MATERNAL SERUM URIC ACID AS A PROGNOSTIC FACTOR FOR PERINATAL OUTCOME IN HYPERTENSIVE DISORDERS OF PREGNANCY

By

Dr GUMMALLAPU AKSHAYA, MBBS

Dissertation submitted to the

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR – 563 101

In partial fulfilment of the requirements for the degree of

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IN

OBSTETRICS AND GYNAECOLOGY

Under the Guidance of

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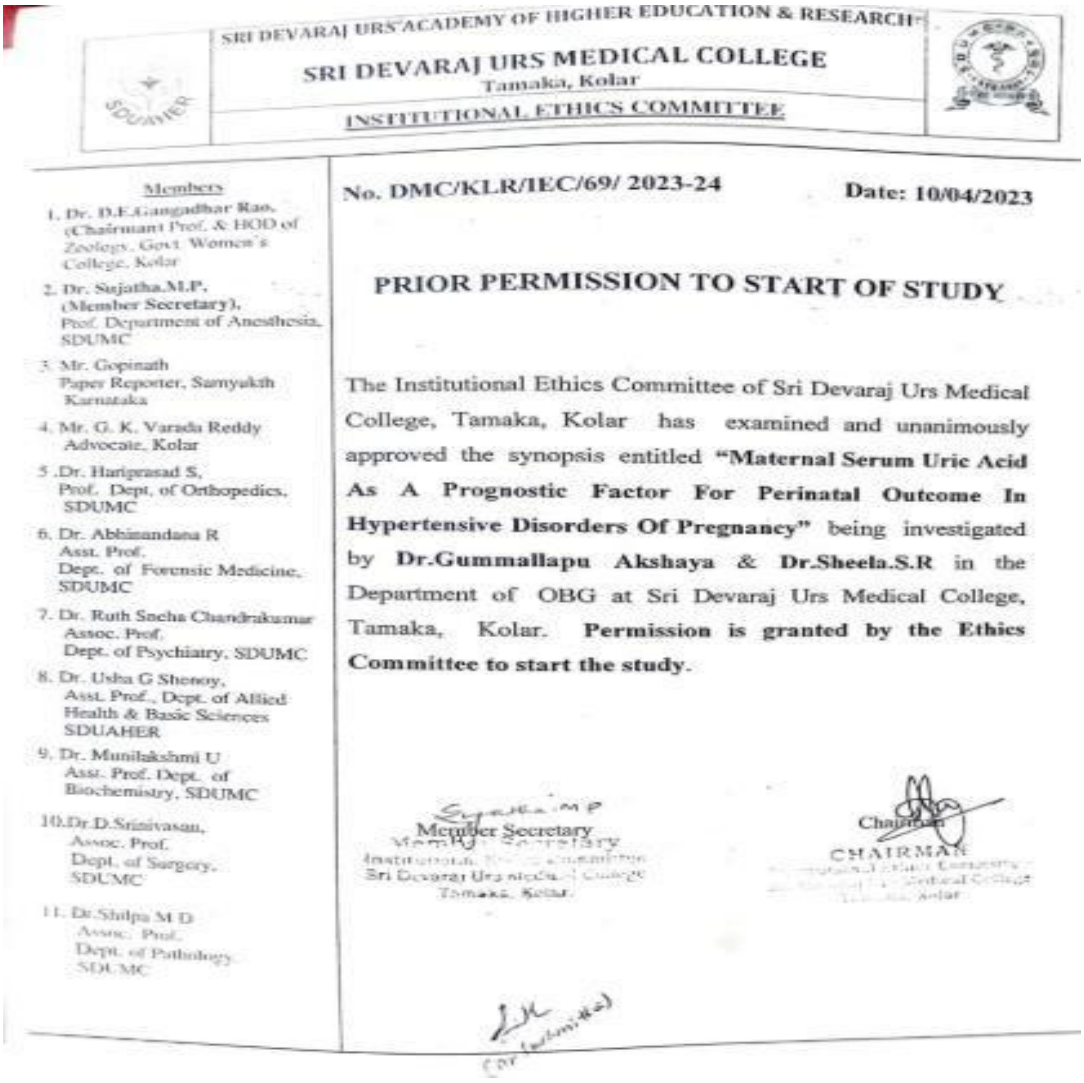

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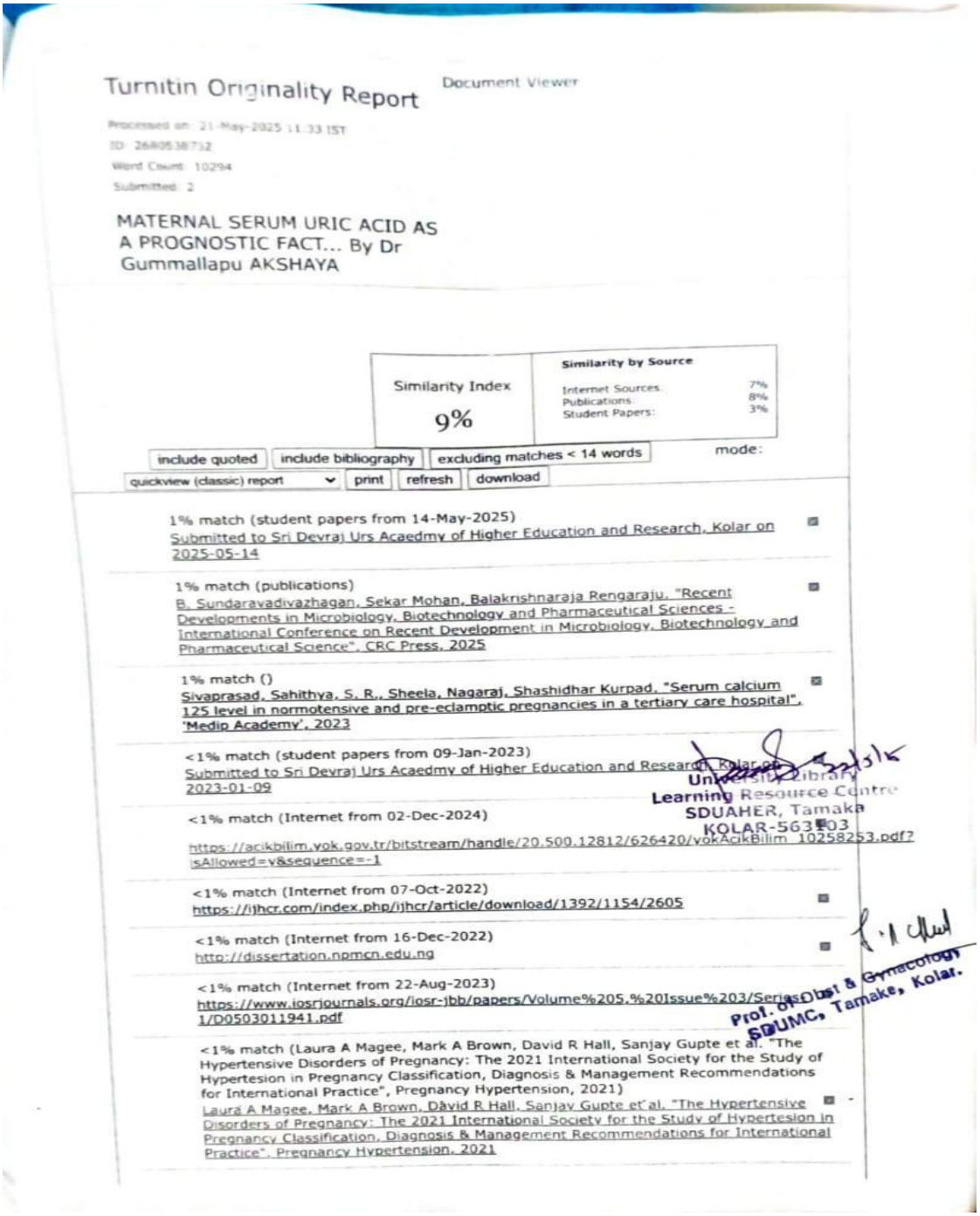

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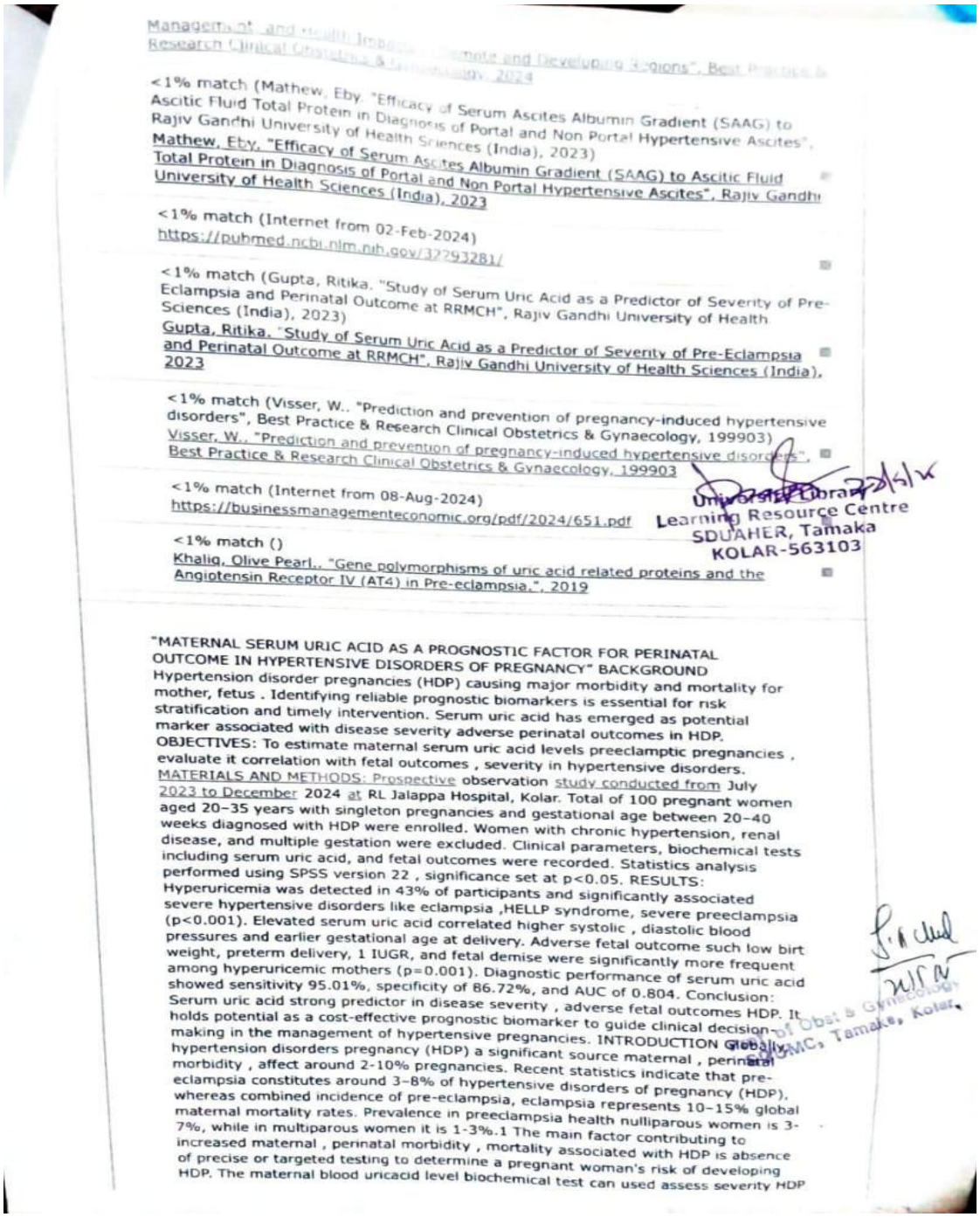

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"MATERNAL SERUM URIC ACID AS A PROGNOSTIC FACTOR FOR PERINATAL OUTCOME IN HYPERTENSIVE DISORDERS OF PREGNANCY" BACKGROUND Hypertension disorder pregnancies (HDP) causing major morbidity and mortality for mother, fetus . Identifying reliable prognostic biomarkers is essential for risk stratification and timely intervention. Serum uric acid has emerged as potential marker associated with disease severity adverse perinatal outcomes in HDP. OBJECTIVES: To estimate maternal serum uric acid levels preeclamptic pregnancies , evaluate it correlation with fetal outcomes , severity in hypertensive disorders. MATERIALS AND METHODS: Prospective observation study conducted from July 2023 to December 2024 at RL Jalappa Hospital, Kolar. Total of 100 pregnant women aged 20–35 years with singleton pregnancies and gestational age between 20–40 weeks diagnosed with HDP were enrolled. Women with chronic hypertension, renal disease, and multiple gestation were excluded. Clinical parameters, biochemical tests including serum uric acid, and fetal outcomes were recorded. Statistics analysis performed using SPSS version 22 , significance set at $p<0.05$. RESULTS: Hyperuricemia was detected in 43% of participants and significantly associated severe hypertensive disorders like eclampsia ,HELLP syndrome, severe preeclampsia ($p<0.001$). Elevated serum uric acid correlated higher systolic , diastolic blood pressures and earlier gestational age at delivery. Adverse fetal outcome such low birt weight, preterm delivery, 1 IUGR, and fetal demise were significantly more frequent among hyperuricemic mothers ($p=0.001$). Diagnostic performance of serum uric acid showed sensitivity 95.01%, specificity of 86.72%, and AUC of 0.804. Conclusion: Serum uric acid strong predictor in disease severity , adverse fetal outcomes HDP. It holds potential as a cost-effective prognostic biomarker to guide clinical decision-making in the management of hypertensive pregnancies. INTRODUCTION Globally, hypertension disorders pregnancy (HDP) a significant source maternal , perinatal morbidity , affect around 2-10% pregnancies. Recent statistics indicate that pre-eclampsia constitutes around 3–8% of hypertensive disorders of pregnancy (HDP), whereas combined incidence of pre-eclampsia, eclampsia represents 10–15% global maternal mortality rates. Prevalence in preeclampsia health nulliparous women is 3-7%, while in multiparous women it is 1-3%.1 The main factor contributing to increased maternal , perinatal morbidity , mortality associated with HDP is absence of precise or targeted testing to determine a pregnant woman's risk of developing HDP. The maternal blood uricacid level biochemical test can used assess severity HDP

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S. No.	Abbreviation	Full Form
1.	HDP	Hypertensive Disorders of Pregnancy
2.	SBP	Systolic Blood Pressure
3.	DBP	Diastolic Blood Pressure
4.	HELLP	Hemolysis, Elevated Liver enzymes, and Low Platelet count
5.	IUGR	Intrauterine Growth Restriction
6.	IUD	Intrauterine Death
7.	LBW	Low Birth Weight
8.	LSCS	Lower Segment Cesarean Section
9.	AUC	Area Under the Curve
10.	SPSS	Statistical Package for the Social Sciences
11.	OPD	Outpatient Department
12.	BMI	Body Mass Index
13.	ALT	Alanine Aminotransferase
14.	AST	Aspartate Aminotransferase
15.	eNOS	Endothelial Nitric Oxide Synthase

ABSTRACT

"MATERNAL SERUM URIC ACID AS A PROGNOSTIC FACTOR FOR PERINATAL OUTCOME IN HYPERTENSIVE DISORDERS OF PREGNANCY"

BACKGROUND

Hypertension disorder pregnancies (HDP) causing major morbidity and mortality for mother, fetus . Identifying reliable prognostic biomarkers is essential for risk stratification and timely intervention. Serum uric acid has emerged as potential marker associated with disease severity adverse perinatal outcomes in HDP.

OBJECTIVES:

To estimate maternal serum uric acid levels preeclamptic pregnancies , evaluate it correlation with fetal outcomes , severity in hypertensive disorders.

MATERIALS AND METHODS:

Prospective observation study conducted from July 2023 to December 2024 at RL Jalappa Hospital, Kolar. Total of 100 pregnant women aged 20–35 years with singleton pregnancies and gestational age between 20–40 weeks diagnosed with HDP were enrolled. Women with chronic hypertension, renal disease, and multiple gestation were excluded. Clinical parameters, biochemical tests including serum uric acid, and fetal outcomes were recorded. Statistics analysis performed using SPSS version 22 , significance set at $p<0.05$.

RESULTS:

Hyperuricemia was detected in 43% of participants and significantly associated severe hypertensive disorders like eclampsia ,HELLP syndrome, severe preeclampsia ($p<0.001$). Elevated serum uric acid correlated higher systolic , diastolic blood pressures and earlier gestational age at delivery. Adverse fetal outcome such low birt weight, preterm delivery,

IUGR, and fetal demise were significantly more frequent among hyperuricemic mothers (p=0.001). Diagnostic performance of serum uric acid showed sensitivity 95.01%, specificity of 86.72%, and AUC of 0.804.

Conclusion:

Serum uric acid strong predictor in disease severity , adverse fetal outcomes HDP. It holds potential as a cost-effective prognostic biomarker to guide clinical decision-making in the management of hypertensive pregnancies.

INTRODUCTION

Globally, hypertension disorders pregnancy (HDP) a significant source maternal , perinatal morbidity , affect around 2-10% pregnancies. Recent statistics indicate that pre-eclampsia constitutes around 3–8% of hypertensive disorders of pregnancy (HDP), whereas combined incidence of pre-eclampsia, eclampsia represents 10–15% global maternal mortality rates. Prevalence in preeclampsia health nulliparous women is 3-7%, while in multiparous women it is 1-3%.[1]

The main factor contributing to increased maternal , perinatal morbidity , mortality associated with HDP is absence of precise or targeted testing to determine a pregnant woman's risk of developing HDP. The maternal blood uricacid level biochemical test can used assess severity HDP , its effect on mother and fetus outcomes. In 1917, relationship between serum uricacid or the severity hypertension disorders of pregnancy was first reported. It remains a recognised indication of HDP severity, since higher maternal serum uricacid level correlate increased illness severity.[2]

Furthermore, was shown in HDP, increase uric acid levels precedes development of hypertension or proteinuria. Elevated blood uric acid levels women with hypertensive disorders of pregnancy (HDP) can be attributed to several factors, including diminished renal clearance, enhanced tissue catabolism, acidosis, heightened activity xanthine oxidase or dehydrogenas enzyme. Primary cause is diminished renal clearance rate of uric acid a lower GFR, coupled with high absorption , low secretion, resulting in elevated blood uric acid levels in women with hypertensive disorders of pregnancy . Serum uricacid level normally decrease by 3 mg/dl during 1st trimester a typical pregnancy. This decrease is caused by uricosuric effects of estrogen and increased renal blood flow. As a result, uricacid level increased during third trimester and reached 4-5mg/dl at term. Pregnant women predisposed to developing HDP exhibit marginally elevated blood uric acid levels during the first trimester. The rise in serum

uric acid levels corresponds illness severity with prognosis.[3,4]

This study aimed investigate function maternal blood uric acid predicting prognosis hypertensive disorders pregnancy (HDP) and impact on overall perinatal outcomes. Outcomes of proposed study will assist the future formulation of policies aimed at illness prevention, mitigation of severity, reduction of mortality, utilising current resources to a certain degree.

Objectives of the study:

1. To estimate Serum uricacid level in preeclamsia pregnancies
2. To correlate the serum uric acid levels with the foutes outcome

REVIEW OF LITERATURE

The four principal hypertensive diseases in pregnant women are:

- Preeclampsia (including associated conditions: ecllampsia ,HEL-LP [haemolysis, high lliver enzymes, low platelets] syndrome)
- Gestational hyprtension
- Chronic hypertnsion
- Preeclampsi occurring in conjunction with chronic hypertension

An accurate diagnosis, when feasible, aids in management decisions (e.g., timing delivery, necessity antiseizure prophylaxis) evaluating maternal prognosis (e.g., risks progression ,current pregnancy, recurrence in future pregnancies, long-term maternal health risk).

Alongside differentiating these four causes of hypertension, healthcare practitioners must also contemplate additional medical conditions (e.g., glomerulonephritis, thrombotic microangiopathies) exhibit clinical or laboratory characteristics similar to hypertensive diseases pregnancy.

Four primary hypertensive diseases pregnant individuals can differentiated by their diagnostic criteria, include unique features despite some overlaps.[5,6]

Comparison of major hypertensive disorders that occur pregnant women

	Normotension before pregnancy	Hypertension during pregnancy (%)	Proteinuria	Thrombocytopenia and/or increased transaminases
Preeclampsia	Yes	100	Usually present	Variable, depending on whether preeclampsia is at the severe end of the disease spectrum
HELLP	Yes	82 to 88	Usually present	100%
Gestational hypertension	Yes	100	No	No
Chronic hypertension	No	100	Variable	No
Preeclampsia superimposed on chronic hypertension	No	100	Usually present	Variable, depending on whether preeclampsia is at the severe end of the disease spectrum

Table no :1

Eleven percent of a cohort of more than 4million delivery hospitalization had a diagnosis related to hypertension, including 4.7percent with preeclampsia, 3.8percent with gestational hypertension, 1.7percent with chronic hypertension, and 0.6percent with unexplained hypertension [5]. One percent had eclampsia, fifteen percent had preeclampsia on top of chronic hypertension, 37percent had preeclampsia with severe symptoms and HELLP syndrome, and nearly 47percent had moderate and nonspecific preeclampsia out of 176,925 birth.

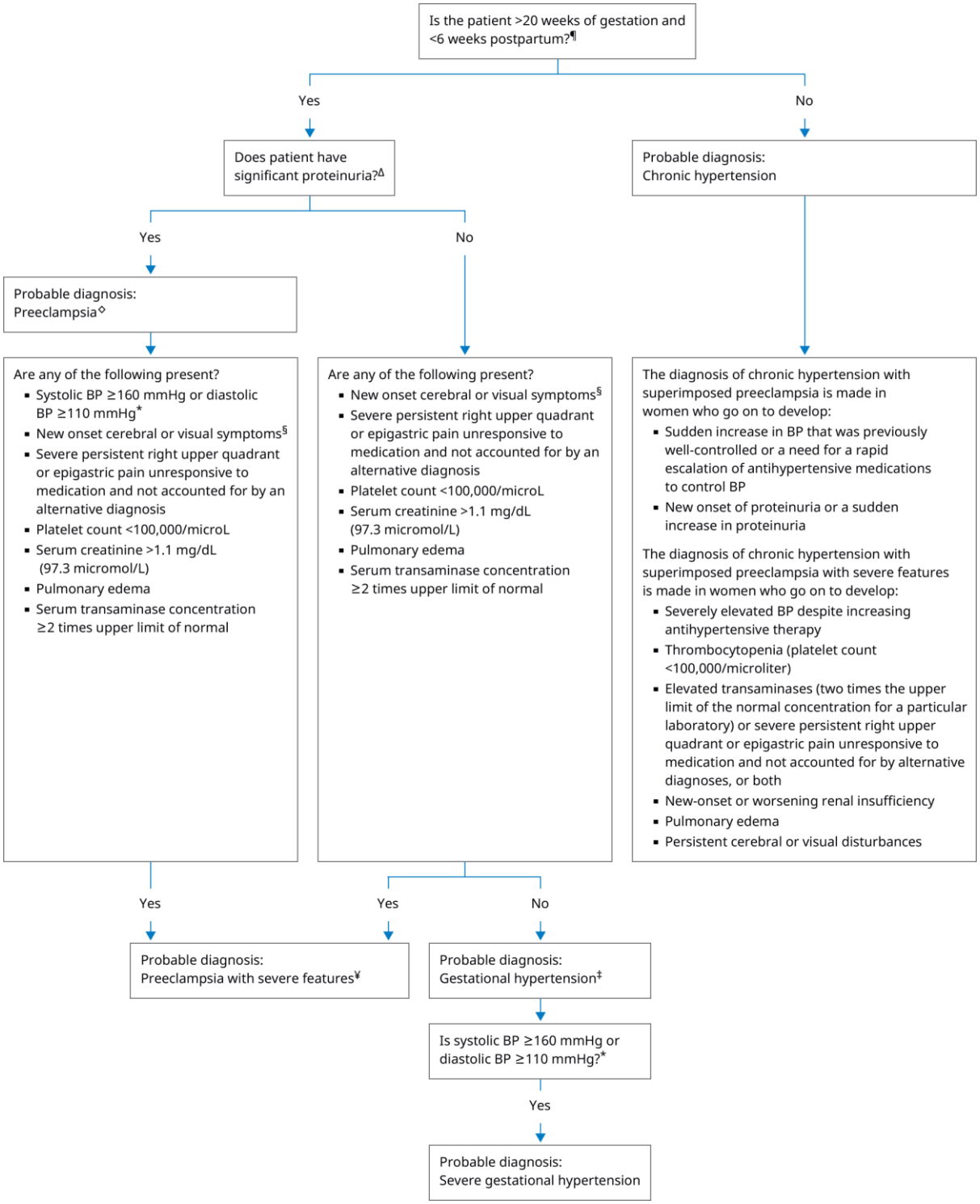


Fig1- Diagnostic evaluation a pregnant / postpartum woman with persistent diastolic blood pressure ≥90mm of Hg or systolic blood pressure ≥140mm of Hg

Decrease blood pressure during early pregnancy typical physiological phenomenon. Consequently, women with persistent hypertension may have normotension at their initial

prenatal appointments. In the later stages of pregnancy, if blood pressures reverts its prepregnancy baseline , the individual seem to exhibiting signs of preeclampsia and gestational hypertension in the absence of recorded prepregnancy blood pressure data.

DEFINITIONS/DIAGNOSTIC CRITERIA

The principal hypertension problems encountered in pregnant individuals are delineated here. Hypertension in pregnancy is characterised by a SBP of ≥140mmHg or a diastolic blood DBP of ≥90mm of Hg. Severe Preclampsia is characterised by a SBP of ≥160mm of Hg or a DBP of ≥110mmHg. American College of Cardiology and the AH Association have approved reduced threshold for diagnosis hypertension in nonpregnant individuals (SBP ≥130mmHg or DBP ≥80mmHg). Some proposed concept may also applicable to pregnant patient. The use lower criterion not been well researched, would elevates prevalence of hypertension pregnancy around 10 %, would lead to possibly superfluous testing, hospitalisation, and intervention without demonstrated benefits. A meta-analysis indicated reducing blood pressure threshold aberrant readings beyond 20 weeks will not aid doctors in recognising individuals at increased maternal or neonatal risk.[7,8]

- Preeclampsia denotes the emergence in hypertension, proteinuria development of hypertension accompanied by substantial end-organs dysfunctions, with or without proteinuria, previously normal bloodpressure individual, generally occurring after 20 weeks of gestation or postpartum. The majority of patients exhibit proteinuria; nevertheless, crucial note that a diagnosis can established in pregnant patients with hypertension in absence proteinuria with new-onset hypertension associated certain symptoms indicative substantial end-organ dysfunctions. [9,10]

Multiple subtypes preeclampsia may present, each associated with distinct pathophysiological mechanisms contribute maternal, foetal death and morbidity. The predominant categories are early onset (less than 34weeksgestational age) and late onset (34weeksgestation or more).

Clinical characteristics coincide; however, illness range , results vary. Early-onset illness is linked to more severe placental and maternal/fetal clinical manifestations, resulting worse mother/ fetus outcomes. Consequently, it been postulated two traits possess distinct origins , pathophysiology. Additional potential subgroups are "HELLP syndrome," "gestational hypertension," "preeclampsia with or without foetal growth restriction." Nonetheless, disparities may also elucidated by biological variance in illness prognosis.[11,12]

Preeclampsia severe characteristics, previously known as severe preeclampsia, refers to individuals exhibiting extreme hypertension or certain signs and symptoms indicative substantial end-organs dysfunctions, representing severe end preeclampsia continuum.

Preeclampsia with superimposed over chronic hypertension identified preeclampsia manifests patient chronic hypertension, defined as hypertension existing before to pregnancy, recorded atleast two occasions less than 20th week of gestational age, and persisting beyond 12 weeks after delivery . It defined by exacerbation , refractory nature of hypertension (particularly in acute cases), the emergence proteinuria ,rapid escalation proteinuria, notable new end-organs dysfunctions occurring more than 20 weeks gestational age or after delivery in patient with pre-existing chronic hypertension.

HEL-LP syndrome (Haemolysis, raised Lliver enzymes, less Platelets) is characterised as severe variant preeclampsia, distinguished by the predominance of haemolysis, raised liver enzymes, and thrombocytopenia. Hypertension, CNS impairment, or renal failure may also be evident. Majority patient, though not, exhibit hypertension (82 to 88 %, with instances showing a first mild rise in blood pressure) or proteinuria (86 to 100 %). In rare cases, patient may exhibit neither condition; other diagnosis linked to comparable lab parameters anomalies must ruled out before to confirming a diagnosis of HELLP in these unusual patients.[13]

Eclampsia denotes the manifestation tonic-clonic seizure preeclamptic patient, excluding other neurological disorders that may explain the seizure.

GHT denotes increases blood pressure occurring more than 20weeks gestational age individual a history of normal blood pressure, characterised by the absent proteinuria and other indicators preeclampsia-relates end-organs dysfunction. As much as 50% these patient eventually exhibit signs and symptoms of preeclampsia. The emergence proteinuria elevates diagnosis preeclampsia. Patients exhibit severe hypertension , other characteristics severe illness handled similarly to those with preeclampsia with severe symptoms, even in the absence of proteinuria. Genuine gestational hypertension often resolves within 12 weeks following childbirth.

"International Society for the Study of Hypertension in Pregnancy"

The categorisation system hypertensive diseases pregnancy established by International Society Study of Hypertension Pregnancy (ISSHP):

●**Pre pregnancy or < 20weeks of gestational age**

•**White-coat hypertension**: SBP ≥140mmHg and/or DBP ≥90mmHg in clinical settings, while exhibiting blood pressure <135/85mmHg in home , 24-hours ambulatory BP monitoring.

•**Masked hypertension**: BP is <140/90mmHg during a clinic or office visits, but ≥ 135/85mm Hg at other times outside the clinic/office.

•**Chronic hypertension**: Hypertension identified prior to pregnancy or before 20weeks of gestational age . Chronic hypertnsion can classified as essential hypertnsion (i.e.,without a identifiable second cause) secondary hypertnsion (i.e., with recognised secondary disease, such as renal illness).

●**Pregnancy ≥ 20 weeks of gestational age**

•**GESTATIONAL HYPERTENSION**: De nvo hypertnsion occurring at or more than 20 weeks of gestational age , absent proteinuria any indicators preclampsia.

• **PREECLAMPSIA**: gestationalhypertnsion accompanied by one , more of the following:

Protein positive in urine

-central nervous system problems (e.g., eclampsia, altered sensorium, blind, stroke, clonus, severe headache, chronic visual distrubances)

Pulomonary edema

-Haematologica problem (e.g., platelets counts <150,000/microLit, DIC , haemolysis)

renal damage (creatinine levels ≥90 mmol/L or 1 mg/dL)

-Liver damge (increase transaminase SGOT or SGPT over 40 IU/L) witth , witthout upper right quadrants , epigstric tenderness.

Utero placental dysfunctions (e.g., placentall abruption, angiogeni imbalnce, foetal developments limitation, aberrant umbilical artery Doppler waveform analysis, or foetus demise)

Uric Acid

Uricacid byproduct purine catabolism facilitated enzyem xanthine dehydrogenas or xanthine oxidases (XDH/XO). XDH transformed into oxidase form, XO, several triggers, including ischaemia. The metabolims of purines xanthine oxidase links uricacid productio generation free radical superoxide (O2-), associated with oxidative stress. XDH/XO present several tissue, with a notable concentration liver , gastrointestinal tract. Circulating population XO recently found significantly rises after ischaemic tissue injury. Elevated circulating uric acid is associated with similar insults. Circulating XO is believed to attach to the endothelium, resulting in localised oxidative damage. [14,15]

Most animals posess enzymeuricase, which degrades uricacid to allantoinn, non-toxic byproduct secreted by the kidneys. Humans and big apes lack uricase, hence uric acid elimination mostly depends on renal excretion. Uric acid has poor solubility, content kept relatively less healthy persons (below 6.0 mg/dl). Nonetheless, minimal amounts uric acid have biologicla action. Uric acid plasma antioxidant can neutralise superoxide , hydroxl radicals, singlet oxygen. Diminishes the nitrosylation tyrosine residues proteins peroxynitrite preserves superoxide dismutase function, demonstrating advantageous benefits soem contexts. Conversely, uric acid act as pro- oxidant (urate radicla) when antioxidant availability is

restricted, especially with diminished ascorbate levels. Uric acid serves as an inflammatory mediator, promoting the synthesis monocytes chemoattractant protein-1,IL-1β,IL-6,and TNF-α. [16,17]

Uric Acid in Pregnancy

Uricacids levels affected dietary factors (such excessive protein , fructose intake), alcohol use, elevated cell turn over, enzymatics deficiencies purines metabolis, or impaired renal functions. Oestrogen has uricosurics properties, and uricacid level elevated males , post-menopause womens. During pregnant, uric acid levels initial decrease by 25-35% because influence oestrogen, increased blood volume, enhanced GFR. Nevertheless, concentration gradually increase in levels seen in non-pregnant women end of term (4-6 mg/dL). [18]

HYPERURICEMIA AND PREECLAMPSIA

In the late 1800s, increased uric acid levels were initially observed in women with preeclampsia. Since then, multiple study has established correlation with uric acid level illness severity. Therapeutic value hyperuricemia in preeclampsia therapy remains contentious. We recently investigated correlation between elevated uric acid level pregnant womens with hypertension outcomes preterms delivery (mostly recommended preterm delivery treatment preeclamptic) growth limitations. Hyperuricmia occurred 16percent of women pregnant hypertnsion withut proteinura in 75% womens clinical confirmed preeclampsia. Pregnanc hypertensio accompanied by hyperuricemia was linked to an increased incidence of these detrimental foetal outcomes. Hypertension women elevated urc acid level has higher incidence of preterm delivery and growth limitations , even abence protein in urine.[19]

Women has subsequent develops preeclampsia, uricacid level are increased before 10 weeks gestational age, significant preceding clinical manifestation condition. Elevated uricacid frequently precede symptoms condition, includes decreased GFR. Historically, hyperuricemia ascribed diminished kidney clearance. The kidney filters, reabsorbs, and secretes uric acid.

Hypovolemia, a first alteration in preeclampsi, rises uric acid reabsorptio, potentially raising blood uricacid level. Nevertheless, elevated uricacid level occur prior to the reduce plasma volume. Elevate uricacid syntheis in maternal, foetus or placenta tissue due to enhanced tissue degradation (i.e., higher substrat availbility) augmented xanthine oxidase activiy may account for elevated levels. The precise cues that elevate XO activity preeclampsia women remain un clear. [20]

URIC ACID AS A PATHOGENIC VASCULAR ELEMENT

The studies supporting the harmful significance uricacid accumulating. Non -pregnant demographic , hyperuricemias serves as independen predictr CVS, kidney disease general population and chronic hypertension population. Uricacid serves as an indicator of negative cardiovascular events in individuals with pre-existing cardiovascular disease.

Experimental findings further substantiate the harmful effect of uricacid. Rats made hyperuricemic with the injection of oxoni acids, inhibitor of uricase, exhibit crystal kidney damage and endothelial illness, alongside glomerular and systemic hypertension. Pharmacological inhibition of uricacid increases allopurinol, axanthine oxidase inibitor, averted alterations. [21]

"Potential Effects of Uric Acid on Placental Vascular Development, Structure and Function"

URIC ACID AND PLACENTAL DEVELOPMENT

Placenta tropho-blast alter phentype throughout gestation, transitioning from a high proliferative high invasive cell subtypes. Facilitates sufficient placenta growth , the invasion maternal deciduas , spiral arteries. No research have been identified to far that investigate the effects uric acid tropho-blast cell. Discourse on the influence uricacid placenta growth development conjectural, relying on phenotypic functional resemblances in endothelium cells and trophoblast cells. Uric acid significantly impacts endothelial cell proliferation & migratin,

reducing serum induced proliferation HUVEC cells 50% limiting HUVEC migrtion by up to 75percent . The observed effects stem directly from uricacid absorption endotheial cell, evidenced by the attenuation of these effects following treatment probenecid, organic anion transport inhibitor. [22,23]

Invasive extravillous cytotrophblast cell penetrate maternl spial arteries , resulting in vasculr remodelling. Leads to larger diameter, flacid vesels lacking responsive smoth musscle vessels walls. Preeclampsia, meticulously controlled remodelling proces is insufficient, leading to impaired oxygen and nutrition supply to the placenta. The generation of nitricoxide (NO) promotes trophoblasts migrations and invasions in both in cell line , animal model. Furthermore, eNOS located invades cytotrophobast cell. Uricacid diminishes nitric oxide generation in endothelial cells, and a comparable action in trophoblasts might alter migratory , invasiveness characteristics trophobast cell. Given that uricacid levels rise womens predisposed preeclampsia before ten week of gestation, conceivable uricacid may have a role in insufficient trophoblast invasion and remodelling of spiral arterioles. Additionally, localised heightened uric acid synthesis may occur, since increases in xanthine oxidase enzyme levels activity found the invasiveness of cytotrophoblast cells of women preeclampsia. [24]

URIC ACID AND THE STRUCTURE AND FUNCTION OF PLACENTAL VASCULATURE

The normal vasculature architecture transports functions of the placenta essential for delivering necessary O_2 , nutrients of growing foetus. Uricacid can harm adults vasculatures analogous effect the placenta women with preeclampsia. Uricacid penetrates smoothmuscle cell via organics anions transporter, subsequently activating intracelluler mitogens activated proteins kinase and nu-clear transcriptions factor (e.g., NFk-B). In cell lines, uricacid induces synthesis platelet derived growth factors, vasoconstrictor thromboxanes and angiotensins II, inflammatory marker including C-reactive protein. Consequently, uricacid treatments smooth

muscles cell induces proliferatively , pro-inflammatory phenotypes. Notably, certain effects uricacid on smoothmuscles cell diminished presences antioxidant, indicating pathogenic function for the urate radicals. [25,26]

Placenta vasculatures is devoid of autonomicinnervation and depends only on locally generated or circulating chemicals for haemodynamic regulation. The principal vasoactive substance that sustains optimal placenta perfusions endothelial derived nitricoxide. Hyperuricemic rat, uricacid diminishes eNOS activities, hence restricting Nitric oxide availabile, upregulates COX-2 expressions, resulting in enhanced production of the powerful vasoconstrictor thromboxane. The same vasoconstrictive impact uricacid the placenta women preclampsia may jeopardise placenta perfusions may hinder foetal development. [27]

URIC ACID AND PLACENTAL REDOX BALANCE

Elevated placenta oxidativ stres wel -established characteristic preeclmpsia. Oxdative imbalnce arises elevated pro oxidant production combined inadequate antoxidant capability. Typical conditions, uricacid neutralises oxidising chemicals implicated in the placental diseases associated with preeclampsia. The antioxidant action of uric acid leads to its conversion to fre radicals, namely urates radicals. Typical conditions, urates rapidly restored antioxidants form throughs activities ascorbates. Conditions diminished ascorbates availabilty, such preeclampsia womens, that urate radicals endures may oxidativey alter placenta protins , lipds. [28,29]

POTENTIAL EFFECTS OF URIC ACID ON MATERNAL VASCULATURE

Uric Acid in Maternal Hypertension

Possible pathogenics effect uricacid on placenta vasculer bed likewise pertinent maternals vasculature. Preeclampia women increased levels of circulate vasopressor, including thromboxne endotelin, alongside corresponding reductions vasodilator such as prostacyclins.

Maternal vasculatures womens with preeclampsia demonstrates heightened susceptibility to pressor drugs. This is partly attributed to diminished nitric oxide availability resulting from endothelial dysfunction. As previously mentioned, increased uric acid levels may contribute to diminished nitric oxide generation, perhaps elucidating the modified endothelium role in vascular tone among women with preeclampsia. Reducing uricacid levels with allopuerinol enhanced endothelial dependent vasodilation individuals with diabetes and heart's failure. Lowering uricacid level may serve viable treatment approach women with preeclampstic.[30]

Uric Acid causing Inflammation

The preeclampsia condition characterised by increased inflammation leads to endothelial impairment vasculer damage. Uricacid powerful modulator inflammations. Uricacid elevate levels monocytes chemoattractants proteins-1 (MCP-1) mRNA , proteins in vasculer smooth muscles cell dose-dependent manner. Uricacid prompts humans monocyte generate the proinflammatory cytokine IL-1β,IL-6,andTNF-α, raised blood both experimental produced hyperuricemia in animals and preeclamptic women. In women with preeclampsia, elevated levels circulates TNF-α were positive linked with circulation uricacid concentration. [31,32]

Uric Acid causing Endothelial Repair

Significant increases circulation uricacid occur after acute ischemia reperfusion episode. The rapid increase uricacid level animal models stimulates mobilisation endothelial progenitor cells (EPCs). Uricacid believed function signals endothelials injury, facilitating the repair of damaged arteries via the mobilisation of endothelial progenitor cells (EPCs). The protective function uricacid limited acute elevation circulation uricacid level. Enhanced mobilisation of EPCs is not seen in rats that have been made chronical hyperuricemia after induce kidney ischemia -reperfusions damage. Consequently, persistently elevated uric acid levels are linked to decreased release of endothelial progenitor cells (EPCs). The progenitors cells diminished preeclamptic. This may come from heightened use the cell injured vasculars bed , as shown

by aforementioned studies, diminished mobilisation of cells.

Uric Acid and Maternal Renal Dysfunction

Kidney impairment prevalent observation womens with preeclampsia. kidney anatomical alterations including juxtaglomerulars hyperplasia , macula dens atrophy, affernt arteriopathy, glomeruler hypertrophies, and glomeruloendothe liosis. Slightly hyperuricemia rats models exhibits notably analogous kidney alterations, includes afferent arteriolopathies , mild tubulointerstitial fibroses, glomerular hypertrophies, ultimately glomerulosclrosis, accompanied by future albuminura , proteinuria. Uricacid induced diseases not contingent upon uricacid crystals formation mitigated reducing uricacid levels. Notably, two decades , Nochy indicated kidney abnormalities seen preeclampsia women only present those with hyperuricemia. [33]

FEED-FORWARD CYCLE OF URIC ACID PRODUCTION IN PREECLAMPSIA

What initiates hyperuricemia in preeclampsia, elevation of uricacid precedes decline in GFR , hypovolemiastate. Therefore, is improbable characteristics of preeclamptic , which significantly diminish uricacid excretion, constitute the underlying cause. Women predisposed to preeclampsia may enter pregnancy with elevated uric acid levels due to metabolic syndrome, or uric acid synthesis may rise during early pregnancy. Multiple credible sources contribute to elevated uricacid levels womens preeclampsia, includes foetus, placental , and maternal organ and vasculatures.

Uricacid may hinder placenta bed vasculature remodelling obstructing trophblast invsion, leading to diminished placenta perfuson and predisposing placenta to ischemia-reperfusion damage oxidatives stres. Maternals tissue suffer ischaemic harm due to vasospasm resulting from endothelial dysfunction, potentially linked to elevated uric acid levels. Ischaemic damage oxidativ stres facilitate self-perpetuating cycle of uricacid synthesis. Event of tissue damage, purines are released, and under hypoxic conditions, ATP degraded into both adenines and

xanthines (substrates). Moreover, hypoxia powerful inducers holoenzymes xanthine oxidases/dehydrogenase selectively elevates oxidases the enzymes [4]. Simultaneous elevation of both substrates, enzymes concentration will enhance uricacid synthesis. Additionally, vasospasm and fluid loss resulting from endothelial dysfunction can promote kidney reabsorptions uricacid. Consequently, hyperuricemic results in increased uricacid synthesis decreased uricacid excretions feed-forwards loops.[34]

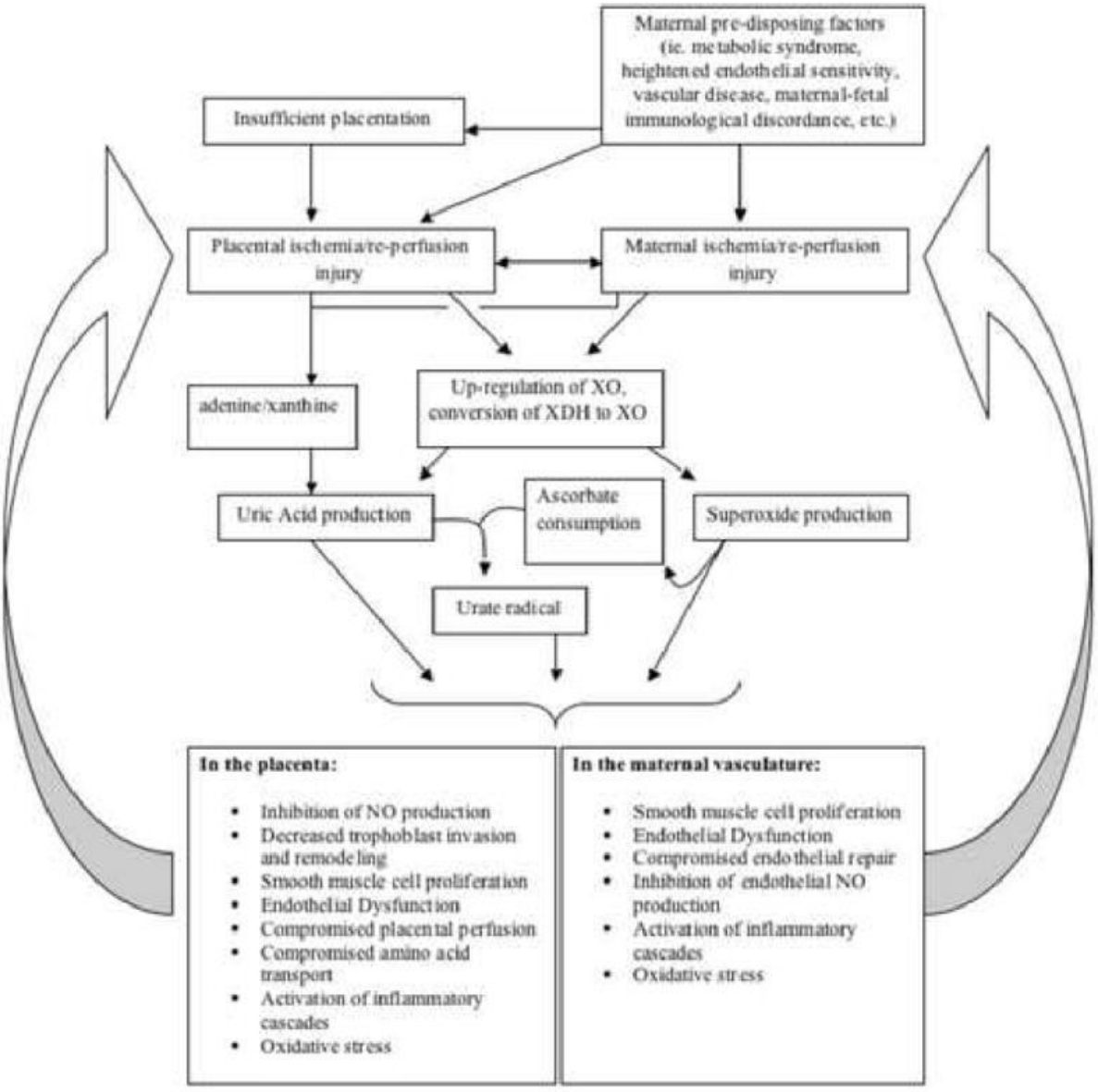


Figure 2.

Feedforward process uricacid synthesis its putative harmful effects in the setting of

preeclampsia.

Literature Review

Kumar , Singh (2019) evaluated correlation between mother blood uricacid levels the severity in hypertensive problems during pregnancy rural tertiary hospital. This study was done the OBG department of a rural tertiary centre in North India for a duration of 7 months (October 2016-May 2017) with 110 mothers hospitalised for hypertension disorders pregnancies (Gestational hypertension, preclampsia, ecllampsia) more than 34 week of gestational age . maternal serum uricacid level wer analysed across 3 group for illness severity, delivery method, and mother outcomes. There was association identified between mother blood uricacid level, illness severity, and mother outcomes.[35]

Kumar and Singh (2019) compared blood uricacid with calciium level between normal blood pressure and hypertension in pregnants evaluate mother and fetal outcomes the 2 group. A prospectiv case control research done following ethica permission at the OBG department of a rural tertiary facility in North India, spanning 7 months. A total of 220 pregnant women at or beyond 34 week gestational age were evaluated, comprising 110 patients with hypertension disorders of pregnancy and 110 control with normal BP, to compare morther uricacid , Ca level, as well as mother and fetal outcomes. Maternal hyperuricemia and hypocalcaemia were linked to worse maternal and fetal outcomes mother with hypertension disorders in pregnanc compare health normotensive women. Kumar [36]

Adu-Bonsaffoh et al., 2024 investigated the correlation between mother blood uricacid levels negative pregnanc outcome preclampsia. Cross-section research was undertaken including womens diagnos preclampsia KorleBu teaching hospital (kbth), tertiay facility in GHANA. Descrptive analyse wer conducted, a multvariable logstic rgression mdel employed investigate relationship betwen mother blood uricacid level pregnancies outcome utilising R software.

Hyper-uricemia preclampsia substantially correlated unfavourable mother outcomes (LSCS and severehypertension) newborn outcome (LBW). Hyperuricmia appears to be clinically significant in forecasting pregnancy outcomes, particularly in cases of preterm pre-eclampsia. Additional longitudinal research is advised to investigate the clinica implication of mother uricacid level , maternal outcomes in preclampsia. [37]

Jummaat et al., 2021 sought to ascertain the correlation between hyperuicaemia preclampsia patient and maternal and perinatal outcome. Prospective cohrt research encompassed 79 patient at tertiay centres between 2016 - 2018. Blood samples collected antenatal , six weeks postpartum to assess renal function, including serum uric acid levels. The data reveal a greater prevalence of adverse mother and perinatal outcome in hyperuricemia groups compared to the normal uricacid groups. Serum uricacid strong predicto of LBW and early deliver women with preeclamptic. A substantial negative connection was identified betwee uricacid levels prenatal creatinin clearanc ($rs = -0.338$, $p = .002$). the evaluation blood uricacid levels appears crucial for improving outcomes in individuals with preeclampsia. [38]

Koopmans et al., 2009 assess the precision clinica significance blood uricacid forecasting mother problems patients with preclampsia. Existing metaanalysis topic has been revised. The precision of blood uric acid in predicting maternal problems was evaluated using a bivariat mode to estimate a summar Receivr Operatig Characteristi (sROC) crve. a clinica decision analysi subsequently conducted, modelling 3 potential strategie: (I) Expectant management monitor till spontanous labur; (II) Induction laour; (III) blood uricacid a predictor for mother problems. Latter technique utilised accurcy dat serum uricacid obtained sROC curve evaluate significance serum uricacid therapy womenS with preclampsia. technique involved inducing labour womens elevated serum uricacid level, while level below threshold handled expectant. Choice to implement Expectant managements, induced labours, assess serum uricacid level determined by anticipated value approach. anticipated utili is contingent upon likelihood

severe maternal problems (such as sevre hypertnsion, hamolysis, high liver enzyme, low platelets count associated with HEL-LP syndromes or ec-lampsia) mode of delivery (LSCS or vaginal birth). The outcome were evaluated using a distres ratios, quantifies the relative severity of a pre-eclampsia problem in comparison to a caesaren delivery. 8 study including 1,565 womens with preclampsia satisfied inclusio crteria. Distress ratio 10, serum uric acid method favoured when complication likelihood ranged from 2.9% to 6.3%. Induction of labour is favoured at elevated complication rates, whereas expectant management is the optimal approach at reduced complication rates. The findings were consistent in sensitivity studies employing various distress ratios. According to decisions analysi, serum uricacid appears an effective test therapy preclampsia unde practical assumption. [39]

Luo et al., 2024 sought to assess the correlation between serum uricacid (SUA) level and hypertensives disorders pregnancy (HDP) 288 women of advaced maternl age, namely those over 35 year. MATERIALS AND Methods. Total of 780 pregnants womens advance mother age participated study, with 288 diagnosis hypertension disorder pregnancies (includes gestationl hyperension and preclampsia) 492 exhibiting normotensive , utilising a 1:2 (84:168) propensiy scoe matchng methodology. The incidence of SUA (collected before to 20 weeks gestational) HDP women of advance mother age was evaluated using multivaiate logitic modelling three proensity score based methodologies. FINDINGS The medien aeg of patients 37 year. likelihood getting hypertension in pregnancy escalates elevated Serum uricacid levels (30.19% versus 13.65%, P<0.001). The propensity score matched sample, odds ratio (OR) hypertensive disorders of pregnancy (HDP) associated with elevated uric acid levels, after controlling for confounding variables, was 2.88 (95% CI: 1.44-5.75, P=0.0027). High uric acid levels are significantly correlated with the incidence of hypertensive disorders of pregnancy (HDP) unadjusted populaion (Odds ratio=3.43, 95% ConfidenceInterval: 2.01-4.66, P<0.0001) adjusted cohort (Odds Ratio=3.62, 95% Confidenceinterval: 2.81-4.66, P<0.0001).

After controlling for clinical covariates, 1-SD rise SUA associated with 135% heightened risk HDP (OR=2.35; 95% CI: 1.57-3.50; P<0.0001) according to the fuly adjustd odel. The sensitivity analysis yielded analogous findings. CONCLUSIONS A notable correlation existed SUA and HDP in womens advanced matenal ae, underscoring necessity for erly identification SUA in pregnant individuals. [40]

MATERIALS AND METHODS

SOURCE OF DATA: The study will be conducted on women who are pregnant with hypertension disorder at department OBG, RL Jalappa Hospital, Kolar during the study period.

Study design: Prospective study

Study period: July 2023-December 2024

METHOD OF COLLECTION OF DATA: Cohort study is conduct on patients coming to department of obstetrics and gynaecology OPD at R L JALAPPA HOSPITAL TAMAKA, KOLAR attached to sri devraj urs medical college under sri devraj urs academy of higher education and research.

Relevant information like age, parity, previous history of preterm pregnancy would be recorded.

INCLUSION CRITERIA:

Study subjects were proposed at the start of study to be

Age between 20 to 35 years

gestational age between 20 weeks – 40 weeks.

Hypertension pregnancy disoders in pregnancy

Singleton pregnancy

EXCLUSION CRITERIA:

Pregnant women risk factors such as

Chronnic hypertenson

Kidney disease

Multiple pregnancy

Sample size: 100

Sample size(n)

Sample size estimate use of the sensitivity of Uricacid in predicating preeclampsia was 90.9%from the study by Ana I. Corominas et al. using the formula [8]

$n=Z_{\alpha 2)}{}^2P\hat{}(1-P\hat{})/d^2$

Where P^ is predetermined value sensitivity (or specificity) that is ascertained by previous published data or clinician experience/judgment and for $\alpha = 0.05$, Zα2 is inserted by 1.96.

P^ = 90.9% or 0.909

d = 6% or 0.06.

Using the above values at 95% CI level a sample size of 89 subjects will be included in the study.

Considering 10% Nonresponse rate sample size 89+ 8.9 = 98 subjects minimum should included study.

Methodology:

This research will take place at the Department of Obstetrics and Gynaecology at RL Jalappa Hospital Tamaka Kolar, affiliated with Sri Devraj Urs Medical College under the auspices of Sri Devraj Urs Academy of Higher Education and Research.

Written informed permission was obtained from study participants following the verification of inclusion and exclusion criteria.

A patient information leaflet was provided to the trial subjects in a comprehensible language.

Subjects with pre-eclampsia are recruited for the research. A comprehensive clinical history of the research participants, in conjunction with prenatal assessment, will be conducted.

The blood pressure of each research subject will be documented.

Subjects with an initial elevated blood pressure reading will have a further measurement after a four-hour period, and this reading will be recorded.

The urine will be analysed for proteinuria.

A complete blood count will be conducted for all trial participants, including assessments of white blood cell count, platelet count, and uric acid levels.

All patients must provide fully informed permission before samples are obtained. Patient involvement in the study will terminate with the acquisition of the final reports.

Comprehensive investigations, including complete blood count, regular urinalysis, liver function tests, renal function tests, uric acid measurement, and coagulation profile, will be conducted.

The data will be compiled in an Excel spreadsheet and evaluated using descriptive statistics.

Does the study require any investigations or interventions to be conducted on patients or other humans or animals?

Yes. blood samples taken from study population complete blood count, liver function test,

coagulation profile, renal function test, uric acid and will be estimated.

Has ethical clearance been obtained from your institution?

Yes

Statistical analysis: SPSS 22 software will be used to analyze the data after it has been entered into a Microsoft Excel data sheet. Frequencies and proportions will be used to represent categorical data. The significance test for qualitative data will be the chi-square test. The mean and standard deviation will be used to represent continuous data. To determine the mean difference between two quantitative variables, the independent t test will be employed as the test of significance.

Graphical representation of data:

MS Excel and MS word will be used to obtain various types of graphs such as bar diagram, Pie diagram.

- Sensitivity = a/(a+c) x 100 = Truepositive / Truepositive + Falsenegative
- Specificity = d/(b+d) x 100 = TrueNegative / Truenegative + Falsepostive
- Positivepredictive value = a/ (a+b) x 100 = Truepostive / Truepositive + Falsepostive
- Negativepredictive value = d/ (c+d) x 100 = Truenegative / TrueNegative + FalseNegative
- Diagnosticaccuracy = a + d / a + b + c + d = Truepostive + TrueNegative / Total

P value (Probability that the result is true) of <0.05 will be considered as statistically significant after assuming all the rules of statistical tests.

Statistical software: MS Excel**,** SPSS version 22 **(**IBM SPSS Statistics, Somers NY, USA)

will be used to analyze data.

The categorical variables will be presented as frequencies and percentages and Chi square test will be used as test of significance. The Quantitative variables will be presented as measures of central tendency and student T test or Mann Whitney U test will be used as test of significance. A p value of less than 0.05 will be considered as statistically significant

RESULTS

Table 2: Age Groups

Age Groups	Frequency	Percent
18 – 25years	62	62.00
26 – 30years	24	24.00
31 – 35years	12	12.00
36 – 40years	2	2.00
Total	100	100.00

Table 1 represents the data of participants across different age groups. The maximum number of participant were within the 18-25 age range, comprising 62% of the sample. The 26-30 age group includes 24% of participants, while the 31-35 age group represents 12%. Only 2% of participants are in the 36-40 age group.

Fig:3 **Age Groups**

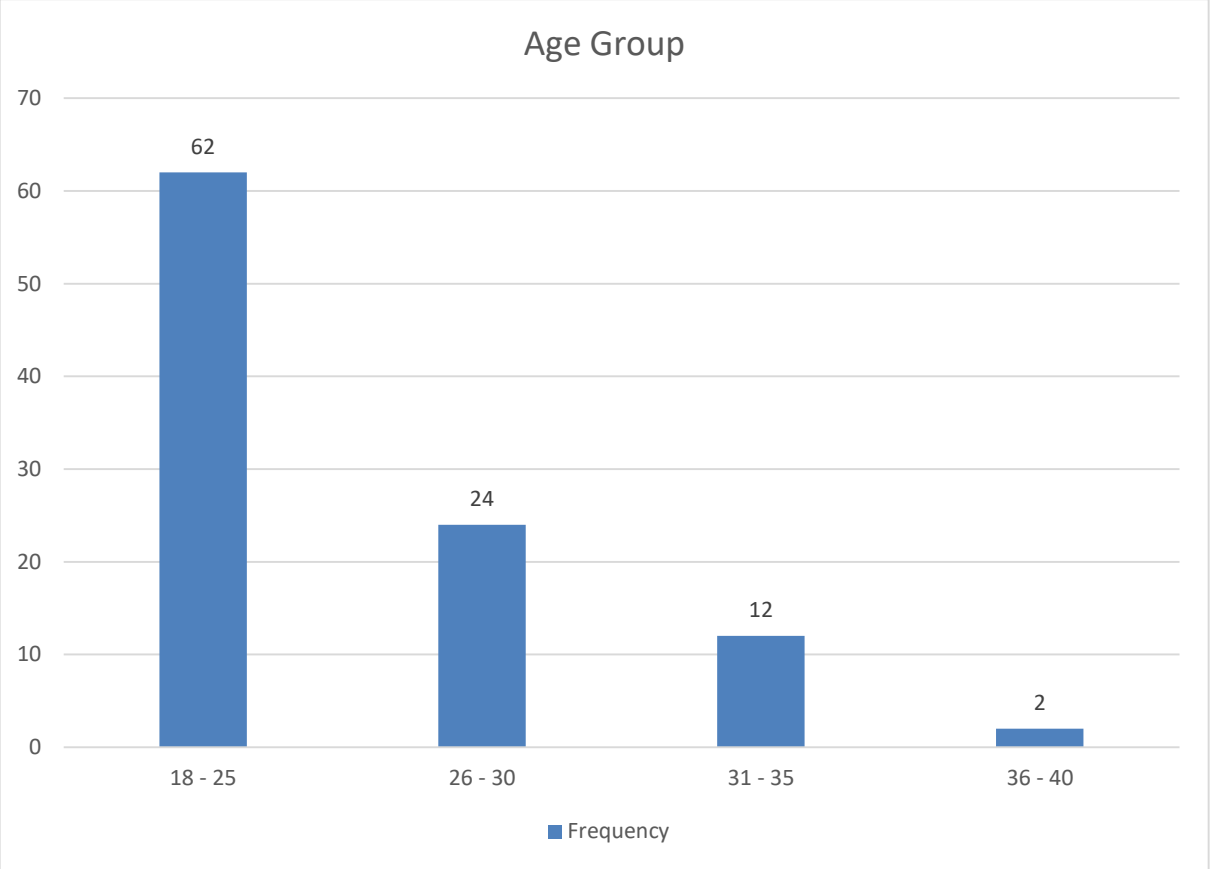


Table 3: Obstetric History

Obstetric History	Frequency	Percent
Primigravida	48	48.00
Multigravida	52	52.00
Total	100	100.00

Table 3 shows the distribution of participants based on their obstetric history. Of the total sample, 48% are primigravida (first-time pregnancies), while 52% are multigravida (having had previous pregnancies).

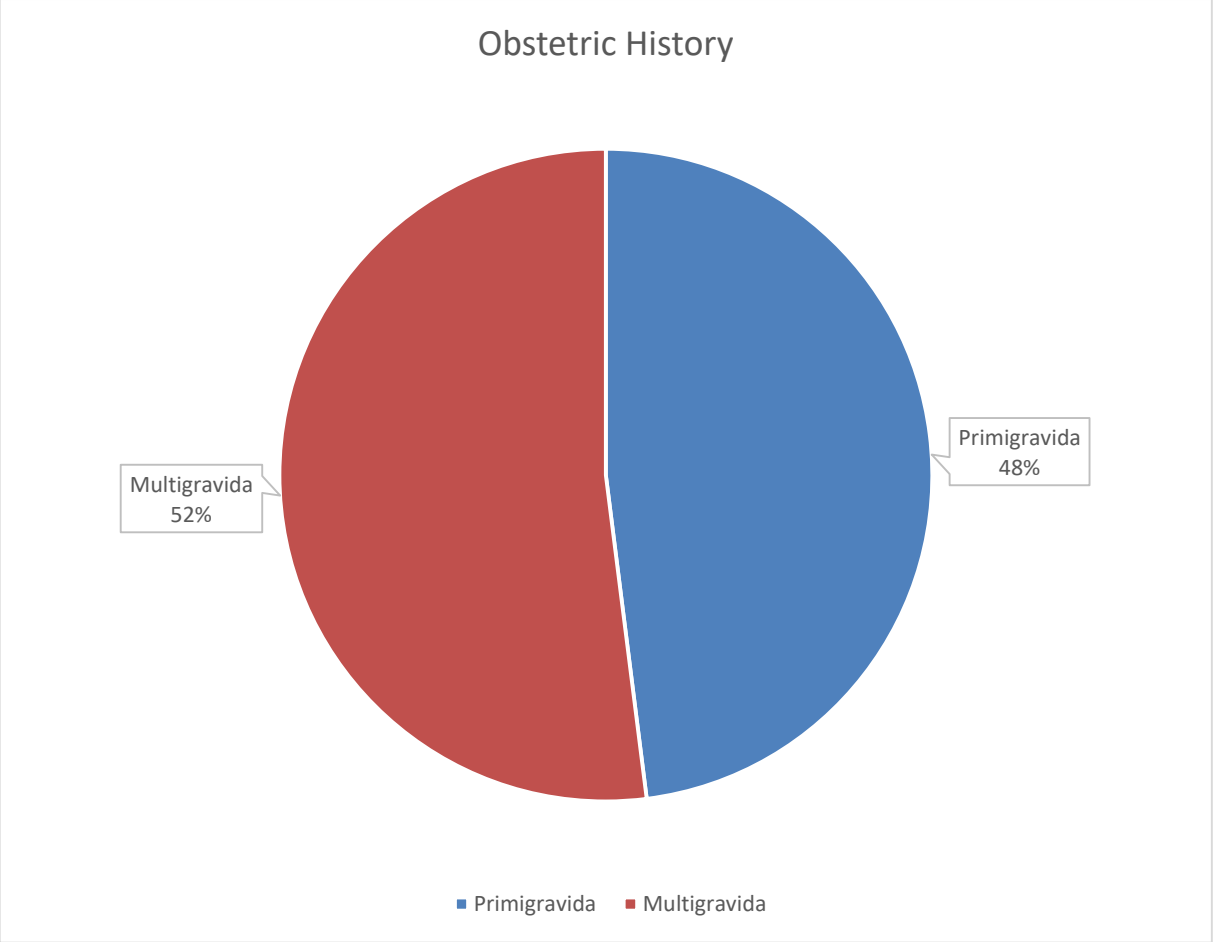


Fig:4 obstretic history

Table 4: Gestational age

Gestational age	Frequency	Percent
< 34 weeks	25	25.00
34 - 37 weeks	44	44.00
> 37 weeks	31	31.00
Total	100	100.00

Table 4 presents the distribution of participants based on their gestational age. It shows that 25% of the participants were born before 34 weeks, 44% were born between 34 and 37 weeks, and 31% were born after 37 weeks. The total number of participants is 100, with the distribution reflecting a higher proportion of births between 34 and 37 weeks.

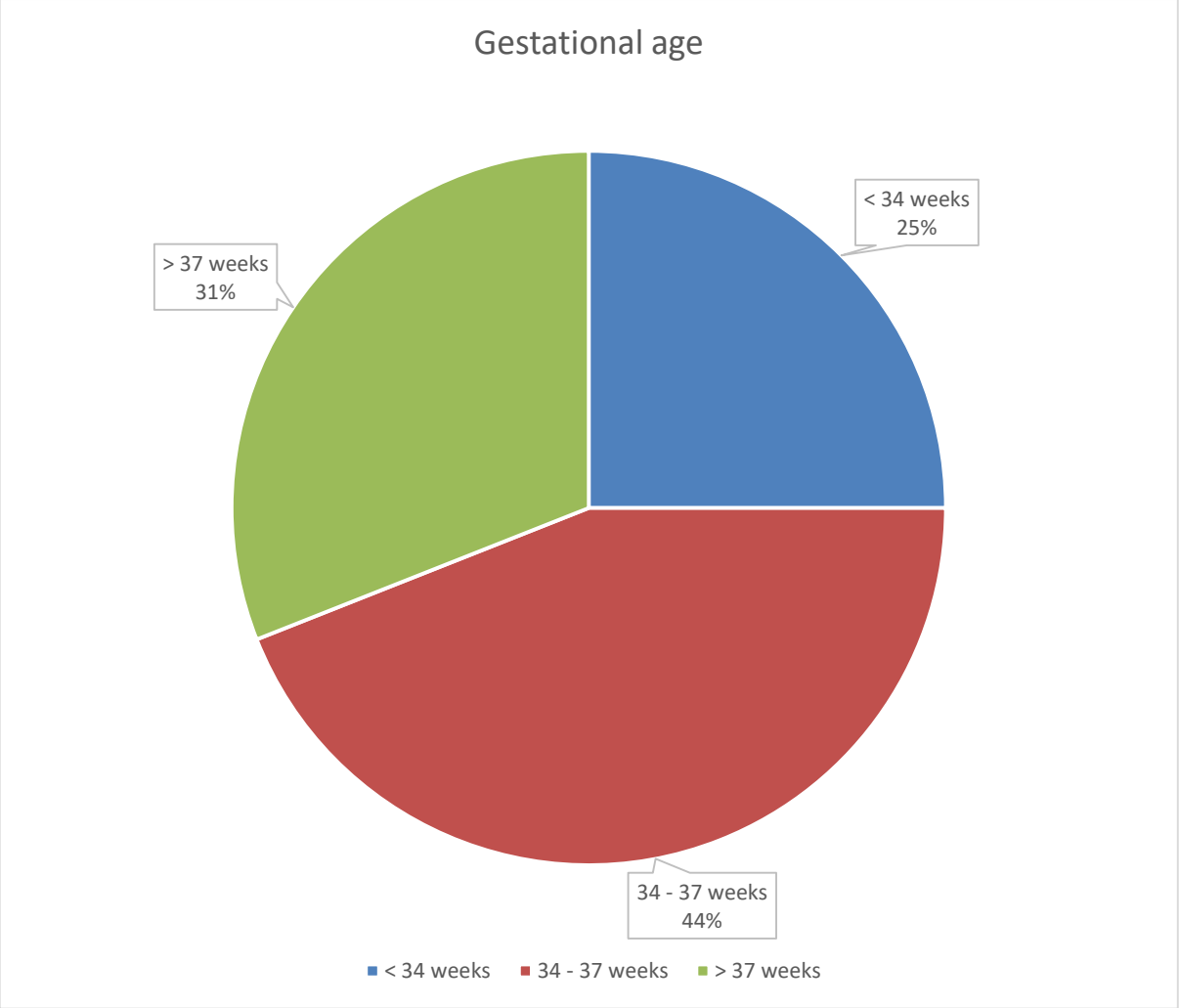


Fig:5 Gestational age

Table 5: Anthropometry

	Weight(kg)	Height(cm)	BMI(kg/m2)
Mean	56.55	152.56	24.50
Std. Deviation	8.09	5.26	3.22

Table 4 summarizes the anthropometric data of the participants, including weight, height, and

BMI. The mean weight is 56.55 kg, with a standard deviation of 8.09 kg; the mean height is 152.56 cm, with a standard deviation of 5.26 cm; and the mean BMI is 24.50 kg/m², with a standard deviation of 3.22.

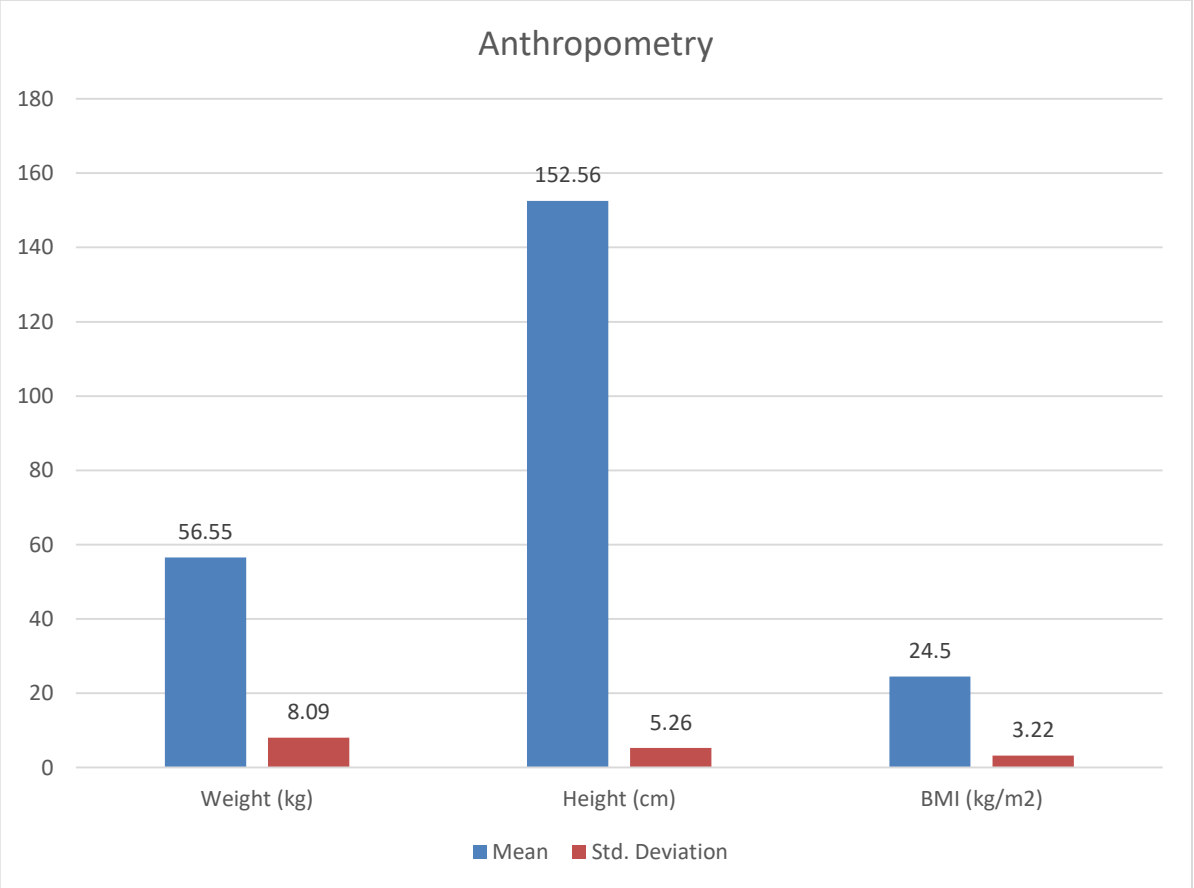


Fig 6: Anthropometry

Table 6: Gestational Hypertension Category

GHT Category	Frequency	Percent
Gestational Hypertension	45	45.00
Preeclampsia	20	20.00
Severe preeclampsia	19	19.00
HELLP Syndrome	3	3.00
Eclampsia	13	13.00
Total	100	100.00

Table 6 outlines the distribution of gestational hypertension categories among 100 patients. The most common condition was **gestational hypertension**, accounting for **45%** of cases, followed by **preeclampsia (20%)** and **severe preeclampsia (19%)**. More critical conditions such as **eclampsia (13%)** and **HELLP syndrome (3%)** were less frequent but clinically significant. This distribution suggests that while the majority of cases involve milder hypertensive disorders, a substantial proportion progress to severe and potentially life-threatening forms, underscoring the importance of early detection, close monitoring, and timely management of hypertensive disorders in pregnancy.

Fig7: Gestational Hypertension Category

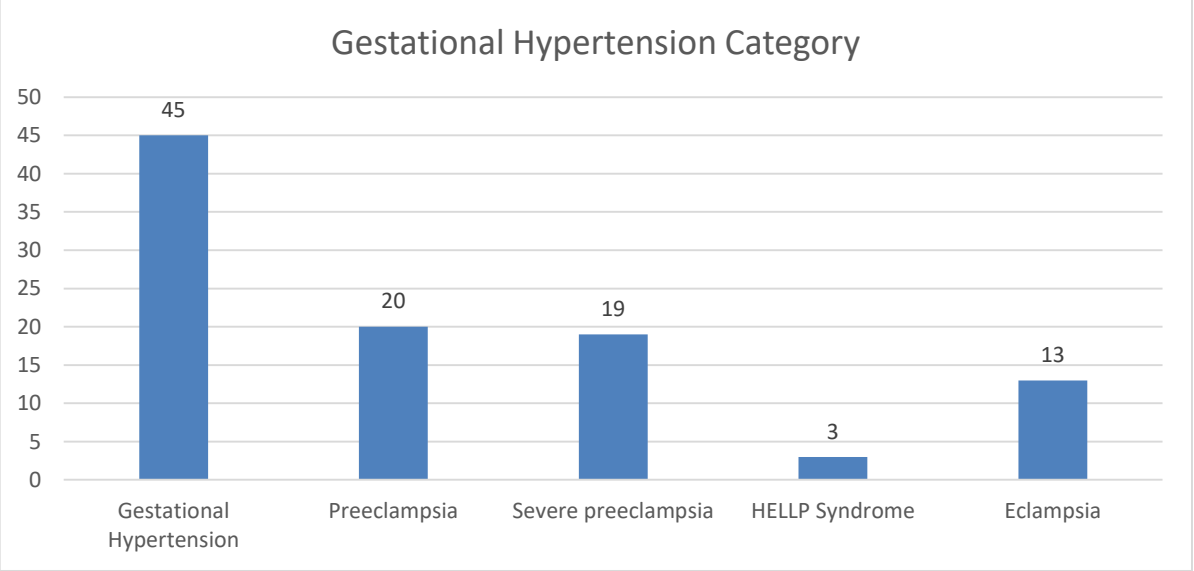


Table 7: Distribution of Systolic Blood Pressure

	SBP				
	GHT	**HELLP Syndrome**	**Preeclampsia**	**Severe preeclampsia**	**Eclampsia**
Mean	147.82	175.67	158.95	168.32	177.46
Std. Deviation	12.34	8.02	5.70	13.33	3.50
	p-value < 0.001				

Table 7 presents the distribution of **mean systolic blood pressure (SBP)** across different categories of hypertensive disorders in pregnancy. The highest mean SBP was observed in **eclampsia (177.46 mmHg)** and **HELLP syndrome (175.67 mmHg)**, followed by **severe preeclampsia (168.32 mmHg)**. **Preeclampsia** and **gestational hypertension** had lower mean SBP values at **158.95 mmHg** and **147.82 mmHg**, respectively. The difference in SBP among the groups was **statistically significant (p < 0.001)**, indicating that more severe hypertensive disorders are associated with markedly elevated systolic pressures.

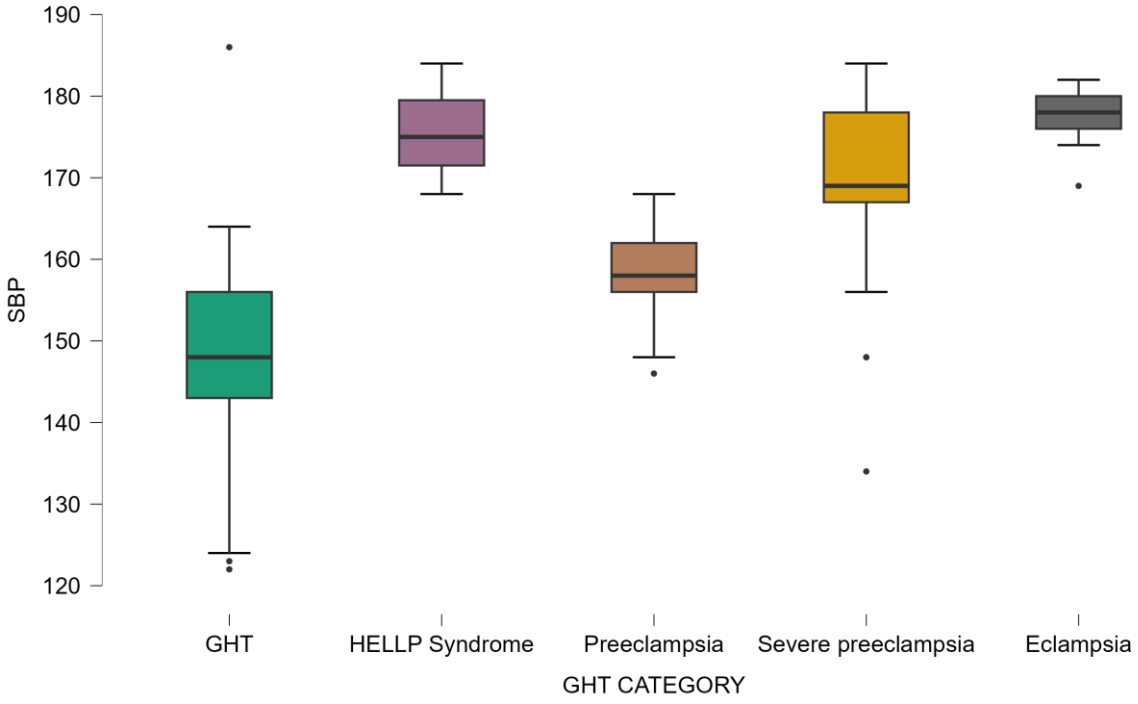


Table 8: Distribution of Diastolic Blood Pressure

	DBP				
	GHT	**HELLP Syndrome**	**Preeclampsia**	**Severe preeclampsia**	**Eclampsia**
Mean	91.33	111.33	100.70	102.16	115.62
Std. Deviation	7.36	5.03	4.03	4.55	3.64
	p-value < 0.001				

Table 8 presents the distribution of **mean diastolic blood pressure (DBP)** across different categories of gestational hypertensive disorders. The highest DBP was observed in **eclampsia (115.62 mmHg)** and **HELLP syndrome (111.33 mmHg)**, followed by **severe preeclampsia (102.16 mmHg)** and **preeclampsia (100.70 mmHg)**. The lowest mean DBP was noted in **gestational hypertension (91.33 mmHg)**. The differences across groups were **statistically significant (p < 0.001)**, indicating a clear correlation between the severity of the hypertensive disorder and elevated DBP.

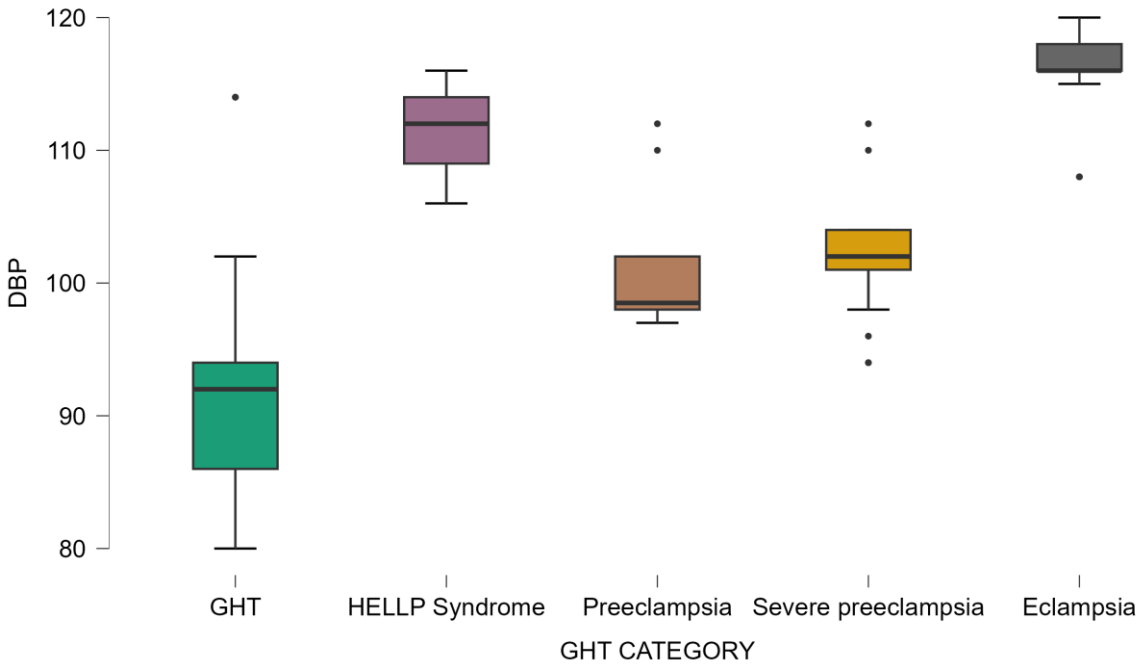


Table 9: Uric Acid Classification

Uric Acid Classification	Frequency	Percent
Hyperuricemia	43	43.00
Normal	57	57.00
Total	100	100.00

Table shows the distribution of participants based on their uric acid classification. It reveals that 43% of participants had hyperuricemia, while 57% had normal uric acid levels.

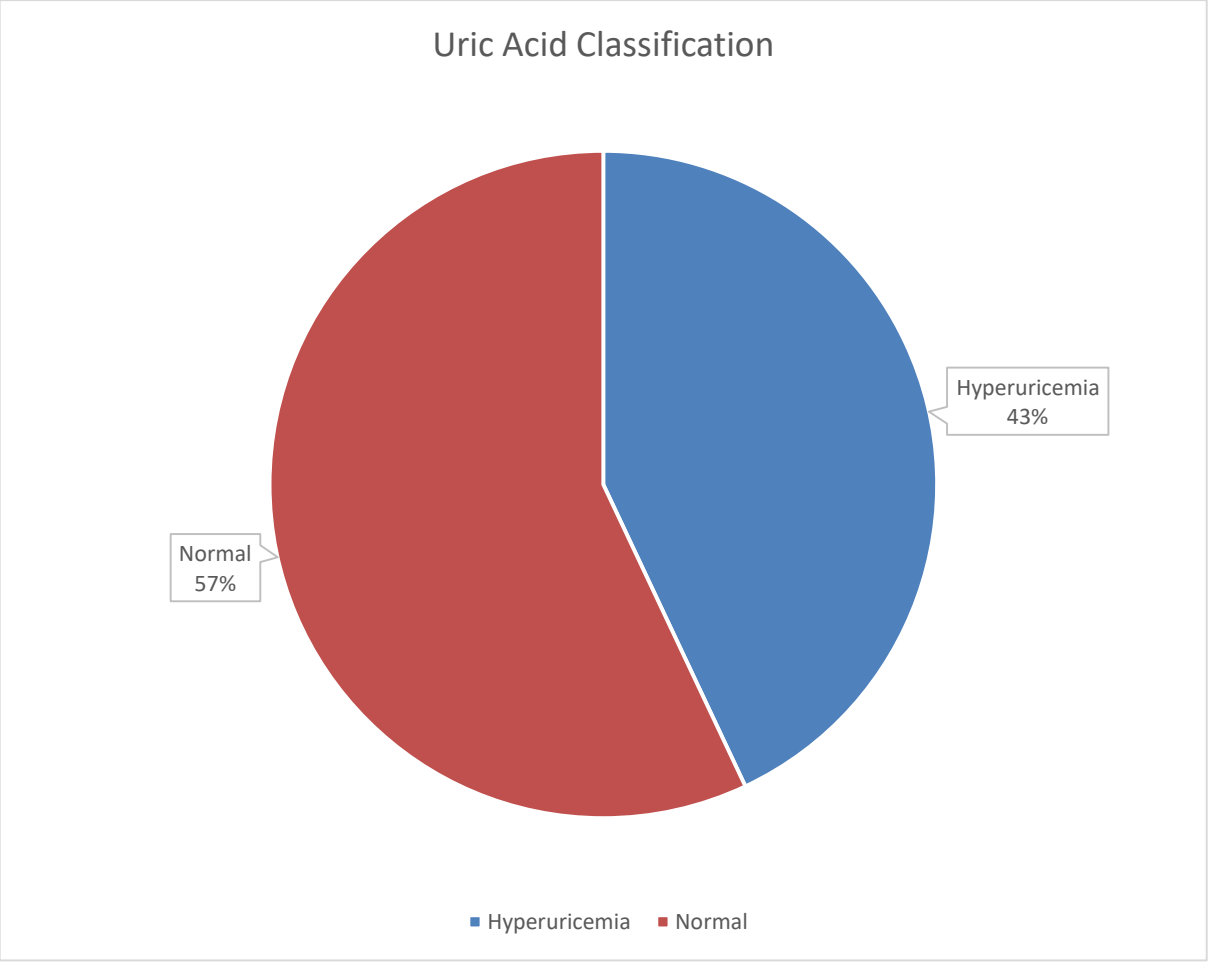


Table 10: Urine Albumin

Urine Albumin	Frequency	Percent
1+	5	5.00
2+	9	9.00
3+	2	2.00
4+	34	34.00
Nil	50	50.00
Total	100	100.00

Table 10 presents the distribution of participants based on urine albumin levels. It shows that 50% of participants had no detectable urine albumin (Nil), while 34% had 4+, 9% had 2+, 5% had 1+, and 2% had 3+ urine albumin.

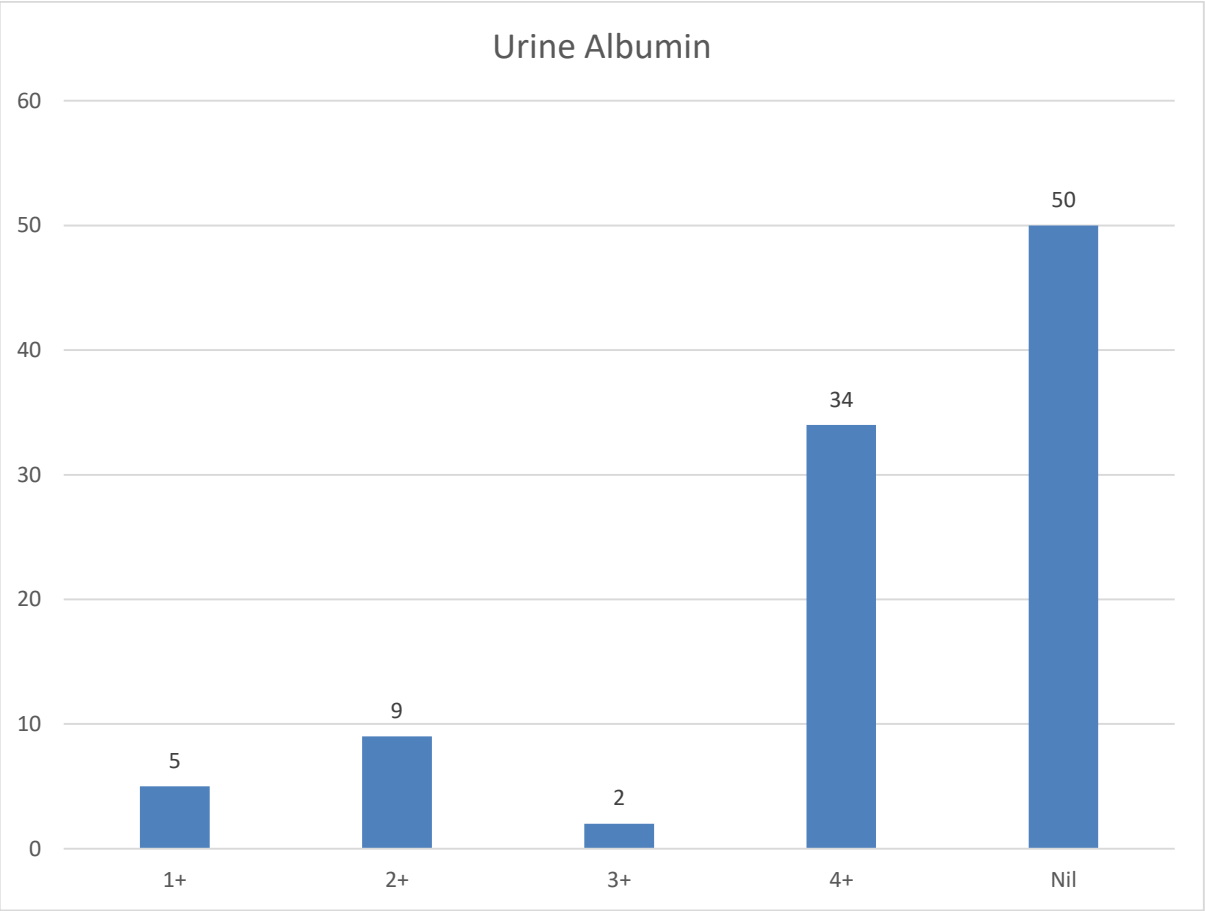


Table 11: Mode of Delivery

Mode of Delivery	Frequency	Percent
Elective LSCS	30	30.00
Emergency LSCS	31	31.00
Labour Naturale	39	39.00
Total	100	100.00

Table 10 displays the distribution of participants based their mode delivery. It shows that 39% participants had a natural labor delivery, 31% underwent an emergency cesarean section (LSCS), and 30% had an elective cesarean section.

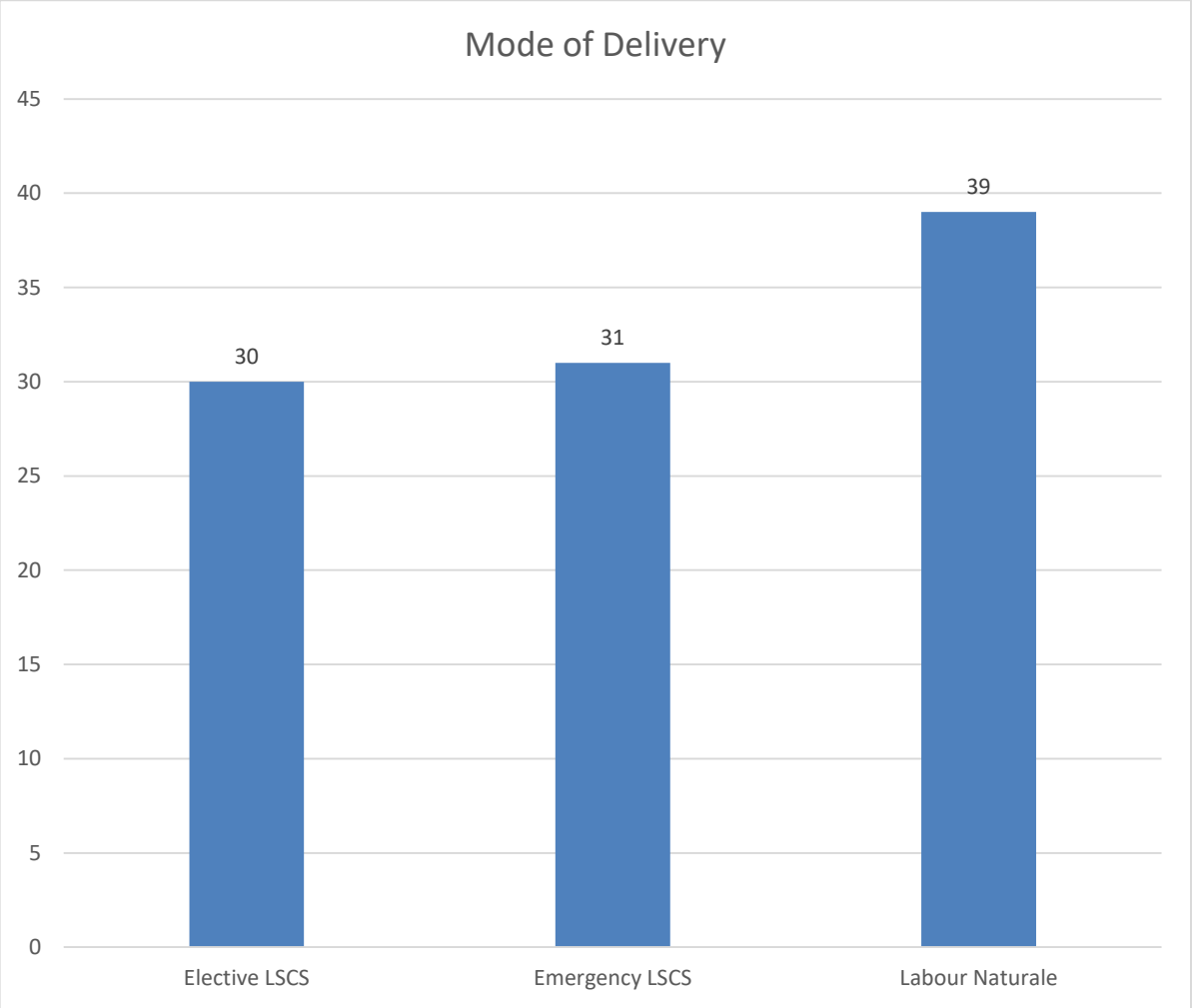


Table 12: Fetal Outcome

Fetal Outcome	Frequency	Percent
Early Preterm	15	15.00
Extreme Preterm	4	4.00
IUD	3	3.00
IUGR	10	10.00
LBW	24	24.00
Late Preterm	5	5.00
Term baby	39	39.00
Total	100	100.00

Table 12 presents the distribution of fetal outcomes among participants. It shows that 39% of the participants had term babies, while 24% had low birth weight (LBW), 15% had early preterm, 10% had intrauterine growth restriction (IUGR), 5% had late preterm, 4% had extreme preterm, and 3% experienced intrauterine death (IUD).

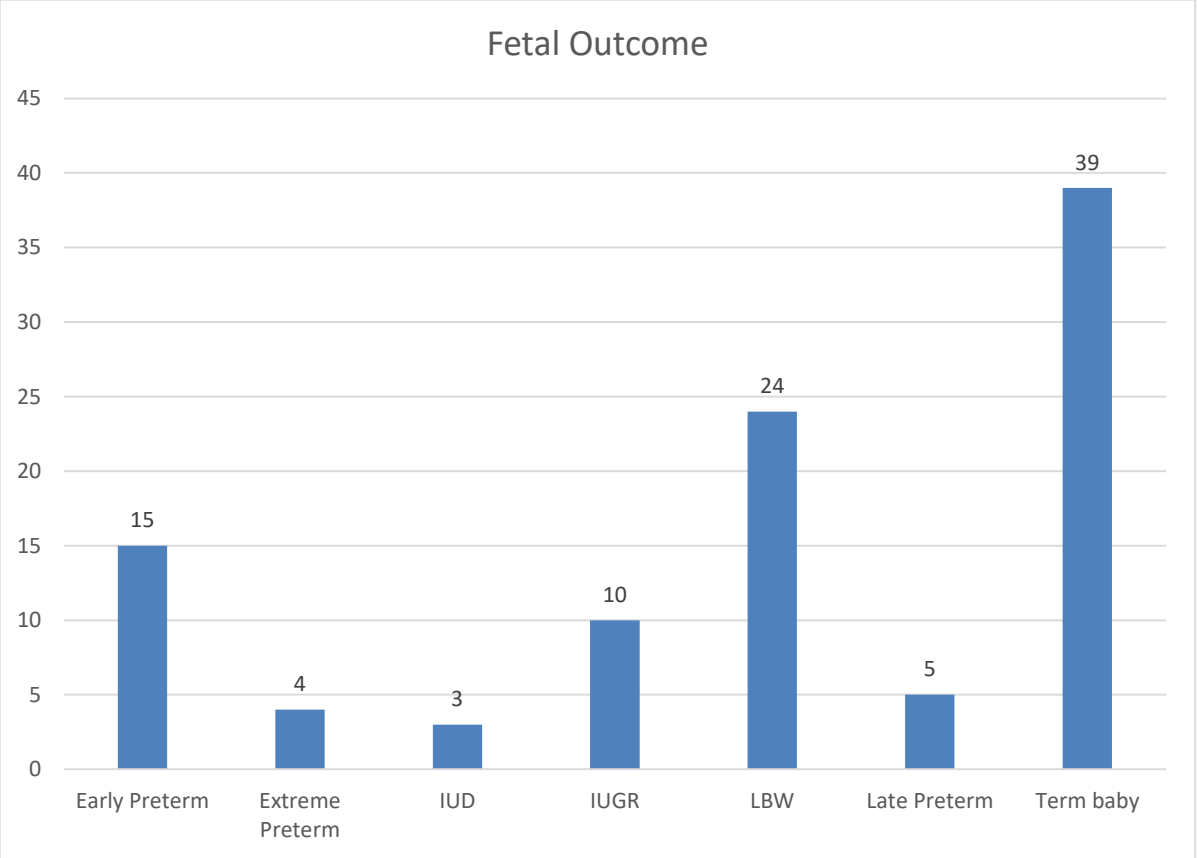


Table 13: Association between gestational hypertension and Uric Acid

GHT CATEGORY	Uric Acid Classification		Total
	Hyperuricemia	Normal	
Gestational Hypertension	14	31	45
HELLP Syndrome	3	0	3
Preeclampsia	9	11	20
Eclampsia	8	5	13
Severe preeclampsia	9	10	19
Total	43	57	100

p-value < 0.001

Table 13 shows the association between gestational hypertension categories and uric acid classification. Among the 58 participants with gestational hypertension, 17 had hyperuricemia, and 41 had normal uric acid levels. In the HELLP syndrome group, all three participants had normal uric acid levels. For preeclampsia, 5 participants had hyperuricemia, and 15 had normal uric acid levels. In severe preeclampsia, 14 had hyperuricemia, and 5 had normal uric acid levels. The total number of participants is 100, with 39 having hyperuricemia and 61 having normal uric acid levels. The p-value (< 0.001) indicates a statistically significant association between gestational hypertension and uric acid classification.

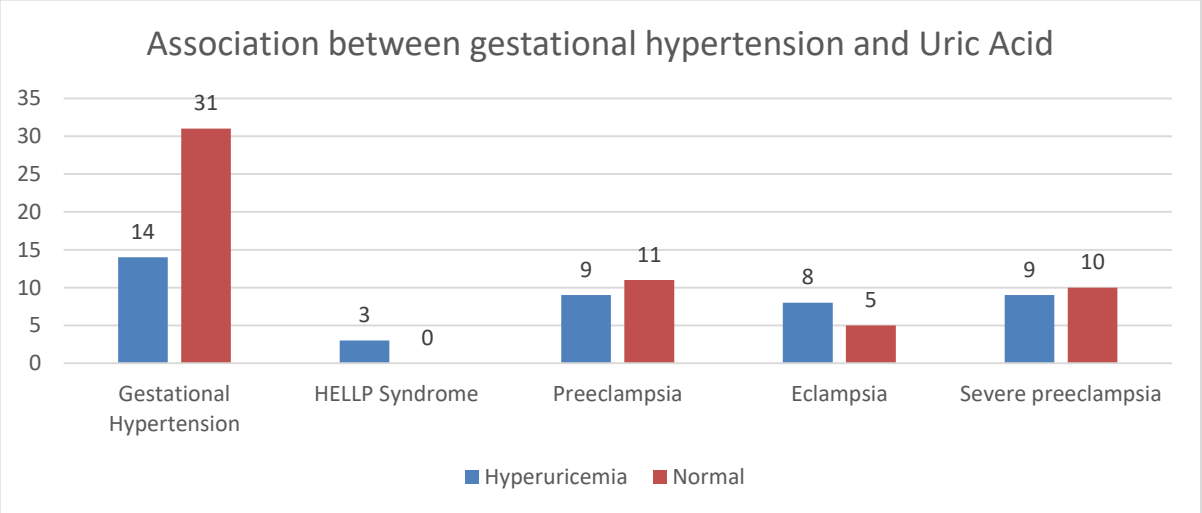


Table 14: Association between Gestational Hypertension and Gestational age

GHT CATEGORY	Gestational age			Total
	< 34 weeks	34 – 37 weeks	> 37 weeks	
GHT	9	20	16	45
HELLP Syndrome	0	2	1	3
Preeclampsia	3	10	7	20
Eclampsia	4	6	3	13
Severe preeclampsia	9	6	4	19
Total	25	44	31	100
p-value = 0.04				

Table 14 examines the association between **gestational hypertension categories** and **gestational age at presentation or delivery**. A statistically significant association was found (**p = 0.04**), indicating that the type of hypertensive disorder influences the gestational age. Notably, **severe preeclampsia** and **eclampsia** were more likely to occur **before 34 weeks** (9 and 4 cases respectively), suggesting earlier onset and more severe disease progression. In contrast, **gestational hypertension** more frequently presented **after 34 weeks**, with 20 cases between 34–37 weeks and 16 cases beyond 37 weeks. These findings underscore that **more severe hypertensive disorders are associated with earlier gestational age**, reflecting increased maternal-fetal risks and the need for timely management in preterm settings.

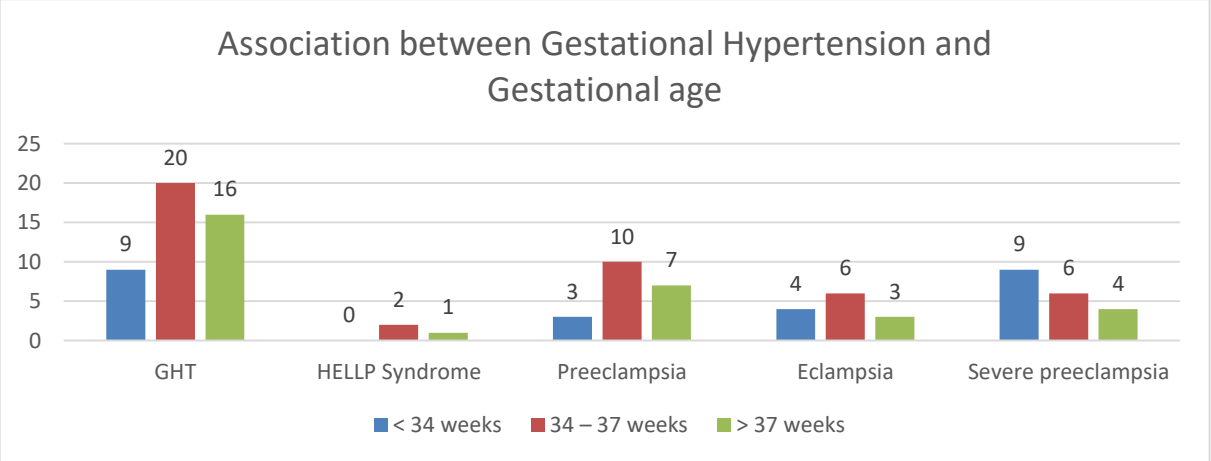


Table 15: Association between fetal outcome and Uric Acid

Uric Acid Classification	Fetal Outcome		
	Abnormal	**Normal**	**Total**
Hyperuricemia	28	15	43
Normal	19	38	57
Total	47	53	100

p-value = 0.001

Table 15 shows a significant association between **serum uric acid levels** and **fetal outcomes** (**p = 0.001**). Among those with **hyperuricemia**, **65.1% (28 out of 43)** had **abnormal fetal outcomes**, while only **34.9%** had normal outcomes. In contrast, among those with **normal uric acid levels**, the majority (**66.7%**) had **normal fetal outcomes**, and only **33.3%** had abnormal outcomes. This statistically significant association suggests that **hyperuricemia is strongly linked to adverse fetal outcomes**, supporting its potential use as a **prognostic biomarker** in pregnancies complicated by hypertensive disorders.

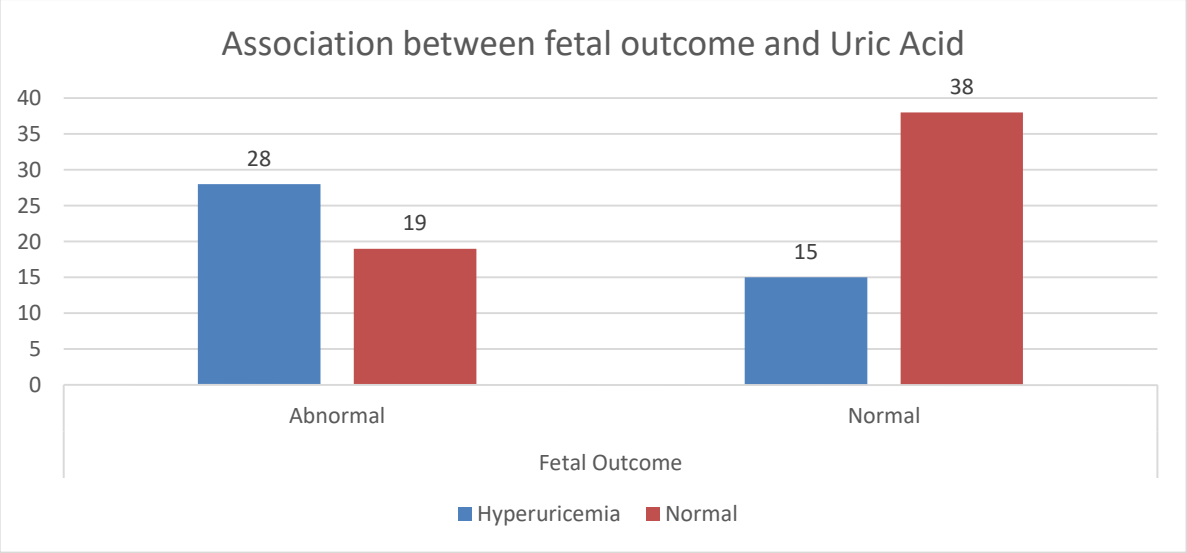


Table 16: Distribution of Serum UricAcid

	Serum uricacid(mg/dL)				
	GHT	**HELLP Syndrome**	**Preeclampsia**	**Severe preeclampsia**	**Eclampsia**
Mean	5.51	7.00	5.20	6.86	5.45
Std. Deviation	0.99	0.00	0.66	1.41	0.74
p-value < 0.001					

Table 16 presents the distribution of **serum uric acid levels** across different categories of gestational hypertensive disorders. The highest mean levels were seen in **HELLP syndrome (7.00 mg/dL)** and **severe preeclampsia (6.86 mg/dL)**, followed by **gestational hypertension (5.51 mg/dL)** and **eclampsia (5.45 mg/dL)**. The lowest mean level was observed in **preeclampsia (5.20 mg/dL)**. The different among group was **statistically significantly (p < 0.001)**, suggesting a strong correlation between elevated serum uric acid and the severity of hypertensive disorders. Clinically, this supports the role of Serum Uric Acid As A Potential Biomarker for identifying and monitoring severe hypertensive complications in pregnancy, particularly HELLP syndrome and severe preeclampsia.

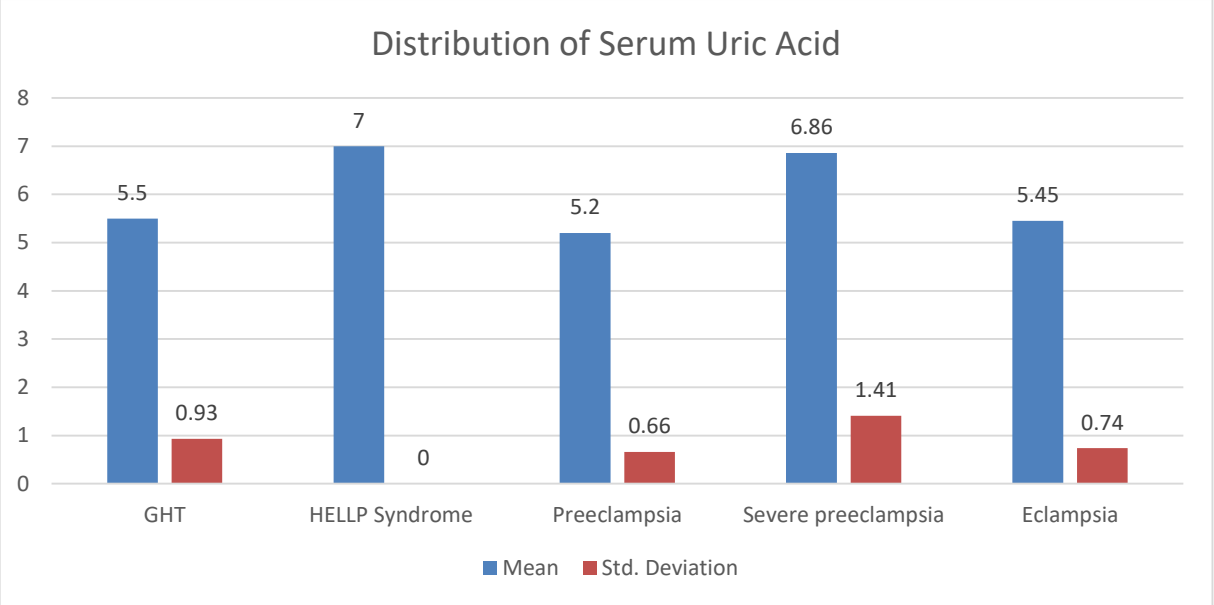
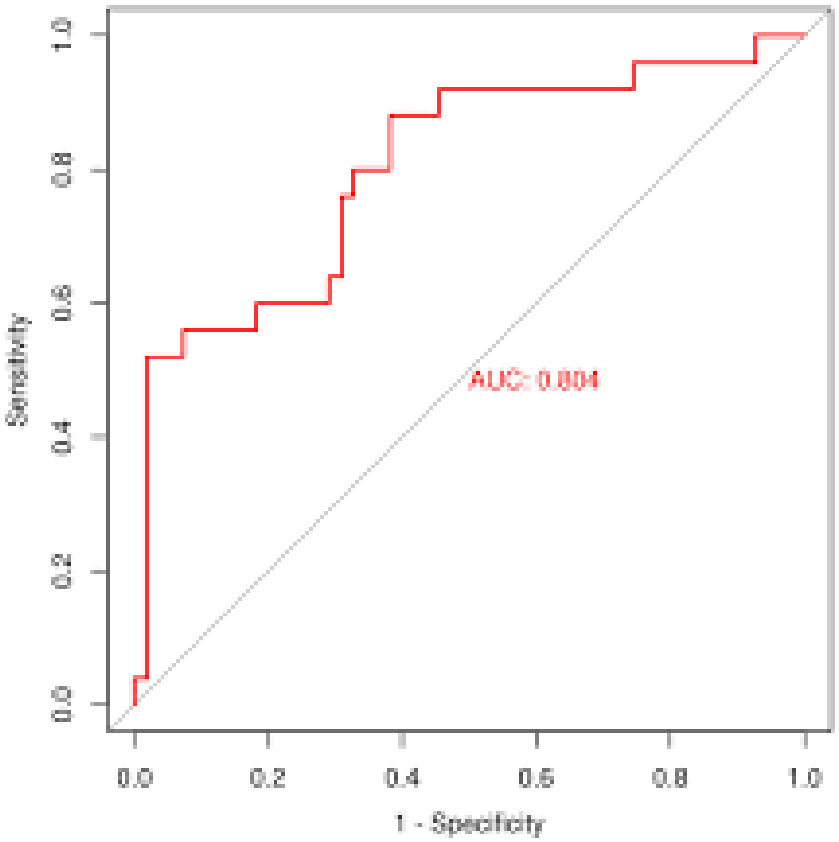


Table 17: Diagnostic Accuracy of Serum Uric acid

Sensitivity	95.01%
Specificity	86.72%
AUC	0.804

Table 16 presents the diagnostic accuracy of **serum uric acid** in identifying adverse outcomes or severe hypertensive disorders in pregnancy. The test demonstrates **high sensitivity (95.01%)**, indicating it correctly identifies most true cases, and **good specificity (86.72%)**, reflecting its ability to correctly rule out non-cases. The **Area Under the Curve (AUC) of 0.804** signifies **excellent overall diagnostic performance**. These values suggest that serum uric acid is a **reliable and clinically useful biomarker** for screening and early detection of severe hypertensive complications in pregnancy.



DISCUSSION

Findings of the present study

Demographic and Obstetric Profile

The age distribution of the study population showed that the majority (62%) were between 18–25years, followed 24% in 26–30age range. Very few participants were older than 30 years, reflecting a younger obstetric population. Regarding obstetric history, a near-equal distribution was seen between primigravida (48%) and multigravida (52%) women.

Gestational Age at Presentation

Analysis of gestational age revealed that 44% of women delivered between 34 and 37 weeks, 31% delivered after 37 weeks, and 25% had preterm births before 34 weeks. This distribution suggests a significant burden of preterm deliveries among those with hypertensive disorders.

Anthropometric Measures

Mean weight, height participants 56.55kg and 152.56cm, respectively. The average BMI was 24.50 kg/m², which falls within the upper range of the normal category, possibly reflecting subtle metabolic changes common in hypertensive pregnancies.

Clinical Diagnosis and Categorization of Hypertensive Disorders

Gestational hypertension was the most prevalent category, affecting 45% of participants. Preeclampsia and severe preeclampsia were present in 20% and 19% respectively. HELLP syndrome (3%) and eclampsia (13%) were less common but represented the more severe end of the disease spectrum.

Blood Pressure Trends

SBP and DBP level significant rise patient severe preeclampsia, eclampsia, and HELLP syndrome. The mean SBP in eclampsia was the highest (177.46 mmHg), followed closely by HELLP syndrome (175.67 mmHg). Diastolic pressures followed a similar pattern, with the highest values observed in eclampsia (115.62 mmHg) and HELLP syndrome (111.33 mmHg). Both SBP and DBP differences across diagnostic categories were statistically significant ($p < 0.001$), reinforcing the strong correlation between elevated BP and disease severity.

Serum Uric Acid and Proteinuria

Hyperuricemia was present in 43% of participants. Additionally, 50% had no proteinuria, but 34% exhibited 4+ albuminuria, indicating significant renal involvement in a subset of patients. statistically significant association ($p < 0.001$) was found between uricacid level the severity of hypertensive disorders, with the highest mean uric acid level observed in HELLP syndrome (7.0 mg/dL) and severe preeclampsia (6.86 mg/dL).

Mode of Delivery and Neonatal Outcomes

Normal vaginal delivery occurred in 39% of cases, while 61% required cesarean sections (30%

elective, 31% emergency). Fetal outcomes were suboptimal in a notable proportion: 24% had low birth weight, 15% were early preterm, 10% had IUGR, and 3% had IUD. Only 39% pregnancies resulted in term deliveries with favorable outcomes.

Association Analysis

statistical significat association (p = 0.04) was observed between the type of hypertensive disorder and gestational age, with severe disorders such as eclampsia and severe preeclampsia more frequently presenting before 34 weeks. Furthermore, there was a strong association between hyperuricemia and abnormal fetal outcomes (p = 0.001), further suggesting the predictive utility of serum uric acid as a biomarker.

Diagnostic Value of SerumUricAcid

Serum uricacid was shown to have a high diagnostic accuracy in predicting severe hypertensive complications and poor fetal outcomes, with a sensitivity of 95.01%, specificity of 86.72%, and an AUC of 0.804. This underlines its potential utility as a cost-effective, easily accessible diagnostic and prognostic tool.

Comparison with similar studies

The present study observed majority participants young women aged 18–25years, nearly equal distribution between primigravida and multigravida cases. This demographic trend aligns with findings from a Nepalese study by Das et al., 2023, which also reported a higher prevalence of hypertensive disorders among younger pregnant women. Mean gestation at delivery was predominantly between 34and37weeks, indicating a significant incidence of late preterm births among women with hypertensive disorders.[41]

A significant finding of this study is the elevated serum uric acid levels in patients with severe preeclampsia and HELLP syndrome, with mean levels reaching 6.86 mg/dL and 7.00 mg/dL, respectively. These results are consistent with those reported by Singh et al., who found mean uric acid levels of 6.20 mg/dL in preeclampsia and 7.38 mg/dL in eclampsia cases. Additionally,

Kumar et al. demonstrated that higher maternal serum uric acid levels were associated with increased disease severity and adverse maternal outcomes. [35,42]

The diagnostic accurac of serum uric acid in predicting severe hypertensive complication was notable current study, with a sensitivity of 95.01% and specificity of 86.72%. These findings support the utility of serum uric acid as a reliable biomarker for risk stratification in HDP.

The study reported statistically significant correlation between elevated blood pressure levels and the severity of hypertensive disorders. Mean systolic and diastolic blood pressures were highest in patients with eclampsia and HELLP syndrome. This observation is corroborated by a study by Xavier et al., 2023 conducted in Ghana, which found that women with preeclampsia/eclampsia had significantly higher risks of adverse maternal and neonatal outcomes, including elevated blood pressure levels. [43]

The present study identified a strong association between hyperuricemia and adverse fetal outcomes, including low birth weight (LBW), intrauterine growth restriction (IUGR), and intrauterine death (IUD). This is in line with findings from a Chinese study by Luo et al., 2024, which reported that elevated serum uric acid levels in women with advanced maternal age were independently associated with an increased risk of LBW. Furthermore, Kumar et al. observed that higher maternal serum uric acid levels were linked to poorer perinatal outcomes, reinforcing the prognostic value of this biomarker. [35,44]

In this study, a higher proportion of women with severe hypertensive disorders underwent cesarean sections (both elective and emergency) compared to those with gestational hypertension. This trend is supported by a retrospective study in Nepal, which found that preeclampsia was strongly associated with an increased likelihood of cesarean delivery.[41]

Clinical Implications

The findings of this study underscore the importance of early identification and management of hypertensive disorders in pregnancy. The significant associations between elevated serum uric

acid levels, increased blood pressure, and adverse maternal and fetal outcomes highlight the need for routine monitoring of these parameters. Implementing such measures could facilitate timely interventions, potentially improving pregnancy outcomes.

The present study highlights a strong correlation between serum uric acid levels and both the severity of gestational hypertensive disorders and adverse fetal outcomes. Blood pressure parameters, gestational age at delivery, and proteinuria were also significantly associated with disease severity. Overall, this data supports the inclusion of serum uric acid as a routine part of risk stratification and monitoring in pregnancies complicated by hypertension.

The present study offers significant insights into the clinical and biochemical spectrum of hypertensive disorders during pregnancy and their impact on maternal and fetal outcomes. While the raw data highlight expected clinical trends, a broader analysis reveals the implications and clinical relevance of the findings in a holistic manner.

The high burden of severe hypertensive disorders observed in the study population, including preeclampsia, severe preeclampsia, and eclampsia, emphasizes the persistent challenges faced in antenatal care despite advances in screening and treatment. The notable representation of these severe categories suggests that a subset of patients either progress rapidly or present late in pregnancy with advanced disease, reflecting potential gaps in timely diagnosis, health-seeking behavior, or access to care.

Elevated serum uric acid levels in patients with more severe disease categories and their statistical association with both maternal condition and fetal outcomes reinforce the hypothesis that uric acid is more than just a metabolic byproduct. Its role as a marker of oxidative stress and endothelial dysfunction becomes clinically significant in the context of preeclampsia and HELLP syndrome. In particular, the higher diagnostic sensitivity and specificity observed for serum uric acid support its potential integration into antenatal monitoring protocols. This would

enable early identification of high-risk patients and potentially inform decisions on hospitalization, timing of delivery, and neonatal care preparedness.

The data on blood pressure trends further validate the known pathophysiological escalation seen in hypertensive disorders of pregnancy. The gradation of both systolic and diastolic values across diagnostic categories underscores how vascular reactivity and systemic involvement intensify with disease severity. This reinforces the clinical need for consistent blood pressure monitoring and aggressive antihypertensive management in selected cases, especially given the clear link to adverse fetal outcomes such as low birth weight, growth restriction, and prematurity.

Mode of delivery statistics offer an indirect reflection of clinical decision-making in the face of maternal compromise or fetal distress. The high cesarean rate, particularly emergency procedures, suggests that many cases of hypertensive disorders escalate quickly, necessitating rapid obstetric interventions. This also raises questions about the thresholds used for induction or surgical delivery in such cases and whether early warning indicators could reduce emergency surgical rates through planned interventions.

The fetal outcome distribution paints a sobering picture. The notable proportion of preterm births, low birth weight infants, and intrauterine demise points toward compromised placental function and uteroplacental insufficiency—hallmarks of preeclampsia-related pathophysiology. That hyperuricemia correlated significantly with adverse fetal outcomes in this cohort adds a valuable dimension, suggesting that biochemical monitoring may offer predictive information that clinical signs alone may not immediately reveal.

The associations between gestational age and the severity of hypertensive disorders provide further evidence that earlier gestational involvement often indicates a more aggressive disease course. These cases may present unique clinical dilemmas, requiring a careful balance between maternal stabilization and fetal viability. This interplay highlights the need for multidisciplinary

management, including neonatology support, particularly in resource-limited settings where the threshold for viability may vary.

Taken together, the findings illustrate the complex and multifaceted nature of hypertensive disorders in pregnancy. They challenge clinicians to go beyond routine monitoring, advocating for biochemical markers such as uric acid to be used alongside blood pressure and proteinuria in risk stratification. Additionally, the study indirectly calls for stronger community outreach and antenatal education to ensure earlier detection and intervention. Ultimately, it underlines the critical importance of an integrated approach combining clinical vigilance, timely investigations, and appropriate obstetric interventions to mitigate maternal and fetal risks in hypertensive pregnancies.

Limitations and Future Directions

While study provides valuable insights, limited by sample size ,single-center design. Future multicenter studies with larger populations warranted validate findings and explore integration serum uric acid measurements into standard prenatal care protocols.

Limitations

While the present study provides valuable insights into the clinical and biochemical profile of hypertensive disorders in pregnancy, several limitations must be acknowledged. Firstly, the study was conducted in a single tertiary care center, which may limit the generalizability of the findings to other populations, particularly rural or primary care settings where resources and healthcare-seeking behaviors may differ. Secondly, the sample size of 100 participants, although sufficient for internal analysis, may not be large enough to capture the full spectrum of variability in clinical presentation and biochemical markers across different sociodemographic backgrounds.

Another limitation lies in the cross-sectional nature of the study design, which restricts the ability to infer causality between elevated uric acid levels and adverse maternal or fetal

outcomes. Longitudinal follow-up data would have allowed for better assessment of temporal relationships and disease progression. Furthermore, the study did not account for potential confounding variables such as preexisting metabolic conditions (e.g., renal dysfunction, diabetes, obesity), dietary influences, or medications that may influence serum uric acid levels or hypertensive status.

The reliance on semi-quantitative assessment of urine albumin through dipstick methods rather than 24-hour urinary protein or spot protein-creatinine ratio may affect the accuracy of proteinuria categorization. Additionally, the study did not incorporate other emerging biomarkers or inflammatory markers, which could have provided a more comprehensive risk assessment model. Lastly, neonatal outcomes were broadly categorized and lacked detailed clinical follow-up on neonatal morbidity and mortality, limiting the scope of pediatric implications.

Conclusion

The present study reinforces the significant clinical and prognostic implications of hypertensive disorders in pregnancy, with particular emphasis on the role of serum uric acid as a reliable biomarker. Elevated uric acid levels were significantly associated with both the severity of maternal hypertensive status and adverse fetal outcomes, including low birth weight, preterm birth, and intrauterine growth restriction. The strong statistical associations between elevated blood pressure readings, gestational age at delivery, and biochemical parameters highlight the multifactorial nature of maternal-fetal compromise in hypertensive pregnancies.

These findings support the incorporation of serum uric acid measurement into routine antenatal care for early risk stratification and timely intervention. Additionally, the data underscore the importance of comprehensive antenatal surveillance, multidisciplinary management, and individualized delivery planning to mitigate maternal and neonatal morbidity. While the study strengthens existing knowledge on hypertensive disorders of pregnancy, it also paves the way

for further research exploring integrated diagnostic models and longitudinal monitoring for improved maternal and perinatal health outcomes.

Summary

This observational study evaluated the clinical, biochemical, and obstetric profiles of 100 pregnant women diagnosed with various forms of gestational hypertension, including preeclampsia, eclampsia, and HELLP syndrome. The study found that a significant number of women presented with severe hypertensive disorders, often leading to early gestational termination and adverse fetal outcomes. Elevated systolic and diastolic blood pressures were strongly correlated with disease severity. Serum uric acid emerged as a highly sensitive and specific marker for predicting both severe maternal complications and poor neonatal outcomes, with a diagnostic AUC of 0.804.

A statistically significant relationship was also observed between serum uric acid levels and the occurrence of low birth weight, preterm delivery, and fetal demise. Additionally, the mode of delivery was influenced by disease severity, with cesarean sections being more common in severe cases. The study concludes that uric acid may serve as an accessible, cost-effective adjunct biomarker in clinical practice for identifying high-risk pregnancies affected by hypertensive disorders. Despite its limitations, the study adds to the growing body of evidence advocating for early screening and proactive management strategies in such high-risk obstetric populations.

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PROFORMA

NAME:

AGE:

UHID NO:

ADDRESS:

I.PNO:

DATE/TIMEOFADMISSION:

DATE/TIMEOFDISCHARGE:

CHIEFCOMPLAINTS:

OBSTETRICAL HISTORY: Booked/
Unbooked/ Referred

Married Life: Consanguinous marriage: Yes/ No

Obstetrical Score:

MENSTRUAL HISTORY:

LMP:

EDD: cEDD:

POG:

PAST HISTORY:

PERSONAL HISTORY:

FAMILY HISTORY:

GENERAL PHYSICAL EXAMINATION:

Pallor/ Icterus/ Cyanosis/ Clubbing/
Lymphadenopathy/ Edema

Pulse: RR:

BP:

RS: Temp:

CNS: CVS:

Per Abdomen:

Per Speculum:

Per Vagina:

PROVISIONAL DIAGNOSIS:

INVESTIGATIONS:

BLOOD GROUP

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

PT,APTT,INR

LIVER FUNCTION TEST:

DAT E	TB (mg/dl)		SGOT(U/L)	SGP T(U/ L)	AL P(U/ L)	TP (g/dl)	Albumi n (g/dl)	A: G	GG T(U/ L)	LDH (mg/dl)

RENAL FUNCTION TEST:

BLOOD UREA

SERUM CREATININE

SODIUM

POTASSIUM

URIC ACID

URINE ROUTINE

Fetal outcome:

PATIENT INFORMATION SHEET

STUDY TITLE: MATERNAL SERUM URIC ACID AS A PROGNOSTIC FACTOR FOR PERINATAL OUTCOME IN HYPERTENSIVE DISORDERS OF PREGNANCY"

STUDY SITE: R.L Jalappa Hospital and Research Centre, Tamaka, Kolar.

This is to inform you that, uric acid used as a prognostic factor for perinatal outcome in hypertensive disoders of pregnancy mothers.

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 relevant investigations such as complete blood count, liver function test, coagulation profile, renal function test, uric acid .which are required for diagnosis and treatment

This will facilitate identifying the extent of severity of Preterm births 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 a part of the study. The investigations needed for the study will be handled by principal investigator. You are free to contact Dr. GUMMALLAPU AKSHAYA or any other member of the above research team for any doubt or clarification you have.

Dr. GUMMALLAPU AKSHAYA

Mobile no:9148312415

E-mail id: akshayachenchu@gmal.com

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9– CF: akshayachenchu@gmal.com

INFORMED CONSENT FORM

I Mrs._____have been explained in my own understandable language, that I will be included in a study which is —: **MATERNAL SERUM URIC ACID AS A PROGNOSTIC FACTOR FOR PERINATAL OUTCOME IN HYPERTENSIVE DISORDERS OF PREGNANCY"**

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:

Signature of the witness:

Name:

Name:

Date:

Relation to the patient:

Place:

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Masterchart

S.No.	AGE (years)	Age Group	Obstetric History	Gestational age during delivery (in eeks)	Gestational age	HEIGHT (m)	WEIGHT (kg)	BMI (kg/m2)	SBP	DBP	GHTCATEGORY	URINEALBUMIN	SERUM URIC ACID (mg/dL)	Uric Acid Classification	MODE OF DELIVERY	FETAL OUTCOME	Fetal Outcome	IUD	IUGR	HELLP	ABRUPTION	ECLAMPSIA	MATERNAL DEATH	ICH	PULMEDEMA	SPONTANEOUS EXPULSION
1	19	18-25	Primi	38	> 37 weeks	1.5	55	24.4	154	92	GHT	++++	6.5	Hyperuricemia	LN	LBW	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
2	28	26-30	Primi	38	> 37 weeks	1.47	93	43	162	87	GHT	Nil	6.6	Hyperuricemia	LN	IUGR	Abnormal	Absent	Present	Absent	Absent	Absent	Absent	Absent	Absent	Absent
3	29	26-30	Multi	38	> 37 weeks	1.42	65	32.2	154	98	GHT	Nil	8	Hyperuricemia	LN	IUGR	Abnormal	Absent	Present	Absent	Absent	Absent	Absent	Absent	Absent	Absent
4	23	18-25	Primi	33	< 34 weeks	1.51	71	31.1	174	102	Severe preeclampsia	++++	7	Hyperuricemia	LN	IUGR	Abnormal	Absent	Present	Absent	Absent	Absent	Absent	Absent	Absent	Absent
5	23	18-25	Primi	32	< 34 weeks	1.63	61	23	146	89	GHT	Nil	5.4	Normal	Elective LSCS	LBW	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
6	22	18-25	Multi	24	< 34 weeks	1.54	52	21.9	168	104	Severe preeclampsia	++++	8.9	Hyperuricemia	Emergency LSCS	IUD	Abnormal	Present	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
7	19	18-25	Primi	39	> 37 weeks	1.43	45	22	176	115	Eclampsia	Nil	5.4	Normal	LN	Term baby	Normal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
8	32	31-35	Multi	38	> 37 weeks	1.5	60	26.7	155	84	GHT	++++	9	Hyperuricemia	Elective LSCS	IUGR	Abnormal	Absent	Present	Absent	Absent	Absent	Absent	Absent	Absent	Absent
9	23	18-25	Primi	39	> 37	1.46	60	28.1	162	86	GHT	+++	6.7	Hyperuricemia	LN	Term baby	Normal	Abs	Pres	Abs	Absent	Absent	Absent	Abs	Absent	Absent

					weeks													ent	ent	ent				ent		
10	26	26-30	Multi	37	34-37 weeks	1.5	55	24.4	160	87	GHT	Nil	4.6	Normal	LN	LBW	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
11	27	26-30	Multi	34	34-37 weeks	1.6	62	24.2	182	120	Eclampsia	++++	6.7	Hyperuricemia	LN	Term baby	Normal	Absent	Present	Absent	Absent	Absent	Absent	Absent	Absent	Absent
12	23	18-25	Primi	28	<34 weeks	1.55	52	21.6	178	94	Severe preeclampsia	++++	10.2	Hyperuricemia	Emergency LSCS	IUD	Abnormal	Present	Absent	Absent	Present	Absent	Absent	Absent	Absent	Absent
13	32	31-35	Primi	32	<34 weeks	1.52	60	26	155	98	Preeclampsia	++++	8	Hyperuricemia	Emergency LSCS	IUGR	Abnormal	Absent	Present	Absent	Absent	Absent	Absent	Absent	Absent	Absent
14	29	26-30	Primi	38	>37 weeks	1.44	58	28	154	99	GHT	+	9	Hyperuricemia	Elective LSCS	LBW	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
15	25	18-25	Multi	37	34-37 weeks	1.51	53	23.2	157	98	Preeclampsia	Nil	6	Hyperuricemia	LN	Term baby	Normal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
16	19	18-25	Primi	34	34-37 weeks	1.38	48	25.2	156	102	GHT	++++	6.3	Hyperuricemia	LN	Early Preterm	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
17	22	18-25	Primi	38	>37 weeks	1.49	64	28.8	168	102	Severe preeclampsia	++++	6.9	Hyperuricemia	Emergency LSCS	IUGR	Abnormal	Absent	Present	Absent	Absent	Absent	Absent	Absent	Absent	Absent
18	20	18-25	Multi	30	<34 weeks	1.57	59	23.9	166	100	Severe preeclampsia	++++	10.4	Hyperuricemia	Emergency LSCS	IUD	Abnormal	Present	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent

19	27	26-30	Multi	39	> 37 weeks	1.55	57	23.7	162	102	Preeclampsia	++++	4.4	Normal	Elective LSCS	Term baby	Normal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
20	18	18-25	Primi	38	> 37 weeks	1.44	44	21.2	132	114	GHT	++	5.1	Normal	LN	LBW	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
21	22	18-25	Primi	36	34 - 37 weeks	1.53	56	23.9	158	89	GHT	++++	6.5	Hyperuricemia	LN	Term baby	Normal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
22	29	26-30	Multi	33	< 34 weeks	1.42	42	20.8	174	118	Eclampsia	++	6.6	Hyperuricemia	Emergency LSCS	Term baby	Normal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
23	30	26-30	Multi	35	34 - 37 weeks	1.59	53	21	186	93	GHT	+	6	Hyperuricemia	Elective LSCS	Late Preterm	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
24	35	31-35	Multi	38	> 37 weeks	1.5	58	25.8	156	94	GHT	Nil	4.7	Normal	LN	Term baby	Normal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
25	23	18-25	Primi	37	34 - 37 weeks	1.55	46	19.1	146	92	GHT	Nil	6.7	Hyperuricemia	LN	Term baby	Normal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
26	21	18-25	Primi	35	34 - 37 weeks	1.56	61	25.1	158	102	Preeclampsia	++	5.6	Normal	LN	Term baby	Normal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
27	29	26-30	Primi	35	34 - 37 weeks	1.52	61	26.4	164	90	GHT	Nil	4.7	Normal	Elective LSCS	Term baby	Normal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
28	25	18-25	Multi	28	< 34	1.63	58	21.8	178	116	Eclampsia	+++	5.9	Normal	Emergency LSCS	Extreme	Abnormal	Abs	Abs	Abs	Absent	Absent	Absent	Abs	Absent	Absent	Absent

					weeks											Preterm		ent	ent	ent				ent		
29	26	26-30	Multi	39	> 37 weeks	1.53	61	26.1	145	93	GHT	Nil	4.4	Normal	Emergency LSCS	Term baby	Normal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
30	20	18-25	Multi	37	34 - 37 weeks	1.45	41	19.5	148	97	GHT	++++	9.8	Hyperuricemia	Emergency LSCS	Term baby	Normal	Present	Absent	Absent	Present	Absent	Absent	Absent	Absent	Absent
31	25	18-25	Multi	35	34 - 37 weeks	1.55	69	28.7	169	118	Eclampsia	Nil	4.6	Normal	Elective LSCS	Late Preterm	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
32	26	26-30	Multi	33	< 34 weeks	1.57	48	19.5	158	95	GHT	Nil	5.4	Normal	Emergency LSCS	Term baby	Normal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
33	27	26-30	Primi	38	> 37 weeks	1.62	71	27.1	155	92	GHT	++	5.2	Normal	Elective LSCS	Term baby	Normal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
34	19	18-25	Primi	37	34 - 37 weeks	1.48	48	21.9	158	97	Preeclampsia	++++	6	Hyperuricemia	LN	Term baby	Normal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
35	23	18-25	Multi	32	< 34 weeks	1.55	57	23.7	136	89	GHT	Nil	4.4	Normal	Emergency LSCS	Early Preterm	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
36	24	18-25	Primi	36	34 - 37 weeks	1.54	66	27.8	176	116	Eclampsia	++++	5.9	Normal	Elective LSCS	IUGR	Abnormal	Absent	Present	Absent	Absent	Absent	Absent	Absent	Absent	Absent
37	23	18-25	Multi	37	34 - 37 weeks	1.56	60	24.7	156	102	Severe preeclampsia	++++	6.7	Hyperuricemia	Emergency LSCS	LBW	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent

38	38	36-40	Primi	37	34-37 weeks	1.5	55	24.4	184	116	HELLP Syndrome	++++	7	Hyperuricemia	Emergency LSCS	Term baby	Normal	Absent	Absent	Present	Absent	Absent	Absent	Absent	Absent	Absent
39	25	18-25	Multi	33	<34 weeks	1.61	69	26.6	162	102	Preeclampsia	Nil	5.2	Normal	Elective LSCS	Early Preterm	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
40	18	18-25	Primi	36	34-37 weeks	1.5	55	24.4	166	98	Preeclampsia	Nil	4.9	Normal	LN	Late Preterm	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
41	37	36-40	Multi	38	>37 weeks	1.48	50	22.8	168	104	Severe preeclampsia	++++	7	Hyperuricemia	Elective LSCS	Term baby	Normal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
42	19	18-25	Primi	33	<34 weeks	1.45	45	21.4	154	92	GHT	Nil	6.5	Hyperuricemia	LN	Term baby	Normal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
43	23	18-25	Multi	30	<34 weeks	1.56	56	23	156	102	GHT	Nil	4.4	Normal	Elective LSCS	Early Preterm	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
44	20	18-25	Primi	37	34-37 weeks	1.56	60	24.7	158	112	Preeclampsia	+	6	Hyperuricemia	LN	LBW	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
45	18	18-25	Primi	32	<34 weeks	1.49	48	21.6	148	103	Severe preeclampsia	++++	6.2	Hyperuricemia	Emergency LSCS	Early Preterm	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
46	32	31-35	Multi	38	>37 weeks	1.5	50	22.2	175	106	HELLP Syndrome	++++	7	Hyperuricemia	LN	Term baby	Normal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
47	29	26-30	Multi	37	34-37	1.6	64	25	168	98	Preeclampsia	Nil	4.9	Normal	Elective LSCS	LBW	Abnormal	Abs	Abs	Abs	Absent	Absent	Absent	Abs	Absent	Absent

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48	24	18-25	Multi	38	> 37 weeks	1.53	57	24.3	156	114	GHT	Nil	4.8	Normal	Elective LSCS	LBW	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
49	19	18-25	Primi	30	< 34 weeks	1.48	60	27.4	168	104	Severe preeclampsia	++++	6.6	Hyperuricemia	Emergency LSCS	Early Preterm	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
50	18	18-25	Primi	37	34 - 37 weeks	1.52	56	24.2	177	118	Eclampsia	++	4.4	Normal	LN	Term baby	Normal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
51	32	31-35	Multi	32	< 34 weeks	1.56	60	24.7	178	116	Eclampsia	++++	5.4	Normal	LN	Early Preterm	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
52	23	18-25	Multi	38	> 37 weeks	1.49	53	23.9	158	98	Preeclampsia	Nil	5	Normal	LN	LBW	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
53	25	18-25	Primi	37	34 - 37 weeks	1.54	58	24.5	166	102	Preeclampsia	Nil	4.7	Normal	LN	LBW	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
54	21	18-25	Primi	34	34 - 37 weeks	1.55	49	20.4	154	88	GHT	Nil	5.2	Normal	LN	Term baby	Normal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
55	28	26-30	Multi	34	34 - 37 weeks	1.52	54	23.4	148	98	Severe preeclampsia	++++	6.2	Hyperuricemia	Elective LSCS	IUGR	Abnormal	Absent	Present	Absent	Absent	Absent	Absent	Absent	Absent	Absent
56	34	31-35	Primi	38	> 37 weeks	1.55	60	25	144	94	GHT	Nil	4.2	Normal	Emergency LSCS	Term baby	Normal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent

57	22	18-25	Multi	30	< 34 weeks	1.45	55	26.2	179	116	Eclampsia	Nil	5.2	Normal	Emergency LSCS	Early Preterm	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
58	26	26-30	Multi	37	34 - 37 weeks	1.5	53	23.6	128	93	GHT	+	4.4	Normal	Elective LSCS	LBW	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
59	24	18-25	Multi	33	< 34 weeks	1.53	56	23.9	134	94	Severe preeclampsia	++++	5.7	Normal	Elective LSCS	Early Preterm	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
60	27	26-30	Primi	38	> 37 weeks	1.6	62	24.2	162	98	Preeclampsia	Nil	4.9	Normal	LN	Term baby	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
61	19	18-25	Primi	37	34 - 37 weeks	1.47	47	21.8	180	118	Eclampsia	Nil	4.8	Normal	LN	LBW	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
62	18	18-25	Primi	38	> 37 weeks	1.52	48	20.8	165	99	Preeclampsia	Nil	6.2	Hyperuricemia	Emergency LSCS	LBW	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
63	29	26-30	Multi	32	< 34 weeks	1.5	53	23.6	143	94	GHT	++	6	Hyperuricemia	Elective LSCS	Early Preterm	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
64	18	18-25	Primi	36	34 - 37 weeks	1.55	62	25.8	158	99	GHT	++++	5.9	Normal	LN	IUGR	Abnormal	Absent	Present	Absent	Absent	Absent	Absent	Absent	Absent
65	20	18-25	Multi	38	> 37 weeks	1.54	50	21.1	182	108	Eclampsia	Nil	4.6	Normal	Elective LSCS	Term baby	Normal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
66	19	18-25	Primi	37	34 - 37	1.51	67	29.4	169	96	Severe preec	++++	5.8	Normal	Emergency LSCS	Term baby	Normal	Abs	Abs	Abs	Absent	Absent	Absent	Abs	Absent	Absent

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67	26	26-30	Multi	37	34-37 weeks	1.58	66	26.4	176	108	Eclampsia	Nil	5.5	Normal	LN	LBW	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
68	33	31-35	Multi	31	< 34 weeks	1.49	56	25.2	156	98	Preeclampsia	Nil	6	Hyperuricemia	Elective LSCS	Early Preterm	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
69	26	26-30	Primi	37	34-37 weeks	1.44	49	23.6	178	102	Severe preeclampsia	++++	6.5	Hyperuricemia	Emergency LSCS	LBW	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
70	23	18-25	Multi	37	34-37 weeks	1.5	56	24.9	124	92	GHT	Nil	6.3	Hyperuricemia	Elective LSCS	Term baby	Normal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
71	18	18-25	Primi	37	34-37 weeks	1.53	48	20.5	122	94	GHT	Nil	5.3	Normal	LN	Term baby	Normal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
72	24	18-25	Multi	33	< 34 weeks	1.42	52	25.8	176	104	Severe preeclampsia	++++	6.1	Hyperuricemia	Emergency LSCS	Early Preterm	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
73	21	18-25	Primi	35	34-37 weeks	1.5	45	20	180	116	Eclampsia	Nil	5.9	Normal	LN	Late Preterm	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
74	19	18-25	Primi	38	> 37 weeks	1.49	54	24.3	154	98	Preeclampsia	Nil	4.4	Normal	Emergency LSCS	LBW	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
75	32	31-35	Multi	36	34-37 weeks	1.6	72	28.1	149	82	GHT	++	6.3	Hyperuricemia	Emergency LSCS	IUGR	Abnormal	Absent	Present	Absent	Absent	Absent	Absent	Absent	Absent	Absent

76	25	18-25	Multi	39	> 37 weeks	1.56	59	24.2	138	84	GHT	Nil	6	Hyperuricemia	LN	LBW	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
77	22	18-25	Primi	38	> 37 weeks	1.47	48	22.2	184	104	Severe preeclampsia	++++	5.9	Normal	Emergency LSCS	Term baby	Normal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
78	24	18-25	Multi	34	34 - 37 weeks	1.49	53	23.9	135	86	GHT	Nil	4.7	Normal	Elective LSCS	Early Preterm	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
79	22	18-25	Primi	37	34 - 37 weeks	1.58	61	24.4	162	102	Preeclampsia	Nil	5	Normal	LN	Term baby	Normal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
80	19	18-25	Primi	36	34 - 37 weeks	1.47	59	27.3	135	83	GHT	Nil	5.8	Normal	LN	LBW	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
81	26	26-30	Multi	38	> 37 weeks	1.55	53	22.1	134	80	GHT	+	6.2	Hyperuricemia	Emergency LSCS	Term baby	Normal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
82	31	31-35	Primi	37	34 - 37 weeks	1.6	75	29.3	148	102	Preeclampsia	Nil	5.9	Normal	LN	Term baby	Normal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
83	24	18-25	Multi	38	> 37 weeks	1.44	48	23.1	135	84	GHT	Nil	6	Hyperuricemia	Elective LSCS	Term baby	Normal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
84	29	26-30	Multi	32	< 34 weeks	1.52	60	26	182	102	Severe preeclampsia	++++	5.9	Normal	Elective LSCS	Early Preterm	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
85	18	18-25	Primi	36	34 - 37	1.42	54	26.8	146	98	Preeclampsia	Nil	4.3	Normal	LN	Term baby	Normal	Abs	Abs	Abs	Absent	Absent	Absent	Abs	Absent	Absent

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86	33	31-35	Multi	33	< 34 weeks	1.6	61	23.8	148	82	GHT	Nil	5.6	Normal	Emergency LSCS	Extreme Preterm	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
87	23	18-25	Multi	37	34 - 37 weeks	1.57	55	22.3	146	84	GHT	++	4.6	Normal	Emergency LSCS	LBW	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
88	20	18-25	Primi	38	> 37 weeks	1.44	49	23.6	143	83	GHT	Nil	5.3	Normal	LN	LBW	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
89	28	26-30	Multi	38	> 37 weeks	1.53	47	20.1	156	102	Preeclampsia	Nil	4.2	Normal	Elective LSCS	Term baby	Normal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
90	25	18-25	Multi	38	> 37 weeks	1.61	77	29.7	123	87	GHT	Nil	4.9	Normal	Emergency LSCS	Term baby	Normal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
91	32	31-35	Multi	37	34 - 37 weeks	1.49	62	27.9	168	112	HELLP Syndrome	++++	7	Hyperuricemia	Emergency LSCS	LBW	Abnormal	Absent	Absent	Present	Absent	Absent	Absent	Absent	Absent	Absent	Absent
92	19	18-25	Primi	28	< 34 weeks	1.55	51	21.2	183	104	Severe preeclampsia	++++	6.2	Hyperuricemia	Emergency LSCS	Extreme Preterm	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
93	18	18-25	Primi	34	34 - 37 weeks	1.52	48	20.8	144	83	GHT	Nil	5.4	Normal	LN	Late Preterm	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
94	31	31-35	Multi	37	34 - 37 weeks	1.47	55	25.5	178	112	Severe preeclampsia	++++	5.9	Normal	Elective LSCS	Term baby	Normal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent

95	20	18-25	Primi	37	34-37 weeks	1.5	56	24.9	172	110	Severe preeclampsia	++++	6.2	Hyperuricemia	Elective LSCS	LBW	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
96	22	18-25	Multi	38	>37 weeks	1.55	64	26.6	148	89	GHT	Nil	5.4	Normal	Emergency LSCS	LBW	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
97	23	18-25	Multi	37	34-37 weeks	1.43	52	25.4	149	92	GHT	Nil	4.6	Normal	Elective LSCS	Term baby	Normal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
98	26	26-30	Primi	38	>37 weeks	1.56	55	22.6	162	110	Preeclampsia	++	4.8	Normal	LN	Term baby	Normal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
99	29	26-30	Multi	32	<34 weeks	1.58	60	24	145	94	GHT	Nil	5.3	Normal	Elective LSCS	Extreme Preterm	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
100	24	18-25	Multi	34	34-37 weeks	1.49	53	23.9	154	93	GHT	Nil	4.7	Normal	Elective LSCS	Early Preterm	Abnormal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent