“ROLE OF SHEAR WAVE ELASTOGRAPHY OF PLACENTA IN NORMAL AND PRE-ECLAMPTIC PREGNANCIES IN THIRD TRIMESTER.”

By

Dr. SHANTALA SAWKAR

DISSERTATION SUBMITTED TO

SRI DEVARAJ URS ACADEMY OF HIGHEREDUCATION AND RESEARCH, KOLAR, KARNATAKA

In partial fulfilment of the requirements for the degree of

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IN

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Under the Guidance of

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SRI DEVARAJ URS MEDICALCOLLEGE,

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LIST OF ABBREVIATIONS

AFI	Amniotic fluid index
AIUM	American Institute of Ultrasound Medicine's
ARFI	Acoustic radiation force impulse
AGA	Appropriate for gestational age
ACOG	The American College of Obstetricians and Gynecologists
AUC	Area under the ROC curve
BMI	Body mass index
DBP	Diastolic blood pressure
DIC	Disseminated intravascular coagulopathy
E	Young's modulus
EFW	Estimated fetal weight
EDD	Expected date of delivery
EVT	Extravillous trophoblast cells
FHR	Fetal heart rate
GA	Gestational age
GH	Gestational hypertension
HELLP	Hemolysis, Elevated Liver enzyme levels, and Low Platelet levels
HTN	Hypertension
IUGR	Intrauterine growth restriction
ISSHP	The International Society for the Study of Hypertension in Pregnancy
kPa	Kilopascal
LMP	Last menstrual period

NPV	Negative predictive value
Pa	Pascal
PI	Pulsatility index
pSWE	Point shear wave elastography
PPV	Positive predictive value
psi	pounds per square inch
RF	Radiofrequency
ROI	Region of interest
ROC	Receiver operating characteristic curve
SWE	Shear wave elastography
SWV	Shear wave velocity
SE	strain elastography
SWI	Shear Wave Imaging
SGA	Small for gestational age
SBP	Systolic blood pressure
SLE	Systemic lupus erythematosus
TI	Thermal Index
USE	ultrasound shear elastography
2D-SWE	Two-dimensional shear wave elastography
TE	One – dimensional transient elastography
US	Ultrasound
USG	Ultrasonography

`

`ROLE OF SHEAR WAVE ELASTOGRAPHY OF PLACENTA IN NORMAL AND PRE-ECLAMPTIC PREGNANCIES IN THIRD TRIMESTER

ABSTRACT

INTRODUCTION

Pre-eclampsia (PE) is a pregnancy-related condition characterized by new-onset hypertension (with systolic blood pressure > 140 mm Hg and diastolic blood pressure > 90 mm Hg on two occasions at least 4 hours apart, or more severe cases with higher values) along with proteinuria (elevated protein levels in urine). This condition resolves within the first 6 weeks after childbirth. This abnormal placental attachment and ineffective invasion of trophoblast cells into the muscular spiral arteries leads to hypoxia and placental ischemia. Shear wave elastography (SWE) is an innovative ultrasound technique designed to assess the elasticity of soft tissues. In the placenta, increased stiffness is a consequence of ischemia-related processes including inflammation, necrosis, infarction and fibrosis. The present study is planned to assess the utility of SWE in evaluation of placental function and can be used as a supplement to existing methods for prediction of PE.

OBJECTIVES

- To assess placental stiffness by shear wave elastography in third trimester.
- To compare placental elastography findings in normal and pre- eclamptic pregnancies in third trimester.

MATERIAL AND METHODS

This prospective case control study which includes 134 pregnant women in third trimester referred for obstetric scan on whom B-mode sonography and shear wave elastography was performed. Obstetric sonography and elastography of placenta will be performed using Philips EPIQ5 system equipped with shear wave point quantification, ELASTPQ, using curvilinear broadband transducer C5-1MHz. 67 women who had clinically normal pregnancies with normal fetal biometric measurements without any perinatal complications formed group A, and 67 women who had a clinical diagnosed preeclampsia formed group B.

RESULTS

Shear wave elastography values for case group 10.98 (9.70-13.13) were significantly higher than those for control group 2.90 (2.78-3.58) ($P<0.001$). No statistically significant difference was found between the elasticity values measured at the centre or edge of the placenta.

CONCLUSION

This study demonstrates that the use of shear wave elastography for detecting Placental stiffness has a good diagnostic performance for detecting Pre-Eclampsia. Shear wave elastography is a novel technique for characterizing tissues that is helpful for assessing tissue characterization, placental function and serves as an addition to current preeclampsia prediction tools.

KEYWORDS

Pre-Eclampsia, Shear Wave Elastography, Placenta, Elasticity, Placental Stiffness.

TABLE OF CONTENTS

`LIST OF TABLES

LIST OF FIGURES

	The mean placental elasticity value was 3.72 kPa & median value was 3.70 kPa. Average SWE value is 3.66 kPa (Normal).	
31.	Ultrasound grey scale images from a 32 year old primigravida at 32 W 2 D Gestation with Pre-eclampsia. 31a. ROI was placed at centre of fundal placenta 31b 5 samples were taken from centre of placenta of placenta in the same patient.The mean placental elasticity value was 12.46 kPa & median value was 12.50 kPa 31c. ROI was placed at edge of fundal placenta 31d. 5 samples were taken from edge of placenta in the same patient. The mean placental elasticity value was 12.78 kPa & median value was 12.80 kPa. Average SWE value is 12.62 kPa (Increased).	49
32.	Ultrasound grey scale images from a 35 year old multipara at 35 W Gestation with Pre-eclampsia. 32a. ROI was placed at centre of fundal right lateral placenta 32b. 5 samples were taken from centre of placenta of placenta in the same patient. The mean placental elasticity value was 13.26 kPa & median value was 13.30 kPa 32c. ROI was placed at edge of fundal right lateral placenta 32d. 5 samples were taken from edge of placenta in the same patient. The mean placental elasticity value was 13.26 kPa & median value was 13.10 kPa. Average SWE value is 13.26 kPa (Increased).	50

INTRODUCTION

INTRODUCTION

Pre-eclampsia (PE) is a disorder of pregnancy characterized by onset of hypertension and proteinuria after 20 weeks of gestation. PE significantly contributes to perinatal and maternal mortality and impacts approximately 5% to 7% of pregnant women globally. The prevalence of PE is approximately seven times higher in developing countries than to developed countries. In Indian population, its reported incidence ranges from 6.9% to 15%. [1,2]

PE is a pregnancy-related condition characterized by new-onset hypertension (with systolic blood pressure (SBP) > 140 mm Hg and diastolic blood pressure (DBP) > 90 mm Hg on two occasions at least 4 hours apart, or more severe cases with higher values) along with proteinuria (elevated protein levels in urine). This condition typically occurs after 20 weeks of gestational age and resolves within the first 6 weeks after childbirth. [3] PE is divided into two distinct types: Early-onset, which occurs before 34 weeks of gestation and Late-onset, which manifests at or after 34 weeks of gestation.

Early onset PE is primarily attributed to inadequate placental implantation in the uterine lining. This abnormal placental attachment results in impaired utero-placental blood flow, inflammation & endothelial dysfunction. Ultimately, ineffective invasion of trophoblast cells into the muscular spiral arteries prevents the transformation of arteries into "low-resistance" capacity vessels. This leads to hypoxia and placental ischemia reducing the supply of nutrients to the fetus.[4,5] Late onset PE is characterized by minimal or superficial alterations in the spiral arteries and may be connected to maternal intrinsic factors. [6]

Early identification and effective management of PE are crucial for enhancing the well-being of mother and fetus. Screening for PE relies on assessing maternal factors and

history, such as a prior or family history of PE, nulliparity, maternal age > 35, diabetes, multiple pregnancies, chronic kidney disease and obesity. This screening approach can detect 35% of cases with a false-positive rate of 10%. [7] The accuracy of PE screening has been improved by combining maternal blood biochemical markers with maternal biophysical indicators, such as uterine artery Doppler and mean arterial pressure.[8]

Sonoelastography is a method that can detect variations in the elasticity or stiffness of tissues. Elasticity, in this context, pertains to the way materials respond when subjected to reversible deformation. Changes in the soft tissue stiffness can occur due to a range of physiological or pathological factors. Shear wave elastography (SWE) is an innovative ultrasound technique to assess the elasticity of soft tissues. It operates by generating mechanical vibrations through acoustic radiation force, capturing the lateral propagation of transverse shear waves emanating from the tissue and measuring their velocity. This dynamic approach offers real-time quantitative data, boasts strong reproducibility, avoids compression-related artifacts and can penetrate deeper into tissues compared to static elastography. [9,10]

SWE is a viable technique for assessing the elasticity of placenta. [11] In the placenta, increased stiffness is a consequence of ischemia-related processes including inflammation, necrosis, infarction and fibrosis.[12, 13] Nonetheless, it has been reported that the placenta in cases of PE exhibits varying elasticity values across different regions. [14,15] As an addition to current techniques for PE prediction, the present study aims to evaluate the usefulness of SWE in placental function assessment.

AIMS & OBJECTIVES

AIMS AND OBJECTIVES

The study aims to evaluate shear wave elastography (SWE) values in relation to alterations in placental elasticity in both PE and normal pregnancies, with the goal of determining its effectiveness as a diagnostic tool for assessing the disease.

Objectives:

1. To assess placental stiffness by shear wave elastography in third trimester.
2. To compare placental elastography findings in normal and pre- eclamptic pregnancies in third trimester.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

PREECLAMPSIA (PE):

PE (previously known as toxaemia of pregnancy) is observed over 200 years. Despite this long history, our knowledge of the pathogenesis of these illnesses is still limited which has hindered the development of effective treatments. It has been established for quite some time that these conditions are primarily related to the placenta and that the symptoms typically resolve after the placenta is delivered. Consequently, from a pathogenesis perspective, these conditions are fundamentally placental disorders. [16]

Epidemiology of Preeclampsia (PE):

Based on data from approximately 39 million pregnancies worldwide, a global estimation indicates an incidence rate of 4.6%.[17] This condition accounts for 2 to 8% of complications related to pregnancy, resulting in more than 50,000 maternal fatalities and over 500,000 fetal deaths on a global scale. It leads to 9% to 26% of maternal fatalities in low-income countries and 16% in high-income nations. [18] In the Indian population, its reported incidence ranges from 6.9% to 15%.[1,2] In the study conducted by Agarwal S et al.(2022) in Kanpur , India, incidence of non-severe PE was 13.2 % and severe PE was 3.19 % (As shown in Figure 1). [19]

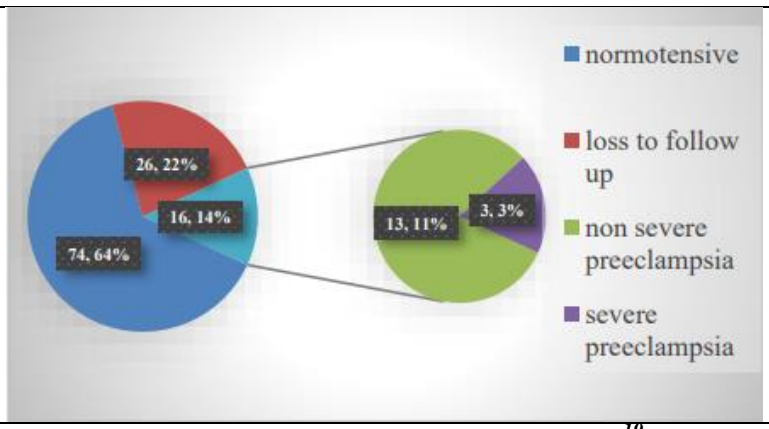

Figure. 1: Incidence of preeclampsia.[19]

Risk factors:[20,21,22]

- Maternal age >40
- Pre-pregnancy BMI >30
- Previous pre-eclampsia
- Previous intrauterine growth restriction
- History of placental abruption
- Chronic hypertension
- Antiphospholipid antibody syndrome
- Systemic lupus erythematosus (SLE)
- Pre-gestational diabetes
- Chronic renal disease
- Nulliparity
- Multifetal pregnancy
- Previous stillbirth
- Increased pre-pregnancy BMI
- Long inter-pregnancy interval (>5 years)
- Assisted reproduction

There are various hypertensive conditions that can occur in pregnancy as mentioned below in Figure 2.

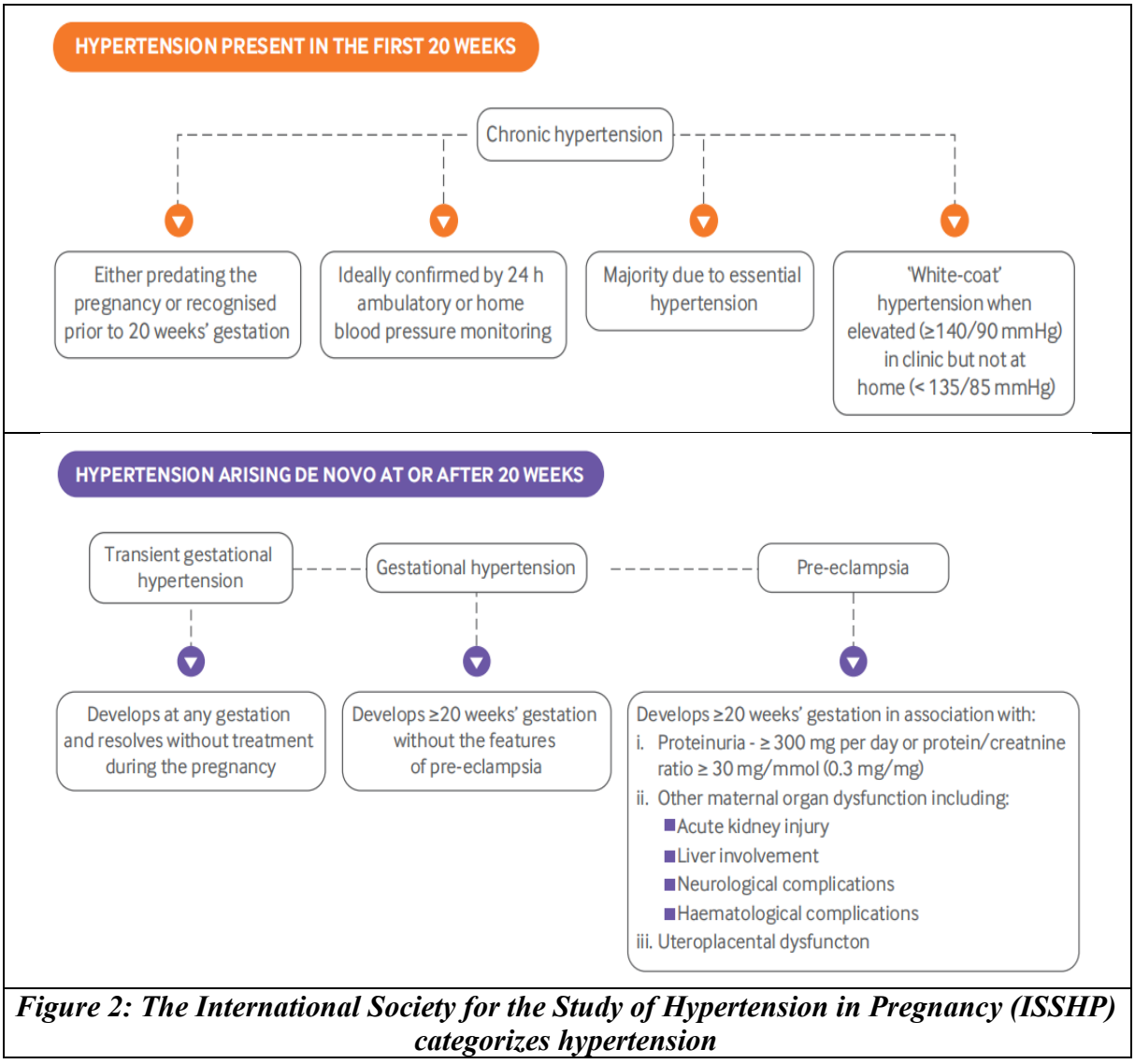

Figure 2: The International Society for the Study of Hypertension in Pregnancy (ISSHP) categorizes hypertension

Placental development, spiral artery remodeling and early onset pre-eclampsia:

Placental development proceeds rapidly, by the end of the third week following fertilization, a protective covering of trophoblast cells has completely encircled the conceptus and made contact with the maternal tissues. The formation of a strong protective barrier is crucial because it isolates the conceptus and shields it from potentially harmful levels of oxygen and foreign substances during the crucial period of organ formation. [23] The initiation

of this development is triggered by histotroph, often referred to as "uterine milk," originating from the endometrial glands.

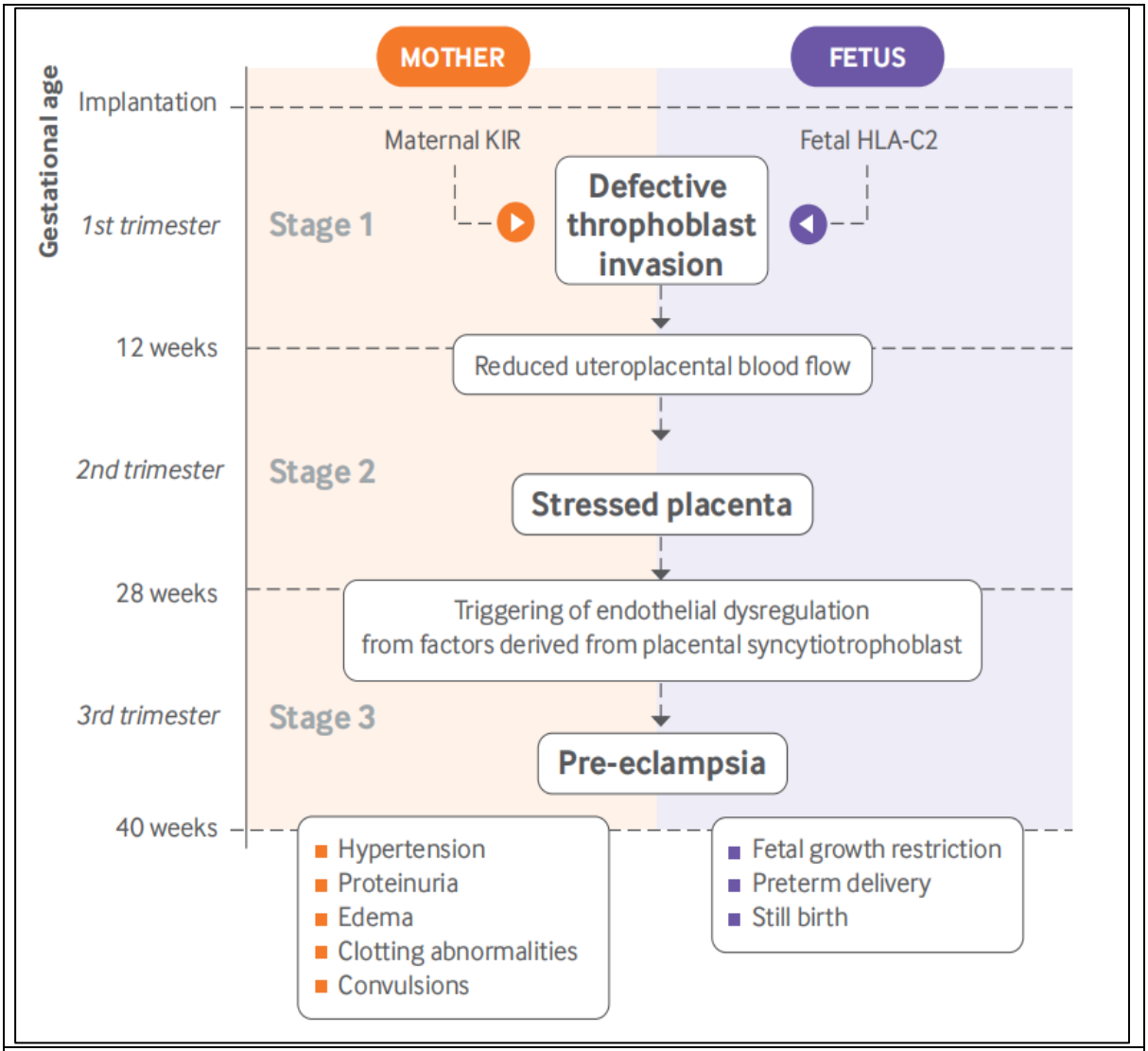

Figure 3: The pathophysiology of pre-eclampsia and its impact on the fetus and mother. The placenta experiences a stress reaction as a result of the trophoblast-uterine interactions failing during the first trimester

Extravillous trophoblast cells (EVT) originate from external surface of the protective shell through a process involving partial epithelial-to-mesenchymal transition. During this transition, these cells transform into invasive cells that are marked by the presence of Human leukocyte antigen-G. Individual EVT migrate by utilizing matrix metalloproteinases through two distinct pathways. Interstitial EVT first migrate toward the

spiral arteries via the stromal tissue. These arteries ultimately provide blood to the placenta since they serve as the terminal branches of the uterine vascular network inside the endometrium. These arteries have a lot of smooth muscle in their walls that is very sensitive to hormonal and vasoactive signals in the non-pregnant state. However, during a normal pregnancy, EVT cells break down the elastin and smooth muscle in these arteries, which are then replaced by non-reactive fibrinoid material. [4]

The remodeling process has two primary effects on the blood flow from the uterus to the placenta. Firstly, end portions of the arteries widen in a funnel-like manner as they get closer to the placenta. The placenta's overall blood flow volume remains relatively unchanged during the remodeling process and consequently, the oxygen supply remains relatively unaffected. However, according to mathematical models, it does have a substantial impact on the speed and pulsation of maternal blood entering the placenta, reducing it by approximately tenfold to around 10 cm/s. [24] (Figure 4).

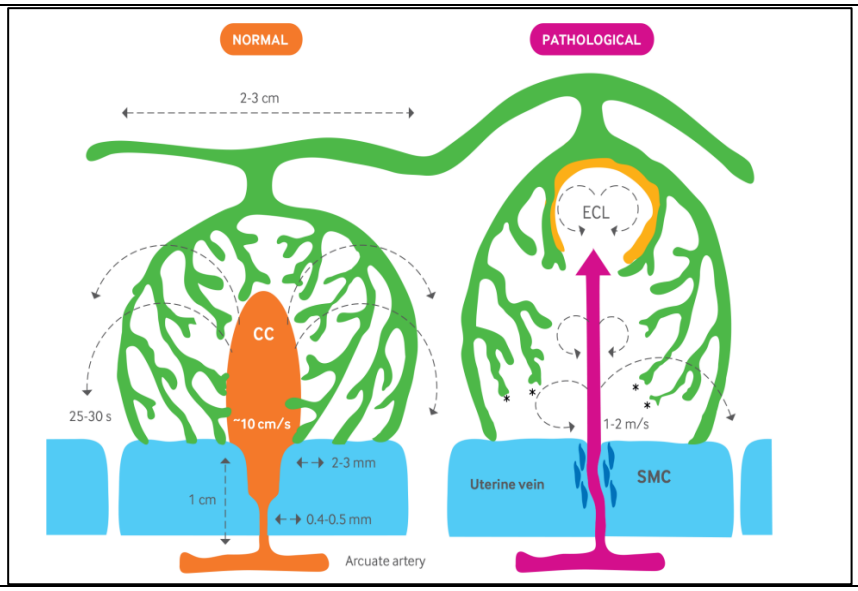

Figure 4: Diagrammatic illustration showing how spiral artery remodeling affects the amount of blood that a mother can inject into the intervillous area during both healthy and pathological pregnancies

The reduction in blood flow velocity, which is end result of the remodeling process, plays a crucial role in safeguarding the microvilli and placental villi from potential damage, particularly when perfusing the placenta in vitro at high flow rates. This slower flow is essential for maintaining the health of the placental tissue. Additionally, trophoblast-driven transformation of spiral arteries typically extends into the inner third of the myometrium. This includes the hypercontractile segment of the artery located at the junctional zone between the endometrium and myometrium, which helps control blood loss during menstruation. During pregnancy, this segment must undergo remodeling to ensure uninterrupted placental blood flow, while other segments of the uteroplacental vasculature dilate in response to different stimuli.

Over the course of pregnancy, the placenta, a dynamic and temporary organ, continuously changes in both structure and function. The placenta usually appears as an oval or round disk after delivery, with a diameter of around 18–20 cm and a weight of about 500 g. Its functional unit is the cotyledon, which is the area where the chorionic villi are submerged between two placental septa. Within these chorionic villi, fetal blood circulates, enabling vital exchanges through the villi structure between the maternal and fetal compartments.

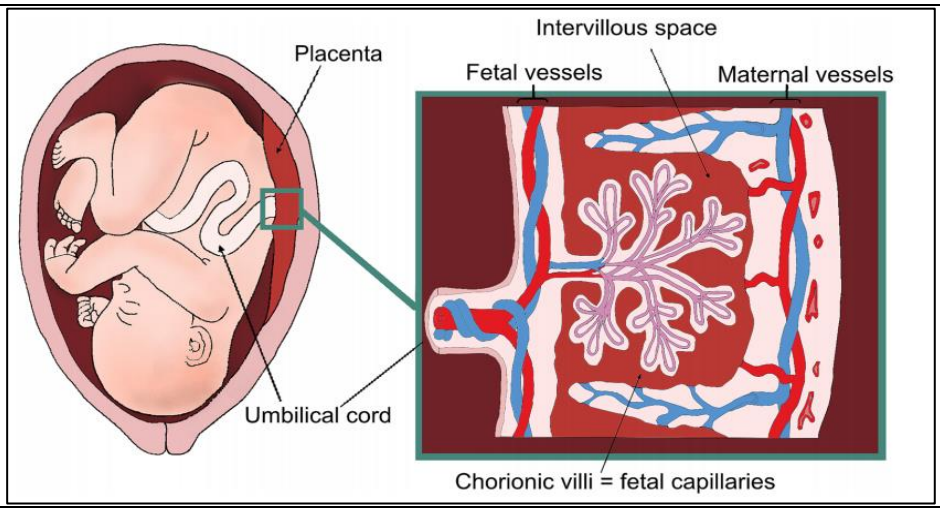

Figure 5: Diagram of the placenta illustrating the branching vascular system of the organ

Morphological changes in the placenta associated with preeclampsia (PE):

The changes in the placenta are expected to occur due to placental ischemia. As anticipated, in preterm PE, the placenta tends to be smaller and may display various forms of infarction. [25]

Preeclamptic placentas often exhibit an oblong shape, unlike the typical circular shape seen in normal pregnancies. This abnormal shape is thought to result from reduced endovascular invasion by trophoblast cells, impacting maternal spiral arteries plugging. Reduced invasion can lead to villus damage, oxidative stress, and villus necrosis, resulting in atypical placental shapes. Additionally, preeclamptic placentas tend to be thicker, possibly due to damage from high-velocity blood flow from untransformed spiral arteries. [26,27]

Pathophysiological of PE:

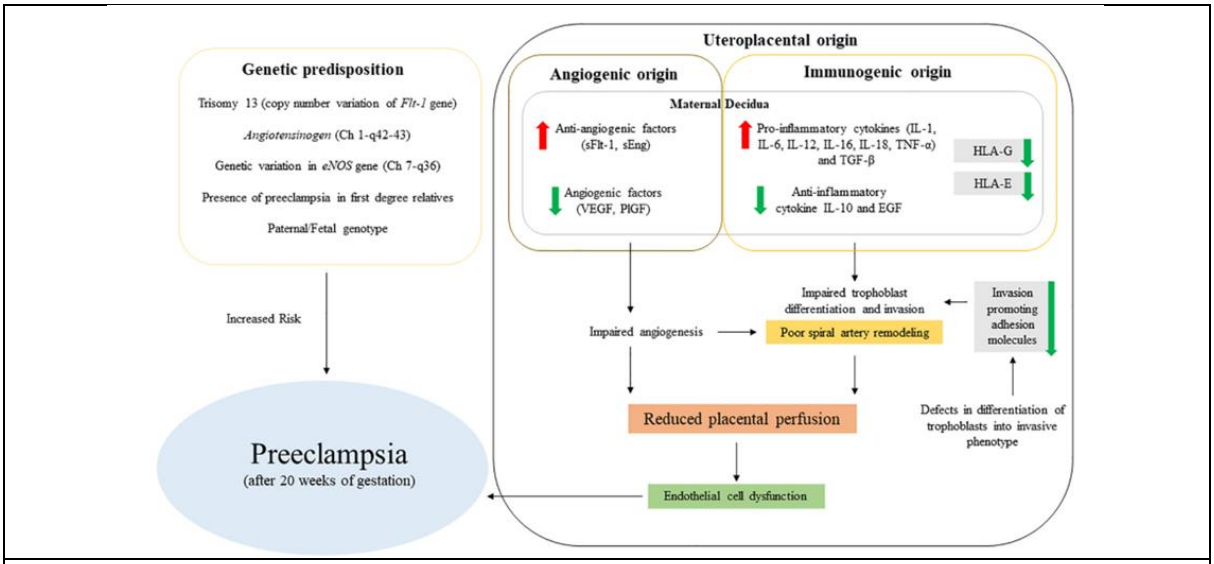

Figure. 6. Theories of origin of preeclampsia. Theories explaining the mechanisms of development of preeclampsia include angiogenic origin, uteroplacental origin, immunogenic origin and genetic predisposition. [28]

The American College of Obstetricians and Gynecologists (ACOG) has established the following diagnostic standards for preeclampsia. [29] :

a. **Blood pressure:**

A diagnosis of hypertension (HTN) in pregnancy is made if a woman who previously had normal blood pressure exhibits SBP of 140 mm Hg or higher or DBP of 90 mm Hg or higher on two separate occasions, each at least 4 hours apart, after the 20 week of gestation.

In cases of severe hypertension, a diagnosis can be promptly confirmed, often within minutes, if systolic blood pressure reaches 160 mm Hg or more or diastolic blood pressure is 110 mm Hg or more. This swift confirmation is crucial to initiate timely antihypertensive treatment.

b. **Proteinuria:**

- Excretion of total protein of 300 mg or higher in a 24-hour urine collection (or an equivalent amount extrapolated from a timed collection).
- A dipstick reading showing 2+ protein levels (to be used only in situations where other quantitative methods are not accessible).
- A protein/creatinine ratio measuring 0.3 mg/dL or greater.

Alternatively, when proteinuria is not present, new-onset hypertension accompanied by new onset of any of the following:

- Renal insufficiency: Elevated serum creatinine levels exceeding 1.1 mg/dL or a twofold increase in serum creatinine concentration without any other underlying renal disease.
- Thrombocytopenia, where platelet count lower than 100,000 x 10^{9} /L.

- Impaired liver function: Elevated blood levels of liver transaminases to twice the normal concentration.
- Pulmonary edema.
- New-onset headache that does not respond to medication and cannot be explained by other medical conditions or visual symptoms.

Preeclampsia with severe features:

- Elevated blood pressure with a systolic reading of 160 mm Hg or higher or a diastolic reading of 110 mm Hg or higher, confirmed on two separate occasions at least 4 hours apart unless antihypertensive treatment is initiated before this interval.
- Thrombocytopenia, where platelet count lower than $100{,}000 \times 10^{9}$/L.
- Renal insufficiency: serum creatinine concentration exceeding 1.1 mg/dL or a twofold increase in serum creatinine concentration when there is no other underlying renal disease.
- Impaired liver function, characterized by abnormally elevated levels of liver enzymes (exceeding twice the upper limit of normal concentrations), or severe persistent upper right quadrant or epigastric pain that does not respond to medications and cannot be attributed to other diagnoses.
- Pulmonary edema.
- New-onset headache that does not improve with medication and cannot be explained by other medical conditions.
- Visual disturbances.

Complications of Pre- eclampsia. [30]:

Maternal complications:

- Hemolysis, Elevated Liver enzyme levels, and Low Platelet levels (HELLP) syndrome with or without liver haemorrhage.
- Placental abruption with or without disseminated intravascular coagulopathy (DIC).
- Acute renal failure (which may require dialysis)
- Pulmonary edema.
- Eclampsia (which may be complicated by aspiration pneumonitis)
- Retinal detachment with or without underlying retinopathy.
- Adult respiratory distress syndrome.
- Stroke (encephalopathy or cerebral haemorrhage)
- Death.

Fetal complications:

- Fetal growth restriction.
- Hypoxia – acidosis.
- Oligohydramnios.
- Preterm delivery
- Death.
- Long term morbidity :
    - Cerebral palsy
    - Cardiovascular disease
    - Neurological deficit

PLACENTA:

The placenta plays a significant role throughout pregnancy and works in coordination with the fetal membranes and amniotic fluid to support the healthy growth and development of the developing fetus. The fetus's capacity to adapt to the intrauterine environment is significantly impacted by changes in placental development and function. Maternal and embryonic cells interact during the highly coordinated process of placenta development and implantation.

The trophoblast cell infiltration of uterine tissues and remodeling of uterine spiral artery walls ensures enough blood supply for the developing feto-placental unit, as well as effective gas & nutrition transfer and waste elimination.[31]

IMAGING OF PLACENTA:

Ultrasonography (USG) is the preferred imaging modality of choice. The placenta becomes apparent at transabdominal US at 10 weeks of gestation, the placenta is seen as a thicker, echogenic rim of tissue encircling the gestational sac. The placenta is well developed and the retroplacental (subplacental) hypoechoic zone is visible by 15 weeks of gestation.

Normal placenta appears discoid, uniformly echogenic, and has rounded edges. It often extends into the lateral walls of the uterus and is found along the posterior or anterior walls. The placenta's mid part normally measures between 2-4 cm. Few focal sonographic lucencies may be present with sluggish flow representing venous lakes. [32]

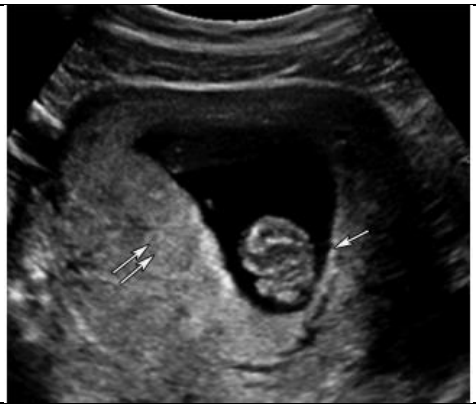

Figure 7. Normal placenta at 10 weeks gestation. Transverse gray-scale US image shows the chorion laeve (right arrow) and chorion frondosum (left arrows) of the placenta

ELASTOGRAPHY:

A non-invasive imaging technique that can be used to measure the stiffness or elasticity of tissues. This is accomplished by measuring the displacement or deformation of tissue in response to a small applied pressure. It is a method of "virtual palpation" of tissue or lesions. It can provide objective and quantitative measures of tissue stiffness. This can be useful for diagnosing and monitoring a variety of conditions.[33]

Basic physics:

a. Stress:

Stress is defined as the force applied per unit area and is typically measured in units such as Pascal (Pa) or pounds per square inch (psi) (1 Pascal equals 1 Newton per square meter). Stress can result from compression, which acts perpendicular to a surface and leads to the shortening of an object. Shear stress, on the other hand, acts parallel to a surface and causes deformation.

In elastography, stress can be induced externally through methods like transducer compression, acoustic radiation force or vibrators. Alternatively, endogenous motion generated by factors like vascular movement, respiratory or cardiac motion can

also be employed. While endogenous sources offer advantages over exogenous sources, such as overcoming issues like attenuation (e.g., due to ascites or obesity), quantifying endogenous stress can be challenging.[34]

b. Strain:

When an object is subjected to stress, it undergoes deformation. The amount of deformation is known as strain. Strain is unitless, and it is expressed as the change in length per unit length of the object. Hard objects have lower strain values than softer objects.

When compression is applied, lesions that are closer to the applied force will undergo more displacement than objects that are lying in a deeper plane. This is similar to the clinical difficulty in palpating deep-seated lesions.[33]

c. Elasticity:

Elasticity is the property of materials to return to their original shape after stress is removed. Elastic materials deform immediately when stressed and also return quickly to their original position. [33]

Hooke's law establishes a relationship where stress is directly proportional to the strain experienced by an object within its elastic limit. Young's modulus (E) is the measure of this relationship, calculated as the ratio of stress to strain, and it shares the same units as stress. Young's modulus quantifies how resistant a tissue is to compression. Hooke's law is applicable to homogeneous isotropic solids. In softer tissues like fat, the application of stress, such as through compression during palpation, leads to a greater degree of deformation (strain). Conversely, harder tissues like muscle and fibrous tissue offer higher resistance to strain, resulting in a higher Young's modulus value.[35-36]

Shear modulus:

Also known as the modulus of rigidity (G), represents the relationship between shear stress and shear strain. Elasticity imaging techniques can be founded on the imaging of various parameters, including strain, stress, Young's modulus, shear modulus, or shear wave velocity.[33]

Elastography techniques can utilize alterations in the elasticity of soft tissues resulting from distinct physiological or pathological conditions. It is well-established that alterations in tissue firmness play a role in numerous medical conditions, including cancerous tumors, fibrosis in liver cirrhosis, and the development of atheromas and calcifications associated with arteriosclerosis. [37] Elastography enhances conventional ultrasound by introducing stiffness as an additional characteristic to the existing ultrasound imaging methods. The main elastography procedure steps can be summed up as shown in figure 8. [38]

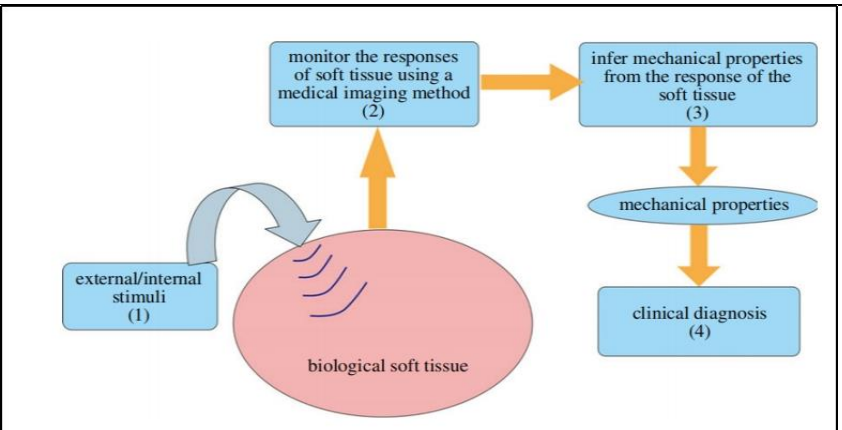

Figure 8: A diagram illustrating the main steps in elastography [38]

Principles and Techniques of Ultrasound Elastography:

Physics of ultrasound elastography: [39,40]

Elastography is an imaging modality used to evaluate soft tissue elasticity. The ability of a tissue to either regain its original shape after a force is removed or to resist deformation in response to an applied force is known as elasticity. Hooke's law can be used to explain elasticity if a material is completely elastic and its deformation is independent of time (i.e., it is not viscous).

$$\sigma = \Gamma \cdot \varepsilon \quad \rightarrow \text{Equation 1}$$

where, σ (Stress) = Force per unit area with unit kilopascal (i.e; N/m2) (Fig 9, top row)

ε (Strain) = Expansion per unit length which is dimensionless (Fig 9, second row)

Γ (Elastic modulus) = Relates stress to strain with unit kilopascal (Fig 9, third row)

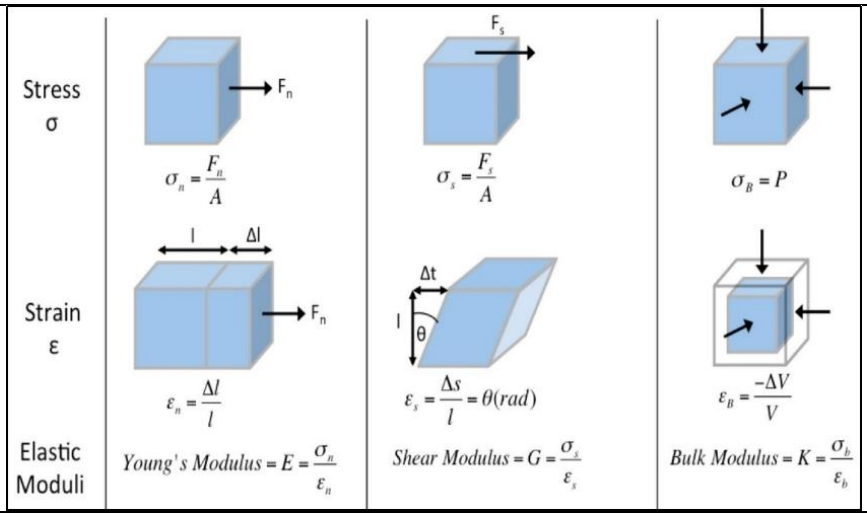

Figure 9: Ultrasound elastography physics, deformation models. Static deformations of entirely elastic materials can be described by stress σ (force per unit area, top row), strain ε (expansion per unit length, middle row), and elastic modulus Γ (stress divided by strain, bottom row). This is applied to normal (perpendicular to surface, first column), shear (tangential to surface, second column), and bulk (normal inward or pressure, third column) forces used in ultrasound elastography

There are two types of wave propagation in ultrasound: longitudinal waves and shear waves as described in Figure 10:

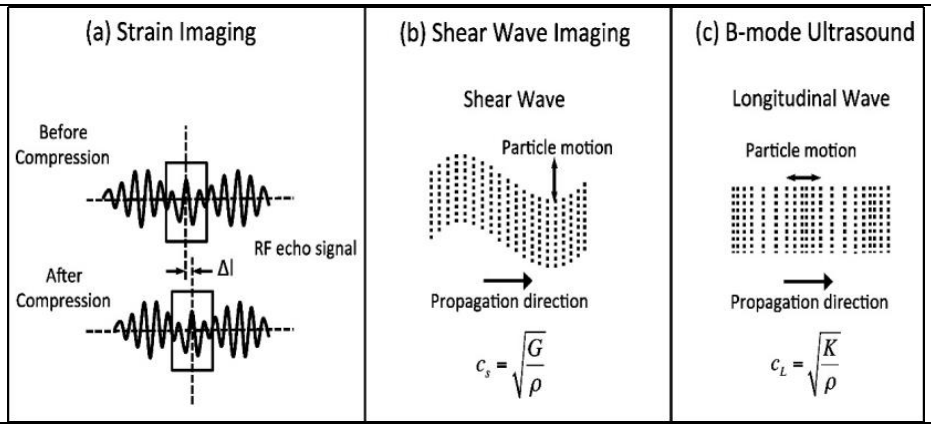

Figure 10: Ultrasound elastography physics, measurement methods. In strain imaging (a), tissue displacement is measured by correlation of RF echo signals between search windows (boxes) in the states before and after compression. In shear wave imaging (b), particle motion is perpendicular to the direction of wave propagation, with shear wave speed C_s related to shear modulus G. In B-mode ultrasound (c), particle motion is parallel to the direction of wave propagation, with longitudinal wave speed C_L related to bulk modulus K

Ultrasound elastography methods:

Various currently accessible ultrasound elastography (USE) techniques can be categorized according to the specific physical quantity they measure as:

1) Strain imaging

2) Shear wave imaging

1) STRAIN IMAGING: In this method, tissue is subjected to a normal stress σ_n, and the resulting normal strain ε_n is measured (as depicted in Figure 11, in the first column). Strain imaging was the initial ultrasound shear elastography (USE) technique to be introduced.[41]

There are two forms of strain imaging like acoustic radiation force impulse (ARFI) and strain elastography (SE) (Figure 11)

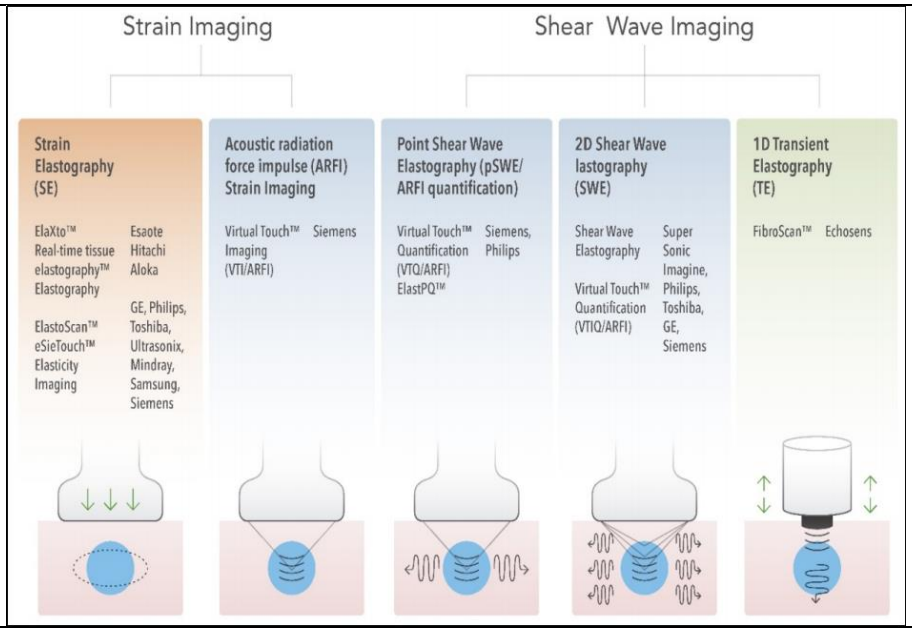

Figure 11: Ultrasound Elastography Techniques

a. Strain elastography (SE): SE divided into two types by excitation method:

i). In the first approach, the operator applies manual compression to the tissue using the ultrasound transducer.[43] While manual compression is effective for evaluating elasticity in superficial organs like the breast and thyroid, it presents difficulties when assessing the elasticity of organs located deeper within the body, such as the liver.[42]

ii. In the second method of excitation, the ultrasound transducer remains stationary, and tissue displacement is induced by internal physiological motions, such as those related to the cardiovascular or respiratory systems. Because this approach doesn't rely on externally applied compression, it can be employed effectively to evaluate the elasticity of organs located at greater depths within the body.[43]

The amount of tissue displacement in the same direction as the applied stress is measured using a variety of methods, depending on the manufacturer. These methods include radiofrequency (RF) echo correlation-based tracking, Doppler processing, or a combination of the two methods. [44]

The strain measurements are presented in the form of a semi-transparent color map referred to as an elastogram, which is superimposed onto the B-mode image. Generally, the elastogram portrays low strain (indicating stiff tissue) in blue and high strain (indicating soft tissue) in red. However, it's important to note that the specific color scale used can vary depending on the ultrasound equipment manufacturer.[43,45]

b. Acoustic radiation force impulse (ARFI) strain imaging: An alternative method for measuring strain involves the use of a short-duration (ranging from 0.1 to 0.5 milliseconds) high-intensity acoustic "pushing pulse" (acoustic radiation force), with spatial peak pulse average energy of 1400 W/cm2 and spatial peak temporal average energy of 0.7 W/cm2. This pulse is employed to induce tissue displacement, typically in the range of approximately 10 to 20 micrometers, in the normal direction, which is perpendicular to the surface.[46]

In ARFI imaging, the displacement of the tissue is measured within a specified ROI. The displacements can then be displayed as an elastogram overlaid on the B-mode image.

Siemens Virtual TouchTM Imaging is a commercial implementation of ARFI imaging. It is used to image the liver, breast, and other soft tissues.[47]

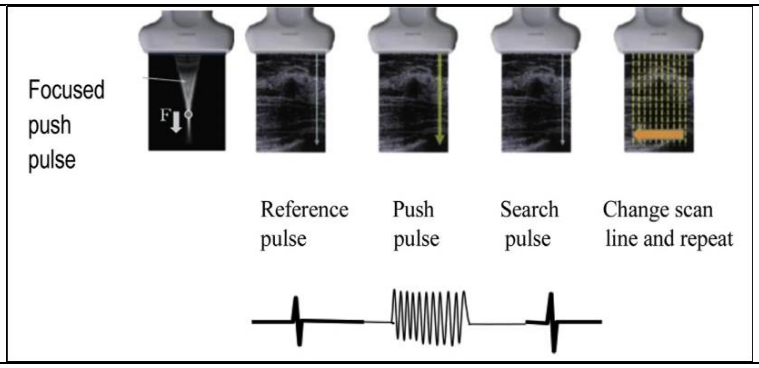

Figure 12. Pulse sequence in ARFI Imaging

2) Shear wave imaging (SWI):

Shear waves in the parallel or perpendicular dimensions are produced by Shear Wave Imaging (SWI), which makes use of a dynamic stress. Both qualitative and quantitative measures of tissue elasticity are produced by measuring shear wave speed in SWI.[48]

In SWE, elastograms are produced by combining an ultrafast imaging sequence that can record the propagation of the ensuing shear waves in real time with a radiation force that an ultrasonic beam induces in a tissue. Shear waves are created by the ultrasonic probe's highly focused radiation force, and they travel straight from the focal point into the tissue of interest. A subsequent change in the depth of focal location leads to interference of shear waves and the generation of a conical shear wave. This technique requires very fast acquisition of ultrasound images, at least 5000 frames per second up to 20,000 frames per second. Such fast acquisition reduces the risk of artifacts made by patient or investigator movements. SWE makes it possible to create a two-dimensional color map, where color codes speed of wave in meters per second or elasticity of the tissue in kilopascals. However, this method has limits on the intensity used to avoid both mechanical and thermal bio-effects so it may cause difficulties in analyzing deeper-located tissues. The technique is performed using a conventional linear array probe, thus can be incorporated into standard diagnostic ultrasound examinations. [49]

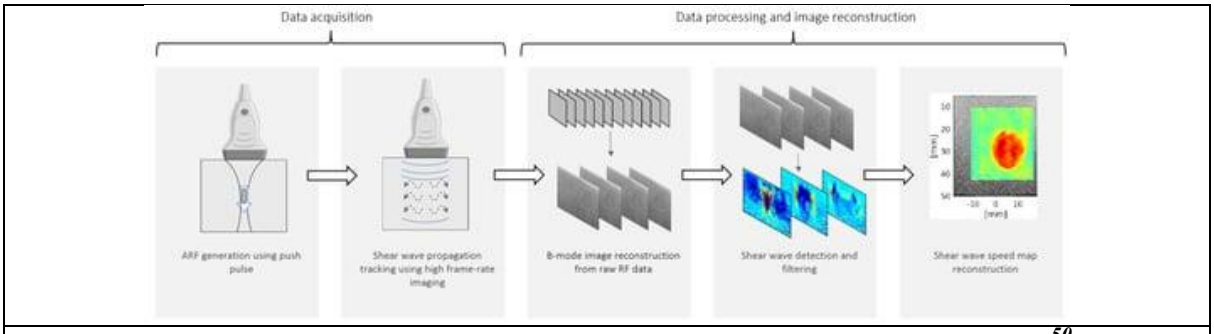

Figure 13: A general measurement procedure of the SWE technique.[50]

There are three approaches for SWI (as summarised in Table 1) :

a. Point shear wave elastography (pSWE)
b. Two-dimensional shear wave elastography (2D-SWE)
c. One – dimensional transient elastography (TE)

pSWE	2D-SWE	1D-TE
- Excitation method: dynamic stress by ARFI, in the normal direction, in a single focal location. - Shear waves measured perpendicular to plane of excitation. - Shear wave speed ***(C_s)*** reported or converted in Young's modulus (***E)*** to provide quantitative estimate of tissue elasticity. - Operator can use B-mode US to directly visualize and select ROI. - Does not show an image of stiffness. - Can be performed on conventional US machine using standard ultrasound probe. - Became available in 2008.	- Excitation method: dynamic stress by ARFI, in the normal direction in multiple focal zones - Shear waves measured perpendicular to ARFI application. - Multiple focal zones are interrogated in rapid succession, faster than the shear wave speed, creating a near cylindrical shear wave cone, allowing real-time monitoring of shear waves in 2D for measurement of C_s or ***E*** and generation of quantitative elastograms. - Operator is guided by both anatomical and tissue stiffness information, has real-time visualization of a color box; quantitative elastogram superimposed on a B-mode image stiffness information. - Currently newest SWI method.	- Excitation method: dynamic stress by a mechanical vibrating device. - Shear waves measured parallel to excitation. - Stiffness estimated along ultrasonic A-line, in a fixed region, neither user adjustable nor image guided. - Operator selects imaging area using time-motion ultrasound, based on multiple A-mode lines in time at different proximal locations forming low quality image. The same probe uses A-mode US to measure C_s and ***E*** is calculated. - First system commercially available. The most widely used and validated technique for assessment of liver fibrosis.

Table 1: Summary of shear wave imaging methods. [48]

The main advantage of SWE is that the usage of the ARFI eliminates the need for external, operator-dependent stress. Moreover, SWE can measure soft tissue stiffness both qualitatively and quantitatively, whereas strain elastography solely offers quantitative maps of soft tissue stiffness. Along with having many uses, including fibrosis assessment for chronic liver disease, breast cancer screening, thyroid nodule assessment, gastrointestinal wall diagnostics, prostate abnormality screening, and cardiovascular system diagnostics, it also shares many characteristics with ultrasound imaging, including being non-invasive, quick, and reasonably inexpensive. [50]

Elastography on placenta:

In clinical practice, mechanical properties of placenta are not explored. Placental elasticity and viscosity could be modified in case of complicated pregnancy. Elastography is relevant tool for studying biomechanical properties of a tissue. Both in vivo and ex vivo sono-elastography studies of normal placenta have been performed and wide range of intraplacental elasticity have been reported.

Before performing in vivo placental Shear Wave Elastography (SWE) on pregnant patients, a series of steps are followed. Patients are advised to empty their bladders, maintain a calm breathing pattern, lie supine, and expose their abdomen. The ultrasound transducer is gently placed on the skin to minimize pressure on the fetus and positioned away from the umbilical cord for effective placental examination. The examiner waits for fetal stillness and, if there are no uterine contractions, instructs the patient to briefly hold her breath. The placenta's center and periphery will be the fixed-size region of interest (ROI), a rectangle of 1 x 0.5 cm. A B-mode ultrasonography was used to illustrate the quantitative placental stiffness value. Five samples are taken from each site, from the centre (sample 1) and periphery (sample 2) of the placenta, and will be averaged to obtain samples 1 and 2. In this method ARFI are used to create shear waves that propagate through tissue. Tracking pulses are used to measure shear wave displacements between two points inside a region of interest (ROI), giving the speed of the shear wave at a certain point, hence the name point SWE. [1] Currently, there is no consensus on measurement standards for placental SWE, but standardization and reference values could enhance the assessment of placental function. [51]

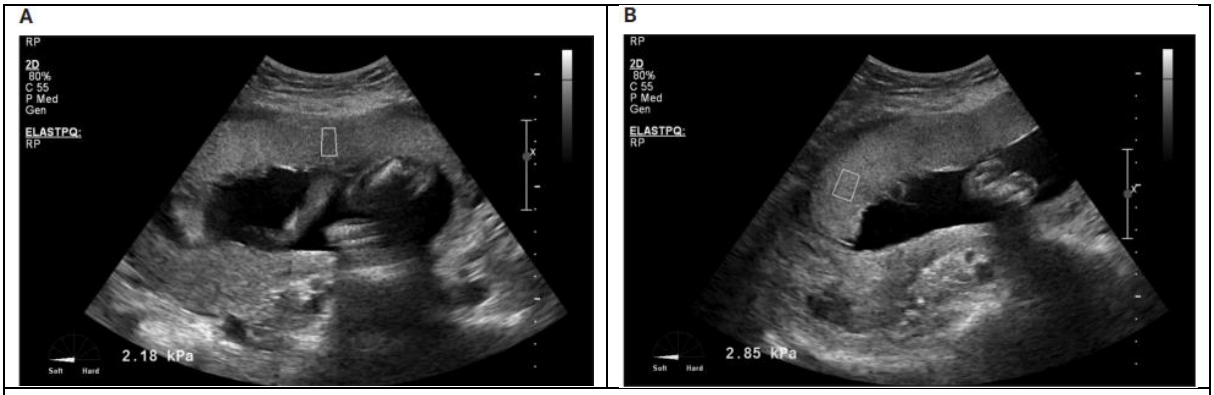

Figure 14: 22-year-old multigravida woman at 21 weeks of gestation. A, The ROI (box) was placed at the center of the anterior placenta (sample 1). The placental elasticity value was 2.18 kPa. B, The ROI (box) was placed at the edge of the anterior placenta (sample 2). The placental elasticity value was 2.85 kPa.[1]

THE VARIOUS FACTORS INFLUENCING ELASTICITY OF PLACENTA:

1) **Maternal BMI**: Spiliopoulos et al.[64] demonstrates that BMI plays a crucial role in predicting placental health in both healthy and preeclampsia pregnancy models, with higher BMI associated with increased placental hardness. Edwards et al. [49] used a linear mixed model to establish that in normal pregnancies, pre-pregnancy obesity leads to a significant increase in placental stiffness and weight gain during pregnancy also contributes to greater placental stiffness.
2) **Deep breathing and fetal movements:** It has been observed that pregnant women with fetal movement's and deep breathing exhibit significantly higher shear wave velocity (SWV) compared to those with no fetal movement and shallow breathing. [52]
3) **Sample depth:** Edwards et al. found that when restricting the sample depth to a range of 2-6 cm, the maximum change in the mean SWV measurement was 0.21 m/s, only slightly higher than the sample difference of 0.71 m/s. However, in clinical practice, it is challenging to consistently achieve the recommended 2-6 cm depth for the placental region of interest. Presently, it is widely accepted that elasticity measurements at a depth of 8 cm are more meaningful, with no notable differences in elasticity values observed across different placental regions[53,54]. Thus limiting its applicability in posterior placenta.

4) **Gestational age (GA):** Ge et al[55]. and Wu et al [56]. found that differences in GA did not significantly impact placental elasticity values. However, Ohmaru et al [53] reported a slight, non-significant increase in SWVs with higher GA. It remains unclear whether this increase is linked to natural placental maturation. [54].

Safety of SWE in pregnancy: Currently, no reports of acoustic radiation force impulse (ARFI) imaging endangering women's safety during pregnancy exist. The equipment used in the process complies with the American Institute of Ultrasound Medicine's (AIUM) guidelines, which specify that TI < 0.7 and MI ≤ 1.9 are the upper limits for thermal index (TI) even though elastic imaging based on radiation force uses a high TI. Scholars such as Ge et al. have proposed that shear waves are not propagated in amniotic fluid, hence reducing the effect on the developing foetus. SWE lowers the danger of chronic placental irradiation by intermittently emitting low-density acoustic radiation. However, it's suggested to follow time guidelines, like the British Medical Ultrasound Society's (15-minute) recommendation. The safety features of should be further investigated in scientific research.[54,55,57,58]

PLACENTAL ELASTOGRAPHY IN PRE-ECLAMPSIA (PE) PATIENTS:

PE can result in fine atherosclerosis, with uterine artery spasms causing constriction of blood vessels, reduced blood volume, increased resistance and diminished blood supply to the placenta. Additionally, the hypercoagulable state of the mother during PE makes placental micro vessels prone to thrombosis, potentially leading to villous embolism or necrosis.

Consequently, the development of PE involves the deposition of calcification and fibrin in placental tissue, histological changes leading to increased placental stiffness and elasticity values.[59,60].

Studies [1,55] identified correlations indicating increased placental modulus values in their groups with preeclampsia (PE). Interestingly, there were no statistically significant differences in elastic modulus values among different placental regions. Fujita et al [62] et al., demonstrated that placental elastic values (YM) increased before the onset of PE, while umbilical artery blood flow parameters remained unchanged. Kilic et al[61]. found that the central placental area of the fetus had the highest diagnostic accuracy (AUC value of 0.895) for diagnosing preeclampsia, with a threshold of 7.35 kPa, offering 90% sensitivity, 86% specificity, and 88% diagnostic accuracy.

Recent studies [61,62] proposed optimal cutoff values for predicting PE at 1.188 m/s (AUC of 0.912) and 7.43 kPa (AUC of 0.924), respectively, indicating changes in placental elasticity occurring before the onset of PE. Therefore, SWE may be a very sensitive method for identifying abnormal placental changes early in gestational hypertension patients, which could help predict the development of PE.

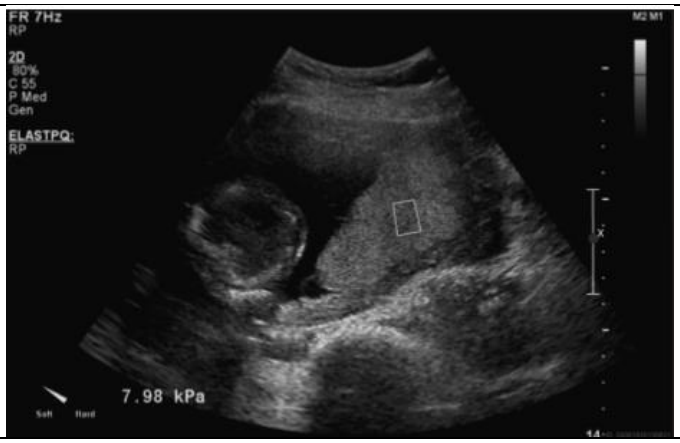

Figure 15: 29-year-old primigravid woman with PE at 23 weeks' gestation. The ROI (box) was placed at the center of the anterior placenta. The placental elasticity value was 7.98 kPa. The placenta is located at the left anterolateral wall. At the bottom left, the scale shows the degree of stiffness

REVIEW OF LITERATURE OF PLACENTAL ELASTOGRAPHY IN PATIENTS WITH PRE-ECLAMPSIA:

Sheeza Imtiaz et al., conducted prospective study from September 15, 2022, to January 15, 2023, Karachi, Pakistan comprised singleton pregnant women during 28-40 weeks of gestation. The subjects were divided into normal pregnancy group A and high-risk pregnancy group B. Risk factors include gestational hypertension, gestational diabetes, intrauterine growth restriction, placenta previa, morbidly adherent placenta, old primigravida, teen age and morbid obesity were noted. Of the 104 subjects, 78(75%) were in group A and 26(25%) were in group B. In group B, mean placental shear wave velocity was 2.34±1.17m/sec and elasticity was 24.41±25.51kPa compared to 1.42±0.55 m/sec and 13.6±10.23kPa in group A ($p<0.05$). Hence, SWE was found to be a useful technique in detecting placental stiffness, and can be used as an adjunct to the currently available ultrasonographic methods in high-risk pregnancies elastography. [63]

Cimcit C et al., conducted a study in 2014 (Istanbul, Turkey), involving 204 singleton pregnancies undergoing anomaly scan between 20 and 23 weeks of gestation, 129 patients received shear wave elastography. Group A consisted of 101 women with normal pregnancies and deliveries, while Group B included 28 women diagnosed with early-onset preeclampsia before anomaly scanning. The study revealed significantly higher shear wave elastographic values in Group B compared to Group A ($P < 0.05$). There was no significant differences found in elasticity values between the centre and edge of the placenta ($P > 0.05$). In conclusion, SWE effectively distinguishes the placental elasticity in normal pregnancies and those affected by preeclampsia during the second trimester. [1]

Raman R et al., conducted a study of sono-elastographic evaluation of placenta, To establish a relationship between placental thickness, the average Pulsatility Index (PI) of the uterine artery, placental firmness, maternal weight during pregnancy, and newborn weight in

three groups: control subjects, individuals with gestational diabetes, and patients with pregnancy-induced hypertension. This study involved 222 pregnant women in the last trimester, who underwent obstetric USG between January 2017 and June 2018. The placental thickness ranged from 27 to 34 mm in the pregnancy-induced hypertension group (mean: 30.36 mm, standard deviation: 1.868), while it ranged from 33 to 51 mm in patients with gestational diabetes (mean: 40.75 mm, standard deviation: 4.181). The Pulsatility Index of uterine arteries was between 1.6 and 2.2 in pregnancy-induced hypertensive patients (mean: 1.824), whereas in gestational diabetes patients, it ranged from 0.6 to 1.1 (mean: 0.866). Placental stiffness was significantly higher in the pregnancy-induced hypertension group (mean: 7.233, standard deviation: 0.7025) compared to controls (mean: 2.906 kPa, standard deviation: 0.2923) and gestational diabetes patients (mean: 2.838 kPa, standard deviation: 0.3424). Infants born to mothers with pregnancy-induced hypertension had lower birth weights, while infants of gestational diabetes patients had higher birth weights. In conclusion, patients with gestational diabetes tend to have larger placentae and larger fetuses, with placental stiffness unaffected by diabetes. Conversely, pregnancy-induced hypertensive patients typically have smaller placentae and fetuses, with increased placental stiffness.[2]

Micheal Spiliopoulos et al., conducted a case control study, 47 singleton pregnancies in the second and third trimesters were enrolled, consisting 24 healthy pregnancies and 23 diagnosed preeclampsia. Placental stiffness was measured once during patient recruitment. Study found that placental elasticity was significantly higher in preeclamptic pregnancies compared to healthy ones in the third trimester (mean difference = 16.8; 95% CI [9.0, 24.5]; $P < 0.001$). No significant differences were observed in placental stiffness between the two groups in the second trimester or between severe preeclampsia and preeclampsia without severe features (mean difference = 9.86; 95% CI [−5.95, 25.7]; $P \geq 0.05$). Furthermore, the peripheral regions of the placenta were significantly stiffer than central regions in the

preeclamptic group (mean difference = 10.67; 95% CI [0.07, 21.27]; $P < 0.05$), whereas this difference was not observed in the control group (mean difference = 0.55; 95% CI [−5.25, 6.35]; $P > 0.05$). Placental stiffness did not correlate with gestational age, maternal age, gravidity, or parity, but it did show a significant correlation with BMI ($P < 0.05$). [64]

A study by Meena R et al., in 2022, New Delhi, to assess the diagnostic capability of placental shear wave elastography in early prediction of preeclampsia between the 16th and 20th weeks of gestation. A study involving 230 pregnant women utilized SWE (ElastPQ) to measure placental shear modulus. Participants were subsequently monitored for the development of preeclampsia and divided into two groups: group A (those who developed preeclampsia) and group B (normotensive individuals). Comparing the elasticity values of these groups, the study identified a statistically significant difference, with group A exhibiting higher placental shear modulus (4.61 kPa) compared to group B (2.51 kPa). The study determined a cut-off value of 2.9667 kPa as the most accurate in predicting preeclampsia, with an area under the curve of 0.970, sensitivity of 92%, specificity of 91.71%, positive predictive value of 57.5%, and negative predictive value of 98.9%. In conclusion, this study suggests that placental stiffness, quantitatively assessed through shear wave elastography at 16 to 20 weeks of gestation, is elevated in women who later develop preeclampsia, making it a potential predictor for this condition. [65]

MATERIALS & METHODS

MATERIALS AND METHODS

STUDY SITE:

This study was done in Department of Radio-diagnosis at R.L Jalappa Hospital and Research centre attached to SDUMC, Kolar.

STUDY POPULATION:

All pregnant women in third trimester referred for obstetric scan to Department of Radio-diagnosis at R.L Jalappa Hospital and Research center were regarded as study population.

STUDY DESIGN: Prospective case control study

SAMPLE SIZE: Canan Cimsit et al.[1] has reported the mean (SD) SWV to be 2.53 among normal pregnant women and 7.01 among Pre-eclampsia patients. Assuming alpha error of 5% (95% Confidence limit), Assuming a standard deviation of 5 units 12 units in each of the normal group and pre-eclampsia group respectively, Power of 80%.

The minimum required sample size to find the difference in mean SWV between normal pregnant women and Pre-eclampsia patients was **67 patients** in pre-eclampsia group and **67 subjects** in normal pregnant women group. The total sample size will be **134 subjects**.

Sample size (n) = $\frac{2S_p{}^2 [Z_{1-\frac{\alpha}{2}} + Z_{1-\beta}]^2}{\mu_d{}^2}$; $S_p{}^2 = \frac{S_1{}^2 + S_2{}^2}{2}$

Where, S_1 : Standard deviation in the normal pregnant women group

S_2 : Standard deviation in the pre-eclampsia group

μ_d : Mean difference between the samples

1-β: Power

α : Significance level

STUDY DURATION:

Between September 2022 – February 2024 data was collected for the study.

INCLUSION CRITERIA:

1. All normal pregnant women
2. All pre-eclampsia patients in third trimester referred to Department of Radio-diagnosis for obstetric scan

EXCLUSION CRITERIA:

1. Placental anomalies
2. Posterior placental location
3. Gross calcification of placenta
4. Twin pregnancy
5. Pre-existing medical conditions like chronic hypertension, chronic renal disease, uncontrolled diabetes mellitus, systemic lupus erythematosus (SLE), maternal infections.

ETHICAL CONSIDERATIONS:

Institution's human ethics committee approved this study. All participants were provided with written informed consent, and only those willing to sign the consent were allowed to take part in the study. Before getting consent, the participants were informed about risks and advantages of study as well as voluntary nature of participation. Privacy of study participants was protected at all times.

METHOD OF DATA COLLECTION:

Written Informed Consent was taken from all the individuals. Once a patient satisfies the inclusion criteria for this study, detailed history was taken from the patient referred to department of Radio-diagnosis. All patients were subjected to B-mode sonography and shear wave elastography in the supine position. Obstetric sonography and elastography of placenta was performed using Philips EPIQ5 system equipped with shear wave point quantification, ELASTPQ, using curvilinear broadband transducer C5-1MHz.Gestational age will be determined with bi-parietal diameter, head circumference, abdominal circumference and femur length.

Shear wave elastography is performed by using shear wave technique. The placental image will centre in the field of view. The fixed-size region of interest (ROI), a rectangle measuring 1 × 0.5 cm, will be placed at the centre and edge of the placenta, and the quantitative placental stiffness value was displayed over a B-mode sonogram. The ROI will be placed at homogeneous areas in the axial plane. The centre and edge of the vessel-free placenta away from the cord insertion will be selected as two sampling sites where fetal movements minimally affect the placenta. During acquisition, patients are asked to hold breaths at natural end-inspiratory phase. Five samples were taken from each site, from the centre (sample 1) and edge (sample 2) of the placenta, and will be averaged to obtain samples 1 and 2. In this method acoustic radiation force impulses are used to create shear waves that propagate through tissue. Tracking pulses are used to measure shear wave displacements between two points inside a region of interest (ROI), giving the speed of the shear wave at a certain point, hence the name point shear wave elastography. This quantification of the shear wave speed is given in meters per second and, depending on user preference, can be automatically converted to kilopascals by approximating the Young's modulus.

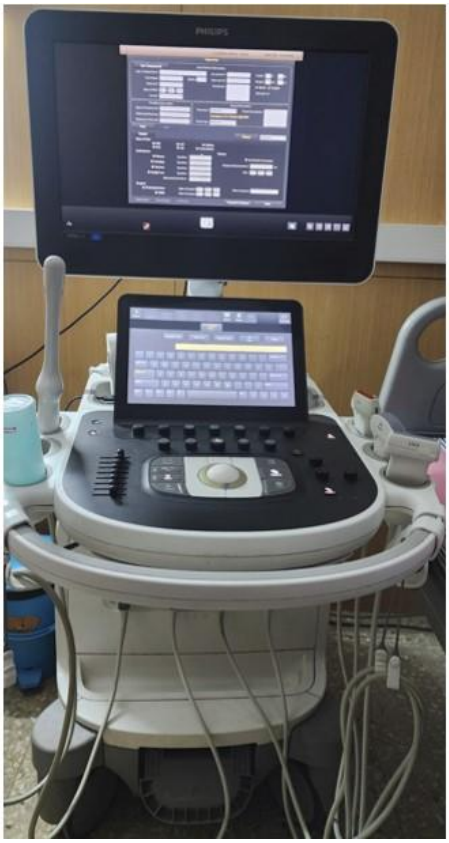

Figure 16: Ultrasound scanner Philips EPIQ5.

Figure 17: C1 - 5 MHz convex transducer (equipped with shear wave point quantification, ELASTPQ)

Data Analysis

Data was entered using Microsoft Excel and analyzed using the Statistical Package for Social Science (SPSS) standard version 20.

All socio-demographic and clinical characteristics of the patient was summarized using Mean (SD) for continuous variables and proportions (%) for categorical variables.

Comparison of continuous variable (age, Placental elasticity value, BMI, gestational age, birth weight) across the two groups (normal pregnancy vs Pre-eclampsia) was performed using the student's t test.

Comparison of categorical variables (smoking, obstetric index, previous history) across study groups (normal pregnancy vs Pre-eclampsia) will be done using Chi square test.

P-value of <0.05 will be considered statistically significant.

Statistical Analysis

The current study encompassed both qualitative and quantitative variables. Qualitative variables were expressed as number (%) while quantitative variables were represented by Median (Interquartile Range). The normality of the data was assessed using the Kolmogorov-Smirnov test. Non-parametric Mann-Whitney U tests were employed to compare the two quantitative variables. The Chi-square test and Fisher's exact test were utilized to determine the association between two independent qualitative variables. ROC analysis was employed to determine the cut-off value and assess the accuracy of predicting pre-eclampsia in third trimester of pregnancy.

A confidence level of 95% was considered for all statistical tests. Data analysis was conducted using SPSS 20 and R Studio statistical software.

RESULTS

RESULTS

Total of 134 subjects were included in final analysis. Control group (N=67) and case group (N = 67)

Table 2: Distribution of median age and gestational age (GA) between case (N=67) and control (N=67)

Group		N	Median	(IQR)	P- value
AGE	Control	67	25	(22-28)	0.90
	Case	67	24	(22-29)	
GA	Control	67	34.00	30-35	0.395
	Case	67	34.00	31-36	

Table 2: Distribution of median age and gestational age (GA) between case (N=67) and control (N=67)

*Mann Whitney U test applied

Table 2: Distribution of median age and gestational age (GA) between the case and control groups. No significant differences were observed in median age and GA between the case and control groups

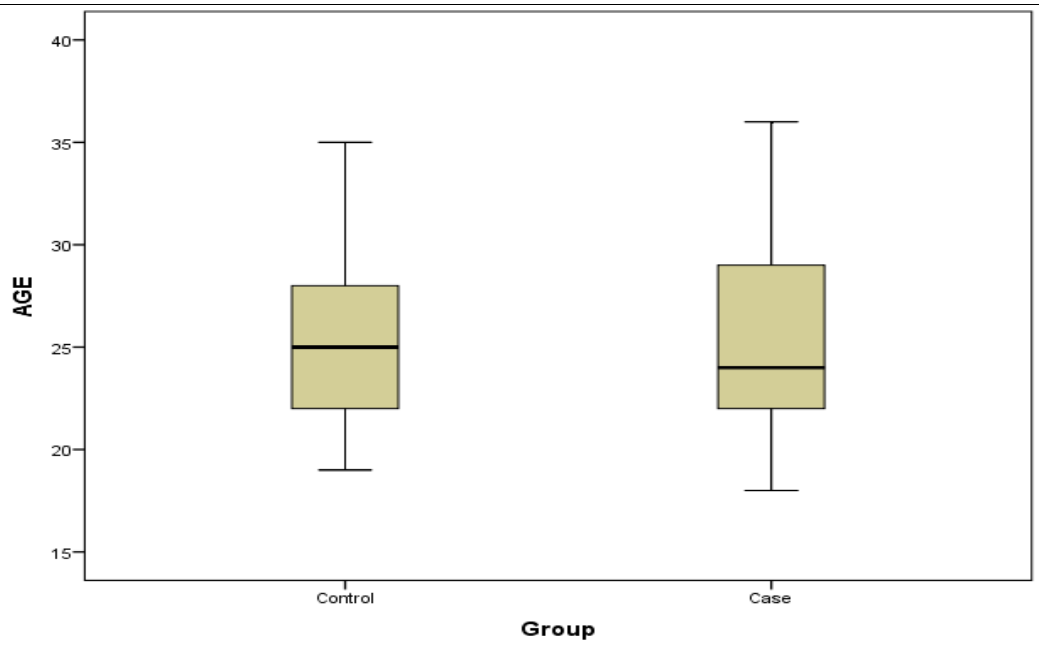

Figure 18: Box plot graph showing the comparison of median age between control (N=67) and case (N=67)

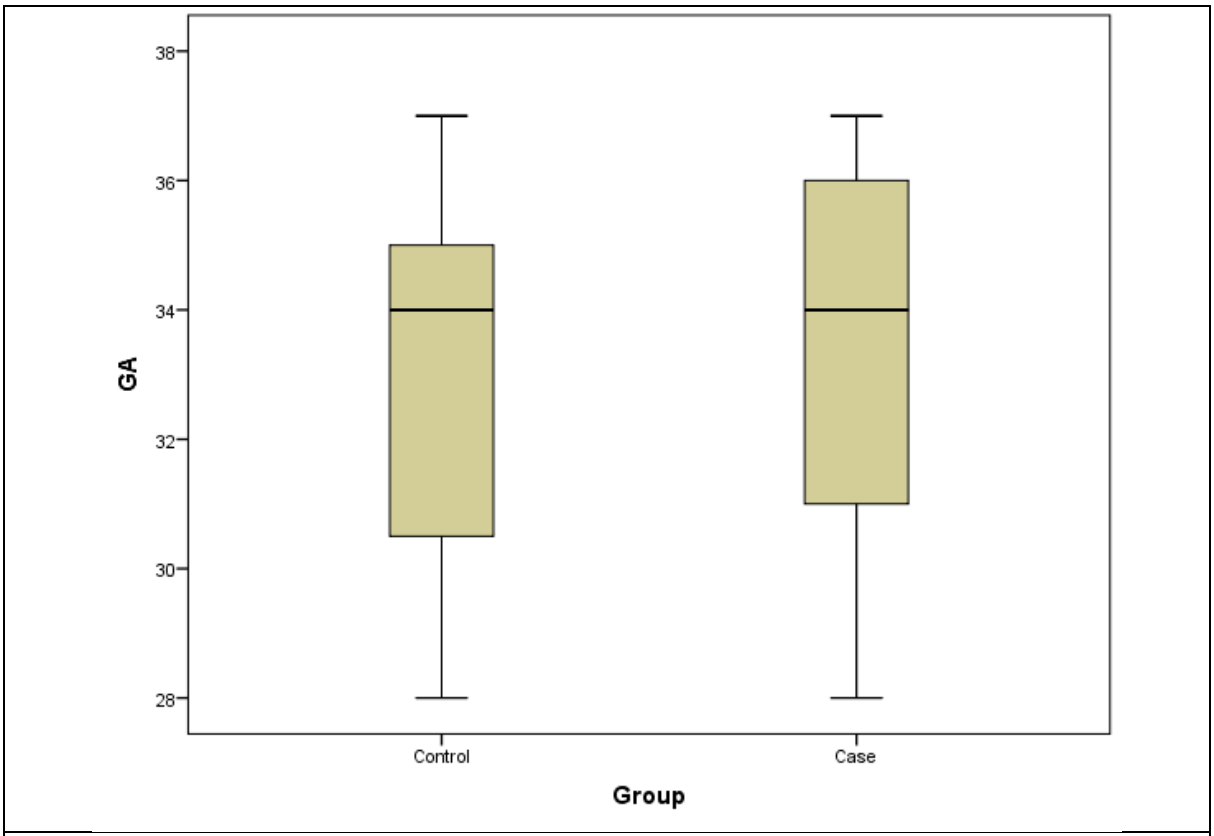

Figure 19: Box plot graph showing the comparison of gestational age (GA) between control (N=67) and case (N=67)

Age Group	Case	Control	Total
18-22	19	18	37
	28.4%	26.9%	27.6%
22-27	27	26	53
	40.3%	38.8%	39.6%
>27	21	23	44
	31.3%	34.3%	32.8%
Total	67	67	134
	100.0%	100.0%	100.0%

Table 3: Group-wise distribution of age between case (N=67) and control (N=67)

P >0.05 (Chi square test)

Table 3: Shows the details of age group classification according to case and control. Out of the total cases highest patients were from the age group 22 – 27 years similarly in the control also the highest patients were observed in the same age group. Age group was not statistically significant with case and control (P >0.05)

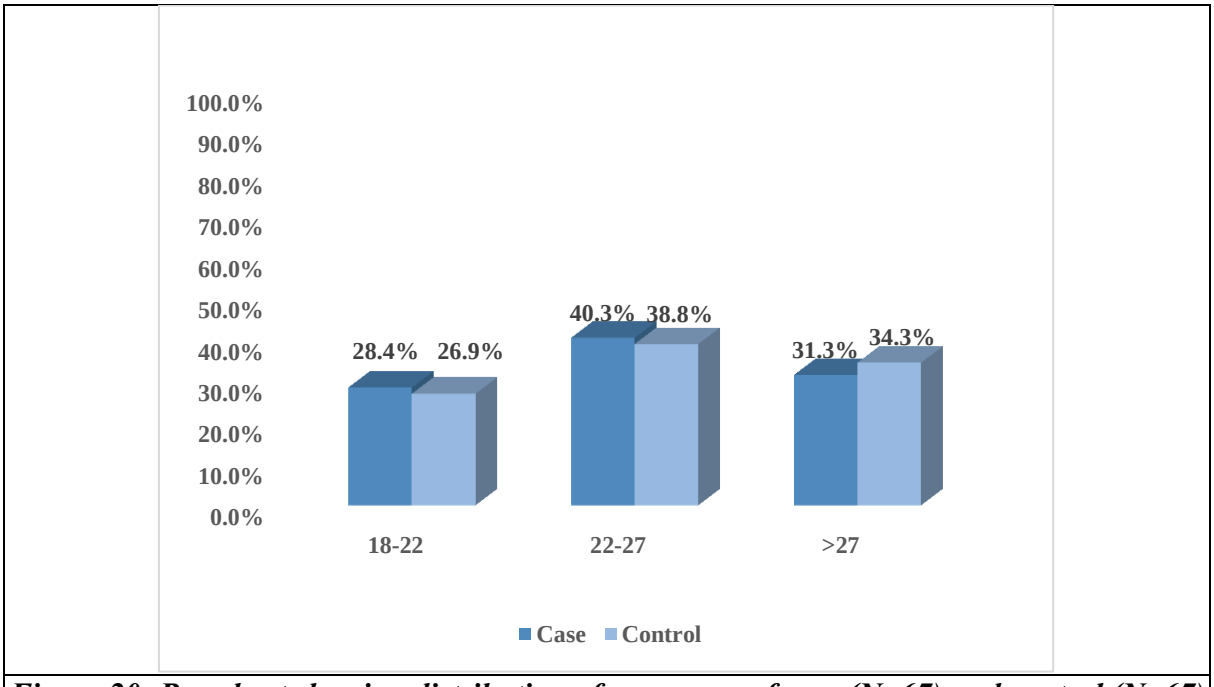

Figure 20: Bar chart showing distribution of age group of case (N=67) and control (N=67)

Group		N	Median	(IQR)	P- value
E1(Centre of placenta)	Control	67	3.02	2.70-3.50	P<0.001
	Case	67	10.88	9.75-13	

Table 4: Comparison of SWE values at centre of placenta (E1) between case (N=67) and control (N=67)

Table 4: Illustrates the Comparison of SWE values at centre of placenta (E1) between the control and case groups. The median of centre of placenta (E1) was higher in the case group at 10.88 (9.75-13.0) compared to the median of the control group at 3.02 (2.70-3.50) with statistical significance (P<0.001).

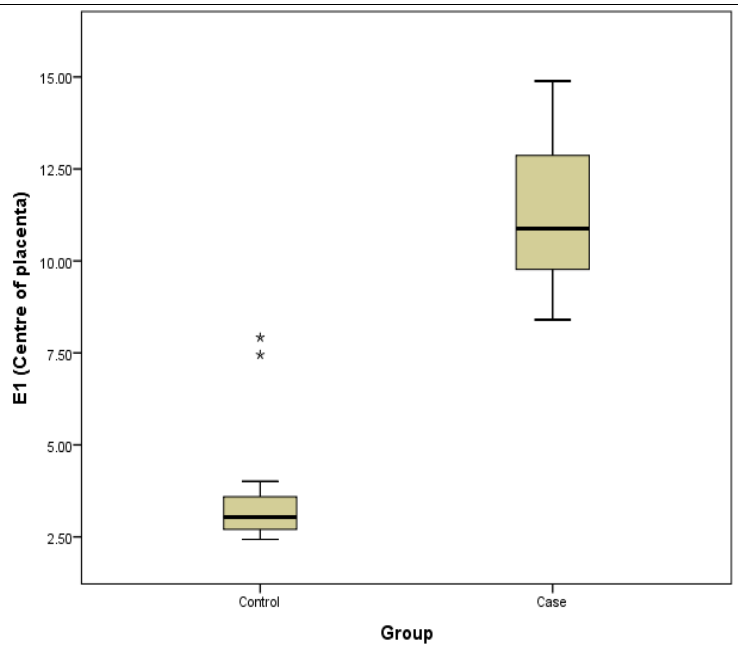

Figure 21: Box plot graph showing the comparison of SWE values at centre of placenta (E1) between case (N=67) and control (N=67)

Group		N	Median	(IQR)	P- value
E2 (edge of placenta)	Control	67	3.04	2.84-3.66	P<0.001
	Case	67	11.07	9.74-13.03	

Table 5: Comparison of SWE values at edge of placenta (E2) between case (N=67) and control (N=67)

Table 5: Illustrates the comparison of SWE values at edge of placenta (E2) between the control and case groups. The median of edge of placenta (E1) was higher in the case group at 11.07 (9.74-13.03) compared to the median of the control group at 3.04 (2.84-3.66), with statistical significance (P<0.001).

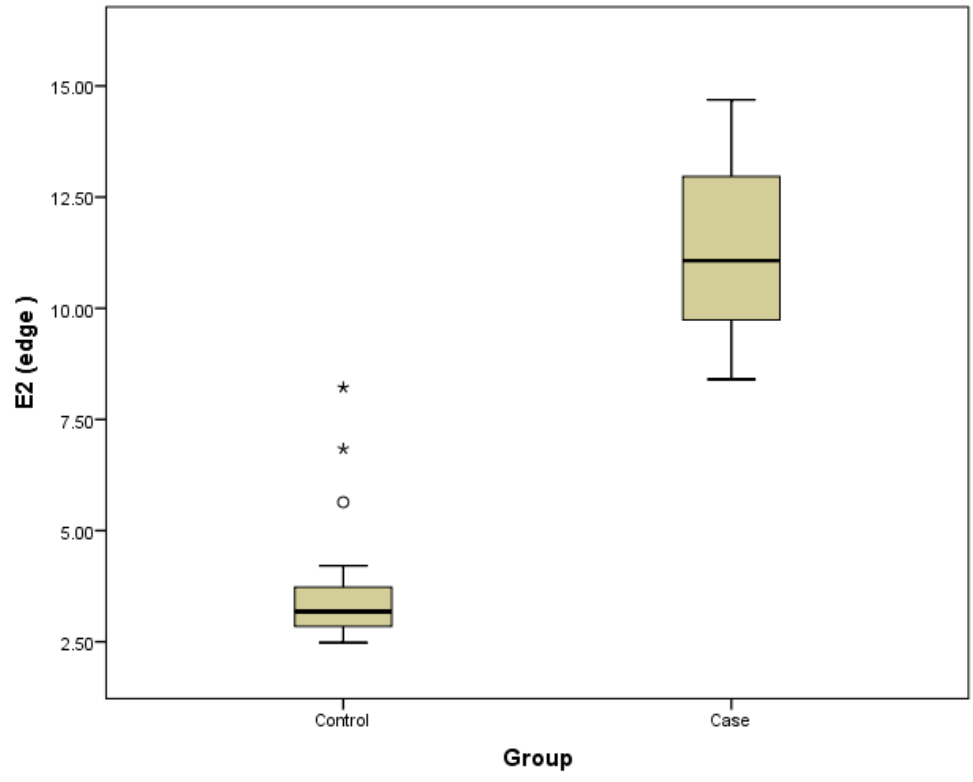

Figure 22: Box plot graph showing the comparison of SWE values at edge of placenta (E1) between case (N=67) and control (N=67)

Group		N	Median	(IQR)	P- value
SWE	Case	67	11.07	9.74-13.03	P<0.001
	Control	67	2.90	2.78-3.58	

Table 6: Median SWE values between case and control

Table 6: Illustrates the median of SWE was higher in the case group at 11.07 (9.74-13.03) compared to the median of the control group at 2.90 (2.78-3.58) with statistical significance (P<0.001).

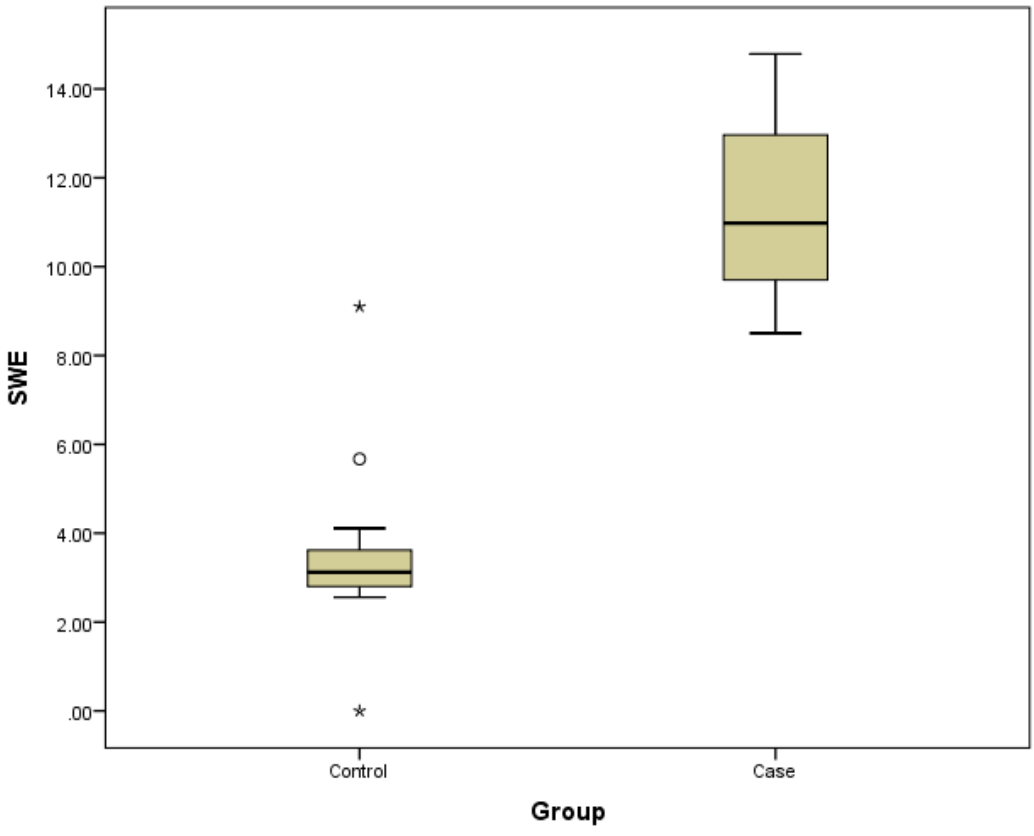

Figure 23: Box plot graph showing the comparison of average SWE values between case and control

	Groups				Total
	Control		Case		
	N	%	N	%	
Primigravida	35	52.2%	36	53.7%	71
Multigravida	32	47.8%	31	46.3%	63
Total	67	100.0%	67	100.0%	134

Table 7: Distribution of parity between case (N=67) and control (N=67)

*Chi square test applied (P value -0.4313)

Table 7: Presents the distribution of parity between the case (N=67) and control groups **(N=67)**. Among the total controls, 52.2% were primigravida, while in the case group, 53.7% were primigravida. No statistical significance was observed in parity between the case and control groups. (P value -0.4313).

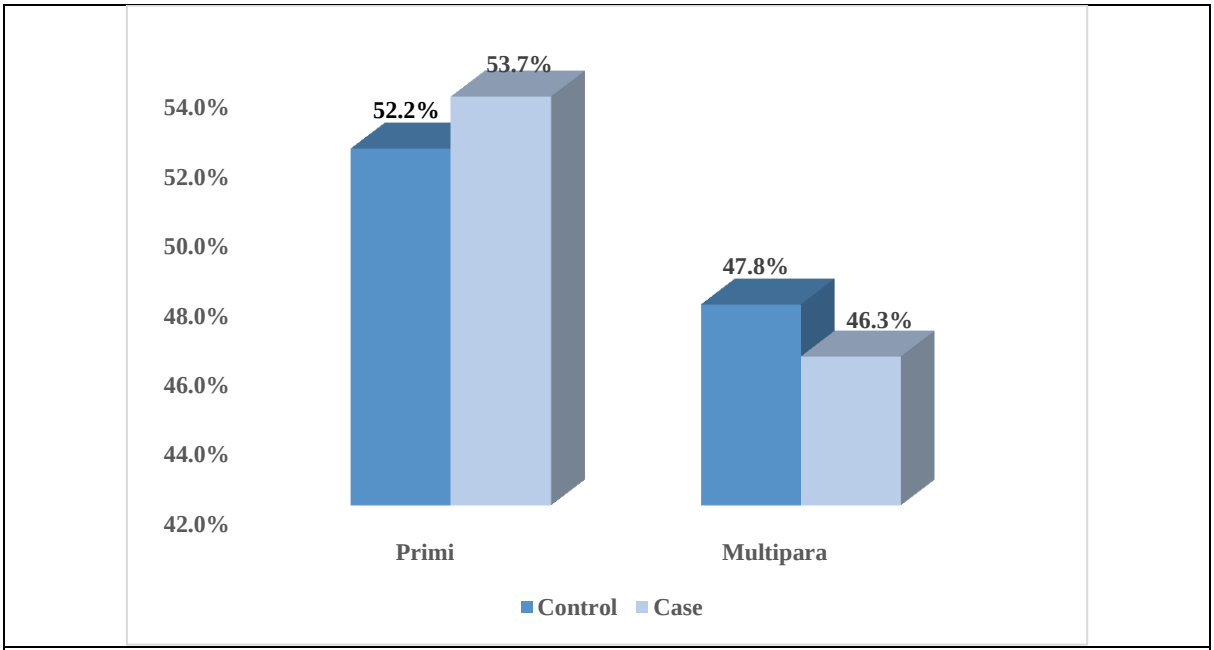

Figure 24: Bar chart showing distribution parity between case (N=67) and control (N=67)

Location of placenta	Group				Total
	Control		Case		
	N	%	N	%	
Anterior	29	43.3%	33	49.3%	62
Anterior left lateral	3	4.5%	3	4.5%	6
Anterior right lateral	7	10.4%	5	7.5%	12
Fundal	7	10.4%	6	9.0%	13
Fundal anterior	14	20.9%	12	17.9%	26
Fundal left lateral	2	3.0%	4	6.0%	6

Table 8: Distribution of placental location in the case (N=67) and control groups (N=67)

*Fisher exact test applied (P- value 0.9275)

Table 8: Presents distribution of placenta location among the case and control groups. In both cases and control groups, the highest number of placentas were located anteriorly. There was no significant association observed between placenta location and case/control status.(P-value 0.9275)

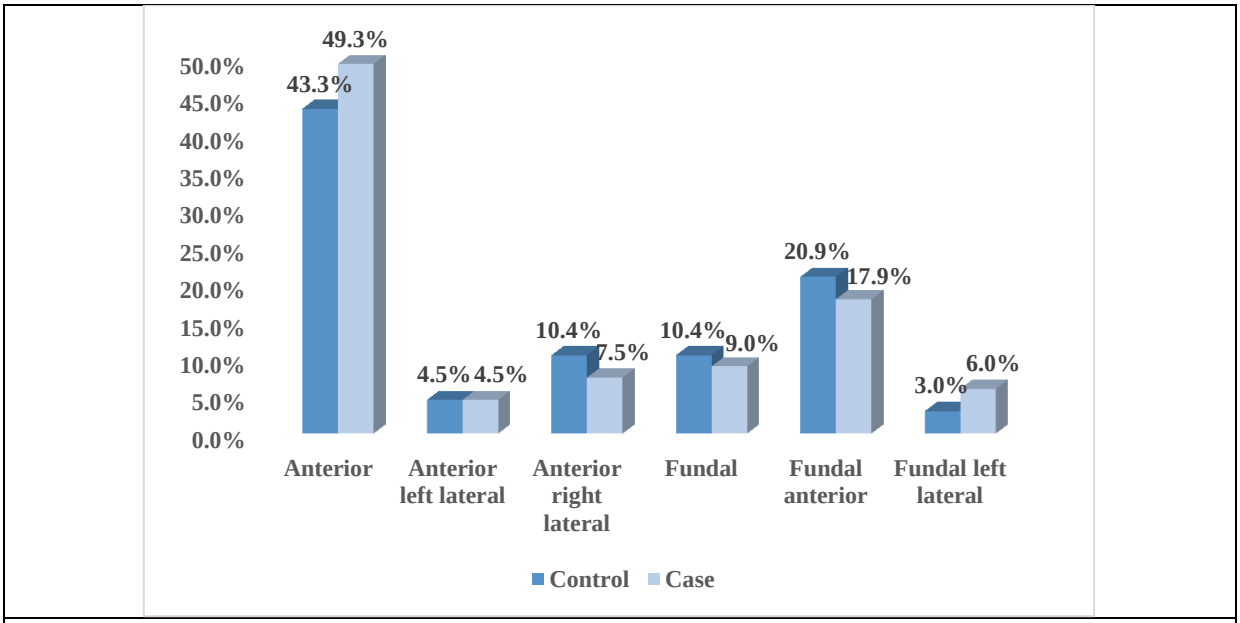

Figure 25: Bar chart showing distribution of placental location in the case and control groups

Placenta Grading	Group				Total
	Control		Case		
	N	%	N	%	
I	8	11.9%	10	14.9%	18
II	56	83.6%	54	80.5%	110
III	3	4.5%	3	4.5%	6
	67	100.0%	67	100.0%	134

Table 9: Distribution of placenta grading in the case (N=67) and control groups (N=67)

*Fisher exact test applied (P- value 0.2515)

Table 9: Displays the distribution of placenta grading in the case and control groups. The majority of patients in both cases (80.5 %) and controls (83.6%) were observed to have grade II placentas. No significant association was found between placental grading and case/control status (P-value 0.2515).

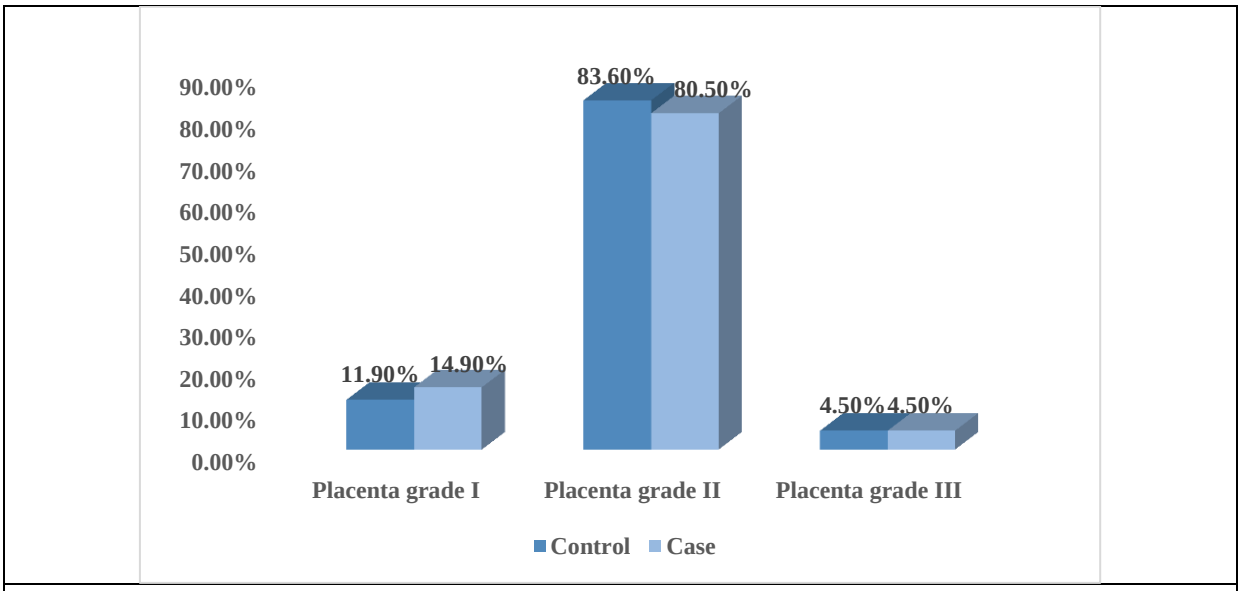

Figure 26: Bar chart showing distribution of placental grading in the case (N=67) and control groups (N=67)

Out Come	Group				Total
	Control		Case		
	N	%	N	%	
Appropriate for gestational age (AGA)	67	100.0%	44	65.7%	111
Small for gestational age (SGA)	0	0.0%	21	31.3%	21
Stillbirth	0	0.0%	2	3.0%	2

Table 10: Details of fetal outcomes between the case and control groups

Fisher Exact test applied (p-value = 0.000000024)

Table 10: presents the details of fetal outcomes within the case and control groups. In the control group, no patients experienced SAG (Small for Gestational Age) or stillbirths. Conversely, in the case group 31.3% and 3% were observed with SAG and stillbirths respectively. SAG and stillbirth were significantly associated with cases.

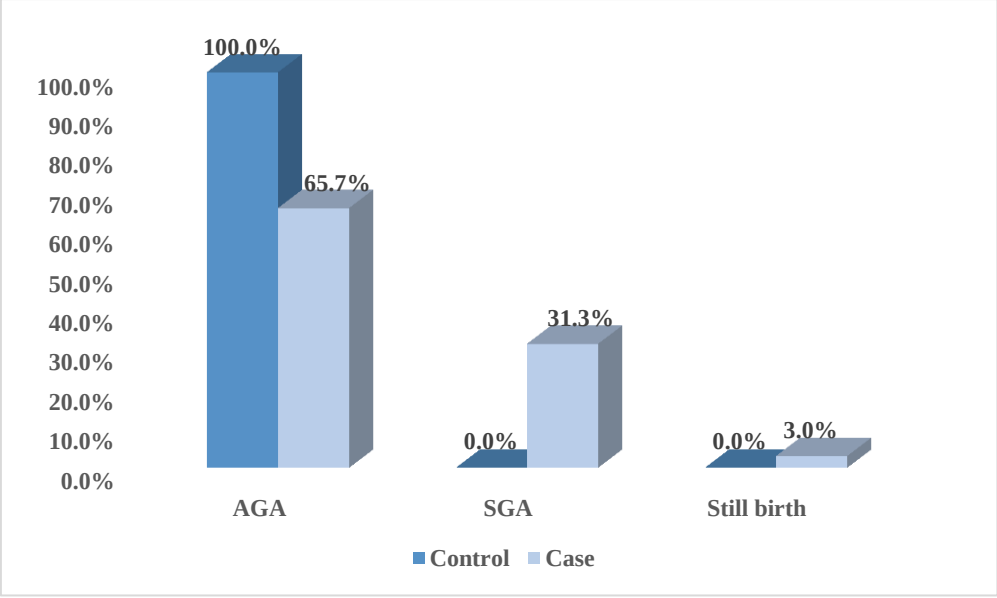

Figure 27: Bar chart showing distribution of fetal outcomes between the case and control groups

Test Result Variable(s)	Area under the ROC curve (AUC)	P-Value	Asymptotic 95% Confidence Interval		Cut-off Value	Sensitivity	Specificity
			Lower Bound	Upper Bound			
SWE	0.958	P<0.001	0.913	1.000	4.87	98.5%	92.5%

Table 11: Assess the accuracy of SWE for predicting Pre-eclamptic pregnancies in third trimester and its cut-off value

Table 10: Presents the details of the cut-off values for the SWE to predict the case. ROC analysis was conducted on the SWE to determine the most appropriate cut-offs for predicting pre-eclampsia in the third trimester. Optimal results were observed with a SWE cut-off value of ≥ 4.87 for predicting pre-eclampsia pregnancies in the third trimester.

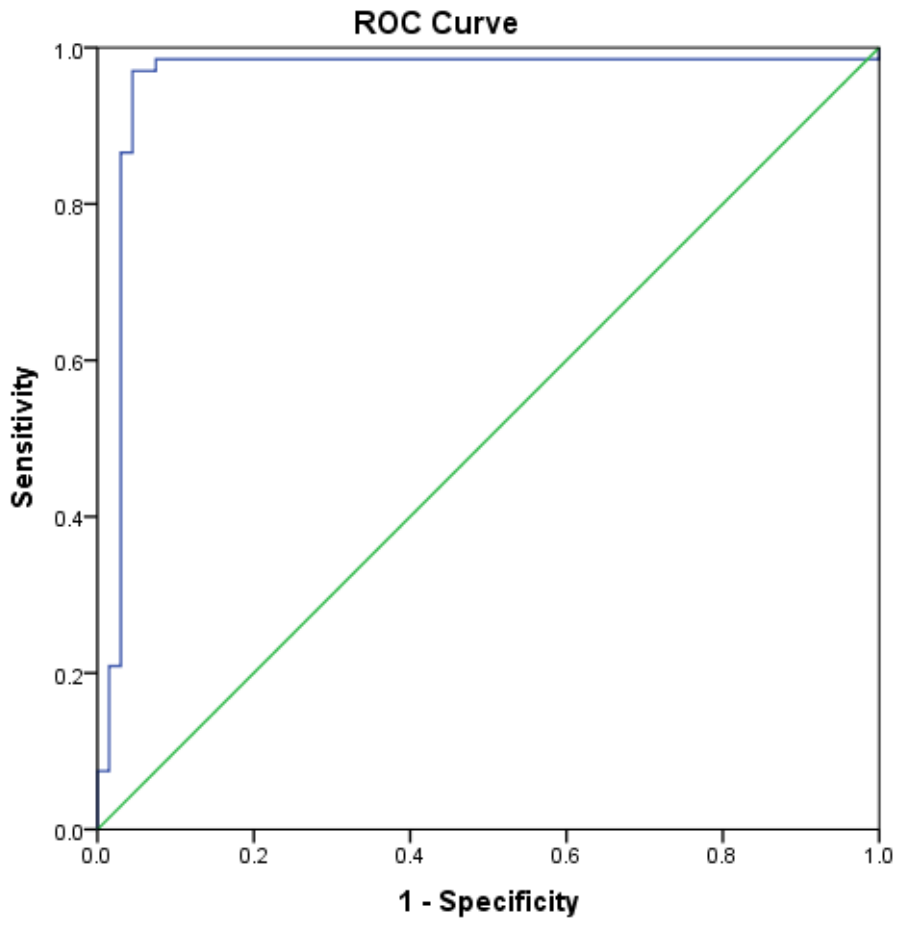

Figure 28: ROC curve constructed to establish the cut-off values for shear wave elastography (SWE) to predict pre-eclampsia

IMAGES

CASE :1. Ultrasound grey scale images from a 21 year old primigravida at 31 w 2 d gestation.

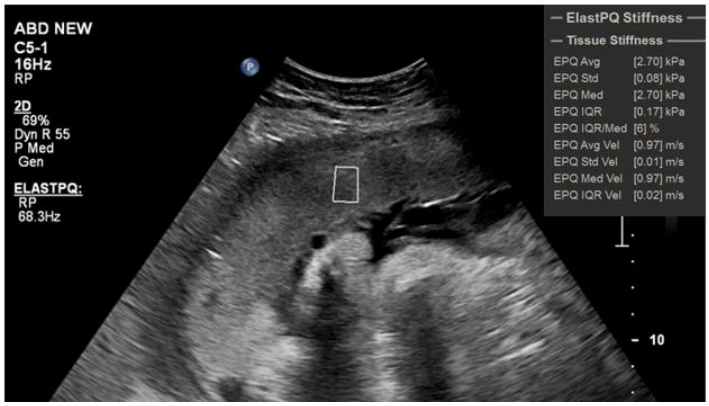

Figure 29a

Figure 29b

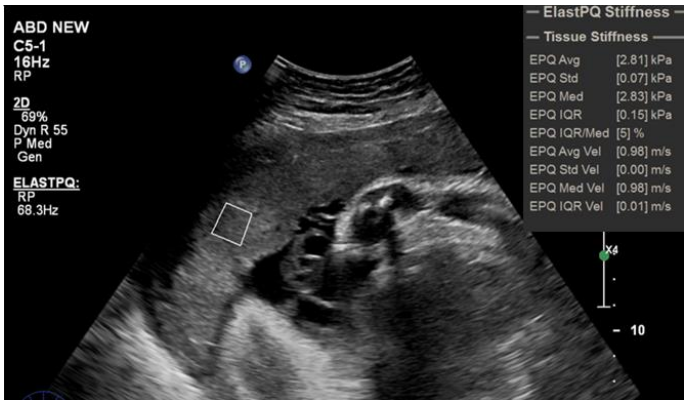

Figure 29c

Figure 29d

Figure 29: (a) ROI was placed at centre of fundal anterior placenta (b) 5 samples were taken from centre of placenta of placenta in the same patient.The mean placental elasticity value was 2.70 kPa & median value was 2.70 kPa (c) ROI was placed at edge of fundal anterior placenta (d) 5 samples were taken from edge of placenta in the same patient. The mean placental elasticity value was 2.81 kPa & median value was 2.83 kPa. **Average SWE is 2.75 kPa (Normal).**

CASE :2. Ultrasound grey scale images from a 28 year old primigravida at 34 W 2 D gestation

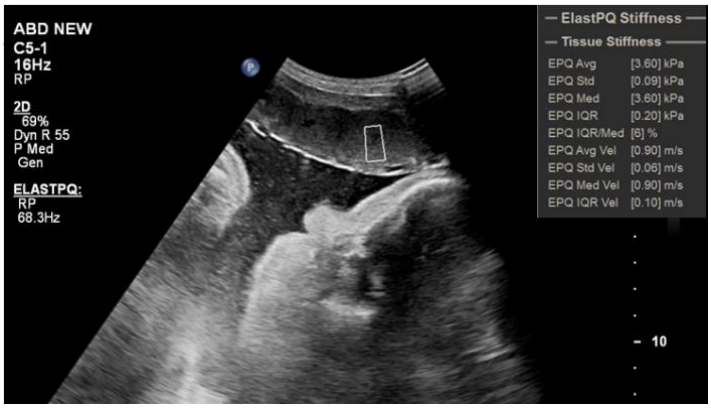

Figure 30a

Figure 30b

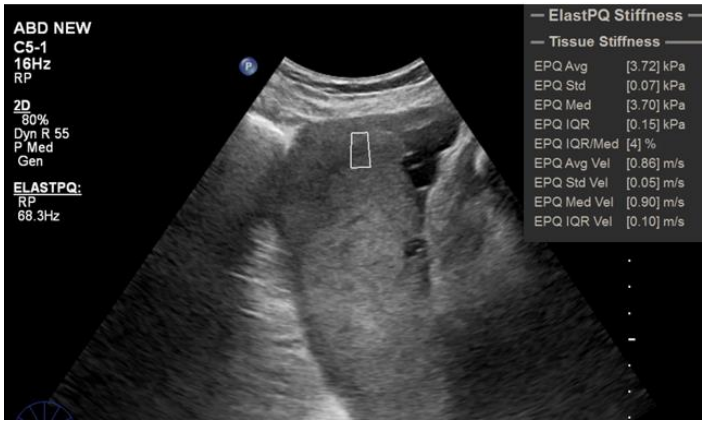

Figure 30c

Figure 30d

Figure 30. (a) ROI was placed at centre of fundal placenta.(b) 5 samples were taken from centre of placenta of placenta in the same patient.The mean placental elasticity value was 3.60 kPa & median value was 3.60 kPa.(c) ROI was placed at edge of fundal placenta (d) 5 samples were taken from edge of placenta in the same patient. The mean placental elasticity value was 3.72 kPa & median value was 3.70 kPa. **Average SWE value is 3.66 kPa (Normal).**

CASE 3: Ultrasound grey scale images from a 32 year old primigravida at 32 W 2 D Gestation with Pre-eclampsia.

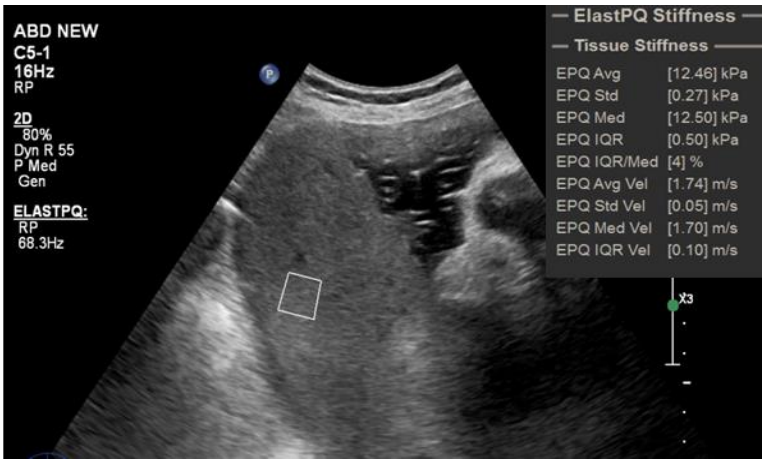

Figure 31a

Figure 31b

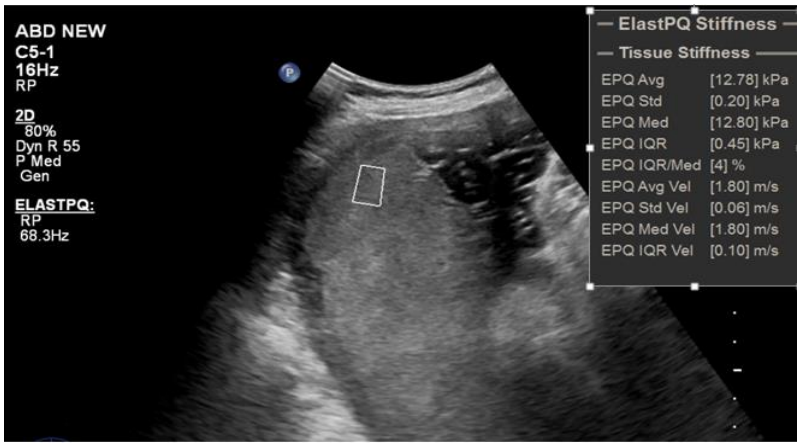

Figure 31c

Figure 31d

Figure 31. (a) ROI was placed at centre of fundal placenta (b) 5 samples were taken from centre of placenta of placenta in the same patient.The mean placental elasticity value was 12.46 kPa & median value was 12.50 kPa.(c) ROI was placed at edge of fundal placenta (d) 5 samples were taken from edge of placenta in the same patient. The mean placental elasticity value was 12.78 kPa & median value was 12.80 kPa. **Average SWE value is 12.62 kPa (Increased) .**

CASE 4: Ultrasound grey scale images from a 35 year old multipara at 35 W Gestation with Pre-eclampsia.

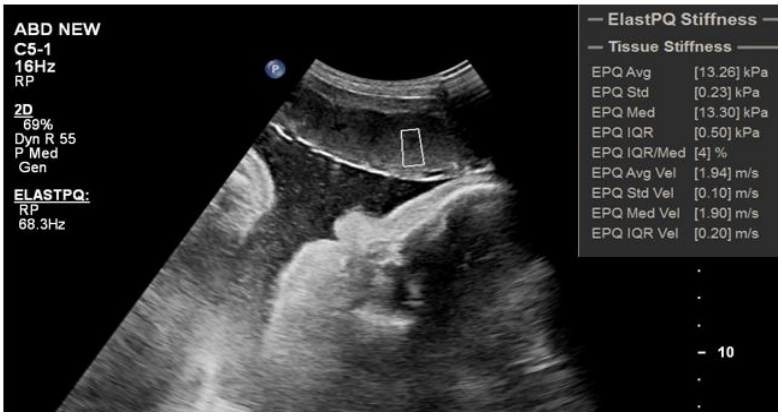

Figure 32a

Figure 32b

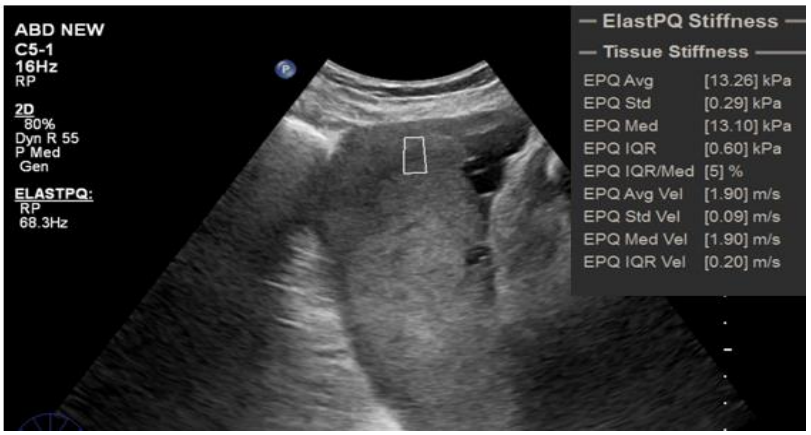

Figure 32c

Figure 32d

Figure 32. (a) ROI was placed at centre of fundal right lateral placenta (b) 5 samples were taken from centre of placenta of placenta in the same patient.The mean placental elasticity value was 13.26 kPa & median value was 13.30 kPa.(c) ROI was placed at edge of fundal right lateral placenta (d) 5 samples were taken from edge of placenta in the same patient. The mean placental elasticity value was 13.26 kPa & median value was 13.10 kPa. **Average SWE value is 13.26 kPa (Increased).**

DISCUSSION

DISCUSSION

Screening for preeclampsia based on maternal characteristics and relevant medical history identifies only 35% of patients with PE. [66] Various maternal serum biochemical indices have been used to predict preeclampsia; however, the predictive value of these indices is low.[67] Initial studies on placental elastography measured the elastic modulus of the human placenta during late pregnancy and found that it was an independent assessment parameter. [68] Currently, shear wave elastography (SWE) is widely used to quantitatively evaluate placental stiffness and has been employed as an adjunct diagnostic tool for various perinatal diseases.[69-71]

Additionally, the stiffness of the placenta is regarded as a potential biomarker for placenta-mediated disease detection.[72] In recent years, there have been an increasing number of studies examining the usage of ultrasonic elastography in diagnosing PE. The aim of study was to provide a comprehensive evaluation of the diagnostic performance of ultrasonic elastography in PE.

Out of the total cases in the present study, majority of the patients were from the age group 22 – 27 years. Similarly, in the controls majority of the patients were observed in the same age group. Age group was not statistically significant with case and control (P >0.05). Similar finding was found in the study conducted by Meena, R et al **[65]** on 230 participants between 16 and 20 weeks. The age of the participants ranged from 16 to 35 years. The average age of participants in group B (controls) was 24.89 years, and in group A (cases) was 23.92 years.

In the current study among the subjects (N=134), 71 (52.9%) participants were primigravida, and 63 (47.05 %) were multigravida. In 53.7 % cases with preeclampsia were primigravida in the current study. No statistical significance was observed in parity between the case and control groups. In the study by Vikas Singh et al **[75] ,** among the total controls, 52.2% were primigravida, while in the case group, 53.7% were primigravida. No statistical

significance was observed in parity between the case and control groups (P value -0.4313) which similar to current study.

In the present study, among both cases and controls, the highest number of placentas were located anteriorly. There was no significant association observed between placenta location and case/control status. (P- value 0.9275) The majority of patients in both cases (85.1%) and controls (83.6%) were observed to have grade II placentas. No significant association was found between placental grading and case/control status (P-value 0.2515).

In our study, the median of SWE was higher in the case group at 10.98 (9.70-13.13) compared to the median of the control group at 2.90 (2.78-3.58), with statistical significance (P<0.001). The median of E1 (center of placenta) was higher in the case group at 10.88 (9.75-13.0) compared to the median of the control group at 3.02 (2.70-3.50), with statistical significance (P<0.001).Median of E2 (edge of placenta) was higher in the case group at 11.07 (9.74-13.03) compared to the median of the control group at 3.04 (2.84-3.66), with statistical significance (P<0.001). In the **Meena, R.et al** [65] study, the SWE value of the placenta was calculated from the average of measurements obtained from at least three places: E1 – Right peripheral part of placenta, E2 – Center of placenta and E3 – Left peripheral part of placenta. The average shear modulus value was 2.74 kPa. The average value of placental shear modulus in group B (controls) was 2.51 kPa and in group A (cases) it was 4.61 kPa (p value <0.0001). The average SWE value of the women included in the **Vikas Singh et al** [75] study during the initial screening was (10.06 ± 15.06) at the center of placenta and (10.49 ± 15.62) at the placental edge. The average elasticity values in both the central (27.98 ± 16.12 vs. 4.57 ± 6.57 kPa) and peripheral areas of placenta (29.14 ± 16.12 vs. 4.80 ± 7.70 kPa) were significantly elevated as compared to normal pregnancies. The findings in studies conducted by Meena, R.et al and Vikas Singh et al were similar to current study.

In the present study, in the control group, no patients experienced SAG (Small for Gestational Age) or stillbirth. Conversely, in the case group, 31.3% and 3% were observed with SAG and stillbirths respectively. SAG and stillbirth were significantly associated with cases. In the study by Cimsit et al [1] **,** out of 28 cases, 64.2% (18 of 28) were small for gestational age, 3.5% (1 of 28) were stillborn, and 32.1% (9 of 28) were appropriate for gestational age. The findings in studies conducted by Cimsit et al [1] were similar to present study.

In the present study, Sensitivity of SWE was 98.5% and Specificity was 92.5% ROC analysis was conducted on the SWE to determine the most appropriate cut-offs for predicting pre-eclampsia in the third trimester. Optimal results were observed with a SWE cut-off value of ≥4.87 for predicting preeclampsia pregnancies in the third trimester. Similar studies were conducted and diagnostic performance of the studies are described below in table 12.

Author	Technique Value	Cutoff Value	AUROC	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
Spiliopoulos M et al.[64]	mean SWE value	16 kPa	0.82	75	83	82	76
Alan B et al.[13]	mean SWV value	1.5 m/s	0.99	91	5	50	33
Sirinoglu HA et al.[62]	mean SWE value	7 kPa	0.82	89	79	81	88
Fujita Y et al.[59]	mean SWV value	1.2 m/s	0.91	92	91	40	99
Meena R et al.[65]	mean SWE value	3 kPa	0.97	92	92	58	99
Kılıç F et al.[73]	median SWE value	7 kPa	0.90	90	86	82	92
Hefeda MM et al.[74]	mean SWV value	1.4 m/s	0.91	91	86	73	75

Table 12: The summarized results of comparable studies show the Diagnostic Performance of SWE.

SWE, shear wave elastography; SWV, shear wave velocity; AUROC, area under the receiver operating characteristic curve; PPV, positive predictive value; NPV, negative predictive value.

In the present study, controls (N=67) and cases (N=67) underwent sonoelastography scan in third trimester. There was no significant differences were found in elasticity values between the centre and edge of the placenta. Similar study by Cimcit C et al. [1] conducted a study involving 204 singleton pregnancies undergoing routine anomaly scanning between 20 and 23 weeks gestation. No significant differences were found in elasticity values between the centre and edge of the placenta.

Author	Gestational Weeks	Technique	Representative Values	PE Group			Control Group		
				n	PSM	Range	*n*	PSM	Range
Spiliopoulos M. [64]	>20	2D-SWE	Mean	23	22 ± 3 kPa	NA	24	11 ± 2 kPa	NA
Alan B. [13]	27–35	P-SWE	Mean	42	1.4 m/s	1.3–1.5	44	1.1 m/s	1.00–1.1
Sirinoglu HA. [62]	23–37	2D-SWE	Mean	9	6 ± 2 kPa	2–14	75	9 ± 3 kPa	3–12
Fujita Y. [59]	16–32	P-SWE	Median	13	1.4 m/s	1.1–2.4	208	NA	NA
Meena R. [65]	16–20	2D-SWE	Mean	25	5 kPa	NA	205	3 kPa	NA
Kılıç F. [73]	23–37	2D-SWE	Median	23	21 kPa	2–71	27	4 kPa	2–14
Hefeda MM. [74]	>18	P-SWE	Mean	9	2.1 ± 1.5 m/s	NA	46	0.9 ± 0.4 m/s	NA
		P-SWE	Mean	46	2.2 ± 1.5 m/s	NA	94	0.9 ± 0.6 m/s	NA

Table 13: Placental Stiffness in Preeclampsia and Control Groups of Similar Studies

PE, preeclampsia; PSM, placental stiffness measurement; NA, not available; *n*, number

SUMMARY

SUMMARY

This prospective case control study was carried out to evaluate shear wave elastography (SWE) values in relation to alterations in placental elasticity in both PE and normal pregnancies, with the goal of determining its effectiveness as a diagnostic tool for assessing the disease. Total of 134 pregnant ladies in third trimester were assessed. Out of which 67 subjects in control group and 67 subjects in case group.

- In the present study, majority of the study group belonged to the age group 22 – 27 years and gestational age was between 30- 35 weeks. No significant differences were observed in age and GA between the case and control groups.
- In the present study, majority of subjects were primigravida (53 %). No statistical significance was observed in parity between the case and control groups.(P value - 0.4313).
- There was no significant association observed between placenta location and case/control status (P- value 0.9275).
- In my current study, in control group fetal outcome in all subjects were appropriate AGA whereas, in cases group 65.7% were AGA , 31.3% were SGA and 3.0% were still births. SAG and stillbirth were significantly associated with cases (p-value = 0.000000024).
- In my current study, the median SWE values at centre of placenta (E1) was higher in the case group at 10.88 (9.75-13.0) compared to the median SWE values of the control group at 3.02 (2.70-3.50) with statistical significance (P<0.001).
- In the present study, the median SWE values at the edge of placenta (E2) was higher in the case group at 11.07 (9.74-13.03) compared to the median SWE values of the control group at 3.04 (2.84-3.66) with statistical significance (P<0.001).

- In this study, the median SWE values was higher in the case group at 10.98 (9.70-13.13) compared to the median SWE values of the control group at 2.90 (2.78-3.58), with statistical significance (P<0.001).
- Optimal results were observed with a SWE cut-off value of ≥4.87 for predicting pre-eclampsia pregnancies in the third trimester.
- By ROC analysis, Sensitivity of SWE was 98.5% and Specificity was 92.5% .

Thus, the study demonstrates statistically significant differences between patients with preeclampsia and those with normal pregnancies. The placental stiffness can be used as an additional prognostic parameter in the outcome of hypertension in pregnancy. Thus, it determines the effectiveness of SWE as a diagnostic tool for assessing the disease and placental function.

CONCLUSION

CONCLUSION

This study demonstrates that the use of ultrasound elastography for detecting placental stiffness has a good diagnostic performance for detecting Pre-Eclampsia. Shear wave elastography is a novel technique for characterizing tissues that is helpful for assessing tissue characterisation, placental function and serves as an addition to existing methods in prediction preeclampsia.

LIMITATIONS

Our study included a few additional drawbacks. First, because the maximum entry depth was 8 cm, women whose placentas lay posteriorly were not included to participate in the study. This factor can be interpreted as a technical limitation for the use of shear wave elastography in generalized screening. The lack of a histological evaluation of the placentas is another research drawback. Histopathological analysis could reveal a connection between elastography results and structural alterations in the pathology. Although the standardization of the elastography technique was satisfactory, we did not evaluate interobserver variability because we aimed to minimize repeated examinations of the same fetus. Due to the fixed dimensions of the SWE sample box, information about a small area only could be obtained for the elastographic values.

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ANNEXURE

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND

RESEARCH, TAMAKA, KOLAR, KARNATAKA

ANNEXURE I - PATIENT PROFORMA

STUDY TITLE: "ROLE OF SHEAR WAVE ELASTOGRAPHY OF PLACENTA IN NORMAL AND PRE-ECLAMPTIC PREGNANCIES IN THIRD TRIMESTER."

Date:

Time:

DEMOGRAPHIC DETAILS

1. Name:
2. Age:
3. UHID No / IP No:

Consent taken: Yes / No

OBSTETRIC HISTORY

1. Obstetric score:
2. Last menstrual period (LMP):
3. Expected date of delivery (EDD):
4. Presenting complaints:
5. Previous obstetric history:
6. Clinical examination:

ULTRASONOGRAPHIC FINDINGS

1. Fetal heart rate (FHR):
2. Estimated fetal weight (EFW):
3. Liquor:

4. Amniotic fluid index (AFI):
5. Gestational age by ultrasound:

CONVENTIONAL B- MODE ULTRASOUND FEATURES

1. Placenta location:
2. Placenta grading:

SHEAR WAVE ELASTOGRAPHY

Elastography values (kPa) at the center of placenta (Sample 1): / / / /

Elastography values (kPa) at the edge of placenta (Sample 2): / / / /

Average kPa values at the center of placenta (Sample 1):

Average kPa values at the edge of placenta (Sample 2):

Average elastography reading (Sample 1 & 2):

FETAL OUTCOME:

1. Appropriate for gestational age (AGA)
2. Small for gestational age (SGA)
3. Still birth

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR, KARNATAKA

INFORMED CONSENT FORM

PG guide's name: Dr. ADARSH A D

Principal investigator: Dr. SHANTALA SAWKAR

I Mrs. _____ have been explained in my own understandable language, that I will be included in a study which is **"ROLE OF SHEAR WAVE ELASTOGRAPHY OF PLACENTA IN NORMAL AND PRE-ECLAMPTIC PREGNANCIES IN THIRD TRIMESTER."** I have been explained that my clinical findings, investigations, postoperative findings will be assessed and documented for study purpose.

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

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

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

I have principal investigator mobile number for enquiries.I in my sound mind give full consent to be added in the part of this study.

Signature of the patient:

Name:

Signature of the witness:

Name:

Relation to patient:

Date:

Place:

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR, KARNATAKA

PATIENT INFORMATION SHEET

STUDY TITLE: ROLE OF SHEAR WAVE ELASTOGRAPHY OF PLACENTA IN NORMAL AND PRE-ECLAMPTIC PREGNANCIES IN THIRD TRIMESTER

This is to inform you that,

I, Dr. Shantala Sawkar, post-graduate student in Department of Radiodiagnosis at Sri Devaraj Urs Medical College. I will be conducting a study titled "Role of shear wave elastography of placenta in normal and pre-eclamptic pregnancies in third trimester." for my dissertation under the guidance of Dr. Adarsh A D , Professor, Department of Radiodiagnosis In this study, we will assess the role of shear wave elastography of placenta in normal and pre-eclamptic pregnancies in third trimester

You are free to opt-out of the study at anytime if you are not satisfied or apprehensive to be a part of the study. Your treatment and care will not be compromised if you refuse to be a part of the study. The study will not add any risk or financial burden to you if you are part of the study. Your identity and clinical details will be confidential. You will not receive any financial benefit for being part of the study.You are free to contact Dr. Shantala Sawkar or any other member of the above research team for any doubt or clarification you have.

Dr. Shantala Sawkar

Mobile no: 8884746278

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MASTER CHART

KEY TO MASTER CHART

SL NO	ABBREVIATION	FULL FORMS
1.	UHID	Unique Health Identification Number.
2.	GA	Gestational age
3.	LMP	Last menstrual period
4.	FHR	Fetal heart rate
5.	AUA	Arithmetic ultrasound age
6.	E1	Centre of placenta
7.	E2	Edge of placenta
8.	Avg	Average
9.	SWE	Shear wave elastography
10.	kPa	kilopascal
11.	AGA	Appropriate for gestational age
12.	SGA	Small for gestational age
13.	PE	Pre-eclampsia
14.	W	Weeks
15.	D	Days

SL NO	UHID	AGE	GRAVIDA	GROUP	GA BY LMP	FHR (bpm)	GA BY AUA	PLACENTA LOCATION	PLACENTA GRADING	E1	E2	Avg SWE (kPa)	OUTCOME
1	106053	22	1	normal	35 W 4 D	147	35 W	Fundal anterior	II	2.43	2.69	2.56 kPa	AGA
2	151179	24	2	normal	29 W 5	152	29 W 2D	Anterior	I	3.02	3.26	3.14 kPa	AGA
3	152268	21	1	normal	30 W	155	31 W 2 D	Fundal anterior	II	2.7	2.81	2.75kPa	AGA
4	159273	30	3	normal	34 W 4 D	142	34 W 4 D	Anterior, right lateral	II	3.4	3.62	3.51 kPa	AGA
5	180053	24	2	normal	34 W 6 D	151	34 W 5 D	Fundal and left lateral	II	2.71	2.93	2.82 kPa	AGA
6	183830	25	1	normal	36 W 6 D	152	36 W 4 D	Fundal, anterior	II	3.04	3.2	3.12 kPa	AGA
7	186441	29	3	normal	36 W 3 D	143	35 W	Anterior and left lateral	II	2.58	2.74	2.66 kPa	AGA
8	186772	25	1	normal	37 W	133	35 W 1 D	Anterior right lateral	II	3.02	3.26	3.14 kPa	AGA
9	191255	22	1	normal	30 W	160	28 W 3 D	Fundal right lateral	I	3.04	3.2	3.11 kPa	AGA
10	192242	26	1	normal	28 W	145	29 W 4 D	Anterior	I	3.9	4.1	4.00 kPa	AGA
11	204318	22	2	normal	30 W 4 D	141	32 W	Anterior	II	3.8	3.98	3.89 kPa	AGA
12	204975	21	1	normal	33 W 4 D	139	31 W 2 D	Fundal anterior	II	3.58	3.74	3.66 kPa	AGA
13	210097	22	1	normal	34 W 3 D	151	35 W 1 D	Anterior right lateral	II	2.48	2.64	2.56 kPa	AGA
14	212051	24	3	normal	34 W 3 D	144	34 W 5 D	Anterior	II	4.01	4.21	4.11 kPa	AGA
15	213130	25	1	normal	31 W 5 D	158	31 W 4 D	Anterior	I	2.71	2.85	2.78 kPa	AGA
16	75458	27	2	normal	31 W 2 D	144	30 W 4 D	Anterior	II	3.5	3.66	3.58 kPa	AGA
17	108763	25	2	normal	33 W 1 D	147	33 W 3 D	Anterior	II	2.71	2.93	2.82 kPa	AGA
18	113610	28	1	normal	31 W 4 D	148	33 W 2 D	Anterior	II	3.6	3.72	3.66 kPa	AGA
19	118484	28	1	normal	35 W 4 D	138	35 W 2 D	Anterior	II	2.6	2.7	2.65 kPa	AGA
20	125435	28	2	normal	29 W 1 D	144	29 W 3 D	Fundal	II	3.04	3.24	3.14 kPa	AGA
21	171553	23	1	normal	35 W 4 D	141	33 W	Fundal anterior	II	2.71	2.93	2.82 kPa	AGA
22	184223	25	2	normal	33 W 2 D	152	33 W 5 D	Anterior	II	3.7	3.92	3.81 kPa	AGA
23	185494	32	3	normal	33 W 3 D	140	33 W 6 D	Anterior	II	2.8	2.84	2.82 kPa	AGA
24	196321	22	1	normal	35 W 4 D	147	35 W	Fundal anterior	II	2.5	2.62	2.56 kPa	AGA
25	222153	24	3	normal	29 W 5	152	29 W 2D	Anterior	I	3.04	3.24	3.14 kPa	AGA
26	221841	21	1	normal	30 W	155	30 W 2D	Anterior	II	2.71	2.85	2.78 kPa	AGA
27	234273	30	2	normal	34 W 4 D	142	34 W 4 D	Anterior, right lateral	II	3.4	3.62	3.51 kPa	AGA
28	238913	24	2	normal	34 W 6 D	151	34 W 5 D	Fundal, right lateral	II	2.7	2.94	2.82 kPa	AGA
29	216910	25	1	normal	36 W 6 D	152	36 W 4 D	Fundal, anterior	II	3.24	3.01	3.12 kPa	AGA
30	200092	29	3	normal	36 W 3 D	143	35 W	Anterior, left lateral	II	2.7	2.62	2.66 kPa	AGA
31	239005	25	1	normal	37 W	133	35 W 1 D	Anterior right lateral	II	3.1	3.18	3.14 kPa	AGA
32	221375	22	1	normal	30 W	160	28 W 3 D	Fundal right lateral	I	3.2	3.02	3.11 kPa	AGA
33	200232	26	1	normal	28 W	145	29 W 4 D	Anterior	I	3.91	4.09	4.00 kPa	AGA
34	286682	22	2	normal	30 W 4 D	141	32 W	Anterior	II	3.8	3.98	3.89 kPa	AGA

SL NO	UHID	AGE	GRAVIDA	GROUP	GA BY LMP	FHR (bpm)	GA BY AUA	PLACENTA LOCATION	PLACENTA GRADING	E1	E2	Avg SWE (kPa)	OUTCOME
35	198493	21	1	normal	33 W 4 D	139	31 W 2 D	Fundal anterior	II	3.72	3.6	3.66 kPa	AGA
36	242201	22	1	normal	34 W 3 D	151	35 W 1 D	Anterior right lateral	II	2.64	2.48	2.56 kPa	AGA
37	215648	24	2	normal	34 W 3 D	144	34 W 5 D	Anterior	II	4.01	4.21	4.11 kPa	AGA
38	206128	25	1	normal	31 W 5 D	158	31 W 4 D	Anterior	I	2.7	2.86	2.78 kPa	AGA
39	206915	27	3	normal	31 W 2 D	144	30 W 4 D	Anterior	II	3.5	5.64	3.58 kPa	AGA
40	201354	25	1	normal	33 W 1 D	147	33 W 3 D	Anterior	II	2.7	2.94	2.82 kPa	AGA
41	244323	28	2	normal	31 W 4 D	148	33 W 2 D	Fundal anterior	II	7.92	8.22	8.0 kPa	AGA
42	219141	24	1	normal	35 W 4 D	138	35 W 2 D	Anterior	II	3.9	4.08	3.99 kPa	AGA
43	243869	28	2	normal	29 W 1 D	144	29 W 3 D	Fundal	II	2.58	2.72	2.65 kPa	AGA
44	203106	23	1	normal	35 W 4 D	141	33 W	Fundal anterior	II	3.04	3.24	3.14 kPa	AGA
45	211648	32	2	normal	33 W 3 D	140	33 W 6 D	Anterior	II	2.9	2.74	2.82 kPa	AGA
46	239005	22	1	normal	35 W 4 D	147	35 W	Fundal anterior	II	2.88	2.76	2.82 kPa	AGA
47	327364	31	1	normal	29W1D	143	29W 1 D	Anterior right lateral	II	7.45	6.84	7.14 kp	AGA
48	326479	28	3	normal	37W	157	34 W 2D	Right lateral	II	3.7	3.9	3.8 kPa	AGA
49	323363	35	3	normal	36W2D	149	35W1D	Fundal anterior	II	2.81	2.99	2.9 kPa	AGA
50	329787	23	1	normal	35 W 4 D	143	35 W 5 D	Anterior	II	3.6	3.72	3.66 kPa	AGA
51	329503	22	2	normal	35 W	154	35W 4D	Anterior left lateral	III	2.48	2.64	2.56 kPa	AGA
52	328856	22	1	normal	37 W	142	35 W 6 D	Fundal	III	4.01	4.21	4.11 kPa	AGA
53	284741	30	3	normal	36 W 1D	155	34 W 1 D	Anterior	II	2.7	2.86	2.78 kPa	AGA
54	134193	29	1	normal	37 W	155	35 W 3 D	Anterior	II	7.9	8.4	8.15 kPa	AGA
55	312214	34	2	normal	30 W	153	32 W 5 D	Anterior	II	2.7	2.94	2.82 kPa	AGA
56	231188	32	1	normal	33 W 6D	154	35 W 2D	Anterior	II	3.9	4.08	3.99 kPa	AGA
57	309711	32	1	normal	36 W 1 D	132	35 W 5 D	Fundal	II	2.69	2.61	2.65 kPa	AGA
58	298123	28	2	normal	28 W	152	29 W 3 D	Fundal anterior	II	3.24	3.04	3.14 kPa	AGA
59	308712	24	3	normal	29 W 4D	150	30 W 5 D	Anterior	II	2.72	2.92	2.82 kPa	AGA
60	307734	19	1	normal	37 W 2 D	148	35 W 4 D	Anterior	II	2.92	2.72	2.82 kPa	AGA
61	299222	20	3	normal	34 W	147	35 W 4D	Fundal	II	2.5	2.62	2.56 kPa	AGA
62	332445	27	1	normal	35 W 3 D	130	33 W 6 D	Fundal anterior	II	5.6	7.4	6.5 kPa	AGA
63	333761	24	2	normal	36 W	134	35 W 2 D	Right lateral	III	3.7	3.9	3.8 kPa	AGA
64	334056	31	1	normal	36 W	164	32 W 6 D	Fundal	II	2.81	2.99	2.9 kPa	AGA
65	334170	19	1	normal	34 W 1D	147	32 W 5 D	Left lateral	II	3.02	3.26	3.14 kPa	AGA
66	210882	31	2	normal	28 W 3D	136	29 W 4 D	Fundal	II	2.7	2.94	2.82 kPa	AGA
67	336137	19	2	normal	35 W	142	34 W 5 D	Anterior	II	2.94	2.7	2.82 kPa	AGA

SL NO	UHID	AGE	GRAVIDA	GROUP	GA BY LMP	FHR (bpm)	GA BY AUA	PLACENTA LOCATION	PLACENTA GRADING	E1	E2	Avg SWE (kPa)	OUTCOME
1	106055	25	1	PE	35 W 4 D	147	35 W	Fundal anterior	II	9.94	10.14	10.04	AGA
2	151889	27	2	PE	29 W 5	152	29 W 2D	Anterior	I	12.05	12.25	12.15	AGA
3	152468	32	1	PE	30 W	155	30 W 2D	Anterior	II	9.36	9.56	9.46	AGA
4	186773	29	3	PE	34 W 4 D	142	34 W 4 D	Anterior, right lateral	II	9.25	9.45	9.35	SGA
5	180053	24	3	PE	34 W 6 D	151	34 W 5 D	Fundal and left lateral	II	9.8	9.7	9.75	AGA
6	184430	29	1	PE	36 W 6 D	152	36 W 4 D	Fundal, anterior	II	9.54	9.74	9.64	AGA
7	186461	35	2	PE	36 W 3 D	143	35 W	Anterior and left lateral	II	4.9	5.5	5.20	SGA
8	186992	29	1	PE	37 W	133	35 W 1 D	Anterior right lateral	II	8.92	9.22	9.02	SGA
9	193355	25	1	PE	30 W	160	28 W 3 D	Fundal right lateral	I	10.82	11.02	10.92	AGA
10	192242	22	1	PE	28 W	145	29 W 4 D	Anterior	I	10.88	11.08	10.98	AGA
11	204318	22	2	PE	35 W 4 D	141	32 W	Anterior	II	9.8	9.6	9.70	SGA
12	204975	21	1	PE	34 W 3 D	139	31 W 2 D	fundal anterior	II	8.6	8.4	8.50	SGA
13	210097	26	1	PE	34 W 3 D	151	35 W 1 D	Anterior right lateral	III	13.67	13.47	13.57	AGA
14	212051	24	2	PE	34 W 3 D	144	34 W 5 D	Anterior	II	11.68	11.48	11.58	AGA
15	213130	24	1	PE	31 W 5 D	158	31 W 4 D	Anterior	I	10.4	10.6	10.50	AGA
16	212055	29	3	PE	31 W 2 D	144	30 W 4 D	Anterior	II	11.84	12.04	11.94	SGA
17	108763	21	1	PE	33 W 1 D	147	33 W 3 D	Anterior	II	13.74	13.94	13.84	SGA
18	113610	32	2	PE	31 W 4 D	148	33 W 2 D	Fundal anterior	II	10.32	10.52	10.42	AGA
19	118484	28	1	PE	35 W 4 D	138	35 W 2 D	Anterior	II	13.8	14	13.90	AGA
20	125435	28	2	PE	29 W 1 D	144	29 W 3 D	Fundal	II	14.47	14.67	14.57	SGA
21	171553	31	1	PE	35 W 4 D	141	33 W	Fundal anterior	II	11.88	12.08	11.98	AGA
22	184223	23	3	PE	33 W 2 D	152	33 W 5 D	Anterior	II	14.89	14.69	14.79	AGA
23	185494	23	2	PE	33 W 3 D	140	33 W 6 D	Anterior	II	11.8	11.6	11.70	SGA
24	296695	25	1	PE	35 W 4 D	147	35 W	fundal anterior	II	10.06	10.02	10.04	AGA

SL NO	UHID	AGE	GRAVIDA	GROUP	GA BY LMP	FHR (bpm)	GA BY AUA	PLACENTA LOCATION	PLACENTA GRADING	E1	E2	Avg SWE (kPa)	OUTCOME
25	339882	31	2	PE	29 W 5	152	29 W 2D	Anterior	I	12.05	12.25	12.15	AGA
26	206128	23	1	PE	36 W	155	35 W 6 D	Anterior	II	9.56	9.36	9.46	AGA
27	300163	22	3	PE	36 W	142	35 W 6 D	Fundal anterior	III	9.45	9.25	9.35	AGA
28	281787	20	3	PE	36 W	151	33 W	Fundal and left lateral	II	9.65	9.85	9.75	SGA
29	295794	29	1	PE	36 W 6 D	152	36 W 4 D	Fundal, anterior	II	9.74	9.84	9.64	AGA
30	346167	35	2	PE	36 W 3 D	143	35 W	Fundal, right lateral	II	13.26	13.26	13.26	SGA
31	324703	25	1	PE	37 W	133	36 W 2 D	Anterior right lateral	II	9.09	8.95	9.02	AGA
32	215648	21	1	PE	35 W	160	33 W 4 D	Fundal right lateral	I	10.99	10.85	10.92	AGA
33	298911	22	1	PE	28 W	145	29 W 4 D	Anterior	I	10.9	11.07	10.98	AGA
34	211648	24	2	PE	35 W 4 D	141	33 W 5 D	Fundal left lateral	II	9.6	9.8	9.70	AGA
35	200232	25	1	PE	36 W 5 D	139	32 W 2 D	fundal anterior	II	8.4	8.6	8.50	SGA
36	296196	26	1	PE	34 W 3 D	151	35 W 1 D	Anterior right lateral	II	13.5	13.64	13.57	AGA
37	338477	24	3	PE	34 W 3 D	144	34 W 5 D	Anterior	II	11.68	11.48	11.58	SGA
38	316597	24	1	PE	30 W 2 D	158	29 W 4 D	Anterior	I	10.6	10.4	10.50	AGA
39	314744	23	2	PE	31 W 2 D	144	31 W 1 D	Anterior	III	12.04	11.84	11.94	AGA
40	314749	21	1	PE	33 W 1 D	147	32 W 5 D	Anterior	II	13.94	13.74	13.84	AGA
41	334221	21	2	PE	37 W	148	34 W 1 D	right lateral	II	10.52	10.32	10.42	SGA
42	294186	28	1	PE	35 W 4 D	138	35 W 2 D	Anterior	II	14	13.8	13.90	AGA
43	297249	28	2	PE	36 W 2 D	144	29 W 3 D	Anterior left lateral	II	14.67	14.47	14.57	SGA
44	321300	22	1	PE	35 W 4 D	141	35 W 2 D	fundal anterior	II	12.08	11.88	11.98	AGA
45	300177	23	2	PE	33 W 2 D	152	33 W 5 D	Anterior	II	14.89	14.69	14.79	AGA
46	172857	31	3	PE	37 W 2 D	140	33 W 6 D	Anterior	II	11.6	11.8	11.70	SGA
47	393281	30	2	PE	36 W 0 D	138	35 W 6 D	Anterior	II	9.94	10.14	10.04	AGA
48	349092	35	2	PE	37W 0 D	155	34 W 4 D	fundal left lateral	II	9.36	9.56	9.46	SGA

SL NO	UHID	AGE	GRAVIDA	GROUP	GA BY LMP	FHR (bpm)	GA BY AUA	PLACENTA LOCATION	PLACENTA GRADING	E1	E2	Avg SWE (kPa)	OUTCOME
49	348899	32	1	PE	32W 2 D	157	32W 2D	Fundal	II	12.46	12.78	12.50	AGA
50	348415	24	1	PE	36 W 6 D	139	31 W 5 D	Anterior	II	9.25	9.45	9.35	SGA
51	347593	36	2	PE	33 W 0 D	139	33 W 4 D	right lateral	II	9.8	9.7	9.75	AGA
52	347376	24	1	PE	28 W 5 D	142	28 W 2 D	Anterior	II	9.54	9.74	9.64	AGA
53	293227	29	1	PE	32 W 3D	154	31 W	Anterior	I	13.03	13.23	13.13	Still birth
54	156258	24	2	PE	29 W 2 D	147	31 W 4 D	Anterior	II	8.92	9.22	9.02	AGA
55	262554	22	1	PE	32 W 3D	138	33 W 4 D	Fundal anterior	II	10.82	11.02	10.92	AGA
56	289868	22	1	PE	37 W 4 D	135	33 W 3 D	Anterior	II	10.88	11.08	10.98	SGA
57	292742	18	1	PE	30 W 5 D	135	31 W 4 D	Fundal anterior	II	9.8	9.6	9.70	AGA
58	293431	30	3	PE	31 W 5 D	142	35 W 5 D	Fundal	II	8.6	8.4	8.50	AGA
59	206915	20	1	PE	36 W	149	35 W 1 D	Anterior	II	13.67	13.47	13.57	AGA
60	223154	27	2	PE	30 W 1 D	149	28 W 4 D	Anterior	II	11.68	11.48	11.58	AGA
61	250225	20	1	PE	34 W	146	31 W 3 D	Anterior	II	10.4	10.6	10.50	SGA
62	262885	23	2	PE	33 W 4 D	153	34 W 2 D	Anterior	II	11.84	12.04	11.94	AGA
63	293646	19	1	PE	29 W	155	30W 2 D	Fundal	I	13.74	13.94	13.84	Still birth
64	342963	20	1	PE	36 W 3 D	130	35 W	Anterior	II	10.32	10.52	10.42	AGA
65	347525	30	3	PE	35 W 2 D	158	35 W 2 D	Anterior	II	13.81	13.99	13.90	AGA
66	347074	23	1	PE	37 W 3 D	135	34 W 2 D	Fundal	II	14.69	14.45	14.57	SGA
67	347525	30	2	PE	36 W	150	35 W 2 D	Fundal	II	12.7	12.9	12.80	AGA