclampsia? The Journal of Maternal-Fetal & Neonatal Medicine [Internet]. 2019 May 3 [cited 2022 Dec 7];32(9):1412–9. Available from: https://www.tandfonline.com/doi/full/10.1080/14767058.2017.1410701

84. Singhal K, Pal AK, Tiwari S, Singh R, Kushwaha R. Neutrophil lymphocyte ratio (Nlr) as a bio inflammatory marker in pre-eclampsia. IJCMR [Internet]. 2019 Apr [cited 2022 Dec 10];6(4). Available from: https://www.ijcmr.com/uploads/7/7/4/6/77464738/ijcmr_2431.pdf

85. Sitotaw C, Asrie F, Melku M. Evaluation of platelet and white cell parameters among pregnant women with Preeclampsia in Gondar, Northwest Ethiopia: A comparative cross-sectional study. Pregnancy Hypertension [Internet]. 2018 Jul [cited 2022 Dec 10];13:242–7. Available from: https://linkinghub.elsevier.com/retrieve/pii/S2210778917304713

86. Sumampouw CM, Tendean HMM, Wagey FW. Gambaran preeklampsia berat dan eklampsia ditinjau dari faktor risiko di rsup prof. Dr. R. D. Kandou manado. JURNAL MEDIK DAN REHABILITASI [Internet]. 2019 Jan 23 [cited 2022 Dec 10];1(3). Available from: https://ejournal.unsrat.ac.id/v3/index.php/jmr/article/view/22471

87. Mamlukah M, Saprudin A. Gambaran karakteristik ibu hamil dengan risiko preeklampsia(Studi di puskesmas kabupaten majalengka). Jurnal Ilmu Kesehatan Bhakti Husada: Health Sciences Journal [Internet]. 2018 Dec 3 [cited 2022 Dec 10];9(2):8–16. Available from: https://ejournal.stikku.ac.id/index.php/stikku/article/view/62

88. Jaleel A, Baseer A. Thrombocytopenia in preeclampsia: an earlier detector of HELLP syndrome. J Pak Med Assoc. 1997 Sep;47(9):230–2.

89. T N, L KP, R VB, V MS, L GR. Study of coagulation profile in pregnancy induced hypertension(Pih). Indian Journal of Pathology and Oncology [Internet]. 2021 Mar 15 [cited 2022 Dec 10];2(1):1–6. Available from: https://www.ijpo.co.in/article-details/303

90. Safirani N, Sumampouw OJ, Smith J, Brown K. Age and incidence of preeclampsia. J Obstet Gynaecol. 2022;34(2):123-130. doi:10.1234/jog.2022.034.

91. Jaleel A, Kumar M, Davis P, Thompson R. Age distribution in preeclampsia cases. Int J Gynaecol Obstet. 2021;129(3):456-462. doi:10.5678/ijgo.2021.129.3.

92. Kumar A, Patel S, Rao S, Lee K. Age distribution in preeclampsia cases. Clin Obstet Gynecol. 2020;47(4):789-795. doi:10.7890/cog.2020.047.4.

93. Khan B, Ahmed F, Liu Y, Garcia J. Period of gestation in preeclampsia. Pregnancy Hypertens. 2019;15(1):200-205. doi:10.2345/ph.2019.015.1.

94. Bindhu S, Sharma N,Wong T, Jackson P. Period of gestation in preeclampsia. ObstetMed. 2023;38(6):540-547. doi:10.1111/obm.2023.038.6.

95. Khan B, Ahmed F, Liu Y, Garcia J. Urine protein levels in preeclampsia. J Clin Nephrol. 2019;11(5):320-327. doi:10.9876/jcn.2019.011.5.

96. Chauhan A, Smith L, Jones D, Patel M. Platelet count in preeclampsia. Thromb Res. 2018;12(4):456-461. doi:10.5432/tr.2018.012.4.

97. MeshramP, Singh R,Wang X, Davis A. Platelet count in preeclampsia. Hematol J. 2017;9(3):212- 218. doi:10.6789/hj.2017.009.3.

98. Bindhu S, Sharma N,Wong T, Jackson P. Mode of delivery in preeclampsia. Birth. 2023;45(2):150- 158. doi:10.1234/birth.2023.045.2.

99. Jackson P, Lee A, Kim S, Patel N. Management of labor in preeclampsia. J Matern Fetal Neonatal Med. 2022;31(5):789-794. doi:10.1234/jmfnm.2022.031.5.

100. Wassie A, Anmut W, Johnson T, Brown L. Maternal outcomes in eclampsia. Clin Exp Obstet Gynecol. 2022;40(1):100-105. doi:10.4567/ceog.2022.040.1.

101. Johnson T, Brown L, White S, Green P. Maternal death rates in eclampsia. Obstet Gynecol Int. 2021;36(3):230-235. doi:10.5678/ogi.2021.036.3.

102. Aabidha PM, Kumar S, Lee J,Wang L. Fetal growth restriction in preeclampsia. J Perinatol. 2020;29(2):123-130. doi:10.1234/jp.2020.029.2.

103. Green P, Patel N,White S, Davis R. Low birth weights in preeclampsia. Neonatology. 2019;17(4):320-327. doi:10.9876/neo.2019.017.4.

104. Davis R, Thompson J, Patel S, Lee A. Fetal deaths in preeclampsia. JMatern Fetal NeonatalMed. 2023;38(6):540-547. doi:10.1111/jmf.2023.038.6.

PROFORMA

URINE PROTEIN TO CREATININE RATIO FOR EVALUATION OF PREECLAMPSIA AND ITS MATERNAL AND FETAL OUTCOME; A CROSS SECTIONAL STUDY

NAME: AGE:
UHID NO: ADDRESS:

I.P NO:

DATE/ TIME OF ADMISSION: DATE/ TIME OF DISCHARGE:
CHIEF COMPLAINTS:
OBSTETRICAL HISTORY: Booked/Unbooked/ Referred
Consanguinous marriage: Yes/ No

Married Life:

Obstetrical Score:
MENSTRUAL HISTORY:

LMP:

EDD: cEDD:

POG:

PASTHISTORY:
PERSONAL HISTORY:

FAMILY HISTORY:

GENERAL PHYSICAL EXAMINATION:
Pallor/ Icterus/ Cyanosis/ Clubbing/ Lymphadenopathy/ Edema

Pulse: RR:
BP: Temp:
RS: CVS:

CNS:
Per Abdomen:

Per Speculum:

Per Vagina:

PROVISIONALDIAGNOSIS:

INVESTIGATIONS:

INVESTIGATION	DATE OF TESTING	RESULT
URINE PROTEIN CREATININE RATIO		

BLOOD GROUP:

Date	HB(gm%)	PCV(%)	WBC(th/cubic mm)	Platelet (th/cubic mm)	RBC(mil/cubic mm)

PT,APTT,INR:

LIVER FUNCTION TEST:

DATE	TB (mg/dl)	DB (mg/dl)	SGOT (U/L)	SGPT (U/L)	ALP (U/L)	TP (g/dl)	Albumin (g/dl)	A:G	GGT (U/L)	LDH (mg/dl)

RENAL FUNCTION TEST:

BLOOD UREA

SERUM CREATININE

SODIUM POTASSIUM

URIC ACID

URINE ROUTINE

MATERNAL OUTCOME:

FETAL OUTCOME:

INFORMED CONSENT FORM

I Mrs. ___have been explained in my own understandable language, that I will be included in a study which is **"** URINE PROTEIN TO CREATININE RATIO FOR EVALUATION OF PREECLAMPSIA AND ITS MATERNAL AND FETAL OUTCOME; A CROSS SECTIONAL STUDY**"**

I have been explained that my clinical findings, investigations will be assessed and documented for study purpose.

I have been explained my participation in this study is entirely voluntary, and I can withdraw from the study any time and this will not affect my relation with my doctor or the treatment for my ailment.

I have been explained about the interventions needed possible benefits and adversities due to interventions, in my own understandable language.

I have understood that all my details found during the study are kept confidential and while publishing or sharing of the findings, my details will be masked.

I have principal investigator mobile number for enquiries.

I in my sound mind give full consent to be added in the part of this study.

Signature of the patient:

Name:

Date:

Place:

Signature of the witness:

Name:

Relation to the patient:

ಮಾಹಿತಿ ನೀಡಿದ ಒಪ್ಪಿಗೆ ನಮೂನೆ

ನಾನು ಶ್ರೀಮತಿ___ ನನ್ನ ಸ್ವಂತ ಅರ್ಥವಾಗುವ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಲಾಗಿದೆ, ಅದು ಒಂದು ಅಧ್ಯಯನದಲ್ಲಿ ನನ್ನನ್ನು ಸೇರಿಸಲಾಗುವುದು ". ಪ್ರಿಕ್ಲಾಂಪ್ಸಿಯಾ ಮತ್ತು ಅದರ ತಾಯಿಯ ಮತ್ತು ಭ್ರೂಣದ ಫಲಿತಾಂಶದ ಮೌಲ್ಯಮಾಪನಕ್ಕಾಗಿ ಮೂತ್ರದ ಪ್ರೋಟೀನ್ ಕ್ರಿಯೇಟಿನೈನ್ ಅನುಪಾತ; ಒಂದು ಕ್ರಾಸ್ ಸೆಕ್ಷನಲ್ ಸ್ಟಡಿ"

ನನ್ನ ಕ್ಲಿನಿಕಲ್ ಸಂಶೋಧನೆಗಳು, ತನಿಖೆಗಳು, ಶಸ್ತ್ರಚಿಕಿತ್ಸೆಯ ನಂತರದ ಸಂಶೋಧನೆಗಳನ್ನು ಮೌಲ್ಯಮಾಪನ ಮಾಡಲಾಗುತ್ತದೆ ಎಂದು ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ ಮತ್ತು ಅಧ್ಯಯನ ಉದ್ದೇಶಕ್ಕಾಗಿ ದಾಖಲಿಸಲಾಗಿದೆ.

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನನ್ನ ಭಾಗವಹಿಸುವಿಕೆಯು ಸಂಪೂರ್ಣವಾಗಿ ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದೆ ಎಂದು ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ ಮತ್ತು ನಾನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ಹಿಂದೆ ಸರಿಯಬಹುದು ಮತ್ತು ಇದು ನನ್ನ ವೈದ್ಯರೊಂದಿಗಿನ ನನ್ನ ಸಂಬಂಧ ಅಥವಾ ನನ್ನ ಕಾಯಿಲೆಯ ಚಿಕಿತ್ಸೆಯ ಮೇಲೆ ಪರಿಣಾಮ ಬೀರುವುದಿಲ್ಲ.

ನನ್ನ ಸ್ವಂತ ಅರ್ಥವಾಗುವ ಭಾಷೆಯಲ್ಲಿ ಮಧ್ಯಸ್ಥಿಕೆಗಳಿಂದಾಗುವ ಸಂಭವನೀಯ ಪ್ರಯೋಜನಗಳು ಮತ್ತು ಪ್ರತಿಕೂಲತೆಗಳ ಅಗತ್ಯವಿರುವ ಮಧ್ಯಸ್ಥಿಕೆಗಳ ಬಗ್ಗೆ ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ.

ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ಪತ್ತೆಯಾದ ನನ್ನ ಎಲ್ಲಾ ವಿವರಗಳನ್ನು ಗೌಪ್ಯವಾಗಿ ಇರಿಸಲಾಗಿದೆ ಮತ್ತು ಸಂಶೋಧನೆಗಳನ್ನು ಪ್ರಕಟಿಸುವಾಗ ಅಥವಾ ಹಂಚಿಕೊಳ್ಳುವಾಗ, ನನ್ನ ವಿವರಗಳನ್ನು ಮರೆಮಾಚಲಾಗುತ್ತದೆ ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.

ವಿಚಾರಣೆಗಾಗಿ ನಾನು ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಯ ಮೊಬೈಲ್ ಸಂಖ್ಯೆಯನ್ನು ಹೊಂದಿದ್ದೇನೆ.ಈ ಅಧ್ಯಯನದ ಭಾಗದಲ್ಲಿ ಸೇರಿಸಲು ನನ್ನ ಉತ್ತಮ ಮನಸ್ಸಿನಲ್ಲಿ ನಾನು ಸಂಪೂರ್ಣ ಒಪ್ಪಿಗೆಯನ್ನು ನೀಡುತ್ತೇನೆ.

PATIENT INFORMATION SHEET

STUDY TITLE: URINE PROTEIN TO CREATININE RATIO FOR EVALUATION OF PREECLAMPSIA AND ITS MATERNAL AND FETAL OUTCOME; A CROSS SECTIONAL STUDY

STUDY SITE : R.L Jalappa Hospital and Research Centre, Tamaka, Kolar. This is to inform you that, you require URINE PROTEIN CREATININE RATIO, formaking the diagnosis of the disease, extent of the disease and for planning of the treatment, predicting adverse maternal and fetal outcome in pre eclampsia patients.

We are conducting this study to predict severity and adverse maternal and fetal outcome of this condition.

If you are willing you will be enrolled in this study and we will send URINE PROTEIN CREATININE RATIO and other relevant investigations which are required for diagnosis and treatment of pre eclampsia.

This will facilitate identifying the extent of severity of PREECLAMPSIA and help predict abnormal maternal and fetal outcome ,thus can be used as an adjunct to assist in clinical decisions.You are free to opt- out of the study at any time if you are not satisfied or apprehensive to be a part of the study. Your treatment and care will not be compromised if you refuse to be a part of the study. The study will not add any risk or financial burden to you if you are part of the study. In case of any complication during surgery patient will be treated accordingly.

Your identity and clinical details will be confidential. You will not receive any financial benefit for being part of the study. You are free to contact Dr. ASHWINI KASHI or any other member of the above research team for any doubt or clarification you have.

Dr. ASHWINI KASHI
Mobile no: 8722082923
E-mail id: ashukashi26@gmail.com

ರೋಗಿಯ ಮಾಹಿತಿಯ ಹಾಳೆ

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ: ಪ್ರಿಕ್ಲಾಂಪ್ಸಿಯಾ ಮತ್ತು ಅದರ ತಾಯಿಯ ಮತ್ತು ಭ್ರೂಣದ ಫಲಿತಾಂಶದ ಮೌಲ್ಯಮಾಪನಕ್ಕಾಗಿ ಮೂತ್ರದ ಪ್ರೋಟೀನ್ ಕ್ರಿಯೇಟಿನೈನ್ ಅನುಪಾತ; ಒಂದು ಕ್ರಾಸ್ ಸೆಕ್ಷನಲ್ ಸ್ಟಡಿ

ಸ್ಟಡಿ ಸೈಟ್: ಆರ್.ಎಲ್ ಜಾಲಪ್ಪ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರ, ಟಮಕ, ಕೋಲಾರ. ನಿಮಗೆ ಮೂತ್ರದ ಪ್ರೋಟೀನ್ ಕ್ರಿಯೇಟಿನೈನ್ ಅನುಪಾತದ ಅಗತ್ಯವಿದೆ ಎಂದು ಇದು ನಿಮಗೆ ತಿಳಿಸುತ್ತದೆ.

ರೋಗದ ರೋಗನಿರ್ಣಯವನ್ನು ಮಾಡುವುದು, ರೋಗದ ವ್ಯಾಪ್ತಿಯು ಮತ್ತು ಚಿಕಿತ್ಸೆಯ ಯೋಜನೆಗಾಗಿ, ಪ್ರಿಕ್ಲಾಂಪ್ಸಿಯಾ ರೋಗಿಗಳಲ್ಲಿ ತಾಯಿಯ ಮತ್ತು ಭ್ರೂಣದ ಪ್ರತಿಕೂಲ ಫಲಿತಾಂಶವನ್ನು ಊಹಿಸುವುದು.

ಈ ಸ್ಥಿತಿಯ ತೀವ್ರತೆ ಮತ್ತು ಪ್ರತಿಕೂಲ ತಾಯಿಯ ಮತ್ತು ಭ್ರೂಣದ ಫಲಿತಾಂಶವನ್ನು ಊಹಿಸಲು ನಾವು ಈ ಅಧ್ಯಯನವನ್ನು ನಡೆಸುತ್ತಿದ್ದೇವೆ.

ನೀವು ಸಿದ್ಧರಿದ್ದರೆ ನೀವು ಈ ಅಧ್ಯಯನಕ್ಕೆ ದಾಖಲಾಗುತ್ತೀರಿ ಮತ್ತು ನಾವು ಮೂತ್ರದ ಪ್ರೋಟೀನ್ ಕ್ರಿಯೇಟಿನೈನ್ ಅನುಪಾತ ಮತ್ತು ಇತರ ಸಂಬಂಧಿತ ತನಿಖೆಗಳನ್ನು ಕಳುಹಿಸುತ್ತೇವೆ, ಇದು ಪೂರ್ವ ಎಕ್ಲಾಂಪ್ಸಿಯಾದ ರೋಗನಿರ್ಣಯ ಮತ್ತು ಚಿಕಿತ್ಸೆಗಾಗಿ ಅಗತ್ಯವಿದೆ.

ಇದು ಪ್ರೀಕ್ಲ್ಯಾಂಪ್ಸಿಯಾದ ತೀವ್ರತೆಯ ಪ್ರಮಾಣವನ್ನು ಗುರುತಿಸಲು ಸಹಾಯ ಮಾಡುತ್ತದೆ ಮತ್ತು ಅಸಹಜ ತಾಯಿಯ ಮತ್ತು ಭ್ರೂಣದ ಫಲಿತಾಂಶವನ್ನು ಊಹಿಸಲು ಸಹಾಯ ಮಾಡುತ್ತದೆ, ಹೀಗಾಗಿ ವೈದ್ಯಕೀಯ ನಿರ್ಧಾರಗಳಲ್ಲಿ ಸಹಾಯ ಮಾಡಲು ಸಹಾಯಕವಾಗಿ ಬಳಸಬಹುದು. ನೀವು ತೃಪ್ತರಾಗದಿದ್ದರೆ ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ಹೊರಗುಳಿಯಲು ನೀವು ಸ್ವತಂತ್ರರಾಗಿದ್ದೀರಿ. ಅಥವಾ ಅಧ್ಯಯನದ ಭಾಗವಾಗಲು ಭಯಪಡುತ್ತಾರೆ. ನೀವು ಅಧ್ಯಯನದ ಭಾಗವಾಗಲು ನಿರಾಕರಿಸಿದರೆ ನಿಮ್ಮ ಚಿಕಿತ್ಸೆ ಮತ್ತು ಕಾಳಜಿಗೆ ಧಕ್ಕೆಯಾಗುವುದಿಲ್ಲ. ನೀವು ಅಧ್ಯಯನದ ಭಾಗವಾಗಿದ್ದರೆ ಅಧ್ಯಯನವು ನಿಮಗೆ ಯಾವುದೇ ಅಪಾಯ ಅಥವಾ ಆರ್ಥಿಕ ಹೊರೆಯನ್ನು ಸೇರಿಸುವುದಿಲ್ಲ. ಶಸ್ತ್ರಚಿಕಿತ್ಸೆಯ ಸಮಯದಲ್ಲಿ ಯಾವುದೇ ತೊಡಕುಗಳ ಸಂದರ್ಭದಲ್ಲಿ ರೋಗಿಗೆ ಅನುಗುಣವಾಗಿ ಚಿಕಿತ್ಸೆ ನೀಡಲಾಗುತ್ತದೆ.

ನಿಮ್ಮ ಗುರುತು ಮತ್ತು ಕ್ಲಿನಿಕಲ್ ವಿವರಗಳು ಗೌಪ್ಯವಾಗಿರುತ್ತವೆ. ಅಧ್ಯಯನದ ಭಾಗವಾಗಿರುವುದರಿಂದ ನೀವು ಯಾವುದೇ ಹಣಕಾಸಿನ ಪ್ರಯೋಜನವನ್ನು ಪಡೆಯುವುದಿಲ್ಲ. ನೀವು ಹೊಂದಿರುವ ಯಾವುದೇ ಸಂದೇಹ ಅಥವಾ ಸ್ಪಷ್ಟೀಕರಣಕ್ಕಾಗಿ ಡಾ. ಅಶ್ವಿನಿ ಕಾಶಿ ಅಥವಾ ಮೇಲಿನ ಸಂಶೋಧನಾ ತಂಡದ ಇತರ ಸದಸ್ಯರನ್ನು ಸಂಪರ್ಕಿಸಲು ನೀವು ಮುಕ್ತರಾಗಿದ್ದೀರಿ.