"PROSPECTIVE CASE-CONTROL STUDY ON MATERNAL OPHTHALMIC ARTERY DOPPLER VELOCIMETRY IN EVALUATION OF PRE-ECLAMPSIA"

By

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Dissertation submitted to

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In partial fulfilment of the requirements for

MASTER OF SURGERY

OBSTETRICS AND GYNECOLOGY

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SL No.	ABBREVATION	EXPANSION
1.	PIH	PREGNANCY-INDUCED HYPERTENSION
2.	CT	COMPUTED TOMOGRAPHY
3.	MRI	MAGNETIC RESONANCE IMAGING
4.	TCD	TRANSCRANIAL DOPPLER
5.	RI	RESISTIVITY INDEX
6.	PI	PULSATILITY INDEX
7.	HELLP	(HEMOLYSIS, ELEVATED LIVER ENZYMES, AND LOW PLATELETS) SYNDROME.
8.	IUGR	INTRAUTERINE GROWTH RESTRICTION
9.	PE	PRE-ECLAMPSIA
10.	BP	BLOOD PRESSURE
11.	LMIC	LOW- AND MIDDLE-INCOME COUNTRIES

12.	LBW	LOW BIRTH WEIGHT.
13.	sFlt-1	SOLUBLE FMS-LIKE TYROSINE KINASE-1.
14.	sEng	SOLUBLE ENDOGLIN
15.	VEGF	VASCULAR ENDOTHELIAL GROWTH FACTOR
16.	PIGF	PLACENTAL GROWTH FACTOR
17.	ROS	REACTIVE OXYGEN SPECIES
18.	NO	NITRIC OXIDE
19.	TNF- α	TUMOUR NECROSIS FACTOR-ALPHA
20.	PRES	POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME
21.	CBF	CEREBRAL BLOOD FLOW
22.	BBB	BLOOD-BRAIN BARRIER
23.	MRA	MAGNETIC RESONANCE ANGIOGRAPHY
24.	MCA	MIDDLE CEREBRAL ARTERY
25.	PCA	POSTERIOR CEREBRAL ARTERY

26.	ICA	INTERNAL CAROTID ARTERY
27.	PSV	PEAK SYSTOLIC VEOLCITY
28.	EDV	END-DIASTOLIC VELOCITY
29.	SDR	SYSTOLIC-DIASTOLIC RATIO
30.	GA	GESTATIONAL AGE
31.	MAP	MEAN ARTERIAL PRESSURE
32.	OAD	OPHTHALMIC ARTERY DOPPLER
33.	TAMAX	TIME-AVERAGED MAXIMUM VELOCITY,

ABSTRACT

BACKGROUND AND OBJECTIVE

Preeclampsia (PE) is a lethal hypertensive disorder that significantly contributes to maternal as well as fetal morbidity besides mortality, especially in low-resource settings like in India. Early diagnosis and management are crucial in preventing adverse maternal and fetal outcomes. Preeclampsia causes cerebrovascular endothelial dysfunction and impairs cerebral autoregulation due to systemic hypertension. So, monitoring cerebrovascular alterations is crucial for preventing severe neurological complications. In this study, we have used maternal ophthalmic artery Doppler (OAD) as a key tool for monitoring hemodynamic parameters and severity of the disease in preeclampsia and normotensive pregnant women

METHODOLOGY

A prospective observational case-control study took place in a medical facility that provided tertiary care from April 2023 to March 2025, including 170 pregnant women (85 preeclampsia cases and 85 normotensive controls). OAD metrics, such as pulsatility index (PI), resistivity index (RI), peak systolic velocity (PSV), and end-diastolic velocity (EDV), were measured using high-resolution ultrasound with a 7-10MHz transducer. Data were analysed using SPSS version 20, and cutoff values for determining preeclampsia severity were determined using ROC curve analysis.

RESULTS

Preeclampsia patients demonstrated significantly higher RI and PI values compared to controls(p=0.001). ROC analysis identified RI >0.72 and a strong predictor of preeclampsia severity (sensitivity 82.3%, Specificity 79.4%). Increased ophthalmic artery Doppler indices correlated with adverse maternal and fetal consequences, including intrauterine growth restriction (IUGR) as well as preterm birth.

CONCLUSION

This prospective observational case-control study demonstrates the maternal ophthalmic artery doppler velocimetry as a valuable tool in evaluating and predicting preeclampsia in pregnancy. Increased resistance in the right ophthalmic artery and lower pulsatility suggest cerebrovascular dysfunction and impaired autoregulation in pre-eclampsia. As it serves as a promising surrogate marker for cerebrovascular dysfunction, its integration into routine obstetric evaluation may aid in early detection and risk stratification.

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INTRODUCTION

Pre-eclampsia (P.E.) is a complex and life-threatening hypertensive disorder that affects pregnant women, typically manifesting after 20 weeks of gestation.[1] It is characterized by hypertension and multi-system involvement, which, if left untreated, can progress to eclampsia, a severe manifestation that marks the endpoint of the disease spectrum. This progression significantly contributes to maternal mortality and morbidity, especially in low-resource settings such as India, where pre-eclampsia remains a leading cause of adverse maternal and fetal outcomes. Pregnancy-induced hypertension (PIH), a hallmark of pre-eclampsia, is a critical determinant of maternal mortality, further emphasizing the urgent need for improved diagnostic and management strategies for this condition.[1,2]

The pathophysiology of pre-eclampsia is complex and multifactorial, involving abnormal placentation, systemic inflammation, endothelial dysfunction, and immune maladaptation. One of the central theories is the incomplete remodeling of the spiral arteries in the placenta, leading to placental ischemia and the release of anti-angiogenic factors such as soluble fms-like tyrosine kinase-1 (sFlt-1) and soluble endoglin (sEng). These factors disrupt maternal endothelial homeostasis, contributing to widespread vasoconstriction, hypertension, and end-organ dysfunction.[2]

One of the most concerning aspects of pre-eclampsia is its neurological sequelae, which include stroke, eclampsia, and symptoms such as headache and visual disturbances. These complications are largely attributed to cerebrovascular endothelial dysfunction and impaired cerebral autoregulation caused by systemic hypertension. The ability to monitor and

assess cerebrovascular alterations in pre-eclampsia is crucial for preventing severe neurological outcomes.[3] However, real-time assessment of cerebrovascular hemodynamics using advanced imaging techniques such as computed tomography (CT) and magnetic resonance imaging (MRI) is limited by logistical, financial, and safety concerns. CT imaging involves exposure to ionizing radiation, which is undesirable during pregnancy, while MRI is not only expensive but also logistically challenging to perform in acute and longitudinal scenarios in obstetric care.

Transcranial Doppler (TCD) ultrasonography offers a real-time, non-invasive alternative for assessing cerebral hemodynamics, but its utility is constrained to the evaluation of large-caliber intracranial vessels, such as the middle cerebral artery. These vessels may not adequately reflect the microvascular changes and pathological alterations occurring in pre-eclampsia.[4] Consequently, there is a growing interest in exploring alternative vascular territories that can serve as surrogates for cerebrovascular evaluation in pre-eclampsia.

The ophthalmic artery, due to its embryological, anatomical, and functional similarities with the cerebral vasculature, presents itself as a promising candidate.[5] Doppler ultrasonography of the ophthalmic artery has been extensively employed in non-obstetric conditions such as glaucoma, systemic atherosclerosis, multiple sclerosis, and heart failure to study vascular changes. Its non-invasive nature, affordability, and ability to provide real-time hemodynamic data make it a potentially invaluable tool for obstetric use.[4]

In pre-eclampsia, maternal ophthalmic artery Doppler velocimetry has demonstrated alterations in key hemodynamic parameters such as the resistivity index (RI) and pulsatility index (PI). These changes reflect underlying cerebrovascular dysfunction and provide insights into disease severity.[5] Several studies have reported increased RI and PI in pre-

eclamptic women compared to normotensive pregnant women, suggesting compromised downstream perfusion and increased vascular resistance.[4,5]

A critical advantage of ophthalmic artery Doppler over traditional cerebrovascular imaging is its ease of accessibility and potential for early diagnosis. By detecting hemodynamic alterations before clinical symptoms of severe pre-eclampsia appear, ophthalmic artery Doppler may facilitate timely interventions, reducing the risk of complications such as eclampsia, intracranial hemorrhage, and stroke.[2,3]

Another important aspect of maternal ophthalmic artery Doppler evaluation is its ability to differentiate between mild and severe pre-eclampsia. Severe pre-eclampsia is often associated with more pronounced cerebrovascular changes, reflected in further elevated RI and PI values.[5] By stratifying pre-eclamptic patients based on Doppler parameters, clinicians can tailor management strategies and optimize maternal and fetal outcomes.

Furthermore, ophthalmic artery Doppler has shown promise in predicting adverse maternal and neonatal outcomes. Studies have suggested that abnormal ophthalmic artery Doppler indices correlate with higher rates of maternal complications such as eclampsia, cerebrovascular accidents, and HELLP (hemolysis, elevated liver enzymes, and low platelets) syndrome.[4,5] Similarly, fetal complications, including intrauterine growth restriction (IUGR), preterm birth, and perinatal mortality, have been linked to altered maternal ophthalmic artery Doppler findings.[2]

Despite its potential, the clinical application of ophthalmic artery Doppler in pre-eclampsia remains underutilized. Standardized cut-off values for Doppler parameters have not been universally established, limiting its widespread adoption in obstetric practice. Further research is needed to validate its role as a predictive tool and to integrate it into existing pre-eclampsia screening protocols.

This prospective case-control study aims to bridge this gap by evaluating maternal ophthalmic artery Doppler velocimetry as a surrogate marker for cerebrovascular hemodynamics in women with pre-eclampsia. By leveraging this point-of-care imaging modality, we seek to establish a non-invasive, cost-effective, and accessible method for assessing cerebrovascular changes, which could have far-reaching implications for the management and prognosis of pre-eclampsia.

The study will compare ophthalmic artery Doppler parameters between pre-eclamptic and normotensive pregnant women, assessing their correlation with disease severity and maternal-fetal outcomes. By identifying characteristic Doppler patterns in mild and severe pre-eclampsia, this research aims to enhance early detection and risk stratification of the condition.

Additionally, the study will explore the role of ophthalmic artery Doppler in monitoring disease progression and response to treatment. Serial Doppler measurements throughout pregnancy may provide valuable insights into the dynamic changes in cerebral hemodynamics associated with pre-eclampsia, guiding clinical decision-making and improving patient care.

Incorporating ophthalmic artery Doppler into routine obstetric evaluation could revolutionize the approach to pre-eclampsia management. Its integration alongside other established predictive markers such as uterine artery Doppler and biochemical assays may enhance the accuracy of early diagnosis and intervention.

Ultimately, this investigation not only underscores the potential of ophthalmic artery Doppler velocimetry in obstetric care but also sets the stage for its integration into routine clinical practice for the early detection and management of pre-eclampsia. By providing a non-invasive, real-time assessment of cerebrovascular changes, this modality could

significantly improve maternal and fetal outcomes, reducing the burden of pre-eclampsia-related complications worldwide.

AIMS AND OBJECTIVES

Aim:

To determine the association of maternal ophthalmic artery Doppler and hemodynamics of cerebrovasculature in pregnant women with pre-eclampsia.

Objectives:

Primary objective:

To investigate changes in the hemodynamics of cerebral vasculature by the study of maternal ophthalmic artery doppler.

Secondary objective:

- To compare ophthalmic artery doppler parameters between pre-eclamptic and normotensive patients.
- To compare blood pressure and ophthalmic artery doppler metrics in mild and severe Pre-eclampsia
- Maternal and fetal outcome.

REVIEW OF LITERATURE

Definition of pre-eclampsia

Pre-eclampsia is a pregnancy-specific multisystem disorder characterised by new-onset hypertension and proteinuria or other systemic complications occurring after 20 weeks of gestation in a previously normotensive woman.[1,6] It is associated with significant maternal and fetal morbidity and mortality, particularly in low- and middle-income countries. Pre-eclampsia is primarily attributed to abnormal placentation, leading to widespread endothelial dysfunction, systemic inflammation, and impaired vascular autoregulation.

Classification

Pre-eclampsia is broadly classified based on severity and timing of onset:

By Severity:[7]

1. Mild Pre-Eclampsia:
    - Blood pressure (BP) ≥ 140/90 mmHg but < 160/110 mmHg.
    - Proteinuria ≥ 300 mg in a 24-hour urine collection or protein/creatinine ratio ≥ 0.3.
    - Minimal or no systemic symptoms.
2. Severe Pre-Eclampsia:
    - BP ≥ 160/110 mmHg.
    - Proteinuria ≥ 5 g in a 24-hour urine collection or significant systemic symptoms, such as persistent headache, visual disturbances, epigastric pain, or evidence of

organ dysfunction (e.g., elevated liver enzymes, thrombocytopenia, pulmonary oedema).

By Onset:[8]

1. Early-Onset Pre-Eclampsia: Occurs before 34 weeks of gestation and is associated with more severe complications and higher risks of maternal and fetal morbidity.
2. Late-Onset Pre-Eclampsia: Develops at or after 34 weeks and is often milder, with better maternal and neonatal outcomes.

Diagnostic Criteria[9]

The diagnosis of pre-eclampsia requires the presence of hypertension and one or more of the following:

1. Hypertension: Systolic BP ≥ 140 mmHg or diastolic BP ≥ 90 mmHg on at least two occasions, four hours apart, after 20 weeks of gestation.
2. Severe hypertension: Systolic BP ≥ 160 mmHg or diastolic BP ≥ 110 mmHg.
3. Proteinuria: Protein excretion ≥ 300 mg in a 24-hour urine sample, Protein/creatinine ratio ≥ 0.3, Urine dipstick reading of ≥ +1 (if quantitative methods are unavailable).
4. Absence of Proteinuria with End-Organ Dysfunction: In the absence of significant proteinuria, pre-eclampsia can be diagnosed if hypertension is accompanied by any of the following:
    - Hematological Abnormalities: Thrombocytopenia (platelet count < 100,000/μL).
    - Renal Dysfunction: Serum creatinine > 1.1 mg/dL or a doubling of baseline values.

- Hepatic Dysfunction: Elevated liver enzymes (transaminases) to twice the upper limit of normal.
- Neurological Symptoms: Persistent headache, visual disturbances, or seizures.
- Pulmonary Oedema: Evidence of respiratory compromise.

Global Epidemiology

Pre-eclampsia affects approximately 2–8% of pregnancies worldwide, representing a significant cause of maternal and perinatal morbidity and mortality.[10] It accounts for nearly 10–15% of maternal deaths in low- and middle-income countries (LMICs), where limited access to healthcare and antenatal services exacerbates its impact. The prevalence is higher in LMICs due to factors such as delayed diagnosis, poor nutritional status, and inadequate medical interventions, highlighting its role as a pressing global health concern.[11]

Maternal Impact

Pre-eclampsia is a leading cause of maternal mortality globally, particularly in resource-poor settings. Complications such as eclampsia, HELLP syndrome, renal failure, and cerebrovascular accidents contribute to significant morbidity and mortality. Women who survive severe pre-eclampsia face long-term health risks, including chronic hypertension and cardiovascular diseases, making it a condition with both acute and chronic health implications.[12]

Fetal and Neonatal Impact

Pre-eclampsia is a major cause of adverse fetal outcomes, including fetal growth restriction (FGR), preterm birth, and perinatal death. Neonates born to pre-eclamptic mothers are more likely to suffer from low birth weight (LBW) and complications such as respiratory

distress syndrome and neonatal sepsis. Placental insufficiency due to pre-eclampsia also has long-term developmental consequences for children, further emphasizing the need for effective prevention and management strategies.[13-15]

Pre-Eclampsia in India

India bears a disproportionately high burden of pre-eclampsia, with an estimated prevalence of 8–10% of pregnancies.[16] The condition is a leading cause of maternal deaths in India, contributing to nearly 20% of maternal mortality in some regions.[17] Adverse fetal outcomes, including stillbirths and neonatal deaths, are common, primarily due to placental insufficiency and preterm deliveries.[15] Socioeconomic disparities, inadequate antenatal care, and high rates of nutritional deficiencies contribute significantly to the burden of pre-eclampsia in the country.[18]

A study conducted by Shandilya & Rani (2023) found a prevalence of 6.2% among 500 pregnant women, reflecting a moderate occurrence within the population under investigation.[19] In contrast, Tandur et al. (2024) reported a significantly higher prevalence of 15.67% in a sample of 583 women, suggesting that certain groups may be at a greater risk.[20] Furthermore, the National Family Health Survey, as noted by Das et al. (2024), indicated a prevalence of 3.6% for pre-eclampsia or eclampsia among a much larger cohort of 190,797 women, providing a broader national perspective on the condition's occurrence.[21]

Contributing Risk Factors in India

Several risk factors amplify the burden of pre-eclampsia in India. Early marriage and adolescent pregnancies increase the risk of hypertensive disorders during pregnancy. Poor

maternal nutrition, including widespread anemia and deficiencies in calcium and folate, further predispose women to pre-eclampsia. Additionally, inadequate healthcare access and delayed recognition of symptoms in rural and underserved areas exacerbate adverse maternal and fetal outcomes.[18]

Relevance in existing literature:

Several risk factors contribute to the development of pre-eclampsia in India, including obesity, inadequate dietary intake, and lack of iron and folic acid supplementation (Shandilya & Rani, 2023; Das et al., 2024).[19,21] Obesity, in particular, has been identified as a significant risk factor due to its association with metabolic disturbances and increased systemic blood pressure, both of which can exacerbate the condition. Additionally, primigravida status (first-time pregnancies) and lower socioeconomic status also increase the likelihood of developing pre-eclampsia, as these women often face barriers to accessing proper antenatal care and healthcare services (Chauhan & Patel, 2023).[22]

The impact of pre-eclampsia on maternal and fetal health is considerable. Common fetal complications include low birth weight and preterm birth. For example, Shandilya & Rani (2023) found that nearly 48.4% of infants born to mothers with pre-eclampsia had low birth weight.[19] These complications often result in long-term health risks for the newborn, including developmental delays and chronic health conditions. Moreover, maternal complications, such as eclampsia and HELLP syndrome, can have severe consequences, including maternal mortality in extreme cases (Chauhan & Patel, 2023).[22] This emphasizes the critical importance of early detection and timely management to mitigate the risks associated with pre-eclampsia.

Pathophysiology of Pre-Eclampsia

Pre-eclampsia is a complex, multisystem disorder characterised by hypertension and end-organ damage during pregnancy. Its pathophysiology is rooted in abnormal placental development and widespread endothelial dysfunction, which result in systemic inflammation, oxidative stress, and impaired vascular regulation.

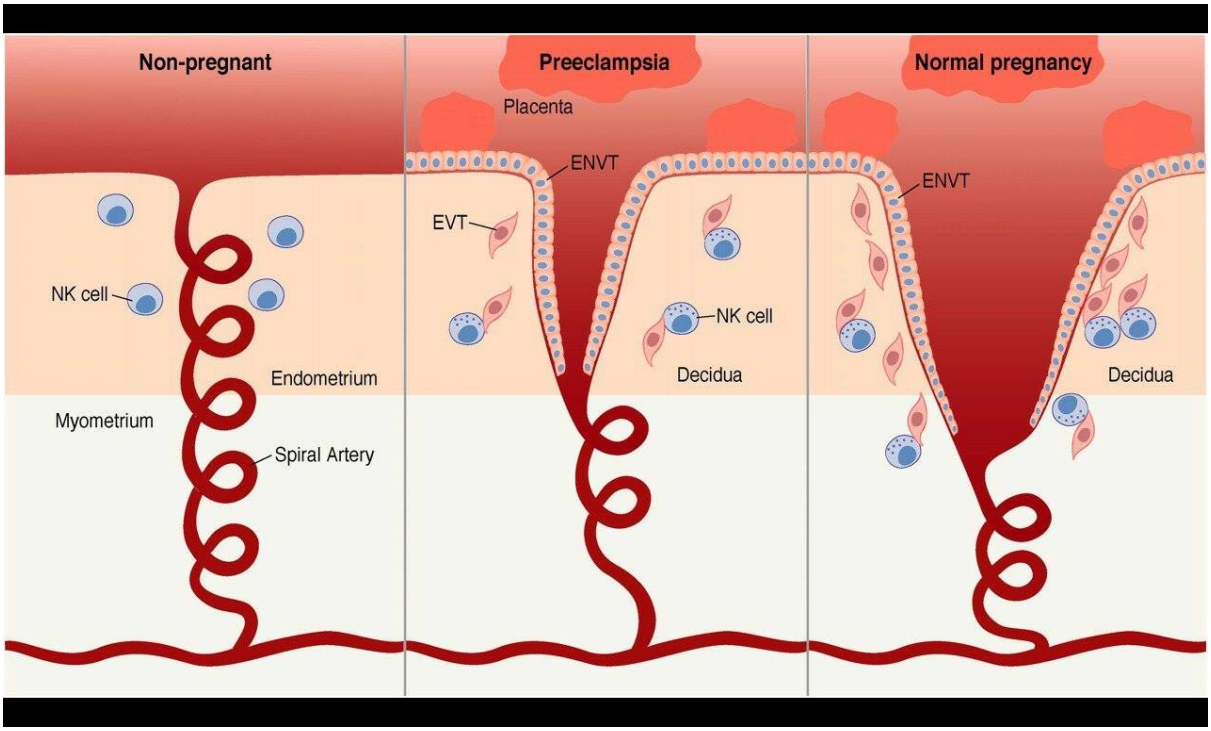


Figure 1: Pathophysiology of pre-eclampsia

Impaired Placental Development

The initial trigger for pre-eclampsia lies in abnormal placentation during early pregnancy. Normal placental development involves the invasion of trophoblasts into the maternal spiral arteries, transforming them into low-resistance, high-capacity vessels capable of supporting the growing fetus. In pre-eclampsia, this transformation is incomplete, leading to shallow trophoblast invasion and the persistence of high-resistance spiral arteries.

As a result, placental perfusion is compromised, causing ischemia and hypoxia. The ischemic placenta releases anti-angiogenic factors such as soluble fms-like tyrosine kinase-1 (sFlt-1) and soluble endoglin (sEng) into the maternal circulation. These factors disrupt the balance of angiogenic and anti-angiogenic signals, particularly vascular endothelial growth factor (VEGF) and placental growth factor (PlGF), which are essential for maintaining vascular health.[23,24]

Endothelial Dysfunction

Endothelial dysfunction is central to the pathophysiology of pre-eclampsia and is driven by placental-derived factors and systemic inflammation. The imbalance of angiogenic signals causes widespread damage to the maternal vascular endothelium, leading to increased vascular permeability, reduced vasodilation, and pro-thrombotic tendencies.[23-25] Key mechanisms of endothelial dysfunction include:

- Oxidative Stress: Hypoxia and reperfusion injury in the placenta generate reactive oxygen species (ROS), which damage endothelial cells and exacerbate inflammation.
- Reduced Nitric Oxide (NO) Bioavailability: NO, a critical vasodilator, is diminished due to oxidative stress and the antagonistic effects of sFlt-1. This contributes to systemic vasoconstriction and hypertension.
- Inflammatory Activation: Systemic inflammation triggered by abnormal placentation increases circulating cytokines, such as tumour necrosis factor-alpha (TNF-α) and interleukins, further impairing endothelial function.

Systemic Effects

- Hypertension: Due to vasoconstriction and increased vascular resistance.

- Proteinuria: Resulting from endothelial damage in the renal glomeruli and increased vascular permeability.
- Organ Damage: Endothelial dysfunction and microvascular injury affect multiple organs, including the brain (cerebral oedema, seizures), liver (elevated enzymes, haemorrhage), and lungs (pulmonary oedema).

Role of Angiogenic Imbalance

The disrupted balance of pro-angiogenic (VEGF, PlGF) and anti-angiogenic factors (sFlt-1, sEng) not only causes endothelial damage but also contributes to placental insufficiency. This imbalance is a hallmark of pre-eclampsia and serves as a potential biomarker for early detection and monitoring of the disease.[25]

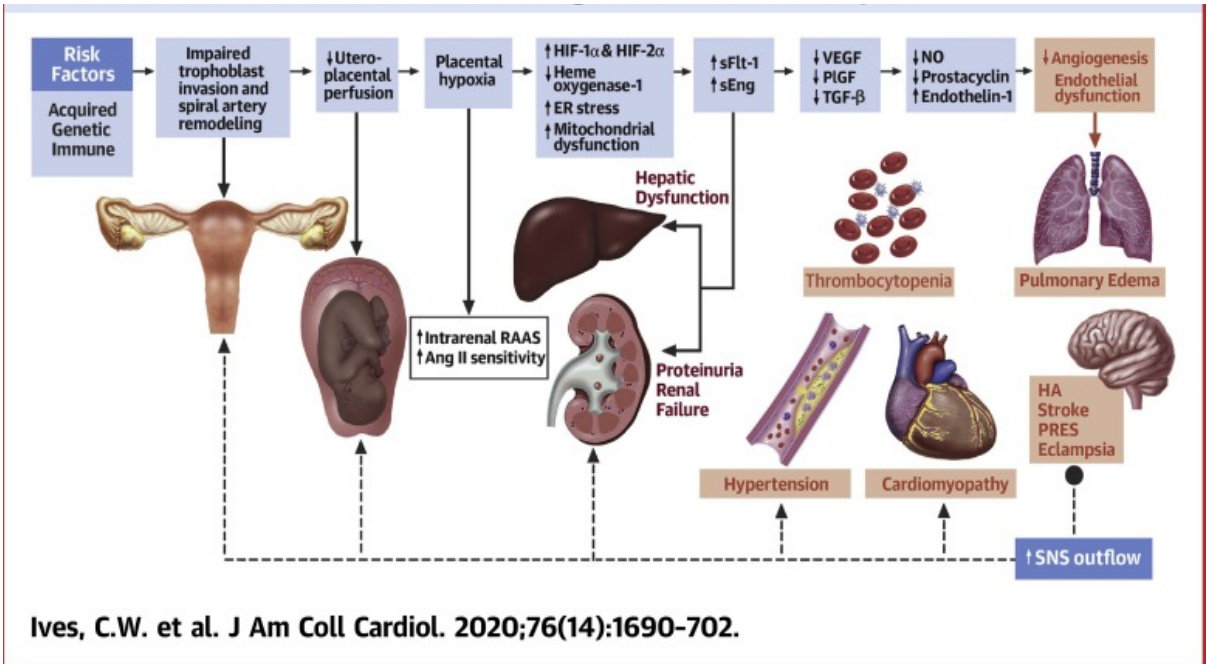


Figure 2: Pathogenesis of pre-eclampsia

Maternal Neurological Complications in Pre-Eclampsia:

Pre-eclampsia is associated with a range of neurological complications that significantly contribute to maternal morbidity and mortality. These complications arise from severe hypertension, endothelial dysfunction, and impaired cerebrovascular autoregulation. The most common neurological sequelae include stroke, eclampsia, and visual disturbances, each with distinct mechanisms and clinical implications.[26]

Stroke

Stroke is a severe and life-threatening complication of pre-eclampsia, resulting from the interplay of hypertension, endothelial dysfunction, and coagulopathy. It can manifest as ischemic or haemorrhagic stroke, with haemorrhagic strokes being more common in the context of severe hypertension. Impaired cerebral autoregulation leads to excessive perfusion pressure, causing vascular rupture and intracranial haemorrhage. Additionally, hypercoagulability associated with pre-eclampsia predisposes women to thrombotic events, increasing the risk of ischemic stroke.[27]

Neurological symptoms such as severe headache, altered mental status, focal neurological deficits, or seizures often precede stroke. Rapid diagnosis and management are essential to prevent irreversible damage and reduce maternal mortality.

Eclampsia

Eclampsia is a defining neurological complication of pre-eclampsia, characterised by the onset of generalised tonic-clonic seizures in a woman with pre-eclampsia. The pathophysiology involves cerebral oedema, hyperperfusion, and endothelial dysfunction

leading to disruption of the blood-brain barrier. These changes result in cortical irritation and seizure activity.

Eclampsia remains a leading cause of maternal death globally, particularly in low-resource settings where delayed diagnosis and inadequate management are common. Seizures can lead to additional complications, including aspiration pneumonia, hypoxia, and brain injury, necessitating prompt treatment with magnesium sulphate and blood pressure control.

Visual Disturbances

Visual disturbances are frequent but often underreported neurological sequelae of pre-eclampsia. They result from hypertensive changes affecting the retinal and occipital vasculature.[28] Common visual symptoms include:

- Blurred Vision: Due to retinal oedema or ischemia.
- Scotomas (Blind Spots): Resulting from retinal involvement or posterior cortical dysfunction.
- Photopsia (Flashes of Light): Caused by retinal irritation or vascular abnormalities.
- Cortical Blindness: A rare but reversible complication due to posterior reversible encephalopathy syndrome (PRES). PRES is characterised by vasogenic oedema affecting the posterior cerebral hemispheres and is closely linked to hypertensive crises.

Pathophysiological Basis

The neurological sequelae in pre-eclampsia share a common pathological basis rooted in cerebrovascular endothelial dysfunction. Severe hypertension leads to dysregulation of

cerebral blood flow, increased permeability of the blood-brain barrier, and subsequent vasogenic oedema or haemorrhage. Additionally, systemic inflammation and oxidative stress further exacerbate vascular and neuronal damage.[26,29]

Pre-eclampsia's neurological complications stem from the pathological interplay between systemic hypertension, impaired cerebral autoregulation, and cerebrovascular dysfunction. These interlinked processes disrupt normal cerebrovascular physiology, leading to significant maternal morbidity.[29]

Systemic Hypertension

Systemic hypertension is a hallmark of pre-eclampsia and a primary contributor to cerebrovascular dysfunction. In normal physiology, cerebral autoregulation maintains consistent cerebral blood flow (CBF) across a range of blood pressures by modulating cerebrovascular resistance. However, in pre-eclampsia, chronic hypertension disrupts this mechanism, pushing the limits of autoregulatory capacity and predisposing the brain to both hyperperfusion and hypoperfusion.

Cerebral Autoregulation in Pre-Eclampsia[30]

Cerebral autoregulation is the process by which small resistance arteries adjust vascular tone to sustain adequate CBF, despite fluctuations in systemic blood pressure. In pre-eclampsia, severe or rapidly escalating hypertension overwhelms this system, leading to:

- Hyperperfusion: When blood pressure exceeds the upper limit of autoregulation, excessive cerebral blood flow causes endothelial damage, increased vascular permeability, and vasogenic oedema.
- Hypoperfusion: Conversely, if vascular tone fails to maintain adequate perfusion, focal ischemia and infarction may occur.

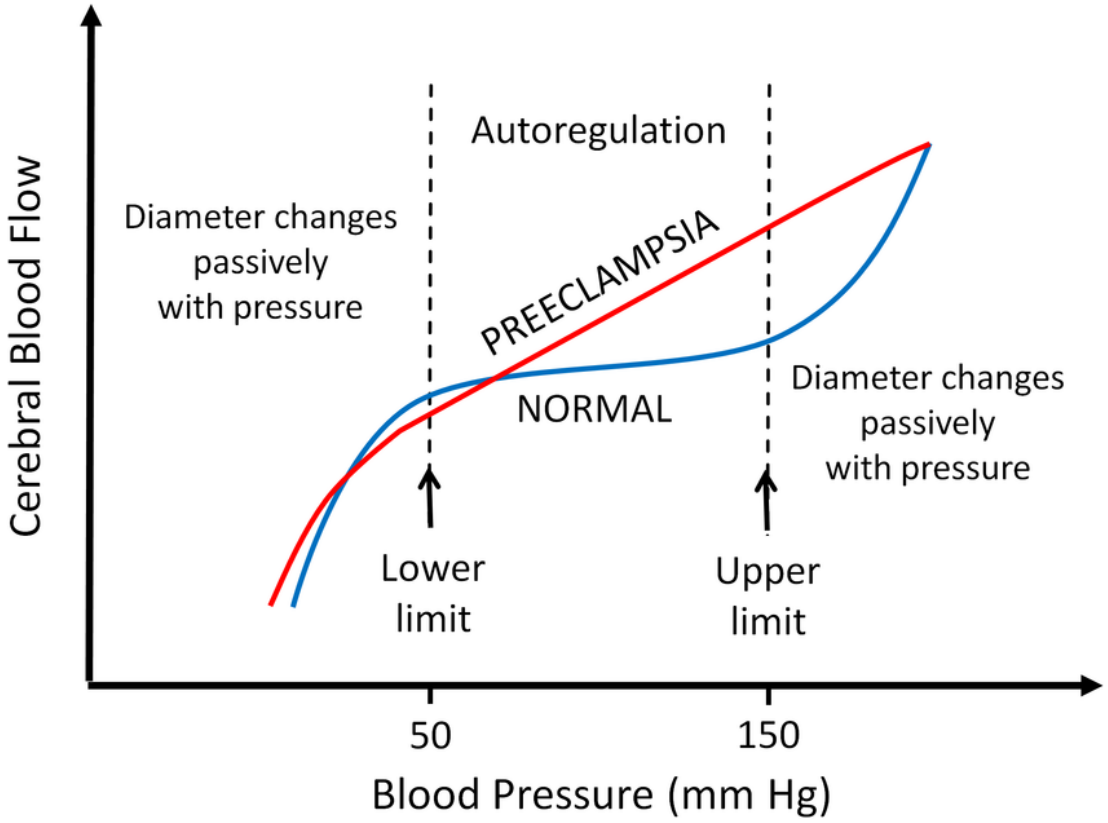


Figure 3: Cerebral Blood Flow curve in Normal and Pre-eclampsia conditions

Cerebrovascular Dysfunction

The disruption of cerebral autoregulation in pre-eclampsia results in significant cerebrovascular dysfunction, which manifests as endothelial damage, blood-brain barrier (BBB) disruption, and vasogenic oedema.[31,32]

- Endothelial Dysfunction: Endothelial cells in the cerebral vasculature are particularly sensitive to oxidative stress, inflammation, and the imbalance of angiogenic factors seen in pre-eclampsia. This dysfunction reduces the production of nitric oxide (NO), a critical vasodilator, further impairing cerebrovascular tone.

- Blood-Brain Barrier Disruption: The breakdown of the BBB allows plasma proteins and fluid to leak into the interstitial space, exacerbating cerebral oedema. This contributes to symptoms such as headache, visual disturbances, and seizures.
- Vasogenic Oedema and Posterior Reversible Encephalopathy Syndrome (PRES): Impaired autoregulation and hyperperfusion are key drivers of PRES, a condition characterised by reversible vasogenic oedema, particularly in the posterior cerebral regions. PRES is closely associated with hypertensive crises in pre-eclampsia and can present with seizures, altered mental status, and cortical blindness.
- Ischemia and Infarction: Chronic cerebrovascular dysfunction, compounded by hypercoagulability, increases the risk of ischemic strokes in pre-eclampsia. Small vessel occlusions and large-vessel thrombotic events are common in severe cases.

History of imaging techniques for pre-eclampsia

The history of imaging techniques in pre-eclampsia traces a path from the early use of basic clinical observation to the more sophisticated and specialized imaging technologies employed today. Pre-eclampsia, as a pregnancy-related hypertensive disorder, has long been a subject of concern due to its potentially severe complications for both the mother and fetus.

Early Clinical Observations and Monitoring

Historically, the diagnosis of pre-eclampsia was primarily based on clinical symptoms, including hypertension and proteinuria, with limited use of imaging. Physicians relied heavily on physical exams and laboratory tests to identify pre-eclampsia and its complications. The first clinical descriptions of pre-eclampsia date back to the 19th century, and while the disease was recognized, diagnostic techniques were rudimentary. Early

management focused largely on symptom control and general observation, without any advanced imaging technologies.

The Emergence of Ultrasonography

The introduction of ultrasonography in the 1950s revolutionized the ability to visualize internal structures during pregnancy, including the assessment of the fetus and maternal organs. Initially, ultrasonography was primarily used for fetal monitoring, with a focus on assessing fetal growth, position, and gestational age. However, as research progressed, the use of Doppler ultrasonography began to gain attention for its potential in evaluating maternal hemodynamics. Doppler studies, which measure the velocity of blood flow in various vessels, were later adapted to assess uterine and placental blood flow, offering valuable information about the vascular changes that occur in pre-eclampsia.[33,34]

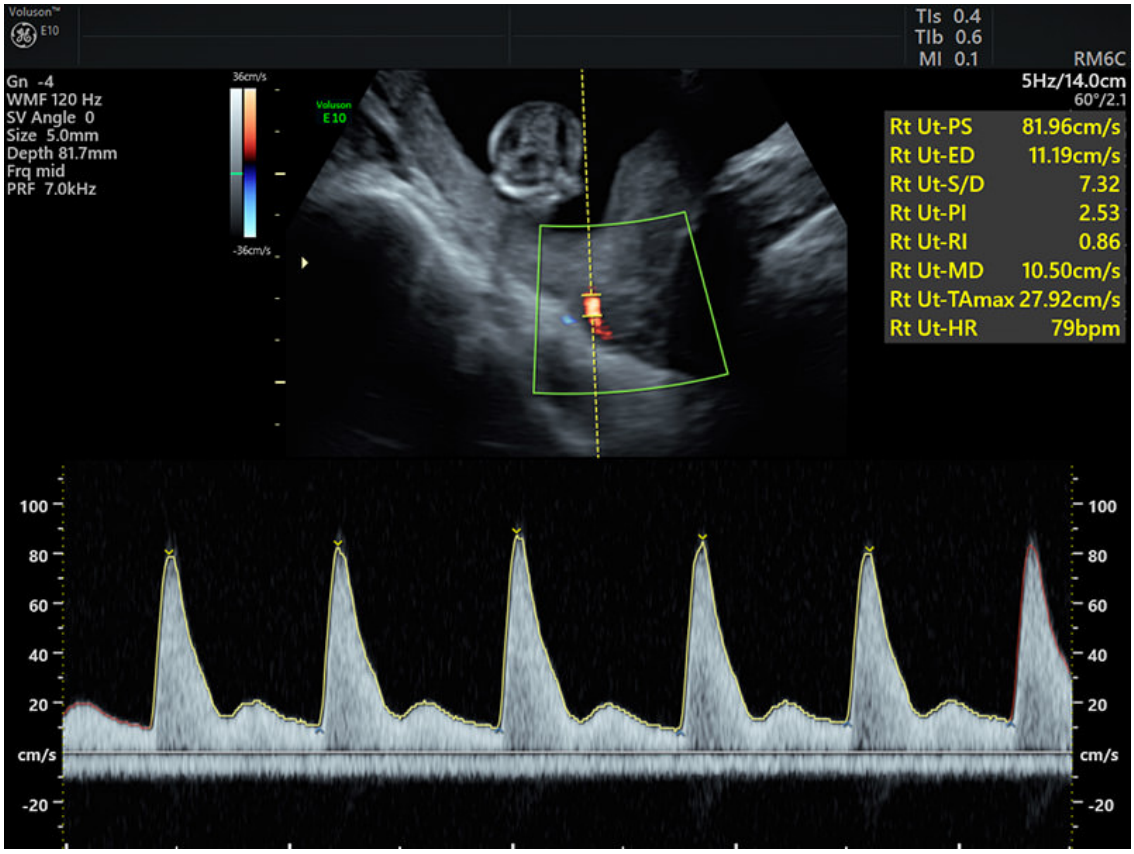


Figure 4: Pre-eclampsia screening by Ultrasound

Doppler Ultrasonography and Early Vascular Assessments

By the 1980s and 1990s, Doppler ultrasonography became a more integral part of managing high-risk pregnancies. The technique was increasingly used to assess maternal vascular changes in pre-eclampsia, particularly the resistance to blood flow in uterine and placental arteries. Studies demonstrated that Doppler indices such as the resistance index (RI) and pulsatility index (PI) could help identify abnormal placental blood flow, a key characteristic of pre-eclampsia. As Doppler ultrasonography proved effective in predicting adverse pregnancy outcomes, its use expanded to monitoring maternal blood flow and early identification of pre-eclampsia in at-risk populations.[33]

Advances in Cerebrovascular Imaging

As the understanding of the neurological complications of pre-eclampsia grew, there was increasing interest in using imaging techniques to assess the cerebrovascular system. Pre-eclampsia is known to impact cerebral circulation, often leading to conditions such as stroke, visual disturbances, and eclampsia. Traditional imaging methods like computed tomography (CT) and magnetic resonance imaging (MRI) were initially employed to evaluate cerebral changes in pre-eclampsia; however, the limitations of these techniques in pregnant women became apparent. CT scans involve ionizing radiation, which is contraindicated during pregnancy, and MRI, while safer, requires specialized equipment and is not easily accessible for real-time monitoring.[35-37]

The Advent of Transcranial Doppler (TCD)

In the 1990s and early 2000s, the development of transcranial Doppler (TCD) ultrasonography provided a new approach to monitoring cerebral hemodynamics in pre-eclampsia. TCD allows for the real-time assessment of blood flow in the large intracranial arteries, offering insights into the cerebrovascular changes associated with pre-eclampsia.

However, TCD is limited in its ability to assess smaller vessels and detect subtle cerebrovascular alterations that might occur in the early stages of pre-eclampsia. Despite these limitations, TCD continues to be a valuable tool in the assessment of cerebrovascular complications in pre-eclampsia.[38]

Ophthalmic Artery Doppler: A New Frontier

In recent years, ophthalmic artery Doppler studies have emerged as a promising alternative for assessing cerebral hemodynamics in pre-eclampsia. The ophthalmic artery shares embryological, anatomical, and functional similarities with the cerebral vasculature, making it an ideal candidate for evaluating cerebrovascular changes associated with pre-eclampsia. Doppler ultrasonography of the ophthalmic artery allows for a non-invasive, real-time assessment of blood flow, reflecting systemic hemodynamics that may be indicative of pre-eclampsia-related cerebrovascular alterations. [39]

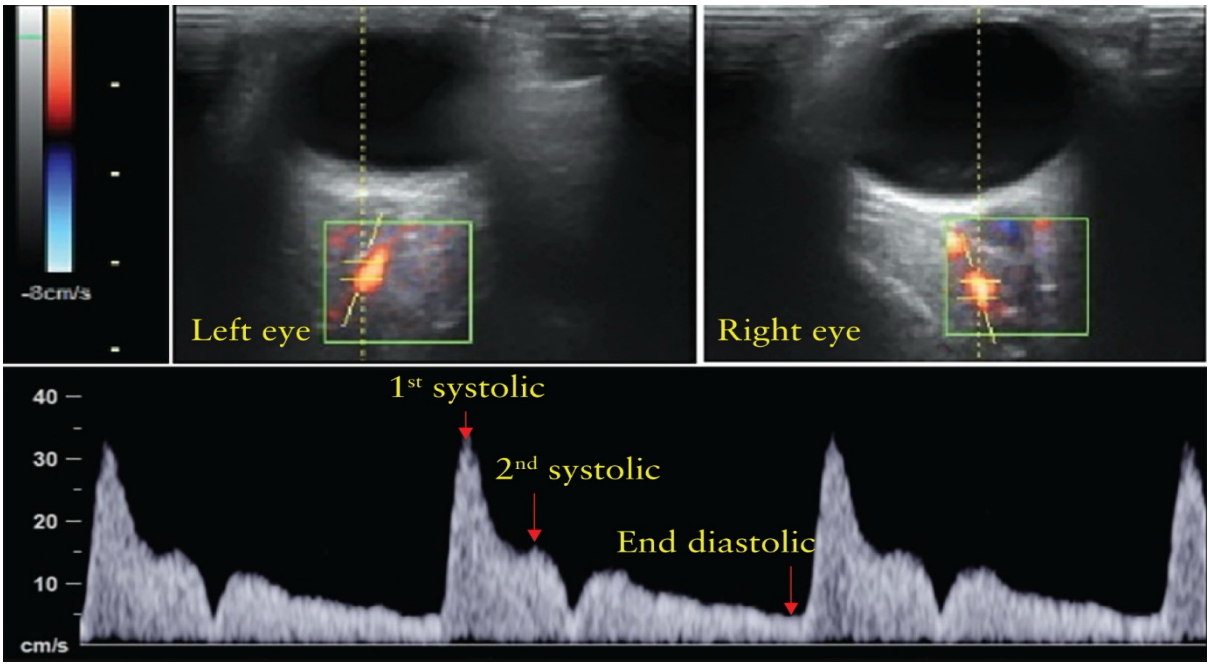


Figure 5: Ultrasound images of left and right orbits with color flow demonstration of left and right ophthalmic arteries. At the bottom is flow velocity waveform from ophthalmic artery obtained by pulsed-wave Doppler, showing first and second peaks of systolic velocity and end-diastolic velocity

Current Trends and Future Directions

Today, imaging techniques such as Doppler ultrasonography, including ophthalmic artery Doppler studies, are considered essential tools in the management of pre-eclampsia. These non-invasive, real-time assessments have enhanced early detection and monitoring capabilities, allowing healthcare providers to identify and manage pre-eclampsia more effectively. As technology continues to evolve, there is growing interest in integrating more advanced imaging techniques, including three-dimensional (3D) Doppler ultrasound and more portable imaging devices, to improve accessibility and accuracy in diagnosing and monitoring pre-eclampsia. The continued refinement of imaging modalities, particularly point-of-care technologies, promises to further enhance the management of pre-eclampsia and improve maternal and fetal outcomes in the future.[33]

Utilisation of Computed Tomography (CT) in Cerebrovascular Assessment

CT is a widely accessible imaging technique that provides rapid results, making it particularly useful in emergency situations where time is of the essence. In pre-eclampsia, CT is often employed to quickly assess for acute neurological events such as intracranial haemorrhage, stroke, or brain infarctions. When stroke is suspected, CT can detect early signs of ischemic changes, although it is less sensitive in the detection of smaller, early infarcts compared to MRI. However, its rapid acquisition time makes CT an invaluable tool in the acute assessment of neurological emergencies, where decisions must be made quickly.[35]

Advantages of CT in Acute Settings

One of the primary advantages of CT is its ability to rapidly evaluate patients in critical or emergency situations. In pre-eclampsia, when patients may present with severe neurological symptoms like headache, visual changes, or seizures, CT can be used as an initial diagnostic tool. The fast nature of the scan—often completed within minutes—enables clinicians to rule out intracranial bleeding and assess the presence of large infarctions. This speed is particularly crucial when immediate interventions, such as the administration of antihypertensive drugs or the initiation of magnesium sulfate therapy, are required to prevent further cerebral damage. Furthermore, CT is effective in identifying conditions like intraventricular or subarachnoid haemorrhage, which can have life-threatening consequences in the context of severe pre-eclampsia.[35,36]

Limitations of CT in Pregnant Women

While CT is highly effective for rapid assessment, its use in pregnant women is limited due to concerns about ionising radiation. Although modern CT scanners utilise low-dose radiation protocols, exposure to X-rays remains a potential risk to the developing fetus, especially in the early stages of pregnancy when organogenesis is occurring. In non-urgent cases, the potential risks of radiation exposure often outweigh the benefits of CT scanning, particularly when the imaging is not immediately necessary for life-saving interventions. Additionally, while CT is excellent at identifying gross structural abnormalities, such as haemorrhage or large infarctions, it is less sensitive than MRI for detecting more subtle changes, such as vasogenic oedema or small infarcts. This limitation makes CT less effective in detecting certain pre-eclampsia-related complications like posterior reversible encephalopathy syndrome (PRES). [35,36]

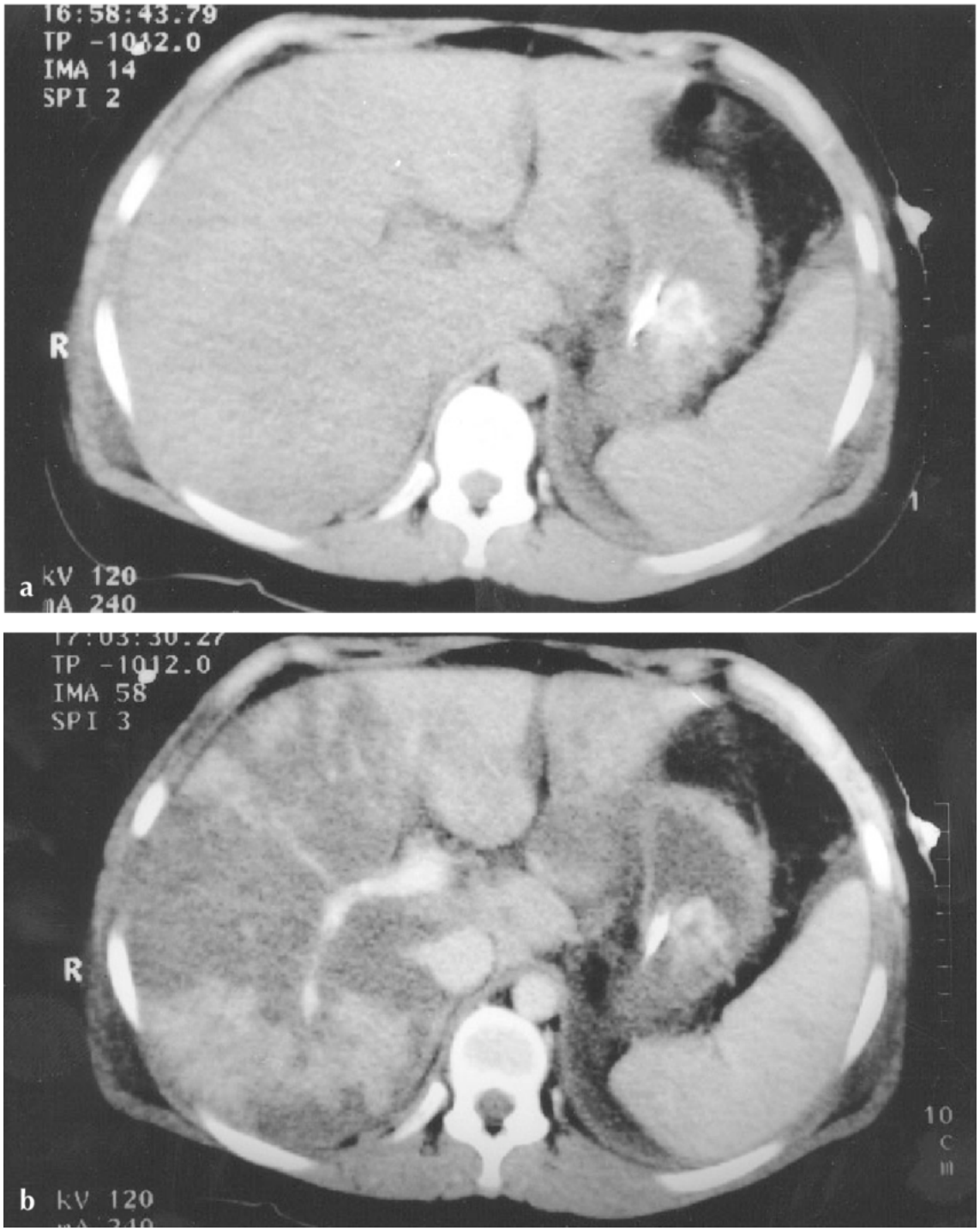


Figure 6: Pre (a) and post (b) contrast CT images of a woman with pre-eclampsia

Magnetic Resonance Imaging (MRI): An Advanced Tool for Cerebrovascular Changes

MRI is a non-invasive imaging modality that uses strong magnetic fields and radiofrequency waves to create detailed images of the brain and vasculature. MRI is particularly valuable in the evaluation of pre-eclampsia-related neurological changes because it offers superior soft tissue resolution compared to CT. It can detect subtle changes in brain tissue, such as microvascular injury, ischemia, and oedema, which may not be visible on CT scans. The high sensitivity of MRI to fluid changes makes it an excellent tool for identifying vasogenic oedema—a hallmark of posterior reversible encephalopathy syndrome (PRES), which is commonly seen in pre-eclampsia. The ability to perform multiple imaging sequences, such as diffusion-weighted imaging (DWI), T2-weighted imaging, and fluid-attenuated inversion recovery (FLAIR), enhances MRI's ability to detect both acute and chronic cerebral changes in pre-eclampsia.[36,37]

MRI Sequences for Detailed Brain Evaluation

In pre-eclampsia, MRI sequences like DWI are particularly useful in the early detection of ischemic changes. DWI is highly sensitive to the movement of water molecules in the brain and can detect early signs of infarction within minutes to hours of onset, long before any permanent damage occurs. This allows for early therapeutic interventions, such as the use of thrombolytics or surgery, which can significantly improve patient outcomes. T2-weighted imaging and FLAIR are other important sequences that can identify vasogenic oedema, which is often seen in PRES, a condition characterised by swelling in the posterior regions of the brain. FLAIR imaging is especially useful in detecting subtle areas of oedema or small infarcts that may be missed with other imaging modalities. Additionally, magnetic resonance angiography (MRA) can be used to evaluate the cerebral vasculature, helping to

identify abnormalities such as arterial stenosis or malformations, which may increase the risk of stroke in pre-eclampsia.

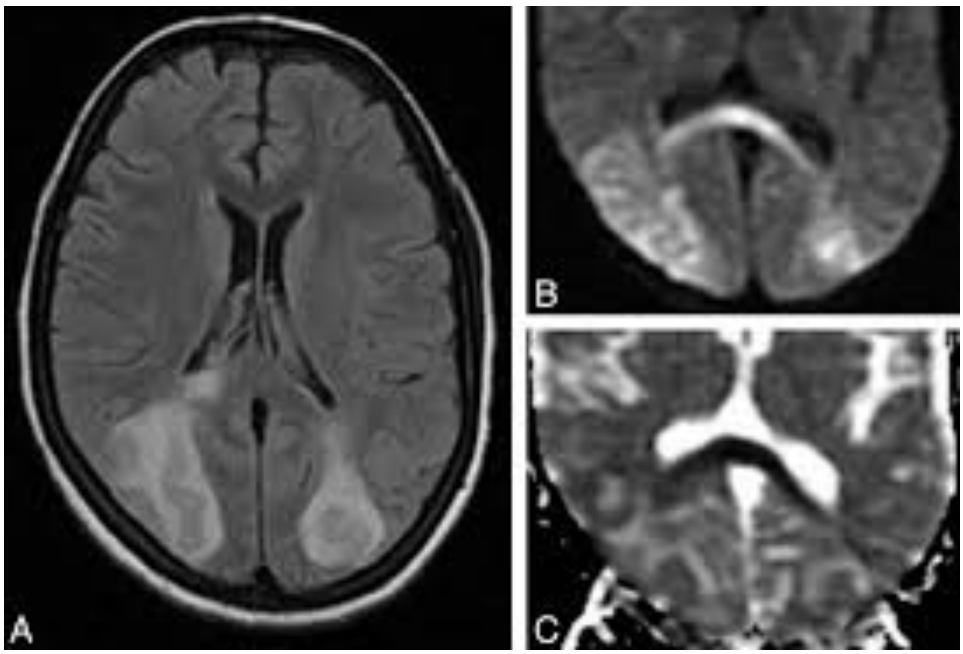


Figure 7: MRI of eclamptic encephalopathy

Limitations of MRI in Pregnant Populations

Despite its advantages, MRI also presents some limitations, particularly when used in pregnant patients. One of the primary concerns is the use of gadolinium-based contrast agents, which are often required for enhanced vascular imaging. Gadolinium agents can cross the placenta and accumulate in fetal tissues, potentially posing risks to fetal development. As a result, their use during pregnancy is generally avoided unless absolutely necessary, and even then, only in cases where the benefits of enhanced imaging outweigh the risks. Additionally, MRI requires patients to remain still for extended periods, typically 30–60 minutes, which may not be feasible for pregnant women with severe pre-eclampsia who may be in pain or discomfort. Furthermore, MRI machines are not as widely available as CT

scanners, particularly in low-resource settings, making access to MRI limited in certain regions. [36,37]

MRI's Role in Long-Term Monitoring of Cerebrovascular Health

One of the key advantages of MRI over CT is its ability to assess long-term changes in cerebral health. In women with pre-eclampsia, ongoing monitoring of the brain's vascular and structural changes can be crucial for preventing long-term neurological complications. MRI is highly effective in detecting chronic changes such as cerebral atrophy, white matter lesions, and microvascular damage, which may not be apparent in the acute phase of pre-eclampsia but could have long-lasting effects on cognitive function and neurological health. Additionally, because MRI does not rely on ionising radiation, it can be used repeatedly for longitudinal monitoring, providing clinicians with a more comprehensive understanding of a patient's cerebrovascular health over time. [36,37]

Ophthalmic Artery Anatomy and Function[40]

The ophthalmic artery is a major branch of the internal carotid artery (ICA) that supplies blood to the eye and its surrounding structures, including the optic nerve, retina, and extraocular muscles. Interestingly, the ophthalmic artery has significant embryological and anatomical parallels with the cerebral vasculature, which has important implications for its use in evaluating cerebral hemodynamics, particularly in the context of disorders like pre-eclampsia.

During fetal development, the ophthalmic artery and cerebral vasculature share a common origin from the internal carotid artery, which is one of the main suppliers of blood to the brain. Early in embryogenesis, the carotid artery gives rise to several branches that develop into the major cerebral arteries, including the ophthalmic artery. This shared origin is

fundamental to the close anatomical and functional relationship between the ophthalmic and cerebral circulations. As both systems evolve, the ophthalmic artery continues to supply blood to the eye, while the cerebral arteries, including the middle cerebral artery (MCA) and posterior cerebral artery (PCA), extend into the brain.

In terms of anatomical location, the ophthalmic artery branches off the internal carotid artery just before it enters the cranial cavity. This positioning places the ophthalmic artery in close proximity to the cerebral vessels, reinforcing their functional interdependence. The ophthalmic artery then passes through the optic canal and enters the orbit, where it gives rise to several branches that supply the retina, optic nerve, and other structures involved in vision. Notably, the ophthalmic artery's blood flow characteristics—such as its resistance and velocity—mirror those of larger cerebral arteries, which is why it has been considered an indicator of cerebral hemodynamics in certain clinical contexts.

Embryologically, the ophthalmic artery's developmental pathway is tightly connected to the brain's vasculature. Both the ophthalmic artery and the cerebral arteries form from a common vascular plexus that develops during the early stages of embryogenesis. This vascular plexus eventually differentiates into the internal carotid artery and its branches, including the ophthalmic artery and the major cerebral arteries that supply the brain. Because of this shared embryological origin, both systems share similar physiological characteristics, including how they respond to changes in systemic blood pressure and blood flow dynamics. This similarity makes the ophthalmic artery an important marker for monitoring cerebral perfusion in various clinical conditions, including pre-eclampsia.

The anatomical parallels between the ophthalmic and cerebral vasculature extend to the regulatory mechanisms that control blood flow. For instance, both systems are influenced by similar neurovascular regulation, including factors such as autoregulation, vascular tone,

and response to sympathetic and parasympathetic nervous system stimuli. These common regulatory mechanisms mean that alterations in the hemodynamics of the ophthalmic artery can reflect changes in the cerebral vasculature, which is particularly useful when assessing cerebral complications associated with conditions like pre-eclampsia.

Functional Relevance of the Ophthalmic Artery in Reflecting Cerebrovascular Hemodynamics

The functional relevance of the ophthalmic artery in reflecting cerebrovascular hemodynamics is rooted in its anatomical and physiological connections with the cerebral circulatory system. As a branch of the internal carotid artery (ICA), the ophthalmic artery shares a direct vascular pathway with the cerebral arteries, notably the middle cerebral artery (MCA) and other large-caliber vessels that supply the brain. This proximity allows the ophthalmic artery to serve as an indirect yet valuable indicator of cerebral blood flow dynamics, particularly in cases where real-time cerebrovascular monitoring is crucial.[41]

One of the key aspects of cerebrovascular hemodynamics is cerebral autoregulation, which ensures stable blood flow to the brain despite fluctuations in systemic blood pressure. When systemic blood pressure increases or decreases, cerebral blood vessels adapt by constricting or dilating, maintaining consistent perfusion pressure. The ophthalmic artery, given its embryological and anatomical similarities to the cerebral vasculature, responds to these changes in blood pressure in much the same way as larger cerebral arteries. This shared autoregulatory behaviour makes the ophthalmic artery a sensitive marker for detecting disruptions in cerebral blood flow, particularly in pathological conditions such as pre-eclampsia, where cerebrovascular autoregulation is often impaired.[30]

In pre-eclampsia, endothelial dysfunction and abnormal placental development lead to systemic hypertension, which can compromise cerebral autoregulation and affect the hemodynamics of both cerebral and ophthalmic circulations. The ophthalmic artery's blood flow velocity, resistance index (RI), and pulsatility index (PI) have been found to correlate with changes in cerebral blood flow in pre-eclamptic patients. For instance, in cases where systemic hypertension leads to increased resistance in the vasculature, the ophthalmic artery may exhibit higher resistance and lower diastolic flow, which reflects impaired cerebral perfusion. These changes in the ophthalmic artery are indicative of similar alterations in the cerebral vasculature, making it a reliable proxy for evaluating cerebrovascular dysfunction.[33]

The use of Doppler ultrasonography to measure blood flow in the ophthalmic artery offers a real-time, non-invasive method for monitoring these hemodynamic changes. Doppler indices such as the RI and PI in the ophthalmic artery can provide insights into the degree of vascular resistance and blood flow patterns. Elevated PI and RI values in the ophthalmic artery have been shown to correlate with poor cerebral perfusion, which is a hallmark of pre-eclampsia. [33]

Moreover, the ophthalmic artery's functional relevance extends beyond just the assessment of blood flow velocity. The artery is also sensitive to changes in vascular tone and elasticity, which are often altered in pre-eclampsia and other hypertensive disorders of pregnancy. Impaired endothelial function, commonly seen in pre-eclampsia, leads to increased arterial stiffness and reduced capacity for vasodilation. These changes can affect both the cerebral and ophthalmic circulations, leading to abnormal hemodynamic readings in the ophthalmic artery.[35]

In addition to its role in evaluating blood flow, the ophthalmic artery provides insights into systemic vascular health due to its shared embryological origin with the cerebral

circulation. Changes in the hemodynamics of the ophthalmic artery are often indicative of broader systemic alterations, such as increased arterial resistance or reduced compliance, which can affect both the brain and other organs. This is particularly important in conditions like pre-eclampsia, where systemic hypertension can lead to widespread vascular changes. Thus, monitoring the ophthalmic artery can offer a broader understanding of maternal cardiovascular function, complementing other diagnostic approaches like blood pressure measurements and fetal monitoring.

The ophthalmic artery also offers advantages over traditional cerebrovascular imaging techniques, such as CT and MRI, in that it is easily accessible and can be evaluated in real-time at the point of care. This makes it a practical tool for monitoring cerebrovascular health in pregnant women, especially in settings where advanced imaging modalities may be unavailable or impractical. Additionally, ophthalmic artery Doppler ultrasonography does not carry the risks associated with ionizing radiation (as seen with CT) or the logistical challenges of MRI, making it a safer and more feasible option for pregnant patients.

Principles of Doppler Ultrasonography and Its Application in Maternal-Fetal Medicine

Doppler ultrasonography is a non-invasive imaging technique that uses the Doppler effect to assess blood flow and vascular resistance within the body. It works by emitting high-frequency sound waves through tissues, which are reflected back to the transducer by moving red blood cells. The frequency shift in the returned sound waves—caused by the motion of these blood cells—is measured and analysed to determine the velocity and direction of blood flow. The principle of Doppler ultrasonography is based on the shift in frequency of the ultrasound waves as they reflect off moving objects, in this case, red blood cells in the bloodstream. This frequency shift provides valuable information about the speed of blood

flow, the presence of obstructions or stenosis, and the overall hemodynamic characteristics of the circulatory system.[33,42-44]

In obstetrics, Doppler ultrasonography plays a critical role in evaluating both maternal and fetal health. It is particularly useful in monitoring the blood flow within the uteroplacental and fetal circulations. By measuring the velocity and resistance in the blood vessels of the uterus, placenta, and umbilical cord, Doppler ultrasonography helps assess placental perfusion, fetal well-being, and the risk of pregnancy complications such as pre-eclampsia, intrauterine growth restriction (IUGR), and fetal distress. In the context of maternal health, Doppler ultrasonography also provides insight into the maternal vasculature, allowing for the detection of conditions such as gestational hypertension and pre-eclampsia, both of which can have significant impacts on maternal and fetal outcomes.[44]

One of the primary applications of Doppler ultrasonography in maternal-fetal medicine is the assessment of uteroplacental blood flow. The placental circulation is critical for providing oxygen and nutrients to the fetus, and any compromise in blood flow to the placenta can result in adverse outcomes. Doppler studies of the uterine arteries, for example, can help identify abnormal flow patterns indicative of placental insufficiency, a condition that is commonly associated with pre-eclampsia and IUGR. Abnormal Doppler findings in the uterine arteries, such as increased pulsatility index or absent or reversed end-diastolic flow, are markers of impaired placental perfusion and can guide clinical decision-making regarding the management of high-risk pregnancies.[43,44]

Similarly, Doppler ultrasonography of the umbilical artery is a powerful tool for evaluating fetal well-being. The umbilical artery Doppler waveform is an important indicator of fetal oxygenation, and abnormal waveforms, such as an increased resistance or absent end-diastolic flow, can indicate fetal distress or hypoxia. Such findings prompt early interventions

to prevent further complications and improve neonatal outcomes. Doppler ultrasonography is also used to assess the middle cerebral artery (MCA) in cases where there is concern about fetal anemia or other conditions that may affect the fetal circulation, as the MCA is one of the primary arteries supplying blood to the fetal brain.

In addition to its use in fetal evaluation, Doppler ultrasonography is invaluable for monitoring maternal vascular health, particularly in cases of hypertensive disorders such as pre-eclampsia. The measurement of blood flow velocity and resistance in maternal vessels, including the uterine arteries and ophthalmic artery, allows for the early detection of vascular changes associated with pre-eclampsia, such as increased vascular resistance and impaired blood flow.

The utility of Doppler ultrasonography extends beyond its ability to detect abnormal blood flow. It can also be used to monitor the progression of disease and the effectiveness of treatment. For example, in patients with pre-eclampsia, repeated Doppler studies can track changes in uterine and umbilical artery flow, providing valuable information about the condition's evolution. Similarly, in cases of IUGR, Doppler monitoring can assess the response to interventions such as corticosteroids or changes in maternal blood pressure, helping clinicians determine the best course of action to optimise fetal health.[43]

A significant advantage of Doppler ultrasonography is its ability to provide real-time, dynamic information about blood flow. This is particularly beneficial in high-risk obstetric situations where timely decisions are crucial. Doppler imaging can be performed at the point of care, without the need for invasive procedures or complex equipment, making it a convenient and accessible tool for routine monitoring and emergency situations alike.[44] Furthermore, Doppler ultrasonography does not involve ionizing radiation, making it a safer

alternative to techniques like CT or X-ray, especially when monitoring pregnant women and their fetuses.

Parameters Assessed in Doppler Velocimetry[42]

Doppler velocimetry involves the measurement of various parameters that provide crucial information about blood flow and vascular resistance. These parameters are especially useful in evaluating maternal and fetal circulatory health, particularly in high-risk pregnancies.

- Peak Systolic Velocity (PSV): This refers to the highest blood flow velocity achieved during the systolic phase of the cardiac cycle, when the heart pumps blood into the arteries. PSV is an important indicator of the degree of arterial stenosis or resistance. In obstetrics, assessing the PSV in arteries like the umbilical artery or uterine arteries can provide insight into placental and fetal perfusion. An increased PSV can be indicative of reduced compliance or increased vascular resistance in the blood vessels, which may signal conditions like pre-eclampsia or intrauterine growth restriction (IUGR).
- End-Diastolic Velocity (EDV): EDV measures the blood flow velocity during the diastolic phase of the cardiac cycle, after the heart has relaxed and filled with blood. In healthy pregnancies, there is often a noticeable amount of blood flow during diastole in the umbilical artery, indicating good placental perfusion. In pathological conditions such as pre-eclampsia, the EDV may decrease or even reverse, suggesting impaired placental perfusion and potential fetal distress. The absence or reversal of EDV is a key sign of compromised fetal oxygenation and is associated with poor outcomes in pregnancies affected by hypertension.

- Resistive Index (RI): The RI is a measure of the resistance to blood flow within a vessel and is calculated as the difference between the peak systolic velocity and the end-diastolic velocity, divided by the peak systolic velocity. A higher RI indicates increased vascular resistance, which can be seen in conditions like pre-eclampsia, where there is endothelial dysfunction and altered vascular tone. A normal RI suggests low resistance to blood flow, typical of healthy, well-perfused vessels. An elevated RI in the uterine arteries, for instance, is often associated with placental insufficiency and can signal the risk of pre-eclampsia, IUGR, or fetal hypoxia.
- Pulsatility Index (PI): The PI is similar to the RI but is a more general measure of blood flow pulsatility within a given artery. The PI is calculated by measuring the difference between the peak systolic velocity and the end-diastolic velocity and dividing it by the mean velocity. Higher PI values are indicative of increased vascular resistance and can be associated with reduced perfusion, while lower PI values suggest improved or normal blood flow dynamics. In obstetrics, abnormal PI values in the uterine or umbilical arteries can be early indicators of complications like pre-eclampsia, preterm labor, or fetal growth restriction.
- Systolic-Diastolic Ratio (SDR): The SDR compares the peak systolic velocity to the end-diastolic velocity and is another measure used to assess blood flow resistance. An increased SDR can indicate high resistance in the placental or uterine vasculature, while a lower ratio typically reflects a more favourable blood flow profile. This parameter is often used in conjunction with RI and PI to gain a comprehensive understanding of maternal and fetal circulatory health.

Advantages of Doppler Ultrasonography in Point-of-Care Assessments

Doppler ultrasonography offers several significant advantages as a point-of-care diagnostic tool, particularly in obstetrics. Its ability to provide real-time, dynamic information about blood flow and vascular resistance makes it indispensable in monitoring both maternal and fetal health during pregnancy.

- Non-invasive and Safe: Unlike other imaging techniques such as CT or MRI, Doppler ultrasonography is non-invasive and does not involve the use of ionizing radiation. This makes it particularly safe for use in pregnant women, who are at risk of fetal exposure to harmful radiation. The safety profile of Doppler ultrasonography is one of the reasons it is widely used in obstetrics for routine and emergency monitoring of both maternal and fetal circulations.
- Real-Time Monitoring: One of the most significant advantages of Doppler ultrasonography is its ability to provide real-time data about blood flow dynamics. This is crucial in high-risk pregnancies where timely decisions need to be made to ensure maternal and fetal health. Clinicians can immediately assess changes in blood flow patterns, making it possible to respond rapidly to any abnormalities or concerns, such as reduced placental perfusion or fetal hypoxia.
- Accessibility: Doppler ultrasonography can be performed in a wide range of clinical settings, including outpatient clinics, labor and delivery units, and emergency rooms. Its portability and ease of use make it accessible for point-of-care assessments, enabling healthcare providers to conduct quick evaluations without the need for specialized imaging equipment or complicated procedures. This is particularly beneficial in resource-limited settings or when rapid evaluation is needed.
- No Need for Contrast Agents: Unlike some other imaging techniques, Doppler ultrasonography does not require contrast agents, which can sometimes carry risks of

allergic reactions or complications, especially in pregnant women. This further contributes to its safety and feasibility as a diagnostic tool in obstetrics.

- Cost-Effectiveness: Doppler ultrasonography is generally more cost-effective than other imaging modalities such as MRI or CT. Given its ability to provide accurate, real-time information without the need for expensive equipment or contrast agents, Doppler ultrasonography is a more affordable option for monitoring maternal and fetal health, especially in resource-limited settings.
- Versatility: Doppler ultrasonography can be used to assess a variety of blood vessels in both the mother and fetus. It is commonly used to evaluate the umbilical artery, uterine arteries, middle cerebral artery, and ophthalmic artery, among others. This versatility allows clinicians to obtain a comprehensive view of maternal and fetal circulatory health, aiding in the diagnosis of various pregnancy complications such as pre-eclampsia, intrauterine growth restriction (IUGR), and fetal distress.
- Timely Intervention: The real-time nature of Doppler ultrasonography allows for the early detection of abnormal blood flow patterns, which can be crucial in preventing serious complications such as pre-eclampsia-related stroke, eclampsia, or fetal hypoxia. By providing immediate feedback, Doppler ultrasonography enables healthcare providers to initiate timely interventions, such as blood pressure management, corticosteroid administration, or early delivery, to improve maternal and fetal outcomes.

Use of Ophthalmic Artery Doppler in Non-Obstetric Conditions

Ophthalmic artery Doppler studies have long been utilised in a variety of non-obstetric conditions due to their ability to provide valuable insights into cerebrovascular health and systemic circulatory dynamics. The ophthalmic artery, which supplies blood to the eye and parts of the brain, serves as a critical vessel for assessing hemodynamics that reflect the broader cerebral vasculature. Doppler ultrasonography of the ophthalmic artery provides an effective, non-invasive means of assessing blood flow and resistance, making it a useful diagnostic tool in numerous systemic and ocular disorders.[45]

- ✓ Glaucoma: One of the most established uses of ophthalmic artery Doppler is in the management of glaucoma, a condition characterised by elevated intraocular pressure and potential damage to the optic nerve. Abnormal blood flow in the ophthalmic artery can contribute to the pathophysiology of glaucoma, as impaired perfusion may exacerbate optic nerve damage. Doppler studies can assess the resistive index (RI) and pulsatility index (PI) in the ophthalmic artery, providing an indication of vascular insufficiency that may compromise optic nerve health. Research has shown that patients with glaucoma often exhibit higher RI values, suggesting increased vascular resistance in the ophthalmic artery, which is linked to impaired ocular blood flow and greater susceptibility to optic nerve damage. Thus, ophthalmic artery Doppler studies offer a valuable tool for evaluating the vascular health of patients with glaucoma, aiding in both diagnosis and monitoring of disease progression.
- ✓ Systemic Atherosclerosis: The ophthalmic artery is also an important vessel for assessing systemic atherosclerosis, a condition in which the arteries become narrowed and hardened due to plaque buildup. Atherosclerosis can lead to diminished blood flow and vascular resistance, which in turn can increase the risk of cardiovascular events such as stroke. Ophthalmic artery Doppler studies can provide early indications of atherosclerotic

changes in the vasculature by measuring alterations in flow velocity, pulsatility, and resistance within the artery. For example, increased RI or PI values in the ophthalmic artery may suggest the presence of systemic atherosclerosis, as these changes reflect increased vascular resistance due to plaque formation or endothelial dysfunction. As a result, ophthalmic artery Doppler studies can serve as an adjunctive tool in the assessment of patients at risk for cardiovascular diseases and cerebrovascular events, helping to identify individuals who may require further diagnostic evaluation or therapeutic intervention.

- ✓ Heart Failure: In patients with heart failure, the heart's ability to pump blood effectively is compromised, leading to reduced perfusion of various organs, including the eyes and brain. The ophthalmic artery, being a major supplier of blood to the eye and related cerebral structures, can be affected by the reduced cardiac output seen in heart failure. Doppler studies of the ophthalmic artery have been used to assess the impact of heart failure on ocular and cerebral circulation. In heart failure, ophthalmic artery Doppler studies typically reveal increased resistance to blood flow, as evidenced by elevated RI and PI values. These changes reflect the systemic reduction in perfusion pressure and altered hemodynamics, which are characteristic of heart failure. By providing early indicators of circulatory impairment, ophthalmic artery Doppler studies can help clinicians assess the extent of cardiovascular dysfunction and monitor treatment response in patients with heart failure.
- ✓ Cerebrovascular Disorders: Ophthalmic artery Doppler ultrasonography has also been used in the evaluation of cerebrovascular disorders, such as transient ischemic attacks (TIAs), stroke, and cerebral aneurysms. Given the embryological and anatomical similarities between the ophthalmic artery and the cerebral vasculature, Doppler measurements in the ophthalmic artery can provide valuable information about

cerebrovascular health. For instance, reduced blood flow or increased vascular resistance in the ophthalmic artery may indicate impaired cerebral perfusion, which is a risk factor for stroke and other cerebrovascular events. Doppler studies of the ophthalmic artery, therefore, serve as an adjunct to other imaging techniques, such as CT or MRI, in the assessment of patients with suspected cerebrovascular diseases. By identifying early signs of vascular dysfunction, ophthalmic artery Doppler studies can aid in the early detection of cerebrovascular diseases, facilitating timely intervention and improving patient outcomes.

Existing studies on ophthalmic artery Doppler in pre-eclampsia and related conditions:

Predictive and Diagnostic Value

Resistivity and Pulsatility Indices: Both the resistivity index (RI) and pulsatility index (PI) have been shown to effectively distinguish between mild and severe forms of PE, offering high sensitivity and specificity. A study conducted in a rural South Indian population highlighted the resistivity index and peak ratio as particularly effective for early detection of PE (Shetty et al., 2024).[46] These indices have proven to be reliable in identifying abnormal hemodynamics associated with PE, which can assist in early diagnosis and intervention.

Peak Systolic Velocity (PSV) Ratio: The PSV ratio, when combined with other biomarkers, provides an effective prediction of PE, particularly when assessed between 35 and 37 weeks of gestation. This approach has demonstrated a high detection rate for PE, often identifying the condition within three weeks of the assessment (Mansukhani et al., 2024).[47] By incorporating the PSV ratio alongside other clinical indicators, healthcare providers can enhance the accuracy of PE prediction and improve patient outcomes.

According to a study by Selima et al., Ophthalmic artery PI had a sensitivity of 89.7% and specificity of 75.12% at a cutoff value < 1.76 in predicting pre-eclampsia. Also, ophthalmic artery RI had a sensitivity of 80.2% and specificity of 74.9% at a cutoff value < 0.77 in predicting pre-eclampsia. The peak Ratio (PR) of the ophthalmic artery had a sensitivity of 77.3% and a specificity of 65.8% at a cutoff value > 0.59 in predicting pre-eclampsia. In addition, 2nd systolic peak (P2) ophthalmic artery had a sensitivity of 91.7% and specificity of 61.7% at a cutoff value > 20.1 in prophesying pre-eclampsia.[48]

Correlation with Blood Pressure

- ✓ Mean Arterial Pressure (MAP): Changes in ophthalmic artery Doppler indices, such as a decrease in resistivity and pulsatility indices, have been shown to correlate with rising blood pressure in patients with PE. This suggests that ophthalmic artery Doppler parameters can serve as early indicators of PE before clinical symptoms, such as hypertension and proteinuria, fully develop (Singh, 2024).[49] The ability to detect these early hemodynamic changes can enable timely intervention and monitoring in high-risk pregnancies.

Meta-Analysis Findings

- ✓ Diagnostic Performance: A systematic review and meta-analysis examining the diagnostic performance of ophthalmic artery Doppler in PE found that the peak ratio (PR) and the second systolic velocity peak (P2) were the most reliable parameters for PE detection. These indices demonstrated high sensitivity and specificity, outperforming other Doppler indices like the resistance index (RI) and pulsatility index (PI) in terms of diagnostic accuracy (Roever et al., 2023). This meta-analysis highlights the growing body of evidence supporting the use of ophthalmic artery Doppler as a valuable tool in the clinical management of PE.[50]

Correlation Between Doppler Parameters and Disease Severity in Pre-Eclampsia

Doppler ultrasonography has become a valuable tool in assessing the severity of pre-eclampsia (PE) and its potential complications. Doppler parameters, which include indices such as the resistivity index (RI), pulsatility index (PI), peak systolic velocity (PSV), and the presence or absence of end-diastolic flow (EDV), provide insight into the systemic and uteroplacental hemodynamics that are altered in PE. These parameters reflect the degree of vascular resistance and impaired perfusion in critical organs such as the placenta, kidneys, and brain, which are directly affected by the pathophysiology of PE. A correlation between these Doppler parameters and the severity of PE has been established, making Doppler ultrasonography an essential tool for predicting maternal and fetal outcomes.

Resistivity Index (RI) and Pulsatility Index (PI)

Both the resistivity index (RI) and pulsatility index (PI) are commonly used to evaluate vascular resistance in various arteries, including the uterine, umbilical, and ophthalmic arteries. In PE, elevated values of RI and PI indicate increased vascular resistance, which reflects the impaired ability of blood vessels to dilate and maintain optimal blood flow. Studies have demonstrated that higher RI and PI values in the uterine and umbilical arteries are associated with more severe forms of PE, particularly in cases with fetal growth restriction (FGR), placental insufficiency, and adverse pregnancy outcomes.[43]

In the ophthalmic artery, increased RI and PI values have been linked to the severity of maternal hypertension and endothelial dysfunction in PE. These indices reflect changes in the cerebral vasculature, which are crucial for understanding the neurological complications associated with PE, such as visual disturbances and eclampsia. Elevated RI and PI in the ophthalmic artery correlate with more severe hypertension and are predictive of a higher risk of complications such as stroke or seizure.

Peak Systolic Velocity (PSV) and Disease Progression

The peak systolic velocity (PSV) is another important Doppler parameter that reflects the speed at which blood flows through a particular vessel. In PE, changes in PSV can indicate the degree of circulatory impairment and the presence of vascular resistance. A decrease in PSV, particularly in the uterine arteries, is often seen in the early stages of PE and is associated with reduced placental perfusion. On the other hand, an increase in PSV, especially when combined with other abnormal Doppler findings, may suggest more severe vascular changes and a higher risk of maternal and fetal complications.

When used in conjunction with other Doppler indices, the PSV can provide valuable insights into the progression of PE. For example, a low PSV combined with elevated RI or PI in the uterine or umbilical arteries may indicate placental insufficiency, which is often seen in severe PE cases. Similarly, in the ophthalmic artery, elevated PSV can indicate increased blood flow to the brain as a compensatory mechanism in response to impaired cerebral perfusion due to hypertension. However, persistent elevated PSV in the context of rising blood pressure may signal a more severe form of PE, warranting closer monitoring and more aggressive interventions.[45]

End-Diastolic Flow (EDV) and Its Role in Predicting Severity

The presence or absence of end-diastolic flow (EDV) in the Doppler assessment of various arteries is another critical parameter in evaluating disease severity in PE. In normal pregnancies, the umbilical artery typically exhibits continuous EDV throughout the cardiac cycle. However, in PE, particularly in cases with severe hypertension or placental insufficiency, the umbilical artery may show absent or reversed EDV, which is associated with higher vascular resistance and impaired placental perfusion. This absence of EDV has

been correlated with an increased risk of fetal compromise, including intrauterine growth restriction (IUGR), preterm birth, and stillbirth.

Similarly, in the ophthalmic artery, the presence of EDV is an important indicator of cerebrovascular function. The absence of EDV in the ophthalmic artery can indicate severe impairment in cerebral perfusion, which is commonly observed in pre-eclamptic women with significant endothelial dysfunction and elevated blood pressure. The absence or reduction of EDV in the ophthalmic artery may also serve as a marker for more severe forms of PE, such as those with neurological complications like eclampsia or visual disturbances.

Combined Doppler Indices for Severity Prediction

To enhance the diagnostic and prognostic value of Doppler ultrasonography in pre-eclampsia, many studies have combined multiple Doppler parameters to assess disease severity more accurately. A composite index that includes RI, PI, PSV, and EDV has been shown to improve the ability to predict adverse maternal and fetal outcomes. For instance, a combination of high RI and PI values in the uterine arteries, along with absent EDV in the umbilical artery, is strongly associated with the development of severe PE and fetal compromise.[33] Similarly, combining Doppler measurements of the ophthalmic artery with uterine and umbilical artery Doppler can offer a more comprehensive assessment of the severity of PE and the associated risks for maternal and fetal health.

Prognostic Value of Doppler Parameters

Several studies have demonstrated the prognostic value of Doppler parameters in PE. Elevated RI and PI, particularly in the uterine and ophthalmic arteries, have been shown to correlate with poor maternal outcomes, including the risk of eclampsia, stroke, and organ damage. In addition, abnormal Doppler findings in the umbilical artery, such as absent or reversed EDV, are predictive of adverse fetal outcomes, including intrauterine growth

restriction (IUGR), preterm birth, and stillbirth. Monitoring these parameters during pregnancy allows for early identification of high-risk cases and facilitates timely interventions to reduce complications.

Studies from literature evaluating the maternal ophthalmic doppler velocimetry in pre-eclampsia:

1. Barbosa AS et al. (2010)[2] investigated the association between ophthalmic artery resistive index (OARI) and clinical evidence of posterior reversible encephalopathy syndrome (PRES) in 112 patients with severe pre-eclampsia. PRES, characterized by headache and blurred vision, is linked to cerebral autoregulation impairment. Among the patients, 46 (41%) exhibited clinical evidence of PRES. These patients had significantly lower OARI (P<0.0001), higher mean blood pressure at admission (P<0.0001), greater blood pressure elevation after the first trimester (P<0.0001), and increased lactate dehydrogenase levels (P<0.0001). Receiver operating characteristic (ROC) analysis showed an area under the curve (AUC) of 0.810±0.039 (95% CI: 0.742–0.895; P<0.0001), indicating strong predictive value. An OARI threshold of <0.56 was associated with a 12.67-fold higher risk of PRES (P<0.0001). The study concluded that OARI is a valuable biomarker for identifying PRES in severe pre-eclamptic patients, highlighting its potential for early detection and risk stratification.
2. Olatunji RB et al. (2015)[1] conducted a study to evaluate hemodynamic changes in the ophthalmic artery using Doppler ultrasound in 42 pre-eclamptic women and 41 healthy pregnant controls at the University College Hospital, Ibadan. They measured key Doppler parameters, including peak systolic velocity, peak diastolic velocity, end diastolic velocity, pulsatility index, and peak ratio using transorbital triplex ultrasound

with a 7–10 MHz multifrequency linear transducer. Their findings showed that the resistivity index, pulsatility index, and peak systolic velocity were significantly lower in pre-eclamptic patients, whereas peak diastolic velocity, end diastolic velocity, and peak ratio were significantly higher. Receiver operating characteristic (ROC) analysis revealed that the resistivity index (sensitivity 75%, specificity 77.8%) could distinguish mild from severe PE, while the peak ratio (sensitivity 90.5%, specificity 81.3%) was highly accurate in detecting PE. The study concluded that ophthalmic artery Doppler (OAD) ultrasound can serve as a valuable tool for monitoring disease progression in pre-eclamptic women and detecting early hemodynamic alterations in high-risk pregnancies, potentially preventing severe cerebral complications.

3. Kane SC et al. (2017)[4] highlight that pre-eclampsia, affecting 3–4% of pregnancies globally, is a major cause of maternal mortality, accounting for 12% of such deaths. Fatal complications include intracranial hemorrhage, eclampsia, hepatic rupture, and acute pulmonary edema. Neurological complications contribute significantly to both maternal mortality and long-term morbidity. These include visual impairment, stroke-related neurological deficits, and cognitive impairment later in life. The study underscores the severe short- and long-term consequences of pre-eclampsia, particularly its neurological impact.
4. Sarno M et al. (2020)[3] conducted a prospective observational study to assess the predictive value of maternal ophthalmic artery Doppler at 35–37 weeks' gestation for pre-eclampsia (PE) development. A total of 2,287 pregnancies were analyzed, with 60 (2.6%) developing PE, including 19 (0.8%) within three weeks of assessment. Ophthalmic artery Doppler parameters, including peak systolic velocity (PSV) ratio, were measured in both eyes. The detection rate (DR) of PE using maternal factors alone was 25%, which improved to 50% (P=0.005) when the PSV ratio was included.

For PE within three weeks, DR increased from 31.6% to 57.9%. Other Doppler indices did not significantly enhance prediction. PSV ratio measurements showed strong repeatability within the same eye ($r>0.8$) but lower correlation between eyes ($r\approx0.69$). The best predictive accuracy was achieved using the average of one measurement from each eye. The study concluded that ophthalmic artery PSV ratio at 35–37 weeks can effectively predict PE risk, particularly for cases occurring within three weeks.

5. Menêses VFS de C in 2020 established that ophthalmic artery (OA) Doppler velocimetry is an effective tool for diagnosing pre-eclampsia (PE). Key parameters, particularly the second peak systolic velocity (P2) and peak ratio (PR), demonstrated superior diagnostic performance. Cutoff values of $P2 \geq 21.5$ cm/s and $PR \geq 0.70$ yielded high sensitivity and specificity, with the combination of both parameters achieving the best diagnostic accuracy. Thus, OA Doppler sonography serves as a valuable complementary imaging examination in PE evaluation.[51]
6. Melo D et al. (2023)[5] conducted a meta-analysis to assess the accuracy of ophthalmic artery Doppler (OAD) parameters in diagnosing pre-eclampsia (PE). The study included eight studies with 1,425 pregnant women, stratifying results into mild, severe, early, and late PE. Key OAD parameters analyzed were peak systolic velocity (PSV), end-diastolic velocity (EDV), second systolic velocity peak (P2), resistance index (RI), pulsatility index (PI), and peak ratio (PR). PR and P2 demonstrated the best diagnostic performance, with PR showing an area under the summary receiver operating characteristic curve (AUsROC) of 0.885, 84% sensitivity, and 92% specificity, while P2 had an AUsROC of 0.926, 85% sensitivity, and 88% specificity. RI, PI, and EDV also performed well but had lower AUsROC values of 0.833, 0.794, and 0.772, respectively. PR and P2 exhibited the lowest false-positive rates, making

them the most reliable indicators. The study confirmed that OAD is a valuable complementary tool for diagnosing PE, especially in severe cases.

7. Adlakha E et al in 2024[52] stated that Maternal ophthalmic artery Doppler parameters, particularly the peak systolic velocity (PSV2) and PSV ratio, are significant predictors of pre-eclampsia (PE) development. In the study, PSV2 and PSV ratio were higher in PE cases, with sensitivities of 83.3% and specificities of 58.3% and 61.7%, respectively. Combining these parameters with uterine artery pulsatility index and mean arterial pressure enhanced predictive accuracy, achieving a maximum accuracy of 97.6%. Thus, ophthalmic artery Doppler is a promising tool for PE evaluation.
8. A study by Singh MB et al in 2024 evaluated maternal ophthalmic artery Doppler indices as potential early indicators of preeclampsia. It found that changes in resistivity and pulsatility indices correlated with rising mean arterial blood pressure (MAP) before clinical symptoms appeared. Specifically, a mean PSV ratio of 0.55 or above indicated a MAP of 100 mmHg or higher. These findings suggest that ophthalmic artery Doppler measurements could be useful for early detection of preeclampsia, warranting further research with larger sample sizes.[48]
9. Shetty S et al (2024) evaluated maternal ophthalmic artery Doppler velocimetry as a diagnostic tool for preeclampsia (PE). It found significant differences in Doppler parameters, such as mean peak systolic velocity, pulsatility index, and resistivity index, between preeclamptic cases and controls ($p < 0.001$). Notably, the resistivity index and peak ratio effectively distinguished between mild and severe PE. The findings suggest that ophthalmic artery Doppler is a valuable, non-invasive method for early detection of hemodynamic changes associated with preeclampsia in high-risk pregnancies.[49]

10. Elitsa H et al (2024) found that the ophthalmic ratio was significantly higher in high-risk patients compared to low-risk women ($p = 0.000$). Additionally, there was a notable relationship between PSV2/PSV1 and gestational age at birth in women with PE. Incorporating this Doppler measurement as a screening tool in Bulgaria could enhance early detection, risk stratification, and improve maternal and fetal health outcomes.[53]

MATERIALS AND METHODS

Study design: Prospective case-control study

Source of Data:

Pre-eclamptic patients were recruited from the antenatal clinic and labour ward, where the diagnosis of pre-eclampsia was confirmed by elevated blood pressure (BP) and proteinuria. The control subjects were normotensive pregnant patients, also seen in the antenatal clinic at the Department of Obstetrics & Gynaecology at B L D E (DEEMED TO BE UNIVERSITY) Shri B M Patil medical college, hospital and research centre, Vijayapura, Karnataka, India.

Study Period:

The study was conducted from April 2023 to March 2025.

Method of Data Collection:

Data collection followed stringent inclusion and exclusion criteria to ensure the reliability of the results.

Inclusion Criteria

- **Cases (Pre-eclampsia patients):** Pregnant women more than 28 weeks period of gestation who met the following criteria:
    - Elevated BP ≥140/90 mmHg on two measurements taken six hours apart.
    - Proteinuria >300 mg in a 24-hour urine sample or at least 1+ on a dipstick random urine test.
- **Controls (Normotensive patients):** Pregnant women more than 28 weeks period of gestation who met the following criteria:
    - Normal BP <140/90 mmHg on at least two separate antenatal visits, spaced 2-4 weeks apart.

- No proteinuria (1+) on dipstick random urine testing.

Exclusion Criteria

- History of diabetes or chronic hypertension.
- History of smoking, alcohol abuse, or drug abuse.
- History of ocular disease.
- Patients on medication for hypertension or corticosteroids.
- Vasculitis or other vascular disorders that might have affected Doppler measurements.

Sample Size

- **Pre-eclampsia patients:** 85
- **Normotensive patients:** 85
- **Total sample size:** 170

Sample Size Calculation

The sample size was calculated based on an anticipated mean ± SD of the Right Ophthalmic Artery Doppler Velocity (PDV) in pre-eclampsia patients of 22.56 ± 7.13 and in normotensive patients of 19.06 ± 6.82 (1). A minimum of 85 participants per group (total sample size of 170) was required to achieve a study power of 90% and a significance level of 5% (two-tailed) to detect a true difference in means between the two groups.

- **Level of significance:** 95%
- **Power of the study:** 90%
- **Clinically significant difference (d):** Difference between two parameters
- **SD (Standard deviation):** Common standard deviation

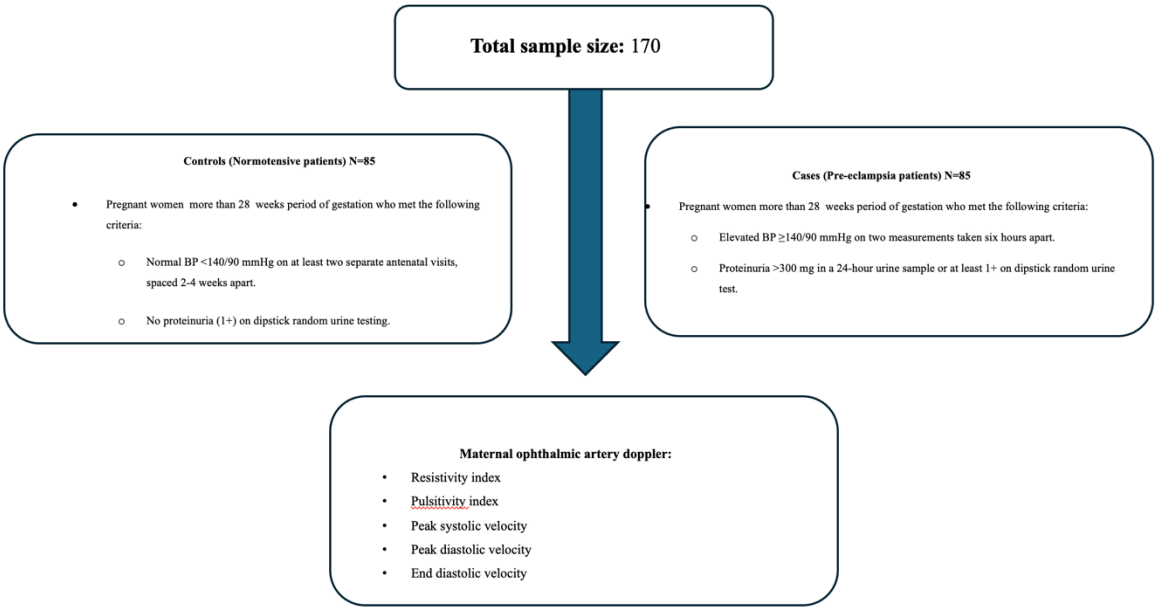
Statistical Analysis

- The data were entered into Microsoft Excel, and statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS) version 20.
- Results were presented as mean ± SD, counts, percentages, and diagrams.
- For normally distributed continuous variables between two groups, the Independent t-test was used. For non-normally distributed variables, the Mann-Whitney U test was applied.
- Categorical variables were compared between the two groups using the Chi-square test.
- The Receiver Operating Characteristic (ROC) curve was used to determine the cutoff value of relevant ophthalmic artery Doppler parameters.
- A p-value of <0.05 was considered statistically significant, and all statistical tests were performed in two-tailed mode.

METHODOLOGY

The study was initially submitted for clearance to the institutional review board. Informed consent was obtained from all patients prior to the procedure. Pre-eclamptic patients who met the inclusion criteria and provided consent were recruited as cases, while normotensive pregnant women who gave their consent served as controls. Each participant was assigned a unique study number. The control group and case group were matched for maternal age, gestational age (G.A.), and parity. Gestational age was determined using fetal head circumference measured between 19–24 weeks or crown–rump length at 11–13 weeks. In cases where this was not possible, G.A. was calculated based on the most recent menstrual cycle and confirmed by an obstetric ultrasound performed prior to the study.

Blood pressure (BP) measurements were taken with the patient at rest using a DIAMOND mercury sphygmomanometer. The systolic and diastolic BP were recorded based on the first and fifth Korotkoff sounds, respectively. The mean arterial pressure (M.A.P.) was then calculated using the formula: M.A.P. = 1/3 (systolic BP) + 2/3 (diastolic BP). All participants provided urine samples, which were tested for proteinuria using MISSION URINALYSIS REAGENT STRIPS.



OPHTHALMIC ARTERY DOPPLER :

According to the method outlined by Lieb et al., the patient was positioned supine with a slight lateral left rotation. The ophthalmic artery was then located using colour Doppler flow imaging after the transducer had been carefully positioned horizontally over the upper eyelid and tilted upward and downward. After relaxing for 10 minutes with both eyes closed, a little amount of acoustic gel was placed to the upper eyelid. The orbit was scanned using a GE VOLUSON S8 BT18 ultrasound machine with a 50/60Hz, 900 VA linear transducer, and the O.A.D. parameters were recorded. The right ophthalmic artery was insonated before the left. On the medial side of the optic nerve, flow velocity was assessed around 15 mm from the optic disc. Three waveforms were measured after six consecutive spectral waveforms of identical size and shape had been recorded. On each of the three waveforms, the P.I., RI, E.D.V., P.D.V., P.S.V. and were measured, and an average value derived from the three measurements was used for each parameter. In the systolic phase of

the cardiac cycle, the P.S.V. was the blood flow's quickest recorded velocity, and the E.D.V. was its slowest recorded velocity[1].

The peak ratio was manually obtained by dividing the P.D.V. values (as determined by manually tracing the spectral waveform at the peak following the proto-diastolic notch) by the values for the initial peak (i.e., P.S.V.).

Care was taken not to overly squeeze the eyelid with the transducer during the examination.

The filter was set to 50 Hz, the pulse repetition frequency was set, and the Doppler sample volume was adjusted to 2 mm while the angle of insonation was kept below 20°.

All of the O.A.D. velocimetry indices (P.S.V., E.D.V., P.D.V., RI, P.I., and peak ratio) were sonographically measured and recorded for all patients in the control group throughout the third trimester. For each case and control, six O.A.D. parameters were recorded: P.I., RI, P.S.V., E.D.V., P.D.V., and peak ratio.

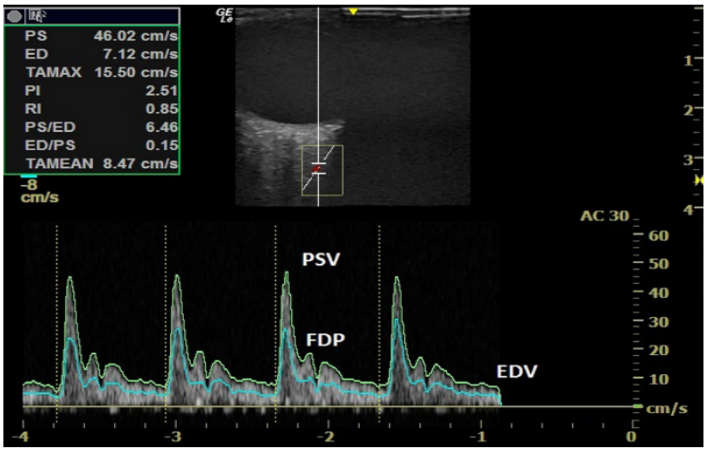


Figure 1 B-mode ultrasound image of orbit and retro-orbital structures, with superimposed color Doppler for identification of ophthalmic artery, and pulsed-wave Doppler to acquire its spectral waveform. EDV, end-diastolic velocity; FDP, first diastolic peak velocity; PSV, peak systolic velocity.

The end-diastolic velocity (E.D.V), Pulsatility index (P.I.), resistivity index (R.I.), peak diastolic velocity (P.D.V.), peak systolic velocity (P.S.V.), and peak ratio are the Doppler parameters that are used to characterize flow in the ophthalmic artery.

The peak ratio, or the ratio of the P.D.V. to the P.S.V., has been discovered to be quite helpful in assessing the blood flow via the ophthalmic arteries in the hypertensive state. The P.I. and R.I. gauge a blood vessel's resistance to blood flow. PSV-EDV divided by TAMAX, where TAMAX stands for time-averaged maximum velocity, yields P.I. The RI can only be collected at a certain period in the cardiac cycle and measures resistance distal to the place of measurement.

A PSV, EDV, PSV, RI and PI Measurement of O.A.D. pressure is crucial in assessing the hemodynamic changes in P.E. since visual symptoms and signs are linked to the disease's progression. In the examination of the ocular artery, the RI and P.I. are preferable to the P.S.V. and E.D.V. because both parameters, unlike the P.S.V. and E.D.V, are independent of the angle of insonation. In contrast to the strong association between the P.I. and R.I. values obtained from both eyes, there is also significant variation between the P.S.V. and E.D.V. for both eyes. The RI is suitable for assessing waveforms with high systolic and diastolic flows, as are frequently observed in ocular arteries. In the examination of the ocular artery, it has been proven to be more helpful than the P.I., which is more suitable in vascular beds with mild-to-moderate resistance.

DOPPLER MATERNAL OPHTHALMIC ARTERY (NORMAL VALUES)

Peak systolic velocity was 32+/-11.30, peak diastolic velocity was 19.06 +/- 6.82, Pulsatility index (PI) was 1.6+/-0.40 and resistivity index (RI) was 0.70 +/- 0.10

It is believed that this vessel's (ophthalmic artery) Doppler characteristics directly reflect the hemodynamics of the system.[4] Ophthalmic artery ultrasonography is a well-known imaging technique. It has been used to research several non-obstetric illnesses, such as multiple sclerosis, glaucoma, heart failure, and systemic atherosclerosis[2]. As a result, the current investigation of maternal ophthalmic artery Doppler suggests prospective applications for this point-of-care imaging technology in the future and serves as a tool to evaluate the cerebrovascular and hemodynamics in women with pre-eclampsia[1].

RESULTS

Demographic characteristics of normal population (controls)

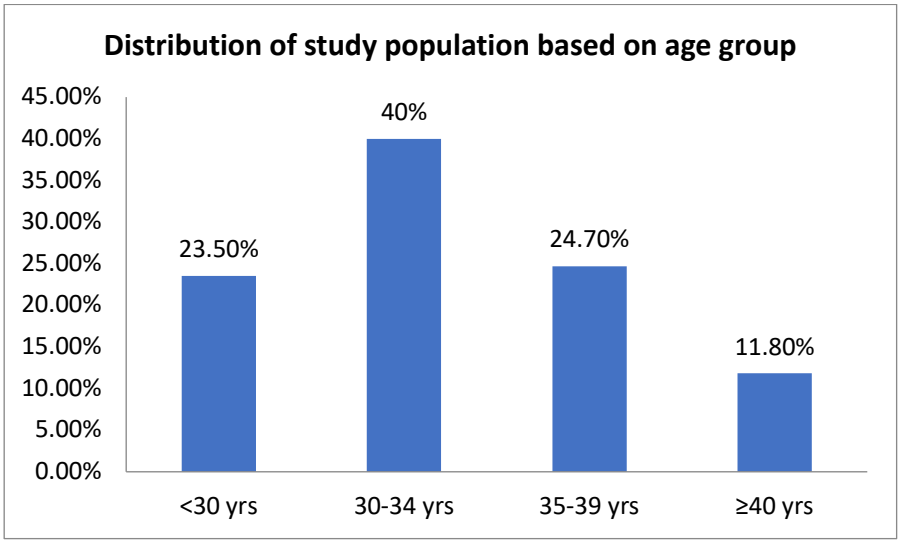
Among the 85 normal patients, the majority (40%) were aged between 30-34 years, while 23.5% were below 30 years, 24.7% were between 35-39 years, and 11.8% were aged 40 years or older. Regarding gestational age (GA), most patients (63.5%) had a GA between 35-39 weeks, followed by 29.4% with GA ≥40 weeks, while 7% had a GA between 30-34 weeks, and none had a GA below 30 weeks. Parity distribution showed that 41.2% of patients were para 1, 28.2% were nulliparous, 21.2% had parity 2, and 9.4% had parity of three or more. Systolic blood pressure (BP) measurements indicated that 40% of patients had values between 110-119 mmHg, 31.7% had systolic BP ≥120 mmHg, 17.6% had BP between 100-109 mmHg, and 10.6% had values below 100 mmHg. For diastolic BP, 44.7% of patients had values between 70-79 mmHg, 29.4% had BP between 60-69 mmHg, and 25.9% had BP ≥80 mmHg, while none had values below 60 mmHg.

Table 1: Demographic characteristics of normal population (controls)

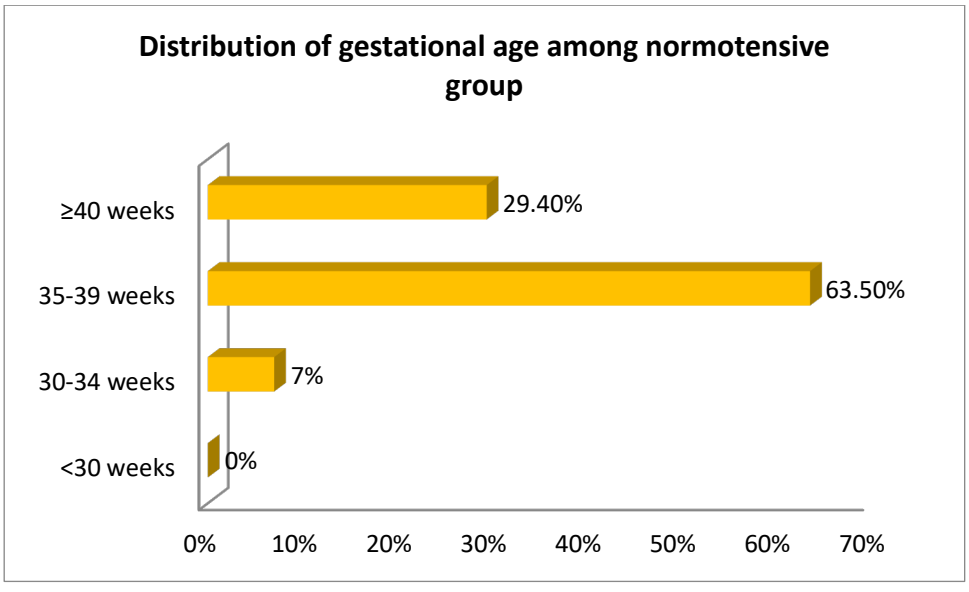
Characteristics	**Frequency (n=85)**	**Percentage**
Age in years		
<30	20	23.5
30-34	34	40
35-39	21	24.7
≥40	10	11.8
GA (weeks)		
<30	0	0
30-34	6	7
35-39	54	63.5
≥40	25	29.4
Parity		
0	24	28.2
1	35	41.2
2	18	21.2
≥3	8	9.4

Systolic BP (mmHg)		
<100	9	10.6
100-109	15	17.6
110-119	34	40
≥120	27	31.7
Diastolic BP (mmHg)		
<60	0	0
60-69	25	29.4
70-79	38	44.7
≥80	22	25.9

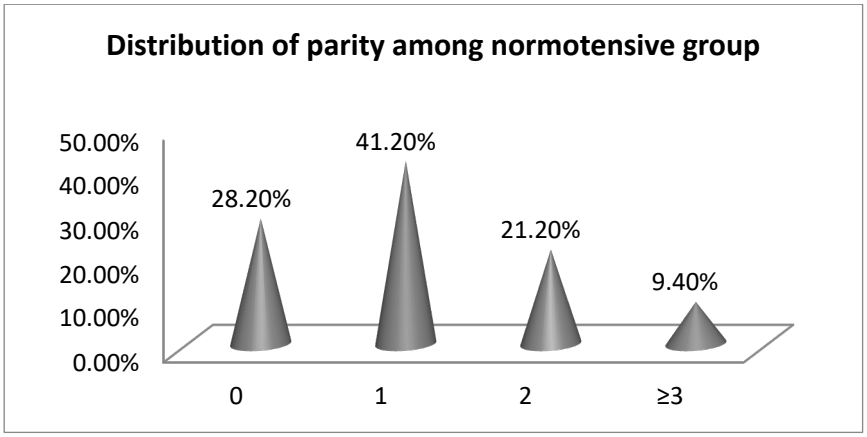
Graph 1a: Distribution of controls based on age



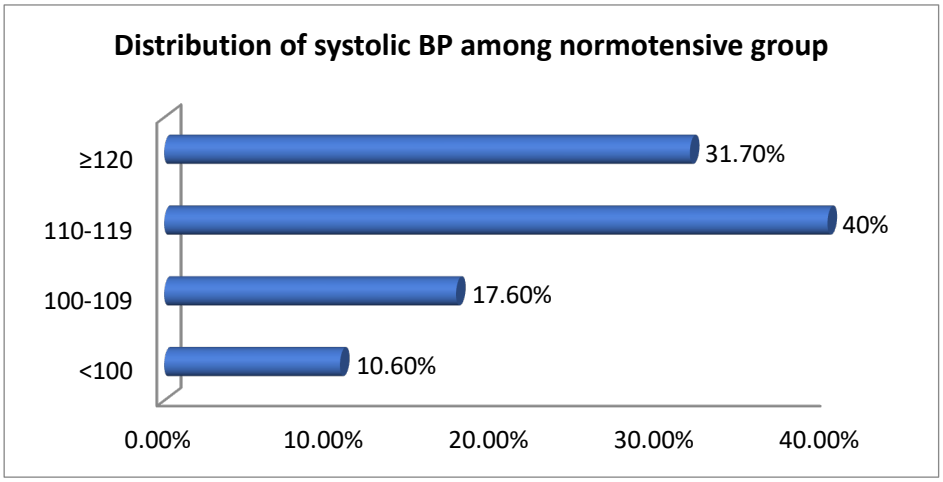
Graph 1b: Distribution of controls based on gestational age



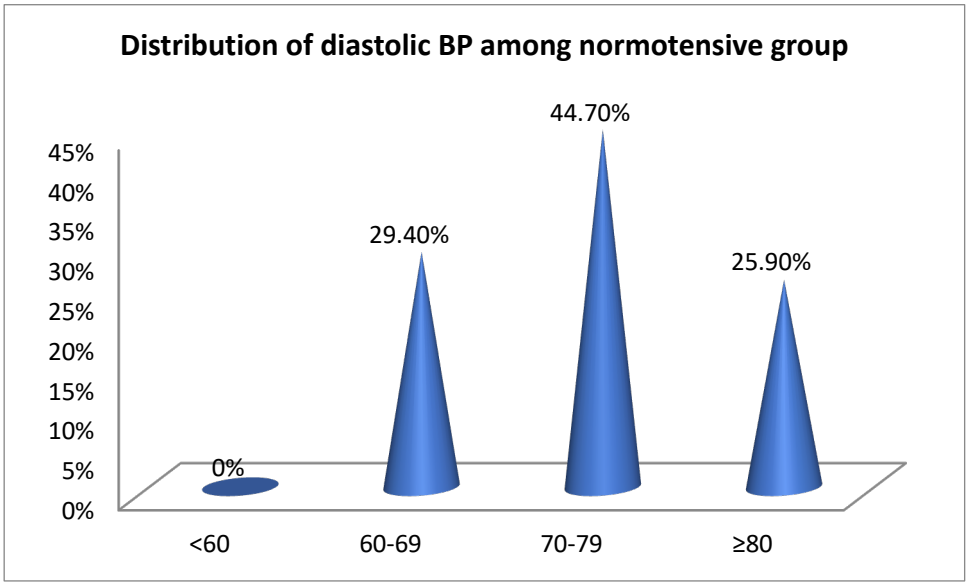
Graph 1c: Distribution of controls based on parity



Graph 1d: Distribution of controls based on systolic BP



Graph 1e: Distribution of controls based on diastolic BP



Characteristics of pre-eclampsia patients (cases)

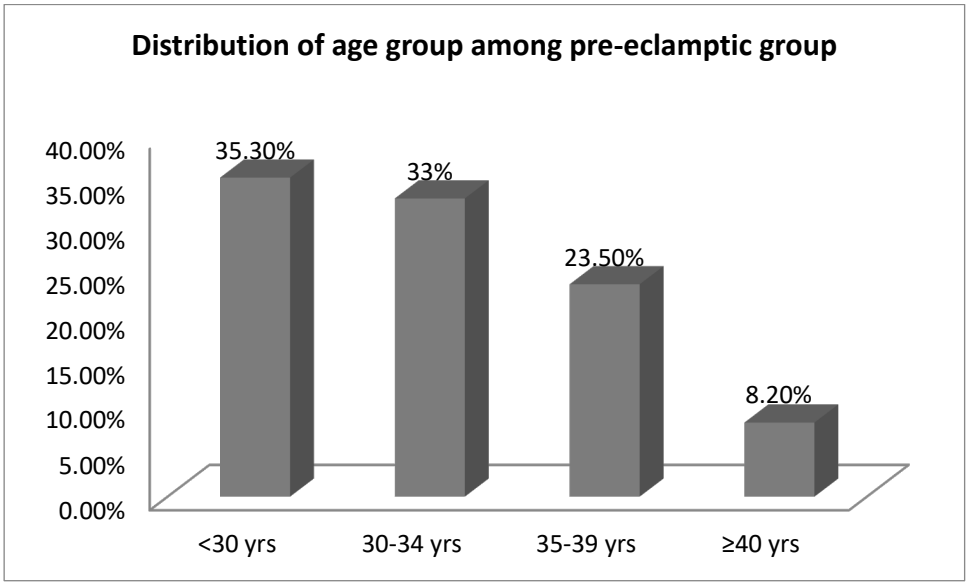
Among the 85 pre-eclampsia patients, the largest proportion (35.3%) were aged below 30 years, followed by 33% in the 30-34 age group. Additionally, 23.5% were aged 35-39 years, while 8.2% were aged 40 years or older. Gestational age (GA) distribution showed that more than half (53%) had a GA between 30-34 weeks, 35.3% had a GA between 35-39 weeks, and 11.7% had a GA of 40 weeks or more. None of the patients had a GA below 30 weeks. Parity distribution indicated that 35.3% were nulliparous, 29.4% had one previous delivery, 11.8% had parity 2, and 23.5% had parity of three or more. Systolic blood pressure (BP) was most commonly in the range of 160-179 mmHg (54.1%), followed by 22.3% with BP between 180-199 mmHg. Additionally, 17.6% had systolic BP in the range of 140-159 mmHg, while 5.9% had values of 200 mmHg or more. For diastolic BP, 58.8% had values between 90-109 mmHg, 17.6% had BP between 110-119 mmHg, and 18.8% had values between 120-139 mmHg. A small proportion (4.7%) had diastolic BP of 140 mmHg or higher.

Table 2: Demographic characteristics of cases

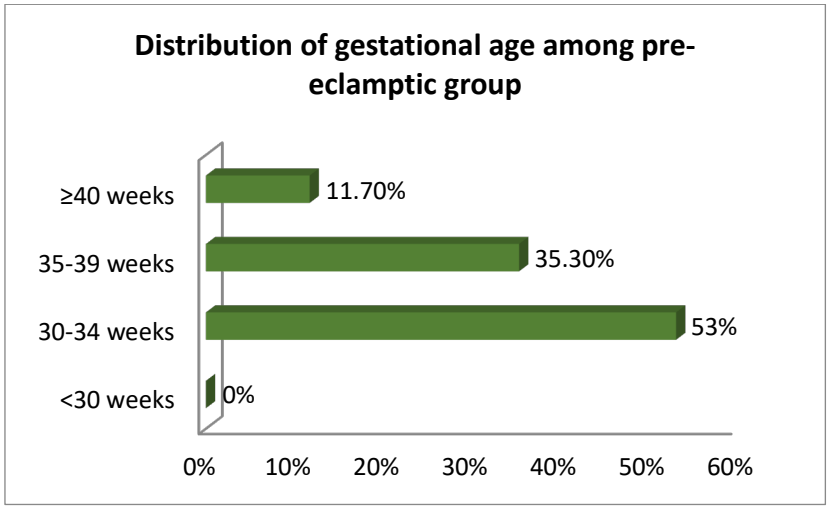
Characteristics	**Frequency (n=85)**	**Percentage**
Age in years		
<30	30	35.3
30-34	28	33
35-39	20	23.5
≥40	7	8.2
GA (weeks)		
<30	0	0
30-34	45	53
35-39	30	35.3
≥40	10	11.7
Parity		
0	30	35.3
1	25	29.4
2	10	11.8
≥3	20	23.5

Systolic BP		
140-159	15	17.6
160-179	46	54.1
180-199	19	22.3
≥200	5	5.9
Diastolic BP		
90-109	50	58.8
110-119	15	17.6
120-139	16	18.8
≥140	4	4.7

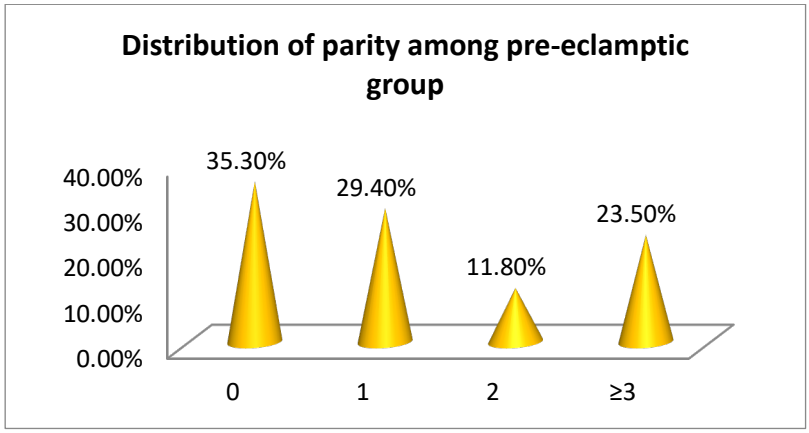
Graph 1a: Distribution of cases based on age



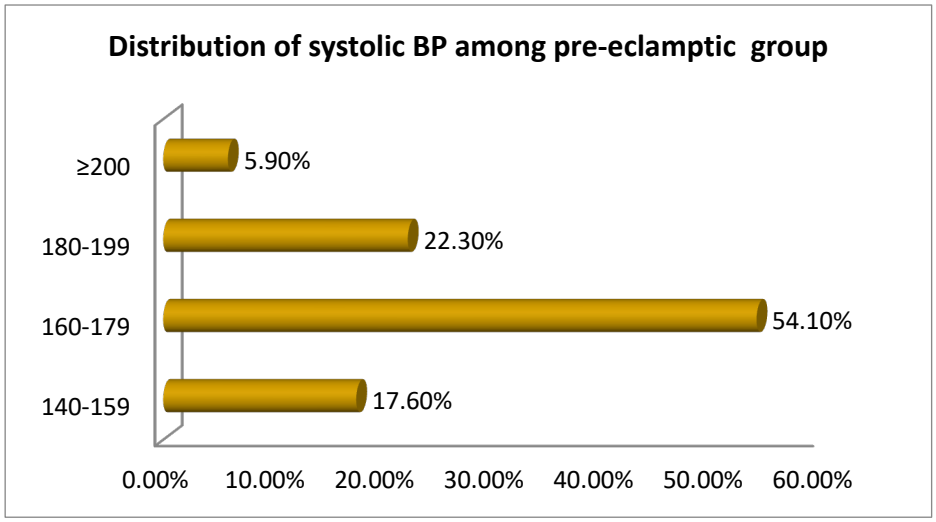
Graph 1b: Distribution of cases based on gestational age



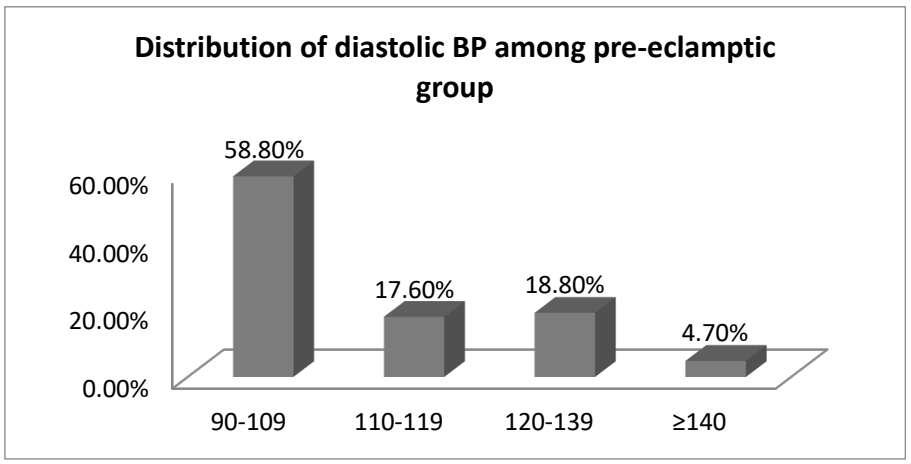
Graph 1c: Distribution of cases based on parity



Graph 1d: Distribution of cases based on systolic BP



Graph 1e: Distribution of cases based on diastolic BP



Characteristics of normotensive participants

Among normotensive participants, the mean pulse rate was 88.9 ± 8.42 beats per minute, while the mean hemoglobin level was 12.0 ± 11.9 g/dL. The total count (TC) had a mean value of 121.0 ± 947.9 cells/mm³, and the platelet count (PL) averaged 193.6 ± 64.8 × 10^3/mm³. Additionally, the mean serum albumin level was 0.38 ± 0.48 g/dL.

Table 3: Clinical and hematological parameters in normotensive patients

Characteristics	**Mean±SD**
Pulse rate	88.9±8.42
Haemoglobin	12.0±11.9
TC	121.0±947.9
PL	193.6±64.8
Albumin	0.38±0.48

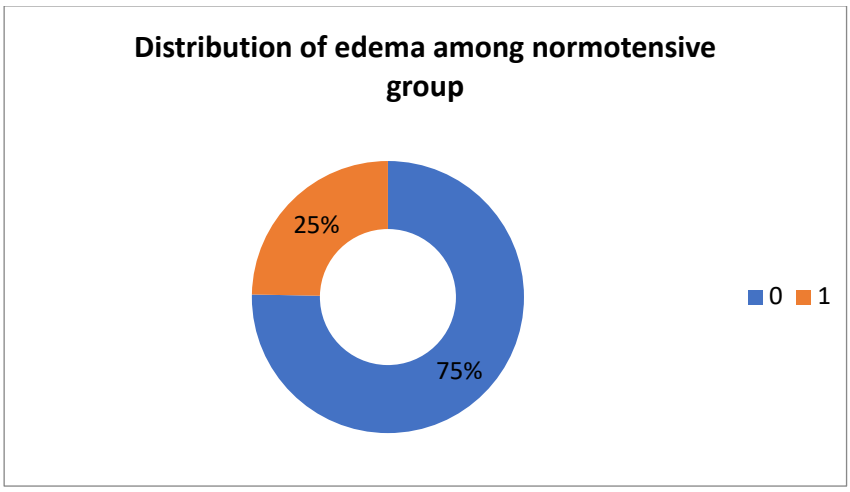
Distribution of edema among normotensive group

Among the normotensive group, the majority of participants (75.3%) had no edema, while 24.7% exhibited edema.

Table 4: Distribution of edema among normotensive group

Edema	**Frequency**	**Percentage**
No edema (0)	64	75.3
Edema present (1)	21	24.7
Total	85	100

Graph 3: Distribution of edema among normotensive group



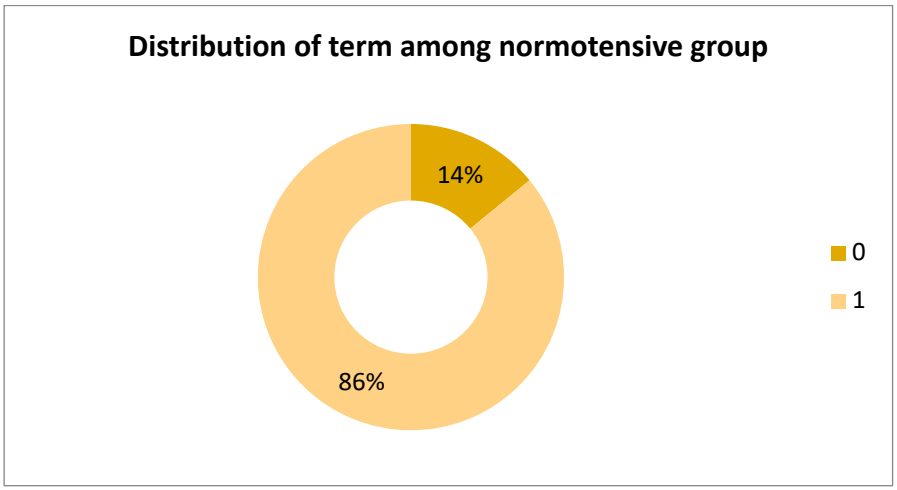
Distribution of term pregnancy among normotensive group

Among the normotensive group, 85.9% had term pregnancy.

Table 5: Distribution of term pregnancy among normotensive group

Term	Frequency	Percentage
Pre Term pregnancy (0)	12	14.1
Term pregnancy (1)	73	85.9
Total	85	100

Graph 4: Distribution of term pregnancy among normotensive group



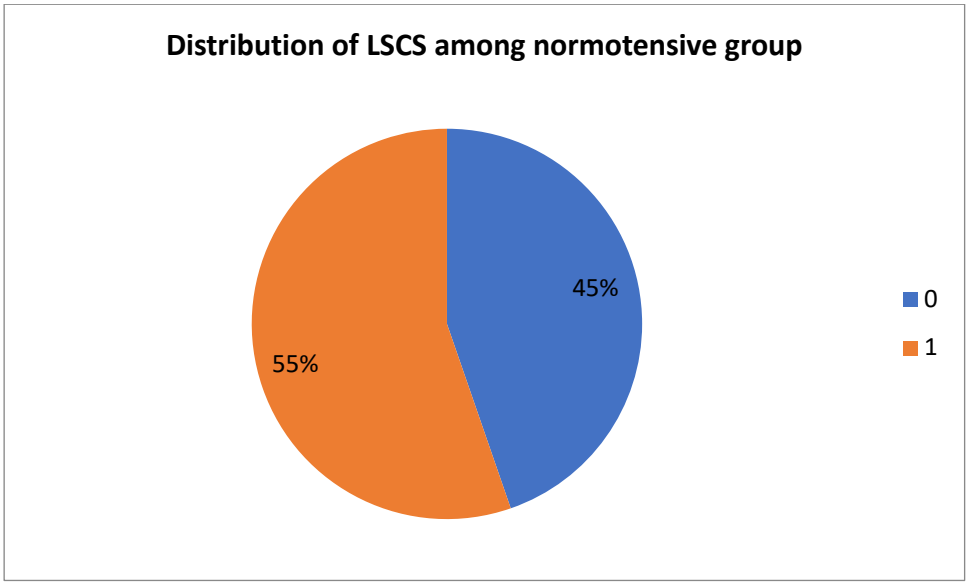
Distribution of LSCS among normotensive group

Among the normotensive group, 55.3% underwent LSCS, while 44.7% did not.

Table 6: Distribution of LSCS among normotensive group

LSCS	Frequency	Percentage
Vaginal delivery (0)	38	44.7
LSCS underwent (1)	47	55.3
Total	85	100

Graph 5: Distribution of LSCS among normotensive group



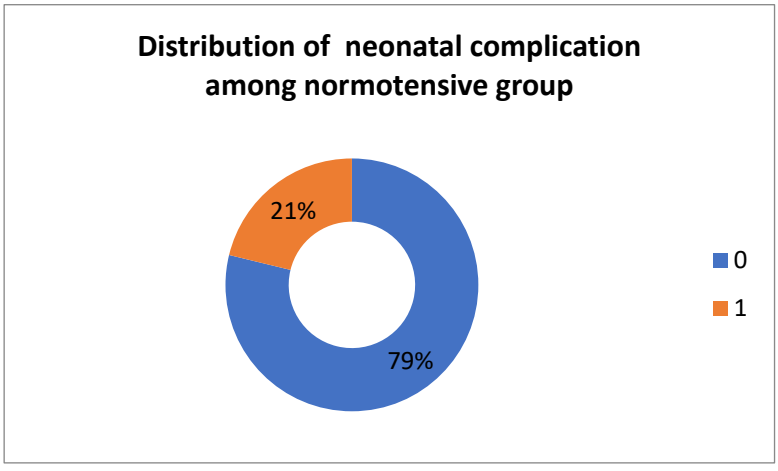
Distribution of neonatal complication among normotensive group

Among the normotensive group, neonatal complications were absent in 78.8% of cases, while 21.2% had neonatal complications.

Table 7: Distribution of neonatal complication among normotensive group

Neonatal complication	Frequency	Percentage
Absent (0)	67	78.8
Present (1)	18	21.2
Total	85	100

Graph 6: Distribution of neonatal complication among normotensive group



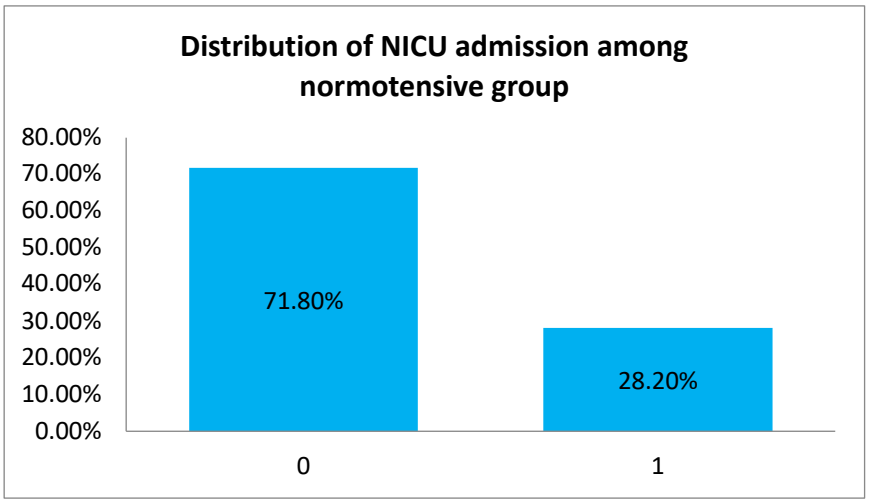
Distribution of NICU admission among normotensive group

Among the normotensive group, 71.8% of neonates did not require NICU admission, while 28.2% were admitted to the NICU.

Table 8: Distribution of NICU admission among normotensive group

NICU admission	Frequency	Percentage
Absent (0)	61	71.8
Present (1)	24	28.2
Total	85	100

Graph 7: Distribution of NICU admission among normotensive group



Clinical and haematological characteristics of pre-eclampsia participants

Among pre-eclampsia participants, the mean pulse rate was 89.23 ± 4.56 beats per minute, while the mean haemoglobin level was 10.39 ± 1.99 g/dL. The total count (TC) had a mean value of 13.4 ± 3.6 × 10^3/mm^3, and the platelet count (PLT) averaged 189.1 ± 71.3 × 10^3/mm^3. The mean serum albumin level was 0.96 ± 1.9 g/dL. Liver function parameters showed a mean SGOT level of 46.9 ± 20.5 U/L, SGPT of 36.6 ± 18.1 U/L, and alkaline phosphatase (ALP) of 133.7 ± 41.9 U/L. Renal function markers included a mean blood urea level of 26.7 ± 8.6 mg/dL and a mean serum creatinine level of 0.77 ± 0.22 mg/dL.

Table 9: Clinical and hematological characteristics of pre-eclampsia participants

Characteristics	Mean±SD
Pulse rate	89.23±4.56
Haemoglobin	10.39±1.99
TC	13.4±3.6
PLT	189.1±71.3
Albumin	0.96±1.9
SGOT	46.9±20.5
SGPT	36.6±18.1
ALP	133.7±41.9
Urea	26.7±8.6
Creatinine	0.77±0.22

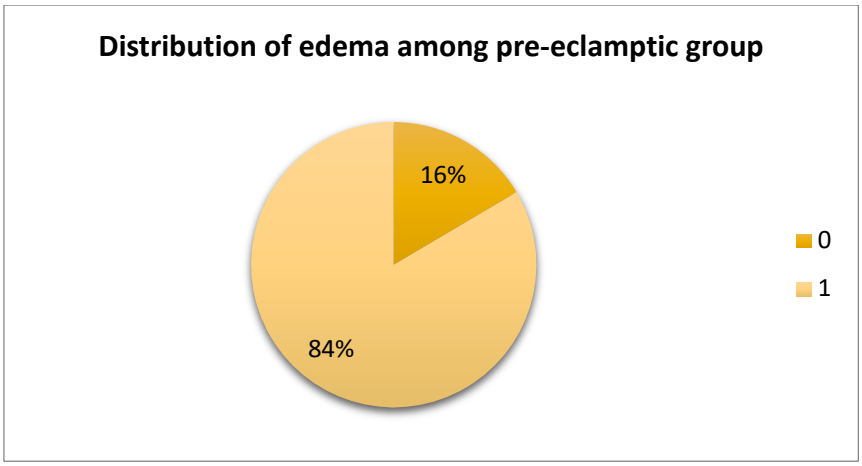
Distribution of edema among pre-eclamptic group

Among the pre-eclamptic group, edema was present in 83.5% of participants, while 16.5% had no edema.

Table 10: Distribution of edema among pre-eclamptic group

Edema	Frequency	Percentage
Absent (0)	14	16.5
Present (1)	71	83.5
Total	85	100

Graph 8: Distribution of edema among pre-eclamptic group



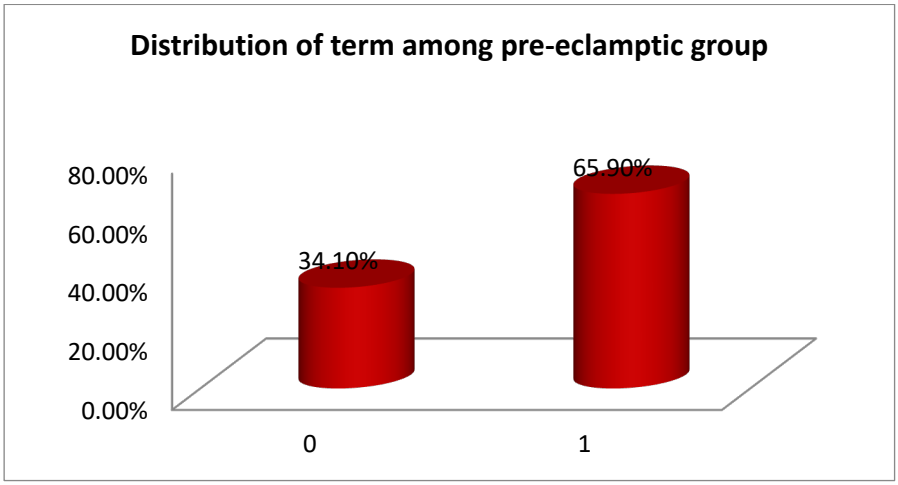
Distribution of term pregnancy among pre-eclamptic group

Among the pre-eclamptic group, 65.9% had a term pregnancy, while 34.1% were pre-term.

Table 11: Distribution of term pregnancy among pre-eclamptic group

Term	Frequency	Percentage
Preterm	29	34.1
Term	56	65.9
Total	85	100

Graph 9: Distribution of term pregnancy among pre-eclamptic group



Distribution of type of delivery among study population

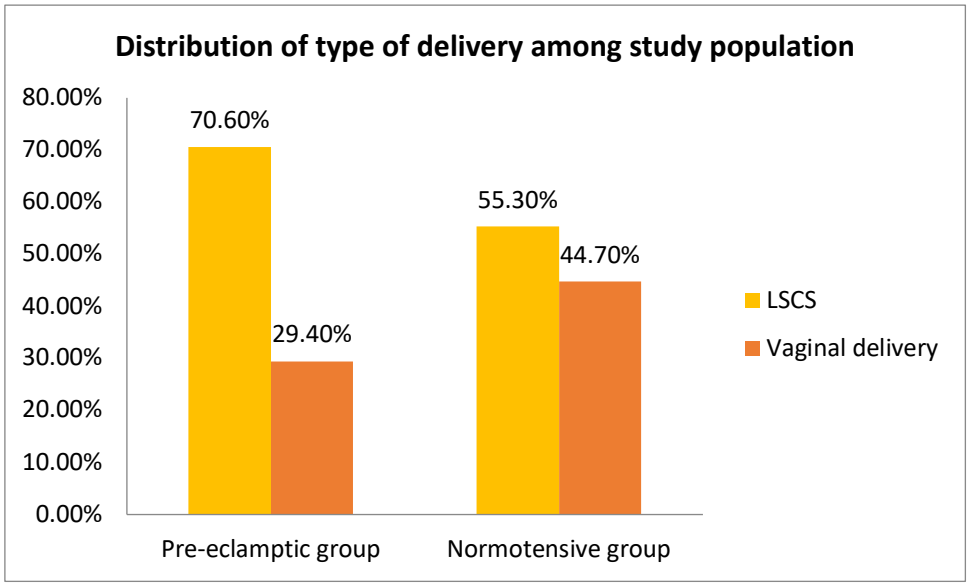
Among the pre-eclamptic group, 70.6% underwent LSCS, while 29.4% had vaginal delivery.

Among the normotensives, 55.3% had undergone LSCS, while 44.7% had vaginal delivery.

Table 12: Distribution of type of delivery among study population

Type of delivery	Pre-eclamptic group		Normotensive group	
	n	%	n	%
LSCS	60	70.6	47	55.3
Vaginal delivery	25	29.4	38	44.7
Total	85	100	85	100

Graph 10: Distribution of type of delivery among study population



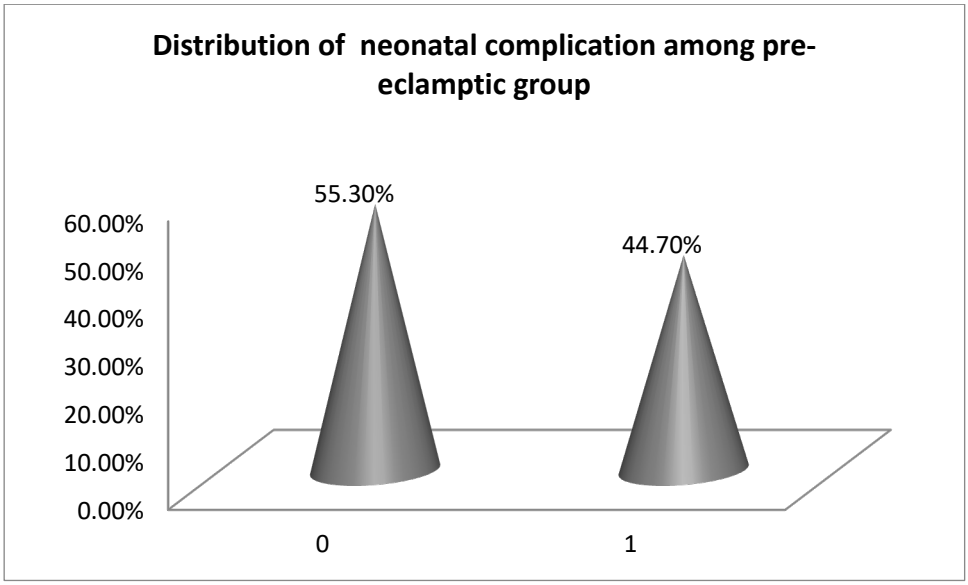
Distribution of neonatal complication among pre-eclamptic group

Among the pre-eclamptic group, neonatal complications were present in 44.7% of cases, while 55.3% had no complications.

Table 13: Distribution of neonatal complication among pre-eclamptic group

Neonatal complication	Frequency	Percentage
Absent (0)	47	55.3
Present (1)	38	44.7
Total	85	100

Graph 11: Distribution of neonatal complication among pre-eclamptic group



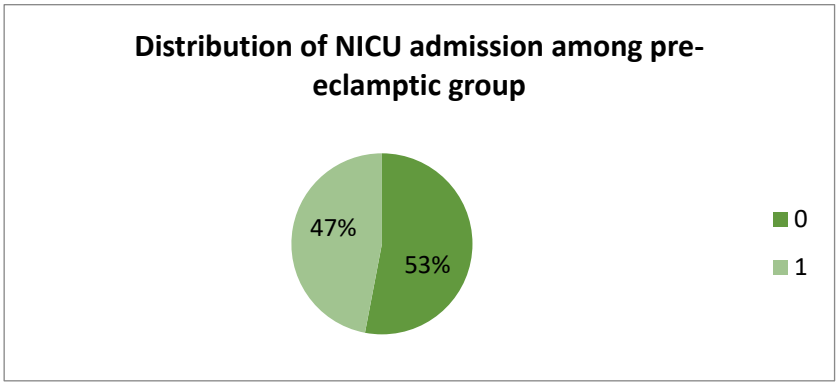
Distribution of NICU admission among pre-eclamptic group

Among the pre-eclamptic group, 47% of neonates required NICU admission, while 53% did not.

Table 14: Distribution of NICU admission among pre-eclamptic group

NICU admission	Frequency	Percentage
Absent (0)	45	53
Present (1)	40	47
Total	85	100

Graph 12: Distribution of NICU admission among pre-eclamptic group



Comparison of mean values of Ophthalmic Artery Doppler (OAD) parameters between the pre-eclampsia group and control group

The mean values of ophthalmic artery Doppler (OAD) parameters were compared between the pre-eclamptic and control groups. The right resistive index (RI) was higher in the pre-eclamptic group (0.73 ± 0.54) compared to the control group (0.71 ± 0.21), with a statistically significant p-value of 0.00. Similarly, the left RI was significantly lower in the pre-eclamptic group (0.69 ± 0.65) than in the control group (0.71 ± 0.12, p = 0.01). For pulsatility index (PI), both right and left PI were significantly lower in the pre-eclamptic group (1.3 ± 0.62 and 1.24 ± 0.45) than in the control group (1.78 ± 0.56 and 1.58 ± 0.53), with p-values of 0.00. Regarding peak systolic velocity (PSV), the right and left PSV values were lower in the pre-eclamptic group (29.25 ± 8.45 cm/sec and 29.76 ± 9.67 cm/sec) compared to the control group (32.10 ± 13.45 cm/sec and 34.02 ± 12.78 cm/sec), but the differences were not statistically significant (p = 0.16 and p = 0.18, respectively). For pre-diastolic velocity (PDV), the right PDV was significantly higher in pre-eclamptic participants (23.10 ± 6.45 cm/sec) than in the control group (19.87 ± 7.85 cm/sec, p = 0.04). Similarly, the left PDV was significantly higher in the pre-eclamptic group (25.67 ± 9.95 cm/sec) than in the control group (20.45 ± 7.56 cm/sec, p = 0.00). Finally, end-diastolic velocity (EDV) values were slightly higher in the pre-eclamptic group for both right (11.02 ± 4.56 cm/sec) and left (11.67 ± 3.54 cm/sec) sides, compared to the control group (8.23 ± 4.67 cm/sec and 7.98 ± 4.12 cm/sec). The statistical significance was also observed for the left EDV (p = 0.05) as well as right EDV (p = 0.04).

Table 15: Comparison of mean values of Ophthalmic Artery Doppler (OAD) parameters between the pre-eclampsia group and control group

Variables	Pre-eclamptic	Control group	T test	P value
Right RI Mean±SD	0.73±0.54	0.71±0.21	-4.43	0.00
Left RI Mean±SD	0.69±0.65	0.71±0.12	-3.89	0.01
Right PI Mean±SD	1.3±0.62	1.78±0.56	-3.43	0.00
Left PI Mean±SD	1.24±0.45	1.58±0.53	-4.56	0.00
Right PSV (cm/sec) Mean±SD	29.25±8.45	32.10±13.45	-2.02	0.16
Left PSV (cm/sec) Mean±SD	29.76±9.67	34.02±12.78	-1.85	0.18
Right PDV (cm/sec) Mean±SD	23.10±6.45	19.87±7.85	-2.37	0.04
Left PDV(cm/sec) Mean±SD	25.67±9.95	20.45±7.56	3.89	0.00
Right EDV(cm/sec) Mean±SD	11.02±4.56	8.23±4.67	3.46	0.04
Left EDV(cm/sec) Mean±SD	11.67±3.54	7.98±4.12	3.32	0.05

Comparison of BP and OAD metrics between patients with mild and severe pre-eclampsia

The comparison of blood pressure (BP) and ophthalmic artery Doppler (OAD) parameters between patients with mild and severe pre-eclampsia revealed significant differences in certain variables.

Blood Pressure (BP):

Patients with severe pre-eclampsia had significantly higher systolic blood pressure (SBP) (179.34 ± 22.53 mmHg) compared to those with mild pre-eclampsia (148.78 ± 8.65 mmHg, p = 0.00). Similarly, diastolic blood pressure (DBP) was significantly elevated in the severe pre-eclampsia group (110 ± 15.48 mmHg) compared to the mild group (98.23 ± 5.08 mmHg, p = 0.02).

Ophthalmic Artery Doppler (OAD) Parameters:

The right resistive index (RI) was significantly lower in the severe pre-eclampsia group (0.62 ± 0.58) than in the mild pre-eclampsia group (0.71 ± 0.76, p = 0.01). Similarly, the right pulsatility index (PI) was significantly reduced in severe cases (1.06 ± 0.37) compared to mild pre-eclampsia (1.27 ± 1.78, p = 0.03), suggesting altered vascular resistance in severe cases.

Other OAD parameters, including right peak systolic velocity (PSV), right pre-diastolic velocity (PDV), and right end-diastolic velocity (EDV), showed no significant differences between the groups (p > 0.05), indicating that while vascular resistance was affected, overall blood flow velocities did not significantly differ.

Table 16: Comparison of BP and OAD metrics between patients with mild and severe pre-eclampsia

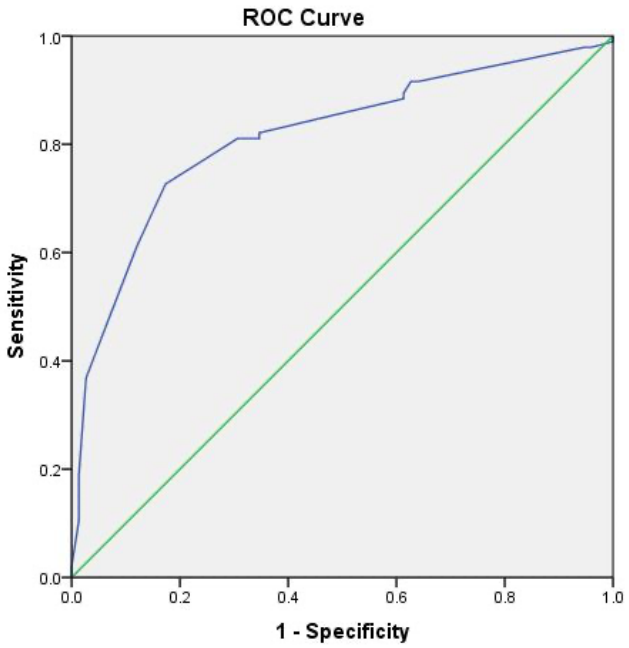
Variables	**Mild PE (n=46)**	**Severe PE (n=39)**	**t value**	**P value**
SBP (mmhg) Mean±SD	148.78±8.65	179.34±22.53	5.67	0.00
DBP (mmhg) Mean±SD	98.23±5.08	110±15.48	6.45	0.02
Right RI Mean±SD	0.71±0.76	0.62±0.58	-2.98	0.01
Right PI Mean±SD	1.27±1.78	1.06±0.37	-2.89	0.03
Right PSV (cm/sec) Mean±SD	29.55±9.56	26.80±9.76	-0.44	0.78
Right PDV (cm/sec) Mean±SD	22.39±6.87	22.46±9.85	0.87	0.88
Right EDV(cm/sec) Mean±SD	9.73±3.03	10.41±4.39	0.98	0.57

ROC curve of Resistivity Index

The ROC curve confirms that Resistivity Index (RI) is a reliable marker for diagnosing pre-eclampsia, with an optimal cut-off of 0.77 and 83% sensitivity. This suggests that ophthalmic artery Doppler (OAD) can play a significant role in assessing cerebral hemodynamic changes in pre-eclamptic patients.

Resistivity Index (RI): AUC = 0.83 (95% CI: 0.75 - 0.90)

Graph 13: ROC curve of Resistivity Index

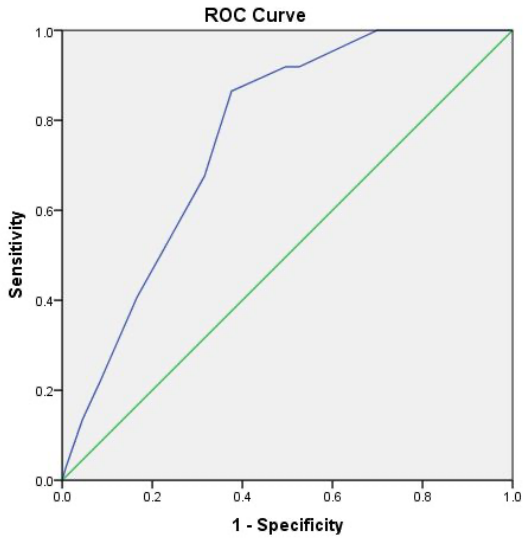


ROC curve of pulsatility index

The ROC curve confirms that Pulsatility Index (PI) is a highly effective marker for diagnosing pre-eclampsia, with a cut-off of 1.21 and 90% sensitivity. The findings suggest PI can reliably indicate altered cerebrovascular resistance in pre-eclamptic patients.

Pulsatility Index (PI): AUC = 0.90 (95% CI: 0.84 - 0.95)

Graph 14: ROC curve of pulsatility index

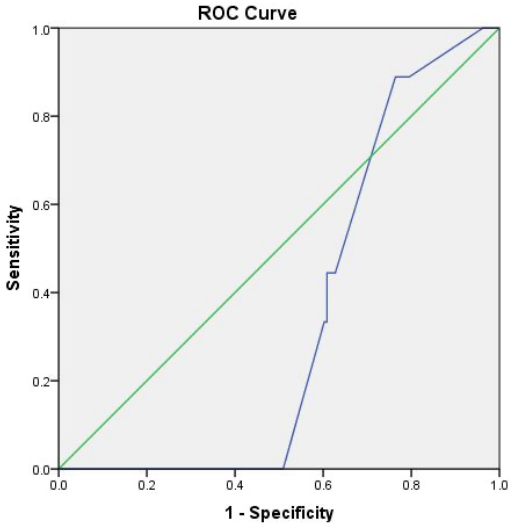


ROC curve of PSV

The ROC curve suggests that Peak Systolic Velocity (PSV) at a cut-off of 35 has a sensitivity of 60%, making it a less effective standalone marker for pre-eclampsia.

Peak Systolic Velocity (PSV): AUC = 0.60 (95% CI: 0.50 - 0.70)

Graph 15: ROC curve of PSV

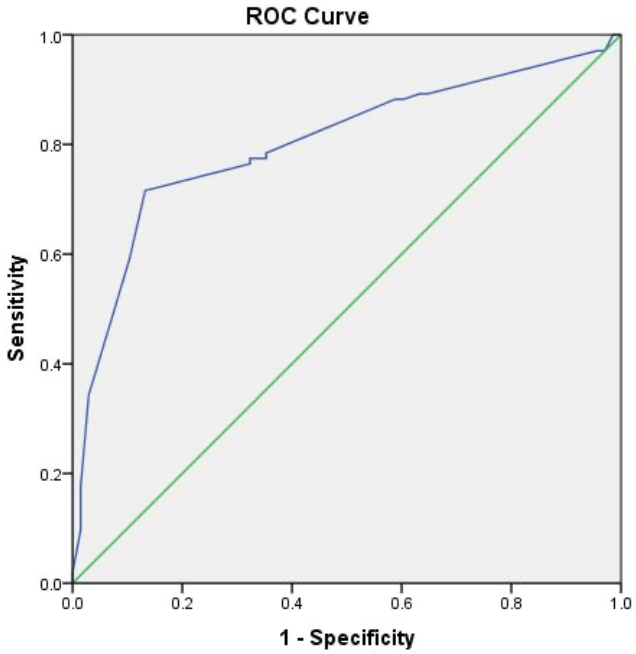


ROC curve of EDV

The ROC curve suggests that End-Diastolic Velocity (EDV) at a cut-off of 7 has a sensitivity of 75%, making it a moderate predictor for pre-eclampsia.

End-Diastolic Velocity (EDV): AUC = 0.75 (95% CI: 0.66 - 0.83)

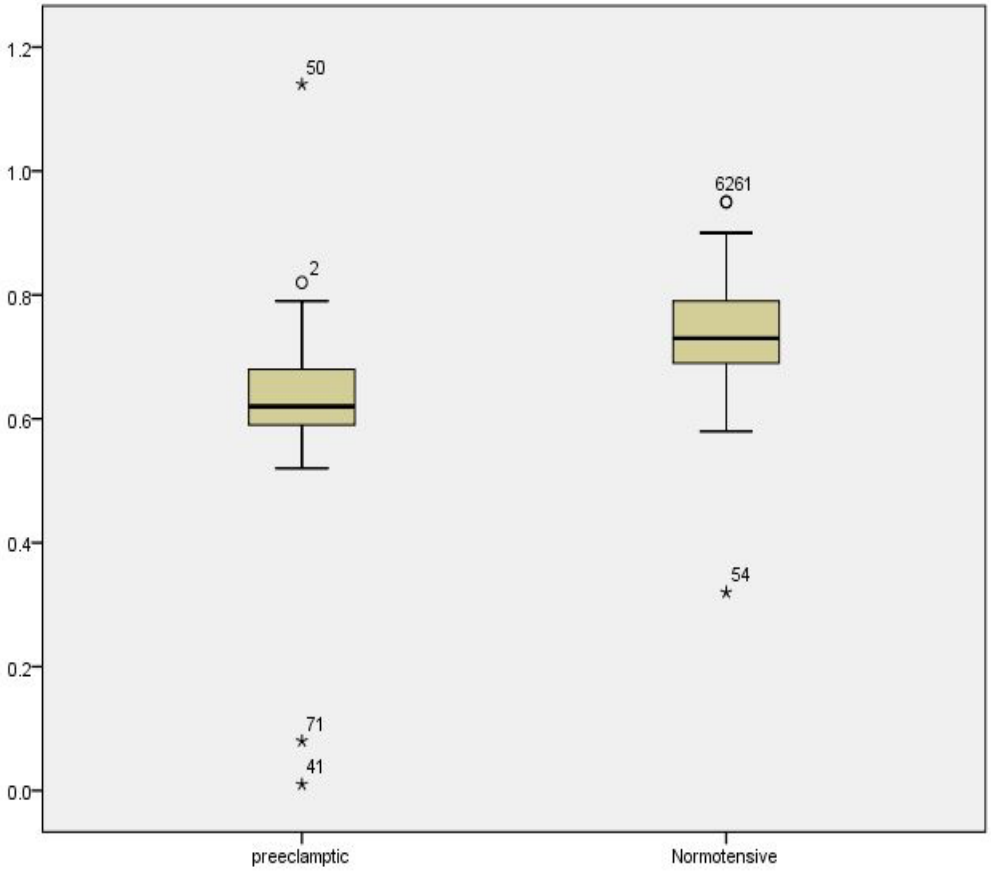
Graph 16: ROC curve of EDV



Box Plot of RI

The box plot shows that preeclamptic patients tend to have lower RI values, while normotensive individuals have higher RI values. This suggests that blood flow resistance is different between the two groups. The greater variation in RI among preeclamptic patients indicates that their blood flow patterns are more irregular. The presence of extremely low values in preeclampsia may reflect more severe cases. Overall, this implies that preeclampsia affects blood circulation, potentially leading to complications.

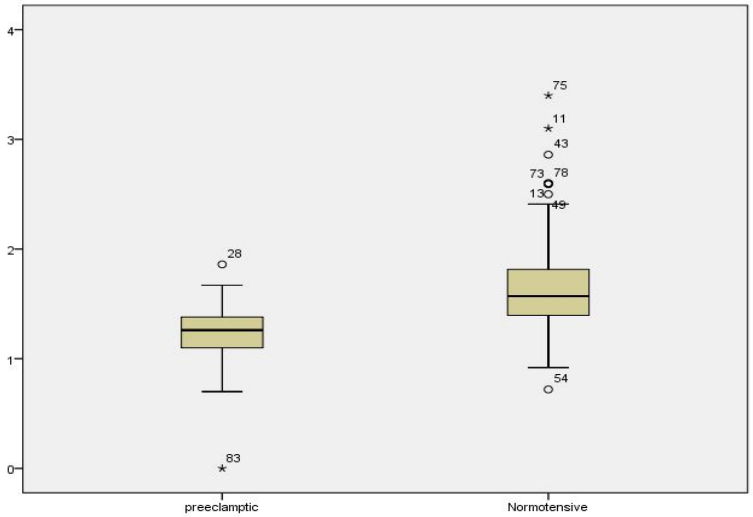
Graph 17: Box Plot of RI



Box plot of pulsatility index

This box plot compares a specific measurement between preeclamptic and normotensive individuals. Normotensive individuals show greater variation and higher values, while preeclamptic individuals have more stable and lower values. The wider range and presence of outliers in the normotensive group suggest that their values are more spread out, while the preeclamptic group has a more consistent pattern. This implies that preeclampsia affects this measurement, possibly leading to reduced blood flow efficiency.

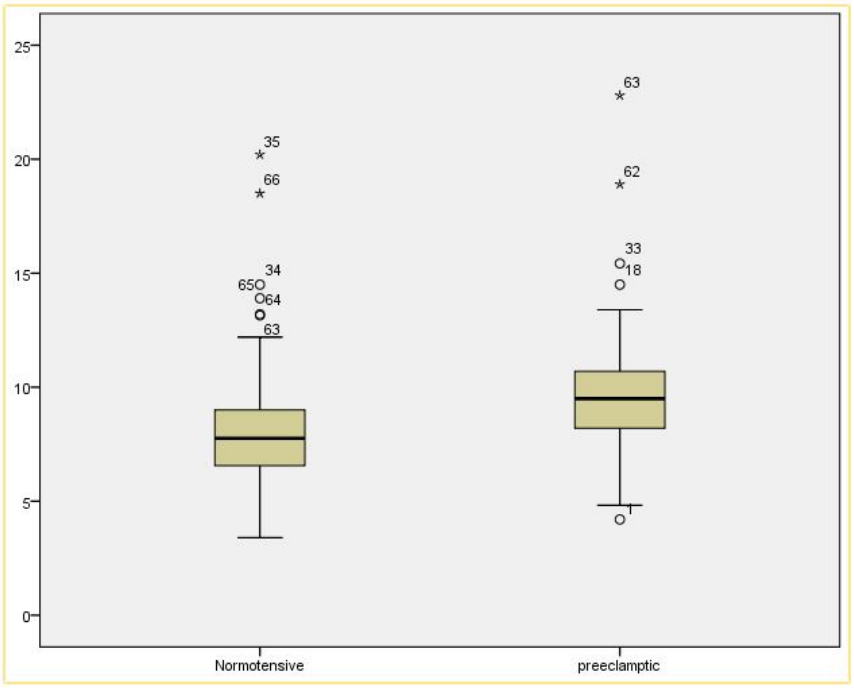
Graph 18: Box plot of pulsatility index



Box plot of EDV

The box plot compares the distribution of End-Diastolic Volume (EDV) between normotensive and preeclamptic patients. The median EDV appears slightly higher in preeclamptic patients, while the interquartile range (IQR) is similar between both groups, indicating a comparable spread of values. The whiskers extend to a similar range in both groups, suggesting that most EDV values fall within a stable range. However, both groups exhibit multiple outliers, with some patients showing significantly higher EDV values, reflecting individual variations in cardiac function. The slightly elevated median EDV in preeclamptic patients may indicate altered cardiac function due to increased vascular resistance or fluid shifts associated with the condition. Despite this, the overall distribution remains similar, suggesting that significant deviations in EDV are likely influenced by individual physiological differences rather than preeclampsia alone.

Graph 19: Box plot of EDV



DISCUSSION

Pre-eclampsia is a leading cause of maternal mortality, and reducing its associated risks depends on three key factors: accurately predicting its onset, monitoring its progression, and identifying preventive measures. However, there is currently no effective intervention that significantly prevents the condition. Therefore, research efforts should prioritize enhancing predictive methods for early detection and assessing potential complications.

This study evaluated the association between maternal ophthalmic artery Doppler parameters and pre-eclampsia by comparing Doppler indices between pre-eclamptic and normotensive pregnant women. Additionally, differences between mild and severe pre-eclampsia were analyzed, along with maternal and neonatal outcomes.

The study included a total of 170 participants, with 85 pre-eclamptic patients (case group) and 85 normotensive pregnant women (control group).

Demographic and Clinical Characteristics

The most common age group in this study among normotensive women was 30-34 years (40%), whereas in pre-eclamptic patients, it was below 30 years (35.3%). This may be because younger women are more likely to have endothelial dysfunction and an exaggerated inflammatory response, predisposing them to pre-eclampsia. This is in contrast to the study by Ananth et al that documented increased PE among 30–34 year old women.[54] Another study by Olatunji RB et al,[1] stated higher mean age of pre-eclamptic patients compared to our study.

In our study, Gestational age (GA) was lower in pre-eclamptic women, with 53% having GA between 30-34 weeks compared to 63.5% of normotensive women having GA

between 35-39 weeks. This is in accordance with the studies by other researchers where the mean GA was 32±2 weeks for patients with pre-eclampsia.[55-58] The increased frequency of preterm delivery in pre-eclamptic patients in our study may be due to placental insufficiency and the need for early intervention to prevent maternal and fetal complications.

Parity distribution in our study showed that para 1 was the most common among normotensive women (41.2%), whereas nulliparity was more frequent in pre-eclamptic patients (35.3%). Nulliparous women have a higher risk of developing pre-eclampsia due to an immunological maladaptation to paternal antigens, leading to abnormal placentation.

Blood pressure differences were significant. Among normotensive women, the highest proportion (40%) had systolic BP of 110-119 mmHg, while in pre-eclamptic patients, most (54.1%) had BP between 160-179 mmHg. Similarly, diastolic BP was predominantly between 90-109 mmHg (58.8%) in pre-eclampsia cases, while in normotensive women, 44.7% had BP of 70-79 mmHg. The higher blood pressure in pre-eclampsia results from increased systemic vascular resistance, endothelial dysfunction, and an imbalance between vasodilatory and vasoconstrictive factors.

Similarly, edema was observed majorly among pre-eclamptic patients (83.5%) compared to 24.7% in normotensive patients. This can be attributed to endothelial damage and increased capillary permeability, leading to fluid extravasation and tissue swelling.

Hematological and Biochemical Parameters

Mean haemoglobin levels were lower in pre-eclamptic patients (10.39 ± 1.99 g/dL) compared to normotensive women (12.0 ± 11.9 g/dL). Anaemia in pre-eclampsia may result

from hemolysis due to microangiopathic changes and increased plasma volume expansion. The total count was elevated in pre-eclampsia ($13.4 \pm 3.6 \times 10^3/mm^3$), likely due to an inflammatory state and increased leukocyte activation in response to endothelial dysfunction. Platelet count was slightly lower ($189.1 \pm 71.3 \times 10^3/mm^3$ vs. $193.6 \pm 64.8 \times 10^3/mm^3$), possibly due to platelet consumption in microthrombi formation and the pro-coagulant state associated with pre-eclampsia. Serum albumin was significantly higher in pre-eclampsia (0.96 ± 1.9 g/dL vs. 0.38 ± 0.48 g/dL), which may be linked to increased protein leakage due to endothelial dysfunction and altered hepatic function in severe cases.

Maternal and Neonatal Outcomes

Preterm delivery was more common in pre-eclampsia (34.1%) compared to normotensive women (14.1%). This is often due to iatrogenic preterm birth induced to prevent maternal and fetal complications arising from worsening hypertension and placental insufficiency. The rate of LSCS was higher in pre-eclampsia (70.6% vs. 55.3%), possibly due to fetal distress, failure to progress in labor due to vascular compromise, and the higher incidence of complications such as abruptio placentae. Neonatal complications were more frequent (44.7% vs. 21.2%), with NICU admissions required in 47% of neonates born to pre-eclamptic mothers compared to 28.2% in the normotensive group. This could be due to prematurity, intrauterine growth restriction (IUGR), and respiratory distress syndrome secondary to placental insufficiency.

Comparison of Ophthalmic Artery Doppler (OAD) Parameters

OAD parameters showed significant differences. Right RI was higher in pre-eclamptic patients (0.73 ± 0.54 vs. 0.71 ± 0.21, $p = 0.00$), while left RI was lower ($0.69 \pm$

0.65 vs. 0.71 ± 0.12, p = 0.01). The higher resistance in the right ophthalmic artery suggests increased cerebrovascular resistance, possibly due to vasoconstriction secondary to elevated blood pressure. Both right and left PI were lower in pre-eclampsia (1.3 ± 0.62 and 1.24 ± 0.45 vs. 1.78 ± 0.56 and 1.58 ± 0.53, p = 0.00), indicating altered cerebral autoregulation and compensatory vasodilation in response to hypertension-induced hypoperfusion. The lower values of RI and PI found in our study are similar to the findings of several studies by other researchers, where they noted lower values of PI and RI in pre-eclampsia.[59-61] However, our study contradicts those studies in the fact that right RI was higher in our pre-eclampsia population.

Pre-diastolic velocity (PDV) was significantly higher in pre-eclampsia for both right (23.10 ± 6.45 cm/sec vs. 19.87 ± 7.85 cm/sec, p = 0.04) and left (25.67 ± 9.95 cm/sec vs. 20.45 ± 7.56 cm/sec, p = 0.00). EDV was also significantly higher among the pre-eclampsia group for both right and left eyes. This may reflect increased downstream resistance due to endothelial dysfunction. However, there were no significant differences observed in right and left PSV between normotensive and pre-eclamptic patients, possibly due to individual variations in vascular response and autoregulation mechanisms.

Differences Between Mild and Severe Pre-Eclampsia

Blood pressure was significantly higher in severe pre-eclampsia (SBP: 179.34 ± 22.53 mmHg vs. 148.78 ± 8.65 mmHg, p = 0.00; DBP: 110 ± 15.48 mmHg vs. 98.23 ± 5.08 mmHg, p = 0.02). This may be due to greater endothelial dysfunction and exaggerated vasoconstriction in severe disease. Right RI was lower in severe cases (0.62 ± 0.58 vs. 0.71 ± 0.76, p = 0.01), and right PI was also lower (1.06 ± 0.37 vs. 1.27 ± 1.78, p = 0.03), which may indicate cerebral autoregulatory mechanisms attempting to maintain perfusion in response to systemic hypertension. But these values are in contrast to several studies that

state that higher level of RI was observed in severe pre-eclampsia cases.[62,63] There were no significant differences between mild and severe pre-eclampsia groups in terms of right PSV, right PDV, and right EDV, suggesting that beyond a certain severity threshold, the cerebrovascular compensatory mechanisms reach a plateau.

Diagnostic Value of OAD Parameters

ROC analysis revealed RI (cut-off: 0.77, 83% sensitivity) and PI (cut-off: 1.21, 90% sensitivity) as reliable markers for pre-eclampsia. This shows that RI values below 0.77 suggest progression to severe PE. This is in close comparison to the cutoff value of 0.657 (sensitivity 73.3%, specificity 88.8%) defined by de Oliveira et al[56] in their Brazilian population. The higher sensitivity of RI and PI may be due to their direct correlation with increased cerebrovascular resistance and impaired vasodilation. PSV (cut-off: 35, 60% sensitivity) was less effective, while EDV (cut-off: 7, 75% sensitivity) showed moderate predictive ability. The limited predictive value of PSV may be attributed to individual hemodynamic variations, while increased variability in RI suggests altered cerebrovascular hemodynamics in pre-eclamptic patients, possibly due to persistent hypertension and endothelial dysfunction.

SUMMARY

Age Distribution

The most common age group among normotensive women was 30-34 years (40%), while in pre-eclamptic patients, 35.3% were below 30 years. This suggests that younger women may have a higher predisposition to pre-eclampsia, possibly due to endothelial dysfunction and an exaggerated inflammatory response.

Gestational Age (GA)

The majority of normotensive women had GA 35-39 weeks (63.5%), whereas pre-eclamptic women had a lower GA, with 53% between 30-34 weeks. This indicates a higher incidence of preterm delivery in pre-eclamptic patients, likely due to placental insufficiency and the need for early intervention.

Parity Distribution

Among normotensive women, 41.2% were para 1, whereas among pre-eclamptic patients, nulliparity was more common (35.3%). Nulliparous women are at a higher risk of developing pre-eclampsia due to immunological maladaptation and abnormal placentation.

Blood Pressure (BP)

- Systolic BP: Most normotensive women had 110-119 mmHg (40%), whereas pre-eclamptic patients predominantly had 160-179 mmHg (54.1%).
- Diastolic BP: Among normotensives, 44.7% had BP 70-79 mmHg, while in pre-eclamptic patients, 58.8% had 90-109 mmHg.
- The higher BP in pre-eclampsia is due to increased systemic vascular resistance, endothelial dysfunction, and an imbalance between vasodilatory and vasoconstrictive factors.

Edema

83.5% of pre-eclamptic patients had edema, compared to only 24.7% in normotensive women. The presence of edema in pre-eclampsia is linked to endothelial damage, increased capillary permeability, and fluid extravasation.

Hematological and Biochemical Parameters

- Hemoglobin levels were lower in pre-eclampsia (10.39 ± 1.99 g/dL) than in normotensive women (12.0 ± 11.9 g/dL), likely due to hemolysis and plasma volume expansion.
- Total count (TC) was significantly elevated in pre-eclampsia (13.4 ± 3.6 × 10^3/mm^3) compared to normotensives (121.0 ± 947.9 cells/mm^3), suggesting an inflammatory state.
- Platelet count (PLT) was slightly lower in pre-eclampsia (189.1 ± 71.3 × 10^3/mm^3 vs. 193.6 ± 64.8 × 10^3/mm^3), reflecting platelet consumption in microthrombi formation.
- Serum albumin levels were significantly higher in pre-eclamptic patients (0.96 ± 1.9 g/dL) compared to normotensive women (0.38 ± 0.48 g/dL), likely due to protein leakage and hepatic dysfunction.

Maternal and Neonatal Outcomes

- Preterm delivery was more frequent in pre-eclampsia (34.1% vs. 14.1% in normotensives), likely due to placental insufficiency.
- LSCS rate was higher in pre-eclampsia (70.6% vs. 55.3% in normotensives), often due to fetal distress and labor complications.
- Neonatal complications were more common in pre-eclampsia (44.7% vs. 21.2% in normotensive women).

- NICU admissions were required in 47% of pre-eclamptic neonates compared to 28.2% in normotensives, possibly due to IUGR and respiratory distress syndrome.

Ophthalmic Artery Doppler (OAD) Parameters

- Resistive Index (RI) was higher on the right in pre-eclampsia (0.73 ± 0.54 vs. 0.71 ± 0.21, p = 0.00) but lower on the left (0.69 ± 0.65 vs. 0.71 ± 0.12, p = 0.01).
- Pulsatility Index (PI) was significantly lower in pre-eclamptic patients (1.3 ± 0.62 and 1.24 ± 0.45) compared to normotensive controls (1.78 ± 0.56 and 1.58 ± 0.53, p = 0.00), suggesting altered cerebral autoregulation.
- Pre-diastolic velocity (PDV) was significantly higher in pre-eclamptic patients for both right and left eyes (p = 0.04 and p = 0.00, respectively), possibly reflecting increased downstream resistance.
- End-diastolic velocity (EDV) was also significantly higher in pre-eclamptic women for both eyes.

ROC Curve Analysis

- Resistivity Index (RI) had an AUC of 0.83, with a cut-off of 0.77 and 83% sensitivity, making it a reliable marker for pre-eclampsia.
- The Pulsatility Index (PI) was even more effective, with an AUC of 0.90, a cut-off of 1.21, and 90% sensitivity, indicating significant cerebrovascular alterations.
- Peak Systolic Velocity (PSV) had a cut-off of 35 cm/sec but only 60% sensitivity, making it a less effective standalone marker.
- End-diastolic velocity (EDV) had a cut-off of 7 cm/sec and 75% sensitivity, suggesting moderate predictive ability.

Box Plot Analysis

- Resistivity Index (RI) Box Plot: Pre-eclamptic patients had lower RI values, indicating altered blood flow resistance and greater variability, which may reflect disease severity.
- Pulsatility Index (PI) Box Plot: Normotensive individuals had higher and more variable PI, while pre-eclamptic patients had more stable but lower values, signifying reduced cerebrovascular efficiency.
- End-Diastolic Velocity (EDV) Box Plot: There was a greater range of EDV values in pre-eclampsia, with some outliers showing extreme variations, which might suggest underlying circulatory dysfunction.

Comparison of Mild and Severe Pre-Eclampsia

- Severe pre-eclampsia had significantly higher SBP (179.34 ± 22.53 mmHg) and DBP (110 ± 15.48 mmHg) compared to mild pre-eclampsia (148.78 ± 8.65 mmHg and 98.23 ± 5.08 mmHg, $p < 0.05$).
- Right RI was lower in severe cases (0.62 ± 0.58 vs. 0.71 ± 0.76, $p = 0.01$), while Right PI was also reduced (1.06 ± 0.37 vs. 1.27 ± 1.78, $p = 0.03$), indicating more severe cerebral vascular alterations.
- No significant differences were observed in PSV, PDV, and EDV between mild and severe cases.

CONCLUSION

This study highlights the significant alterations in maternal ophthalmic artery Doppler parameters in pre-eclamptic women compared to normotensive pregnant women. Increased resistance in the right ophthalmic artery and lower pulsatility indices suggest cerebrovascular dysfunction and impaired autoregulation in pre-eclampsia. The study also demonstrates that resistivity index and pulsatility index serve as reliable markers for predicting the severity of pre-eclampsia, with lower RI values indicating disease progression. Additionally, pre-eclamptic patients exhibited higher rates of adverse maternal and neonatal outcomes, including preterm delivery, increased cesarean section rates, and neonatal complications requiring NICU admission. Despite its limitations, this study reinforces the potential role of ophthalmic artery Doppler as a non-invasive tool for assessing cerebrovascular changes in pre-eclampsia and aiding in early identification of high-risk patients. Further large-scale, multicenter studies are needed to validate these findings and explore their clinical applicability in routine obstetric care.

LIMITATIONS

- Being a single-centre study, its findings may not be generalizable to a broader population.
- The small sample size reduces statistical power and may limit the robustness of the conclusions.
- The absence of longitudinal follow-up prevents the assessment of postpartum changes in Doppler parameters.
- Potential confounding factors, such as comorbidities and medication use, were not accounted for, which may have influenced the results.
- Neonatal outcomes were not extensively evaluated due to a lack of detailed follow-up.

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ANNEXURES

CONSENT FORM

BLDE. (DEEMED TO BE UNIVERSITY)

S.H.R.I. B.M.PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTER, BIJAPUR-586103

INFORMED CONSENT FOR PARTICIPATION IN DISSERTATION/RESEARCH

I, the undersigned, _____, D/O W/O _____, aged ____ years, ordinarily resident of _____ do hereby state/declare that Dr SHARANABASAVA S KULKARNI of Shri. B. M. Patil Medical College Hospital and Research Centre have examined me thoroughly on _____ at _____ (place), and it has been explained to me in my own language about the intervention being performed on me, its progression and possible complications. Further, Dr Sharanabasava S Kulkarni informed me that he/she is conducting a dissertation/research titled " PROSPECTIVE CASE CONTROL STUDY ON MATERNAL OPHTHALMIC ARTERY DOPPLER VELOCIMETRY IN EVALUATION OF HEMODYNAMICS IN PRE ECLAMPSIA.*"* under the guidance of Dr.Shreedevi Kori, requesting my participation in the study. The doctor has also informed me that adverse results may be encountered during this procedure. Among the above complications, most of them are treatable but are not anticipated; hence there is a chance of aggravation of my condition, and in rare circumstances, it may prove fatal despite the anticipated diagnosis and best treatment made available. Further, the Doctor has informed me that my participation in this study would help in the evaluation of the results of the study, which is a useful reference to the treatment of other similar cases shortly, and also, I may benefit from getting relieved of suffering or cure of the disease I am suffering.

The doctor has also informed me that information given by me, observations made, photographs, and video graphs taken upon me by the investigator will be kept secret and will not be assessed by a person other than my legal hirer or me except for academic purposes. The Doctor did inform me that though my participation is purely voluntary, based on the information I gave, I can ask for any clarification during the course of treatment/study related to diagnosis, the procedure of treatment, the result of treatment, or prognosis. At the same time, I have been informed that I can withdraw from my participation in this study at any time

if I want, or the investigator can terminate me from the study at any time study but not the procedure of treatment and follow-up unless I request to be discharged. After understanding the nature of the dissertation or research, the diagnosis made, mode of treatment, I, the undersigned Smt _____, under my fully conscious state of mind, agree to participate in the said research/dissertation.

Date: Signature of the patient:

Place: Signature of Doctor:

B.L.D.E (DEEMED TO BE UNIVERSITY)

ಶ್ರೀ ಬಿ.ಎಂ.ಪಟ್ಟೀಲ್ ಮೆಡಿಕಲ್ ಕಾಲೇಜು, ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರ, ವಿಜಯಪುರ-586103

ಪ್ರಬಂಧ/ಸಂಶೋಧನೆಯಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ಮಾಹಿತಿ ಪಡೆದ ಸಮ್ಮತಿ

ನಾನು, ಕೆಳಗಿನವರು_____ ಸಹಿಯಿಟ್ಟವರು, ಮಗ/ಮಗಳು/ಪತ್ನಿಯ _____ ವಯಸ್ಸು _____ವರ್ಷಗಳು, ಸಾಮಾನ್ಯವಾಗಿ ನಿವಾಸಿಸುವ ಸ್ಥಳದ ಹೆಸರು_____, ಇಲ್ಲಿ ಹೇಳಿದ್ದೇನೆ/ಘೋಷಿಸುತ್ತೇನೆ ಡಾಕ್ಟರ್ ಹೆಸರು_____ ಅವರು ಆಸ್ಪತ್ರೆ ಹೆಸರು_____ ಅವರು ನನ್ನನ್ನು ಪೂರ್ಣವಾಗಿ ಪರೀಕ್ಷಿಸಿದರು ದಿನಾಂಕದಲ್ಲಿ_____ ಸ್ಥಳ ಹೆಸರು_____ ಮತ್ತು ನನಗೆ ನನ್ನ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಲಾಗಿದೆ ನಾನು ಒಂದು ರೋಗ (ಸ್ಥಿತಿ) ಅನುಭವಿಸುತ್ತಿದ್ದೇನೆ. ಮುಂದುವರಿದು ಡಾಕ್ಟರ್ ನನಗೆ ತಿಳಿಸಿದ್ದಾರೆ ಅವರು ಒಂದು ಪದ್ಧತಿ/ಸಂಶೋಧನೆ ನಡೆಸುತ್ತಿದ್ದಾರೆ ಶೀರ್ಷಿಕೆಯುಳ್ಳ_____ ಡಾಕ್ಟರ್_____ ಮಾರ್ಗದರ್ಶನದಲ್ಲಿ ನನ್ನ ಪಾಲ್ಗೊಳ್ಳುವಿಕೆಯನ್ನು ಕೇಳಿದ್ದಾರೆ ಅಧ್ಯಯನದಲ್ಲಿ.

ಡಾಕ್ಟರ್ ನನಗೆ ಇದನ್ನು ಕೂಡಾ ತಿಳಿಸಿದ್ದಾರೆ ಈ ಕ್ರಮದ ನಡೆವಲ್ಲಿ ಪ್ರತಿಕೂಲ ಫಲಿತಾಂಶಗಳನ್ನು ಎದುರಿಸಬಹುದು. ಮೇಲೆ ಹೇಳಿದ ಪ್ರಕಟಣೆಗಳಲ್ಲಿ, ಅಧಿಕಾಂಶವು ಚಿಕಿತ್ಸಿಸಬಹುದಾದರೂ ಅದನ್ನು ನಿರೀಕ್ಷಿಸಲಾಗುತ್ತಿಲ್ಲ ಆದ್ದರಿಂದ ನನ್ನ ಸ್ಥಿತಿಯ ಹಿರಿದಾಗುವ ಅವಕಾಶವಿದೆ ಮತ್ತು ಅಪರೂಪದ ಸಂದರ್ಭಗಳಲ್ಲಿ ಅದು ಮರಣಕಾರಕವಾಗಿ ಪರಿಣಮಿಸಬಹುದು ಹೊಂದಿದ ರೋಗನಿರ್ಧಾರ ಮತ್ತು ಯಥಾಶಕ್ತಿ ಚಿಕಿತ್ಸೆ ಮಾಡಲು ಹೊಂದಿದರೂ. ಮುಂದುವರಿದು ಡಾಕ್ಟರ್ ನನಗೆ ತಿಳಿಸಿದ್ದಾರೆ ನನ್ನ ಪಾಲ್ಗೊಳ್ಳುವಿಕೆ ಈ ಅಧ್ಯಯನದ ಫಲಿತಾಂಶಗಳ ಮೌಲ್ಯಮಾಪನದಲ್ಲಿ ಸಹಾಯಕವಾಗುತತ್ತದೆ ಇತರ ಸಮಾನ ಪ್ರಕರಣಗಳ ಚಿಕಿತ್ಸೆಗೆ ಉಪಯುಕ್ತ ಉಲ್ಲೇಖವಾಗಿದೆ, ಮತ್ತು ನಾನು ಅನುಭವಿಸುವ ರೋಗದಿಂದ ವಿಮುಕ್ತಿ ಅಥವಾ ಗುಣಮುಖಗೊಳ್ಳುವಲ್ಲಿ ನನಗೆ ಪ್ರಯೋಜನವಾಗಬಹುದು.

ಡಾಕ್ಟರ್ ನನಗೆ ಇದನ್ನು ಕೂಡಾ ತಿಳಿಸಿದ್ದಾರೆ ನನ್ನಿಂದ ನೀಡಿದ ಮಾಹಿತಿ, ಮಾಡಿದ ಪರಿಶೀಲನೆಗಳು / ಫೋಟೋಗ್ರಾಫ್‌ಗಳು / ವೀಡಿಯೋ ಗ್ರಾಫ್‌ಗಳು ನನ್ನ ಮೇಲೆ ತೆಗೆದುಕೊಳ್ಳಲಾಗುವ ಅನ್ವೇಷಕರು ರಹಸ್ಯವಾಗಿ ಇಡುವರು ಮತ್ತು ನಾನು ಅಥವಾ ನನಗೆ ಕಾನೂನು ದೃಷ್ಟಿಯಲ್ಲಿ ಸಂಬಂಧಿತrannu ಹೊರತುಪಡಿಸಿ ಇತರ ವ್ಯಕ್ತಿಯಿಂದ ಮೌಲ್ಯಮಾಪನ ಮಾಡಲಾಗುವುದಿಲ್ಲ. ಡಾಕ್ಟರ್ ನನಗೆ ತಿಳಿಸಿದ್ದಾರೆ ನನ್ನ ಪಾಲ್ಗೊಳ್ಳುವಿಕೆ ಶುದ್ಧವಾಗಿ ಸ್ವೇಚ್ಛಾಯಿತ, ನನ್ನಿಂದ ನೀಡಿದ ಮಾಹಿತಿಯ ಆಧಾರದ ಮೇಲೆ, ಚಿಕಿತ್ಸೆ / ಅಧ್ಯಯನದ ಸಂಬಂಧದಲ್ಲಿ ರೋಗನಿರ್ಧಾರ, ಚಿಕಿತ್ಸೆಯ ವಿಧಾನ, ಚಿಕಿತ್ಸೆಯ ಫಲಿತಾಂಶ ಅಥವ ಆ ಭವಿಷ್ಯದ ಪ್ರವೃತ್ತಿಗಳು ಬಗ್ಗೆ ಯಾವುದೇ ಸ್ಪಷ್ಟತೆ ಕೇಳಬಹುದು. ಅದೇ ಸಮಯದಲ್ಲಿ ನನಗೆ ತಿಳಿಸಲಾಗಿದೆ ನಾನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನನ್ನ ಪಾಲ್ಗೊಳ್ಳುವಿಕೆಯನ್ನು ನಿಲ್ಲಿಸಬಹುದು ನಾನು ಬಯಸಿದರೆ ಅಥವಾ ಅನ್ವೇಷಕರು ಅಧ್ಯಯನದಿಂದ ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ನನ್ನನ್ನು ನಿಲ್ಲಿಸಬಹುದು.

ಪ್ರಬಂಧ ಅಥವಾ ಸಂಶೋಧನೆಯ ಸ್ವಭಾವ, ಮಾಡಿದ ರೋಗನಿರ್ಧಾರ ಮತ್ತು ಚಿಕಿತ್ಸೆಯ ವಿಧಾನವನ್ನು ಅರ್ಥಮಾಡಿಕೊಂಡು, ನಾನು ಕೆಳಗಿನ ಶ್ರೀ / ಶ್ರೀಮತಿ_____ ನನ್ನ ಪೂರ್ಣವಾದ ಪ್ರಜ್ಞೆಯ ಸ್ಥಿತಿಯಲ್ಲಿ ಹೇಳಿದ ಸಂಶೋಧನೆ / ಪ್ರಬಂಧದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ಒಪ್ಪುತ್ತೇನೆ.

ರೋಗಿಯ ಸಹಿ ಡಾಕ್ಟರನ ಸಹಿ

ಸಾಕ್ಷಿಗಳು

1)

2)

PERFORMA

"PROSPECTIVE CASE-CONTROL STUDY ON MATERNAL OPTHALMIC ARTERY DOPPLER VELOCIMETRY IN EVALUATION OF PRE-ECLAMPSIA"

NAME:		DATE OF ADMISSION:	
AGE/SEX:		PHONE NUMBER:	
IP NUMBER		ADDRESS:	

CHIEF COMPLAINTS:

ANY IMMINENT SIGNS :

OBSTETRIC HISTORY: MARITAL HISTORY:

LAST MENSTRUAL PERIOD: EXPECTED DATE OF DELIVERY:

PERIOD OF GESTATION:

A.N.C.: 1ST TRIMESTER:

2ND TRIMESTER:

3RD TRIMESTER:

PAST HISTORY:

GENERAL PHYSICAL EXAMINATION:

PULSE: BLOOD PRESSURE:

PALLOR: BREAST:

ICTERUS: SPINE:

CYANOSIS: THYROID:

CLUBBING:

LYMPHADENOPATHY:

EDEMA:

CARDIOVASCULAR SYSTEM:

RESPIRATORY SYSTEM:

PER ABDOMEN:

INVESTIGATIONS:

CBC		U/R		LFT		RFT		OTHERS
HB		ALB		TB		UREA		UA
TC		SUGAR		DB		CREAT		
PLT		RBC		SGOT		NA		
				SGPT		K		
				ALP		CL		

- MATERNAL OPHTHALMIC ARTERY DOPPLER REPORT:

	RIGHT	LEFT
RESISTIVITY INDEX		
PULSATILITY INDEX		
PEAK SYSTOLIC VELOCITY		
PEAK DIASTOLIC VELOCITY		
END-DIASTOLIC VELOCITY		

OBSTETRIC OUTCOME:

PRE-TERM /TERM

VAGINAL DELIVERY/ LSCS

NEONATALOUTCOME
Baby details

- Gender:
- Date of delivery:
- Time of delivery:
- Birth weight:
- APGAR Score:
- Fetal complication
- NICU ADMISSION = YES /NO

MASTERCHART

NAME	AGE	IP NUMBER	ANY IMMINENT SIGNS	OBS HISTORY	POG	PAST HISTORY	PR(BPM)	BP(MM HG)		EDEMA	HB	TC	PLT	U.AL	TB	DB	SGOT	SGPT	ALP	UREA	CREAT	RI	PI	PSV	PDV	EDV	RI	PI	PSV	PDV	EDV	TERM	LSCS	NEONATAL COMPLICATION	NICU ADMISSSION
POOJA	22	123015	0	G2P1L1	38	0	88	110	70	0	11.5	12.09	196	0								0.84	2.41	44.2	7	5.43						1	0	0	0
SWATI	28	990692	0	PRIMIGRAVIDA	38	0	78	110	70	0	10.4	8.51	172	0								0.73	1.39	19.8	5.2	5.29	0.72	1.4	20.01	5.3	5.32	1		0	0
AKSHTHA	23	155606	1	PRIMIGRAVIDA	37	0	84	140	90	1	11.8	13.76	159	1	0.3	0.1	25	15	228	16	0.5	0.61	1.14	39.29	15.4	15.43	0.63	1.08	38.6	15.2	15.6	1	1	0	0
SHRUTI	22	194202	0	PRIMIGRAVIDA	37	0	86	130	90	0	12.1	8.9	162	0								0.63	1.18	18.45	6.8	6.84	0.64	1.2	18.01	6.6	6.8	0	1	1	1
DEEPA	22	1022	0	G3P1L1A1	38	0	72	100	60	0	8.6	11.6	269	0								0.66	1.32	22.9	7.8	7.84	0.7	1.38	22.6	7.9	7.8	1	1	0	0
BHAGYASHREE	20	229437	1	PRIMIGRAVIDA	31	0	90	150	100	0	11.2	17.8	197	1	0.3	0.1	21	14	169	12	0.5	0.57	0.96	21.32	9.07	9.08	0.6	0.95	22.4	9.05	9.85	0	1	1	1
JYOTHI	22	232326	1	PRIMIGRAVIDA	41	0	120	150	100	1	11.3	21	180	1	0.4	0.2	17	17	55	13	0.8	0.6	1.13	27	10.9	11	0.62	1.12	29	11	11.3	1	1	1	1
MAHANANDA	22	226999	0	PRIMIGRAVIDA	41		82	160	90	1	10.4	12.1	251	1	0.7	0.3	44	25	216	14	0.4	0.62	1.33	22.4	8.6	8.51	0.64	1.36	21.1	8.7	8.4	1	1	0	0
SAVITRI	21	237758	0	PRIMIGRAVIDA	41	0	86	130	80	0	9.8	17.1	277	0								0.67	1.01	43	14.8	14.5	0.66	1.12	41.7	14.3	14.2	1	0	0	0
BASAMMA	29	237663	0	G3P2L2	29	0	80	120	70	0	9.8	8.8	223	1								0.67	1.11	42	13.86	13.9	0.66	1.2	41	13.46	13.01	0	1	1	1
PREETI	19	237672	0	G2P1L1	38	0	86	130	70	0	12.1	10.24	211	0								0.84	2.59	37.1	5.84	5.85	0.82	2.6	36.5	5.68	5.8	1	0	0	0
KAVYA	24		0	PRIMIGRAVIDA	41	0	86	120	80	0	13	9	230	0								0.7	1.51	43.2	13.1	13.15	0.71	1.6	40.2	13.3	13.2	1	0	0	0
ASHWINI	24		1	PRIMIGRAVIDA	37	0	88	130	90	0	11.2	10.2	160	1	0.4	0.2	0.2	30	42	12	0.6	0.64	1.28	23.7	8.7	8.57	0.66	1.21	22.6	8.4	8.5	1	1	0	0
MAYAKKA	23		0	G3P2L2	36	0	62	110	70	0	8.9	10.1	201	0								0.8	2.06	31.7	6.4	6.5	0.81	2.13	30.8	6.5	6.4	0	0	1	1
BHAGYA			0	G2P1L1	35	0	86	110	70	0	11.2	8.8	164	1								0.76	2.12	30.1	6.4	6.6	0.78	2.02	29	6.9	7.2	0	0	1	1
SONALI	26	1082	0	G3P2L2	39	0	78	120	80	0	11.1	11.4	186	0								0.9	2.86	35.4	5.86	8.2	0.86	2.6	33.2	5.4	79	1	1	0	0
DANAMMA	25		0	G2P1L1A1	38	0	86	120	80	0	11.2	9.4	14									0.86	2.4	33.8	6.8	7.4	0.84	2.9	38.6	7.1	8.1	1	0	1	1
AARTI	24	466	0	G2P1L1	42	0	86	110	70	0	12.1	6.8	168									0.84	3.1	35.1	3.6	7.8	0.86	3.4	33.2	3.4	7.4	1	1	0	0
SWATI	24	1068	0	PRIMIGRAVIDA	40	0	84	130	70	0	9.8	11.1	186	0								0.79	3.4	38.1	3.8	8.2	0.8	3.6	36.9	3.6	8.1	1	1	0	0
KALAMMA	24	271782	0	G2P1L2	37	0	86	120	80	0	10.5	9.25	222	0								0.84	2.3	44	7.1	7.2	0.86	2.2	42	7.8	8.1	1	1	0	0
LAXMI	25	263057	0	G4P3L3	41	0	80	110	80	0	10.8	11.2	146									0.68	1.34	23	7.9	7.9	0.7	1.48	24	7.8	8.2	1	0	0	0
POOJA	24	268455	0	PRIMIGRAVIDA	38	0	80	120	80	0	8.8	15.43	180									0.73	1.4	20.1	5.4	5.6	0.76	1.5	21	5.6	5.8	1	1	0	0
BHISMILLA	18	259372	0	PRIMIGRAVIDA	40	0	94	110	80	0	10.6	14.51	236	0								0.84	2.6	37	6.1	6.2	0.82	2.4	35	6.2	6.2	1	1	1	1
POOJA	23	1115460	0	G3P1L1A1	39	0	86	130	80	0	9.6	14.56	152	0								0.7	1.52	43.1	13.2	13.4	0.68	1.6	42.6	14.2	14.6	1	0	0	0
RAJESHWARI	26	276133	0	G4P2L2A1	37	0	88	180	100	1	9.8	12.61	182	1	0.6	0.2	3.4	16	271	22	0.8	0.57	0.96	22.4	9.8	9.6	0.54	0.8	24.3	9.9	8.6	0	1	0	0
KALAVATI	24	266025	0	G3P2L2	37	0	84	120	80	0	10.8	11.31	183	1								0.72	1.4	19.86	5.2	5.4	0.77	1.8	22.3	5.6	5.5	1	1	1	
SHILPA	23	274806	0	PRIMIGRAVIDA	39	0	86	110	70	0	11.4	14.58	231	0								0.84	2.41	44.2	7	7.02	0.79	2.5	42.2	6.9	7.01	1	0	0	0
FARANA	27	183474	0	G3P2L2	40	0	88	120	80	0	14.2	8700	259	1								0.66	1.34	23.1	7.9	7.92						1	0	1	0
JYOTHI	24	274876	0	G2P1L1	38	0	88	110	70	0	11.2	5.4	264	1								0.72	1.4	34	8.7	8.76	0.72	1.6	32.1	19.8	8.7	0	0	0	0
SANGEETA	22	257690	0	PRIMIGRAVIDA	41	0	98	110	80	0	11.3	18.5	307	0								0.74	1.7	34.1	20.1	20.2	0.72	1.68	34.5	27.2	21.4	1	0	0	0
MAYAKKA	38	259438	0	PRIMIGRAVIDA	33	0	86	110	60	0	9	289	246	1								0.73	1.39	19.86	5.2	5.3	0.74	1.5	20.5	5.4	5.45	0	1	1	1
LAXMI	25		0	G4P3L3	41	0	86	120	70	0	10.2	12.1	160	0								0.81	2.5	44.2	7.1	7.2	0.84	2.4	40.1	7.06	7.05	1	0	0	0
AARTI	27	199439	0	G2P1L1	39	0	86	110	70	0	10.6	8.9	140	0								0.68	1.6	43	15.1	14.5	0.7	1.67	40	15.2	15.3	1	0	0	0
SARASWATI	24	252785	1	G2P1L1	35	0	92	160	100	0	11	11.1	3.5	0	1.1	0.1	19	14	201	13	0.4	0.6	1.13	27	10.9	11	0.64	1.4	28	10.6	10.7	1	1	1	1
LAVANYA	24	271923	1	PRIMIGRAVIDA	37	0	92	150	100	1	13.2	11.91	148	1	0.2	0.1	28	18	365	20	0.8	0.64	1.3	24.6	18.8	18.9	0.65	1.2	25.1	18.6	18.8	1	1	1	1
SHAHIN	22	274424	0	PRIMIGRAVIDA	38	0	86	120	70	0	7.1	7.65	369	0								0.76	1.52	43	13.1	13.2	0.98	1.6	40	12.6	127	0	0	1	1
NAZRIN	27	266089	0	G3P2L2	37	0	88	120	80	0	13.2	12.5	240	1	0.5	0.1	30	27	275	15	0.5	0.64	1.3	24.6	18.6	18.5	20.7	1.4	25.4	8.5	8.6		1	0	0
ASUTA	21	20139	0	G2A1	40	0	112	130	70	0	8.1	9.69	247	0								0.71	1.4	19.8	5.6	5.7	0.74	1.6	20.1	5.5	5.6	1	1		
AMUNA	29	281789	0	G2P1L1	39	0	78	110	70	0	120	10.96	292	0								0.79	2.05	30.6	6.6	6.56	0.78	2.1	29.1	6.5	6.4	1	1	0	0
SUPRIYA	26	287795	0	G2A1	39	0	110	130	80	0	10.7	16.13	227	0								0.69	2.6	40	13.1	13.2	6.72	2.8	40.1	12.6	12.65	1	1	0	0
LAXMI	23	308846	0	PRIMIGRAVIDA	39	0	90	130	80	0	9.8	12.4	160	0								0.82	1.82	18.2	10.2	5.5	0.74	1.45	18.4	8.4	5.56	1	0	0	0
ROOPA	21	34988	0	G2P1L1	35	0	90	130	80	1	10.6	16.4	198	0								0.58	1.78	17.9	7.4	6.9	0.61	1.86	17.8	7.4	6.92	0	1	0	0
TANJUM	20	38848	0	PRIMIGRAVIDA		0	88	120	86	0	10.6	14.8	154	0								0.76	1.54	18.9	8.2	5.9	0.77	2.03	18.4	8.3	6.9	1	1	0	0
LALITHA	25	40784	0	G3P1L1A1	38	0	86	130	80	0	10.8	17.4	163	0								0.82	1.45	17.2	3.8	4.2	0.83	1.45	17.4	3.9	4.5	1	0	0	0
AKSHATHA	26	40789	0	G2P1L1	40	0	90	120	80	0	9.8	16	163	0								0.71	1.42	19.2	8.2	6.2	0.74	1.42	19.8	8.3	6.2	1	1	0	0
SHRUTHI	25	41686	0	G2P1L1	39	0	96	130	80	0	11.2	14	210	0								0.78	1.34	1.9	6.2	5.9	0.78	1.35	1.9	6.2	5.9	1	0	0	0
VASANTA	26	42082	0	G3P1L1A1	38	0	90	120	70	0	11.2	15,4	196	0								0.32	0.72	19.1	7.2	7.9	0.33	0.79	19.2	7.2	7.4	1	1	0	0
VAISHALI	33	376517	0	G5P2L2A2	39	0	92	120	80	0	11.2	12.9	460	0								0.74	1.34	1.72	5.4	5.5	0.75	1.34	1.74	5.6	5.5	1	1	0	0
MAHANANDA	24	43427	0	G2L1P1	39	0	98	150	90	1	9.2	14.2	169	1	0.5	0.2	40	36	98	26	1.2	0.58	0.92	15.9	6.9	7.45	0.59	0.93	15.9	6.92	7.42	1	1	0	0
RESHMA	26	43431	0	G3P2L2	39	0	88	120	70	0	11.2	10.2	289	0								0.82	1.52	19.4	3.2	3.4	0.82	1.52	19.5	3.4	3.5	1	0	1	1
MALLAMMA	27	45843	0	G3P2L2	39	0	102	160	100	1	9.8	11.2	140	1	0.8	0.3	31	40	130	39	0.8	0.68	0.94	16.2	8.1	8.2	0.69	0.95	16.3	8.1	8.2	1	1	0	1
PRIYANKA	24	45822	0	PRIMIGRAVIDA	40	0	82	120	80	0	10.2	9.8	210	0								0.74	1.82	21.1	6.89	5.4	0.78	1.84	22	6.88	5.5	1	1	0	1
JYOTHI	21	45952	1	PRIMIGRAVIDA	39	0	92	140	90	1	10.2	11.2	146	1	0.8	0.2	30	40	118	30	1	0.82	0.92	18.4	9.2	8.4	0.84	0.93	18.5	9.2	8.5	1	0	0	1
RUFIYA	20	46403	1	PRIMIGRAVIDA	38	0	82	150	100	1	8.9	12.46	96	1	0.9	0.3	52	48	120	26	1.2	0.64	0.72	1.78	1.148	4.82	0.65	0.74	1.79	1.2	4.82	1	1	0	1
RANI	21	47148	1	PRIMIGRAVIDA	38	0	86	150	90	1	11.2	17.2	99	1	1.2	0.4	38	42	112	22	1.2	0.78	0.84	18.4	10.2	8.4	0.79	0.88	19.2	10.4	8.8	1	1	1	0
SUREKHA	33	47738	0	G3P2L2	39	0	86	120	70	0	11.2	9.2	224	0								0.74	0.92	22.4	14.2	8.6	0.74	0.95	22.5	14.2	8.8	1	1	0	0
GEETA	30	49013	0	G4P3L2A1	40	0	92	140	100	1	13.8	12.9	208	1	0.7	0.2	34	18	140	34	0.8	0.69	0.92	21.4	14.2	8.9	0.69	0.94	22	14.3	8.8	1	0	0	0
SHOBHA	26	49038	0	G4P2L2A1	38	0	102	130	70	0	13	11.2	340	0								0.84	1.34	19.2	9.8	6.2	0.85	1.35	19.4	9.9	6.3	1	0	0	0
KAVERI	19	50328	0	PRIMIGRAVIDA	38	0	90	140	100	1	10.8	21.8	192	1	0.8	0.4	38	48	130	50	1	0.79	0.84	16.2	11.2	8.2	0.79	0.86	16.3	11.3	8.3	1	1	0	0
AMREEN	36	57553	0	G3P1L1A1	38	0	86	150	100	1	10.8	16.8	128	1	1.1	0.2	31	38	120	34	0.9	0.71	0.92	17.9	9.2	8.2	0.72	0.92	18.2	9.4	8.4	1	1	0	0
SANGEETHA	19	51604	0	G2A1	39	0	100	150	100	1	9.2	21.2	162	1	0.7	0.3	42	48	98	45	1.2	0.72	0.84	15.4	12.4	8.1	0.73	0.85	15.5	12.4	8.4	1	0	0	0
SANGEETHA	19	51604	0	G2A1	39	0	100	150	100	1	9.2	21.2	162	1	0.7	0.3	42	48	98	45	1.2	0.72	0.84	15.4	12.4	8.1	0.73	0.85	15.5	12.4	8.4	1	0	0	0
PANDUNYA	25	516041	0	G2P1L1	39	0	86	110	80	0	10.9	16.2	192	0								0.82	1.52	20.2	11.2	6.4	0.82	1.56	20.2	11.5	6.6	1	0	0	0
MAHANANDA	22	52741	0	G2A1	40	0	90	160	90	1	11.2	17.2	108	1	0.8	0.2	34	28	100	30	0.9	0.52	0.84	24.2	11.4	9.2	0.52	0.85	25.2	11.5	9.2	1	0	0	0
MAHANANDA	22	52688	0	G2P1L1	40	0	89	140	90	0	10.9	16.2	206	0	0.5	0.1	53	40	130	26	1.2	0.72	0.82	16.2	9.8	10.2	0.73	0.84	16.2	9.9	10.3	1	0	0	0
SAVITRI	23	52622	0	PRIMIGRAVIDA	39	0	82	110	80	0	11.2	15.6	216	0								0.83	1.38	18.2	9.2	6.3	0.84	1.39	18.2	9.3	6.34	1	1	0	0
SAVITA	24	51997	1	PRIMIGRAVIDA	39	0	95	180	100	1	10.1	13.1	130	1	1	0.2	40	38	138	21	0.9	0.64	0.94	19.2	12.2	8.2	0.65	0.95	19.3	12.2	8.2	1	1	0	1
SAVITRI	21	55293	0	PRIMIGRAVIDA	40	0	82	110	70	0	9.9	13.2	210	0								0.74	1.32	17.9	11.2	5.2	0.75	1.32	17.39	10.9	5.9	1	0	0	0
POOJA	22	55448	0	PRIMIGRAVIDA	39	0	100	150	100	1	9.8	11.2	112	1	0.8	0.2	35	40	120	40	1.1	0.62	0.92	21.2	11.2	8.9	0.65	0.98	21.4	11.3	8.9	1	0	0	1
ANITA	24	59408	0	PRIMIGRAVIDA	39	0	90	120	70	0	10.1	12.1	262	0	0.3	0.1	12	15	100	10	0.2	0.84	1.34	18.2	12.1	5.92	0.86	1.33	18.9	12.3	6.01	1	0	0	0
SHILPA	26	59606	0	G2P1L1	39	0	86	110	80	0	11.2	16.2	172	0	0.4	0.1	15	28	130	12	0.3	0.72	1.23	20.2	8.9	5.92	0.73	1.21	20.3	8.98	5.92	1	1	0	0
AKSHATA	25	49052	0	PRIMIGRAVIDA	31	0	112	120	80	0	10.2	17.5	178	0								0.81	1.4	34.1	19.06	9.76	0.82	1.45	34.9	20.01	9.01	0	0	0	1
PUSHPAVATI	33	69288	0	PRIMIGRAVIDA	37	1	90	150	90	0	11.6	12.6	288	1								0.75	1.21	27.4	21.9	9.87	0.78	1.2	27.92	22.01	9	1	1	0	0
DANAMMA	21	70641	0	G2P1L1	42	0	98	120	80	0	12.9	17.3	109	1								0.95	2.13	30.1	17.06	7.61	0.96	2.24	30.6	17.2	7.24	1	1	1	1
DEEPALI	24	72535	0	G4P2L2A1	38	0	100	120	80	0	11.8	11.53	127	0								0.95	1.8	29.4	18.06	9.79	0.96	1.8	29.86	18.27	9.86	1	1	0	0
SANGEETA	25	118426	0	PRIMIGRAVIDA	36	0	94	120	70	0	10.3	11.07	276	0								0.68	1.7	31.9	18..72	7.46	0.67	1.72	31.7	18.62	7.01	1	0	0	0
VIDYA	28	114352	0	G4P2L2A1	36	0	90	110	70	0	9.8	7.26	196	0								0.71	1.25	36.2	22.6	10.26	0.7	1.24	36.26	22.79	10.27	1	0	0	0
PALLAVI	20	111727	1	PRIMIGRAVIDA	39	0	90	150	100	1	9.76	10.6	156	1	1.06	0.46	4.2	35	126	20	1.01	0.71	1.25	26.17	20.56	9.1	0.72	1.25	26.26	20.76	9.56	1	1	0	0
ROOPA	28	12653	0	G2P1L1	36	0	88	110	70	0	10.2	11.26	226	0								0.65	1.64	28.26	20.26	7.72	0.69	1.69	28.96	20.96	7.01	1	0	0	0
RUKSANA	22	65025	0	PRIMIGRABIDA	41	0	80	116	80	0	11.6	9.86	126	0								0.69	1.56	27.1	20.26	7.26	0.66	1.28	27.26	20.76	7.66	1	0	0	0
LAXMI	19	12311	0	PRIMIGRAVIDA	37	0	90	150	100	1	9.6	12.26	100	1	1	0.2	31	36	130	19	1	0.69	1.46	27.6	23.6	10.21	0.52	1.45	27.36	23.7	10.36	1	1	0	1
CHANDRAKALA	22	12132	0	PRIMIGRAVIDA	38	0	84	106	90	0	10.2	11.2	146	0	0.9	0.4	36	32	130	22	0.9	0.78	1.56	34.2	23.2	9.96	0.74	1.53	34.6	23.6	10.01	1	0	0	0
BASAMMA	19	31313	0	PRIMIGRAVIDA	40	0	82	122	74	0	12.6	9.76	146	0								0.58	1.7	36.2	24.6	12.2	0.45	1.51	36.8	24.7	12.76	1	1	0	0
CHAITRA	27	13451	1	PRIMIGRAVIDA	37	0	90	180	100	1	11.8	12.53	224	1	0.8	0.5	21	23	169	20	0.5	0.74	1.3	22.6	20.56	10.26	0.69	1.29	22.7	20.22	10.71	1	1	1	1
LAXMI	19	121321	0	G2P1L1	37	0	90	110	70	0	9.6	12.6	216	0								0.79	1.49	33.26	21.86	7.86	0.81	1.69	33.86	22.2	7.2	1	1	0	0
LAKSHMI	25	148762	0	G2P1L1	42	0	88	120	70	0	11.3	11.29	262	0								0.84	1.56	31.6	20.08	10.86	0.8	1.6	31.76	20.76	10.76	1	1	0	0
SUJATA	26	12121	1	PRIMIGRAVIDA	37	0	86	140	100	1	9.86	12.6	196	1	1.1	0.6	46	48	132	26	0.9	0.58	1.6	27.26	21.5	9.21	0.46	1.62	27.86	21.01	9.86	1	1	1	1
SHEHNAZ	36	40374	0	G4P3L3	39	0	95	150	90	1	9.2	17.72	527	1	0.6	0.4	46	47	166	5	0.4	0.68	1.86	27.26	23.6	11.62	0.62	1.82	28.01	23.86	11.86	1	1	0	0
PREETI	24	74668	0	G2P2L2	38	0	89	130	70	0	11.2	14.2	172	0								0.52	1.62	30.26	21.6	7.26	0.59	1.62	31.02	21.8	7.46	1	0	0	0
LAXMI	28	9693	1	G4P3L3	37	0	100	160	100	1	13.6	19.2	150	1	0.9	0.3	40	42	132	30	1.02	1.14	0.7	27.2	20.06	9	1.2	0.69	26.9	21.2	8.86	1	1	1	1
BHUVANESHWARI	23	230193	0	G3P2L2	39	0	88	120	70	0	9.7	11.2	218	0								0.75	1.34	30.2	20.26	9.86	0.68	1.42	29.86	20.76	10.01	1	0	0	0
AKSHATA	21	38842	1	PRIMIGRAVIDA		0	96	160	100	1	12.1	11.1	234	1	0.4	0.2	55	32	111	23	0.6	0.55	1.2	31.9	21.3	11	0.58	1.1	28.1	22.9	11.1	1	1	0	0
BHAGYASHREE	21	39510	0	G2P1	40	0	90	140	90	1	10.8	18.6	201	1	0.6	0.2	52	33	111	28	0.5	0.62	1.5	28.1	22.9	10.7	0.63	1.17	30.9	24.4	9.5	1	1	0	0
LAXMI	21	38807	0	G3P2L2	36	0	90	180	90	0	11.6	10.7	148	0	0.9	0.2	71	56	184	32	0.7	0.63	1.17	30.9	21.4	9.5	0.6	1.28	29.8	21.1	10.3	1	0	0	0
POOJA	25	230289	0	G3P2L2	27	0	108	130	80	0	9.8	16.9	160	0								0.69	1.6	33.1	20.1	7.8	0.76	1.79	31.4	17.6	6.7	1	1	0	0
LAXMI	22	9693	1	PRIMIGRAVIDA	37	0	90	160	90	1	9.1	7.8	130	1	1.1	0.9	60	70		24	0.9	0.6	1.38	29.8	21.1	10.3	0.66	1.3	27.5	23.4	9.9	1	0	0	1
BHUVANESHWARI	23	18982	0	G3P2L2	32	0	80	180	120	1	10.1	12.4	170	1	1	0.9	88	106		24	1	0.59	1.26	28.3	18.9	10.5	0.7	1.42	32.7	22	10.8	0	1	1	1
JYOTHI	30	240203	0	G3P2L2	38	0	86	108	70	0	11.2	11.4	180	0								0.74	1.57	31	18	7.6	0.75	1.79	31.5	19.4	9.2	1	1	0	0
PREETI	33	251412	1	G2P1L1	36	1	88	140	90	1	10	9.3	273	1	0.9	0.6	34	42			1.1	0.62	1.44	31.7	23.3	9.4	0.64	1.2	30.2	22.1	10.1	0	0	1	1
GEETA	32	34414	0	G3P1L1A1	37	0	82	110	82	0	9.1	12.64	324	0								0.67	1.53	30.3	18.8	8.9	0.73	1.24	32.1	18.4	7.5	1	1	0	0
RESHMA	27	251341	0	G3P1L1A1	38	0	84	110	70	0	11.1	12	189	0								0.72	1.77	30.9	18.4	7.5	0.7	1.6	30.5	17	9.6	1	1	0	0
POOJA	21	34224	0	PRIMIGRAVIDA	38	0	80	150	100	1	9.8	10.1	311	1	0.7	0.5	23	40		28	0.8	0.61	1.39	29.2	19.9	10.2	0.62	1.12	26.9	21.6	10.6	1	1	0	0
PREMA	20		1	PRIMIGRAVIDA	38	0	90	180	90	1	7.9	4.02	199	1	2	0.3	63	29	158	38	1	0.68	1.35	28	20.9	9.7	0.61	1.46	27	23.5	11	1	1	1	1
YALLAMMA	19	12123	0	PRIMIGRAVIDA	38	0	90	130	70	0	11.1	12.1	214	0								0.75	1.95	31.7	15.8	7.2	0.71	1.64	32.7	17.9	8.6	1	0	0	0
RADHIKA	24	1212	1	G2P1L1	34	1	86	170	110	1	11	13.92	76	1	1.7	0.5	52	104	146	8	0.4	0.6	1.18	30.3	21.2	10	0.62	1.31	32.2	19.5	10.5	0	1	1	1
RUKMINI	22	400	1	PRIMIGRAVIDA	30	1	90	150	100	1	12.6	12.9	325	1	0.70.2	0.2	32	30	173	17	0.6	0.58	1.29	29.4	22.4	9.9	0.63	1.56	28.4	21.6	10.4	0	1	1	1
SUDHARANI	27	574	0	G2P1L1	39	0	84	110	70	0	11.1	7.8	211	0								0.72	1.62	31.6	16.8	6.9	0.75	1.56	30	17.2	7.8	1	0	0	0
NALAMBI	35	937	1	G4P3L3	36	1	90	170	100	1	9	11.4	143	1	1.1	0.3	90	38	112	12	0.9	0.66	1.24	26.5	20.7	11.3	0.59	1.15	27.9	24.1	10.5	0	1	1	1
SUPRITA	30	1069	0	G2P1L1	40	0	90	120	80	0	10.1	10.8	190	0								0.69	1.92	33.8	20.2	9.4	0.71	1.61	30.9	18.9	8.6	1	0	0	0
PADMAVATI	26	1051	0	PRIMIGRAVIDA	39	0	83	106	70	0	11.2	17.1	210	0								0.7	1.84	31.2	17.4	8	0.69	1.74	31.1	16	6.7	1	1	0	0
SANIYA	20	110	0	PRIMIGRAVIDA	40	1	78	140	90	1	8.9	11.4	196	1	0.4	0.1	40	28	110	16	0.7	0.72	1.47	30.8	23.7	10.7	0.65	1.22	33	20.2	9.6	1	1	0	0
RENUKA	23	1271	1	PRIMIGRAVIDA	36	1	94	160	100	1	10.4	18.1	200	1	1.1	0.4	72	39	116	30	1	0.63	1.18	25.3	18.8	10.2	0.6	1.33	30.7	21.4	10.9	0	1	1	1
POOJA	22	1591	0	PRIMIGRAVIDA	38	0	90	130	80	0	10.4	12.1	206	0								0.68	1.68	30.4	18.2	8.1	0.75	1.72	31	20.3	7.4	1	0	0	0
SHREEDEVI	26	1605	0	G3P1L1A1	41	0	80	110	70	0	11.4	14.7	216	0								0.73	1.79	32.3	17.9	7.5	0.74	1.53	33.4	19	9.1	1	0	0	0
NANDA	22	1416	1	PRIMIGRAVIDA	34	1	90	150	100	1	10.4	13.1	184	1	1.41	0.4	81	37	116	20	1.2	0.57	1.14	29.6	21.5	10.8	0.61	1.42	31.6	20.1	10.1	0	0	0	0
SHARDA	30	2855	0	G2P1L1	37	0	78	116	84	0	9.9	11.1	206	0								0.69	1.69	32.9	16.6	8.9	0.72	1.76	30.8	18	7.2	1	1	0	0
RENUKA	30	3223	0	PRIMIGRAVIDA	40	0	106	160	110	1	10	14.8	176	1	0.7	0.3	76	40	134	32	1.2	0.65	1.26	30.4	21.3	9.4	0.62	1.25	28.4	19.1	10.3	1	1	1	1
MOKSHITA	26	3891	0	PRIMIGRAVIDA	40	0	104	124	70	0	10.4	11.4	204	0								0.71	1.81	3.7	16.1	7.6	0.7	1.8	32	19.6	8.9	1	0	0	0
SUDHARANI	20	3336	1	PRIMIGRAVIDA	35	1	104	150	100	1	10.2	16.4	121	1	0.7	0.2	60	42	121	16	0.9	0.58	1.35		27.9	22.8	0.64	1.2	33.3	21.9	10.6	0	1	1	1
RENUKA	28	4223	0	G2P1L1	38	0	90	110	78	0	10.4	17.1	216	0								0.68	1.57	31.9	18.3	8.3	0.71	1.73	34.2	17.7	7.9	1	1	0	0
JYOTHI	20	4222	0	PRIMIGRAVIDA	39	0	104	126	88	0	11	10.1	280	0								0.74	1.88	32.4	17.8	9	0.73	1.54	30.6	19.1	6.8	1	0	0	0
SANIYA	22	4218	0	G3P2L2	37	0	102	114	80	0	10.8	9.8	198	0								0.71	1.81	30.9	16.1	7.6	0.7	1.8	32	19.6	8.7	1	0	0	0
ASHWINI	23	4493	0	G2P1L1	39	0	88	106	70	0	9.9	16.1	174	0								0.69	1.69	32.9	16.6	8.9	0.72	1.76	30.8	18	7.2	1	0	0	0

SANI	22	4218	0	G3P2L2	37	0	102	114	80	0	10.8	9.8	198	0								0.71	1.81	30.9	16.1	7.6	0.7	1.8	32	19.6	8.7	1	0	0	0
ASHW[illegible]	23	4493	0	G2P1L1	39	0	88	106	70	0	9.9	16.1	174	0								0.69	1.69	32.9	16.6	8.9	0.72	1.76	30.8	18	7.2	1	0	0	0
BHAVANA	25	4567	0	PRIMIGRAVIDA	37	0	98	140	90	1	9.8	11.4	146	1	0.6	0.2	56	48	114	32	0.6	0.62	1.55	31.1	19.6	10.6	0.69	1.29	29.9	24	10	1	0	1	1
PADMINI	22	4663	0	G2P1L1	37	0	86	118	74	0	12.1	13.6	206	0								0.68	1.57	31.9	18.3	8.3	0.71	1.73	32.2	17.7	7.9	1	0	0	0
RAJESHWARI	25	4884	0	G2A1	37	1	102	150	100	1	9	8.4	98	1	1.4	0.4	88	46	148	40	1.1	0.66	1.32	31.3	23.6	9.8	0.74	1.88	32.4	17.8	9	1	0	1	1
RENUKA	25	4905	0	G3P3L3	38	0	90	110	70	0	11.1	10.9	198	0								0.74	1.88	32.4	17.8	9	0.73	1.54	30.6	19.1	6.7	1	0	0	0
ROOPA	23	4981	0	G2P1L1	40	0	104	116	70	0	10.6	14.1	202	0								0.7	1.65	30.5	20	7.8	0.72	1.9	33.1	18.7	8.5	1	1	0	0
BASAMMA	24	5605	0	G2P1L1	37	0	86	110	70	0	10.1	11.1	219	0								0.75	1.58	32.2	17.4	9.2	0.69	1.83	21.7	19.3	6.9	1	0	0	0
SAVITRI	24	5346	1	PRIMIGRAVIDA	36	1	106	150	100	1	10.4	14.1	190	1	0.7	0.3	52	23	106	24	0.6	0.62	1.41	26.6	21.8	10.4	0.68	1.23	28.6	20	9.7	0	1	0	0
CHINAMMA	22	5331	0	PRIMIGRAVIDA	40	0	90	130	80	0	11	15.4	194	0								0.75	1.58	32.2	17.7	9.2	0.7	1.79	31.1	19.3	6.9	1	0	0	0
ASHWINI	24	5493	0	G2P1L1	37	0	90	110	70	0	11	11.4	160	0								0.73	1.6	33.5	19.4	9	0.72	1.82	30.2	18.8	8.5	1	1	0	0
GEETA	27	5529	0	G3P2L2	38	0	90	106	70	0	11	16.4	201	0								0.74	1.55	31.3	16.4	9.4	0.68	1.8	30.2	18.8	7.5	1	1	0	0
SANA	21	5987	0	PRIMIGRAVIDA	39	0	96	110	70	0	10.4	12.4	198	0								0.68	1.8	31.1	18.1	7.3	0.7	1.74	30.3	17.6	8.6	1	0	0	0
BHAGIRATHI	23	5814	0	G2P1L1	40	0	90	124	70	0	10.6	15.4	211	0								0.72	1.63	31.8	20.1	7	0.69	1.71	31	18.9	7.6	1	1	0	0
AISHA	25	5517	1	G2P1L1	36	1	96	170	100	1	9.6	7.1	123	1	1.1	0.3	40	32	109	24	0.8	0.61	1.33	29.1	19.2	10.4	0.62	1.41	31.8	21	10.5	0	1	1	1
SHABHANA	33	5362	1	PRIMIGRAVIDA	34	1	106	170	106	1	10.6	18.1	160	1	1	0.2				28	0.9	0.58	1.23	30.3	22.7	9.7	0.63	1.25	22.3	20.6	9.9	0	1	1	1
NANDINI	26	5755	1	G4P3L3	37	1	116	180	110	1	11.1	13.1	214	1	0.6	0.1	42	40	136	40	1.1	0.58	1.23	30.3	22.7	9.7	0.63	1.25	28.3	20.6	9.9	1	1	1	1
SHAMSHAD	24	6183	0	G2P1L1	38	0	96	160	90	1	11.6	17.4	246	1	0.8	0.1	46	34	148	30	0.7	0.6	1.54	28.8	21.8	9.6	0.62	1.54	29.8	23.8	10.6	1	0	0	0
INDIRA	34	5812	0	G3P1L1A1	35	0	90	140	90	1	12.6	21.1	214	1	0.6	0.1	41	28	131	28	0.6	0.69	1.21	30.1	20.5	10.2	0.63	1.32	30.5	19.5	10.8	0	1	0	0
INDRA	22	6894	1	PRIMIGRAVIDA	39	0	104	160	100	1	8.8	14.1	186	1	0.6	0.1	84	56	116	30	0.8	0.66	1.55	33.6	23.4	9.8	0.6	1.4	30.2	22	10.5	1	1	0	0
PREETI	34	7351	0	PRIMIGRAVIDA	38	0	94	140	90	1	8.1	16.4	210	1	1.2	0.2	46	24	116	34	0.6	0.61	1.12	27.1	23.1	11.3	0.75	1.5	27.4	22.4	11.4	1	1	0	0
NIDA	22	7304	0	PRIMIGRAVIDA	36	0	90	140	90	0	12.1	9.1	260	1	0.6	0.2	52	30	110	34	0.6	0.62	1.67	24.7	18.2	9.8	0.61	1.62	24.5	18.24	9.7	0	1	0	0
ROOPA	24	7320	1	G3P2L1A1	37	1	90	180	100	1	10.4	9.4	180	1	0.7	0.2	96	58	134	40	0.9	0.55	1.1	28.9	19.7	10.6	0.57	1.12	27.1	18.9	10.5	1	1	1	1
JYOTHI	24	7342	0	G2P1L1	39	1	92	150	100	1	0.9	16.1	190	1	1.4	0.4	91	52	161	39	0.7	0.7	1.35	34.1	20.3	10.1	0.69	1.43	33.5	20.4	10.5	1	1	1	1
ANJUME	20	7543	0	PRIMIGRAVIDA	39	0	90	150	90	1	11.3	11.2	206	1	0.7	0.2	66	41	106	26	0.6	0.58	1.4	26.3	22.5	11	0.6	1.3	25.3	21.8	11.2	1	1	0	0
POOJA	22	7710	0	PRIMIGRAVIDA	39	0	90	160	100	1	10.1	17.1	183	1	0.7	0.2	90	42	118	34	0.6	0.6	1.53	31.5	21.9	9.4	0.61	1.55	30.8	20.6	9.8	1	1	0	0
VIDYA	27	7740	0	PRIMIGRABIDA	39	0	98	164	98	1	9.9	10.53	168	1	5.1	1.1	58	110	134	25	0.8	0.68	1.01	29.7	24.3	10.3	0.66	1.09	28.7	24.1	10.1	0	1	1	1
NIDA	22	7929	0	PRIMIGRAVIDA	36	0	90	150	90	0	10.8	12.02	190		0.6	0.1	28	13	130	15	0.4	0.57	1.28	33.2	19.8	10.8	0.6	1.25	32.1	19.7	10.5	0	0	0	0
LAXMI	25	8404	1	PRIMIGRAVIDA	38	1	90	190	110	1	13.1	12.08	136	1	0.5	0.1	28	13	114	18	0.6	0.64	1.15	25.4	25.1	9.5	0.6	1.1	24.9	25.2	9.8	1	1	1	1
PARVATI	31	8005	0	PRIMIGRAVIDA	35	0	92	150	96	1	10.3	12.2	209	1	0.5	0.1	16	12	129	10	0.5	0.63	1.32	27.8	21.4	11.2	0.61	1.28	25.1	22.4	11	0	1	1	0
SABIYA	25	8621	1	PRIMIGRAVIDA	28	1	102	190	110	1	12	8.04	140	1	0.8	0.2	80	48	136	30	0.9	0.59	1.6	32.4	22.8	10.9	0.6	1.58	31.4	21.8	10.2	0	0	1	1
BHAGYASHREE	20	8606	1	PRIMIGRAVIDA	24	1	118	180	110	1	9.9	12.1	150	1	0.5	0.1	64	44	160	26	0.9	0.61	1.23	28.2	23.2	9.7	0.62	1.39	26.1	20.3	10.2	0	0	1	0
BISMILLA	29	8743	0	G3P1L1A1	36	0	88	150	90	0	12.5	10.25	162	1	0.7	0.2	36	24	110	26	0.4	0.66	1.39	26.1	20.6	10.2	0.72	1.46	30.6	19.3	10	0	1	0	0
NASIMA	27	8929	0	G3P2L2	37	0	88	150	90	1	9.1	13.2	180	1	0.6	0.1	34	26	108	26	0.6	0.72	1.46	30.6	19.3	10	0.69	1.29	28.5	22.1	10.4	1	1	0	0
ASMA	22	9696	0	PRIMIGRAVIDA	40	0	91	140	100	0	11.2	13.1	184	1	0.5	0.2	34	28	112	22	0.6	0.69	1.29	28.5	22.1	10.4	0.61	1.26	29.1	20.8	11.1	1	0	0	0
BHAGYASHREE	22	9703	0	G2P1	39	0	88	150	110	1	11.6	10.1	231	1	0.8	0.2	40	28	114	30	0.7	0.01	1.26	29.1	20.8	11.1	0.58	1.32	31	21.5	9.9	1	1	0	0
PREMA	20	9735	1	PRIMIGRAVIDA	35	0	98	160	110	1	9.9	11.44	361	1	0.6	0.2	39	26	110	28	0.6	0.58	1.32	31	29.5	9.9	0.62	1.1	27.3	20.4	10.4	0	1	1	1
SWATI	24	9925	0	G5P4L4	39	0	98	140	100	0	12.6	19.1	209	1	1	0.2	40	28	121	27	0.8	0.62	1.1	27.3	20.4	10.7	0.63	1.5	30.1	18.5	10.2	1	0	0	0
KAVYA	37	10158	0	G5P4L3A1	39	0	76	150	90	0	12.4	16.3	250	1	0.4	0.1	40	31	109	28	0.6	0.64	1.51	30	18.7	10.3	0.62	1.5	30.1	18.5	10.2	1	0	0	0
LAXMI	20	10019	1	PRIMIGRAVIDA	35	0	110	160	120	1	12.4	7.4	188	1	0.8	0.2	46	32	116	40	0.9	0.62	1.08	33.5	22.3	9.6	0.58	1.4	25.4	21.2	10.5	0	0	1	0
SIDDALINGAMMA	21	10124	1	PRIMIGRAVIDA	38	0	106	170	100	1	12.4	16.34	154	1	0.7	0.2	62	31	170	30	0.8	0.56	1.42	25.6	21	10.5	0.62	1.4	31.2	20.5	10.5	1	1	0	0
SHRIDEVI	22	10256	0	PRIMIGRAVIDA	39	0	106	150	110	1	10.1	20.1	230	1	0.8	0.2	77	51	109	32	0.7	0.63	1.38	31.2	20.5	11	0.61	1.27	32.1	23.8	9.4	1	1	1	0
SANGEETA	24		0	PRIMIGRAVIDA	36	0	106	160	100	0	9.9	12.4	186	1	0.5	0.1	42	30	110	30	0.6	0.6	1.28	32.3	23.9	9.3	0.64	1.4	26.4	21.7	10.4	0	0	0	0
VEENA	23	10288	0	G2P1L1	40	0	104	160	100	1	11	12.1	260	1	0.4	0.1	38	24	110	31	0.5	0.65	1.6	27.4	21.7	10.8	0.64	1.34	33.6	24.1	11.4	1	1	0	0
SUMA	22	10264	1	PRIMIGRAVIDA	34	0	90	160	110	1	12.1	8.1	208	1	0.9	0.2	52	31	108	30	0.8	0.67	1.34	33.7	24	11.5	0.58	1.26	28.7	19.1	9.7	1	1	1	1
SHANTAMMA	37	10504	1	G5P4L2A2	33	0	110	170	100	1	10.1	12.4	192	1	0.6	0.2	48	26	109	28	0.6	0.59	1.27	28.8	19.2	9.8	0.68	1.2	30.1	21.7	10	0	1	1	1
TANYA	20	10122	0	PRIMIGRAVIDA	35	0	106	170	106	1	8.8	13.1	196	1	0.4	0.1	42	28	123	28	0.6	0.08	1.21	30.1	21.8	10.4	0.61	1.44	34.6	22.7	10.3	0	1	1	1
NETRAVATI	19	10889	0	PRIMIGRAVIDA	29	1	88	150	110	1	7.4	15.34	300	1	0.9	0.2	42	31	129	33	0.8	0.67	1.1	26.7	19.4	10.9	0.57	1.32	32.9	23	10.4	0	0	1	1
AKSHATA	22	11192	1	G2P1L1A1	39	0	88	160	110	1	10.1	13.08	90	1	0.8	0.2	39	24	112	29	0.7	0.57	1.32	32.9	23	10.4	0.62	1.33	29.3	20	9.6	1	1	0	0
POOJA	20	11302	0	PRIMIGRAVIDA	31	0	104	150	100	1	11	20.9	337	1	0.5	0.2	49	32	116	32	0.6	0.62	1.33	29.3	20	9.6	0.56	1.2	31.9	21.3	10.7	0	0	1	1

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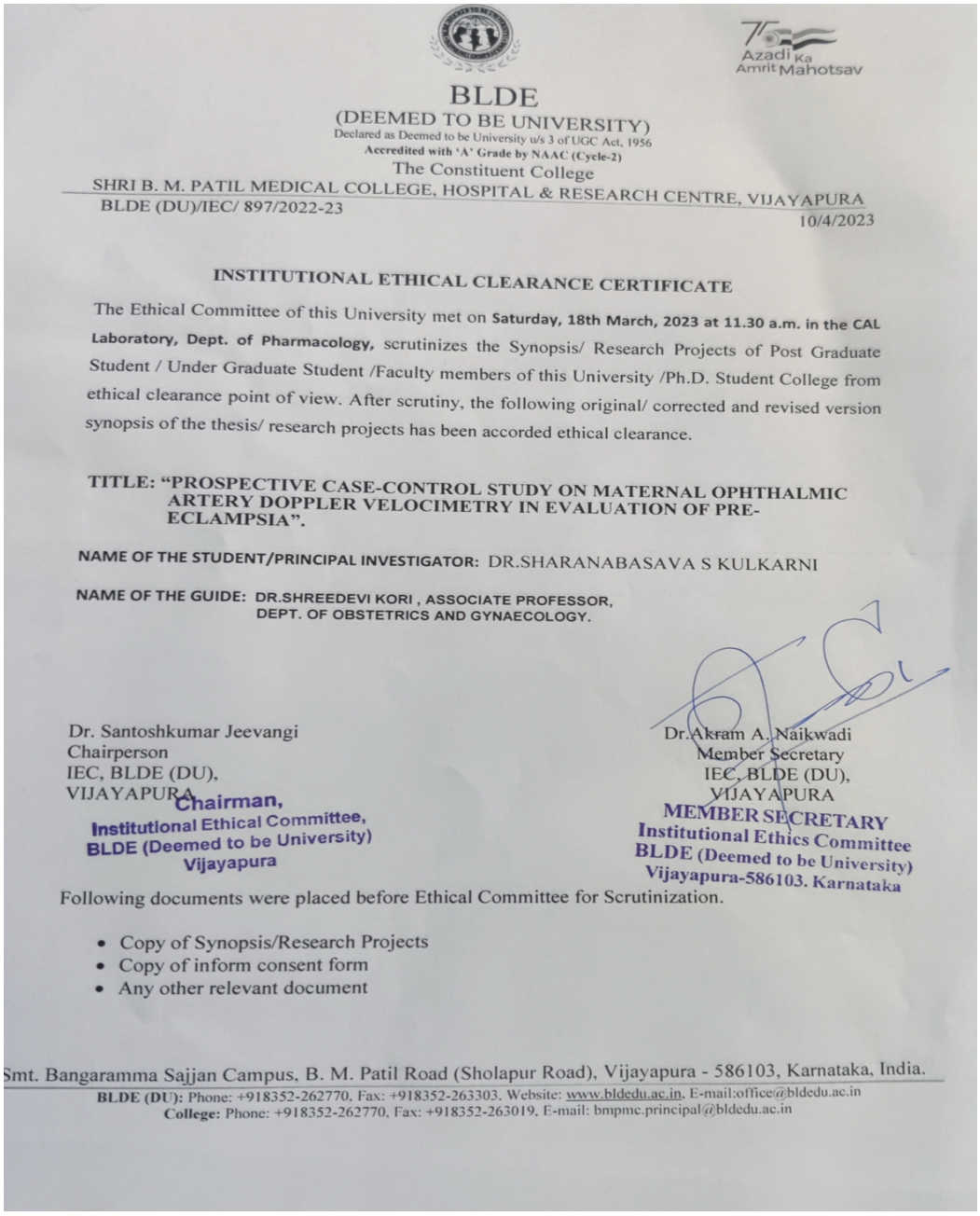

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The Ethical Committee of this University met on **Saturday, 18th March, 2023 at 11.30 a.m. in the CAL Laboratory, Dept. of Pharmacology,** scrutinizes the Synopsis/ Research Projects of Post Graduate Student / Under Graduate Student /Faculty members of this University /Ph.D. Student College from ethical clearance point of view. After scrutiny, the following original/ corrected and revised version synopsis of the thesis/ research projects has been accorded ethical clearance.

TITLE: "PROSPECTIVE CASE-CONTROL STUDY ON MATERNAL OPHTHALMIC ARTERY DOPPLER VELOCIMETRY IN EVALUATION OF PRE-ECLAMPSIA".

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: DR.SHARANABASAVA S KULKARNI

NAME OF THE GUIDE: DR.SHREEDEVI KORI , ASSOCIATE PROFESSOR, DEPT. OF OBSTETRICS AND GYNAECOLOGY.

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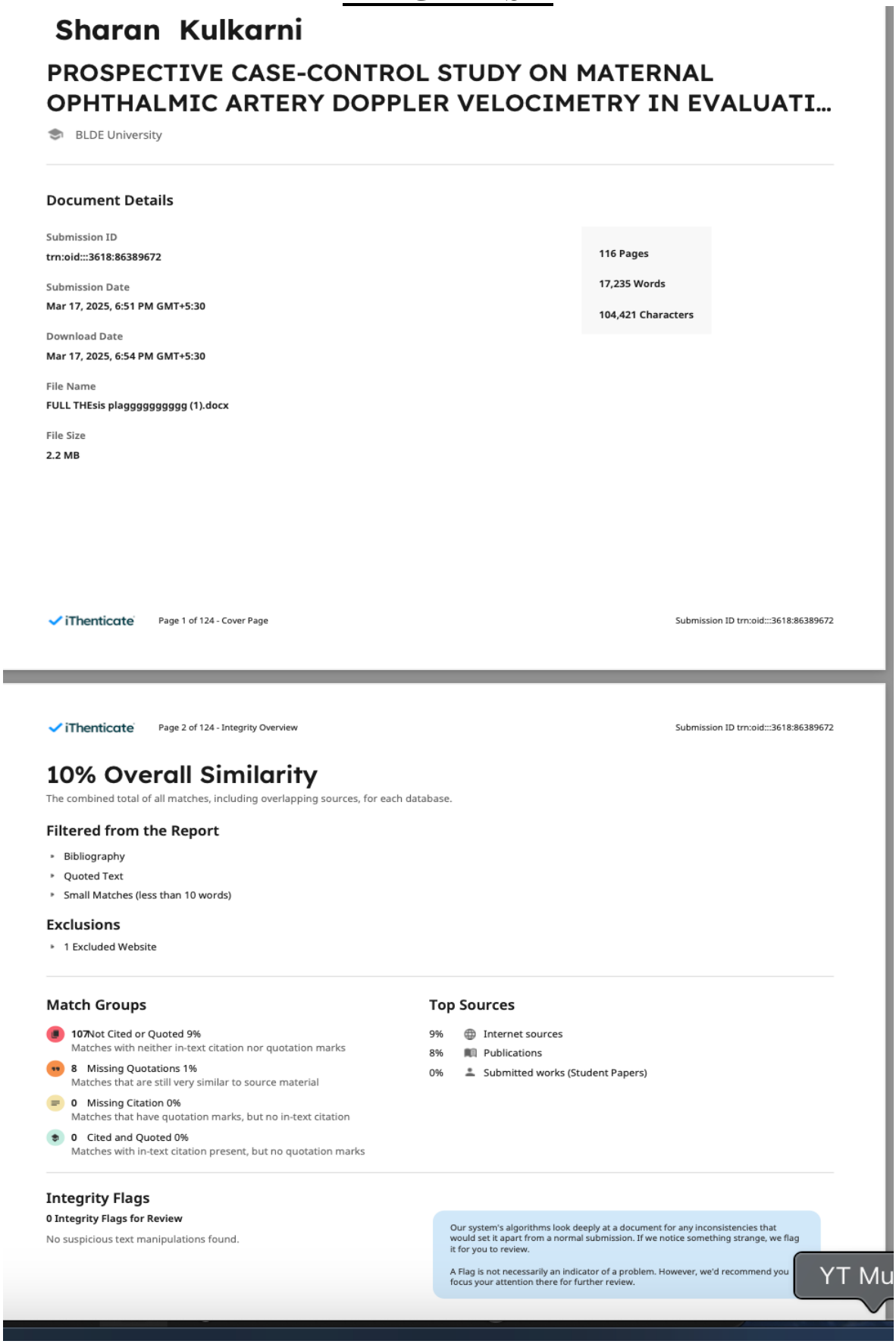

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