"ROLE OF UTERINE ARTERY DOPPLER IN PREDICTING PREECLAMPSIA"

By

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LIST OF ABBREVIATIONS

AEDF	-	Absent end diastolic Flow
FVW	-	Flow velocity Waveform
FGR	-	Fetal Growth Restriction
IUGR	-	Intra Uterine Growth Restriction
LMP	-	Last Menstrual Period
LSCS	-	Lower Segment Caesarian Section
NICU	-	Neonatal intensive care unit
NPV	-	Negative Predictive value
NVD	-	Normal vaginal Delivery
PPV	-	Positive Predictive Value
RI	-	Resistance Index
S/D	-	Systolic/ Diastolic ratio
SES	-	Socio Economic Status

ABSTRACT

ROLE OF UTERINE ARTERY DOPPLER IN PREDICTING PRE-ECLAMPSIA

AIMS AND OBJECTIVES

1. To evaluate whether abnormal uterine artery Doppler study can be used as an effective screening test to predict the development of pre-eclampsia at 14-20 weeks of gestation.

2. To study the flow velocity waveforms in the uterine arteries in mid-trimester.

METHODS

This study is a prospective study including105 woman with singleton pregnancy between 14 to 20 weeks of gestation over the period of 2 years. Bilateral uterine artery Doppler flow velocity was studied. In both uterine arteries, RI, PI and S/D ratio were studied. The presence of early diastolic notch is noted in both the uterine arteries.

RESULTS

100 patients were taken for analysis as five patients were excluded from the study. Out of 100 patients, 7 patients developed preeclampsia. Uterine artery Doppler was abnormal in 9 patients with sensitivity of 87% and specificity of 97% for pre eclampsia.

INTERPRETATION AND CONCLUSION

Uterine artery Doppler in 2nd trimester (14-20 wks) can be used as a good screening test for prediction of preeclampsia. Therefore, it helps in early detection of at risk women and early treatment and favorable outcome.

KEYWORDS

Pre eclampsia, Uterine artery, RI, PI, S/D

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INTRODUCTION

5 to 10% of pregnancies are complicated by hypertensive disorders. Hemorrhage, infections and hypertensive disorders form the deadly triad which majorly effects the maternal mortality and morbidity rates[1]. Preeclampsia involves multiple systems which affects about 2% of pregnancies[2] and is of great threat to mother and fetus. Other than its most fearful complication of developing eclampsia, preeclampsia on its own can cause significant maternal and perinatal morbidity.

As stated by Bringman et al., 2006, preeclampsia is the major source of both maternal and fetal morbidity and mortality. It is approximated that > 14% of maternal deaths / year worldwide are because of preeclampsia and eclampsia, although in developed countries, it mainly affects baby[3]. The incidence of preterm is about 15%[4].

Trophoblast generally invades decidua of spiral arteries at 8th week of gestation and generally completes by 13th week. Then the 2nd stage of spiral artery invasion starts, wherein the myometrial spiral arteries are likewise invaded by the trophoblasts. This process generally completes by 18 - 19 weeks, sometimes my extend up to 22 - 24 weeks. In majority of pre-eclampsia patients, this transition does not occur in the spiral artery bed causing increased resistance to flow into the intervillous space. Uterine artery Doppler waveform indirectly monitors the status of the spiral artery bed, which is the method of choice[4]. Abnormal uterine artery Doppler determined by ultrasound in the 1st and 2nd trimester provide indirect evidence of subsequent development of preeclampsia.

Low peak flow velocity and early diastolic notch is seen in uterine artery Doppler in the non-pregnant female. Gestational weeks of 18 to 20 wks have high flow velocity with no diastolic notch. Abnormal flow is taken when there are high resistance utero-placental waveforms and the presence of notch which is the due to arterial vessel tone and elasticity of the vessel and vasospasm. It disappears in 2nd trimester. High resistance flow is associated with increased rate of pregnancy complication, 70% risk of developing protienuric hypertension and 30% risk of a small for gestational age fetus[7].

Varying sensitivity are noted on the basis of type of Doppler, region of interest, abnormal uterine artery resistance, gestational age[7].My study helps in evaluating the usefulness of UA Doppler in the gestational age of 14-20 weeks, which is before the clinical presentation Hence, helping the obstetrician in taking early precautions and treatment.

OBJECTIVE OF THE STUDY

1. To evaluate whether abnormal uterine artery Doppler study can be used as an effective screening test to predict the development of pre-eclampsia at the gestational age of 14-20 weeks.

2. To study the flow velocity waveforms in the uterine arteries in mid-trimester.

REVIEW OF LITERATURE

The history of hypertensive disorders in pregnancy is as old as human existence. Since olden age, seizures were seen in 3rdtrimister, during the labor and post-partum. Hippocrates had also acknowledged the poor prognosis of seizures during labor. Indian atharva-veda and sushruta, both mentioned about preeclampsia and eclampsia.

This disorder was known long time ago. This disorder was named eclampsia and for many yrs was known as pregnancy specific seizure disorder.

In the later part of 17th century, Francis Mauericeua found preeclampsia as a specific disorder of pregnancy. He could see that the seizures mostly stopped after delivery and suggested early cessation of pregnancy as the best therapy.

In the later part of 1800s the interrelation of proteinuria and later increased BP with eclampsia was understood. John Charles lever in 1843 saw that many women with seizures had albumin in their urine. It was also observed that increased blood pressure and protein in their urine preceded the convulsions. Because of this, the term preeclampsia was derived. Even in the absence of seizures, maternal and infant risk was increased[8].

Figure 1- Johaann Christian Andreas Doppler

Today's applications of the modality of color Doppler superimposed on two dimensional ultrasonography are diverse. Johaann Christian Andreas Doppler a physicist and mathematician in Austria demonstrated the Doppler Effect. He established the principle of frequency shift and developed the formula for calculating the velocity from the shift.

The principles applied to Doppler technology include the physical property described by Christian Doppler that the frequency of waves (sound) produced by objects (moving RBC) approaching the observer (transducer) is higher than that of objects moving away from the observer. When the source and transducer move closer, the frequency increases, when they move away, the frequency decreases. This is known as "Doppler effect", the change in frequency is known as Doppler frequency shift or just the Doppler shift.

Doppler Effect is also observed in relation to reflected ultrasound. This basic principle provides the foundation for constructing Doppler ultrasound devices to measurebloodflowvelocity.8 The magnitude and direction of the frequency shift depends on the relative motion of the moving target, their velocity and direction can be determined[9].

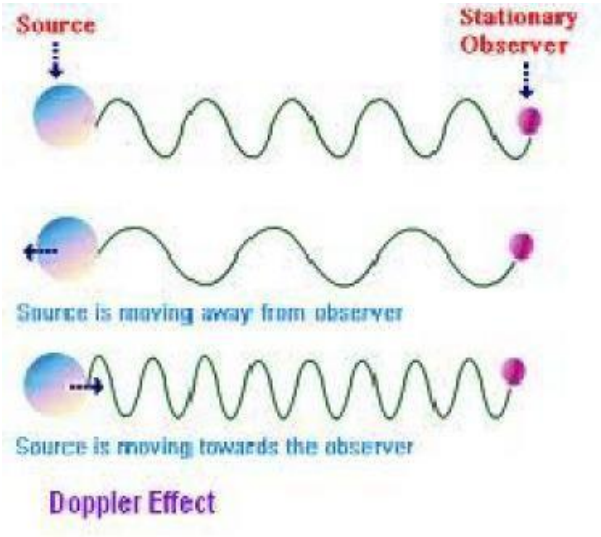


Figure 2: Doppler effect

Where,

Fd = Doppler shift frequency

Ft = Transmitted frequency

Fr = Received frequency

When the source and the observer come closer, the wavelength decreases and the frequency increases. Conversely, when the source and the observer move apart, the wavelength increases and the frequency decreases.

The utility of the Doppler Effect originates from the fact that the shift in frequency is proportional to the speed of movement between the source and the receiver and therefore can be used to assess this speed.

The 1st pulsed wave Doppler apparatus was made by Seattle research team in 1966. Remarkable contribution was done by Dennis Watkins, Donand Baker and John Reid. For the first time, the

ultrasound operator could determine the target of Doppler insonation due to Duplex Doppler technique. This was the important landmark in the obstetric applications as with this, fetal and maternal circulation could be evaluated.

Campbell, who was a pioneer in obstetric ultrasound, was the first to prospect the capability of uterine artery Doppler in predicting preeclampsia. In the beginning, he used a handheld continuous wave Doppler tool to find the predictable waveform at about 18wks. Even though his findings helped, others could not get the same results. Hence, it became understandable that the continuous wave did not allow in specifying the sampling site (as in pulse wave) and more importantly, 25% of patients who previously had abnormal Doppler's at 18weeks of gestation did have normal Doppler by 24wks. These type do not have the same predilection for preeclampsia as in those waveforms which remain abnormal at24 Wks[10].

The indices used to predict adverse outcome include a Resistive index of >0.55, a Systolic /Diastolic ratio of >2.6, a Notch in early diastole, a Systolic notch and a large difference of the right and left sides of the uterine circulation[44.]

HYPERTENSIVE DISORDERS IN PREGNANGY

Hypertensive disorders in pregnancy is normally used to express a wide range of patients who may be having only mild increase in blood pressure or severe hypertension with various organ impairment.

Incidence

5 to 10 percent of all pregnancies are complicated by hypertensive disorders. Incidence in India is 5-15%[11], the incidence is more in nulliparous (15%)than multiparas (10%)[9,11].

TABLE-1 [9,12]

Gestational HTN	5%
Pre-eclampsia	5-7%
Eclampsia	0.5-2%
Pre-eclampsia superadded on chronic HTN	25%
Chronic HTN	1-2%

Categorizing hypertensive disorders in pregnancy

A number of classifications and definitions of the various hypertensive disorders of pregnancy exist and new ones are being put forward constantly. the failure to achieve an agreed classification results from a lack of knowledge of the precise nature and cause of the disorder, the absence of clinical or pathological features or tests by which they can be clearly separated, and want of an agreed nomenclature.

The classification of hypertensive disorders which complicate pregnancy include[9]:

1. Gestational hypertension— previously termed pregnancy-induced HTN. When hypertension reduces by 12 weeks post-partum and there is no proteinuria.
2. Preeclampsia syndrome superadded on chronic hypertension
3. Preeclampsia and eclampsia syndrome
4. Chronic hypertension

Importantly, this classification differentiates the preeclampsia syndrome from other hypertensive disorders because it is potentially more ominous. This concept aids interpretation of studies that

address the etiology, pathogenesis, and clinical management of pregnancy-related hypertensive disorders

Definitions

Gestational hypertension:

- Systolic Blood Pressure of 140 or diastolic Blood Pressure of 90 mm of Hg for the first time during pregnancy after 20weeks of gestation.
- No evidence of protein in urine
- Resolution of Blood Pressure after 12 weeks of delivery
- Final diagnosis made in postpartum period
- May have other signs or symptoms of preeclampsia like abdominal discomfort or reduced platelets.

Preeclampsia:

Minimum criteria:

- Blood Pressure of 140/90 mm of Hg after 20 weeks of gestation
- Proteinuria - 300 milligram per 24 hrs or persistent 30 mg/dL (1 + dipstick)protein

in random urine samples or a urine protein : creatinine ratio ≥ 0.3;

or

- Thrombocytopenia - Platelets 100,000/L

- Renal insufficiency - Creatinine 1.1 mg/dL or doubling of baseline Liver involvement
- Cerebral symptoms
- Serum transaminase levels twice normal Headache, visual disturbances, convulsions ,Pulmonary edema

Features of severe preeclampsia (any one of these)

- Systolic Blood Pressure 160 mm Hg or higher, or diastolic blood pressure of 110 mm Hg or higher on 2 occasions at least 6 hours apart when the patient is on bed rest
- Thrombocytopenia (Platelets < 100,000/ml)
- Impaired liver function as indicated by abnormally elevated blood concentration of liver enzymes (to twice normal concentration), severe persistent right Upper quadrant or epigastric pain.
- Progressive Renal Insufficiency (Serum creatinine of more than 1.1 mg/dl unless known to be previously elevated, or doubling of S. creatinine levels in the absence of other renal disease
- Pulmonary edema
- Constant headache or other visual or cerebral impairments

Eclampsia:

- Seizures that cannot be attributed to other causes in a woman with preeclampsia.

Or

- Eclampsia is the occurrence of convulsions or coma during pregnancy or postpartum unrelated to other cerebral conditions with signs and symptoms of preeclampsia.

Superimposed preeclampsia on chronic hypertension:

- Proteinuria of300 milligram per 24 hours in hypertensive female with no evidence of protein in urine before 20 weeks of gestation
- Unexpected increase in proteinuria or BP or platelet count of less than100,000/l in hypertensive women and proteinuria before 20 weeks of gestation

Chronic hypertension:

- Blood Pressure of 140/90 mm of Hg before pregnancy or identified before 20 wks of gestation which is not accredit able to gestational trophoblastic disease *or*

- Hypertension identified for the first time after 20 weeks pregnancy and continued till 12 weeks post-delivery.

Risk factors for preeclampsia[13,14]

Pregnancy associated factors

- Hydatidiform mole

- Chromosomal abnormalities
- Multiple pregnancy
- Hydrops fetalis
- Oocyte donation or donor insemination

Maternal specific factors

- Age greater than 35years
- Age less than 20years
- Black race
- Family background of preeclampsia
- Primi
- Preeclampsia developed in a previous pregnancy
- Medical conditions like pregnancy induced DM, type 1 DM, renal disease, obesity, chronic hypertension & Thrombophilias
- Stress

Paternal specific factors

- First time father
- Previously fathered a preeclampsia pregnancy in another woman

Etio-pathogenesis of preeclampsia

Hypertensive disorders in pregnancy possibly develop more in women who:

Are exposed to abundance of placental villi as in twin pregnancy or hydatidiform mole

In primigravidae, who are exposed to placental villi for the first time

Have preexisting cardiovascular or kidney diseases

Genetically inclined to developing hypertension during pregnancy

Preeclampsia is a pregnancy specific condition that resolves with delivery. The placenta appears to be the pregnancy component that leads to the disorder. Uterine distension had once been considered important but preeclampsia can occur with abdominal ectopic pregnancies without increased uterine size, eliminating this possibility. Likewise, preeclampsia is actually more common in pregnancies without a fetus (hydatidiform moles). Thus the fetus is not the contributor. The placenta is necessary for preeclampsia but all pregnant women have placentas and only 5% become preeclamptic. Many years ago Page suggested that the important placental feature in preeclampsia was poor perfusion. This is supported by the abnormal implantation with subsequent reduced vascularization of the placental site characteristic of preeclampsia. In addition, obstetrical conditions associated with large placentas such as hydatidiform moles or multiple gestations all predispose to preeclampsia. It is postulated that with the large placenta the normal vasculature of the placental site is inadequate to perfuse the very large fetus/fetuses leading to relative placentalhypoperfusion[9].

Currently thinking about preeclampsia characterizes the reduction in perfusion as a two stage

disease

Stage 1 is caused by faulty endovascular trophoblastic remodeling that downstream causes the **Stage 2** clinical syndrome. There certainly is evidence that *some* cases of preeclampsia fit this theory. Although recognized by these two changes preeclampsia is much more than this[9].

Theories of developing preeclampsia

DISEASE OF THEORIES WITHOUT ANY CAUSE[15]. Description of eclampsia in writing has been dated as far back as 2200 BC by lindheimer and colleagues in 1999.It is not of great surprise that a lot of theories have been put forth to explain its cause. Presently reasonable likely causes include[9].

1. Abnormal trophoblastic invasion of uterine arteries
2. Endothelial cell activation
3. Immunological factors
4. Dietary deficiencies
5. Genetic influences

Abnormal trophoblastic invasion of uterine vessels

Preeclampsia is known for deficient trophoblastic iinvasion[9,15]. With such shallow invasion, only the decidual vessels become lined by endovascular trophoblasts. As a result, the deeper myometrial arterioles are not deprived of their endothelial lining, hence their caliber is only ½ that of vessels in normal placenta.

The cytotrophoblast converts from an epithelial to an endothelial phenotype, a process known as pseudo-vasculogenesis or vascular mimicry.

De wolf and coworkers (1980) examined arteries taken from the uteroplacental implantation site, and saw that early changes included destruction of endothelium, accumulation of plasma constituents into walls of vessel, proliferation, necrosis and lipid accumulation first in myo-intimal cells and later on in macrophage. Such lipid filled cells and related findings have been named atherosis. Usually, the vessels affected by atherosis lead to aneurismal dilatation and are commnly found in relation with spiral arterioles that have not undergone normal adaptation. Obstruction of the spiral arteriolar lumen by atherosis may impair placental blood flow. It is thought that these changes cause placental perfusion to be pathologically reduced, which later causes preeclampsia syndrome[9,16].

2. Immunological factors

a. Immune dysregulation: During gestation, there is immune tolerance to the placental and fetal antigens derived from father. Another theory for preeclampsia could be

loss of this tolerance or probably its dysregulation. Acute graft rejection at the maternal placental interface is noted at microscopic level. The probability of preeclampsia is increased considerably in situations where development of antibodies to placental antigens is uncommonly more than compared to the quantity of antibodies, as in multiple gestations. Immunity from previous abortion doesn't seem to happen. This concept of immunity was reinforced by their observations that preeclampsia developed less commonly in multiparas who had a prior termpregnancy[9].

b. Iimmune maladaptation: Beginning of the early second trimester, women bound to develop preeclampsia have a considerably less number of Helper t cells in comparison with women that don't develop. This th1/th2 disparity with th2 predominance may be arbitrated by adenosine which is seen in increased serum levels in preeclamptics compared with normotensive women.[16]

c. Endothelial cell activation:

Inflammatory changes are thought to be extension of stage 1 changes caused by faulty placentation. The placental factors released by ischemic changes or by any other inciting causes lead to a cascade of events set in motion. Thus an antiangiogenic and metabolic factors and other inflammatory mediators are thought to provoke endothelial cell injury.

Preeclampsia is considered a disease due to an extreme state of activated leucocytes in the maternal circulation. Briefly, cytokines such as tumor necrosis factor and the interleukins may add to the oxidative stress correlated with preeclampsia. The oxidative stress is characterized by reactive oxygen species and free radicals which leads development of lipid peroxides which further produce highly toxic radicals that damage endothelium, alter their nitric oxide generation and hinder prostaglandin equilibrium.

Angiogenic imbalance is due to the abundant anti angiogenic factors such as soluble endoglin and

placental soluble fms like tyrosine kinase 1. The generation of these factors is invoked by the aggravating hypoxia at the uteroplacental junction. Sflt-1 antagonizes vascular endothelial growth factor and placental growth factor, stopping the generation of NO and vasodilator, prostacyclins in the endothelium. An increase in sflt-1 levels and a corresponding decrease in vegf and pigf levels can be evaluated 5 to 6 wks before the presentation of clinical preeclampsia and have been taken as predictors for the further development of pre-eclampsia[9,17].

4. Genetic factors

Preeclampsia is a caused by multiple factors and multiple genes (polygenes). Ward & Lindeimer quote an raised risk for preeclampsia with family history of preeclampsia in a 1st degree kin[9]. The underneath table depicts the incidence of preeclampsia in a patient with positive background of preeclampsia.

Table 2

Relatives with positive history of preeclampsia	Incidence in the patient
Mother	20-40%
Sister	11-37%
Twin Heterozygous Monozygous	 22-47% 60%

This hereditary susceptibility is the result of inherited gene by both mother and father, which control enzymatic and metabolic functions of all organ systems. About seventy genes are found to have likely association.

Because of variety of preeclampsia syndrome, it is unlikely that any one gene will be found accountable[9].

Nutritional factors

Over the centuries a number of dietary deficiencies or excesses have been blamed as the cause of preeclampsia. The hypotheses have been diverse and often mutually exclusive. Thus increased and reduced sodium, protein, fats or carbohydrates were proposed as possible etiological factors. Rarely were these hypotheses appropriately tested in trials. Not surprisingly many care providers became disenchanted with these hypotheses and the role of nutrition was not extensively studied.

Over a period of time, it was noted that there was high incidence of preeclampsia in developing countries which forced some authors to conclude that malnutrition is a risk factor in the etiology of preeclampsia. Various studies were carried out in this direction, which showed that changes in the levels of blood trace elements in preeclamptic patients may implicate its pathogenesis.

First it was hypothesized that lowered s. magnesium levels during pregnancy might lead to development of seizures during pregnancy in at risk women, such as those with a propensity to epilepsy[8].

In 1980, An opposite association between ca++ intake and hypertensive disorders of pregnancy was first explained. Studies led to the theory that an increased ca++ intake during pregnancy might decrease the incidence of high BP and preeclampsia among women with less ca++ intake. An interrelation has been discovered between pre eclampsia and hypocalciuria, low plasma and high

membranous calcium, low urine calcium to creatinine ratio, low dietary milk intake. The reduction of serum calcium and the increase in intracellular calcium may cause arise of BP in preeclamptic mothers, this was accompanied by studies in which addition of calcium and magnesium was made to prevent preeclampsia. These studies have shown good results, in the form of reduction in the incidence of preeclampsia in the population whose diet was deficient in calcium[9].

PATHOGENESIS

Vasospasm: The concept of vasospasm was advanced by Volhard in 1918 based on study of small blood vessels. The vascular narrowing leads to resistance and further hypertension. Also, endothelial cell injury causes interstitial leakage through which blood constituents are placed in the sub endothelium. With reduced blood flow because of faulty distribution, ischemia of adjacent cells leading to necrosis, hemorrhage and other end organ disturbances. Paradoxically, vasoconstriction may be bad in women with preeclampsia than in those with the hemolysis, elevated liver enzymes, and a low platelet count syndrome[16].

Endothelial cell activation: has become the crux in the modern understanding of the pathological process of pre-eclampsia. Unidentified factors, likely from the placenta, are excreted into the maternal blood flow and incite activation and dysfunction of the vascular endothelium. This extensive endothelial cell changes is thought to lead to clinical syndrome of preeclampsia.[9,16,17].

Intact endothelium functions:

It basically helps in clotting and reduces the response to vasospasm.

Damaged /activated endothelial cells indirectly increase coagulation and the responsiveness to vasopressors. [17]

Vasoactive agents: generally pregnant women have reduced sensitivity to vasopressor substances. In preeclampsia, this refractoriness is lost and there is increased ascular response[9].

The vasoactive agents that cause these alterations are,

Prostaglandins: lot of prostaglandins is principal to the causation of the preeclampsia syndrome. Precisely, the reduced presser response noted in uneventful pregnancy is at least partly due to reduced vascular response mediated by vascular endothelial prostaglandin synthesis. Endothelial prostacyclini (pgi2) production is reduced in preeclampsia in comparison to normal pregnancy. This action appears to be mediated by phospholipase A2. At the same time, thromboxane A2 secretion by platelets is increased, and the prostacyclin: thromboxane A2 ratio decreases. The net result favors decreased sensitivity to infused angiotensin 11 and ultimatelyvasoconstriction[9,16].

Endothelins: these 21 amino acid peptides are potential vasoconstrictors, and endothelin 1 is the main isoform produced by endothelium. The plasma ET1 is the main isoform produced by endothelium in humans. Plasma ET1 is raised in non-hypertensive carrying women, but preeclamptics have even more plasma ET1. Amusingly, treatment of preeclampsia with MgSO4 reduces ET1plasma levels[9].

Angiogenic factors: Placental vasculogenesis is appreciable by 21days after fertilization. Angiogenic abnormality is used to explain large amounts of anti-angiogenic factors that are postulated to be due to increased hypoxia at the utero-placental junction.

Soluble fms like tyrosinee kinase 1(sFlt-1): a variant of the sFLT 1 receptor for placental growth factor and vascular endothelial growth factor. Raised maternal sFLT 1 levels disable and reduce

circulating free VEGF &PLGF concentrations causing endothelial dysfunction. sFLT 1 level begin to increase in maternal serum months before preeclampsia is obvious[9,16].

Soluble endogliin(seng): a 65 kda molecule from placenta which blocks endoglin which is a co-receptor for the TGF b.[9,16].

The reason of placental overproduction of anti-angiogenic peptides is still a mystery. The soluable forms aren't raised in the fetal circulation or amniotic fluid, and their levels in maternal blood disappear after delivery. Retrospective studies showed that third trimester increase in sFLT 1 levels and decreased plgf concentration correlate with development of preeclampsia post 25wks.

Nitric oxide is a powerful vasodilator which is produced from L arginine by endothelium of maternal and fetal blood vessels. It sustains the normal low pressure dilated state which is typical of feto-placental perfusion.

The outcome is not clear. It appears that the syndrome is associated with reduced endothelial nitric oxide isynthetase production thus increasing nitric oxide deactivation. It may be associated to race, with African American women giving rise to more nitric oxide[9,17].

To conclude, the cause of preeclampsia remains unclear although more and more proof is resulting to support that placenta has an important role. Few explain cause of preeclampsia as a 2 step process. The 1st involves abnormal placentation which is later followed by placental development of solublee factors that enter the blood vessels of mother and produce extensive endothelial damage.[18]

SCREENING FOR HYPERTENSIVE DISORDERS OF PREGNANCY

The significant mortality and grave late consequences of preeclampsia could be greatly reduced if we could precisely predict, prevent complications and better treat preeclampsia. It is obvious at present that there is no clinically helpful test to accurately predect preeclampsia.

Uterine artery Doppler

It is a noninvasive method to examine the uteroplacental circulation that provides indirect evidence of blood flow and is proposed a predictive test for preeclampsia.[19]

Serum markers

Several groups of fetal placental proteins have been evaluated for predicting the risk of preeclampsia.

Table3. Shows the number of markers studied since 1980s[20]

Placental perfusion and vascular resistance dysfunction related tests:
Roll over test
Mean BP in 2nd trimester
Isometric exercise test
Platelet angiotensiin II binding
Intravenous infusion of angiotensin II
Platelet calcium response to arginine vasopressin
Rennin
24 hours ambulatory BP monitoring
Doppler USG
Feto-placental unit endocrinology dysfunction- related tests:
AFP
HCG
InhibinA
Estriol
activinA
Pregnancy related plasma protein A
CRH
Renal dysfunction associated tests:
Microalbuminuria
Serum uric acid

Doppler in pregnancy induced hypertension

Pregnancy induced hypertension is often expected with medical conditions like DM, twin pregnancy, older women, few autoimmune diseases and renal diseases. Observant obstetrician can always think of and diagnose it early and treat accordingly in these conditions. Therefore, in routine ANC checkup, if any predictive test can be used as screening test for all women then this multisystem condition can be managed intime[9].

Principal behind uteroplacental waveform

Uteroplacental waveforms are obtained from the UA by color & pulsed Doppler ultrasound. As it is difficult to know whether these waves were from the UA or the arcuate A.[21] by using only pulsed wave Doppler, they are still generally referred to as above. With the use of color Doppler, the UA can be reliably recognized since pulsed wave reduces with advancing gestation. For example, high-resistance, notched uterine artery waveforms are 3 times more likely to be seen at 20 than at 24 weeks, when they occur in about 5% of pregnancies. Assessment of uterine artery waveforms is an accepted screening test in such pregnancy conditions[7].

UtA-PI and the proportion of SGA are lower in IVF/ICSI pregnancies conceived after FBT as compared to fresh blastocyst transfer.[22]

Figure 3 **Uteroplacental waveform analysis**

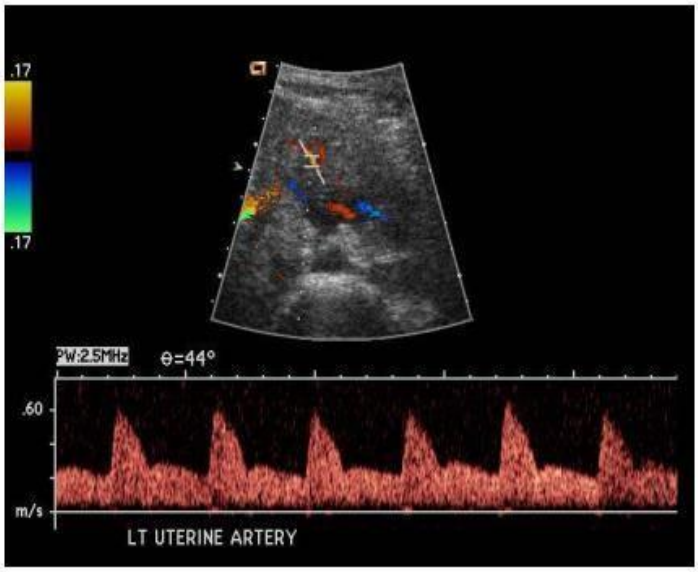
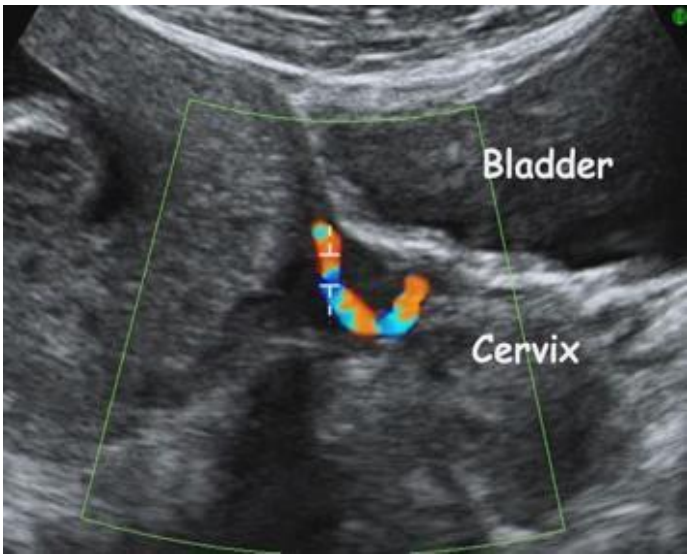


Figure 4 **Finding the uterine artery waveform**



Locating the uterine artery waveform

Ideally, a 4 megahertz probe has to be taken and the wall filter of vessel has to be set at 50 hertz, the frequency range of 4 kilohertz.[23] Make sure that the balance control is absolutely at its centre and the gain control is kept at about half of maximum.

Use color flow Doppler to recognize the bifurcation of the common iliac artery in longitudinal section. The internal iliac artery give uterine artery and joins the uterus just above the cervix. The main uterine artery divides into the arcuate arteries, which course anteriorly and posteriorly and stretch inward for about 1/3rd of the myometrial thickness. They vary in thickness and are tortuous in the region they supply. The arcuate artery web conjoin near the midline. The radial arteries that arise from this network, are run towards the uterine cavity, and become spiral arteries when they course the endometrium[24].

The probe is coursed medially and angled slightly towards the symphysis pubis to visualize the uterine artery just medial to the bifurcation, as it runs toward the uterus. The uterine artery sample gate of the pulsed wave Doppler should be placed at the point of maximum color near the bifurcation. When the waveform is visualized, the frequency range is manipulated on the apparatus until the waveform takes about 2/3rd of the height of the screen.[25] Waveform itself will have a range of frequencies, depicted by a range of different colors within it. If the waveform acquired appears very bright, contains less colors and the background is noisy, then the Doppler gain is lessened until the proper balance is acquired[26]

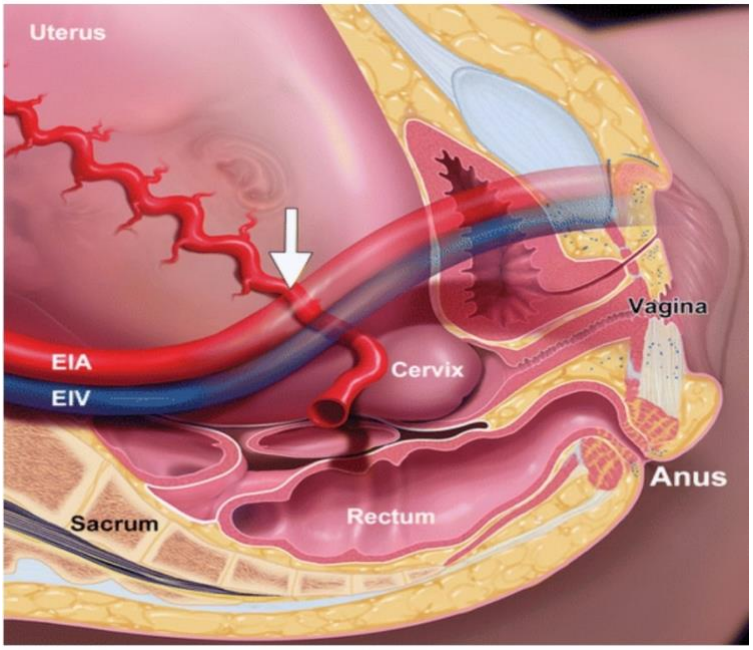


Fig 5: **Ilustrating location of uterine artery at the cervico-isthmic junction**.

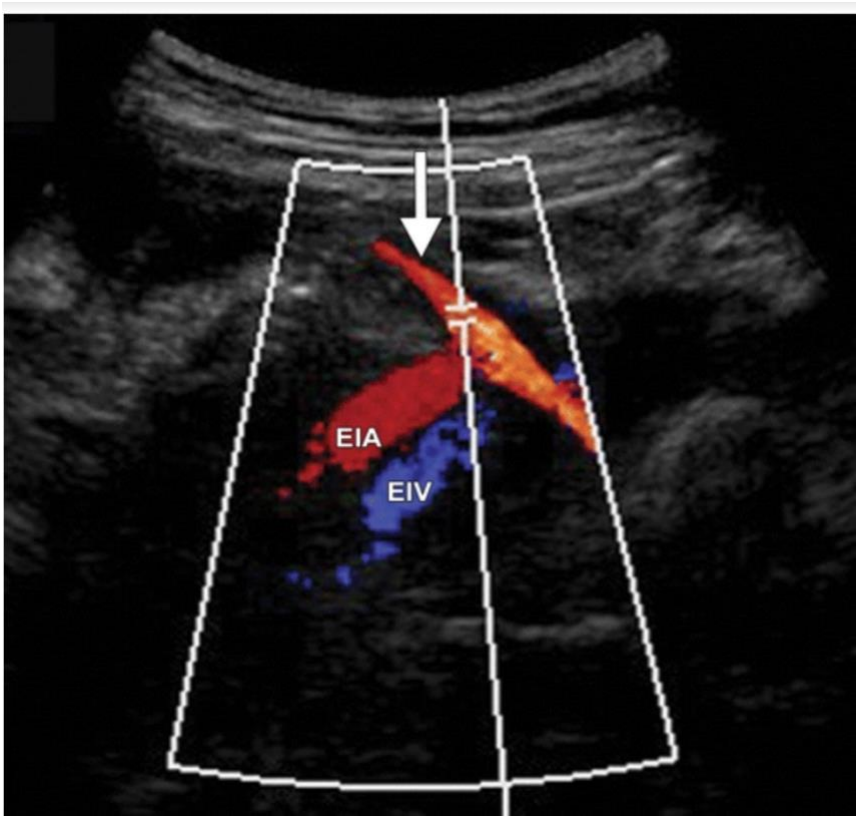


Fig 6 **Color Doppler flow showing sample volume placed in the uterine artery**

Taking measurements

After getting an ideal waveform, the image has to be freezed, then the automatic computation are presented. The 3 waves that the equipment has taken should have less noise. If the equipment Isn't having a frequency follower, then the image is freezed and Doppler values are taken manually. Variety of measurements of the uterine artery waveform can be studied, generally used is pulsatility index (PI), resistance index (RI) & systolic/diastolic (S/D) Ratio.[27]

Figure 7 **Relationship between uterine artery flow velocity waveform and various Doppler indices (RI, PI, Systolic peak B, end diastole ,Vm, mean velocity, Start of Diastole, D, Maximum diastole)**

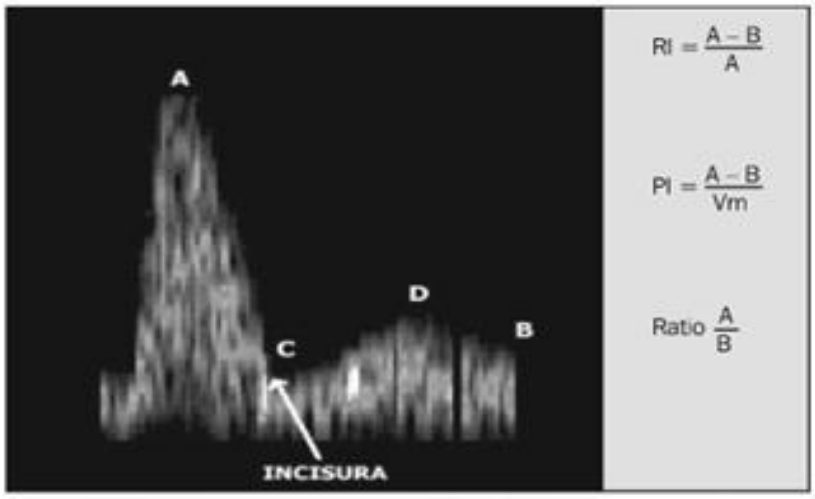


Figure 8 **Doppler indices**

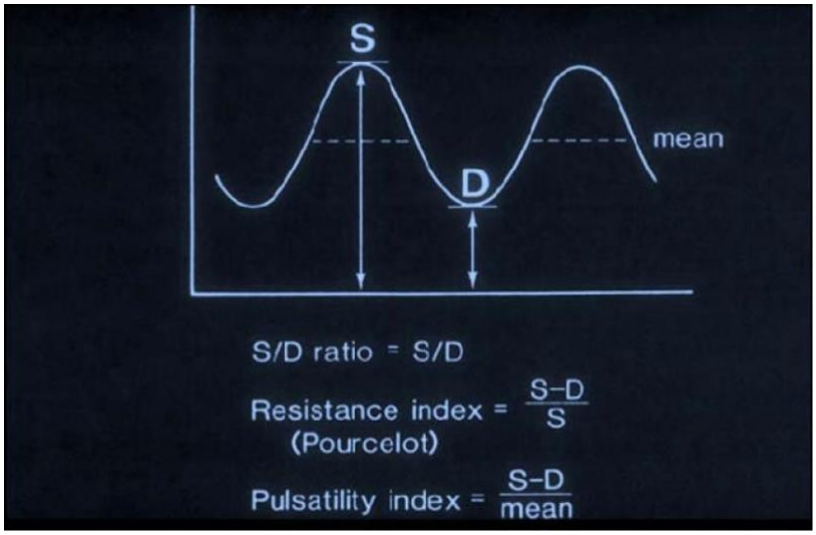
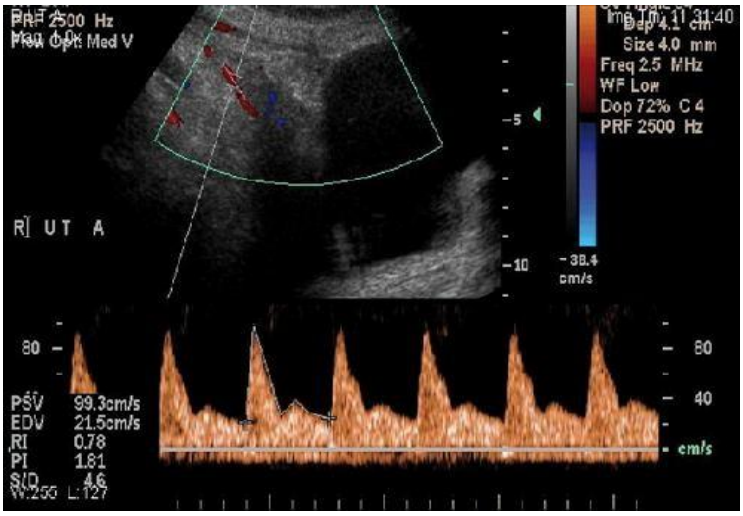


Figure 9 **Uteroplacental waveforms**



How to report utero-placental waveforms[28]

We recommend that you describe them as follows:

High resistance pattern: Mean RI > 0.55 with bilateral notches or Mean RI > 0.65 with unilateral notch.

Low resistance pattern: all other conditions.

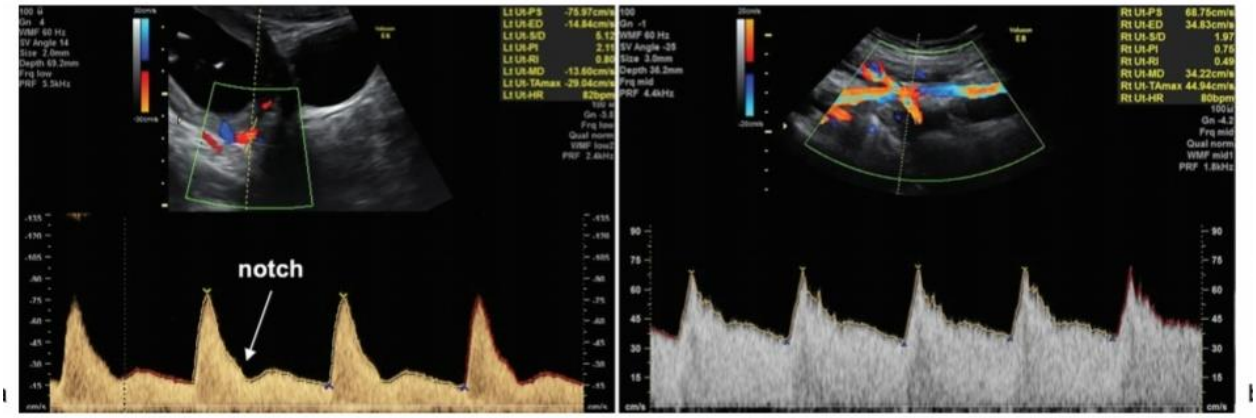


FIGURE 10 – **Doppler waveforms of the uterine arteries showing a high resistance pattern with notching between the systole and the diastole (a) characteristic of the 1st trimester, and a low resistance pattern (b) characteristic of the 2nd trimester.**

Problems

If a signal is not visualized, check the machine settings. Restart the signal acquisition process. The wall filter of vessel, sweep speed, frequency range and gain controls should be reevaluated.

Problem in differentiating waveforms from the internal iliac artery from pathological Utero-placental waveforms (UPW):Pathologic UPW have a biphasic deceleration decline in systole & internal iliac artery have a steep smooth decline.[29]

METHODOLOGY

This is a prospective study conducted at Shri B.M Patil medical college, hospital and research Centre over a period of 2 years from October 2018 to July 2020. 105 women with singleton pregnancy attending ANC were evaluated for uterine artery Doppler with biometry &morphology.

Study design:

Present study is a prospective cohort study to see the usefulness of uterine artery Doppler in predicting pre-eclampsia.

METHOD OF COLLECTION OF DATA:

Sample size

With anticipated Proportion of preeclampsia 24.54% [(ref)] the minimum sample size is 73 patients with 5% level of significance and 10% absolute error.

Formula used

$$n=\frac{z^2\ p*q}{d^2}$$

Where Z= Z statistic at α level of significance

d^2= Absolute error

P= Proportion rate

q= 100-p

Inclusion criteria

All the pregnant women of 14-20 weeks of Gestation.[30]

Exclusion criteria

1. Those with multiple gestations, congenital abnormality of fetus, renal disease, chronic hypertension & cardiac diseases.

2. Eliminate patient who are not delivered at Shri. B.M.Patil Medical College Hospital and Research Centre, Vijayapur.

3. Eliminate patients with unreliable last menstrual period details and not confirmed by 1st trimester ultrasound.

When above criteria are reached, study group underwent Doppler study.

Procedure

Information is collected by SIEMENS ACUSON X700 and PHILIPS HD11-XE machines

The fetal biometry and morphology scan is done; later Doppler mode is switched on. Patient is asked to lie in supine position with transducer in the longitudinal plane. External iliac artery is visualized with color Doppler.[31] The probe is then angulated medially towards the tortuous uterine artery. The right and left uterine arteries spectral waveforms are taken, when three or four waves of same height were visualized, the image was frozen and calculations taken by automatic trace /manually/ trace method.

The findings are noted down.

PI, RI, S/D ratio and early diastolic notching are noted.

The flow velocity waveforms were taken as abnormal if they had persistent diastolic notch in one of the uterine arteries (Rt/Lt), S/D, PI and RI exceeds 95thpercentile of the reference range for that population.

A followed up till delivery with details of pregnancy, labor and neonatal outcomes were noted.

The abnormal outcomes were defined as:

a) Pre-eclampsia is defined as:

Hypertension that is, ≥ 140/90mm Hg of BP documented at least on 2 occasions, four hours apart or diastolic pressure ≥ 110 mm of Hg.

Proteinuria of ≥1+ albumin as recorded by dipstick method.[32]

b) IUGR is defined as < 10thcentile of birth rate of that gestational age.[33]

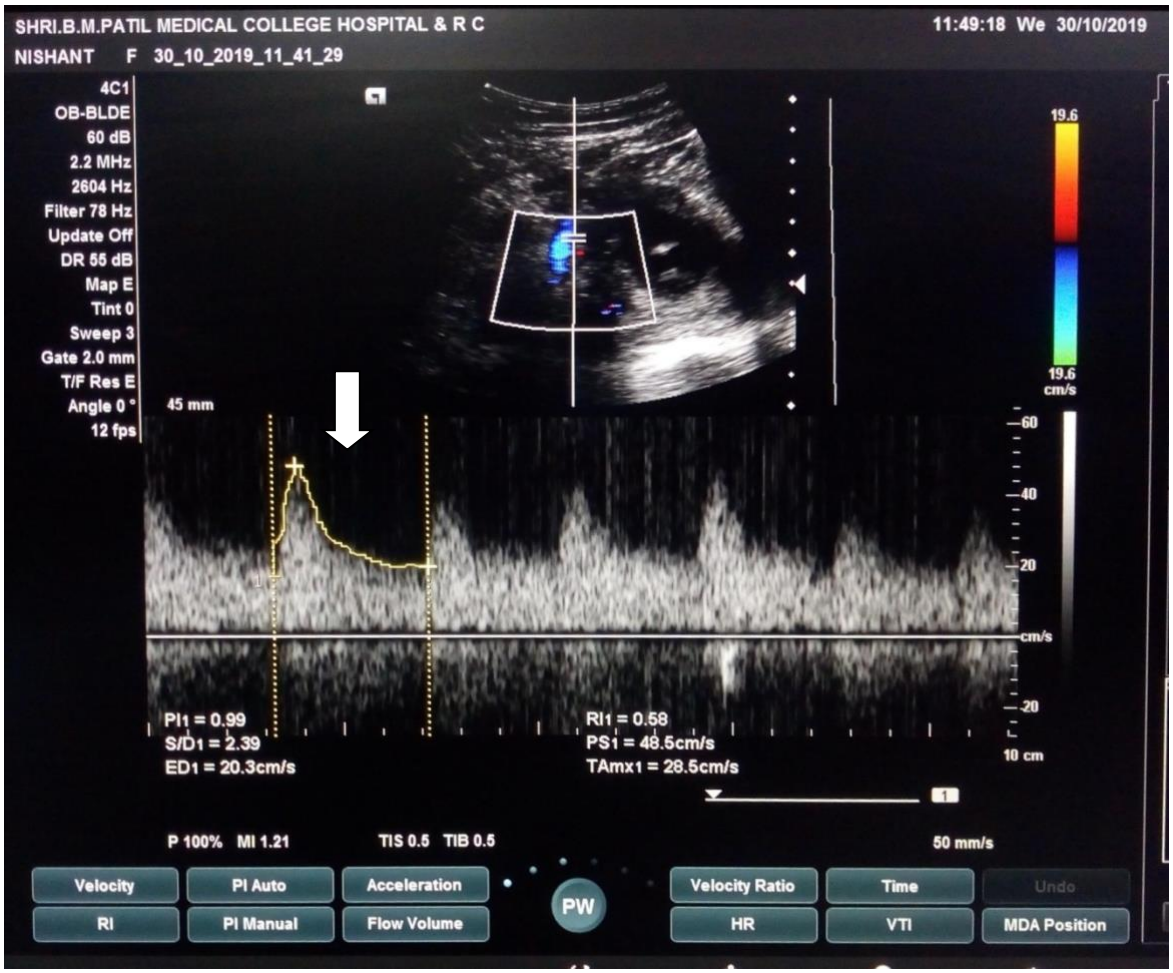


Fig 11: Uterine Artery Doppler showing normal uterine artery wave form (arrow)

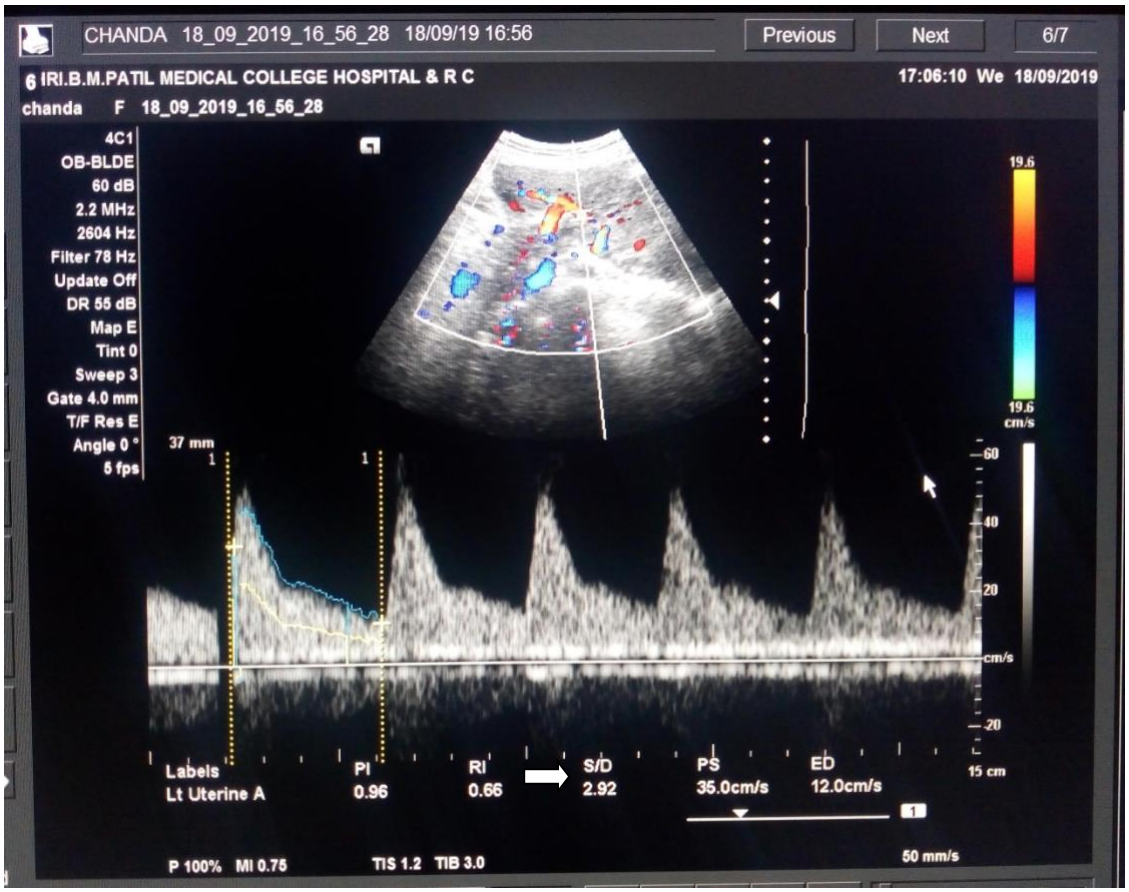


Fig 12: Uterine Artery Doppler with Increased S/D ratio – 3.49 (arrow).

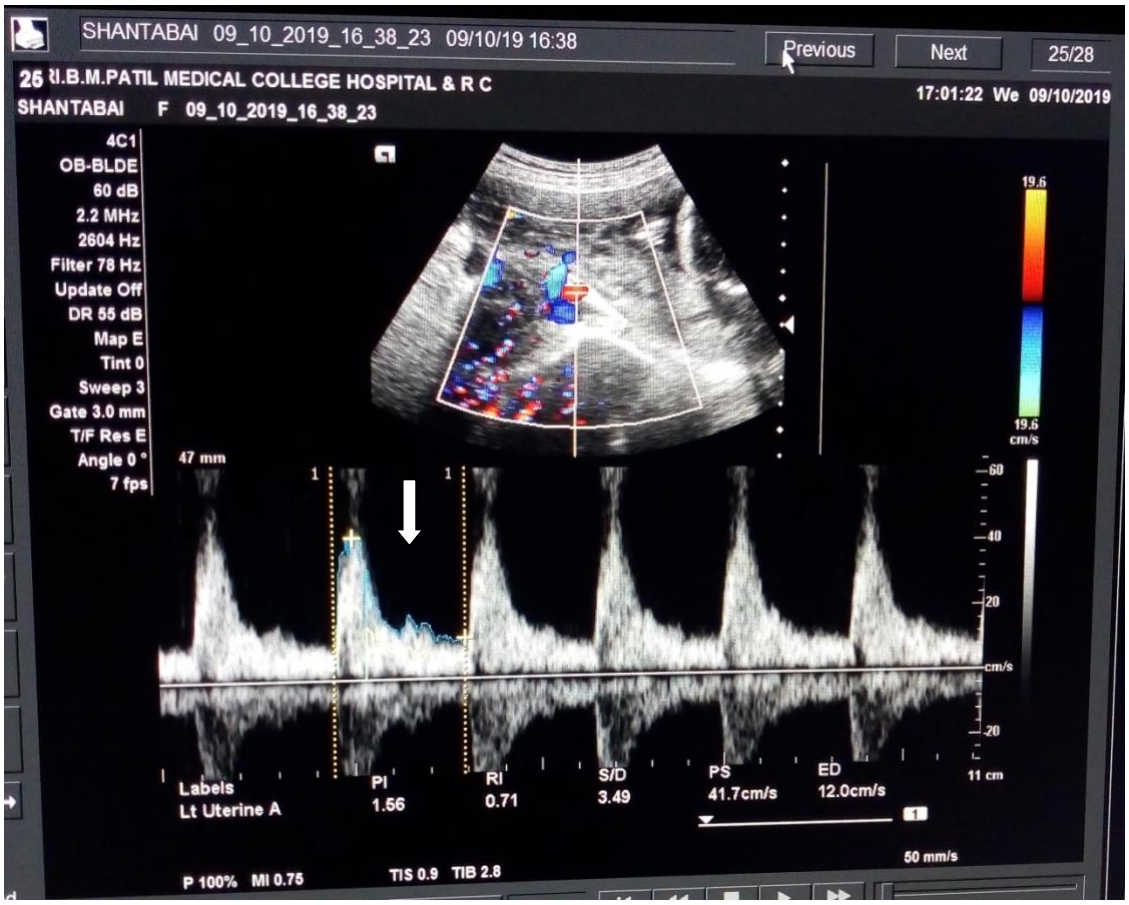


Fig13: Uterine Artery Doppler with Increased S/D ratio – 3.49, increased PI – 1.56 and Increased RI – 0.71

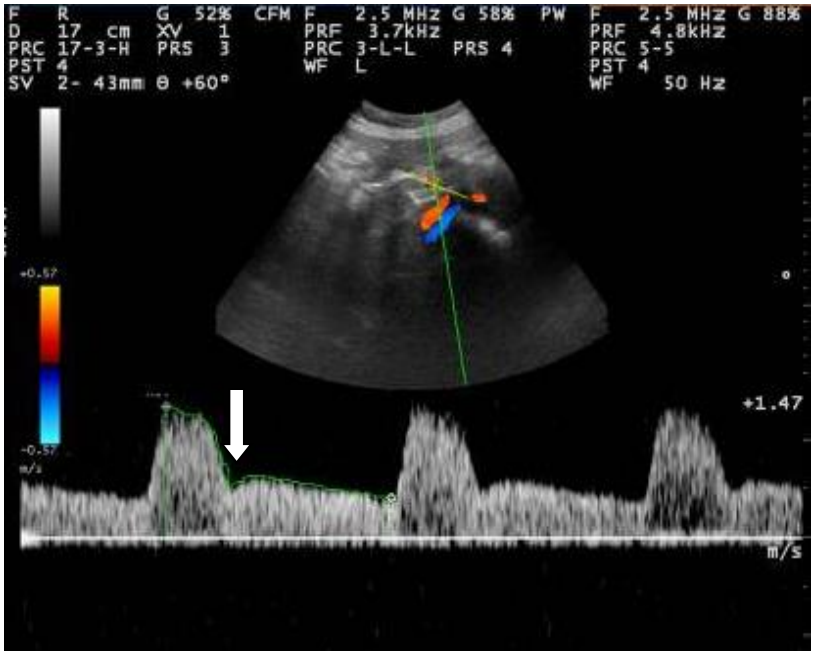
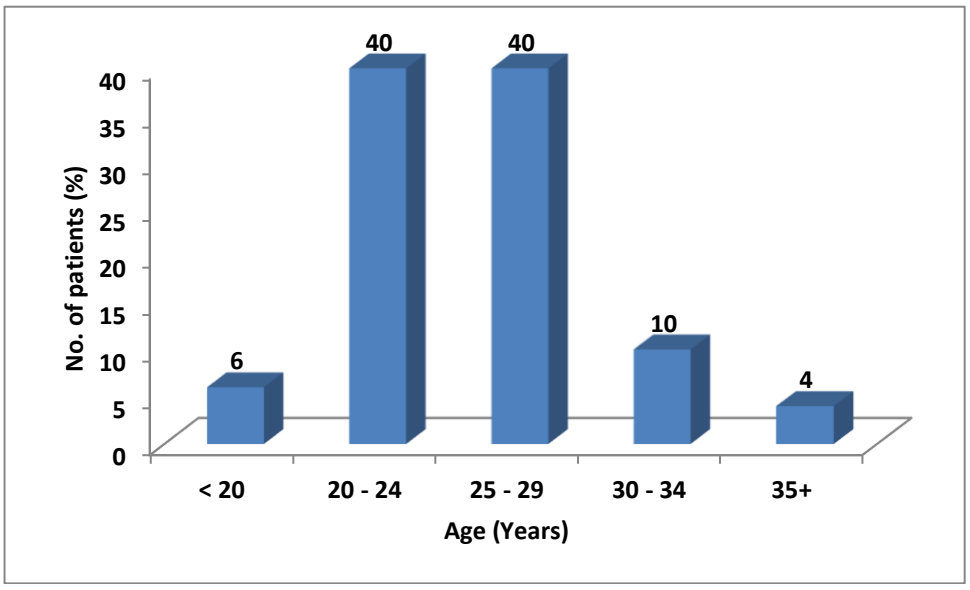


Fig: 14: Uterine Artery Doppler showing Early Diastolic Notch (arrow).

RESULTS AND OBSERVATIONS

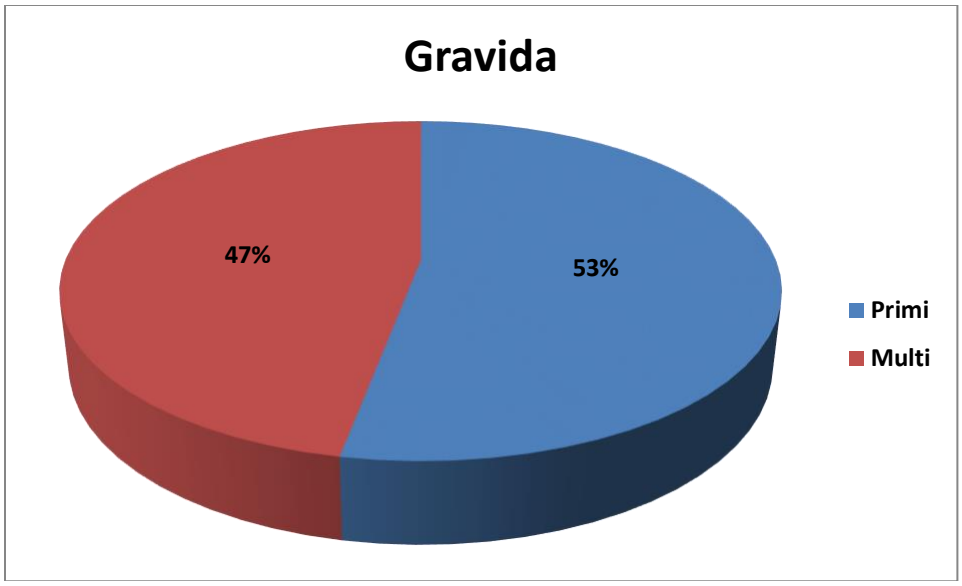
Distribution of patients according to Age (Years)

Age(Years)	No. of patients (%)	Percentage
< 20	6	6.0
20 - 24	40	40.0
25 - 29	40	40.0
30 - 34	10	10.0
35+	4	4.0
Total	100	100.0



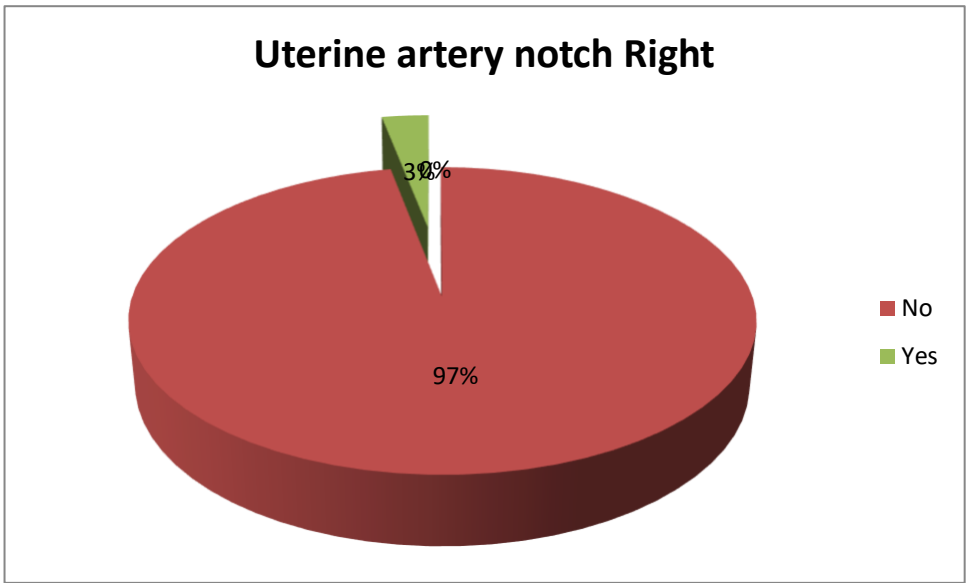
Distribution of patients according to Gravid

Gravid	No. of patients (%)	Percentage
Primi	53	53.0
Multi	47	47.0
Total	100	100.0



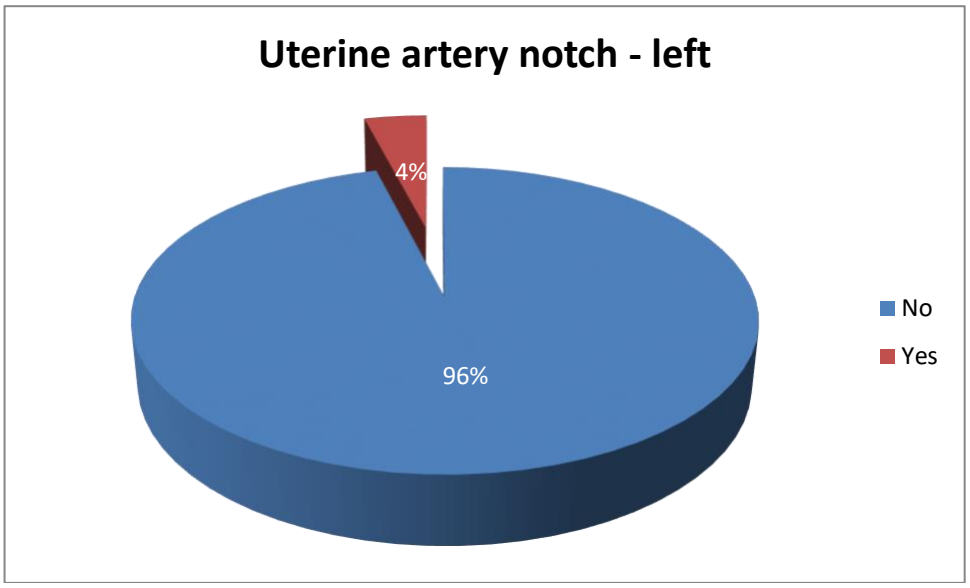
Distribution of patients according to uterine artery notch right

Uterine artery notch Right	No. of patients (%)	Percentage
No	97	97.0
Yes	3	3.0
Total	100	100.0



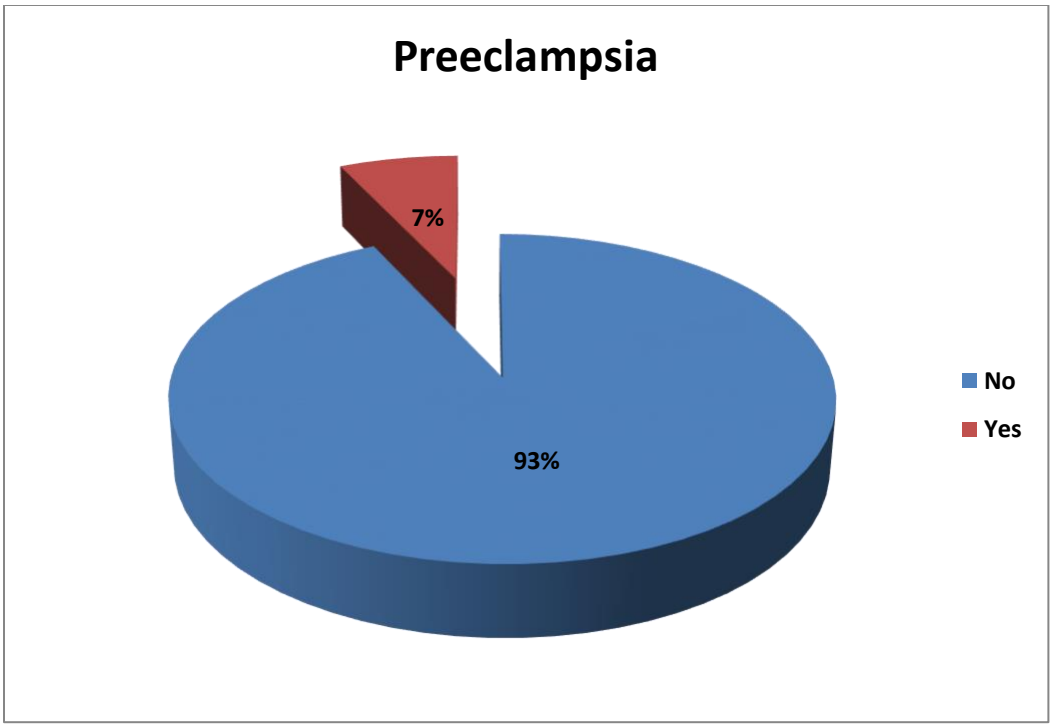
Distribution of patients according to uterine artery notch left

Uterine artery notch left	No. of patients (%)	Percentage
No	96	96.0
Yes	4	4.0
Total	100	100.0



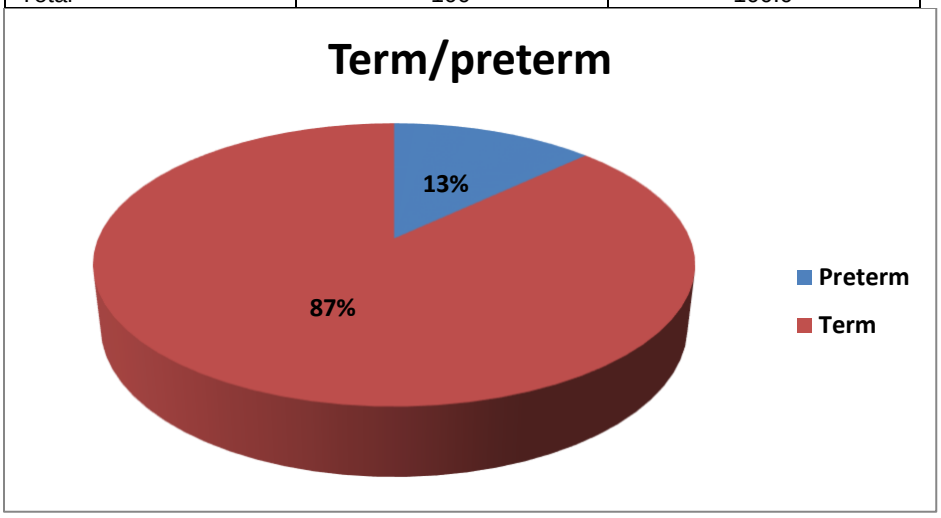
Distribution of patients according to Preeclampsia

Preeclampsia	No. of patients (%)	Percentage
No	93	93.0
Yes	7	7.0
Total	100	100.0



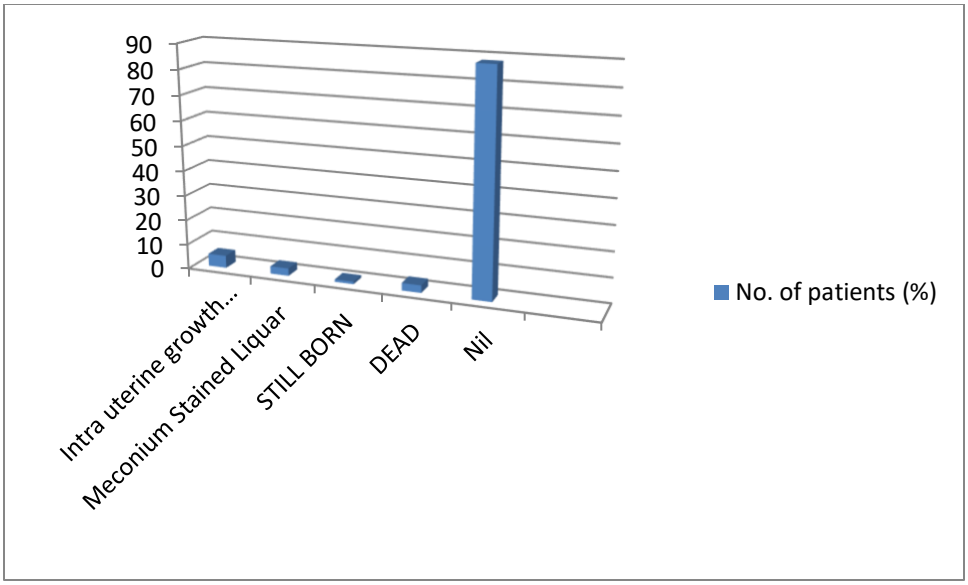
Distribution of patients according to Term/Preterm

Term/preterm	No. of patients (%)	Percentage
Preterm	13	13.0
Term	87	87.0
Total	100	100.0



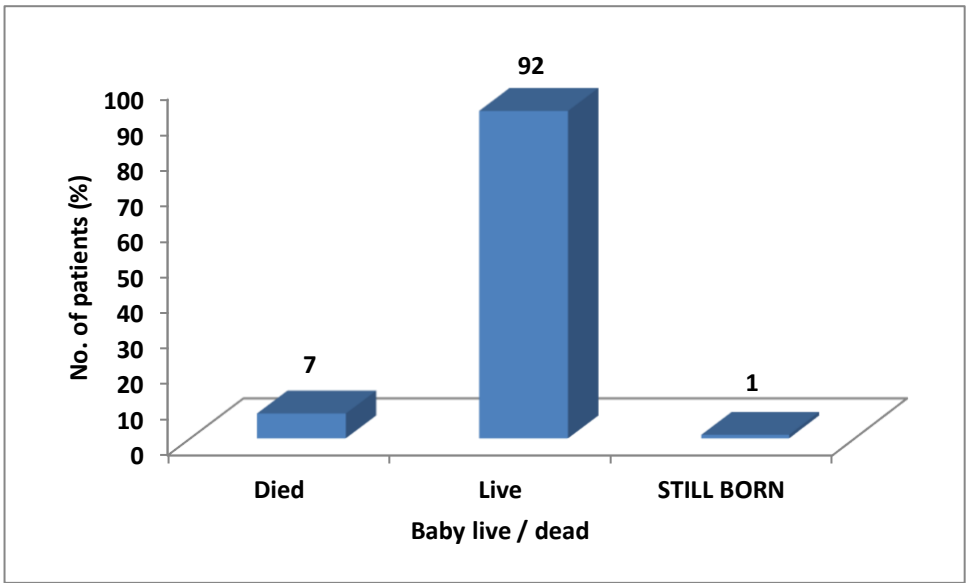
Distribution of patients according to complications

Complications	No. of patients (%)	Percentage
Intra uterine growth restriction	5	5.0
Meconium Stained Liquor	3	3.0
STILL BORN	1	1.0
DEAD	3	3.0
Nil	88	88.0
Total	100	100.0



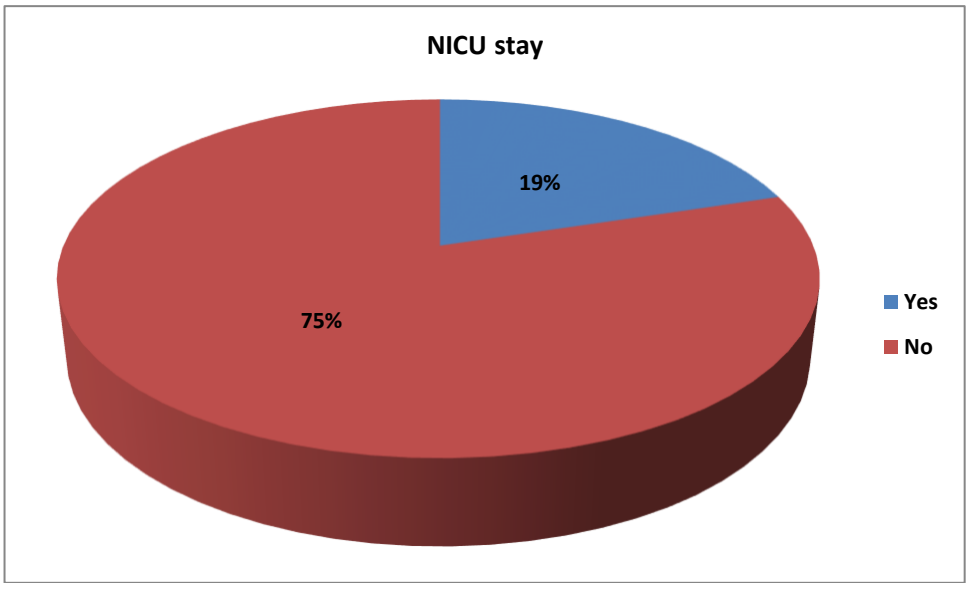
Distribution of patients according to baby Live / Dead/Stillborn

Baby live / dead	No. of patients (%)	Percentage
Died	7	7.0
Live	92	92.0
STILL BORN	1	1.0
Total	100	100.0



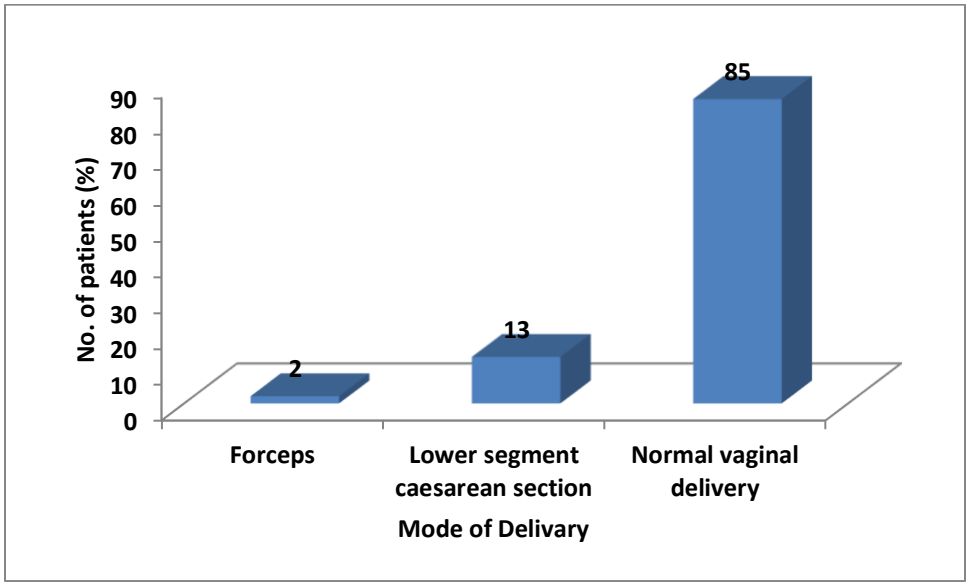
Distribution of patients according to Stay in NICU

Stay in NICU	No. of patients (%)	Percentage
Yes	19	19.0
No	75	75.0
Total	94	100.0

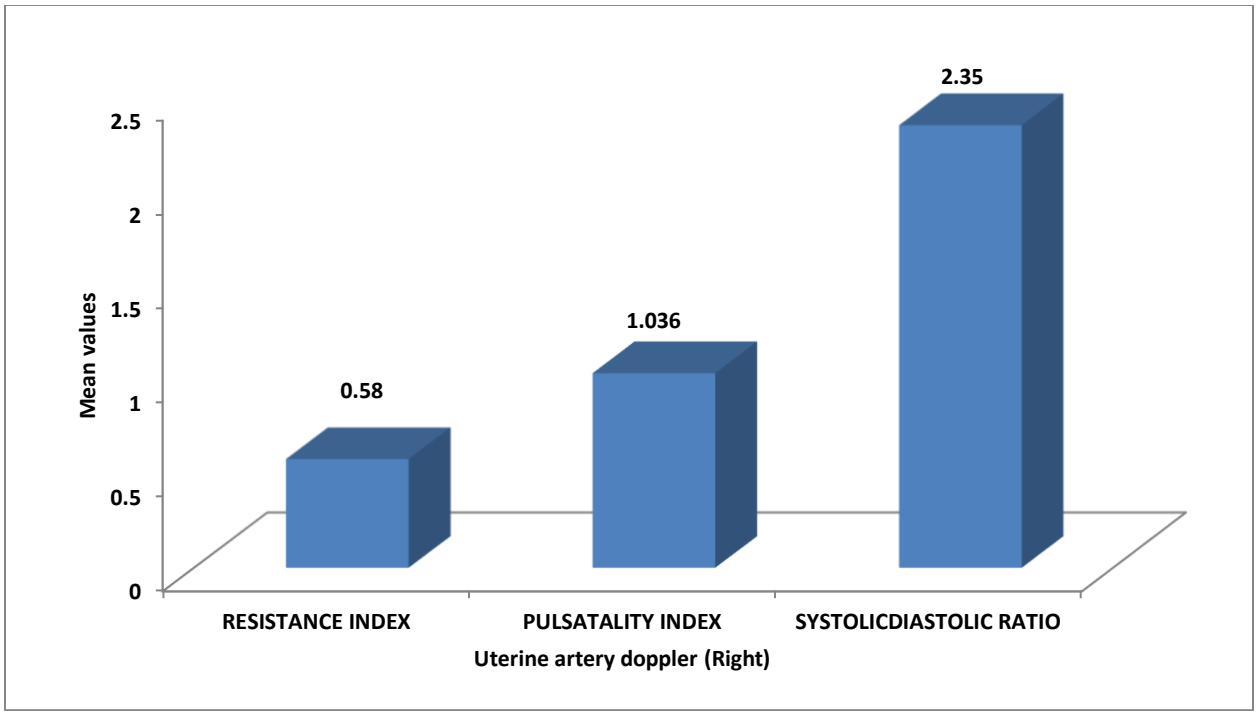


Distribution of patients according to Mode of Delivery

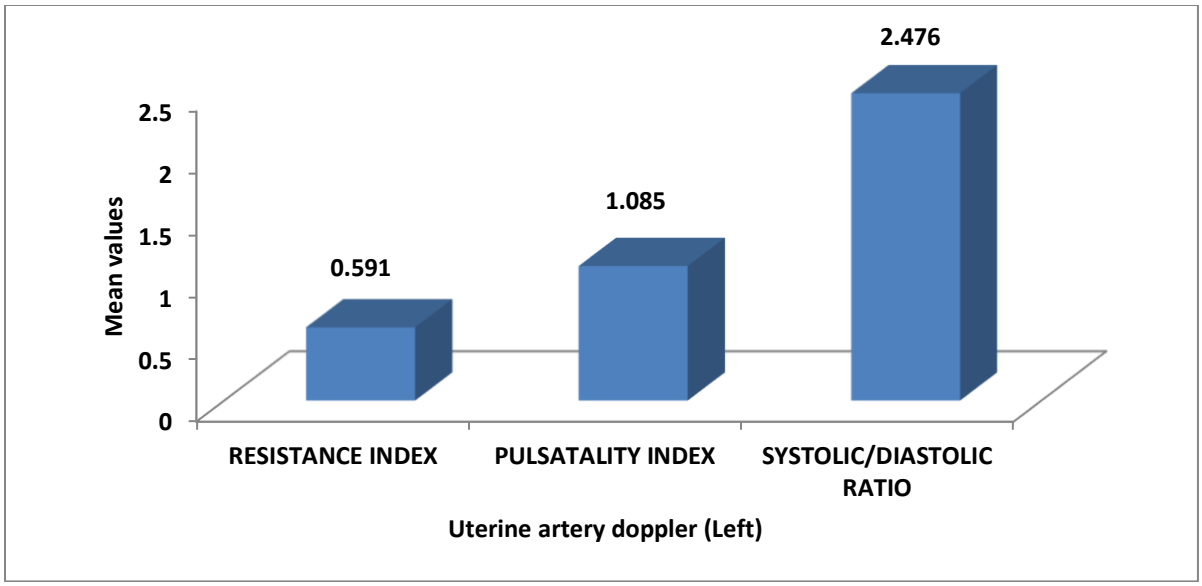
Mode of Delivery	No. of patients (%)	Percentage
Forceps	2	2.0
Lower segment caesarean section	13	13.0
Normal vaginal delivery	85	85.0
Total	100	100.0



Uterine artery Doppler (Right)	Minimum	Maximum	Mean	SD	95th Percentile
RESISTANCE INDEX	0.40	1	0.58	0.096	0.76
PULSATALITYINDEX	0.57	2.66	1.036	0.315	1.72
SYSTOLIC/DIASTOLICRATIO	1.70	6.40	2.35	0.735	4.456

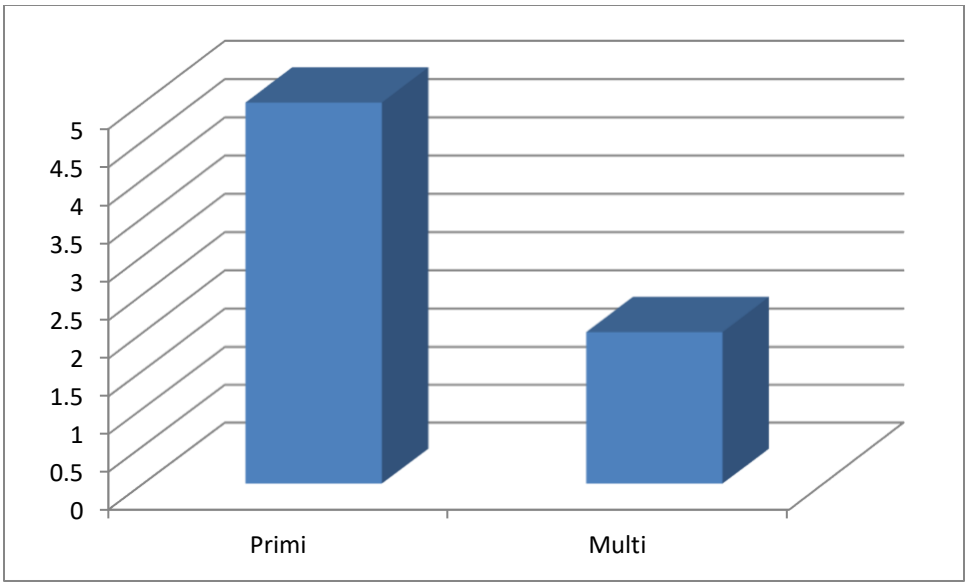


Uterine artery Doppler (Left)	Minimum	Maximum	Mean	SD	95th Percentile
RESISTANCE INDEX	0.45	0.89	0.591	0.101	0.80
PULSATALITY INDEX	0.62	2.90	1.085	0.346	1.600
SYSTOLIC/DIASTOLIC RATIO	1.56	7.20	2.476	1.053	5.00



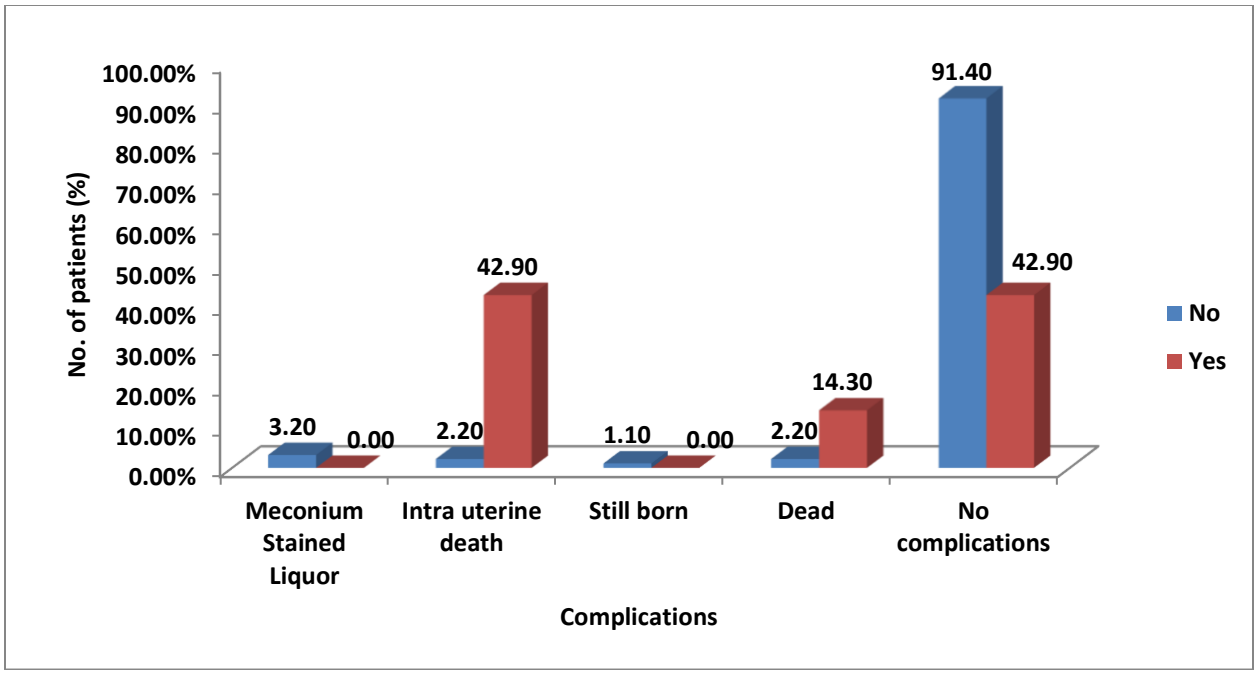
Association between Gravid and Preeclampsia

Gravid	Preeclampsia		
	No	Yes	Total
Primi	48	5	53
Multi	45	2	47
	93	7	100



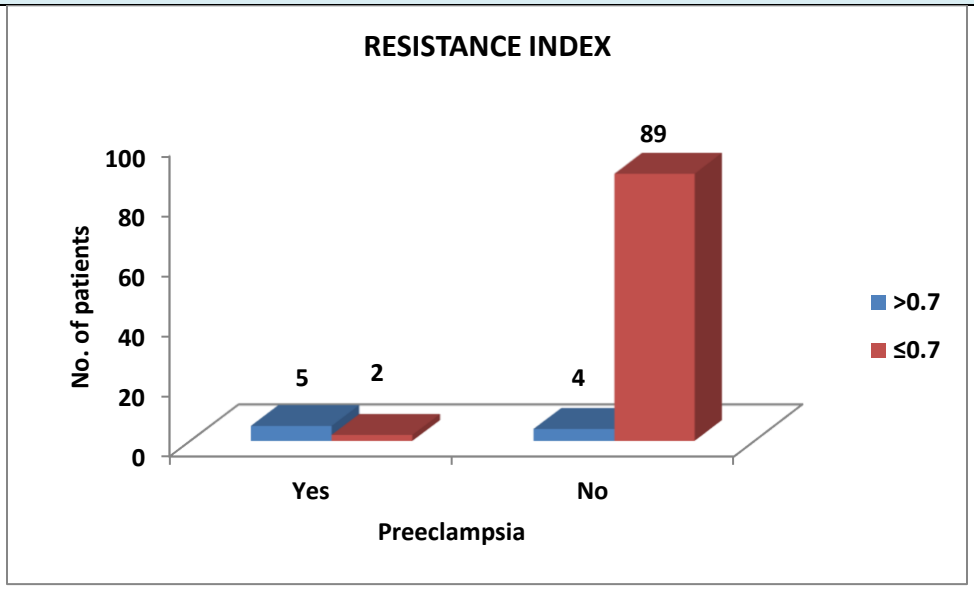
NICU Stay	Preeclampsia			Chi square test	P value
	No	Yes	Total		
No	77	4	75	X^2=5.433	P=0.143
%	77%	4%	75.0%		
Yes	16	3	19		
%	16%	3%	19.0%		
Total	93	7	100		

Complications	Preeclampsia			Chi square test	P value
	No	Yes	Total		
Meconium Stained Liquor	3	0	3	X^2=26.851	P=0.001*
%	3.2%	0.0%	3.0%		
Intra uterine death	2	3	5		
%	2.2%	42.9%	5.0%		
Still born	1	0	1		
%	1.1%	0.0%	1.0%		
Dead	2	1	3		
%	2.2%	14.3%	3.0%		
No complications	85	3	88		
%	91.4%	42.9%	88.0%		
Total	93	7	100		
*:Highly significant					



Association between RI (Right) and Preeclampsia

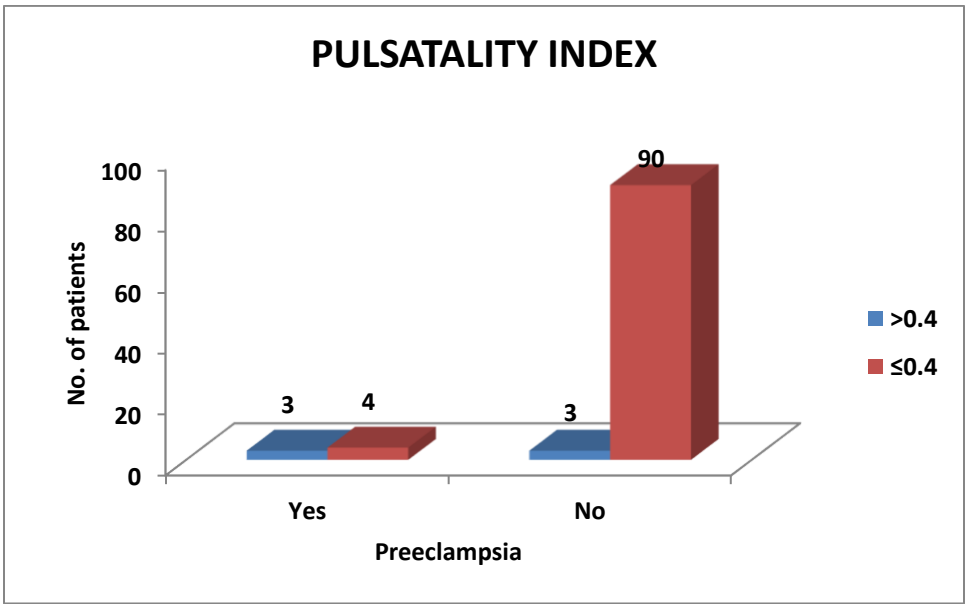
RI >0.7	Preeclampsia			Fisher's Exact test	P value
	Yes	No	Total		
Yes	5	4	9	P<0.0001*	P=0.143
No	2	89	91		
Total	7	93	100		
*:Highly significant					



Diagnostic test	
Sensitivity	71%
Specificity	95%
Positive Predictive value	55%
Negative predictive value	97%

Association between PI (Right) and Preeclampsia

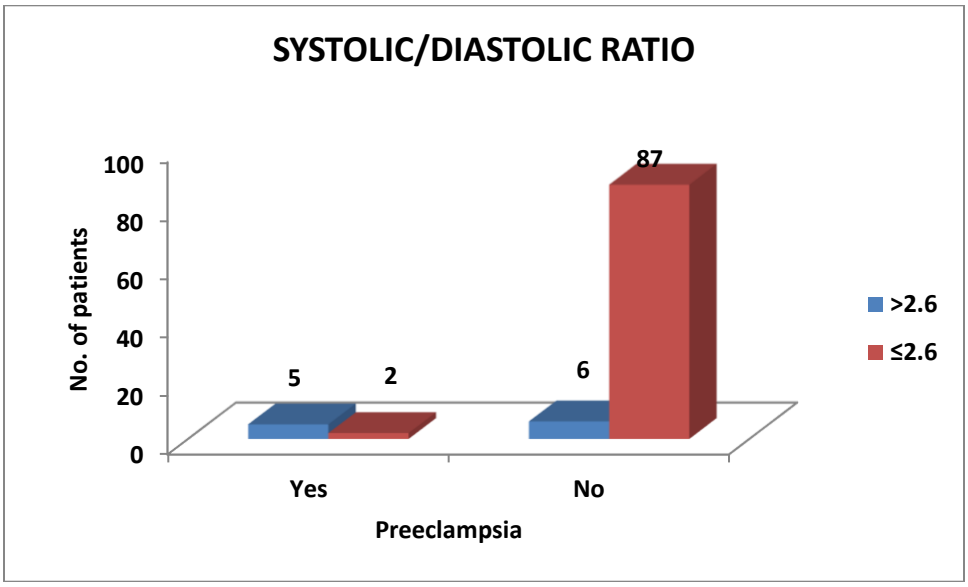
PI >1.4	Preeclampsia			Fisher's Exact test
	Yes	No	Total	
Yes	3	3	6	P<0.0039*
No	4	90	94	
Total	7	93	100	
*:Highly significant				



Diagnostic test	
Sensitivity	43%
Specificity	97%
Positive Predictive value	50%
Negative predictive value	95%

Association between S/D (Right) and Preeclampsia

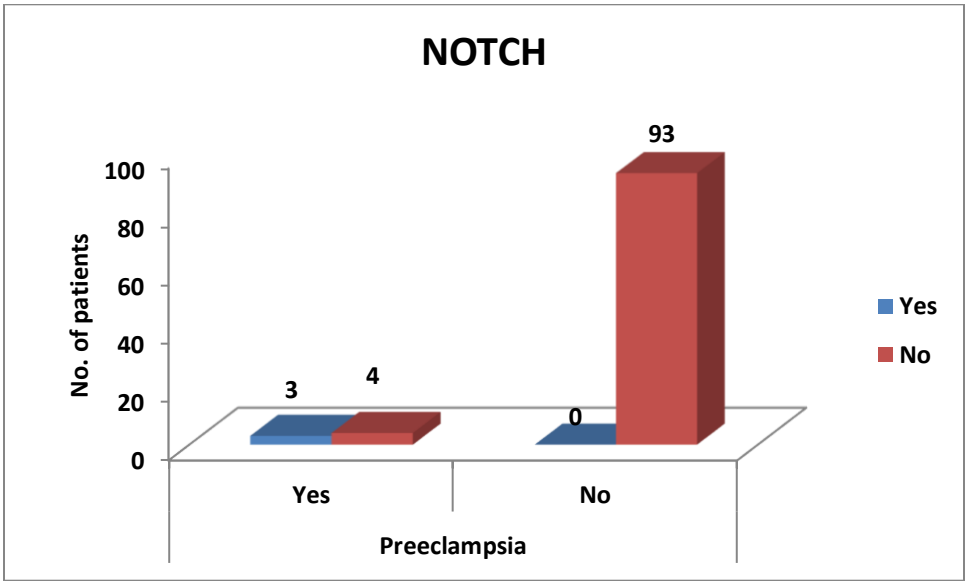
S/D >2.6	Preeclampsia			Fisher's Exact test
	Yes	No	Total	
Yes	5	6	11	P=0.0001*
No	2	87	89	
Total	7	93	100	
*:Highly significant				



Diagnostic test	
Sensitivity	71%
Specificity	94%
Positive Predictive value	45%
Negative predictive value	97%

Association between NOTCH (Right) and Preeclampsia

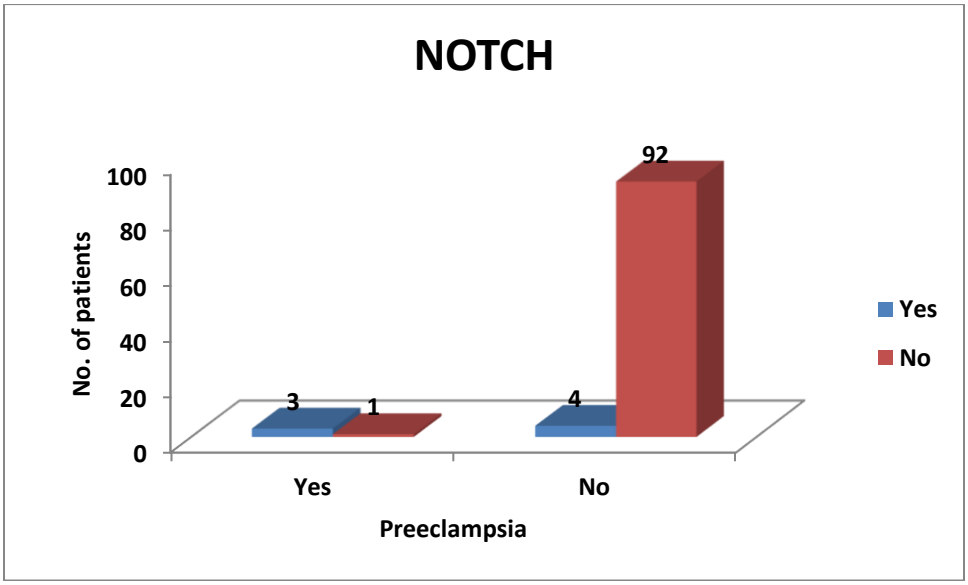
NOTCH	Preeclampsia			Fisher's Exact test
	Yes	No	Total	
Yes	3	0	3	P=0.0001*
No	4	93	97	
Total	7	93	100	
*:Highly significant				



Diagnostic test	
Sensitivity	43%
Specificity	100%
Positive Predictive value	100%
Negative predictive value	95%

Association between NOTCH (Left) and Preeclampsia

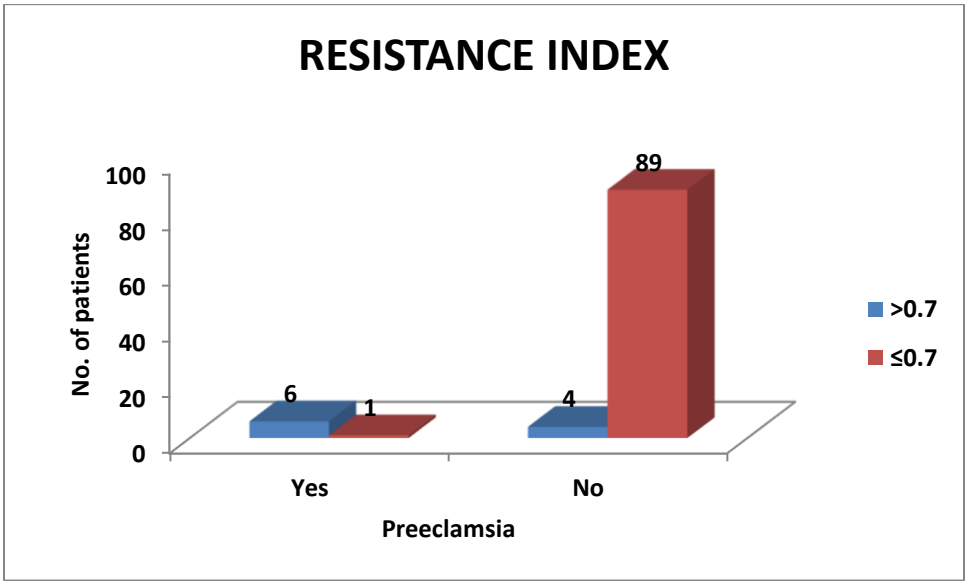
NOTCH	Preeclampsia			Fisher's Exact test
	Yes	No	Total	
Yes	3	1	4	P=0.0001*
No	4	92	96	
Total	7	93	100	
*:Highly significant				



Diagnostic test	
Sensitivity	43%
Specificity	98%
Positive Predictive value	755%
Negative predictive value	95%

Association between RI (Left) and Preeclampsia

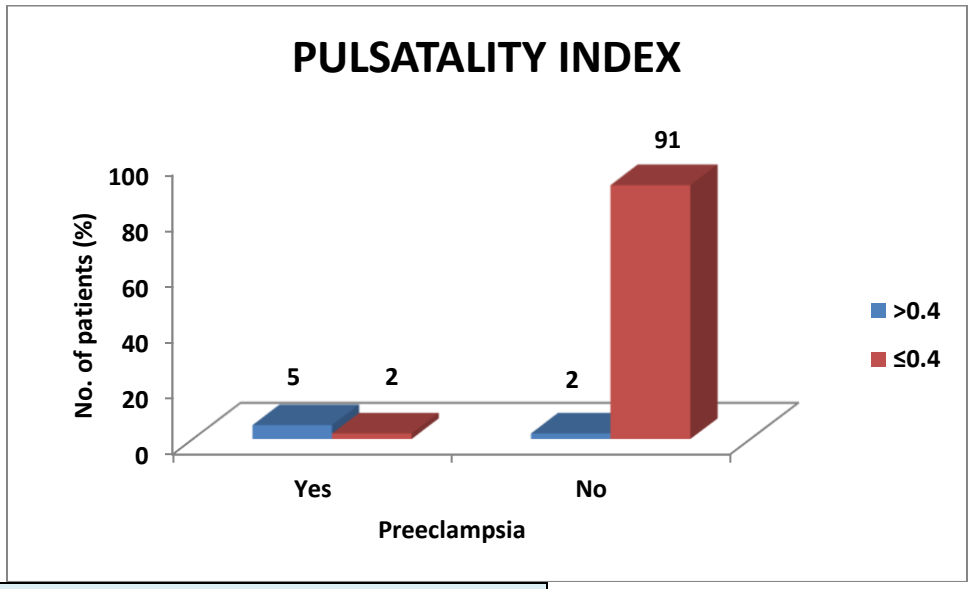
RI >0.7	Preeclampsia			Fisher's Exact test	P value
	Yes	No	Total		
Yes	6	4	10	P<0.0001*	P=0.143
No	1	89	90		
Total	7	93	100		
*:Highly significant					



Diagnostic test	
Sensitivity	85%
Specificity	95%
Positive Predictive value	60%
Negative predictive value	98%

Association between PI (Left) and Preeclampsia

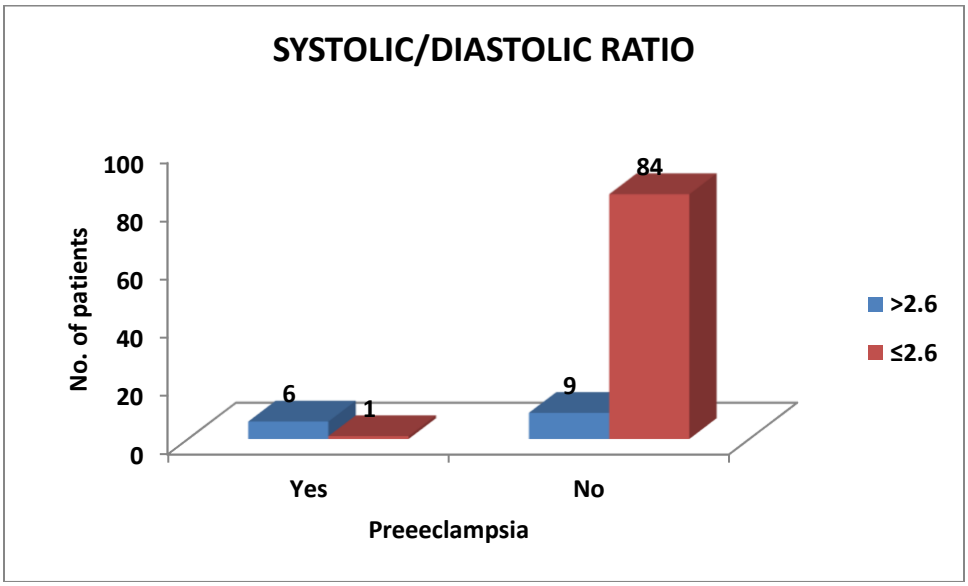
PI >1.4	Preeclampsia			Fisher's Exact test
	Yes	No	Total	
Yes	5	2	7	P<0.001*
No	2	91	93	
Total	7	93	100	
*:Highly significant				



Diagnostic test	
Sensitivity	71%
Specificity	97%
Positive Predictive value	71%
Negative predictive value	97%

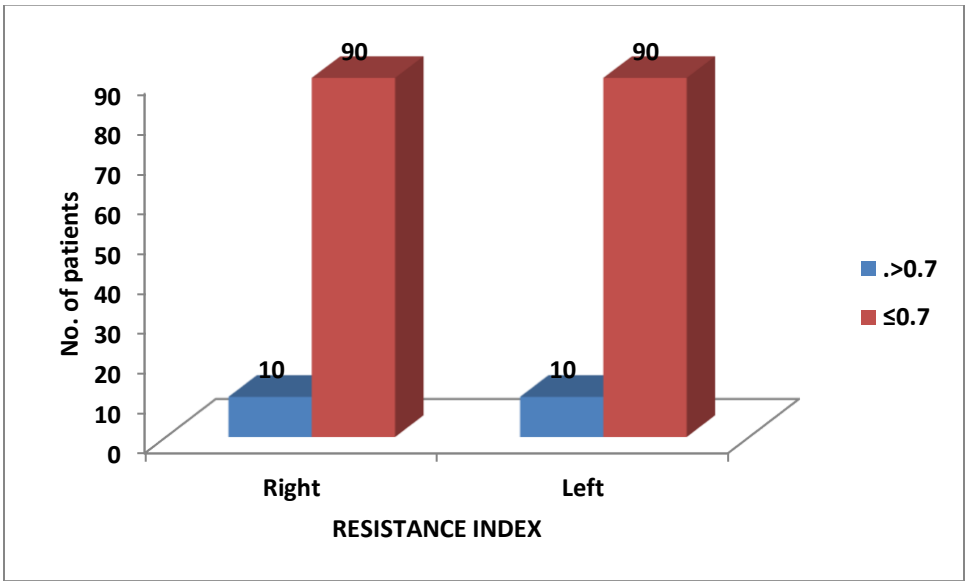
Association between S/D (Left) and Preeclampsia

S/D >2,6	Preeclampsia			Fisher's Exact test
	Yes	No	Total	
Yes	6	9	11	P=0.0001*
No	1	84	89	
Total	7	93	100	
*:Highly significant				

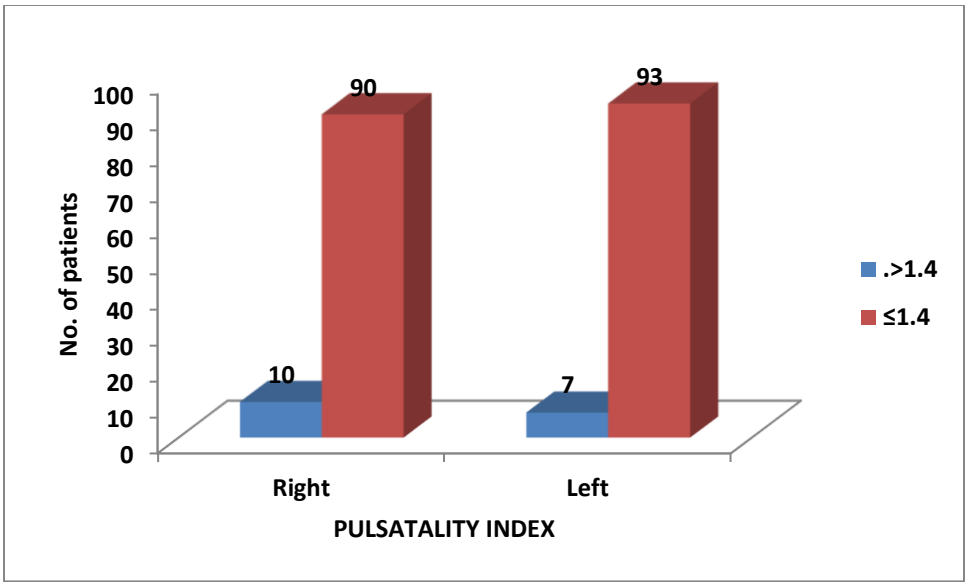


Diagnostic test	
Sensitivity	85%
Specificity	90%
Positive Predictive value	40%
Negative predictive value	98%

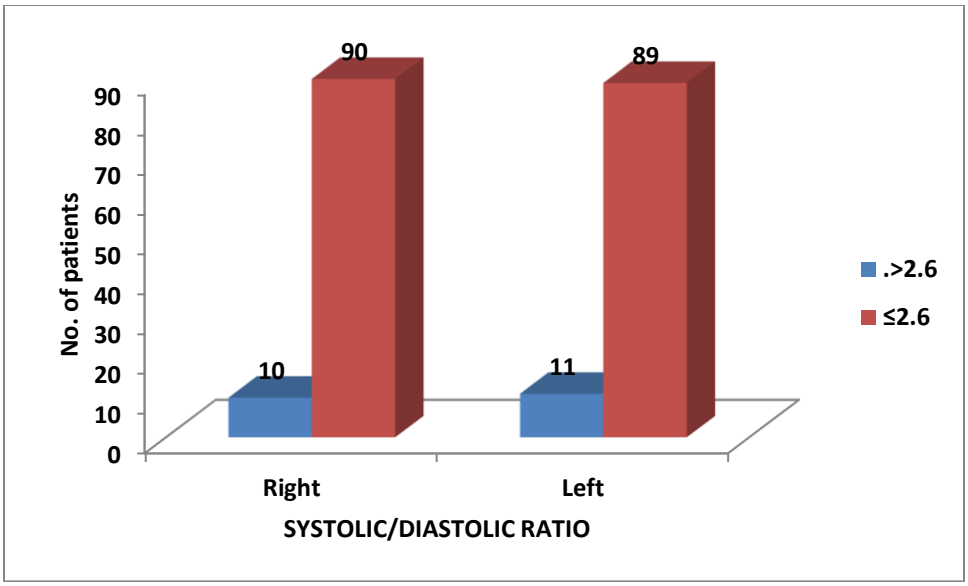
RI	Right		Left	
	No. of patients	%	No. of patients	%
.>0.7	9	10	10	10
≤0.7	91	90	90	90
Total	100	100	100	100



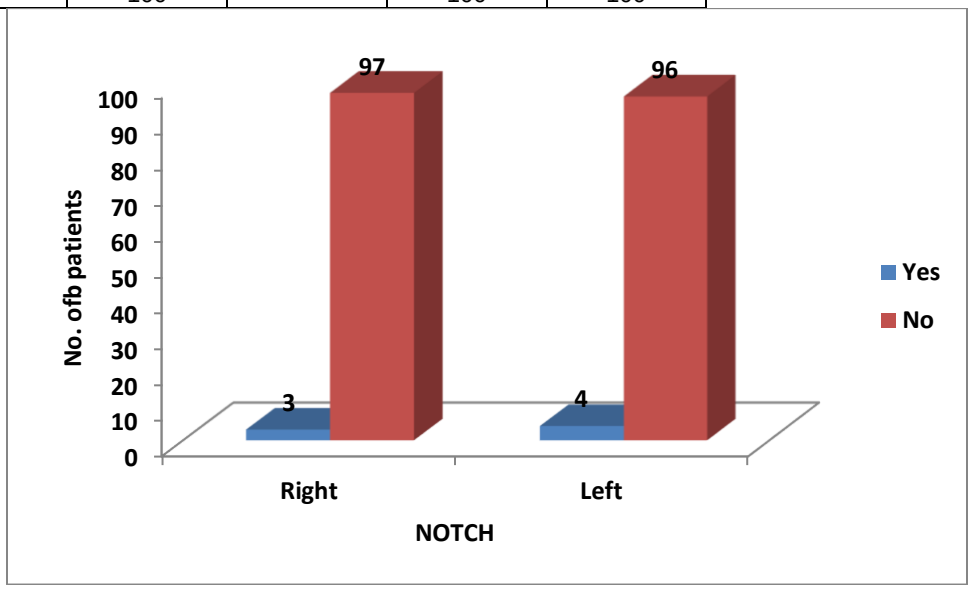
Pi	Right		Left	
	No. of patients	%	No. of patients	%
.>1.4	6	10	7	7
≤1.4	94	90	93	93
Total	100	100	100	100



S/D	Right		Left	
	No. of patients	%	No. of patients	%
.>2.6	11	10	11	11
≤2.6	89	90	89	89
Total	100	100	100	89



NODCH	Right		Left	
	No. of patients	%	No. of patients	%
Yes	3	3	4	4
No	97	97	96	96
Total	100	100	100	100



DISCUSSION

In this prospective study conducted in B.L.D.E.U's Shri B. M. Patil Medical College Hospital and Research Centre, Vijaypura for 2 years, the outcomes of the study, the predictive values of various Doppler indices have been discussed.

INCIDENCE AND PREVALANCE

The incidence of Preeclampsia is 7%, the incidence of IUGR is 5%.

AGE DISTRIBUTION

In this study most of the cases are between 21 to 29 years of age

PARITY

In our study most of the patients are primigravida

SOCIOECONOMIC STATUS

81% belong to SES- V

PROTEINURIA

In 100 patients 7 had albuminuria

PLACENTALPOSITION

60% Centrally Located, 40%Unilateral

TYPE OF DELIVERY

Among 9 Patients with abnormal Doppler indices, 3 of them Delivered vaginally & 4 of them were induced.

1 Patient underwent Emergency LSCS

1 Patient underwent Elective LSCS

GA at the time of delivery:

Mean Gestational at the time of delivery age 38.62 weeks

Out of 7 patients who had pre-eclampsia, 4 babies were Pre term (57%).

Stay in NICU 19%

UTERINE ARTERY DOPPLER INDICES IN PREECLAMPSIA

Among the 100 patients studied there were 9 patients with abnormal uterine artery Doppler when 95th percentile was taken as cut off. Among them 10 Patients had abnormal right S/D ratio, 9 patients had abnormal left S/D ratio, 16 patients had abnormal right RI, 9 patients had abnormal Right RI, 10 patients had abnormal Left RI, 9 patients had abnormal Right PI, 8 patients had abnormal Left PI, 3 patients had early diastolic notch on right side, 4 patients had early diastolic notch on left side.

Sensitivity and specificity of abnormal uterine Doppler in Preeclampsia

Out of these 9 Patients with abnormal Doppler, 7 patients developed Preeclampsia with a sensitivity of 71% & 85% for uterine S/D (Rt, Lt) ratio, specificity of 97 % & 90% for uterine S/D (Rt, Lt).

Sensitivity, Specificity, Positive Predictive value & Negative Predictive Value of RI (Rt, Lt) are 71%, 95%, 55%, 97% & 85%, 95%, 60%, 98% respectively.

Sensitivity, Specificity, Positive Predictive value & Negative Predictive Value of PI (Rt, Lt) are 43%, 97%, 50%, 95% & 71%, 97%, 71%, 97% respectively.

Sensitivity, Specificity, Positive Predictive value & Negative Predictive Value of Notch (Rt, Lt) are 43%, 100%, 100%, 95% & 43%, 98%, 75%, 95% respectively.

This indicates that notch is the better predictor of Preeclampsia, this is similar to opinions by Bower

et al1993[34], Chan et al 1995[35] and Antsakliset al2000[36].

The role of Doppler ultrasound in the study of utero-placental circulation is well known. It helps in detecting the extent of placental pathology and also predicts the maternal and fetal outcome.[37] Numerous studies have been conducted to know the association between Doppler waveforms and maternal perinatal outcome and have had variable results.

The present study showed that abnormal Doppler waveforms were associated with adverse maternal and perinatal outcome.

The application of uterine artery Doppler velocimetry is now being considered as a useful adjunct to screening programs for prediction of adverse pregnancy outcomes.[19]

Konchak et al proved that an increased uterine RI and uterine artery notch were both associated with raised relative risk of preeclampsia. Sensitivity, specificity, PPV and NPV of a uterine notch in their study was noted to be 83.3, 95.6, 55.6, and 98.9 %.[38]

Woschitz MC et al[39] proved that persistant uterine artery notch was related with raised relative risk of preeclampsia. In their study, sensitivity, specificity, PPV, and NPV of uterine artery notch were noted to be 40 %, 78 % , 56 %, and 65 %, respectively.

Coleman et al studied screening of uterine artery Doppler in high risk women showed the sensitivity and specificity for preeclampsia of RI >0.58 to be 91 and 42 %, respectively. In women with RI > 0.7, preeclampsia was noted in 58%[40]

Prajapati et al in their study of uterine artery Doppler screening in HR women showed sensitivity of Uterine artery PI was the best in the prediction of pre-eclampsia with SGA and gestational hypertension at 33.33 %. The specificity of Uterine artery PI [90th percentile was the best for pre-eclampsia at 94 %.[41]

Woschitz MC et al[61] in their study had a sensitivity of Uterine artery PI of 8 % and specificity of 95 %.

In our present study the sensitivity for PI of Uterine artery was higher compared to both Prajapatiet al[41] and Woschitz MC et al[39].

Study conducted by kulkarni et al showed Uterine artery Doppler specificity of 96.30%, sensitivity of 90%, positive predictive value of 94% and negative predictive value of 80%.[42]

Study conducted by k.Sahoo et al showed that by detecting abnormal uterine artery Doppler indices (High RI and PI) between 14 to 20 weeks of pregnancy, we can identify women at risk for development of preeclampsia.[43]

Bayesian analysis was used to calculate the posterior probability of adverse perinatal outcome following an abnormal or normal uterine artery Doppler assessment.[44]

CONCLUSION

Preeclampsia accounts for 10% of perinatal mortality and 14% of maternal mortality and morbidity. Early recognition of women of preeclampsia will help in identifying high risk women who may benefit from early prophylaxis & enhanced surveillance.

Abnormal uterine artery Doppler studies in the first and second trimester have been associated with subsequent adverse pregnancy outcomes including preeclampsia, fetal growth restriction, and perinatal mortality.

Mid trimester uterine artery Doppler velocimetry can be used as a reliable screening test for prediction of preeclampsia especially in the high risk group and it helps to reduce maternal and fetal complications by elective delivery.

Increased pulsatility index with notching in second trimester predicted overall preeclampsia in high risk and low risk patients, increased pulsatility index or bilateral notching predicted severe preeclampsia.

SUMMARY

Preeclampsia is a pregnancy specific disorder of unknown etiology accounting for 14% of maternal deaths worldwide. Incidence of this disorder is around 8-10%. Uterine artery Doppler screening meets all the requirements of a worthwhile screening program in prediction of preeclampsia. Uterine artery screening at 14-20 weeks gestation is superior to first trimester screening in prediction of preeclampsia and other adverse pregnancy outcomes. Despite these impressive results, few hospitals have established uterine artery screening programs in the second trimester as there is no effective preventive therapy when treatment is commenced after 24 weeks and also patients may develop adverse pregnancy outcome before 24 weeks gestation. A study was conducted in our hospital to know the predictive value of uterine artery Doppler at 14 o 20 weeks gestation using RI, PI, S/D ratio and diastolic notching. The results showed that abnormal uterine artery Doppler had a good predictive value in predicting women who developed preeclampsia, and the persistent diastolic notch is a better Doppler index in the prediction of preeclampsia. This was in accordance to various other studies. Doppler ultrasound is a non-invasive and reliable method for prediction of preeclampsia and adverse pregnancy outcome, but currently there are no effective interventions to prevent adverse outcomes based on an abnormal result. Studies are needed to find out such an intervention. Until such time, routine uterine artery Doppler screening of women is not required. Only screening in high risk women will suffice as to be more cautious during the pregnancy.

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B.L.D.E.Us, SHRI.B.M.PATIL MEDICAL COLLEGE, VIJAYAPUR – 586103

INSTITUTIONAL ETHICAL COMMITTEE, Date : 13/11/2018

1. Name of UG/PG Student/Researcher : Dr. Prakruti R. Patil
2. Department : of Radiodiagnosis
3. Title : Role of Uterine Artery Doppler in predicting preeclampsia.
4. Glude/Co-Guide/Principal Researcher : Dr. Shivanand Patil
5. Date of Admission (PG Only) : 02/05/2018

Observation:

OK

I.E.C. Remarks: Ethical clearance accorded/be chairman after corrected revised version is submitted by stipulated time.

1. Any alternation in Synopsis protocol should be intimated to E.C. in writing for review and approval.
2. Any adverse effects to subject of the study should be intimated in writing to E.C.
3. If study is stopped or an included patient is out of study inform E.C. the same with reason.

Signature of the Committee Members:

1. DR RAGHAVENDRA KULKARNI
2. DR TEJASWINI VALLABHA
3. DR.B.R.YELIKAR
4. DR P.BJAJU
5. DR CHANDRASHEKHAR BHUYYAR
6. DR PRANESH JAHAGIRDAR
7. SHRI.SURESH HAKKI
8. DR G V KUKARNI
9. DR.MOHD SHANNAWAZ
10. DR RAGHAVENDRA RAO

PROFORMA

Name IP.NO-

Age DOA-

Husbands name DOD-

Occupation Date of delivery-

Socio-economic status –

ADDRESS:

1) AGE
2) H/ODIABETES
3) H/O CHRONICHYPERTENSION
4) H/O CHRONIC RENALDISEASE
5) PAST BAD OBSTETRIC HISTORY OF PREECLAMPSIA, IUGR ANDIUFD
6) FAMILY HISTORY OF PREECLAMPSIA/IUGR

Obstetric history –

ML- Years, G P L A, Con/Non-Consanguineous Marriage Present
Obstetric history:
Booked / Unbooked LMP
Period of Amenorrhoea EDD
Menstrual History: Cycle regular / Irregular
Corrected EDD:
Spontaneous Conception/ Induced
I Trimester Details
II Trimester Details III Trimester Details Past Obstetric History:

Complication during past pregnancy and delivery: like PIH / GDM
Mode of delivery
Normal Vaginal delivery/Instrumental LSCS

Outcome of past delivery: Weight of baby, Term, Preterm, IUGR, Stillborn, Living, Dead.
PAST HISTORY Medical History of DM, HTN, TB, RHD, Renal Disease, Rickets, BT, Allergy to drug, Asthma, Epilepsy

FAMILY HISTORY

GPE: Pallor/Icterus / Cyanosis / Clubbing / Lymphadenopathy/Edema

SYSTEMIC EXAM'S

PR-:	CVS:	Thyroid
Temp:	RS:	BREAST
BP:		Height
RR :		Weight

PER ABDOMEN: FUNDAL HEIGHT

Date	**GESTATIONAL AGE**	**ANY COMPLAINTS**	**Wt.**	**Edema**	**BP**	**P/A**	**URINARY PROTIEN**

INVESTIGATIONS

1. Routine

HB%:	HIV:	Urine Routine:
Blood Grouping		
RBS:	VDRL:	CT

2. Ultrasound Gestational age:

Fetal biometry: BPD:	AC :	FL
EDD ACCORDING TO SCAN	EFW :	HC :

AFI
Placental grading:
Doppler Report
Doppler Velocity Values in uterine artery

	SD Ratio	RI	PI	Presence or Absence of Notch in Uterine artery
Left Uterine				
Right Uterine				

OUTCOME:

COMPLICATIONS: PERINATAL OUTCOME:

CONSENT FORM

TITLE OF RESEARCH: ROLE OF UTERINE ARTERY DOPPLER IN PREDICTING PREECLAMPSIA

GUIDE: DR SHIVANAND V PATIL

P.G. STUDENT: DR. PRAKRITI R PATIL

PATIENT'S NAME:

PURPOSE OF RESEARCH:

I have been informed that the purpose of this study is to study the role of uterine artery Doppler in predicting preeclampsia.

I understand that I will undergo detailed history and clinical examination and investigations.

RISKS AND DISCOMFORTS:

I understand that there is no risk involved and I may experience mild pain, after the above mentioned procedures.

BENEFITS:

I understand that my participation in this study will help in studying the role of uterine artery Doppler in predicting preeclampsia.

CONFIDENTIALITY:

I understand that the medical information produced by the study will become a part of hospital record and will be subjected to confidentiality and privacy regulations of hospital. If the data is used for publications the identity of the patient will not be revealed.

REQUEST FOR MORE INFORMATION:

I understand that I may ask for more information about the study at any time.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and I may refuse to participate or withdraw from study at any time.

INJURY STATEMENT:

I understand that in the unlikely event of injury to me/my ward, resulting directly to my participation in this study, if such injury were reported promptly, then medical treatment would be available to me, but no further compensation will be provided.

I understand that by my agreement to participate in this study, I am not waiving any of my legal rights.

I have explained to _____ the purpose of this research, the procedures required and the possible risks and benefits, to the best of my ability in patient's own language.

Date:

Dr Shivanand V Patil Dr Prakriti R Patil

(Guide) (Investigator)

STUDY SUBJECT CONSENT STATEMENT:

I/my ward confirm that Dr. Prakriti R Patil has explained to me the purpose of this research, the study procedure that I will undergo and the possible discomforts and benefits that I may experience, in my own language.

I/my ward have been explained all the above in detail in my own language and I understand the same. Therefore I agree to give my consent to participate as a subject in this project.

_____ _____

(Participant) Date

_____ _____

(Witness to above signature) Date

MASTERCHART

Si.no	Name	Age	BMI	Gravid p/m	UA dopp Rt				UA dopp Lt				Preeclampsia	Ga at the time of scan	SBP	DBP	Term/preterm	Ind/spo	Moden/c/i	Complication	Babd/d	bw	Nicu y/n
					N -/+	RI	PI	S/D	n-/+	RI	PI	S/D											
1	NAGARATHNA	26	19.7	M	**Y**	**0.79**	**2.1**	**4.8**	**Y**	**0.86**	**2.9**	**7.2**	**Y**	16	130	90	P	I	N	IUGR	D	0.8	N
2	RAJAMMA	28	19.7	P	N	0.67	1.0	2.2	N	0.7	1.1	2.3	N	18	110	70	T	LSCS	C	-	L	2.25	Y
3	REKHA	30	22.3	P	N	0.44	0.59	1.8	N	0.66	1.3	2.0	N	19	110	74	T	S	N	-	L	2.6	N
4	HAZEERA BEGUM	35	22.3	M	N	0.56	1.23	2.1	N	0.60	1.24	2.2	N	15	100	68	T	S	N	-	L	3	N
5	SHRUTI	19	21.3	P	N	0.69	1.0	2.0	N	0.55	0.99	2.1	N	19	112	76	T	S	N	-	L	3.1	N
6	ANITA	19	22.3	P	N	**0.65**	**1.1**	**2.8**	N	**0.76**	**1.6**	**4.1**	**Y**	19	138	100	T	I	N	IUGR	L	2.3	Y
7	SUSHMA	25	22.3	P	N	0.53	0.83	2.1	N	0.63	1.2	2.6	N	15	100	70	P	S	N	-	L	0.7	N
8	RUKSAR	25	31	M	N	0.64	1.1	2.2	N	0.75	1.2	2.5	N	15	120	80	T	S	N	-	L	0.7	N
9	NAYANA	24	26	M	N	0.70	1.3	2.8	N	0.7	1.3	2.8	N	14	120	78	T	LSCS	C	IUGR	L	2	Y
10	SUMA L	26	25	M	N	0.68	1.1	1.8	N	0.60	1.36	2.2	N	16	110	70	T	S	N	-	D	0.8	N
11	SHEWTA	18	24	P	N	0.60	1.2	2.8	N	0.60	0.99	2.6	N	17	110	70	T	S	N	-	L	1.8	N
12	GEEETA	26	23	M	N	0.61	1.07	1.9	N	0.54	1.26	1.9	N	19	126	76	T	S	N	-	L	1.9	N
13	SHABENA	25	23	M	N	0.52	1.05	2.5	N	0.52	1.25	1.88	N	18	112	76	T	S	N	-	L	0.8	N
14	KAVITA	20	22	P	N	0.55	0.88	2.23	N	0.49	0.62	1.73	N	20	120	80	T	LSCS	C	-	L	2.3	N
15	JAYA	19	22	M	N	**0.8**	**1.5**	**3.3**	N	**0.8**	**1.45**	**3.0**	N	19	120	80	P	S	N	DEAD	D	0.5	-
16	RAKSHA	18	19	P	N	0.45	0.63	1.8	N	0.59	1.2	2.5	N	20	116	80	T	S	N	-	L	3.2	N
17	MANASA	26	18	P	N	0.7	1.3	**3.63**	N	0.7	1.08	2.33	N	17	110	76	P	I	N	-	L	1.3	N
18	SEEMA	20	19	M	N	0.65	0.97	1.9	N	0.55	1.06	2.8	N	16	100	70	T	I	FORCEPS	MSL	L	1.9	N
19	DEVIKA	25	26	M	N	0.72	0.89	2.32	N	0.52	1.28	2.2	N	18	110	60	T	S	N		L	2.4	Y
20	BHAGYASHREE	20	34	P	**N**	0.68	1.4	2.5	N	0.70	1.14	2.4	**N**	14	120	80	T	I	N	-	L	1.8	N
21	SHEHNAZ	26	33	M	N	0.7	0.99	**5**	N	0.6	1.02	2.5	N	15	110	70	T	S	n	-	L	2.2	N
22	BAGAMATI	29	24	M	N	0.59	0.86	2.4	N	0.65	0.89	2.3	N	17	110	72	T	S	N	-	L	2	N
23	MAHDEVI	20	25	M	N	0.55	1.0	2.12	N	0.58	1.06	2.5	N	18	120	76	T	I	N	-	L	2.5	N
24	BHARATI	28	31	M	N	0.66	1.05	2.5	N	0.7	1.3	2.3	N	19	120	80	P	S	N	-	L	1.5	N
25	GORAMMA	24	30	M	N	0.7	0.79	2.6	N	0.7	1.02	2.26	N	20	122	80	T	S	N	-	L	1	N
26	SHAILAJA	30	24	P	N	0.58	1.22	2.4	N	0.65	1.07	1.94	N	20	114	74	T	S	N	-	L	2.1	N
27	REEMA	20	32	M	N	0.7	1.26	2.5	N	**0.8**	1.4	**5**	N	19	120	80	T	I	N	-	L	0.6	N
28	GIRIJA	34	25	M	N	0.67	0.97	2.5	N	0.62	0.88	1.95	N	18	110	70	T	I	N	-	L	1.5	N
29	SALEEMA BEGUM	28	24	P	N	0.66	1.1	2.22	N	0.66	0.79	1.63	N	14	100	80	T	S	N	-	L	1.5	N
30	ROSHNI BANU	37	25	P	N	0.7	0.57	2.6	N	0.7	0.77	1.6	N	15	120	80	T	S	N	-	L	2	N
31	KAMALAMMA	30	36	P	N	0.53	0.95	2.6	N	0.55	1.2	1.56	N	16	110	70	P	S	LSCS	DEAD	D	1.5	-
32	SUREKHA	36	26	P	N	0.50	1.2	2.5	N	0.57	1.0	1.65	N	19	110	70	T	S	LSCS	-	L	3.3	N
33	BHAGYA LAXMI	29	24	P	N	0.49	1.3	2.2	N	0.7	1.0	1.82	N	15	120	78	T	S	N	-	L	2.1	N
34	KAVYASHREE	28	25	P	N	**0.7**	**2.1**	**5**	N	**0.8**	**2.2**	**6**	**N**	15	110	70	T	S	N	-	L	3.25	N
35	RADHA LAXMI	20	19	M	N	0.68	1.1	2.2	N	0.64	0.8	2.2	N	19	110	76	T	S	N	-	L	1.5	N
36	AFREEN	19	23	M	N	**0.85**	**1.9**	**4.5**	N	**0.89**	**2.2**	**5.5**	**Y**	18	144	98	P	S	N	-	L	1.9	N
37	RUKSANA	20	22	M	N	0.5	1	2	N	0.5	1	2	N	20	116	78	T	S	N	-	L	2	N
38	LEELA	25	34	M	N	0.49	0.8	1.9	N	0.49	0.8	1.9	N	20	120	80	T	LSCS	LSCS	-	L	3.25	N
39	NETRA	20	22	P	N	0.5	0.9	1.8	N	0.5	0.9	1.8	N	19	100	68	T	I	N	-	L	2.5	Y
40	SHANTA	24	24	M	N	0.51	0.8	2	N	0.51	0.8	2	N	19	110	70	T	S	N	-	L	2.7	N
41	JAGADEVI	32	25	M	N	0.5	0.9	1.9	**Y**	0.5	0.9	1.9	N	16	120	80	T	I	N	-	L	1	N
42	RATNAMALA	22	24	P	N	0.45	0.9	1.8	N	0.45	0.9	1.8	N	18	120	80	P	I	N	-	D	0.6	-
43	TASNEEM	27	26	M	N	0.5	1	2.4	N	0.5	1	2.4	N	17	110	70	T	S	N	-	L	0.9	N
44	MANJULADEVI	28	28	M	N	0.46	1	2.3	N	0.46	1	2.3	N	15	110	66	T	I	N	STILL BORN	STILL BORN	1.4	-
45	SOUMYASHREE	29	33	P	N	0.5	1	2.2	N	0.5	1	2.2	N	14	120	80	T	LSCS	LSCS	IUGR	L	2.25	Y
46	GOWRAMMA	25	31	P	N	0.51	0.9	2	N	0.51	0.9	2	N	19	116	72	T	S	N	-	L	2.5	N
47	GEETHANJALI	27	32	P	N	0.54	1	2	N	0.54	1	2	N	18	110	80	T	LSCS	N	-	L	3	N
48	PRIYANKA	29	25	P	**Y**	**0.76**	**1**	**3.3**	**Y**	**0.76**	**1.31**	**4.15**	**Y**	20	**140**	**90**	P	S	N	DEAD	D	0.8	-
49	VARSHA	27	28	M	N	0.76	1.04	2.13	N	0.70	1.14	2.13	N	18	120	80	T	I	N	-	L	2	N
50	SAMYA	28	24	M	N	0.7	1.26	2.3	N	0.48	1.11	2.13	N	20	116	80	T	I	N	-	L	3	N

Si.no	Name	Age	BMI	Gravidp/m	UA doppRt				UA dopp Lt				Preeclampsia	Ga at the time of scan	SBP	DBP	Delivt/prt/pot	Ind/spo	Modem/c/i	Compa/l/e/sp/mp/gh	Babyl/d	bw	Nicuy/n
					N -/+	RI	PI	S/D	n-/+	RI	PI	S/D											
51	DEEPIKA	21	19.7	M	N	0.52	0.75	2	N	0.52	0.72	2	N	20	112	80	T	S	N	-	L	2.7	N
52	PREMA	20	19.7	P	N	0.72	0.82	2.6	N	0.7	0.99	2.3	N	18	120	80	T	S	N	-	L	3.1	N
53	GAYATRI	22	22.3	M	N	0.54	0.88	2.2	N	0.62	1.12	2.2	N	18	110	70	T	S	N	-	L	3.27	N
54	ASHA	20	22.3	P	N	0.5	1.1	2.3	N	0.5	1.1	2.3	N	16	120	80	T	lscs	c	-	L	3.75	N
55	SHANTA	25	21.3	M	N	0.52	0.74	2	N	0.61	0.94	2	N	20	126	86	T	S	N	-	L	2.6	N
56	SHOBHARANI	24	22.3	P	N	**0.61**	**0.92**	**1.9**	N	**0.64**	**1.14**	**1.9**	**Y**	18	136	90	T	S	N	-	L	2.5	N
57	LAKSHMAMMA	27	22.3	P	N	0.54	0.98	1.9	N	0.58	1.13	1.9	N	16	110	80	T	I	forceps	-	L	2.5	y
58	NAZNEEN	21	20.8	p	N	0.51	1.29	2.1	N	0.53	1.14	2.1	N	17	100	66	T	S	N	-	L	2.7	N
59	YAMANAVVA	36	21.6	M	N	0.67	1.23	2.2	N	0.64	1.14	2.2	N	19	100	70	T	S	N	-	L	2.9	N
60	HEMALATHA	21	20.4	M	N	0.52	0.78	2.3	N	0.52	0.76	2.3	N	17	100	70	T	S	N	-	L	2.5	N
61	ALEKYA	20	20.7	P	N	0.55	0.96	2.4	N	0.55	0.82	2.4	N	20	126	90	T	S	N	-	L	1.6	Y
62	RAJAMMA	28	18.6	P	N	0.54	0.96	2.1	N	0.52	0.82	2.1	N	19	120	80	T	I	N	-	L	2.5	N
63	LAVANYA	23	23.8	M	N	0.49	0.77	2	N	0.52	0.75	2	N	20	120	80	T	I	N	-	L	3	N
64	SHILPA	20	22.3	P	N	0.70	1.4	2.1	N	0.66	1.4	2	N	18	110	80	T	S	N	-	L	2.6	N
65	LALITHA	23	31.1	P	N	0.52	0.77	2.5	N	0.54	0.86	2.5	N	19	110	80	T	S	N	MSL	L	2.8	Y
66	PRAMATHA	22	22.3	M	N	0.54	0.86	2.2	N	0.52	0.8	2.2	N	20	120	74	T	S	N	-	L	2.4	N
67	CHETANA	20	22	P	N	0.54	0.89	1.9	N	0.55	0.94	1.9	N	15	116	70	T	S	N	-	L	2.8	N
68	RAMABAI	25	21.9	M	N	0.5	0.86	2	N	0.5	0.78	2	N	16	130	80	T	S	N	-	L	2.4	N
69	SIRIDEVI	24	20.4	P	N	0.51	0.78	2.1	N	0.56	0.94	2.1	N	18	120	76	P	S	N	-	L	1.8	Y
70	SAVITRI	20	21.9	P	N	0.5	0.7	2	N	0.6	1.13	2	N	14	120	80	P	I	N	-	L	0.95	Y
71	NANDA	22	23.4	M	N	0.52	0.8	2	N	0.5	0.74	2	N	19	128	80	T	S	N	-	L	3.11	N
72	MANJULADEVI	27	22.2	M	N	0.5	0.7	2.2	N	0.65	1	4	N	20	120	80	T	S	N	-	L	3.1	N
73	RAMA	24	24.6	P	N	0.54	1.1	2.4	N	0.54	1.14	2.4	N	20	120	80	T	S	N		L	1.7	Y
74	RAJANI	21	21.3	M	N	**0.8**	**2.66**	**2.1**	N	**0.85**	**2.49**	**5**	**Y**	19	160	110	T	lscs	C	IUGR	L	2	Y
75	BAVANI	27	25.5	P	N	0.54	0.96	2	N	0.52	0.78	2	N	14	110	80	T	I	N	-	L	2.9	N
76	SHRADDHA	32	22.2	M	N	0.51	0.75	2	N	0.49	0.74	2	N	15	110	70	T	S	N	-	L	2.6	N
77	AYESHA	23	20.9	M	N	0.5	0.75	2	N	0.52	0.78	2	N	20	110	70	T	S	N	-	L	2.7	N
78	SUSHMA R	28	17.5	P	N	0.52	1.14	2.1	N	0.52	0.94	2.1	N	20	110	80	T	S	N	-	L	3.1	N
79	PRAJACTA	26	24	M	N	0.55	1.12	2.2	N	0.52	0.86	2.2	N	15	120	80	T	S	N	-	L	2.9	N
80	ANUSHKA	28	22.2	P	N	0.5	0.69	2.3	N	0.66	1	4.2	N	19	118	80	T	S	N	-	L	2.49	N
81	SUSHILABAI	22	20.2	P	N	0.5	0.69	2	N	0.54	0.98	2	N	20	110	70	T	S	N	-	L	3.1	N
82	KALAVVA	24	22.6	P	N	0.54	0.92	2.3	N	0.53	0.79	2.3	N	19	110	70	T	lscs	C	-	L	2.75	N
83	JAMALA	29	25.7	M	N	0.54	0.9	2.1	N	0.67	1	4	N	14	126	80	P	S	N	-	L	2.5	Y
84	KALAVATI BAI	34	22	M	N	0.59	1.1	1.9	N	0.5	0.7	1.9	N	20	120	80	T	I	N	-	L	3.1	N
85	PARVATI	32	21.6	M	N	0.5	0.7	1.8	N	0.54	0.94	1.8	N	18	120	80	T	S	N	-	L	2.9	Y
86	USHA	21	25.2	P	N	0.54	1.1	1.7	N	0.54	1.1	1.7	N	20	110	70	T	S	N	-	L	2.7	N
87	RAMYA	31	22.8	M	N	0.5	1	1.9	N	0.5	1	1.9	N	20	120	74	T	I	N	-	L	2.5	N
88	AMBIKA	26	21.4	P	N	0.45	1	1.8	N	0.47	1	1.8	N	16	124	80	T	lscs	C	-	L	2.8	Y
89	SONIKA	20	24.3	P	N	0.5	1.1	1.9	N	0.55	1.3	1.9	N	40	120	80	T	S	N	-	L	3.2	N
90	BORAMMA	24	20.4	P	N	0.7	1.73	2	N	0.7	1	4.5	N	17	110	80	T	I	N	-	L	2.92	N
91	SHABANA	26	22.5	P	N	0.5	1.12	2.4	N	0.5	1.12	2.4	N	20	110	70	T	lscs	C	-	D	2.7	-
92	DEVAKAMMA	30	22	M	N	0.55	0.8	2.5	N	0.55	0.8	2.5	N	20	110	70	T	S	N	-	L	2.8	Y
93	ZUBEENA	24	30.3	P	**Y**	**0.72**	**0.96**	**6.4**	**Y**	**0.82**	**1.6**	**6.8**	**Y**	18	148	116	T	S	N	-	L	2.1	Y
94	PALLAVI	25	21.5	P	N	0.48	1.1	2.5	N	0.48	1.1	2.5	N	14	110	70	T	I	N	-	L	2.22	N
95	PRIYA	29	30.29	P	N	0.5	1.1	2.2	N	0.5	1.1	2.2	N	16	120	80	P	I	N	-	L	2.3	N
96	POOJA	28	21.7	P	N	0.5	0.7	2.2	N	0.5	0.75	2.2	N	20	116	70	T	S	N	-	L	2.75	N
97	SHRIDEVI	28	18.4	P	N	0.52	1.13	2	N	0.52	1.13	2	N	18	124	82	T	S	N	-	L	3.12	N
98	SUMALATA	23	22.3	P	N	0.5	1.1	2	N	0.5	1.1	2	N	19	120	80	T	S	N	-	L	2.7	N
99	IRAMMA	22	20	P	N	0.52	1.1	2	N	0.52	1.1	2	N	20	120	80	T	S	N	-	L	3	N
100	ARUNA	24	22	M	N	0.54	1.1	2	N	0.52	1.1	2	N	20	110	80	T	lscs	C	MSL	L	3.1	Y

KEY TO MASTER CHART

BMI	-	BODY MASS INDEX
N	-	UTERINE ARTERY NOTCH
PI	-	PULSATALITY INDEX
RI	-	RESISTANCE INDEX
S/D RATIO	-	SYSTOLIC/DIASTOLIC RATIO
PE	-	PRECLAMPSIA
GA	-	GESTATIONAL AGE
SBP	-	SYSTOLIC BLOOD PRESSURE
DBP	-	DIASTOLIC BLOOD PRESSURE
N	-	NORMAL VAGINAL DELIVERY
I	-	INDUCED
LSCS	-	LOWER SEGMENT CAESAREAN SECTION
C	-	CAESAREAN SECTION
IUGR	-	INTRA UTERINE GROWTH RESTRICTION
IUD	-	INTRA UTERINE DEATH
L	-	LIVE
D	-	DEAD
MSL	–	MECONIUM STAINED LIQUOR