بسم الله الرحمن الرحيم

Sudan University of Science and Technology
College of Graduate Studies

Assessment of Late and Postterm Pregnancies using Doppler ultrasound

تقييم حالات الحمل المتأخر والفترة التي تليها بواسطة الموجات فوق الصوتية دوبلر الطيفية

A thesis Submitted for PhD degree in Medical Diagnostic Ultrasound

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بسم الله الرحمن الرحيم

الآيــة

قال تعالى :

"وَوَصَّيْنَا الْإِنْسَانَ بِوَالِدَيْهِ إِحْسَانًا حَمَلَتْهُ أُمُّهُ كُرْهًا وَوَضَعَتْهُ كُرْهًا وَحَمْلُهُ وَفِصَالُهُ ثَلَاثُونَ شَهْرًا حَتَّى إِذَا بَلَغَ أَشُدَّهُ وَبَلَغَ أَرْبَعِينَ سَنَةً قَالَ رَبِّ أَوْزِعْنِي أَنْ أَشْكُرَ نِعْمَتَكَ الَّتِي أَنْعَمْتَ عَلَيَّ وَعَلَى وَالِدَيَّ وَأَنْ أَعْمَلَ صَالِحًا تَرْضَاهُ وَأَصْلِحْ لِي فِي ذُرِّيَّتِي إِنِّي تُبْتُ إِلَيْكَ وَإِنِّي مِنَ الْمُسْلِمِينَ "

صدق الله العظيم

سورة الاحقاف الاية (15)

Dedication

"Dedicated to my beloved
Parents & family"
For their love, endless
Support, encouragement
& sacrifices

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Abstract

This was prospective study carried out in -Khartoum state –Sudan, Khartoum Bahri Teaching hospital in the period from august 2016 to December 2018. The problem of study was the management of late-term and post term pregnancy creates a challenge to clinicians on the other hand the Doppler ultrasound can be used as an excellent tools to aid in management. The main aim of the study was to assess late-term and post term pregnancies by spectral Doppler ultrasound. The study was done in 197 pregnant women with normal singleton pregnancy with mean age 28 years, with gestational age by Last Menstrual period (GA LMP) 33-46 weeks and no maternal medical condition that may affect pregnancy outcome such as diabetic and hypertension. The data was collected by data collection sheet designed especially for this study and including all variables; then analyzed by statistical package for social sciences (SPSS).

The study found that most of them were nulliparous (78.8%), most of fetus was cephalic presentation (94%), (49.7%) with GA of (33-38w6d) , (17.8%) term with GA of (39-40) weeks and postdate was (32.5%). The study found that there no significant difference in Doppler indices of UA in these three group of gestational age ($p > 0.05$), except for S\d ratio and PI which shows significant difference in different age group ($p< 0.05$), UA S\D ratio

had significant weak negative correlation with GA ($p < 0.05$) and UA PI,RI had no significant correlation with GA LMP ($p > 0.05$) and PSV and EDV shows positive significant weak correlation with GA LMP but in generally decreased by GA , Concerning linearity the study clarified that as GA increased Doppler indices of UA decreased RI, PI, S\D ratio and MCA increased PI decreased S\D ratio and RI remain constant .The study found that no significant difference in Doppler indices of MCA in these three group of gestational age. The study found that MCA PSV and EDV shows positive significant weak correlation with GA LMP ($p < 0.05$) , while PI and S\D ratio shows negative weak correlation with GA ,the mean MCA \ UA ratio was 1.87±1.31. There is significant relation between GA and adverse outcome as prevalence of oligohydramnios, macrosomia and cesarean section increased in postdate pregnancy. The study confirmed that no significant differences in all these Doppler indices in patients post term with adverse versus post term with normal outcome.

The study concluded that Doppler indices in advance gestation and post term decreased with gestational age and increased in post term pregnancy with adverse outcome than in normal outcome but all were not significant. The study recommended that further studies with larger sampling of postdate to predict ratio for cases of adverse outcome in postdate pregnancy.

مستخلص البحث

كانت هذه الدراسة الاستطلاعية التي أجريت في ولاية الخرطوم ، السودان ، مستشفى الخرطوم بحري التعليمي في الفترة من أغسطس 2016 إلى ديسمبر 2018. كانت مشكلة الدراسة هي ان إدارة الحمل المتأخر والبعيد يمثل تحديا للأطباء و من ناحية أخرى ، يمكن استخدام الموجات فوق الصوتية دوبلر كأداة ممتازة للمساعدة في الإدارة. كان الهدف الرئيسي من هذه الدراسة هو تقييم حالات الحمل المتأخر والفترة التي تليها بواسطة الموجات فوق الصوتية دوبلر الطيفية. أجريت الدراسة في 197 امرأة حامل مع الحمل المفرد الطبيعي متوسط اعمارهن 28 سنة ، مع عمر الحمل بتاريخ اخر دورة شهرية (33–46) اسبوع وليس هناك حالة طبية تعاني منها الام ممكن ان تؤثر على نتائج الحمل مثل السكري وارتفاع ضغط الدم. تم جمع البيانات عن طريق ورقة جمع البيانات المصممة خصيصا لهذه الدراسة وشملت جميع المتغيرات؛ ثم تم تحليلها بواسطة الحزمة الإحصائية للعلوم الاجتماعية (SPSS).

ووجدت الدراسة أن معظمهن كان ليس لهن اولاد 78.8 ٪ ، وكان معظم الاجنة في الوضع الرأسي (94 ٪) ، (49.7 ٪) من الحالات كانت في فترة الحمل (33–38 اسبوع) ، (17.8 ٪) 39–40 أسبوعا ، كان (32.5٪) اكتر من 40 اسبوع ، ووجدت الدراسة أنه لا يجد هناك اختلاف كبير في مؤشرات دوبلر شريان السري في هذه المجموعات الثلاث من عمر الحمل (قيمة تنبئية <0.05) باستثناء مؤشر الانبساطي علي الانقباضي و مؤشر النبض حيث انه يوجد فرق دال في هذه المجموعات الثلاث مع عمر الجنين (قيمة تنبئية > 0.05) ، وجدت الدراسة أن هناك علاقة سلبية ضعيفة بين نسبة مؤشر الانبساطي علي الانقباضي في الشريان السري مع عمر الجنين (قيمة تنبئية <0.05) ، أوضحت الدراسة أنه مع زيادة عمر الجنين مؤشرات الدوبلر لم يتاثر بالنسبة الى مؤشرالنبض والمقاومة في الشريان السري وانه يوجد علاقه بين سرعة الدم الانقباضي ، سرعة الدم الانبساطي مع عمر الجنين . وجدت الدراسة أنه لا يوجد فرق كبير في مؤشرات دوبلر من الشريان الاوسط المخى في هذه المجموعة الثلاث مع عمر الحمل .وجدت الدراسة أن هناك علاقة إيجابية ضعيفة بين عمر الجنين بتاريخ اخر دورة شهرية و سرعة الدم الانقباضي ، سرعة الدم الانبساطي (قيمة تنبئية <0.05) ، وانه يوجد علاقة سلبية ضعيفة بين عمر الجنين ومؤشر النبض ومؤشر الانبساطي علي الانقباضي وانه

يوجد علاقه بين مؤشر النبض وعمر الجنين. وكان متوسط نسبة النبض للمخي على السري 1.87±1.31 . هناك علاقة معنوية بين عمر الجنين والنتائج السلبية مثل زيادة قلة السائل الأمنيوني ، زيادة وزن الجنين و الولادات القيصرية في الحمل بعد تاريخ الولادة. أكدت الدراسة أنه لا توجد فروق ذات دلالة إحصائية في جميع مؤشرات دوبلر هذه عند المرضى ذوى زيادة فترة الحمل مع التأثير العكسي مقابل فترة ما بعد الحمل مع النتائج الطبيعية .

وخلصت الدراسة إلى أن مؤشرات دوبلر في الحمل المتقدم وتقدم الفترة تنخفض مع التقدم عمر الجنين ، وفي فترة ما بعد الحمل ارتفعت مؤشرات دوبلر ونسبة النبض للشريان المخي الاوسط على الشريان السري في حالة وجود نتائج سلبية مقارنة بالنتائج الطبيعية ولكن لم تكن جميعها كبيرة او ذات دلالة معنوية . أوصت الدراسة بإجراء مزيد من الدراسات مع أخذ عينات أكبر من زائدات فترة الحمل الطبيعية للتنبؤ بالنسبة لذوات زائدات الفترة الاتي لديهن تأثيرات عكسية.

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Abbreviation

AC	abdominal circumference
AFI	amniotic fluid index
BPD	fetal biparietal diameter
BPP	Biophysical profile
CP ratio	cerebro-placental ratio
CRH	corticotrophin releasing hormone
CRL	crownrump-length
CS	caesarean section
CST	Contraction stress test
CTG	Cardiotocography
CW	continuous wave
DU	Doppler ultrasonography
EDD	estimated date of delivery
FHR	fetal heart rate
FL	femur length
FSH	Follicle stimulating hormone
GA	gestational age
HC/AC ratio	head circumference-to-abdominal circumference ratio
hCG	human chorionic gonadotropin
IOL	induction of labour
IUFD	intra-uterine fetal death
IUGR	intrauterine growth-restriction
LGA	large-for-gestational-age
LH	luteinizing hormone
LMP	last menstrual period
MA	menstrual age
MCA	middle cerebral artery
NT	nuchal translucency
PI	pulsatility index
RI	Resistance Index
UA	the umbilical artery
BSF	brain-sparing flow
CBF	cerebral blood flow
CDI	color Doppler imaging
CPR	Cerebro placental ratio
MCA/UA ratio	middle cerebral artery to umbilical artery
PD	power Doppler

PSV	Peak systolic velocity
S/D ratio	systolic-diastolic ratio

Chapter one

1.1 Introduction

For many years the biological possibility that a pregnancy could exceed the 42 week was questioned. It was not until the early 1960s that conclusive evidence of an increased risk of fetal mortality in Prolonged pregnancy was presented (Mc ClureBrowne.,1963) Many obstetricians met this information with criticism. However, prolonged pregnancy was gradually recognized as a problem when the paediatricians became interested in the matter. In 1954, in a postmature classification system, Clifford described the degree of affliction suffered by Prolonged neonates, and eventually in the late 1960s and 1970s, with the advent of ultrasound dating, the fetal risk associated with Prolonged pregnancy was fully established **(**Kitlinski M., 2007).

Prolonged pregnancy is defined as a pregnancy lasting more than 294 days (42 weeks gestation). Traditionally the diagnosis has been based on calculation of 42 weeks from the first day of the last menstrual period. However, recent years have seen use of first trimester ultrasound as the basis for definition of prolonged pregnancy because it has been found to be accurate and often more reliable in estimating gestation. Prolonged pregnancy is associated with increased risks to both mother and the fetus, thus making it one of the commonest indications for induction of labour (IOL). Most obstetric units throughout the world offer routine IOL between 41 and 42 weeks of pregnancy to minimize the maternal and perinatal risks of a prolonged pregnancy. (Srikumar S et al., 2017)

Doppler ultrasonography (DU) velocimetry of fetal and uterine vessels is a well-established method for antenatal monitoring. Certain Doppler

waveforms indicating circulatory changes can be used to predict adverse perinatal outcomes. DU was successfully introduced in obstetric imaging and fetal monitoring way back in 1977. Fitzgerald et al. were the first to report noninvasive demonstration of the umbilical cord (UC) blood flow pattern and suggested that the umbilical artery (UA) waveforms could be abnormal in fetuses with intrauterine growth-restriction (IUGR). This breakthrough concept of studying waveforms also resulted in several important clinical applications. Doppler assessment of the UA has now become standard of care in antenatal surveillence. Doppler assessment of the fetal middle cerebral artery (MCA) had also been widely used for the diagnosis of fetal anemia.

DU waveforms not only reflect blood velocity but also provide information on various aspects of blood flow like presence and direction of flow, velocity profile, flow volume, and impedance. (Srikumar S et al., 2017)

Among all vessels studied in DU, the UA and MCA are relatively easier to access and evaluate and are reported to be more reproducible. MCA of fetuses had been extensively studied for evaluation of placental compromise and fetal anaemia. Combining the Doppler indices of the MCA with that of the UA by the ratio of their pulsatility indices, also known as cerebro-placental ratio (CP ratio), is a useful tool for monitoring fetal health. A low CP ratio indicates relative redistribution of the blood flow to the cerebral circulation and is found to increase accuracy in envisaging complications and adverse outcomes as compared to MCA or UA Doppler indices alone. This ratio has now been increasingly used in the surveillance of the fetus at risk by repeating the Doppler studies at regular intervals. (Srikumar S et al., 2017)

Several studies have examined the potential value of Doppler assessment in the prediction of adverse outcome (usually defined as fetal distress in labor) in post-term pregnancies and provided conflicting results there was four studies examining uterine arteries reported no significant changes in pregnancies with adverse outcome(Rightmire & Campbell, 1987; Farmakides et al, 1988; Brar et al, 1989; Stokes et al., 1991). Impedance to flow in the umbilical arteries of pregnancies with adverse outcomes was normal in five studies, increased in three studies (Rightmire & Campbell, 1987; Fischer et al., 1991; Anteby et al., 1994) and decreased in one study (Olofsson et al., 1997). Impedance in the fetal cerebral circulation was reported as being decreased in three studies (Brar et al., 1989; Anteby et al., 1994Devine et al., 1994) and normal in two studies(Bar-Hava et al., 1995; Zimmermann et al., 1995)

1.2 Problem of the study:

The management of Late-Term and postterm pregnancy constitutes a challenge to clinicians; knowing who to induce, who will respond to induction and who will require a caesarean section (CS) and the risk of stillbirth increases beyond 41 weeks , additional fetal risks of postterm pregnancies include macrosomia, cesarean deliveries and shoulder dystocia, as well as meconium aspiration syndrome and Oligohydramnios. On the other hand the Doppler ultrasound can be used as an excellent tools to management Late-Term and Postterm Pregnancies. In the centre of health care in Sudan there is rare application of Doppler ultrasound for middle cerebral artery and umbilical artery.

1.3 Justification:

There main goal of prenatal testing to identify foetuses at risk for prenatal morbidity and mortality

The traditional methods of fetal surveillance like non stress test, fetal heat monitoring and fetal biophysical profile are no more ideal tests because of their inability to detect early stages of fetal distress, significant number of false positive tests and low predictive value.

Observational study that Doppler ultrasound of the umbilical artery is more helpful than other tests of fetal wellbeing (e.g. heart rate variability and biophysical profile score) so the rule of colour Doppler come to detect these abnormal vascular resistance patterns. The important issue is not the identification of small fetuse, but rather than"fetus at risk" for compromise

1.4Objectives of the study:

1.4.1 General objective:

To assess Late-term and Postterm Pregnancies using spectral Doppler ultrasound.

1.4.2 Specific objectives:

1. To evaluate the S/D ratio, pulsatility index(PI) , resistance index (RI) and middle cerebral artery to umbilical artery (CU) ratio of the middle cerebral and umbilical artery in Late-Term and post term pregnancy and those with preterm pregnancy
2. To determine the accuracy of middle cerebral artery in decreasing the risks of perinatal morbidity and mortality.

3. To determine the accuracy of umbilical artery in decreasing the risks of perinatal morbidity and mortality.
4. To establish reference ranges of S/D ratio, pulsatility index (PI), resistance index (RI) and middle cerebral artery to umbilical artery (CU) ratio of the middle cerebral and umbilical artery.

Chapter Two
Theoretical background and Literature Review

2.1 Maternal physiology and embryology:

Early in the menstrual cycle, the pituitary secretes rising levels of follicle-stimulating hormone (FSH) and luteinizing hormone (LH), which cause the growth of 4 to 12 primordial follicles into primary ovarian follicles. When a fluid-filled cavity or antrum forms in the follicle, it is eferred to as a secondary follicle. the primary oocyte is off to one side of the follicle and surrounded by follicular cells or the cumulus oophorus. One follicle becomes dominant, bulges on the surface of the ovary and becomes a "mature follicle" or Graafian follicle. It continues to enlarge until ovulation, with the remainder of the follicles becoming atretic. The developing follicles produce estrogen. The estrogen level remains relatively low until 4 days before ovulation, when the dominant or active follicle produces an estrogen surge, after which an LH and prostaglandin surge results in ovulation. Ovulation follows the LH peak within 12 to 24 hours. Actual expulsion of the oocyte from the mature follicle is aided by several factors, including the intrafollicular pressure, possibly contraction of the smooth muscle in the theca externa stimulated by prostaglandins, and enzymatic digestion of the follicular wall. (Oehninger S, Hodgen GD., 1993)

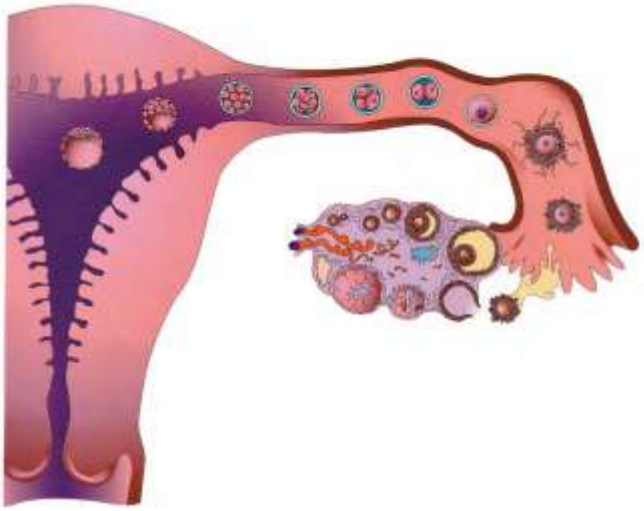
Ovulation occurs on approximately day 14 of the menstrual cycle with expulsion of the secondary oocyte from the surface of the ovary. In women with a menstrual cycle longer than 28 days, this ovulation occurs later, so that the secretory phase of the menstrual cycle remains at about 14 days. After ovulation, the follicle collapses to form the corpus luteum, which secretes progesterone and, to a lesser degree, estrogen. If a pregnancy does

not occur, the corpus luteum involutes. In pregnancy, involution of the corpus luteum is prevented by human chorionic gonadotropin (hCG), which is produced by the outer layer of cells of the gestational or chorionic sac (syncytiotrophoblast) (rumack., 2011).

Before ovulation, endometrial proliferation occurs in response to estrogen secretion .After ovulation, the endometrium becomes thickened, soft, and edematous under the influence of progesterone.(Jones GS, Jones HW,1982)

The glandular epithelium secretes a glycogen-rich fluid. If pregnancy occurs, continued production of progesterone results in more marked hypertrophic changes in the endometrial cells and glands to provide nourishment to the blastocyst. These hypertrophic changes are referred to as the decidual reaction and occur as a hormonal response regardless of the site of implantation, intrauterine or ectopic.

Oocyte transport into the fimbriated end of the fallopian tube occurs at ovulation as the secondary oocyte is expelled with the follicular fluid and is "picked up" by the fimbria. The sweeping movement of the fimbria, the currents produced by the action of the cilia of the mucosal cells, and the gentle peristaltic waves from contractions of the fallopian musculature all draw the oocyte into the tube. (Settlage DS et al.,1973)

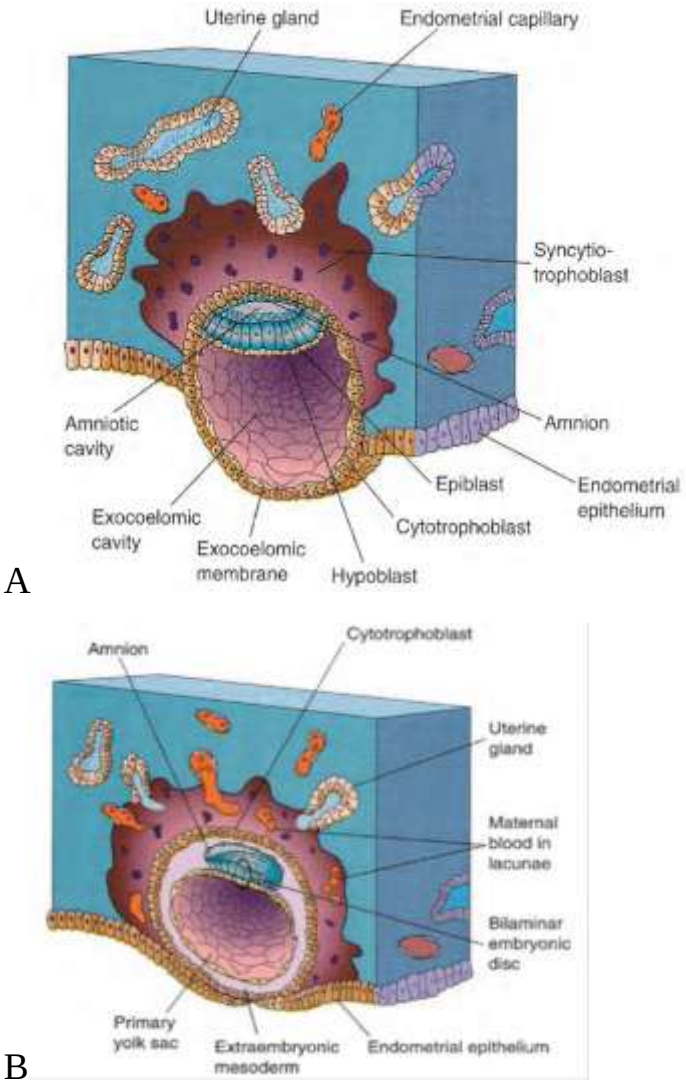


Figure(2.1): Diagram of ovarian cycle, fertilization, and human development to the blastocyst stage. (rumack.,2011)

Fertilization occurs on or about day 14 as the mature ovum and sperm unite to form the zygote in the outer third of the fallopian tube Cellular division of the zygote occurs during transit through the fallopian tube. By the time the conceptus enters the uterus, about day 17, it is at the 12- to 15-cell stage (morula). By day 20, the conceptus has matured to the blastocyst stage.

The blastocyst is a fluid-filled cyst lined with trophoblastic cells that contain a cluster of cells at one side called the inner cell mass. On day 20, the blastocyst at the site of the inner cell mass burrows through the endometrial membrane into the hyperplastic endometrium, and implantation begins12 (Hustin J., 1995)

Implantation is completed by day 23 as the endometrial membrane re-forms over the blastocyst .



Figure(2.2): Implantation of the blastocyst into endometrium. Entire conceptus is approximately 0.1 mm at this stage. A, Partially implanted blastocyst at approximately 22 days. B, Almost completely implanted blastocyst at about 23 days. (rumack.,2011)

During implantation, the amniotic cavity forms in the inner cell mass. A bilaminar embryonic disk separates the amniotic cavity from the exocoelomic cavity. The primary (primitive) yolk sac forms at about 23 days of gestational age as the blastocyst cavity becomes lined by the exocoelomic membrane and hypoblast. As the extraembryonic coelom forms, the primary

yolk sac is pinched off and extruded, resulting in the formation of the secondary yolk sac. Standard embryology texts indicate that the secondary yolk sac actually forms at approximately 27 to 28 days of menstrual age (MA), when the mean diameter of the gestational sac is approximately 3 mm. It is the secondary yolk sac, rather than the primary yolk sac, that is visible with ultrasound.

Later, because of differential growth, the yolk sac comes to lie between the amnion and chorion.

During week 4, there is rapid proliferation and differentiation of the syncytiotrophoblast, forming primary chorionic villi.

Before 12 weeks, the intervillous space contains no blood, only clear fluid, and on histologic examination, the villous tissue is separated from the maternal circulation by a continuous layer of trophoblastic cells. Only after the third month does the trophoblastic shell become broken and the maternal circulation become continuous with the intervillous space. Further, at weeks 8 and 9 of gestation, the trophoblastic shell forms plugs within the spiral arteries, allowing only filtered plasma to permeate the placenta. (Jauniaux E.,1996)

Vascularization of the placenta occurs at the beginning of the fifth week. Oh et al. showed significantincreases in sac size from 5 weeks onward in normal pregnancies versus pregnancy failures. (Oh et al.,2002) .

During the fifth week, the embryo is converted by the process of gastrulation from a bilaminar disk to a trilaminar disk with the three primary germ cell layers: ectoderm, mesoderm and endoderm. During gastrulation, the primitive streak and notochord form. The primitive streak gives rise to the mesenchyme, which forms the connective tissue of the embryo and stromal components of all glands.

The formation of the neural plate and its closure to form the neural tube is referred to as neurulation. This process begins in the fifth week in the thoracic region and extends caudally and cranially, resulting in complete closure by the end of the sixth week (day 42). Failure of closure of the neural tube results in neural tube defects.

During the fifth week, two cardiac tubes (the primitive heart) develop from splanchnic mesodermal cells. By the end of the fifth week, these tubes begin to pump into a primitive paired vascular system. By the end of the fifth week, a vascular network develops in the chorionic villi that connect through the umbilical arteries and vein to the primitive embryonic vascular network

Essentially all internal and external structures present in the adult form during the embryonic period, which ends at 10 menstrual weeks. By the end of the sixth week, blood flow is unidirectional, and by the end of the eighth week, the heart attains its definitive form. The peripheral vascular system develops slightly later and is completed by the end of the tenth week. The primitive gut forms during week 6. The midgut herniates into the umbilical cord from week 8 through the end of week 12. The rectum separates from the urogenital sinus by the end of week 8, and the anal membrane perforates by the end of week 10. The metanephros, or primitive kidneys, ascend from the pelvis, starting at approximately week 8, but do not reach their adult position until week 11. Limbs are formed with separate fingers and toes. Almost all congenital malformations except abnormalities of the genitalia originate before or during the embryonic period.

External genitalia are still in a sexless state at the end of week 10 and do not reach mature fetal form until the end of week 14.

Early in the fetal period, body growth is rapid and head growth relatively slower, with the crown-rump length doubling between weeks 11 and 14. (Rumack .,2011).

2.2 Technique for obstetrical ultrasound:

2.2.1 Routine Approach:

The following is the suggested routine when examining women with an apparently normal pregnancy:

1.Sweep first. A quick scan through the uterus to check for gross pathology and fetal viability allows you to set the tone for how much to share the exam with the patient and family.

2.Lower uterine segment. Do it early, before the bladder fills, potentially distorting the cervical length or its relationship to the placental margin.

3. Long-axis and transverse views of the spine, if the fetus is in a convenient position. This localizes the fetus for the axial views to follow.

4. The head (1t may not be as easy to view later in the exam).

5. Transvers views of the chest to show a four-chamber view.

6. The trunk circumference view, making sure to include the stomach.

7. Transverse and long views of the kidneys.

8.Views of the cord insertion.

9. Bladder views (long view to include the stomach, heart, and diaphragm).

I0. Femur views and evidence that all four extremities are present.

11. A sweep through the entire fetus and an informal biophysical profile.

12. Views of the placental site and amniotic fluid volume. (Roger c. sanders and Thomas c. winter., 2007)

2.2.2 Coupling Agent:

The best acoustic coupling agent is a water soluble gel. Many are commercially available, but they are usually expensive and sometimes

difficult to obtain. It is not necessary to use a particular coupling agent with specified equipment, even though manufactures often suggest that this is essential. Special coupling agents do not give a better image.

2.3 Estimation of gestational age:

2.3.1 Dating by the last menstrual period:

As mentioned earlier in introduction the traditional way to calculate the EDD has been by Naegele´s rule **.**This is done by adding 9 months and 7 days to the date of the first day of the LMP, or by reducting 3 months and adding one year and 7 days, with the assumption that a pregnancy lasts 280 days. This now old fashioned method is associated with considerable uncertainty on account of the major variations in menstrual cycle length. (Harlow & Ephross.,1995)

2.3.2Dating by ultrasound fetometry:

Multiple studies have demonstrated that routine use of ultrasound results in more accurate assessment of the EDD than last menstrual period (LMP) dating or physical examination, even in women with regular and certain menstrual dates. Pregnancy dating is most accurately performed in the first half of pregnancy. Fetal growth should be assessed by comparison to earlier scans in pregnancy. In a Cochrane review of nine trials of routine ultrasound in early pregnancy, routine use of early ultrasound and the subsequent adjustment of the EDD led to a significant reduction of Prolonged pregnancy. A rule of thumb is that in the first trimester, LMP dating should be maintained unless ultrasound yields an EDD more than 7 days off; in the second trimester, ultrasound should be used to change EDD if it is off by more than 2 weeks (and follow-up is then needed to ensure appropriate interval growth); and in the third trimester, a 3-week discrepancy between LMP and ultrasound dating is allowed, but needs to be taken into the clinical

context, with assessment for growth restriction or macrosomia, if appropriate (Rumack ,2011).

a) First trimester dating

The gestational sac is visible by vaginal ultrasound from about 4.5 weeks and can be correlated to GA(Daya, 1991). Measurements of the crownrump-length (CRL) in weeks 5-12 are the most accurate method of determining GA in the first trimester [Selbing, 1983; Wisser et al., 1994; Tunón et al., 2000]. In addition to dating, an ultrasound examination in this period provides important information about fetal viability, chorionicity in twins, placental localization, and to some degree also about fetal malformations.

In addition to the good accuracy of dating by a first trimester ultrasound scan, another advantage of early scans is the possibility of performing nuchal translucency (NT) measurements, as a tool for screening for Down syndrome and other abnormalities at 12-14 gestational weeks, there is an opportunity to visualize fetal anomalies earlier than at the time of the standard 18 to 20–week scan.(Rumack.,2011).

b) Second trimester dating:

When the CRL is more than 60 mm, measurements of the fetal biparietal diameter (BPD), femur length (FL) and abdominal circumference (AC) or abdominal diameter (AD) are more accurate for dating. BPD and FL are used in routine measurements for estimation of gestational age between 15 and 22 weeks (Persson & Weldner, 1986; Geirsson & Have, 1993). Particularly important when performing routine ultrasound examination in the second trimester is to scan for fetal malformations, which are best detected in this period. Ultrasound estimation of the gestational length is based on a standard growth curve and is not recommended after 20 completed weeks, as the variation in fetal growth increases after that time.

c) Third trimester dating:

The fetus has grown to approximately 15 inches in length and 1000 to 1400 g in weight by the beginning of the third trimester and the lungs, organs and vessels are maturing in preparation for birth. third trimester measurements of the fetal is biparietal diameter (BPD), femur length (FL) and abdominal circumference (AC) ,amniotic fluid volume and head circumference-to-abdominal circumference ratio (HC/AC) (During the early third trimester, the head circumference is slightly larger than the circumference of the abdomen and During the late third trimester, with the increase of fetal body fat, the abdominal circumference is typically equal to or slightly larger than the head circumference) and also estimated of fetal weight

(Most commonly calculated using the biparietal diameter, femur length, and abdominal circumference).(Ovel .,2014)

2.3.3 Fetal gender differences in size at ultrasound dating:

It is well known that by the time of ultrasound dating in the early second trimester, male fetuses are already larger than females. Male fetuses may thus be assigned a falsely longer gestational age and female fetuses a falsely shorter (Tunón et al.,1998).

Many investigators (Pedersen, 1980; Wald et al., 1986; Moore et al., 1988) ,but not all (Selbing & McKay, 1985), have found that the BPD of male fetuses is larger than that of female fetuses in early pregnancy. It has been reported that during this period the BPD of male fetuses is 0.8 to 1.1 mm larger than that of female fetuses, and that BPD grows by 0.44 mm per day (Persson et al., 1978).

Measurements of < 1 mm are within the error margin of the ultrasound technique and measurements of fractions of millimeters are currently not used in practice.

Some authors claim that fetal gender is one of many factors (including, parity and maternal age) influencing the accuracy of dating, but the differences are very small and of no clinical importance (Henriksen et al., 1995; Tunón et al., 1998). In an investigation comprising 571617 women, (Källén.,2002) concluded that male fetuses are more likely than female ones to be judged older than the LMP date suggested at early fetometry.

2.3.4 Impact of ethnicity:

Some studies have shown the pregnancy duration to be 2-8 days longer in the white race than in the black (Hendersson & Kay.,1967) but Collins et al.(2001) concluded that African Americans and Mexican Americans have higher postterm delivery rates than Whites. Ethnic group is only one of many demographic variables affecting the fetal size and growth pattern - others include parity and maternal height and weight, and fetal gender.

2.4 Prolonged pregnancy:

2.4.1 Definition:

Although they are used interchangeably – 'prolonged', 'post term' and 'post dates' are different terms used to signify a pregnancy that has extended beyond a certain duration accepted as the upper limit of normal. Besides the difficulties associated with usage of these overlapping terms, problems arise in interpretation of findings from various clinical trials, due to non-uniformity of the definition of prolonged pregnancy (varies between 41 completed to 43 completed weeks). The definition accepted by the World Health Organisation (WHO) is a pregnancy more than 42 completed weeks or 294 days from the first day of the last menstrual period (World Health Organisation.,2003).

2.4.2 Incidence:

The reported incidence of pregnancies reaching 42 weeks ranges from five to ten percent. The incidence of prolonged pregnancy depends on the population characteristics such as percentage of primigravid women, prevalence of obesity, proportion of women with pregnancy complications and the frequency of spontaneous preterm birth (Doherty L and Norwitz E ., 2008). Local hospital management protocols such as use of ultrasound for pregnancy dating, policy of IOL and elective caesarean deliveries also influence these numbers. In the United States (US) in 2005, 14% of all pregnancies progressed beyond 41 weeks of gestation and 6% progressed beyond 42 weeks of gestation. Recent years have seen a decline in the number of prolonged pregnancies largely due to increased use of ultrasound and IOL between 41 to 42 weeks. While in 1998 10% of all pregnancies in the US were prolonged, only 6% continued beyond 42 weeks in 2005(Martin JA et al.,2007). In 1992 a large Canadian multicentre randomised controlled trial compared IOL at 41 weeks with serial fetal monitoring and concluded that routine IOL at 41 weeks possibly reduced perinatal mortality and women's risk of caesarean section. This landmark study, which still forms the main basis for IOL at 41 weeks of pregnancy, has had a long lasting impact on the practice of routine IOL and incidence of prolonged pregnancies **(**Hannah ME et al ., 1992).

Women who have had a prolonged pregnancy have a 20% risk of recurrence in a subsequent pregnancy (Simpson PD, Stanley KP, 2011).

2.4.3 Diagnosis:

Accurate assessment of gestational age is critical for diagnosis of prolonged pregnancy. Reliance on standard clinical criteria such as history, fundal height or fetal heart tones to calculate the estimated date of delivery (EDD)

tends to overestimate the gestational age (Doherty L and Norwitz E., 2008). Traditionally, the expected day of delivery (EDD) has been calculated by using Naegele's rule (Naegele, 1836), with the assumption that a pregnancy lasts for 280 days from the start of the last menstrual period (LMP) to EDD, Calculations of gestation based on the first day of a patient's last normal menstrual period (LMP) are accurate only when the LMP is truly known rather than estimated and when a women's menstrual cycle is regular and predictable. In women with irregular cycles or oligoovulation and those conceived soon after stopping contraception, there is a risk of overestimation of the gestational age.

Measurement of the crown-rump length (CRL) during a first trimester ultrasound is a reliable and accurate method of estimating gestation which has been shown to decrease the incidence of prolonged pregnancy. A study found that the use of ultrasound reduced the incidence of prolonged pregnancy from 12 to 3% (Savitz DA et al .,2002). Another study by Gardosi and colleagues reported a postterm delivery rate of 9.5 % among women dated by LMP, but a rate of 1.5 % if ultrasound dating was used (Gardosi and others.,1997)

2.4.4 Risk factors:

Some of the risk factors are:

2.4.4.1 Primigravidity - First pregnancy has a higher chance of prolongation beyond 42 weeks (Olesen AW et al., 2003) .

2.4.4.2 Previous prolonged pregnancy - Women with a prior prolonged pregnancy have an increased risk of subsequent prolonged pregnancy (Olesen AW et al., 2003; Kistka ZA, et al., 2007) .

2.4.4.3 Family history of prolonged pregnancy - Genetic factors may be involved in the prolongation of pregnancy. Women who themselves were

born after a prolonged pregnancy have been shown to have prolonged pregnancies (Mogren I.,1999).

2.4.4.4 Male fetal gender - Male fetuses have been associated with prolongation of pregnancy (Divon MY et al.,2002).

2.4.4.5 Maternal obesity - Obesity has also been shown to be associated with increased chance of prolongation of pregnancy (Stotland NE et al.,2007).The exact mechanism by which body mass index (BMI) influences timing of labour is unknown, but differences in circulating levels of estradiol and progesterone may be involved.

2.4.4.6 Fetal abnormalities such as anencephaly or adrenal insufficiency - Fetal adrenal insufficiency or hypoplasia and fetal anencephaly (in the absence of polyhydramnios) are other rare contributors to prolonged pregnancy (Doherty L and Norwitz E.,2008). Both adrenal and pituitary glands are thought to play an important part in mechanism of onset of spontaneous labour at term hence their defects are associated with prolongation of pregnancy.

2.4.4.7 Placental sulfatase deficiency - Placental sulfatase deficiency is a rare X-linked recessive disorder that can prevent spontaneous labour due to a defect in placental sulfatase activity that results in decreased estriol levels (Doherty L and Norwitz E.,2008).

Recent evidence also suggests that post term neonates have reduced plasma cortisol levels compared to similar term neonates who have elevated cortisol levels following delivery. This may be due to an intrinsic difference in post term babies, reducing their response to the physiological and metabolic stress of labour and delivery compared to term babies. A relative adrenocortical insufficiency may contribute both to a delay in the onset of

labour and an increased risk of intrapartum hypoxia or death in prolonged pregnancy (Simpson PD, Stanley KP, 2011).

2.4.5 Pathogenesis:

The pathogenesis of postterm pregnancy is not clearly understood. As demonstrated above some risk factors associated with postterm pregnancy were identified with some possible explanations, however, the pathogenesis of the condition is not yet clear. Despite improved understanding of parturition in recent years, we still lack clarity about the exact mechanisms which initiate labour and allow its progression. To have a better understanding of the pathogenesis of postterm pregnancy it is essential to shed some lights on the pathophysiology of parturition and try to understand why these mechanisms fail to be triggered in postterm pregnancies or conversely are triggered earlier in preterm labour. It seems logical that a common ground or a link does exist between these three conditions. The mechanisms of parturition include interactions between hormonal, mechanical and inflammatory processes, in which placenta, mother and fetus each play a vital role.

Placental production of the peptide corticotrophin releasing hormone (CRH) has been related to the length of gestation (McLean et al., 1995). Synthesis of CRH by the placenta increases exponentially as pregnancy advances and peaks at the time of labour. In women who deliver prematurely the exponential rise is more rapid than those delivering at term, while in women who deliver postterm the rate of rise is slower (Ellis et al., 2002; Torricelli et al., 2006). This data suggests that postterm delivery is due to a change in the biological mechanisms regulating the length of gestation. This may be due to an inherited predisposition due to polymorphisms in the genes on the physiological pathway linking CRH to birth. It is also possible that the

maternal phenotype may change the response of maternal tissues to the usual hormonal signals to birth as may occur in the obese woman.

CRH can directly stimulate fetal adrenal production of DHEAs, the precursor for placental oestriol synthesis (Smith et al., 1998). Maternal plasma CRH concentrations correlate with oestriol concentrations (Smith et al., 2009). The rising oestriol driven by CRH increases at the end of gestation more rapidly than oestradiol levels leading to an increase in the oestriol to oestradiol ratio which has been postulated to produce an estrogenic environment in the last weeks of pregnancy. Concurrently the rise in maternal plasma progesterone concentrations that occurs across gestation slows at the end of pregnancy or even falls. This may be due to CRH inhibition of placental progesterone synthesis (Yang et al., 2006). Thus the pro-pregnancy effect of progesterone (promoting relaxation) is declining as the pro-labour actions of oestriol (promoting contraction) are increasing. These changes in ratios have been observed in preterm births, singletons delivering at term and in twin gestations (Smith et al., 2009). The situation in postterm pregnancies is unknown. It is likely to be similar in postterm women who go into spontaneous labour or those who respond to IOL, based on one study of postterm women (Torricelli et al., 2011).

2.4.6 Characteristics of prolonged pregnancy:

2.4.6.1 Prolonged placenta :

The classical explanation of the increased risk of perinatal mortality and morbidity in the prolonged period is a gradual impairment of placental function after term (Kitlinski.,2007).

There is a higher prevalence of placental infarcts and calcium deposition when pregnancy is prolonged. These morphologic changes are associated with the sonographic appearance of the Grade II and III placenta. At the

42nd week of pregnancy, 55% of patients will have Grade II placentas, and 45% will have Grade III placentas (only about 15% of term placentas are Grade II or III**)**

2.4.6.2 Amniotic fluid volume and amniotic fluid index:

Evaluation of amniotic fluid provides important information about fetal renal and placental function. Evaluation of amniotic fluid is a key component of fetal biophysical assessment. After 16 weeks, fetal urine production becomes the major source of amniotic fluid. (Rumack et al., 2011)

The amniotic fluid volume increases steadily from approximately 200 mL at 16 weeks of gestation to 980 mL at 34 to 35 weeks, where after it decreases to approximately 800 mL at 40 weeks and 540 mL at 42 weeks (Queenan et al., 1972). A progressive reduction of the fluid volume by 150 mL/week occurs between gestational weeks 38 and 43 (Elliott & Inman, 1961). Decreases in amniotic fluid volume can occur quickly during the postterm period (Clement et al., 1987).

The amniotic fluid volume can be estimated semiquantitatively by ultrasound measurements. The first ultrasound method to be introduced comprised measurement of only the largest vertical pocket of fluid (free of umbilical cord and fetal small parts) and The following classification has been proposed for the largest single pocket method: vertical depth of the pocket less than 2 cm indicates moderate to severe oligohydramnios, 2 to 8 cm is normal, and greater than 8 cm indicates polyhydramnios (Chamberlain, 1984). However, several investigators questioned the diagnostic accuracy of this method, and Phelan and colleagues (1987) therefore developed the amniotic fluid index (AFI) as a sum of vertical measurements of the largest amniotic fluid pocket in each of four quadrants, dividing the uterus into upper and lower segments and with the linea nigra

indicating the right and left halves. The measured pockets should not contain the umbilical cord. Oligohydramnios is then defined as an AFI of less than 5.0 cm at term, and polyhydramnios as an AFI of 20 cm or greater.

2.4.6.2.1 Oligohydramnios:

Prolonged pregnancies are associated with the development of oligohydramnios and non-reactive fetal heart rate patterns. One possible explanation for the oligohydramnios is decreased fetal renal perfusion due to impaired fetal cardiac function.

The alternative hypothesis for the reduction in renal perfusion and urinary output is redistribution in the fetal circulation, as in intrauterine growth restriction. Supportive evidence for impaired fetal renal perfusion as a cause of oligohydramnios in post-term pregnancies was provided by the study of Veille et al. who examined 50 pregnancies at or after 40 weeks of gestation. In the 17 with oligohydramnios (amniotic fluid index of less than 5 cm) impedance to flow in the fetal renal artery was significantly higher than in the 33 pregnancies with normal amniotic fluid (Veille et al.,1993).

A pronounced reduction of the amniotic fluid volume can occur rapidly and the fluid can even disappear within 24-48 hours. Measurements of AFI therefore need to be performed every second or third day, when AFI is > 50 mm, conservative management is common practice. To identify postterm fetuses that are at greatest risk, presence of oligohydramnios can be used alone (Crowley et al., 1984) or in conjunction with a fetal biophysical profile (Phelan et al., 1984)

2.4.6.3 Meconium stained amniotic fluid and meconium aspiration syndrome:

Meconium stained amniotic fluid is a result of passage of fetal colonic contents into the amniotic cavity. The overall incidence is approximately 12

%, but a figure as high as 30-40 % can be reached in postterm pregnancy (Ahanya et al., 2005). The underlying mechanisms are still not completely understood and are debatable, with a number of different theories. Saling (1968) proposed that passage of meconium is due to mesenteric vasoconstriction, causing hyperperistalsis and anal sphincter relaxation. Walker (1959) observed passage of meconium when the umbilical venous oxygen saturation decreased to below 30 %.

Other theories are that vagal stimulation secondary to cord compression can cause passage of meconium (Hon, 1963), and that presence of meconium in the amniotic fluid is a normal physiological phenomenon even in fetuses without distress

2.4.6.4 Fetal Macrosomia:

Large fetuses may be constitutionally large or they may be large as a consequence of a pathological process, such as maternal diabetes mellitus or obesity, or because of continued growth in prolonged pregnancy. Fetal macrosomia is not equivalent to growth acceleration or large-for-gestational-age (LGA), but occurs when a fetus has grown above a certain limit. There is no consensus on the definition of fetal macrosomia in the literature: some regard a fetal weight above 4000 g as macrosomia, others above 4500 g or 5000 g.

Even with sonographic measurements an accurate diagnosis of macrosomia can be difficult. The error range has been estimated to be up to 15 % (± 2 SD) and the information about estimated weight is therefore of limited clinical value (Gonen et al., 1997). The importance of recognizing macrosomia lies in the complications associated with this condition. These include increased maternal and fetal trauma, shoulder dystocia, perinatal

ashyxia, meconium aspiration, and postpartum hemorrhage (Chervenak et al., 1989).

2.4.7 Complications of prolonged pregnancies:

Prolonged pregnancies are associated with increased fetal and neonatal motality and morbidity as well as maternal morbidity. These risks are greater than it was originally thought. Risks have been underestimated in the past for two reasons. First, earlier studies on prolonged pregnancy were published before the routine use of ultrasound for pregnancy dating. As a result many pregnancies included in the studies was not actually prolonged, the second reason rests within the definition of stillbirth itself. Stillbirth rates were traditionally calculated using pregnancies delivered at a given gestational age rather than ongoing (undelivered) pregnancies. This would lower the stillbirth rates in prolonged pregnancies as once the fetus is delivered it is no longer at risk of intra-uterine fetal death (IUFD).

2.4.7.1 Fetal and neonatal risks:

The perinatal mortality rate displays a U-shaped curve with its nadir at 40 weeks (Campbell et al., 1997; Ingemarsson & Källén, 1997; Divon et al., 1998) (Figure 2.3)

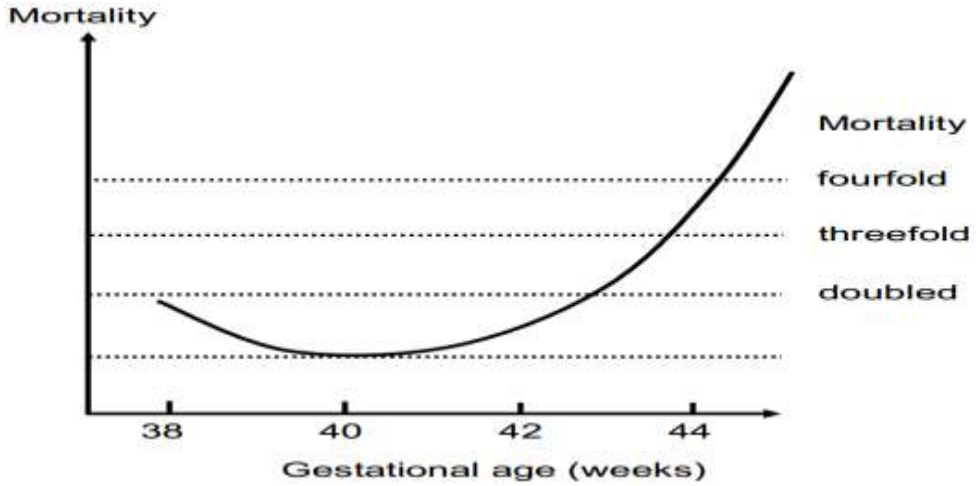


Figure (2.3) Perinatal mortality related to gestational age.

In older studies, with insufficient dating and restrictive use of induction of labor (IOL), the increase in mortality postdate was more steep than it is now days (Evans et al., 1963). The higher mortality is mainly due to intrauterine growth restriction (IUGR), suggesting that much effort should be made to identify such fetuses at an earlier gestational age. In uncomplicated cases with adequate fetal surveillance, the mortality figures in prolonged pregnancy are almost equal to those in term pregnancy (Dyson, 1988), but in the presence of fetal growth restriction the figures are five times higher in postdate than in term pregnancy (Divon et al., 1998). The mortality figures are also correlated to smoking, fetal growth restriction, and maternal age over 35 years (Raymond et al., 1994).

In comparison with term pregnancy, the dominating causes of fetal death in prolonged pregnancy are intrapartum asphyxia and aspiration of meconium (Hovi et al., 2006).

Not only the mortality but also the morbidity is increased in prolonged pregnancy (Divon et al., 1998). The perinatal morbidity panorama includes fetal distress, mainly explained by cord compression, neonatal seizures, meconium aspiration, pneumonia, and shoulder dystocia (Eden et al., 1987). In uncomplicated postdate pregnancy, the fetus gains weight continuously, increasing the risk of macrosomia with difficult labor and traumatic injuries such as cephalhematoma, fractures, and brachial plexus injury (Campbell et al., 1997).

2.4.7.2 Maternal risks:

Postterm pregnancy is associated with significant risks to the mother; there is an increased risk of:

1. Labour dystocia (9-12% versus 2-7% at term)

2. Severe perineal lacerations (3rd & 4th degree tears) related to macrosomia (3.3% versus 2.6% at term)

3. Operative vaginal delivery

4. doubling in caesarean section (CS) rates (14% versus 7% at term) (Rand et al., 2000; Campbell et al., 1997; Alexander et al., 2000; Treger et al., 2002).

Caesarean delivery is associated with higher incidence of endometritis, haemorrhage, and thromboembolic disease (Alexander et al., 2001; Eden et al., 1987).

2.4.8 Fetal Surveillance Program:

The purpose of antenatal fetal testing is to gain reassurance of fetal well-being and prevent fetal death. Therefore any abnormal testing should lead to an intervention either to gain additional reassurance or proceed for delivery , no tailor-made fetal surveillance methods are explicit for the postterm period, and traditional methods are therefore used.

The tests currently employed include:

2.4.8.1 Fetal movement charts – This is used as a screening test for further investigations. The woman is asked to choose a starting time (usually 9 am) and record how long it takes to feel 10 separate movements. If there have been less than 10 movements by 5 pm, she is asked to contact the hospital for further tests. There is great variation in what may be considered as normal and a change in the usual pattern of movements may be more important than absolute numbers. The value of routine movement counting is uncertain and a number of studies have failed to demonstrate any benefit.

2.4.8.2 **Non-stress test (NST) -** Antepartum recording of the fetal heart rate (FHR) continuously on a cardiotocography (CTG) trace for a period of 20 to 40 min, called the NST has become one of the most popular methods of

antepartum fetal surveillance including in prolonged pregnancy. A reactive test, defined as two or more accelerations of 15 beats/min or more, each lasting15 s or more and all occurring within 20 min of beginning the test, is considered to signify fetal well-being. The optimum frequency with which the NST should be performed to assess fetal wellbeing in prolonged pregnancy is not known, but it is generally performed twice weekly. The ability of the NST to detect poor perinatal outcome when used alone is low with a sensitivity of approximately 20% and specificity of 80% (Simpson PD, Stanley KP, 2011). NST is also not adequate to preclude an acute asphyxial event.

2.4.8.3 Contraction stress test (CST) - This not commonly used test, utilises oxytocin induced uterine contractions to assess fetal capacity to withstand stress of labour. It has the disadvantages of the technical difficulties related to the performance of the test and is time consuming (average 90 min). The high incidence of false-positive results can lead to unnecessary obstetric intervention.

2.4.8.4 Amniotic fluid volume estimation by ultrasound - Fetal compromise due to a gradual decline in placental function may be monitored by assessing the amniotic fluid volume. In prolonged pregnancy, an amniotic fluid index (AFI) <5 cm or <2 cm depth of the largest vertical pool is abnormal. In this situation there is a possibility of fetal compromise antenatally or intrapartum due to cord compression. Delivery of the fetus is desirable if the AFI is <5 cm beyond 42 weeks.

2.4.8.5 Biophysical profile (BPP) - The biophysical profile includes the NST plus ultrasound assessment of fetal movements, fetal tone, fetal breathing and quantification of the amniotic fluid. Each of the variables of the biophysical profile is scored 0 or 2, there being no intermediate score of

1. A score of 8 or 10 indicates a fetus in good condition. If the score is 6 then the test has to be repeated 4-6 h later and a decision made based on the new score. A score of 4 or less is an indication for delivery. It is a time consuming procedure and requires a trained ultra-sonographer.

2.4.8.6. Doppler flow studies:

Type of ultrasound that uses sound wave to measure the flow of blood through the blood vessel.

None of these tests has been shown to be superior to the others and there is no clear consensus in the literature regarding optimal fetal surveillance. Current NICE guidelines suggest twice-weekly cardiotocography and ultrasound estimation of maximum amniotic pool depth from 42 weeks gestation (NICE,2008)

2.5 Doppler ultrasound velocimetry:

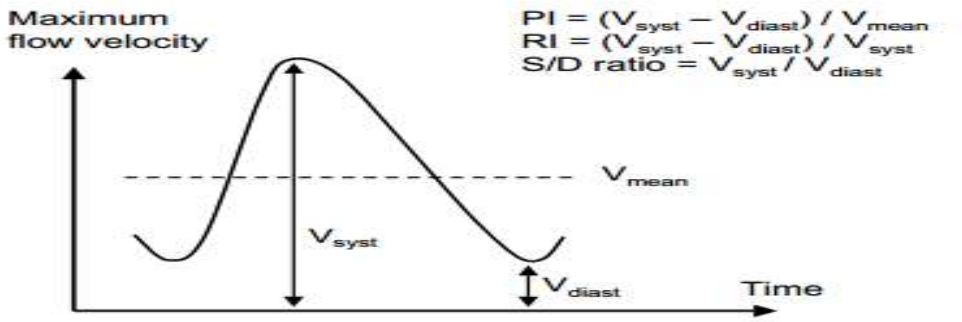
Christian Johann Doppler described the Doppler phenomenon in 1842, and the use of Doppler ultrasound in fetal surveillance was introduced in the 1970s (Fitzgerald & Drumm, 1977; McCallum, 1977).

The Doppler principle is based on changes in the frequency of wave energy when the energy is reflected by a moving object; the frequency shift being proportional to the velocity of the reflector. Different Doppler ultrasound techniques are used in obstetrics, namely continuous wave (CW) Doppler, pulsed wave (PW) Doppler, color Doppler imaging (CDI), and power Doppler (PD). Only PW Doppler ultrasound will be discussed here. PW Doppler transmits short pulse sequencies of ultrasound, where echoes from a moving object (blood cells) are received after a short interval of time, allowing measurements of blood flow velocity. After identification of the vascular anatomy with CDI, the PW technique allows assessment of flow in

a defined site of a defined vessel by placing the sampling volume at the site of interest.

Doppler velocimetry can be carried out in practically all vessels that can be identified with CDI. Numerous Doppler studies have been carried out by measuring blood flow in such vessels as the maternal uterine arteries, umbilical vessels, fetal aorta, renal arteries, and cerebral arteries and veins.

In the early era of obstetric Doppler velocimetry, FVWs were measured in the fetal descending aorta and the umbilical vein (Gill & Kossoff.,1979; Eik-Nes et al., 1984). Lingman & Maršál (1986) presented normal reference values during the third trimester. The peripheral vascular resistance to blood flow is described with different indices, such as the resistance index (RI) (Planiol & Pourcelot, 1974), the systolic-diastolic ratio (S/D) ratio (Stuart et al., 1980), and the pulsatility index (PI) (Gosling et al, 1971) (Fig. 2)



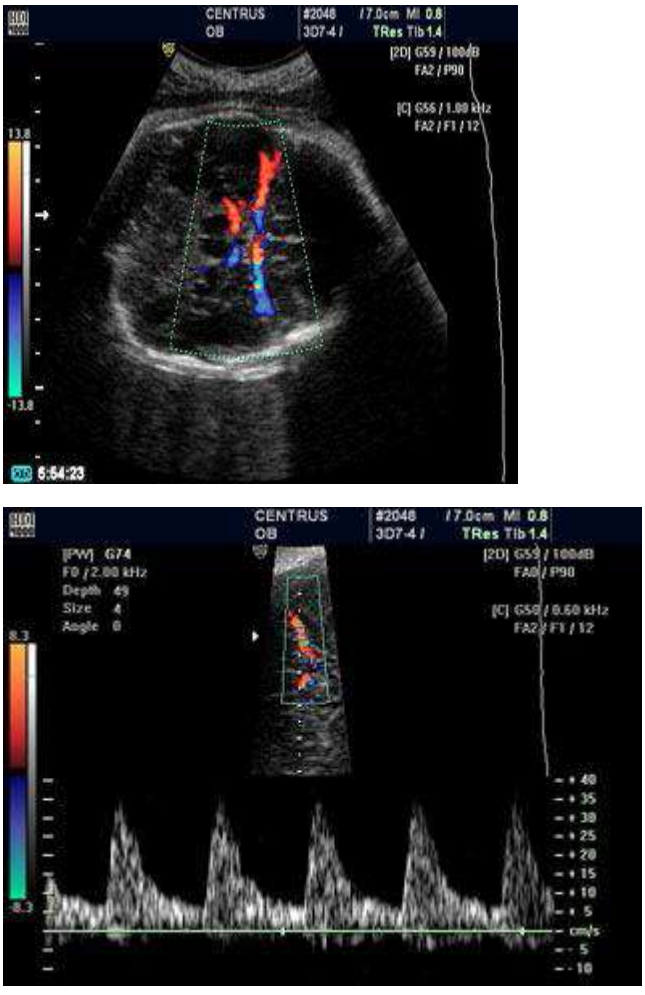
Figur (2.4) Flow velocity waveform analysis. Vsyst denotes peak systolic velocity;

Vdiast = minimum diastolic velocity; Vmean = mean velocity over the heart cycle;

PI = pulsatility index; RI = resistance index; S/D ratio = systolic/diastolic ratio.

2.5.1 Doppler velocimetry in the cerebral circulation:

Anatomy of the cerebral arterial circulation The anterior, middle, and posterior cerebral arteries supply the cerebral hemispheres. The fetal middle cerebral artery (MCA) measures 2-4 cm and is the largest branch of the circle of Willis, running laterally in the Sylvian fissure as a direct continuation of the internal carotid artery. MCA supplies most of the convexity of the cerebral cortex on each side of the hemispheres and the deep parts of the cerebrum, such as the basal ganglia and the internal capsule (Fig. 2.5)



Figure(2.5): Transverse view of the fetal head with color Doppler showing the circle of Willis (left). Flow velocity waveforms from the middle cerebral artery at 32 weeks of gestation (right).

2.5.1.1Cerebral fetal circulation and redistribution of blood flow: The cerebral blood flow (CBF) can be quantified as volume blood flow in milliliters per 100 g brain tissue weight per minute (mL/100 g/min). The CBF adapts to changes in the uteroplacental and umbilicoplacental circulations to maintain the cerebral oxygen supply, and is mainly regulated by changes in pO2 and pCO2. In situations of fetal hypoxia, blood flow is redistributed to vital organs such (the heart, brain, adrenal glands) and the CBF increases to protect the brain from hypoxic injury. In the cerebral arteries, this redistribution of blood flow can be recorded as a decreased vascular resistance in the fetal-MCA and anterior cerebral artery this "brain-sparing" phenomenon is prevalent in growth-restricted fetuses. In addition to development of a brain-sparing flow (BSF), other regulatory systems for protecting the brain from hypoxic injury are activated, such as an increased extraction of oxygen from erythrocytes in peripheral tissues, metabolic downregulation, and mobilization of glucose from glycogen stores (Kitlinski.,2007).

2.5.1.2 Flow velocity in the middle cerebral artery:

The first FVW recordings reflecting the fetal cerebral circulation were obtained from the common carotid artery (Marsál et al., 1984), the internal carotid artery, and MCA (Wladimiroff et al., 1986). Doppler measurements of MCA flow velocities can be carried out in the proximal, middle or distal part of the vessel .The PI tends to be lower in the proximal part than in the two distal parts (Locci et al., 1992; Hsieh et al., 2001). The clinical importance of different sample sites was addressed by Figueras and colleagues (2004), who concluded that the proximal site MCA PI significantly predicts UA pO2 but not pH at delivery, whereas the distal site MCA PI has a weak association with pH. Other observations have confirmed

the correlation between proximal MCA PI and hypoxia during labor (Kassanos et al., 2003).

Studies on MCA velocimetry in the postterm population have shown a continuous decrease in vascular flow resistance at the end of pregnancy (Kirkinen et al., 1987; Hsieh et al., 2001). This might be explained by a physiological change associated with an increase in cerebral metabolic requirements (Dobbing &Sands, 1970; Mari & Deter, 1992), or be secondary to a mild placental insufficiency with fetal hypoxemia, which may occur at this stage of gestation.

However, other studies have not verified any decrease in MCA PI (Battaglia et al.,1991) and suggest that the brain-sparing effect occurs only in situations with acute or chronic hypoxia. Devine and colleagues (1994) demonstrated that a low MCA to UA resistance ratio, also called the cerebroplacental ratio (CPR), is associated with an increased risk of fetal distress.

2.5.1.3measurement technique of the MCA:

Precise methodology is paramount in measurement of MCA indices. The Society for Maternal-Fetal Medicine recently published step-by-step guidelines for the proper technique of measurement of the MCA PSV.

Table(2.1):Methodological guidelines for Doppler assessment of the middle cerebral artery blood flow (SMFM,2015)

- **Obtain a transverse view of the fetal head at the level of the sphenoid bones. Brain landmarks include visualization of the thalami and cavum septum pellucidum.**
- **Visualize the circle of Willis using color Doppler.**
- **Visualize the middle cerebral artery originating off the circle of Willis. The entire length of the MCA should be seen.**
- **Select MCA closest to the transducer if feasible.**
- **Magnify the image such that the MCA occupies >50% of the image.**
- **Pulsed Doppler gate 1–2 mm.**
- **Adjust caliper gate over MCA. The PSV should be sampled close to its origin from the internal carotid artery.**
- **Adjust pulse-wave Doppler scale to fit velocity.**
- **The angle between the ultrasound beam and the direction of blood flow should be as close to 0° as possible, ideally <10°, and parallel to the entire vessel length. If this is not possible, the angle of insonation should be <30°.**
- **Obtain at least 5 uniform waveforms for measurement of PSV. The highest waveform should be used to measure the PSV.**
- **Ensure absence of fetal breathing and movement during measurement.**
- **Repeat sequence at least 3 times. The highest PSV value should be used for clinical care.**
- **Observe the As Low as Reasonably Achievable (ALARA) Principle during evaluation.**

2.5.2 Umbilical cord structure:

The fully developed umbilical cord normally contains two umbilical arteries, one umbilical vein, the remnant of the allantois all embedded in Wharton's jelly and surrounded by a single layer of amnion as shown in **Figure(2.6)** and **Figure(2.7).**

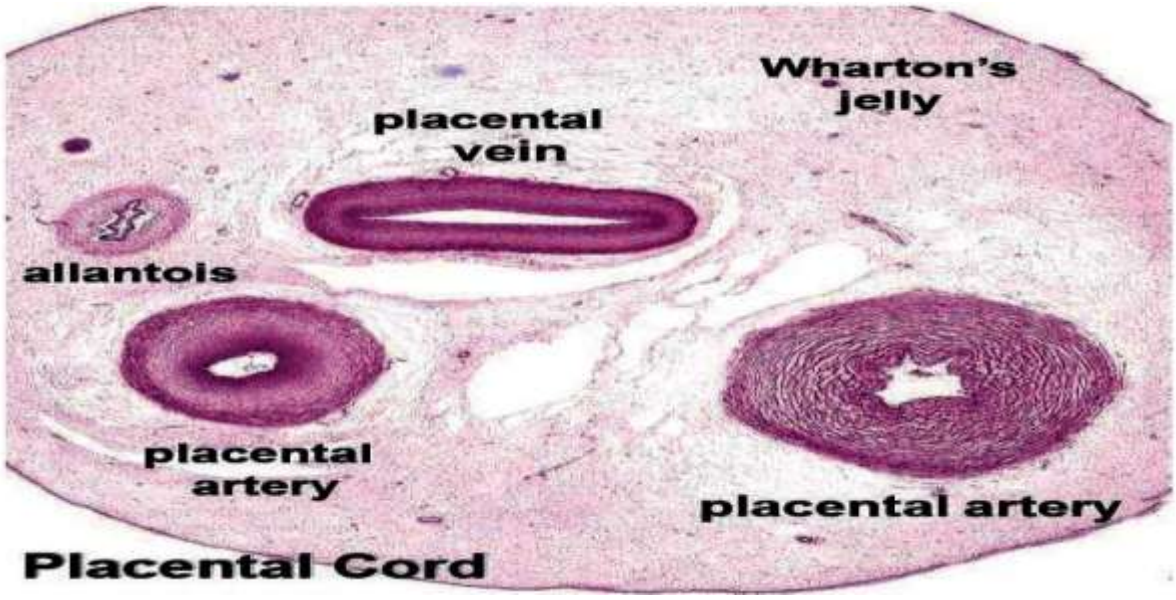
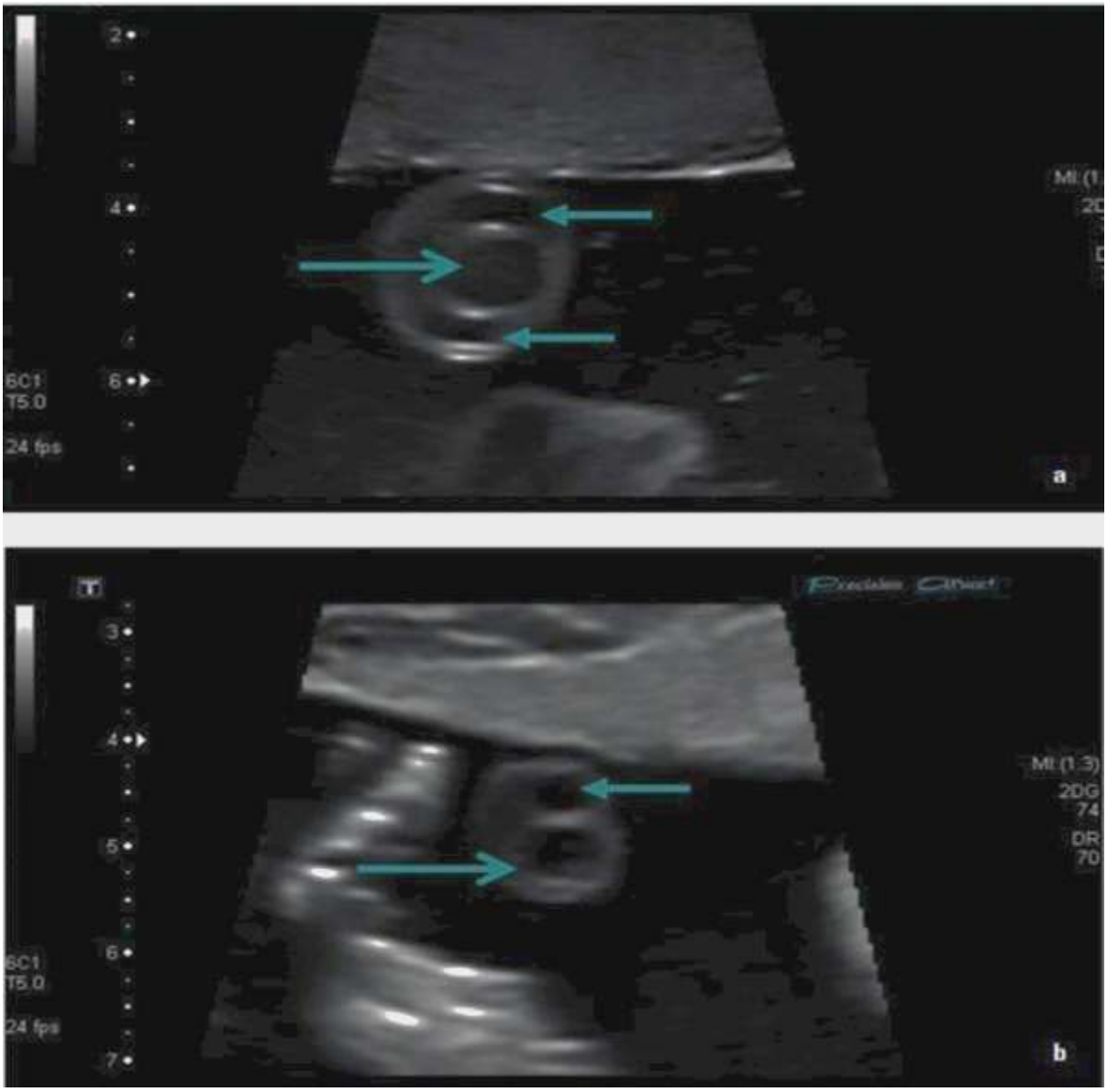


Figure (2.6): A cross-sectional image of a postpartum umbilical cord showing the major structures (Hill M. UNSW Embryology. Placenta Histology. Available from: http://embryology.med.unsw.edu.au/notes/placen-ta5.htm).



Figure(2.7): B mode images of a transverse section of the umbilical cord with (a) showing a normal three-vessel cord and (b) showing a single umbilical artery (←) and vein (→).

At term the umbilical cord has an average length of 50–60 cm. Normal cord length can range from 30 cm to 100 cm, with less than 30 cm considered short. Excessively long cords may be associated with prolapse, looping of the cord around the foetal neck, entanglement, distress and foetal demise. On the other hand very short cords may be associated with premature placental

separation, growth restriction, congenital abnormalities, foetal distress and demise.

The diameter of the umbilical cord vein increases from 4.1 mm at 20 weeks to 8.3 mm at 38 weeks gestation. demonstrated an increase in the cross-sectional area of the umbilical cord vein from 28 mm at 24 weeks gestation to a maximum of approximately 58mm between 34–38 weeks, followed by a slight decline from the 39th week. Another variation in the umbilical cord vein is a decrease in the diameter of vessel by approximately 1 mm between the placental and foetal ends.

The area of the umbilical cord vein is approximately 30% larger than the combined areas of the arteries and as such the velocity in the vein is approximately half the velocity in either artery,⁻with the velocity in the umbilical cord vein ranging from 10–22 cms/s.

The umbilical cord arteries and vein are unlike their counterparts in the remainder of the foetal body as the umbilical cord vein transports oxygenated blood to the foetal heart while the arteries return oxygen-depleted blood to the placenta. The walls of the umbilical cord artery lack an internal and external elastic lamina and the adventitia found in other arteries is replaced by mucous connective tissue. The umbilical cord vein has a thickened muscularis layer with intermingling circular, longitudinal and oblique smooth muscle fibres as well as an internal elastic lamina.

In some cases, one of the umbilical cord arteries may undergo atresia, aplasia or agenesis resulting in a single umbilical cord artery **(Figure 2b),** with the left umbilical artery being absent more frequently. A single umbilical artery may be associated with aneuploid foetuses, or with intrauterine growth restriction and renal anomalies in euploid foetuses.

The two umbilical arteries commonly form a cylindrical helix around the umbilical vein **Figure (2.8a)**. The normal umbilical cord has one coil per 5 cm of cord length. The umbilical cord may develop up to 40 spirals and there may be straight portions or reversal of spiral direction in different segments. In most cases the umbilical arteries twist over the vein, however, in 4.2% of cases the vein may twist around straight or hypocoiled arteries **Figure (2.8b).** The helices or frequently termed "spirals" of the umbilical cord are dextral in approximately 90% of cases and sinistral in the remaining. Spiralling is attributed to the helical muscle layers in the umbilical artery walls, however, foetal rotational movements, asymmetry in the sizes and growth rate of the umbilical arteries and asymmetrical contractions of the uterus have also been proposed as causes of umbilical cord helices. In the clinical setting uncoiled or hypocoiled umbilical cords have been associated with suboptimal pregnancy outcomes including increased incidence of interventional deliveries, higher cord pH and heart rate disturbances. (Spurway, Jacqueline et al 2012)

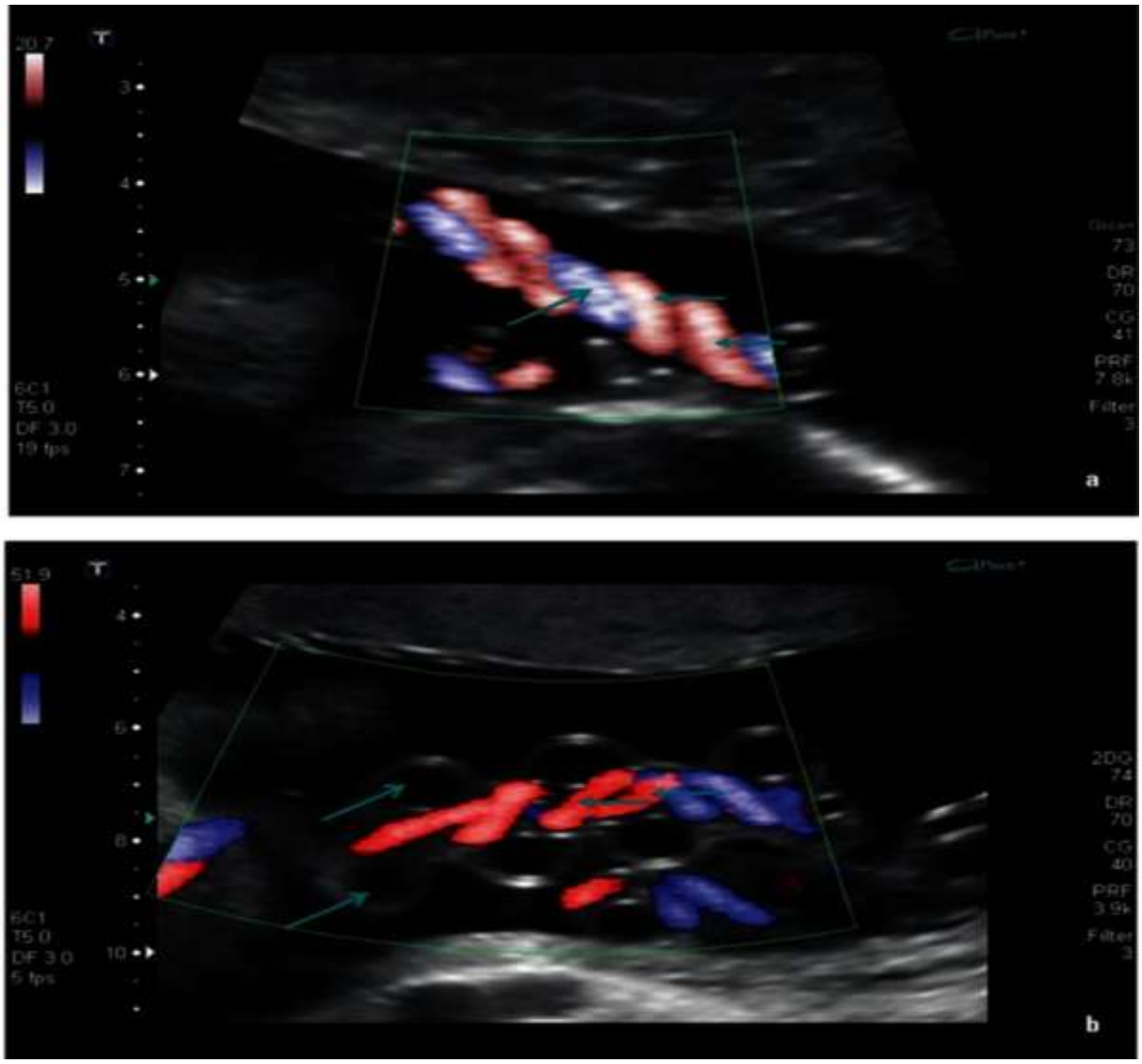


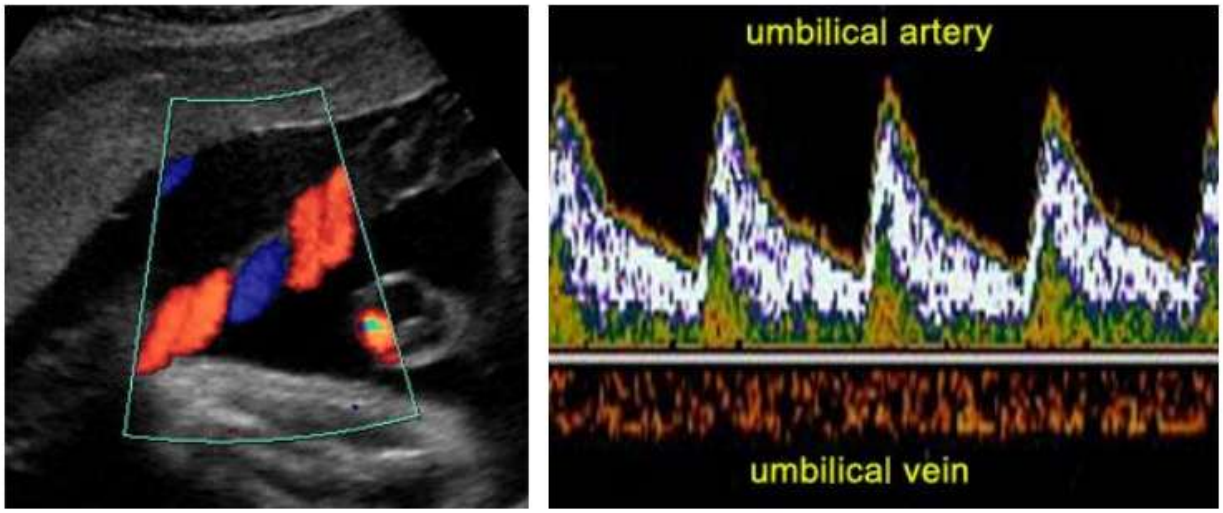
Figure3: Longitudinal images of the umbilical cord with (a) showing paired arteries (←) spiralling around the umbilical vein (→) and (b) showing the vein looping around central coiled arteries.

2.5.2.1 Umbilical Artery Flow:

The umbilical artery was the first fetal vessel to be evaluated by Doppler velocimetry. Flow velocity waveforms from the umbilical cord have a characteristic saw-tooth appearance of arterial flow in one direction and continuous umbilical venous blood flow in the other. Continuous wave Doppler examination of the umbilical artery is simple. The transducer,

usually a pencil-shaped probe, is placed on the mother's abdomen overlying the fetus and is systematically manipulated to obtain the characteristic waveforms from the umbilical artery and vein

With a pulsed wave Doppler system, an ultrasound scan is first carried out, a free-floating portion of the cord is identified and the Doppler sample volume is placed over an artery and the vein Figure (2.9)



Figure(2.9) : Ultrasound image with color Doppler showing the umbilical cord, red umbilical artery and blue umbilical vein (left). Normal flow velocity waveforms from the umbilical vein (bottom) and artery (top) at 32 weeks of gestation (right).

The location of the Doppler sampling site in the umbilical cord affects the Doppler waveform and the impedance indices are significantly higher at the fetal end of the cord than at the placental end. A possible explanation for this finding is that the fetal placental vascular bed is a low impedance system associated with minimal wave reflection, which explains the presence of continuing forward flow in the umbilical artery during diastole. The closer the measurement site is to the placenta, the less is the wave reflection and the greater the end-diastolic flow.(Maulik D, Yarlagadda P et al., 1990)

There are no significant day-to-day variations in pregnancies with normal umbilical arterial Doppler waveforms. Umbilical venous blood flow increases with fetal inspiration and decreases with expiration. There is also a breathing-related modulation of arterial pulsatility, and umbilical artery Doppler studies should be avoided during fetal breathing. Maternal exercise may cause an increase in fetal heart rate but mild to moderate exercise does not affect flow impedance in the umbilical artery. Umbilical arterial flow waveforms are not affected by fetal behavioural states (sleep or wakefulness). Although, in certain pregnancy disorders (such as pre-eclampsia), fetal blood viscosity is increased, the contribution to the increased impedance in the umbilical artery from viscosity is minimal compared to the coexisting placental pathology. Therefore, the viscosity of fetal blood need not be considered when interpreting the umbilical Doppler indices.

With advancing gestation, umbilical arterial Doppler waveforms demonstrate a progressive rise in the end-diastolic velocity and a decrease in the impedance indices Figure(2.10).

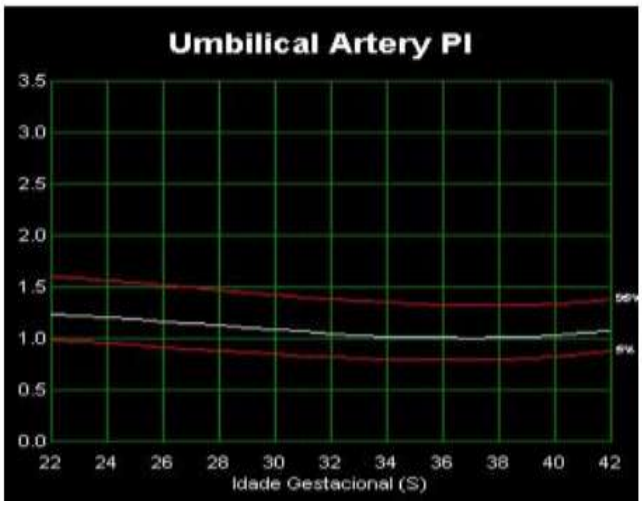


Figure : Pulsatility index in the umbilical artery with gestation (mean, 95th and 5th centiles).

When the high-pass filter is either turned off or set at the lowest value, end-diastolic frequencies may be detected from as early as 10 weeks or in normal pregnancies they are always present from 15 weeks. Human placental studies have demonstrated that there is continuing expansion of the fetoplacental vascular system throughout the pregnancy. (Maulik D., 1989)

2.5.2.2measurement technique of the umbilical artery:

Standard technique should be employed in obtaining the umbilical artery waveform to ensure reproducibility and accuracy of measurements (Table 2.2):

Table(2.2):Methodological guidelines for Doppler assessment of the umbilical artery blood flow that recently published The Society for Maternal-Fetal Medicine(SMFM,2015)
➢ Locate a free loop of uncompressed cord ➢ Identify umbilical artery, using colour Doppler as necessary ➢ Magnify until the loop of cord fills the majority of the image. ➢ Pulsed Doppler gate 1–2 mm ➢ Adjust caliper gate over single umbilical artery. ➢ Adjust power Doppler scale to fit velocity. ➢ Obtain at least 5 uniform waveforms for measurement of indices ➢ Ensure absence of fetal breathing and movement during Measurement ➢ Observe the As Low as Reasonably Achievable (ALARA) principle during evaluation

2.6 Previous Studies:

Rightmire and Campbell (1987), examined 35 pregnancies at more than 42 weeks of gestation and reported that impedance to flow in the uterine and umbilical arteries did not change with gestation, but impedance in the umbilical artery was higher in foetuses with a worse clinical outcome.

Devine et al (1994), evaluate pregnant women of 41 or more weeks' gestation with singleton fetuses and vertex presentations underwent antepartum testing twice a week. Pulsed Doppler ultrasound was used to obtain the flow velocity waveforms from the umbilical and middle cerebral arteries. Adverse post-date related outcome was defined as the occurrence of meconium aspiration syndrome, cesarean delivery for fetal distress, or fetal acidosis. The predictive values of an AFI equal to or less than 5 cm, a biophysical profile score equal to or greater than 6, a nonreactive NST, and a middle cerebral artery to umbilical artery ratio less than 1.05 in identifying adverse outcome were compared. The results was Forty-nine women met the inclusion criteria; ten (20.4%) had an adverse outcome. A middle cerebral artery to umbilical artery ratio of less than 1.05 was found to be the best predictor of adverse outcome, with a sensitivity of 80%, specificity of 95%, positive predictive value of 80%, and negative predictive value of 95%. The other three diagnostic tests had sensitivities equal to or less than 40%. The middle cerebral artery to umbilical artery ratio was also a better discriminator of adverse outcome than either the umbilical artery systolic-diastolic (S/D) ratio or the middle cerebral artery S/D ratio. Their conclusion was that a middle cerebral artery to umbilical artery ratio of less than 1.05 is an accurate method of predicting post-date-related adverse outcome.

Anteby et al (1994), examined 78 women at more than 41 weeks of gestation, who had normal non stress test and amniotic fluid volume. Pregnancies that subsequently developed signs of fetal distress during labor had increased impedance in the umbilical artery, decreased impedance in the fetal middle cerebral artery, and decreased time averaged velocity in the fetal aorta. It was concluded that, in uncomplicated post-term pregnancies, those with abnormal Doppler results are prone to need intervention following fetal distress in labor.

Battaglia et al (1995),compared 16 pregnancies at 40 weeks with 16 pregnancies at more than 41 weeks. In the post-term pregnancies, the time-averaged maximum velocity of the fetal descending thoracic aorta and the ratio of the impedance in the middle cerebral artery to that in the umbilical artery were decreased. Furthermore, post-term pregnancies were associated with an increased incidence of oligohydramnios, increased plasma viscosity and coagulation parameters (decreased fibrinogen, antithrombin III and platelet number). It was concluded that postterm pregnancy may mimic a mild 'fetal growth restriction'.

Some studies suggested that the pathophysiology of placental insufficiency in post-term pregnancies differs from that observed in cases of fetal growth restriction at earlier gestational ages, because, in post-term pregnancies, both placental and fetal Doppler indices are normal. Thus, **Farmakides et al (1988)** examined 149 pregnancies at more than 41 weeks of gestation and

reported that impedance to flow in the uterine and umbilical arteries was not altered, even in the presence of other signs suggestive of fetal compromise.

Similarly, **Stokes et al (1991)**, examined 70 pregnancies at more than 41 weeks of gestation and reported that impedance to flow in the umbilical and uteroplacental arteries was not significantly different in pregnancies associated with fetal compromise and abnormal neonatal outcome from those with normal outcome .

Zimmermann et al (1995), examined 153 pregnancies at 41–43 weeks of gestation and reported that impedance to flow in the umbilical artery, uteroplacental arteries and fetal middle cerebral artery did not change significantly within this gestational range. The majority of Doppler measurements in pregnancies with subsequent asphyxia or otherwise complicated fetal outcome were within the 95% prediction interval for patients with normal fetal outcome. This study also reported that, in the prediction of asphyxia, the sensitivity for oligohydramnios and antepartum cardiotocography was less than 20%

Bar-Hava et al(1995),examined 57 pregnancies at more than 41 weeks of gestation. They measured impedance to flow in the umbilical arteries and the fetal middle cerebral and renal arteries. In 15 pregnancies, there was oligohydramnios and, although in this group the mean birth weight was significantly lower than in the 42 pregnancies with normal amniotic fluid, there were no significant differences between the groups in the Doppler indices. It was concluded that, in post-term pregnancies, oligohydramnios is not associated with a major redistribution in the fetal circulation.

Olofsson et al(1996), examined 34 pregnancies that delivered after 43 weeks of gestation and reported that, at this gestation, compared to 40 weeks, the mean flow velocity and volume flow in the fetal aorta were lower, the flow velocity in the umbilical vein was higher, impedance to flow in the umbilical artery was lower and impedance to flow in the uterine artery was not different. It was suggested that these findings are compatible with physiological circulatory alterations enhancing continued fetal growth until the late post-term period. There were no signs of any general circulatory deterioration.

Also another study done by **Olofsson et al (1997)**, in a subsequent study, these authors examined 44 pregnancies at 42–43 weeks of gestation. In cases that developed fetal distress in labor, the umbilical artery pulsatility index (PI) was significantly decreased and the fetal aortic volume flow significantly increased; uterine flow was not significantly different. It was suggested that, in post-term pregnancies, subclinical fetal hypoxia may trigger vasodilation of placental vessels (with consequent decrease in umbilical artery PI) and indicates an increase of cardiac output with consequent increased aortic volume flow

Gupta Usha et al (2006), evaluated thirty-one pregnant women at 40 weeks or more of gestation with singleton fetus in vertex presentation were enrolled in the study. They underwent biweekly Doppler velocimetry studies of the middle cerebral artery and the umbilical artery of the fetus and from this, the CU ratio was calculated. Routine antepartum tests of fetal surveillance like non stress test, amniotic fluid index and biophysical score were done.

Pregnancies were terminated if any of the routine tests of fetal surveillance were abnormal or when the period of gestation extended beyond 42 weeks. Adverse perinatal outcome was defined as fetal bradycardia or tachycardia requiring cesarean section, presence of meconium stained liquor, fetal acidosis, and meconium aspiration syndrome admission to neonatal intensive care unit and perinatal morbidity and mortality. They found that in 31 women included in the study, 5 (16.1%) had an adverse perinatal outcome. The middle cerebral artery pulsatility index or umbilical artery S/D ratio when used alone had poor predictive value for adverse perinatal outcome.
However when the predictive efficacy of CU ratio was calculated, a cut-off value of 1.3 had a sensitivity of 80% and negative predictive value of 93.3% for predicting adverse perinatal outcome in postdate pregnancies. This assured the obstetricians of the fetal wellbeing. The specificity and positive predictive value of CU ratio however were low, being 53.8% and 25% respectively. Its false positive rate was also high (46%) and they conclude although CU ratio of 1.3 assures the obstetrician of fetal wellbeing, its low specificity and high false Positive value can lead to unnecessary tests and intervention. Hence it is not an ideal test for routine antepartum fetal surveillance in low risk postdate pregnancies.

Subhra Ghosh et al (2016), study 80 pregnant women who are at or beyond 40 weeks of gestation. Doppler indices of umbilical artery and middle cerebral artery were taken. Data were analyzed with obstetrics and perinatal outcome. They found that the Umbilical artery mean RI increased with gestational age (p=0.003). There was no significant difference in PI and S/D ratio in different gestational age groups. Middle cerebral artery Doppler indices did not show any significant difference in different gestational age

groups. In abnormal Doppler group, perinatal outcome was also not significantly different, but neonatal intensive care unit admission was increased, which was statistically significant (p=0.007). They conclude that vascular resistance in the umbilical artery and middle cerebral artery does not change abruptly when gestation exceeds 280 days. It also cannot be taken as the sole method of fetal surveillance when date is crossed.

Chapter Three
Materials and Methods

3.1. Materials:

3.1.1. Study type:

This was a prospective study carried out in the area of the study. Each participant was scanned, list the findings in details according to the study variables recorded in the data collecting sheets.

3.1.2. Study area:

The study was conducted in khartoum state, in the ultrasound departments of Khartoum Bahri Teaching Hospital at the Department of Obstetrics and Gynecology.

3.1.3. Study duration:

From August 2016 to December 2018.

3.1.4. Sample Size:

was caculated by using the following formula

$$n= N\div 1+N(d)^2$$

$n= 33000000\div 1+33000000 (+ \text{ or } - 0.05)^2$

n= 400

n: Sample Size.

N: population in khatoum State.

D: Degree of precission.

3.1.5 Sampling:

The study was conducted on volunteers, selection of participants through simple random sampling and then the data was collected from the participants.

3.1.6 Study population:

The study population was comprised of 197 pregnant women (64 postterm and 133 Normal pregnancies as control group). The two groups were compared according to fetal outcome through the Doppler parameters of the umbilical artery and the middle cerebral artery.

3.1.7 Inclusion criteria:

Normal third trimester pregnancies from 33 weeks onwards.

3.1.8 Exclusion criteria

Diagnosed fetal anomaly before recruitment, multi-fetal pregnancy, history of maternal smoking**,** known complications in the current pregnancy before recruitment, history of any pre-existing maternal medical condition (such as hypertension, diabetes mellitus, renal disease) likely to affect the fetus, cases which develop complication during the present pregnancy during the study period, and inability to obtain perinatal data.

3.1.9 Equipment used for the data collection:

ultrasound machine with capability for Doppler ultrasound with multi-frequency curved linear array transducer (3.5-5 MHZ): Mindray Dc-60 Exp.

3.2 Methods:

3.2.1. Data collection:

The Data were collected by data collecting sheet spesificaly design for the study and including study variable .

3.2.1.1. Source of date collection:

The participant history and data collecting sheet which designed specially for this study.

3.2.2. Ultrasound technique:

All cases were subjected to duplex Doppler examination after the findings of the biometry were confirmed, using a 3.5-MHz curvilinear transducer. Doppler waveforms were recorded from the fetal MCA and UA over three consecutive cardiac cycles.

The patients were examined in semirecumbent position with the fetus in a resting and apneic state. Spectral waveforms were obtained with 4 mm sized sample volume using a medium filter.

• **MCA PI and RI:** The MCA nearer to the probe was identified in each case using color Doppler. Spectral trace was obtained from the MCA immediately after its origin with a sample volume of 4 mm. Angle correction was done in each case and it was ensured that the angle of insonation was between 0 and 60°. PI and RI were measured both manually as well as in auto mode over three consecutive cardiac cycles. The measurements were repeated and two successive readings showing same results were noted for the study.

• **UA PI and RI:** UA was identified in each case using color Doppler. Spectral trace was obtained with a sample volume of 4 mm from the free loop of the UC. In case of difficulty in obtaining the free loop of the UC, the placental insertion of the cord was tracked along to help localizing the free loop. Angle of insonation was maintained between 0 and 60°. PI and RI

were measured both manually and in auto mode over three consecutive cardiac cycles. The measurements were repeated and two successive readings showing same results were finally noted for the study.

• **MCA PI/UA PI ratio:** After ensuring technically satisfactory examination and measurements, the ratio of the MCA PI to the UA PI (CP ratio) was calculated in each case and noted.

3.2.3. Data analysis:

After collecting , the data sheets was coded, classified and analyzed by statistical package for social sciences program of computer version 16 (SPSS), Mean ± Std. Deviation for each measurement was taken then correlation, regression and independent sample t-test were done to assess relation ship and correlation between variables of study (P value significant if less than 0.01 and 0.05 respectively).

3.2.4. Data presentation:

The data was presented as complex tables that was used in the analysis, the frequences were calculated and was carried out the relationship between different variables and the important statistical indicators were drawn from the study.

3.2.5. The study variables:

The study population assessed against the following variables:

-Characteristic of person : Age, Parity, Occupation, LMP

-Specific information: the S/D ratio, pulsatility index(PI) , resistance index (RI) and middle cerebral artery to umbilical artery (CU) ratio of the middle cerebral and umbilical artery.

-Catogorical variable : fetal outcome e.g : macrosomia , Shoulder dystocia and Meconium aspiration syndrome.

3.2.6. Ethical considerations:

The study was not posed any type of risk to the participants. The privacy was protected and the confidentiality was maintained by extracting or dismissing participants personal information. All participant's specific information was settled in compressed dics (CDs), personal computer and box files. All participants were payed a verbal consent and expressing their agreement to be included with in the study , There was official written permission to the ultrasound departments of Khartoum Bahri Teaching Hospital to the Department of Obstetrics and Gynecology to take the data.

Chapter four

Results

Table (4.1) frequency distribution of age \years

Age\years	Frequency	Percent	Valid Percent	Cumulative Percent
17-23	47	23.9	23.9	23.9
24-30	91	46.2	46.2	70.1
31-37	43	21.8	21.8	91.9
38-45	16	8.1	8.1	100.0
Total	197	100.0	100.0	

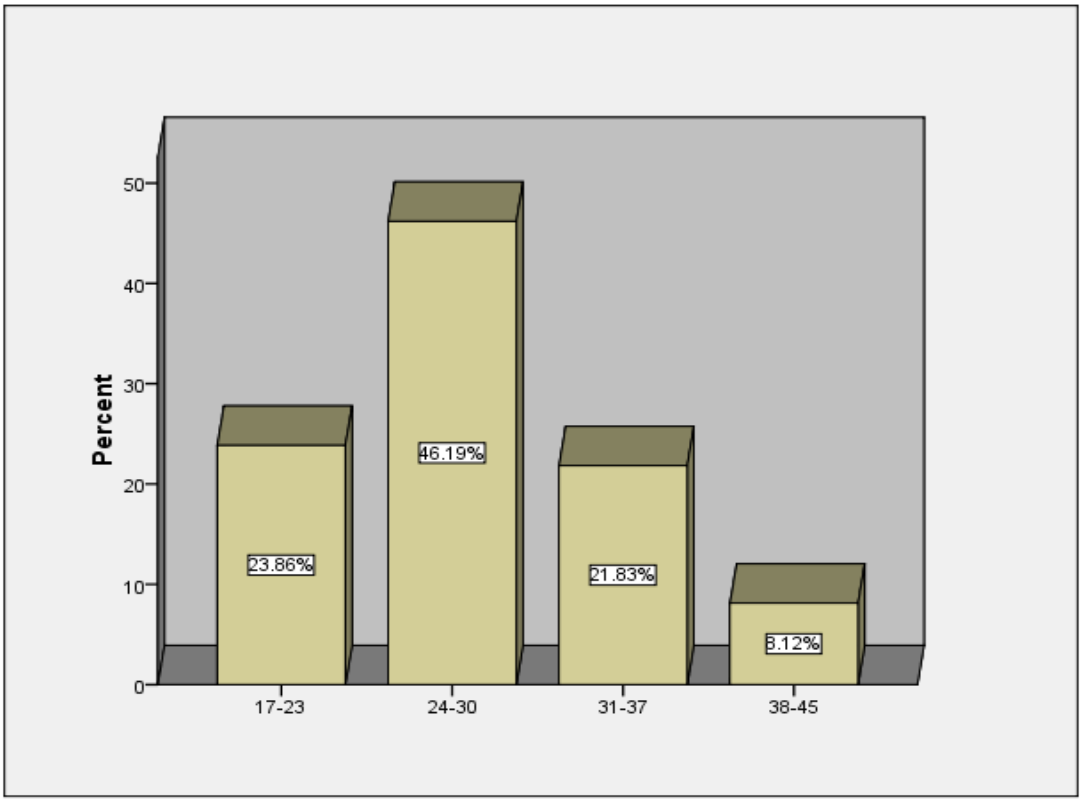


Figure (4.1) frequency distribution of age \year

Table (4.2) frequency distribution of parity

Parity	Frequency	Percent	Valid Percent	Cumulative Percent
Nulliparous	155	78.7	78.7	78.7
Multiparous	42	21.3	21.3	100.0
Total	197	100.0	100.0	

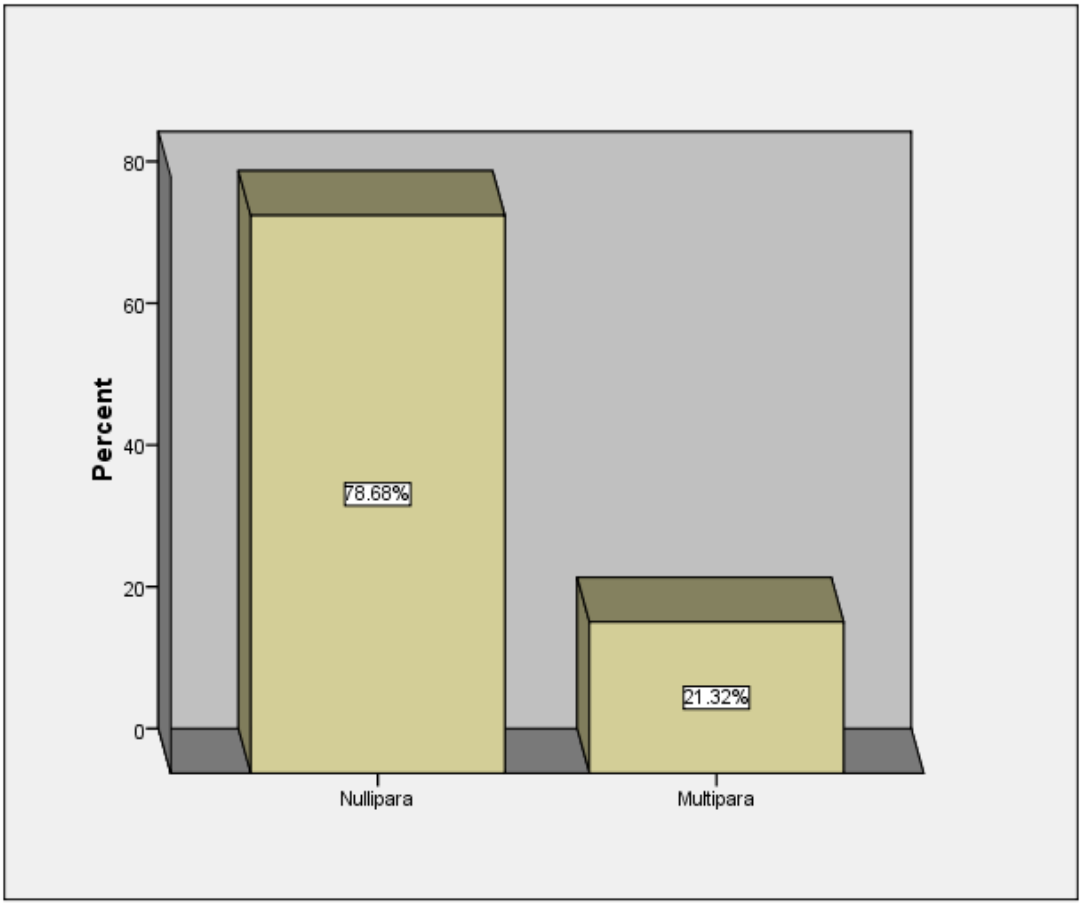


Figure (4.2) frequency distribution of parity

Table (4.3) frequency distribution of occupation

Occupation	Frequency	Percent	Valid Percent	Cumulative Percent
Employee	45	22.8	22.8	22.8
non-employee	152	77.2	77.2	100.0
Total	197	100.0	100.0	

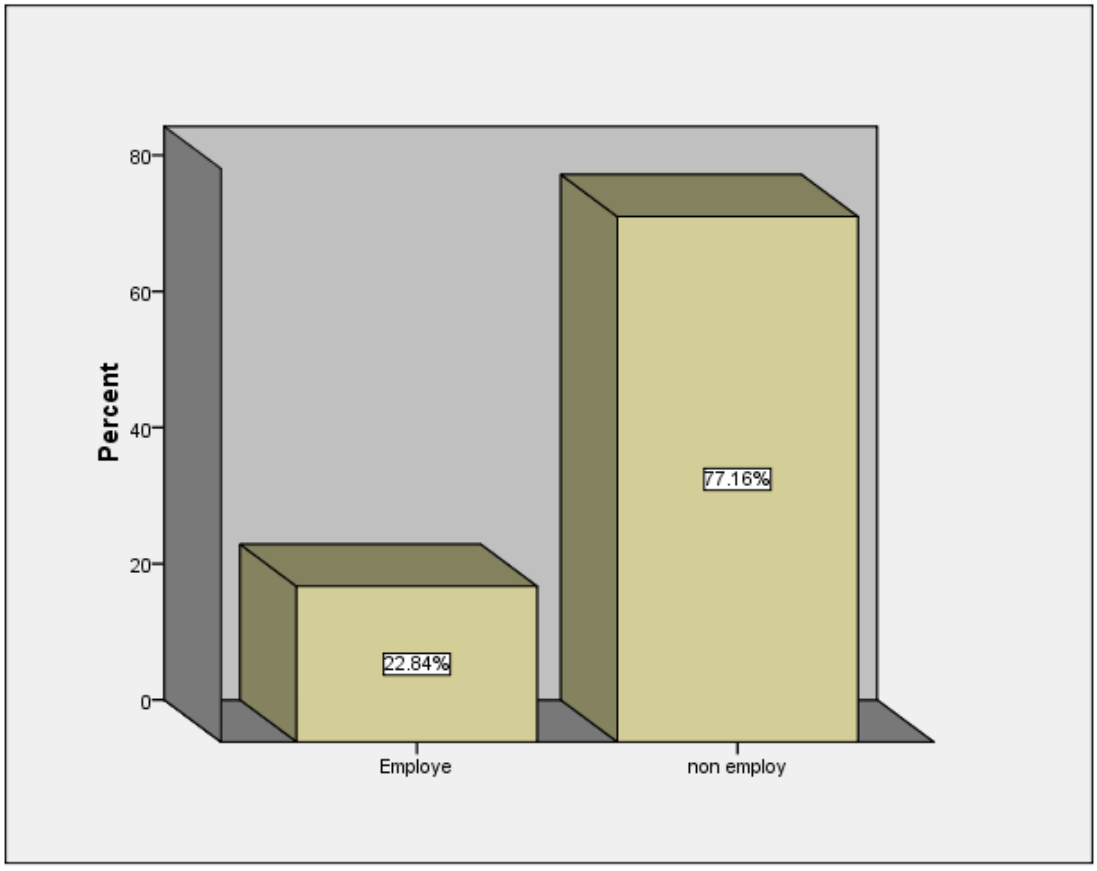


Figure (4.3) frequency distribution of occupation

Table (4.4) frequency distribution of presentation

Presentation	Frequency	Percent	Valid Percent	Cumulative Percent
Cephalic	184	93.4	93.4	93.4
Breech	13	6.6	6.6	100.0
Total	197	100.0	100.0	

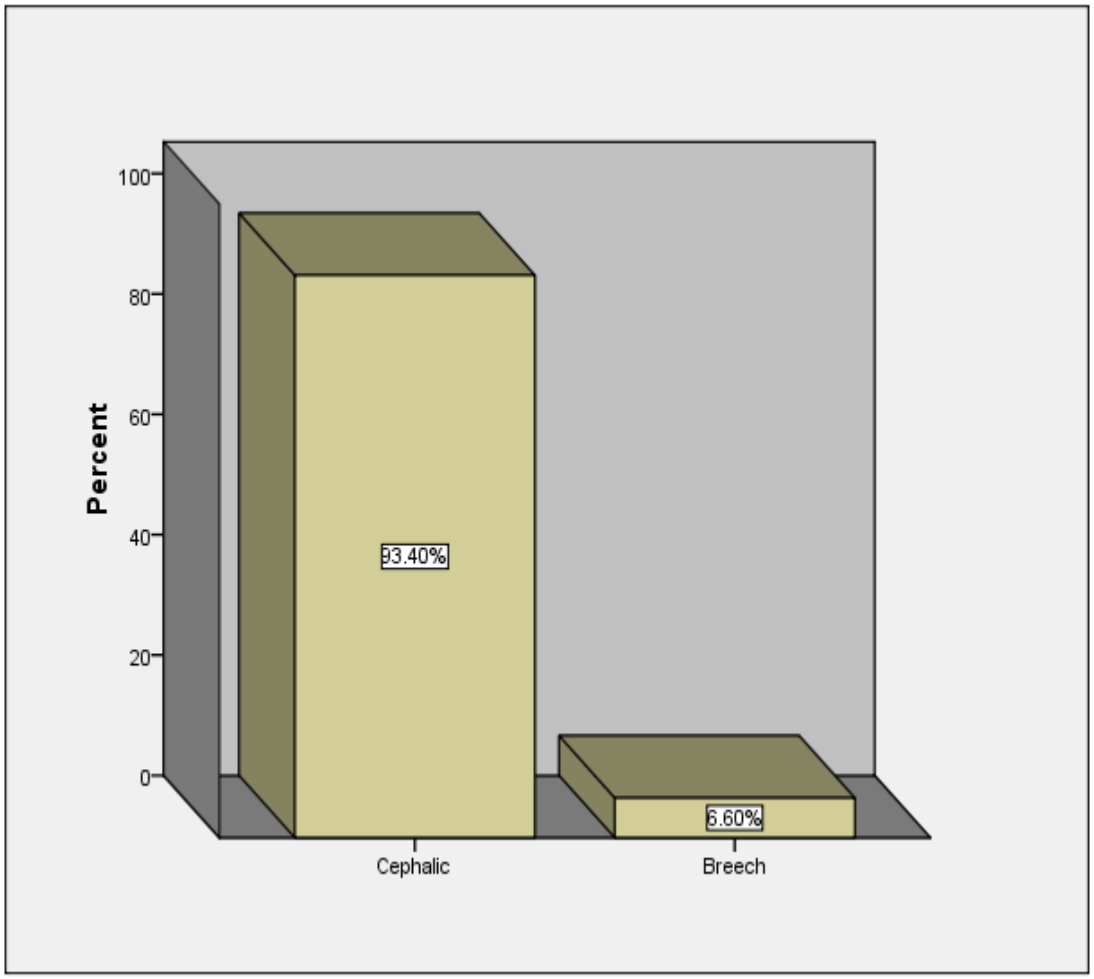


Figure (4.4) frequency distribution of presentation

Table (4.5) descriptive statistic for age and measurements of GA ,Doppler indices of MCA and UA (minimum ,maximum ,mean ± Std. Deviation)

Variables	N	Minimum	Maximum	Mean	Std. Deviation
age	197	17	45	28.07	6.076
FL\ cm	197	5.85	9.62	7.3541	.45261
BPD\ cm	197	7.24	92.70	9.6027	5.97051
AFI	197	0.8	12.0	5.403	2.1227
Ac\ cm	197	24.65	38.07	33.5860	2.56343
EFW	197	1.41	4.44	3.2305	.59814
GA LMP	197	33.43	46.43	39.0753	2.27973
GA FL	197	29.00	41.71	37.6214	2.09028
GA BPD	197	29.00	41.71	37.2923	2.15982
GA AC	197	29.00	42.00	37.5158	2.52691
MCA PSV	197	8.48	57.70	21.3292	8.46838
MCA EDV	197	3.18	34.44	9.6181	4.32694
UA PSV	197	12.7	65.2	29.449	8.8804
UA EDV	197	5.0	43.4	16.184	5.2817
RI MCA	197	.08	3.67	0.57	.30
PI MCA	197	0.21	4.67	1.25	0.73
S\D MCA	197	1.08	6.14	2.3950	.84883
RI UA	197	0.10	1.82	0.46	0.18
PI UA	197	.15	3.96	0.80	0.46
S\D UA	197	1.11	5.08	1.88	0.46
Ratio (PI MCA\PI UA)	197	0.23	7.66	1.87	1.31
Valid N (listwise)	197				

Table (4.6) frequency distribution of outcome

Adverse outcome	Frequency	Percent	Valid Percent	Cumulative Percent
C\S	68	34.5	34.5	34.5
polyhydramnios	4	2.0	2.0	36.5
oligohydramnios	5	2.5	2.5	39.1
macrosomia	3	1.5	1.5	40.6
macrosomia and C\S	5	2.5	2.5	43.1
C\S and polyhydramnios	6	3.0	3.0	46.2
normal outcome	101	51.3	51.3	97.5
oligo and C\S	2	1.0	1.0	98.5
macrosomia, C\S and polyhydramnios	2	1.0	1.0	99.5
prenatal mortality	1	.5	.5	100.0
Total	197	100.0	100.0	

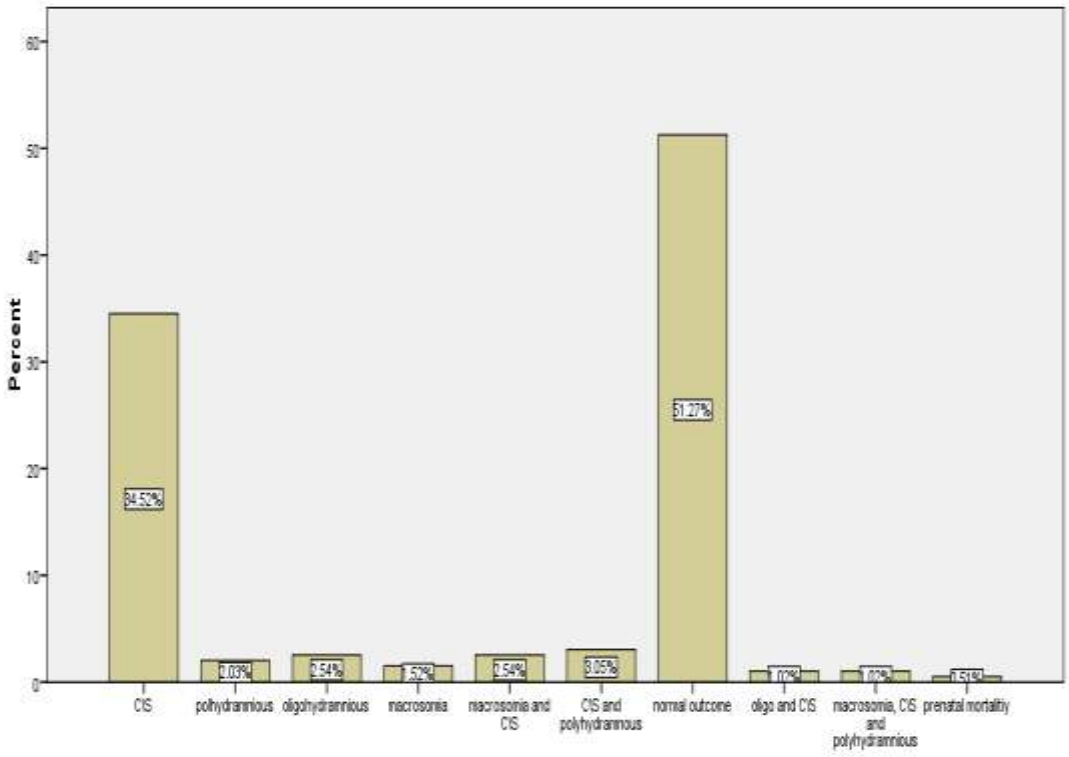


Figure (4.5) frequency distribution of outcome

Table (4.7) frequency distribution of range of GA by LMP

GA (range)	Frequency	Percent	Valid Percent	Cumulative Percent
33-38w6d	98	49.7	49.7	49.7
term (39-40 weeks)	35	17.8	17.8	67.5
post -date (more than 40 weeks)	64	32.5	32.5	100.0
Total	197	100.0	100.0	

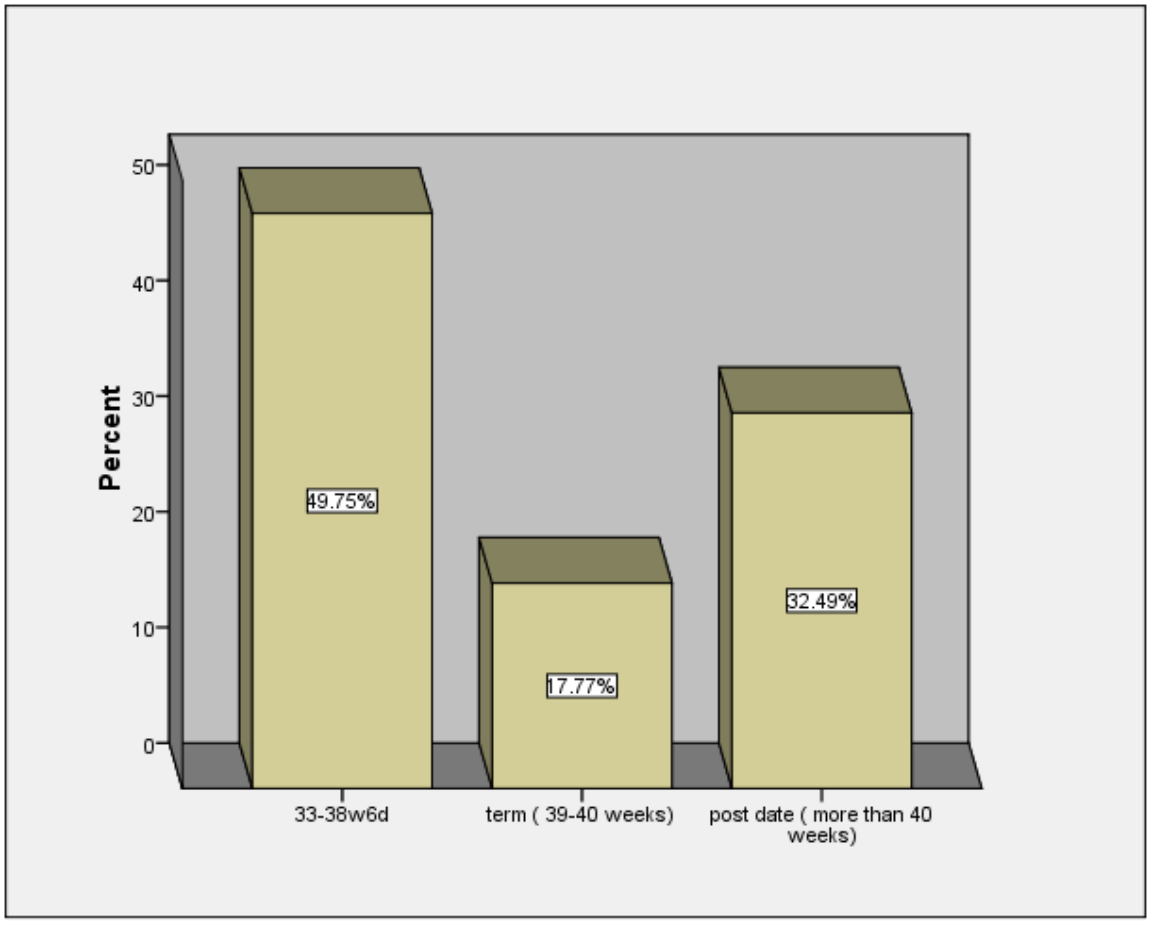


Figure (4.6) frequency distribution of range of GA by LMP

Table (4.8) compare mean Doppler indices of MCA in different range of GA by LMP

GA		PSV MCA	EDV MCA	S\D ratio	PI MCA	RI MCA
33-38w6d	Mean	20.6382	8.9044	2.4879	1.3805	.5752
	N	98	98	98	98	98
	Std. Deviation	7.43908	3.64200	.90775	.78850	.21113
Term (39-40 weeks)	Mean	21.8717	10.6634	2.2340	1.1197	.5340
	N	35	35	35	35	35
	Std. Deviation	8.61248	4.72437	.85429	.64702	.24230
post date (more than 40 weeks)	Mean	22.0906	10.1392	2.2775	1.1286	.5930
	N	64	64	64	64	64
	Std. Deviation	9.81716	4.90960	.67789	.67284	.43875
Total	Mean	21.3292	9.6181	2.3744	1.2523	.5737
	N	197	197	197	197	197
	Std. Deviation	8.46838	4.32694	.83375	.73633	.30745
P value		0.52	0.059	0.16	0.051	0.660

Table (4.9) compare mean Doppler indices of UA in different range of GA by LMP

GA		PSV UA	EDV UA	S\D UA	PI UA	RI UA
33-38w6d	Mean	29.9505	15.8920	1.9681	.8067	.4840
	N	98	98	98	98	98
	Std. Deviation	9.73928	6.01346	.53298	.32869	.19228
term (39-40 weeks)	Mean	29.0446	17.0506	1.7580	.9423	.4594
	N	35	35	35	35	35
	Std. Deviation	7.94979	5.00615	.31973	.84138	.25625
postdate (more than 40 weeks)	Mean	28.9027	16.1586	1.8277	.7095	.4269
	N	64	64	64	64	64
	Std. Deviation	8.02546	4.12757	.40849	.27904	.11837
Total	Mean	29.4491	16.1845	1.8851	.7992	.4611
	N	197	197	197	197	197
	Std. Deviation	8.88037	5.28175	.46835	.45564	.18665
P value		0.733	0.530	0.03	0.05	0.163

Table (4.10) a. compare mean Doppler indices in 33-40 weeks by LMP versus post date

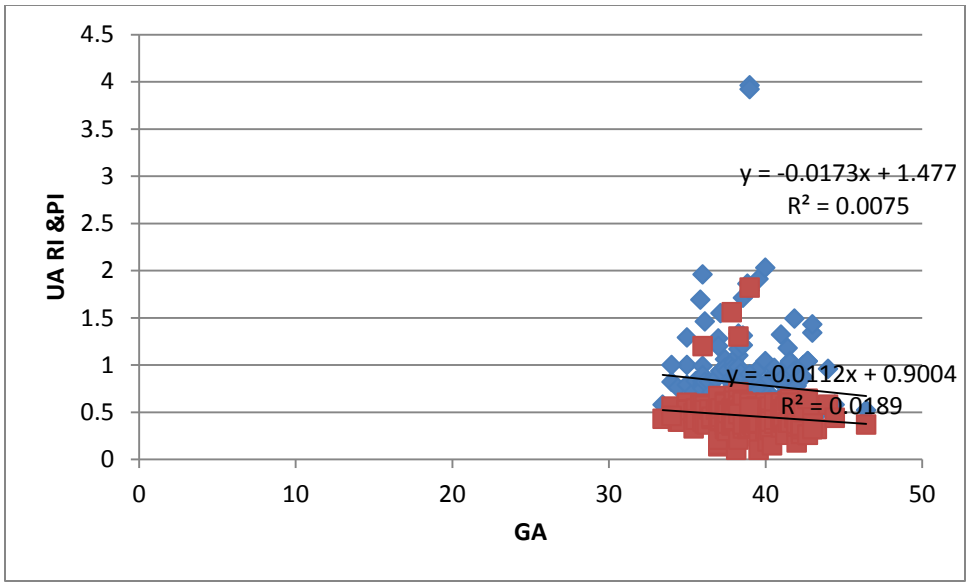
Group Statistics					
	GA	**N**	**Mean**	**Std. Deviation**	**Std. Error Mean**
PSV MCA	**33-40**	**133**	**20.9628**	**7.75044**	**.67205**
	post date	**64**	**22.0906**	**9.81716**	**1.22715**
EDV MCA	**33-40**	**133**	**9.3673**	**4.01257**	**.34793**
	post date	**64**	**10.1392**	**4.90960**	**.61370**
MCA S\D	**33-40**	**133**	**2.4211**	**.89783**	**.07785**
	post date	**64**	**2.2775**	**.67789**	**.08474**
MCA PI	**33-40**	**133**	**1.3119**	**.76026**	**.06592**
	post date	**64**	**1.1286**	**.67284**	**.08411**
MCA RI	**33-40**	**133**	**.5644**	**.21957**	**.01904**
	post date	**64**	**.5930**	**.43875**	**.05484**
UA PSV	**33-40**	**133**	**29.7121**	**9.28127**	**.80479**
	post date	**64**	**28.9027**	**8.02546**	**1.00318**
UA EDV	**33-40**	**133**	**16.1969**	**5.76982**	**.50031**
	post date	**64**	**16.1586**	**4.12757**	**.51595**
UA S\D	**33-40**	**133**	**1.9128**	**.49366**	**.04281**
	post date	**64**	**1.8277**	**.40849**	**.05106**
UA PI	**33-40**	**133**	**.8424**	**.51510**	**.04466**
	post date	**64**	**.7095**	**.27904**	**.03488**
UA RI	**33-40**	**133**	**.4775**	**.21024**	**.01823**
	post date	**64**	**.4269**	**.11837**	**.01480**
Ratio PI MCA\PI UA	**33-40**	**133**	**1.9013**	**1.36802**	**.11862**
	post date	**64**	**1.8226**	**1.20710**	**.15089**

Table (4.10) b. t. test for compare mean Doppler indices in 33-40 weeks by LMP versus post- date .

	t-test for Equality of Means						
	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
						Lower	Upper
PSV	-.875-	195	.383	-1.12784-	1.28907	-3.67016-	1.41447
MCA	-.806-	102.075	.422	-1.12784-	1.39912	-3.90296-	1.64728
EDV	-1.174-	195	.242	-.77193-	.65763	-2.06890-	.52505
MCA	-1.094-	104.839	.276	-.77193-	.70547	-2.17076-	.62691
MCA	1.133	195	.259	.14355	.12675	-.10642-	.39352
S\D	1.248	159.876	.214	.14355	.11507	-.08370-	.37081
MCA	1.643	195	.102	.18329	.11154	-.03669-	.40326
PI	1.715	139.127	.089	.18329	.10686	-.02800-	.39457
MCA	-.611-	195	.542	-.02861-	.04685	-.12100-	.06378
RI	-.493-	78.555	.624	-.02861-	.05805	-.14417-	.08696
UA	.598	195	.550	.80945	1.35320	-1.85933-	3.47823
PSV	.629	142.095	.530	.80945	1.28610	-1.73292-	3.35182
UA	.048	195	.962	.03832	.80557	-1.55042-	1.62707
EDV	.053	166.794	.958	.03832	.71869	-1.38057-	1.45722
UA	1.196	195	.233	.08513	.07117	-.05524-	.22549
S\D	1.278	147.821	.203	.08513	.06663	-.04654-	.21680
UA PI	1.930	195	.055	.13287	.06884	-.00289-	.26864
	2.345	192.268	.020	.13287	.05667	.02110	.24465
UA RI	1.794	195	.074	.05064	.02824	-.00504-	.10633
	2.157	190.227	.032	.05064	.02348	.00433	.09696
Ratio	.393	195	.695	.07877	.20054	-.31672-	.47427
PI MCA\ PI UA	.410	139.506	.682	.07877	.19193	-.30070-	.45825

Table (4.11) correlation Doppler indices with GA LMP,BPD, FL, AC

		GA LMP	GA FL	GA BPD	GA AC
MCA PSV	Pearson Correlation	.145*	.020	.026	-.109-
	Sig. (2-tailed)	.042	.781	.719	.128
	N	197	197	197	197
MCA EDV	Pearson Correlation	.190**	.034	.007	.011
	Sig. (2-tailed)	.007	.637	.920	.880
	N	197	197	197	197
S\D MCA	Pearson Correlation	-.123-	-.072-	-.041-	-.172*
	Sig. (2-tailed)	.084	.317	.564	.015
	N	197	197	197	197
PI MCA	Pearson Correlation	-.187**	-.119-	-.077-	-.175*
	Sig. (2-tailed)	.009	.096	.284	.014
	N	197	197	197	197
RI MCA	Pearson Correlation	.001	.037	.012	-.055-
	Sig. (2-tailed)	.989	.610	.867	.443
	N	197	197	197	197
UA PSV	Pearson Correlation	-.048-	-.054-	-.084-	-.025-
	Sig. (2-tailed)	.503	.449	.238	.728
	N	197	197	197	197
UA EDV	Pearson Correlation	.046	-.009-	-.030-	.015
	Sig. (2-tailed)	.524	.896	.672	.838
	N	197	197	197	197
S\D UA	Pearson Correlation	-.151*	-.069-	-.103-	-.065-
	Sig. (2-tailed)	.034	.334	.152	.366
	N	197	197	197	197
RI UA	Pearson Correlation	-.137-	-.138-	-.141*	-.122-
	Sig. (2-tailed)	.054	.053	.048	.088
	N	197	197	197	197
PI UA	Pearson Correlation	-.087-	-.127-	-.159*	-.142*
	Sig. (2-tailed)	.225	.076	.025	.046
	N	197	197	197	197
**. Correlation is significant at the 0.01 level (2-tailed).					
*. Correlation is significant at the 0.05 level (2-tailed).					



Figure(4.7) scatterplot shows inverse linear relation between GA LMP and RI,PI UA

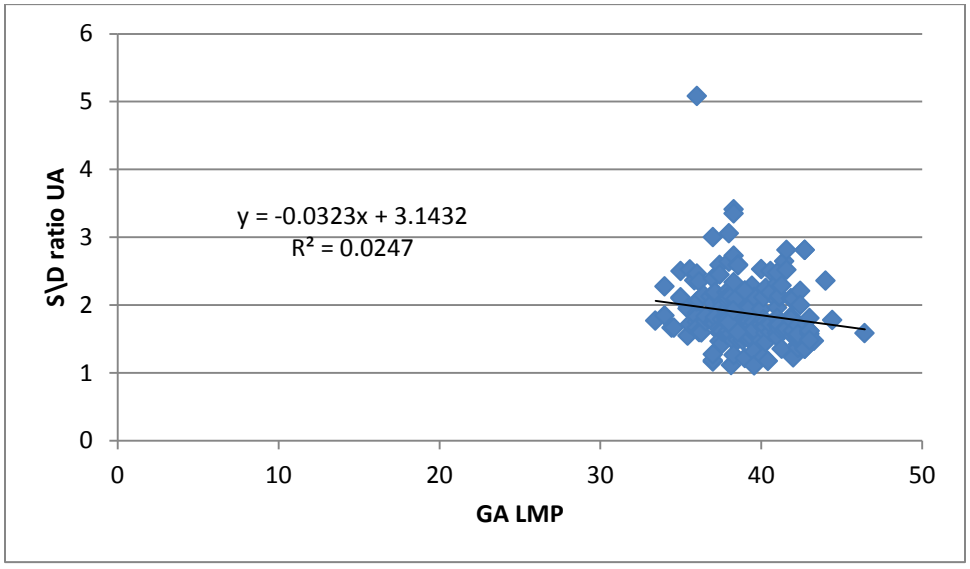


Figure (4.8) scatterplot shows inverse linear relation between GA LMP and S\D ratio of UA

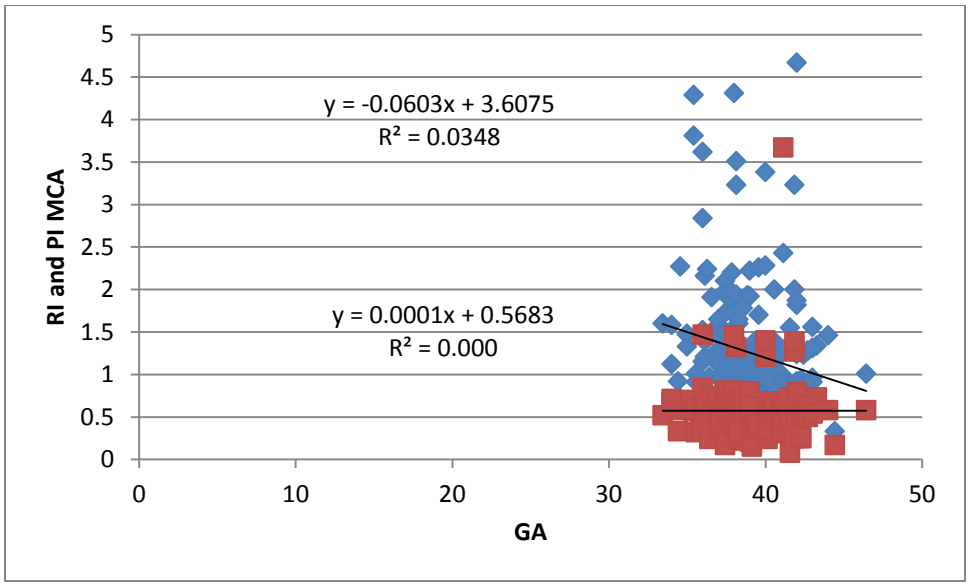


Figure (4.9) scatterplot shows inverse linear relation between GA LMP and RI, PI MCA

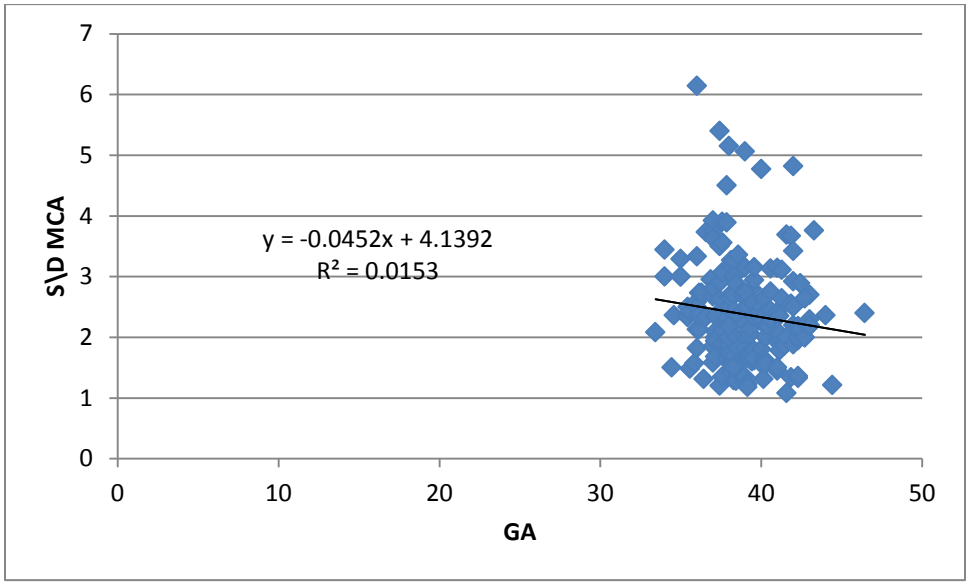


Figure (4.10) scatterplot shows inverse linear relation between GA LMP and S\D ratio of MCA

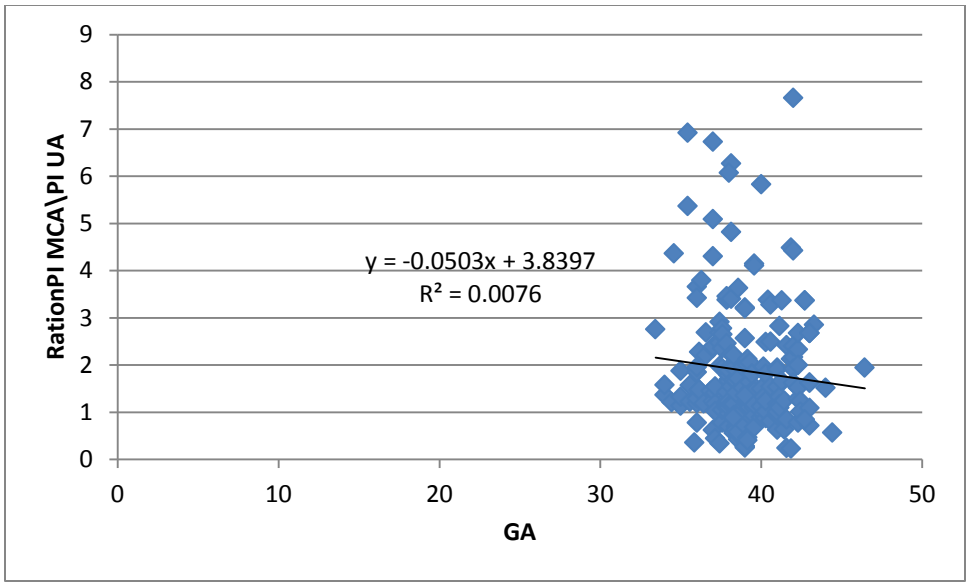


Figure (4.11) scatterplot shows no relation between GA LMP and ratio of MCA\UA

Post term

Table (4.12) descriptive statistic for age and measurements of GA, Doppler indices of MCA and UA (minimum ,maximum ,mean ± Std. Deviation) in post term

	N	Minimum	Maximum	Mean	Std. Deviation
Age	64	17	45	27.17	5.819
Parity	64	1	2	1.27	.445
FL cm	64	7.01	8.16	7.7383	.30052
BPD cm	64	8.68	92.70	10.9081	10.39327
AFI	64	.8	12.0	5.218	2.5423
Ac cm	64	32.07	37.79	35.6581	1.61041
EFW	64	2.08	4.44	3.7494	.48608
GA LMP	64	40.14	46.43	41.7272	1.17929
GA FL	64	36.00	41.71	39.5671	1.46340
GA BPD	64	33.43	41.71	39.1630	1.96165
GA AC	64	36.00	41.71	39.6012	1.63697
MCA PSV	64	9.54	57.70	22.0906	9.81716
MCA ED	64	3.18	34.44	10.1392	4.90960
S\D MCA	64	1.08	4.82	2.2775	.67789
PI MCA	64	.25	4.67	1.1286	.67284
RI MCA	64	.08	3.67	.5930	.43875
UA PSV	64	16.43	56.16	28.9027	8.02546
UA EDV	64	7.95	27.02	16.1586	4.12757
S\D UA	64	1.17	2.81	1.8277	.40849
PI UA	64	.26	1.49	.7095	.27904
RI UA	64	.15	.64	.4269	.11837
Ratio PI MCA\PI UA	64	.23	7.66	1.8226	1.20710
Valid N (listwise)	64				

Table (4.13) frequency distribution of presence of adverse outcome in post term

Adverse	Frequency	Percent	Valid Percent	Cumulative Percent
yes	43	67.2	67.2	67.2
no	21	32.8	32.8	100.0
Total	64	100.0	100.0	

Table (4.14) adverse outcome in post term

Adverse	Frequency	Percent	Valid Percent	Cumulative Percent
C\S	26	40.6	40.6	40.6
oligohydramnios	2	3.1	3.1	43.8
macrosomia	3	4.7	4.7	48.4
macrosomia and C\S	4	6.2	6.2	54.7
C\S and polyhydramnios	3	4.7	4.7	59.4
normal outcome	21	32.8	32.8	92.2
Oligohydramnios and C\S	2	3.1	3.1	95.3
macrosomia, C\S and polyhydramnios	2	3.1	3.1	98.4
prenatal mortality	1	1.6	1.6	100.0
Total	64	100.0	100.0	

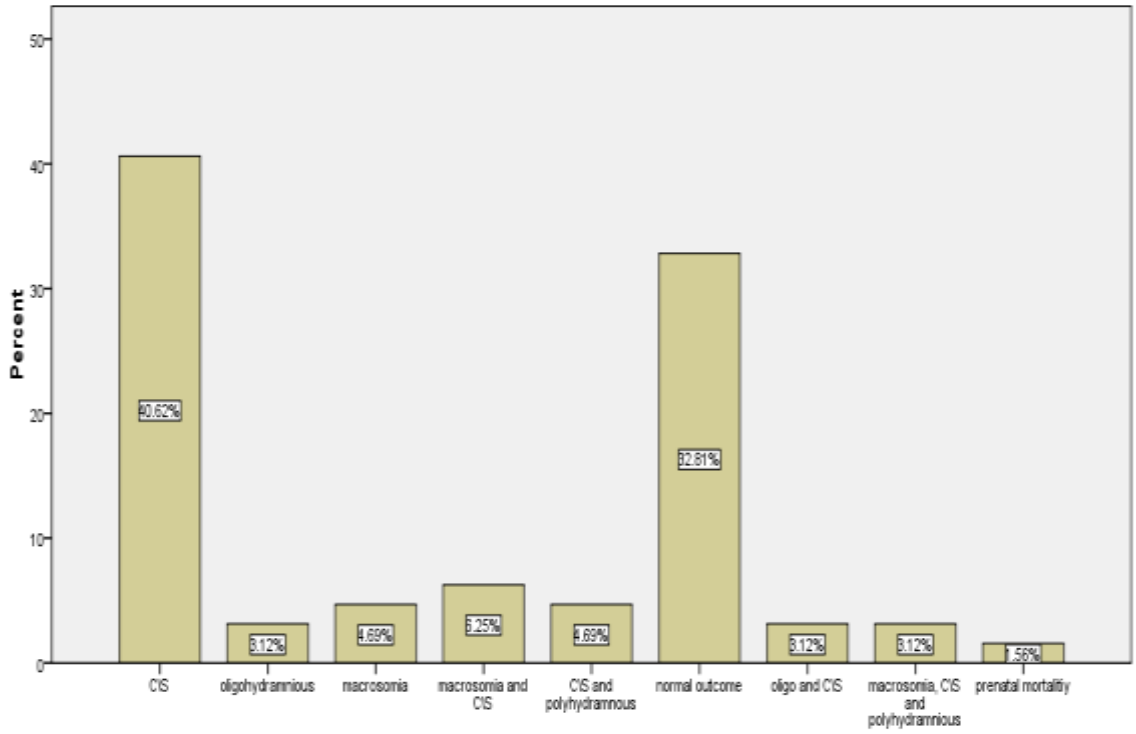


Figure (4.12) adverse outcome in post term

Table (4.15) a. Compare means Doppler indices and outcome in post term

	Adverse	N	Mean	Std. Deviation	Std. Error Mean
MCA PSV	no	21	20.2952	6.16929	1.34625
	yes	43	22.9674	11.13743	1.69844
MCA EDV	no	21	9.4524	3.74726	.81772
	yes	43	10.4747	5.39616	.82291
S\D MCA	no	21	2.2376	.53043	.11575
	yes	43	2.2970	.74441	.11352
PI MCA	no	21	1.0624	.41588	.09075
	yes	43	1.1609	.77035	.11748
RI MCA	no	21	.5276	.11687	.02550
	yes	43	.6249	.52827	.08056
UA PSV	no	21	26.5205	5.82299	1.27068
	yes	43	30.0660	8.73171	1.33157
UA EDV	no	21	15.0229	3.54359	.77328
	yes	43	16.7133	4.31461	.65797
S\D UA	no	21	1.8110	.38749	.08456
	yes	43	1.8358	.42260	.06445
PI UA	no	21	.7457	.29723	.06486
	yes	43	.6919	.27157	.04141
RI UA	no	21	.4248	.11474	.02504
	yes	43	.4279	.12143	.01852
Ratio PI MCA\PI UA	no	21	1.6711	.95434	.20825
	yes	43	1.8965	1.31714	.20086

Table (4.15) b. student t-test to compare Doppler indices and outcome in post term

	t-test for Equality of Means						
	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
						Lower	Upper
MCA PSV	-1.023-	62	.310	-2.67220-	2.61260	-7.89472-	2.55031
	-1.233-	60.885	.222	-2.67220-	2.16728	-7.00611-	1.66170
MCA EDV	-.780-	62	.439	-1.02227-	1.31114	-3.64319-	1.59865
	-.881-	54.436	.382	-1.02227-	1.16010	-3.34771-	1.30317
S/D MCA	-.327-	62	.745	-.05936-	.18176	-.42270-	.30398
	-.366-	53.436	.716	-.05936-	.16213	-.38448-	.26577
PI MCA	-.547-	62	.586	-.09855-	.18013	-.45862-	.26153
	-.664-	61.266	.509	-.09855-	.14845	-.39537-	.19827
RI MCA	-.831-	62	.409	-.09726-	.11709	-.33133-	.13680
	-1.151-	49.790	.255	-.09726-	.08450	-.26701-	.07248
UA PSV	-1.683-	62	.097	-3.54557-	2.10613	-7.75566-	.66452
	-1.926-	55.928	.059	-3.54557-	1.84058	-7.23279-	.14165
UA EDV	-1.556-	62	.125	-1.69040-	1.08668	-3.86264-	.48184
	-1.665-	47.570	.103	-1.69040-	1.01532	-3.73232-	.35152
S/D UA	-.227-	62	.821	-.02486-	.10958	-.24390-	.19418
	-.234-	43.064	.816	-.02486-	.10632	-.23926-	.18954
PI UA	.722	62	.473	.05385	.07457	-.09521-	.20292
	.700	36.725	.488	.05385	.07695	-.10211-	.20982
RI UA	-.099-	62	.921	-.00315-	.03176	-.06664-	.06035
	-.101-	41.893	.920	-.00315-	.03114	-.06600-	.05971
Ratio PI MCA\PI UA	-.699-	62	.487	-.22542-	.32267	-.87043-	.41960
	-.779-	52.772	.439	-.22542-	.28934	-.80581-	.35498

Table (4.16) cross tabulation adverse outcome and GA

Outcome	GA			Total
	33-38w6d	term (39-40 weeks)	postdate (more than 40 weeks)	
C\S	31	11	26	68
polyhydramnios	4	0	0	4
oligohydramnios	2	1	2	5
macrosomia	0	0	3	3
macrosomia and C\S	0	1	4	5
C\S and polyhydramnios	3	0	3	6
normal outcome	58	22	21	101
oligo and C\S	0	0	2	2
macrosomia, C\S and polyhydramnios	0	0	2	2
prenatal mortality	0	0	1	1
	98	5	64	197
P =0.007				

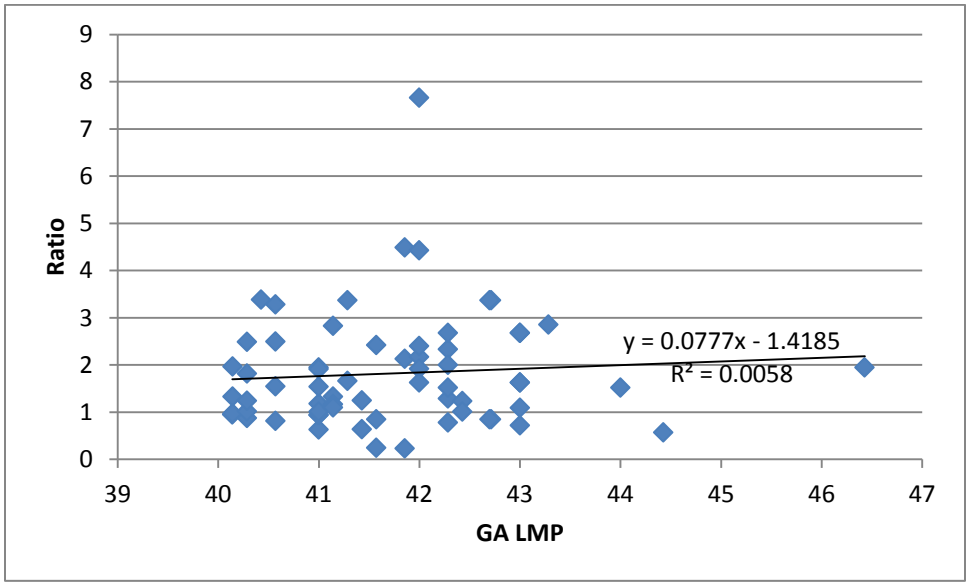


Figure (4.13) scatterplot shows linear relation between GA LMP and ratio of MCA\UA in post term

Table (4.17) correlation Doppler indices and GA LMP in post term

		GA LMP
MCA PSV	**Pearson Correlation**	**.270***
	Sig. (2-tailed)	**.031**
	N	**64**
MCA EDV	**Pearson Correlation**	**.175**
	Sig. (2-tailed)	**.168**
	N	**64**
S/D MCA	**Pearson Correlation**	**.101**
	Sig. (2-tailed)	**.425**
	N	**64**
PI MCA	**Pearson Correlation**	**.080**
	Sig. (2-tailed)	**.527**
	N	**64**
RI MCA	**Pearson Correlation**	**-.024-**
	Sig. (2-tailed)	**.851**
	N	**64**
UA PSV	**Pearson Correlation**	**.072**
	Sig. (2-tailed)	**.570**
	N	**64**
UA EDV	**Pearson Correlation**	**.134**
	Sig. (2-tailed)	**.291**
	N	**64**
S/D UA	**Pearson Correlation**	**-.065-**
	Sig. (2-tailed)	**.610**
	N	**64**
PI UA	**Pearson Correlation**	**.028**
	Sig. (2-tailed)	**.824**
	N	**64**
RI UA	**Pearson Correlation**	**-.071-**
	Sig. (2-tailed)	**.578**
	N	**64**
Ratio PI MCA\PI UA	**Pearson Correlation**	**.076**
	Sig. (2-tailed)	**.551**
	N	**64**
*. Correlation is significant at the 0.05 level (2-tailed).		

Chapter five

Discussion, Conclusion and Recommendations

5.1 Discussion:

5.1.1 Whole sample

This was prospective study done in 197 pregnant women in late third trimester and post term pregnancy with age ranged 17-45 years, 46.2% of them in age ranged 24-30 years and 23.9% of them in age group 17-23 years as shown in table and figure 4.1, mean age 28.07±6.07 years. Most of them were nulliparous 87.7%, most of them were non-employee 77.2%.(table and figure 4.2,4.3)

Concerning fetal presentation most of them were cephalic presentation 93.4% , table 4.4,figure 4.4.

Concerning fetal presentation most of them had cephalic presentation 93.4% as shown in table and figure 4.4. The mean FL , BPD ,AC ,GA LMP,GA AC ,GA FL,GABPDwere7.35±.45,9.60±5.97,33.58±2.56,39.07±2.27,37.51±2.52,37.62±2.09,37.29±2.15 respectively. Table (4.5)

The study clarified that the mean Doppler indices for UA were 0.46±.18,0.80±0.46,29.44±8.88,16.18±5.28,1.88±0.46 for RI,PI ,PSV,EDV,S\D ratio respectively. Table (4.5)

The study clarified that the mean Doppler indices for MCA were0.57±0.30, 1.25±0.73, 21.3±8.46,9.62±4.32,2.39±0.84 for RI, PI ,PSV, EDV, S\D ratio respectively. Table (4.5)

The study found that the mean MCA \ UA ratio was 1.87±1.31, table (4.5)

The study found that 51.3 % of these cases had normal outcome , while 48.7% had adverse outcome . 34.5% delivered by CS, 2.5% had oligohydramnios , 6% had macrosomia , 0.5 % prenatal mortality . Table (4.6)

The study classified cases according to their gestational age GA in to three group .the first group consisted of (98 pregnant women , 49.7%) with GA of (33-38w6d) , the second group consist of (35 pregnant women , 17.8%) consider as term with GA of (39-40 weeks) and the third group consisted of 64 pregnant women , 32.5% with GA of (more than 40 weeks) which consider as post –date or prolong pregnancy(table 4.7) and (figure 4.6).

The study found that no significant difference in Doppler indices of MCA in these three group of gestational age , the mean Doppler indices for each were20.6±7.4, 8.90±3.64, 0.57±0.21,1.38±0.78, 2.48±0.90, PSV ,EDV , RI, PI , S\D ratio respectively for 34-38 w 6d , 21.8±8.61, 10.6±4.72, 0.53±0.24,1.11±0.64, 2.23 ± 0.85, PSV ,EDV , RI, PI , S\D ratio respectively for 39-40 weeks and 22.0±9.81, 10.1±4.90, 0.59±0.43, 1.12±0.67, 2.27±0.67 PSV ,EDV , RI, PI , S\D ratio for postdate (more than 40 weeks) respectively. Table (4.8)

The study found that there was no significant difference in Doppler indices of UA in these three group of gestational age ($p > 0.05$), except for S\d ratio and PI which shows significant difference in different age group ($p< 0.05$) ,the mean Doppler indices for each were 29.95±9.73,15.8±6.01,0.48±0.19 , 0.81±0.32, 1.97±.53 for PSV ,EDV , RI, PI , S\D ratio respectively for 34-38 w6d , 29.04±7.94, 17.05±5.0, 0.45±0.25, 0.94±0.84, 1.75±0.31 PSV ,EDV , RI, PI , S\D ratio respectively for 39-40 weeks and 28.9±8.02, 16.15±4.12, 0.42±0.11, 0.71±0.27,

1.82±0.40 PSV ,EDV , RI, PI , S\D ratio respectively for postdate respectively. Table (4.9)

The study found that MCA PSV and EDV shows positive significant weak correlation with GA LMP ($p< 0.01$) , while PI shows negative weak correlation with GA , RI and S\D ratio had no significant correlation with GA , UA S\D ratio had significant weak negative correlation with GA ($p <0.05$) UA PI,RI , PSV , EDV had no significant correlation with GA LMP ($p>0.05$) but in generally decreased by GA , except UA EDV increased slightely. Table (4.10)

Concerning linearity in relationship between Doppler indices of UA with GA the study clarified that as GA increased Doppler indices of UA decreased slightly RI, PI, S\D ratio figure (4.7,4.8). The study also clarified that as GA of MCA increased PI and S\D ratio decreased in generally but RI remain constant and had no any relationship with GA, figure(4.9,4.10)

In three group the study found that the incidence of post term increased in nulliparous than multiparous women 23.8% versus 8.6% , this results go online with literature as stated that post term increased in no parity women. Table (4.11)

5.1.2 Post term pregnancy

The study found that the mean measurements of GA LMP and Doppler indices of post term were 41weeks 5days GA LMP , for MCA 22.9,10.13,2.27,1.12 and0.59 for PSA,EDV,S\D ratio ,PI and RI respectively and for UA 28.09,16.15,1.82,0.71,0.42 for PSA,EDV,S\D ratio ,PI and RI respectively , the mean ratio of CU was 1.82±1.2. table (4.12)

The study shows that in post term there was 67.2% of cases with abnormal outcome (40.6% delivered by C\S, 4.7 % macrosomia, 3.1

oligohydramnios only. 3.1 oligohydramnios and C\S, 3.1 % macrosomia, polyhydramnious and C\S, prenatal mortality was 0.5%, this results go online with all previous studies whom found that C\S and oligohydramnios increased in post term pregnancy. Table (4.13, 4.14)

The study found that the MCA PSV , EDV , S\D ratio RI and PI increased in post term patients with adverse outcome slightly than with normal outcome 22.97± 11.13 versus 20.30± 6.16, 10.47 ±5.40, 9.45± 3.74 , 2.29 ±0.74 , 2.23± 0.53 , 0.62±0.52, 0.52±0.11 and 1.16±0.77, 1.06+0.41 also the UA Doppler indices were increased in patients with adverse outcome than in normal (PSV ,EDV, ,S\D ratio) 30.07±8.73versus 26.52±5.82 and 16.52±4.31versus 15.02± 3.54 and 1.83±0.42 versus 1.81±0.38 respectively. These results disagree with Anteby et al (1994) who state that impedance decreased. Gupta Usha et al (2006) stated the mean values of UA S/D ratio, MCA PI and CU ratio were 2.46, 1.31 and 1.36 respectively. The ratio in this study higher than in study of Placio et al observed mean CU ratio to be 1.36 at 41 weeks and 1.27 at 42 weeks , a cutoff value of CU ratio of 1.3 was obtained and used for correlating perinatal outcome in these postdate pregnancies. The CU ratio of 1.3 for predicting adverse perinatal outcome in the present also considerably higher than the value established by Devine et al **.**

The PI of UA decreased in post term with adverse than in normal outcome 0.69 versus 0.74 respectively while RI similar in both 0.42, the CU ratio increased in adverse than in normal outcome 1.89 versus 1.67 respectively. Table (4.15. a) this ratio is more than that mention in previous studies such as Devine et al (1994) whom found the ratio less than 1.01 associated with adverse outcome, also disagree with Battaglia et al (1995)

whom state that the ratio of the impedance in the middle cerebral artery to that in the umbilical artery were decreased .

Independent sample t - test confirmed that no significant difference in all these Doppler indices in patients post term with adverse versus post term with normal out come with 95% confidence interval p value >0.05 respectively, this results agree with Zimmermann et al (1995) , Gupta Usha et al (2006) and Subhra Ghosh et al (2016) which they reported that impedance to flow in the fetal middle cerebral artery did not change significantly within this gestational range. Also agree with Stokes et al (1991) whom reported that impedance to flow in the umbilical and uteroplacental arteries was not significantly different in pregnancies associated with fatal compromise and abnormal neonatal outcome from those with normal outcome. Table (4.15 b)

The study found that there was significant difference in complication of pregnancy in post and preterm as macrosomia, oligohydramnios, C\S increased in postdate than in term and preterm ($p<0.01$) , table (4.15)

Concerning linearity in relationship between GA and CU ratio in post term, the study found that CU ratio slightly increased with GA in post term but no linearity (R^2 0.005) , figure (4.11)

Table (5.1) shows comparison of previous studies with present studies results in Doppler indices of post term with abnormal outcome related to normal outcome.

Author	N	Blood vessel examined	Impedance to flow in Adverse outcome
Rightmire & Campbell, 1987	**35**	**umbilical artery**	**increased**
Brar et al., 1989	**45**	**internal cerebral artery**	**decreased**
Fischer et al., 1991	**75**	**umbilical artery increased**	**increased**
Anteby et al., 1994	**78**	**umbilical artery**	**increased**
Olofsson et al., 1997	**44**	**umbilical artery**	**decreased**
Anteby et al., 1994	**78**	**middle cerebral artery**	**decreased**
Devine et al., 1994	**49**	**middle cerebral : umbilical artery**	**decreased**
Rightmire & Campbell, 1987	**35**	**uterine artery**	**normal**
Farmakides et al., 1988	**149**	**umbilical and uterine arteries**	**normal**
Brar et al., 1989	**45**	**umbilical and uterine arteries**	**normal**
Stokes et al., 1991	**70**	**umbilical and uterine arteries**	**normal**
Bar-Hava et al., 1995	**57**	**umbilical and middle cerebral arteries**	**normal**
Zimmermann et al., 1995	**153**	**umbilical, uterine & middle cerebral arteries**	**normal**
Present study (Eman Hassan 2019)	**64**	**middle cerebral artery**	**Increased**
	64	**umbilical artery**	**PSV,EDV,S\D ratio increased**
			RI remain same
			PI decreased

The study found that no significant difference in Doppler indices in MCA and UA in post term with GA, $p > 0.05$ except for MCA PSV which increased as GA increased (p value =0.03, r=0.270), this result similar to Subhra Ghosh et al (2016) whom stated no significant difference but disagree with them in PSV only, also disagree with them in RI UA whom stated significant different was found, also the present study stated increased RI of UA but not significant with GA in post term. Table (4.16)

5.2 Conclusion:

The study concluded that the most common fetal presentation is cephalic presentation.

Most of post term is nulliparous; the mean age of post term is 27 years. The mean GA is 41 weeks 5 days; the range is 40weeks 1day to 46 weeks by LMP.

In general the study found that there was inverse linear relationship between Doppler indices and GA.

The study found that there was significant difference in adverse outcome with GA as caesarian section, oligohydramnios, prenatal mortality, macrosomia all of them was more in post term than in preterm and term pregnancy.

The study found that the MCA PSV , EDV , S\D ratio RI and PI increased in post term patients with adverse outcome slightly than with normal outcome , the UA Doppler indices were increased in patients with adverse outcome than in normal (PSV ,EDV, ,S\D ratio) , The PI of UA decreased in post term with adverse than in normal outcome versus respectively while RI similar in both entities. The study found that the ratio of MCA PI \UA PI was slightly increased in patients with adverse than in normal outcome this ratio is more than that mention in previous studies.

The study concluded that no significant difference in all these Doppler indices in patients post term with adverse versus post term with normal out come with 95% confidence interval p value >0.05 respectively

5.3 Recommendations

1. As there is not agreement by different studies done in what happen to

Doppler indices in post term pregnancies so further studies should be done

with larger sampling is necessary.

2. Further studies should be done taking biophysical profile should be done

3. Further studies to confirm the cut of ratio in Sudanese should be done.

References

Ahanya, S.N. Lakshmanan, J. Morgan, B.L. Ross, M.G (2005) Meconium passage in utero: mechanisms, consequences, and management. Obstet Gynecol Surv, 60(1), pp.45-56.

Alexander, J.M. Mcintire, d.d. Leveno, K.J (2000) Forty weeks and beyond: pregnancy outcomes by week of gestation. Obstet Gynecol, 96(2), pp.291-4.

Alexander, J.M. Mcintire, D.D.and Leveno, K.J (2001) Prolonged pregnancy: induction of labour and caesarean births. Obstet Gynecol, 97(6), pp.911.

Anteby, E.Y. Tadmor, O. Revel, A. Yagel, S (1994) Post-term pregnancies with normal cardiotocographs and amniotic fluid columns: the role of Doppler evaluation in predicting perinatal outcome. Eur J Obstet Gynecol Reprod Biol, 54(2), pp.93–8.

Bar-Hava, I. Divon, M.Y. Sardo, M. Barnhard, Y (1995) Is oligohydramnios in post term pregnancy associated with redistribution of fetal blood flow? Am J Obstet Gynecol, 173(2), pp519–22

Battaglia, C. Artini, P.G. Ballestri M, Bonucchi, D. Galli, P.A. Bencini, S. Genazzani, A.P(1995) Hemodynamic, ematological and hemorrheological evaluation of post-term pregnancy. Acta Obstet Gynecol Scand, 74(5), pp 336–40.

Battaglia, C. Larocca, E. Lanzani, A. Coukos, G. Genazzani, A. R (1991) Doppler velocimetry in prolonged pregnancy. Obstet Gynecol, 77(2), pp 213-6.

Bergholt, T. Stenderup, J. K. Vedsted-Jakobsen, A. Helm, P. Lenstrup, C (2003). Intraoperative surgical complication during caesarean section: an observational study of the incidence and risk factors. Acta Obstet Gynecol Scand, 82(3), pp251-6.

Brar, H.S. Horenstein, J. Medearis, A.L. Platt, L.D. Phelan, J.P. Paul, R.H (1989) Cerebral, umbilical, and uterine resistance using Doppler velocimetry in post term pregnancy, J Ultrasound Med, 8, pp.187–91.

Campbell, M.K. Ostbye, T. Irgens, L.M (1997) Post-term birth: risk factors and outcomes in a 10-year cohort of norwegian births. Obstet Gynecol, 89(4), pp. 543-8.

Campbell, S. Warsof, S.L. Little, D. Cooper, D.J (1985) Routine ultrasound screening for the prediction of gestational age. Obstet Gynecol , 65(5), pp. 613-20.

Chamberlain, P.F. Manning, F.A. Morrison, I. Harman, C.R. Lange, I.R(1984) Ultrasound evaluation of amniotic fluid volume. I. The relationship of marginal and decreased amniotic fluid volumes to perinatal outcome. Am J Obstet Gynecol, 150(3), pp. 245-9.

Chervenak, J.L. Divon, M.Y. Hirsch, J. Girz, B.A. Langer, O (1989). Macrosomia in the postdate pregnancy: is routine ultrasonographic screening indicated, Am J Obstet Gynecol, 161(3), pp. 753-6.

Clement, D. Schifrin, B.S. Kates, R.B (1987). Acute oligohydramnios in postdate pregnancy. Am J Obstet Gynecol, 157(4), pp. 884-6.

Collins, J.W. Schulte, N. F. George, L. Drolet, A (2001) Postterm delivery among African Americans, Mexican Americans and Whites in Chicago. Ethn Dis, 11(2), pp. 181-7.

Crowley, P. O'Herlihy, C. Boylan, P (1984). The value of ultrasound measurement of amniotic fluid volume in the management of prolonged pregnancies. Br J Obstet Gynaecol, 91(5), pp. 444-8.

Daya, S.(1991) Evaluation of normal gestational sac growth: appearance of embryonic heartbeat and embryo body movements using the transvaginal technique. Obstet Gynecol, 78(4), pp. 729-30.

Devine, P.A. Bracero, L.A. Lysikiewicz, A. Evans, R. Womack, S. Byrne, D.W (1994) Middle cerebral to umbilical artery Doppler ratio in post-date pregnancies. Obstet Gynecol, 84(5), pp. 856–60.

Divon, M.Y, Haglund, B. Nisell, H. Otterblad, P.O. Westgren, M. (1998) Fetal and neonatal mortality in the postterm pregnancy: the impact of gestational age and fetal growth restriction. Am J Obstet Gynecol, 178(4), pp. 726-31.

Divon, M.Y. Ferber, A. Nisell, H. Westgren, M. (2002) Male gender predisposes to prolongation of pregnancy. Am J Obstet Gynecol, 187(4), pp. 1081–83.

Dobbing, J. Sands, J. (1970) Growth and development of the brain and spinal cord of the guinea pig. Brain Res, 17(1), pp. 115-23.

Doherty, L. and Norwitz, E. (2008) Prolonged pregnancy: when should we intervene, Current Opinion in Obstetrics and Gynecology, 20(6), pp. 519–27

Dyson, D.C. (1988) Fetal surveillance vs. labor induction at 42 weeks in postterm gestation. J Reprod Med, 33, pp. 262-70.

Eden, R.d. Seifert, l.S. Winegar, A. (1987a). Perinatal characteristics of uncomplicated postdate pregnancies. Obstet Gynecol, 69(3), pp. 296-9.

Eden, R.D. Seifert, L.S. Winegar, A. Spellacy, W.N. (1987b). Postdate pregnancies: a review of 46 perinatal deaths. Am J Perinatal, 4(4), pp. 284-7.

Eik-Nes, S.H. Marsal, K. Kristoffersen, K. (1984) Methodology and basic problems related to blood flow studies in the human fetus. Ultrasound Med Biol, 10(10), pp. 329-37.

Ellis, M.J. Livesey, J.H. Inder, W.J. (2002) Plasma corticotrophin-releasing hormone and unconjugated estriol in human pregnancy: gestational patterns and ability to predict preterm delivery. Am J Obstet Gynecol, 186(1), pp. 94–99.

Evans,T. Koeff, S. Morely, G.(1963) Fetal effects of prolonged pregnancy. Am J Obstet Gynecol, 85, pp. 701-02.

Farmakides, G. Schulman, H. Ducey, J. Guzman, E. Saladana, L. Penny, B. Winter,D. (1988) Uterine and umbilical artery Doppler velocimetry in postterm pregnancy. J Reprod Med, 33(3), pp. 259–61.

Figueras, F. Lanna, M. Palacio, M. Zamora, L. Puerta, B. Coll, O. Cararach, V. Vanrell, J.A. (2004) Middle cerebral artery Doppler indices at different sites: prediction of umbilical cord gases in prolonged pregnancies. Ultrasound Obstet Gynecol, 24(5), pp. 529-33.

Fischer, R.L. Kuhlman, K.A. Depp, R. Wapner, R.J. (1991) Doppler evaluation of umbilical and uterine arcuate arteries in the postdates pregnancy. Obstet Gynecol, 78(3Pt1), pp. 363–8.

Fitz Gerald DE, Drumm JE. (1977) Non-invasive measurement of human fetal circulation using ultrasound: a new method. Br Med J; 2:1450-1.

Gardosi, J. (1998) the application of individualised fetal growth curves. J Perinat Med, 26(4), pp. 137-42.

Geirsson, R.T. Have, G.(1993) Comparison of actual and ultrasound estimated second trimester gestational length in in-vitro fertilized pregnancies. Acta Obstet Gynecol Scand, 72(5), pp. 344-6.

Gill, R.W. Kossoff, G. (1979) Pulsed doppler combined with B-mode imaging for blood flow measurement. Contrib Gynecol Obstet, 6, pp. 139-41.

Gonen, O. Rosen, D.J. Dolfin, Z. Tepperm, R. Markov, S. Fejgin, M.D. (1997) Induction of labour versus expectant management in macrosomia: a randomized study. Obstet Gynecol, 89(6), pp. 913-7.

Gosling, R.G. Dunbar, G. King, D.H. Newman, D.L. Side, C.D. Woodcock, J.P. Fitzgerald, D.E. Keates, J.S. MacMillian, D. (1971)The

quantitative analysis of occlusive peripheral arterial disease by a non-intrusive ultrasonic technique. Angiology, 22(1), pp. 52-5.

Guideline, N.I.C.E., 2008. Induction of Labour. National Collabourating Centre for Women's and Children's Health. National Institute for Health and Clinical Excellence.

Gupta Usha. Chandra Suhasini. Narula, M.K. (2006) Value of middle cerebral artery to umbilical artery ratio by doppler velocimetry in pregnancies beyond term, j Obstet Gynecol India ,56(1), pp. 37-4.

Hannah, M.E. Hannah, W.J. Hellmann, J. et al. (1992). Induction of labour as compared with serial antenatal monitoring in post term pregnancy. A randomized controlled trial. The Canadian Multicenter Postterm Pregnancy Trial Group. N Engl J Med, 326, pp. 1587–92.

Harald, T. Lutz Klinikum Bayreuth. Hassen, A. Gharbi. (2006) Manual of Diagnostic Ultrasound in Infectious Tropical Diseases.1st ed, New York: Springer.

Harlow, S.D. Ephross, S.A.(1995) Epidemiology of menstruation and its relevance to women's health, Epidemiol Rev, 17(2), pp. 265-86.

Henderson, M. Kay, J. (1967). Differences in duration of pregnancy. Negro and white women of low socioeconomic class. Arch Environ Health, 14(6), pp. 904-11.

Hon, E.H. (1963) Fetal heart rate monitoring. Biomed Sci Instrum, 1, pp. 287-91.

Hovi, M. Raatikainen, K. Heiskanen, N. Heinonen, S. (2006). Obstetric outcome in post-term pregnancies: time for reappraisal in clinical management. Acta Obstet Gynecol Scand, 85(7), pp. 805-9.

Hsieh, Y.Y. Chang, C.C. Tsai, H.D. Tsai, C.H. (2001) Longitudinal survey of blood flow at three different locations in the middle cerebral artery in normal fetuses. Ultrasound Obstet Gynecol, 17(2), pp. 125-8.

Hustin J. (1995) Vascular physiology and pathophysiology of early pregnancy. In: Bourne T, Jauniaux E, Jurkovic D, editors.Transvaginal color Doppler. Heidelberg: Springer-Verlag, pp. 47-56.

Jauniaux, E.(1996) Intervillous circulation in the first trimester: the phantom of the color Doppler obstetric opera. Ultrasound Obstet Gynecol, 8(2), pp. 73-76.

Jones GS, Jones HW. (1982) Cyclical cytology and histology. In Jones GS, Jones HW, Gynecology. 3rd ed. Baltimore: Williams & Wilkins; p. 46-68.

Kassanos, D. Siristatidis, C. Vitoratos, N. Salamalekis, E. Creatsas G. (2003) The clinical significance of Doppler findings in fetal middle cerebral artery during labor. Eur J Obstet Gynecol Reprod Biol, 109(1), pp. 45-50.

Kirkinen, P. Muller, R. Baumann, H. Mieth, D. Duc, G. Huch, R. Huch, A. (1987) Fetal cerebral vascular resistance. Lancet, 2(8555), pp. 392-3.

Kistka ZA, Palomar L, Boslaugh SE, et al.(2007) Risk for postterm delivery after previous postterm delivery. Am J Obstet Gynecol, 196(3), pp. 241 e1–6.

Kitlinski, M (2007) Fetal surveillance and outcome in postterm pregnancy, Doctor, Obstetric, Gynaecological and Prenatal Ultrasound Research.

Lingman, G. Marsal, K. (1986) Fetal central blood circulation in the third trimester of normal pregnancy--a longitudinal study.1. Aortic and umbilical blood flow. Early Hum Dev, 13(2), pp. 137-50.

Locci, M. Nazzaro, G. De Placido, G. Montemagno, U. (1992) Fetal cerebral haemodynamic adaptation: a progressive mechanism? Pulsed and color Doppler evaluation. J Perinat Med;20(5),pp. 337-43.

Mari, G. Deter, R.L. (1992). Middle cerebral artery flow velocity waveforms in normal and small-for-gestational-age fetuses. Am J Obstet Gynecol, 166(4) , pp.1262-70.

Mari, G., et al. (2015), Society for Maternal-Fetal Medicine (SMFM) Clinical Guideline #8: the fetus at risk for anemia diagnosis and management, Am J Obstet Gynecol, 212 (6), pp. 697-710.

Marsal, K. Lindblad, A. Lingman, G. Eik-Nes, S.H. (1984) Blood flow in the fetal descending aorta; intrinsic factors affecting fetal blood flow, i.e. fetal breathing movements and cardiac arrhythmia. Ultrasound Med Biol,10(3), pp. 339-48.

Martin, J.A. Hamilton, B.E. Sutton, P.D. (2007) Births: final data for 2005, Natl Vital Stat Rep, 56(6), pp. 1–103.

Maulik, D. (1989) Basic principles of Doppler ultrasound as applied in obstetrics. Clin Obstet Gynecol, 32, pp. 628–44.

Maulik, D. Yarlagadda, P. Downing, G. (1990) Doppler velocimetry in obstetrics. Obstet Gynecol Clin North Am,17(1), pp. 163–86.

McCallum, W.D. (1977) Qualitative estimation of blood velocity changes in human umbilical arteries after delivery. Early Hum Dev, 1(1), pp. 99-106.

McLean, M. Bisits, A. Davies, J. (1995) A placental clock controlling the length of human pregnancy. Nat Med, 1(5), pp. 460–463.

Mogren, I. Stenlund, H. Hogberg, U. (1999) Recurrence of prolonged pregnancy. Int J Epidemiol, 28(2), pp. 253–57.

Montan, S. Malcus, P. (1995) Amniotic fluid index in prolonged pregnancy: cohort study. J Mat Fetal Invest, 5, pp. 4-7.

Moore, W.M. Ward, B.S. Jones, V.P. Bamford, F.N. (1988) Sex difference in fetal head growth. Br J Obstet Gynaecol, 95(3), pp. 238-42.

Oehninger S, Hodgen GD. (1993) Hypothalamic-pituitary-ovarian uterine axis. In: Copeland LJ, Textbook of gynecology. Philadelphia: Saunders.

Oh, J.S. Wright, G. Coulam, C.B. (2002) Gestational sac diameter in very early pregnancy as a predictor of fetal outcome. Ultrasound Obstet Gynecol, 20(3), pp.267-269.

Olesen, A.W. Basso, O. Olsen, J.(2003) Risk of recurrence of prolonged pregnancy. BMJ, 326(787), pp.476.

Olofsson, P. Saldeen, P. Marsal, K. (1996) Fetal and uteroplacental circulatory changes in pregnancies proceeding beyond 43 weeks. Early Hum Dev,46(1-2) , pp.1–13

Olofsson, P. Saldeen, P. Marsal, K. (1997) Association between a low umbilical artery pulsatility index and fetal distress in labor in very prolonged pregnancies. Eur J Obstet Gynecol Reprod Biol, 73(1), pp. 23–9.

Ovel, S. (2014) Sonography exam review, 2nd ed, phildliphia: Mosby Elsevier Inc. p (379-380)

Pedersen, J.F. (1980) Ultrasound evidence of sexual difference in fetal size in first trimester. Br Med J, 281(6250), pp. 1253

Persson, P.H. Grennert, L. Gennser, G. Gullberg, B. (1978). Normal range curves for the intrauterine growth of the biparietal diameter. Acta Obstet Gynecol Scand Suppl, 78, pp. 15-20.

Persson, P.H. Weldner, B.M. (1986) Reliability of ultrasound fetometry in estimating gestational age in the second trimester. Acta Obstet Gynecol Scand, 65(5), pp. 481-3.

Phelan, J.P. Smith, C.V. Broussard, P. Small, M. (1987) Amniotic fluid volume assessment with the four-quadrant technique at 36-42 weeks' gestation, J Reprod Med,32(7), pp. 540-2.

Planiol, T. Pourcelot, L. Itti, R. (1974) Radioisotopes, ultrasonics and thermography in the diagnosis of cerebral circulatory disorders. Rev Electroencephalogr Neurophysiol Clin, 4(2), pp. 221-36.

Queenan, J.T. Thompson, W. Whitfield. C.R. Shah, S.I. (1972). Amniotic fluid volumes in normal pregnancies. Am J Obstet Gynecol, 114(1), pp. 34-8.

Rand, l. Robinson, J.n. Economy, Ke, et al (2000). Post-term induction of labour revisited. Obstet Gynecol, 96(5Pt1), pp.779-83.

Raymond, E.G. Cnattingius, S. Kiely, J.L. (1994). Effects of maternal age, parity, and smoking on the risk of stillbirth. Br J Obstet Gynaecol, 101(4), pp. 301-6.

Rightmire, D.A. Campbell, S. (1987) Fetal and maternal Doppler blood flow parameters in postterm pregnancies. Obstet Gynecol, 69(6), pp.891–4

Roger, C. Sanders, Thomas, C. Winter. (2007) Clinical Sonography a Practical Guide. 4th ed. Lippincort: Williams & Wilkins. p (416-417)

Rumack, Carol. M. Wilson, Stephanie. R. Charboneau, J. W.(2011) Diagnostic ultrasound. 4th ed. Philadelphia: Elsevier Mosby Inc.

Saling, E. (1968) Electronic and biochemical surveillance of the fetus during birth. Bull Soc R Belge Gynecol Obstet, 38(4), pp. 289-99.

Sandra, L. H, (2012) Textbook of Diagnostic Sonography .7th Ed. V1 Missouri: Mosby Inc

Savitz, D.A. Terry, J.W. Dole, N. (2002) Comparison of pregnancy dating by last menstrual period, ultrasound scanning, and their combination. Am J Obstet Gynecol, 187(6), pp.1660–66.

Selbing, A. McKay, K. (1985) Ultrasound in first trimester shows no difference in fetal size between the sexes. Br Med J, 290(6470), pp.750

Selbing, A.(1983) The pregnant population and a fetal crown-rump length screening program. Acta Obstet Gynecol Scand, 62(2), pp.161-4.

Settlage, D.S. Motoshima, M. Tredway, D.R.(1973) Sperm transport from the external cervical os to the fallopian tubes in women: a time and quantitation study. Fertil Steril , 24(9), pp.655-661.

Simpson, P.D. Stanley, K.P. (2011) Prolonged pregnancy. Obstetrics, Gynaecology & Reproductive Medicine, 21 (9), pp. 257-62.

Spurway, Jacqueline et al.(2012) The development, structure and blood flow within the umbilical cord with particular reference to the venous system. Australasian journal of ultrasound in medicine,15(3), pp. 97-102.

Srikumar, S. Debnath, J. Ravikumar, R. Bandhu, H. Maurya, V. (2017). Colour Doppler indices of the umbilical and fetal middle cerebral artery at 18–40 weeks of normal gestation. Med J Armed Forces India, 73(3),pp. 232–241.

Starks, G.C. (1980) Correlation of meconium-stained amniotic fluid, early intrapartum fetal pH, and Apgar scores as predictors of perinatal outcome. Obstet Gynecol, 56(5), pp.604-9.

Stokes, H.J. Roberts, R.V. Newnham, J.P. (1991) Doppler flow velocity waveform analysis in postdate pregnancies. Aust N Z J Obstet Gynaecol, 31(1), pp.27–30

Stotland, N.E.Washington, A.E. Caughey, A.B. (2007) Prepregnancy bodymass index and the length of gestation at term. AmJObstetGynecol, 197(4), pp.3781–5.

Subhra, Ghosh. Kirtirekha, Mohapatra. Sanjukta Samal. Pallabi Nayak, (2016) Study of Doppler indices of umbilical artery and middle cerebral artery in pregnancies at and beyond forty weeks of gestation, International Journal of Reproduction, Contraception, Obstetrics and Gynecology, 5(12), pp.4174-4179

Torricelli, M. Ignacchiti, E. Giovannelli, A. (2006) Maternal plasma corticotrophin-releasing factor and urocortin levels in postterm pregnancies. Eur J Endocrinol, 154, pp. 281–285.

Tunon, K. Eik-Nes S.H. Grottum, P. vonDuring, V. Kahn, J.A. (2000) Gestational age in pregnancies conceived after in vitro fertilization: a comparison between age assessed from oocyte retrieval, crown-rump length and biparietal diameter.Ultrasound Obstet Gynecol, 15, pp.41-6.

Tunon, K. Eik-Nes, S.H. Grottum, P. (1998) The impact of fetal, maternal and external factors on prediction of the day of delivery by the use of ultrasound. Ultrasound Obstet Gynecol,11(2), pp.99-103.

Veille, J.C. Penry,M. Mueller-Heubach, E. (1993). Fetal renal pulsed Doppler waveform in prolonged pregnancies. Am J Obstet Gynecol, 169(4), pp.882–4.

Ventura, S.J. Martin, J.A. Curtin, S.C. (2000) Births: final data for 1998. Natl Vital Stat Rep, 48(3), pp.1–100.

Wald, N. Cuckle, H. Nanchahal, K. Turnbull, A.C. (1986) Sex differences in fetal size early in pregnancy. Br Med J, 292(6513), pp.137.

Walker, J. (1959) The human foetal dissociation curve for oxygen. Scott Med J, 4, pp.405-14.

Wisser, J. Dirschedl, P. Krone, S. (1994) Estimation of gestational age by transvaginal sonographic measurement of greatest embryonic length in dated human embryos. Ultrasound Obstet Gynecol, 4(6), pp.457-62.

Wladimiroff, J.W. Tonge, H.M. Stewart, P.A. (1986) Doppler ultrasound assessment of cerebral blood flow in the human fetus. Br J Obstet Gynaecol, 93(5), pp.471-5.

World Health Organisation (2003). International Classification of Disease. ICD-10. Chapter XV: 048. Geneva.

Zimmermann, P. Alback, T. Koskinen, J. Vaalamo, P. Tuimala, R. Ranta,T. (1995) Doppler flow velocimetry of the umbilical artery, uteroplacental arteries and fetal middle cerebral artery in prolonged pregnancy. Ultrasound Obstet Gynecol, 5(3), pp.189–97.

Appendices (A)

Sonographic images selected as the sample of the study:

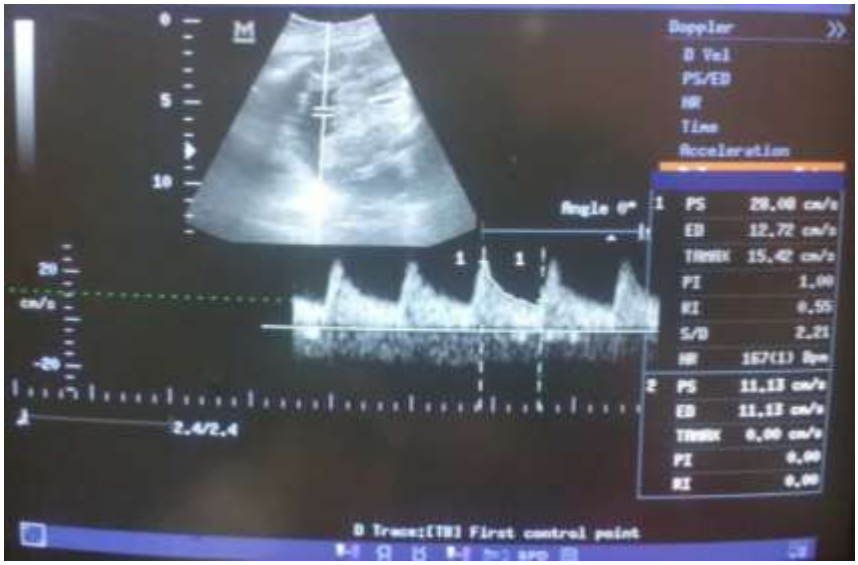


Image (1.a) shows color Doppler umbilical artery waveform at 42 WKs

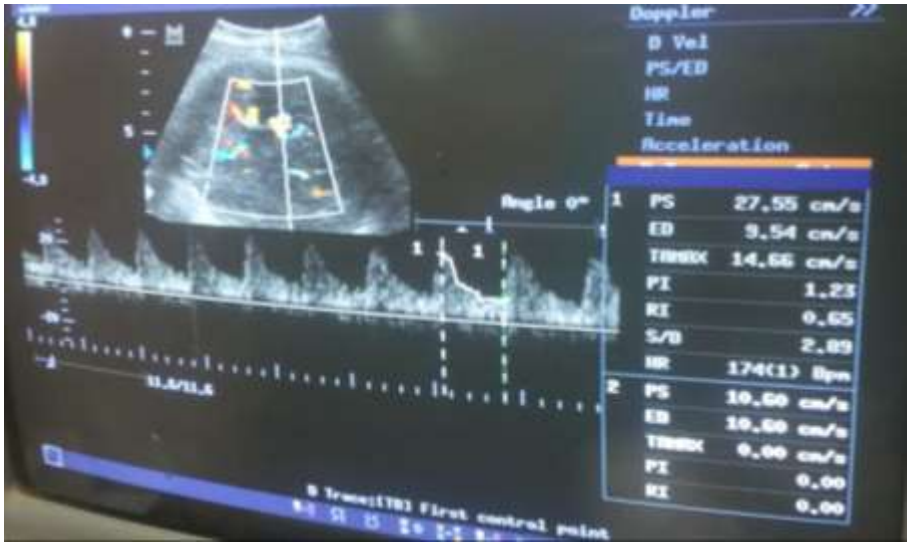


Image (2.b) shows color Doppler middle cerebral artery waveform at 42 WKs

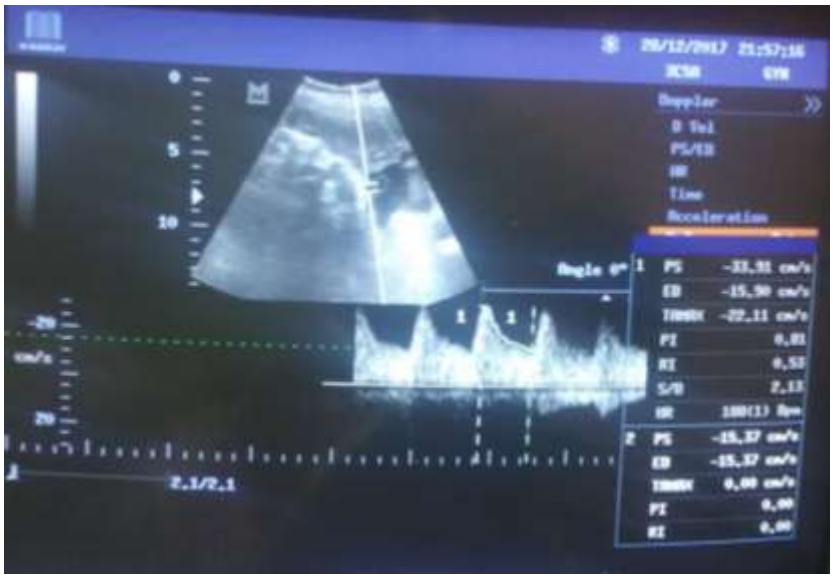


Image (2.a) shows color Doppler umbilical artery waveform at 41WKs

Image (2.b) shows color Doppler middle cerebral artery waveform at 41 WKs

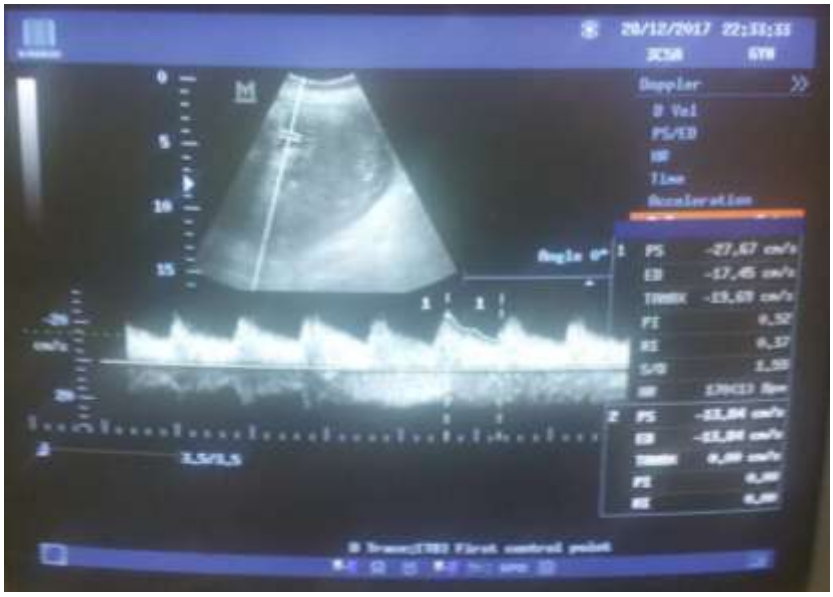


Image (3.a) shows color Doppler umbilical artery waveform at 41 WKs

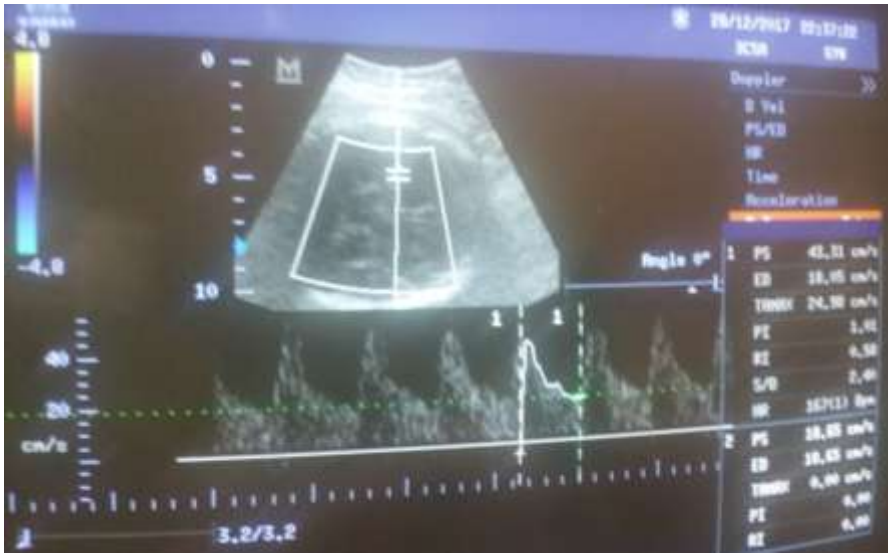


Image (3.b) shows color Doppler middle cerebral artery waveform at 41 WKs

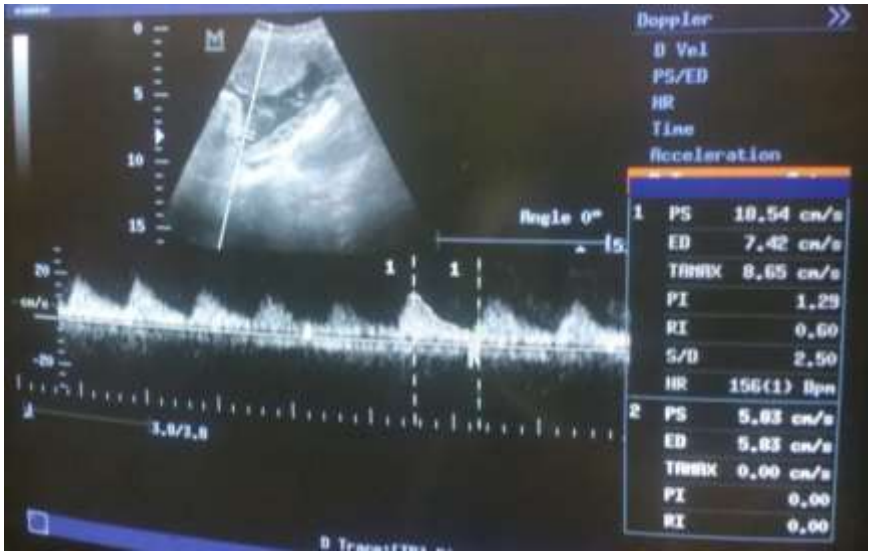


Image (4.a) shows color Doppler umbilical artery waveform at 35 WKs

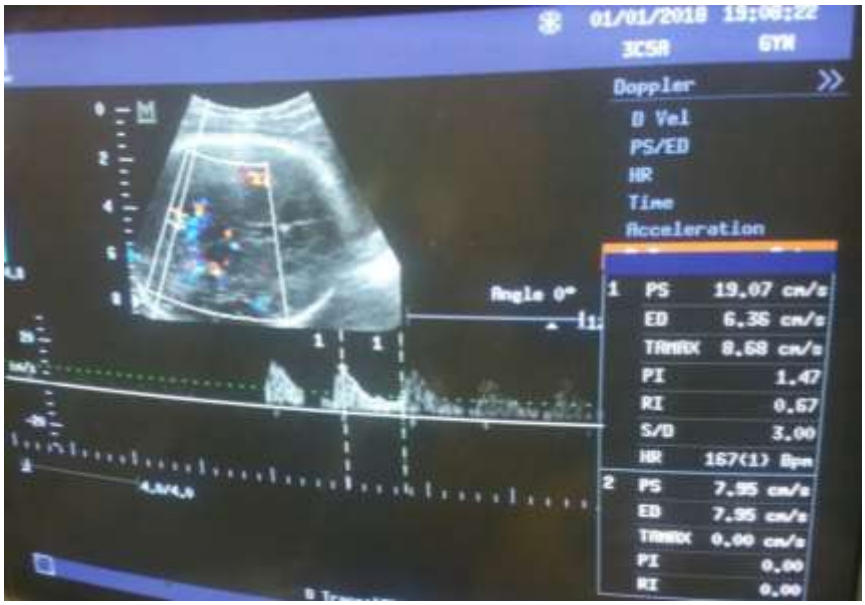


Image (4.b) shows color Doppler middle cerebral artery waveform at 35 WKs

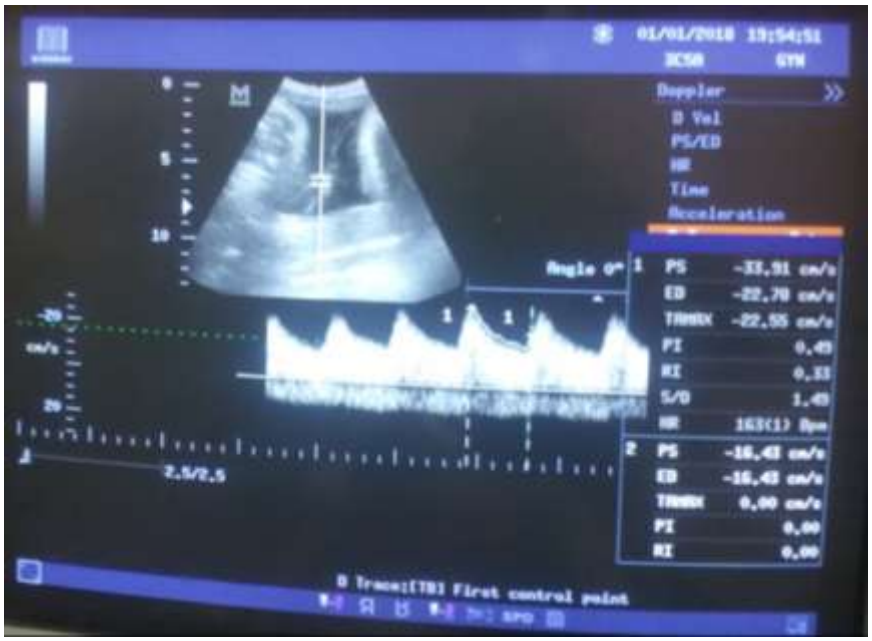


Image (5.a) shows color Doppler umbilical artery waveform at 38 WKs

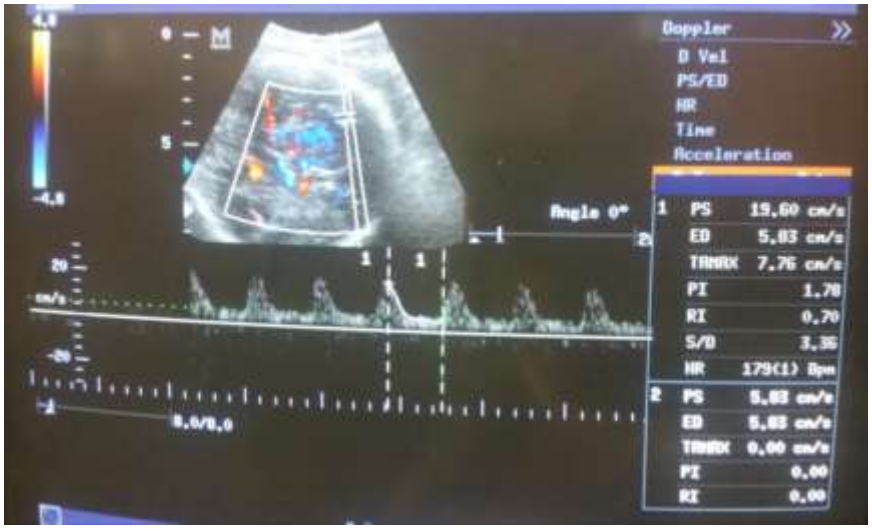


Image (5.b) shows color Doppler middle cerebral artery waveform at 38 WKs

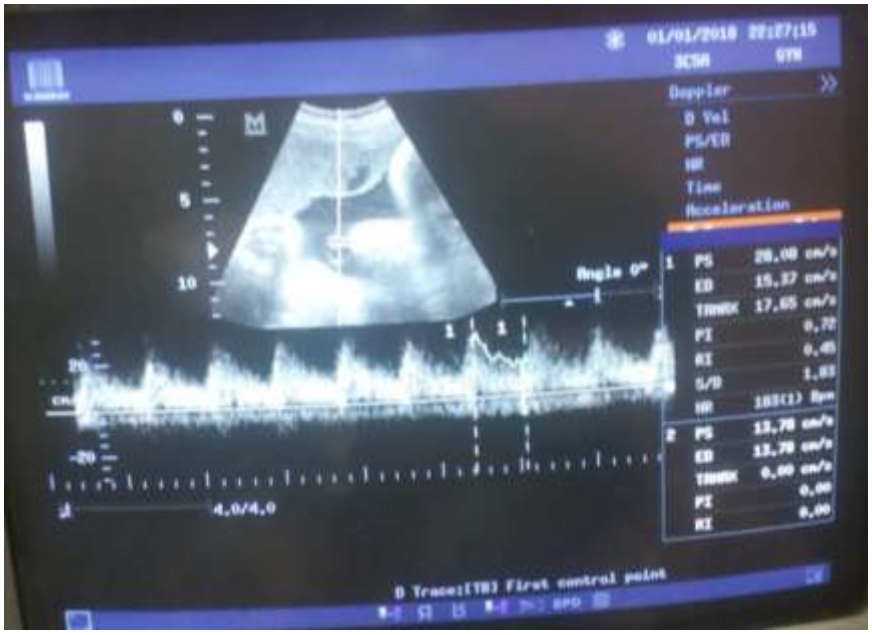


Image (6.a) shows color Doppler umbilical artery waveform at 42 WKs

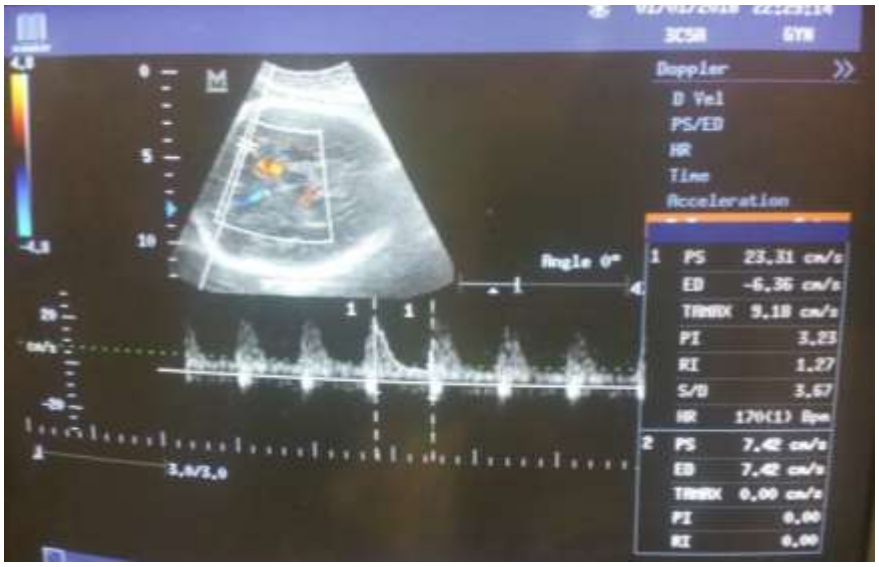


Image (6.b) shows color Doppler middle cerebral artery waveform at 42 WKs

Image (7.a) shows color Doppler umbilical artery waveform at 38 WKs

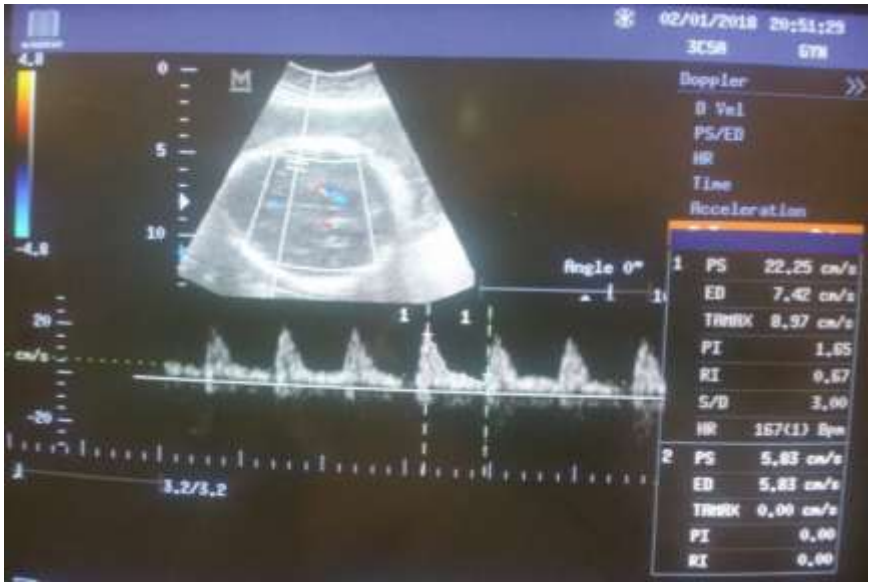


Image (7.b) shows color Doppler middle cerebral artery waveform at 38 WKs

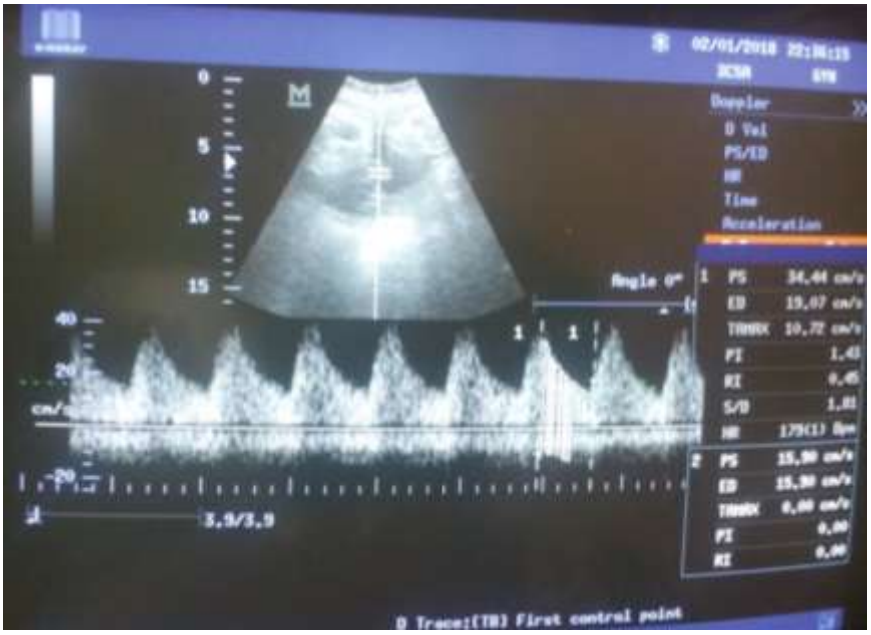


Image (8.a) shows color Doppler umbilical artery waveform at 43WKs

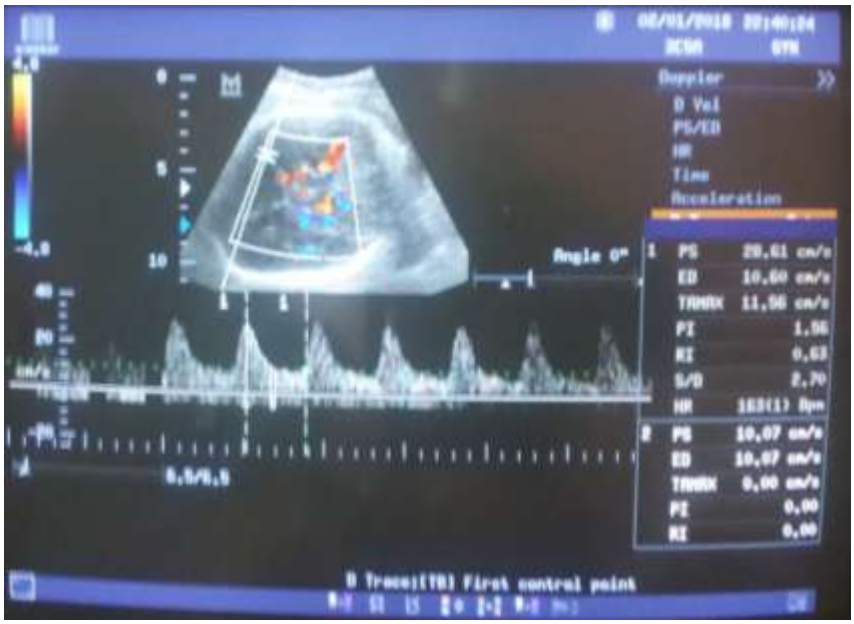


Image (8.b) shows color Doppler middle cerebral artery waveform at 43 WKs

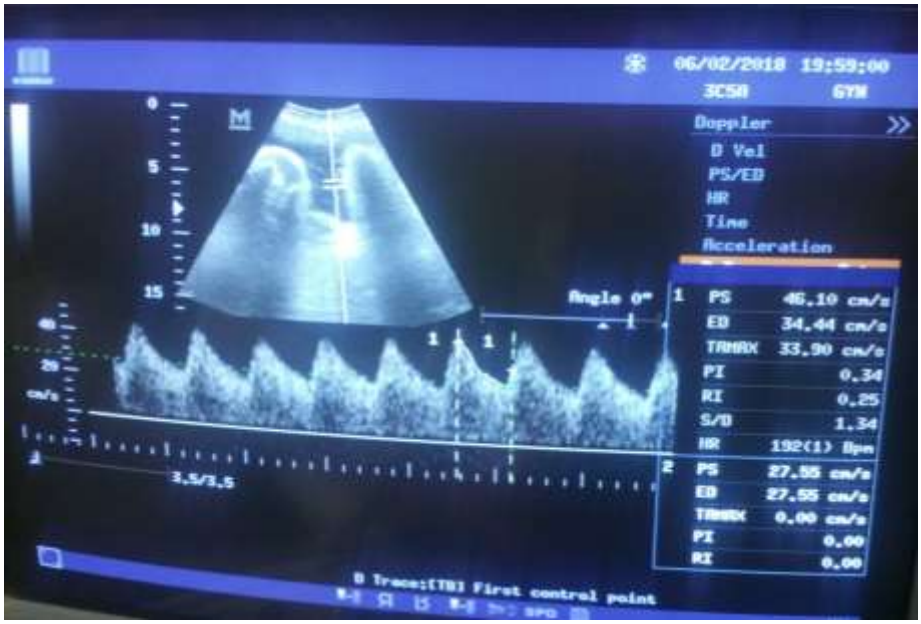


Image (9.a) shows color Doppler umbilical artery waveform at 42 WKs

Image (9.b) shows color Doppler middle cerebral artery waveform at 42 WKs

Image (10.a) shows color Doppler umbilical artery waveform at 39 WKs

Image (10.b) shows color Doppler middle cerebral artery waveform at 39 WKs

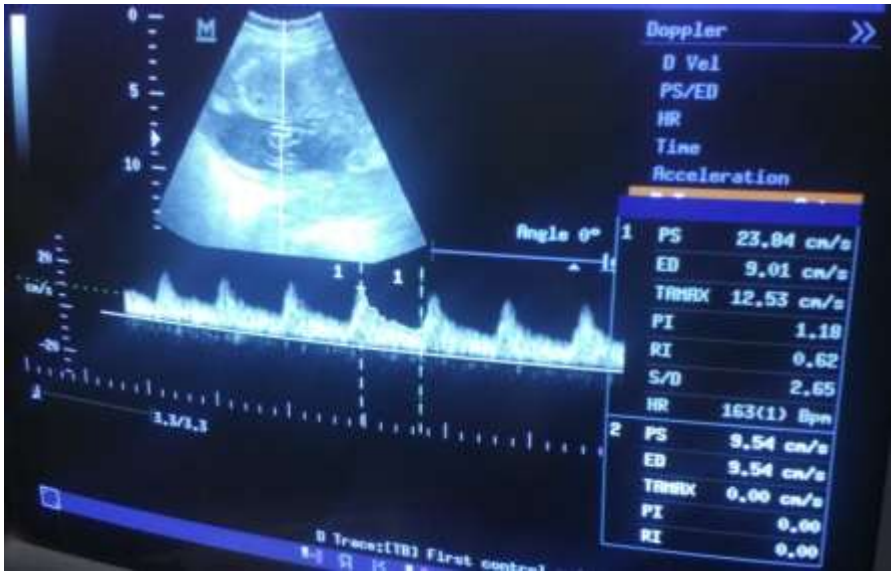


Image (11.a) shows color Doppler umbilical artery waveform at 41 WKs

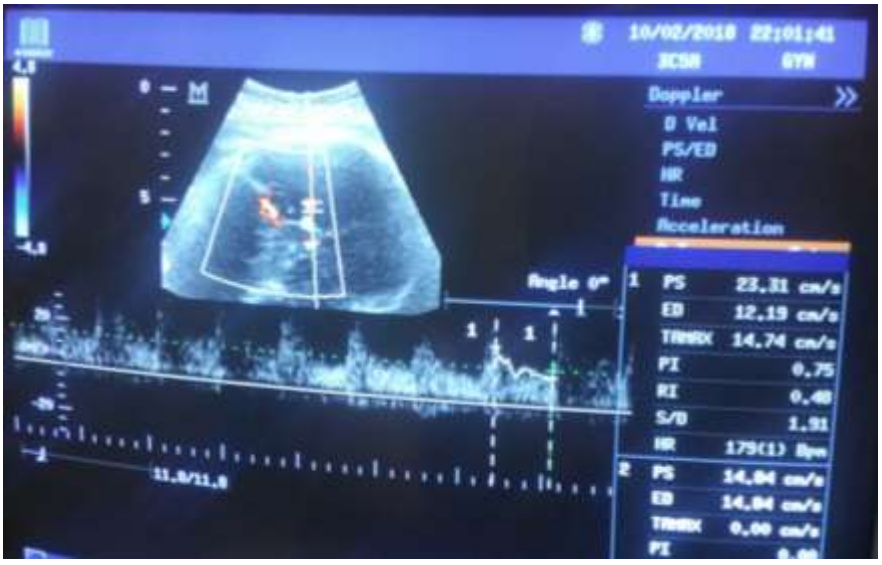


Image (11.b) shows color Doppler middle cerebral artery waveform at 41 WKs

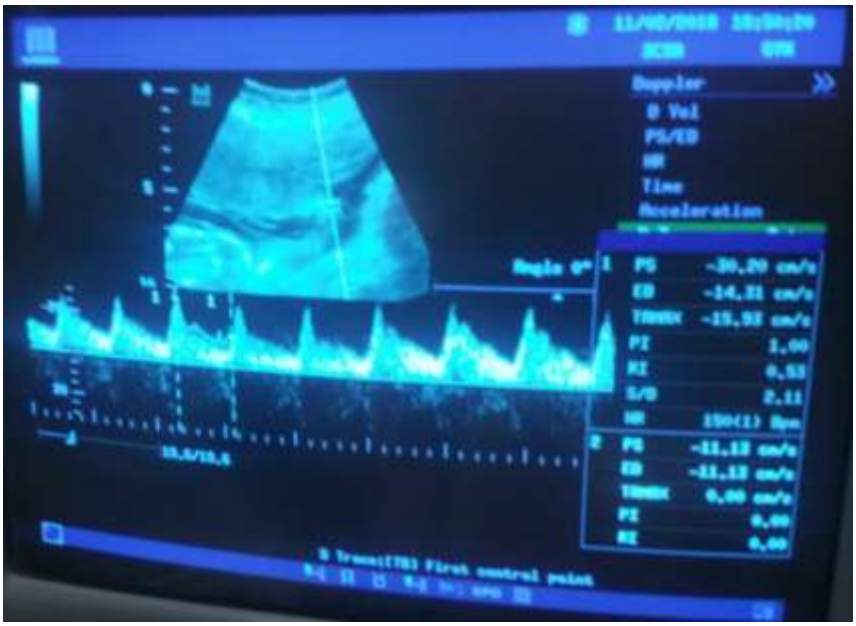


Image (12.a) shows color Doppler umbilical artery waveform at 35 WKs

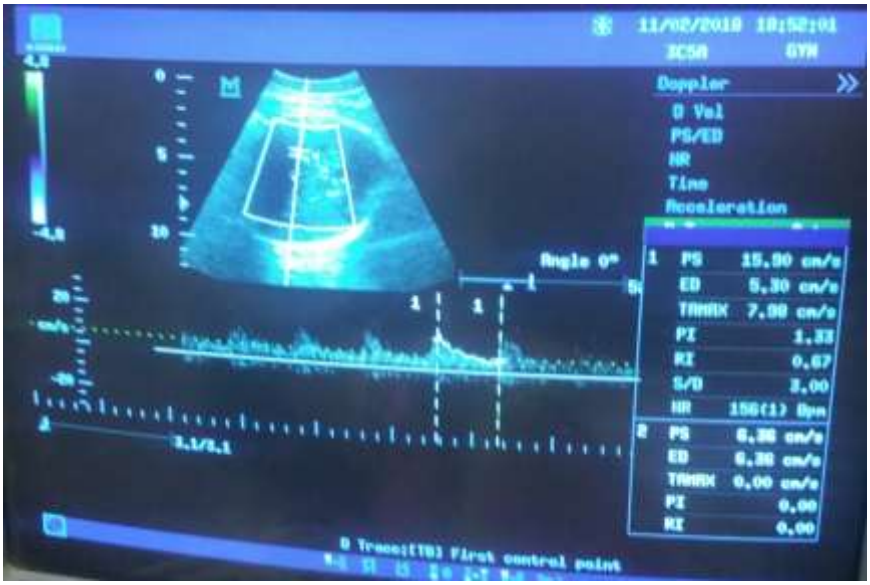


Image (12.b) shows color Doppler middle cerebral artery waveform at 35 WKs

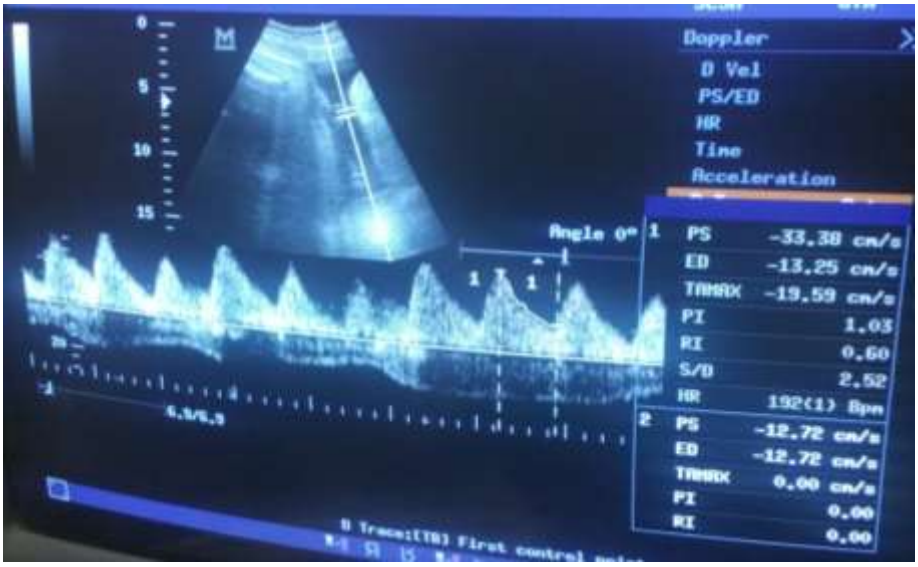


Image (13.a) shows color Doppler umbilical artery waveform at 41 WKs

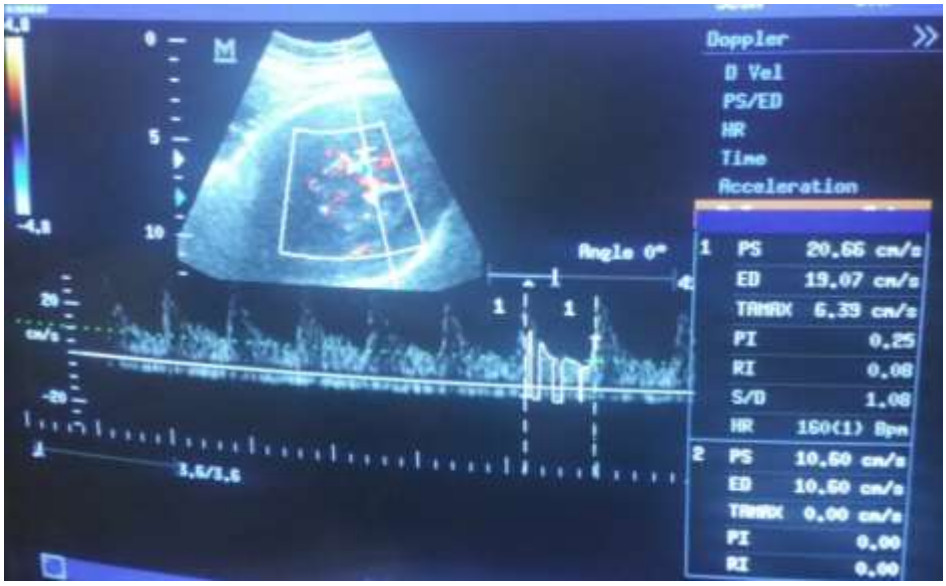


Image (13.b) shows color Doppler middle cerebral artery waveform at 41 WKs

Image (14.a) shows color Doppler umbilical artery waveform at 38 WKs

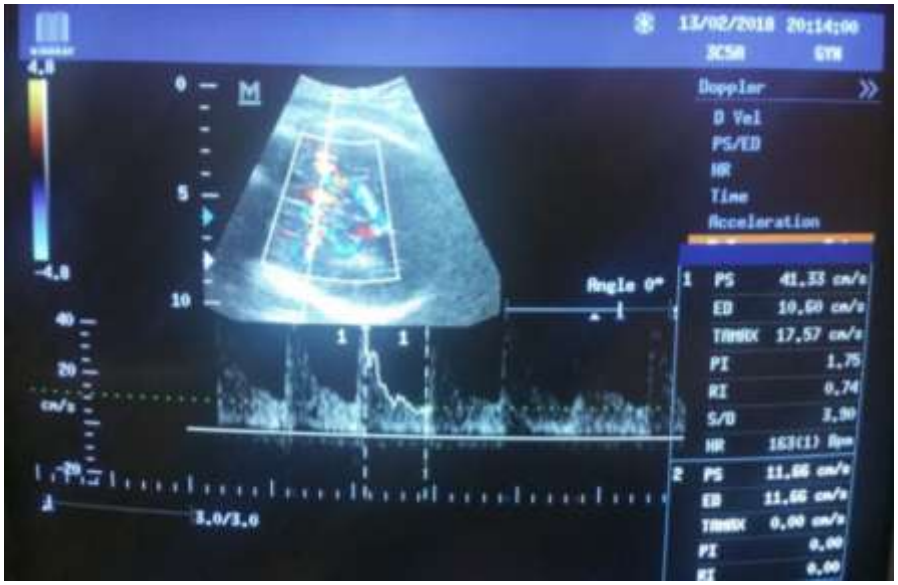


Image (14.b) shows color Doppler middle cerebral artery waveform at 38 WKs

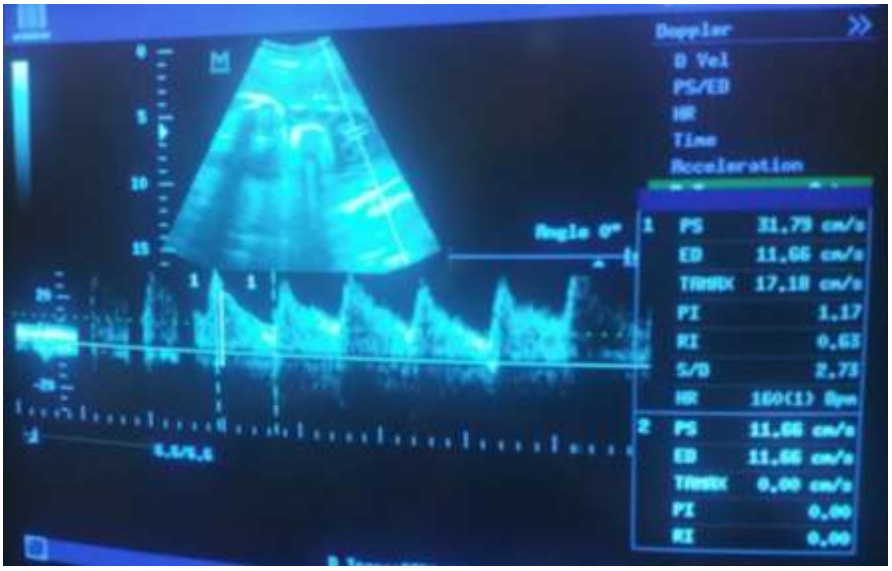


Image (15.a) shows color Doppler umbilical artery waveform at 38 WKs

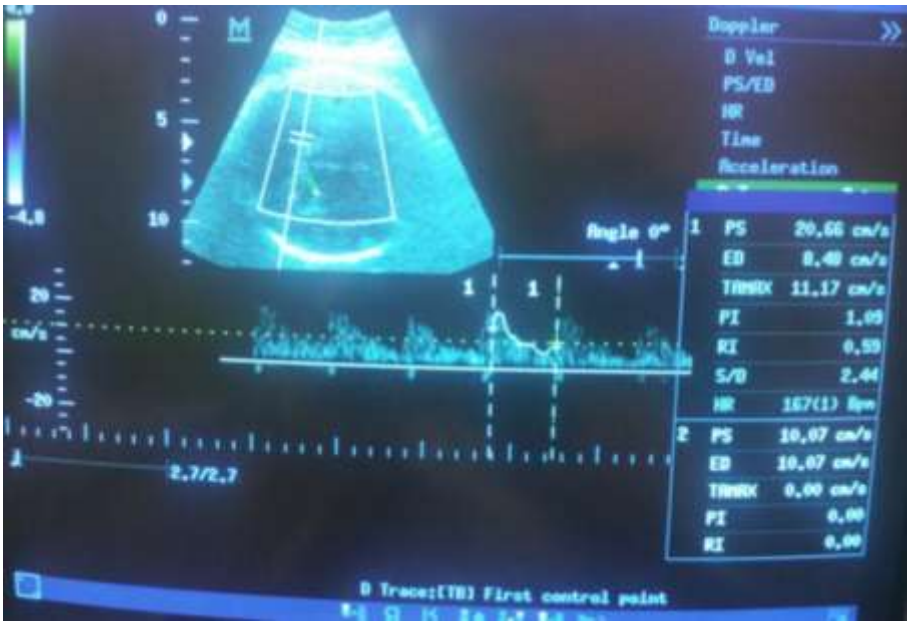


Image (15.b) shows color Doppler middle cerebral artery waveform at 38 WKs

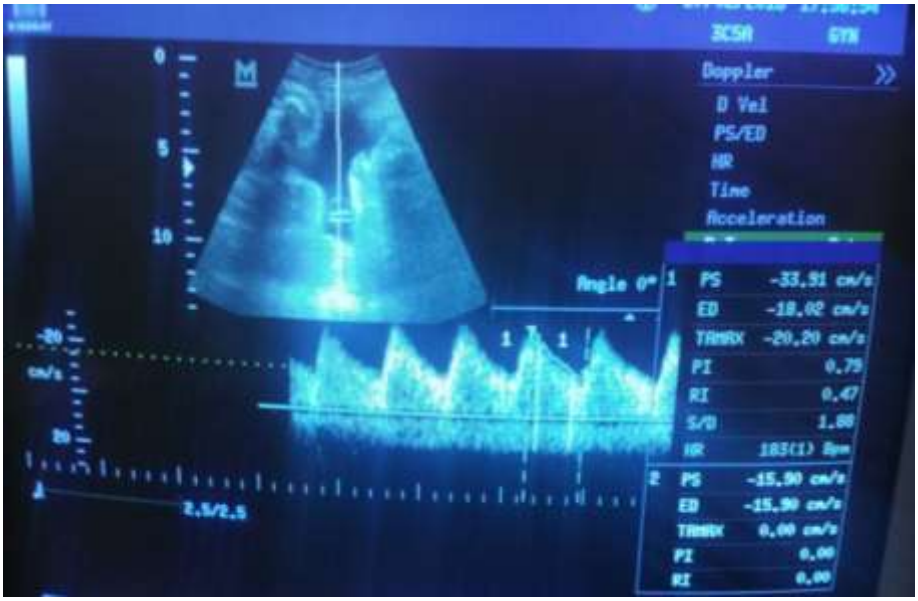


Image (16.a) shows color Doppler umbilical artery waveform at 36WKs

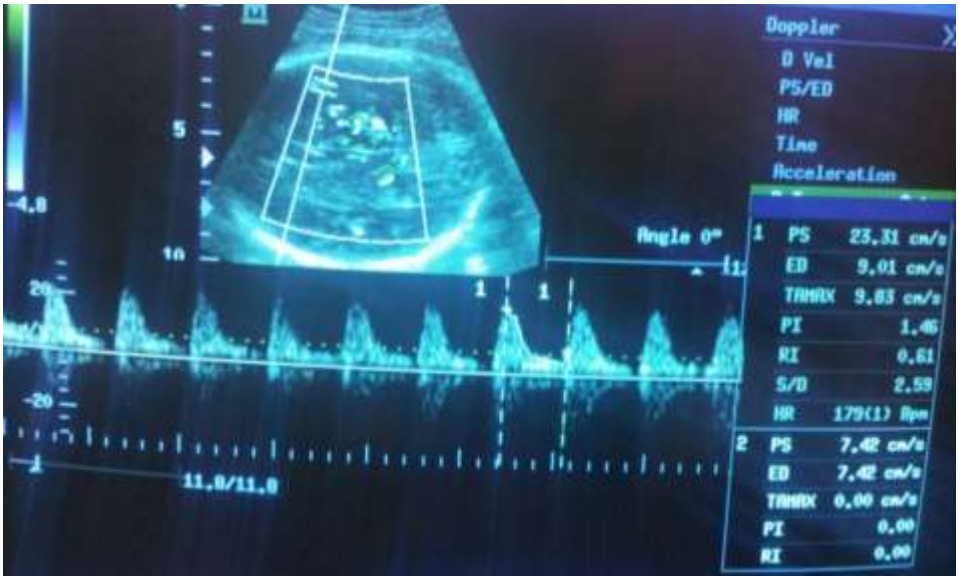


Image (16.b) shows color Doppler middle cerebral artery waveform at 36 WKs

Image (17.a) shows color Doppler umbilical artery waveform at 38 WKs

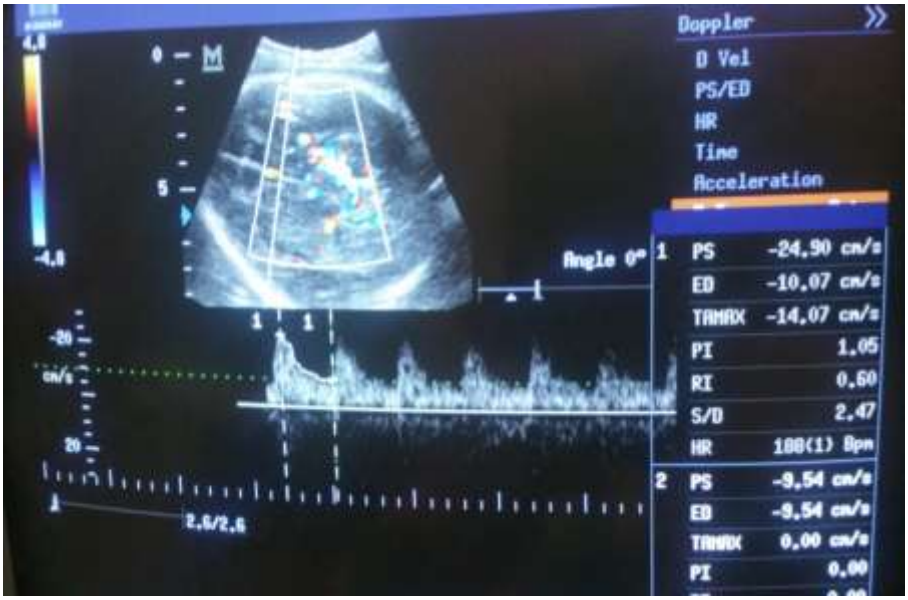


Image (17.b) shows color Doppler middle cerebral artery waveform at 38 WKs

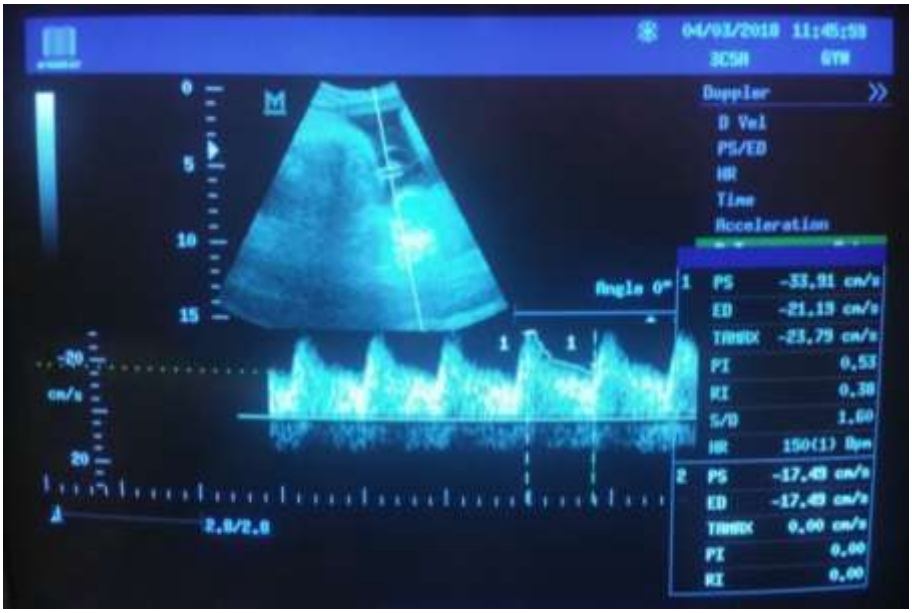


Image (18.a) shows color Doppler umbilical artery waveform at 40 WKs

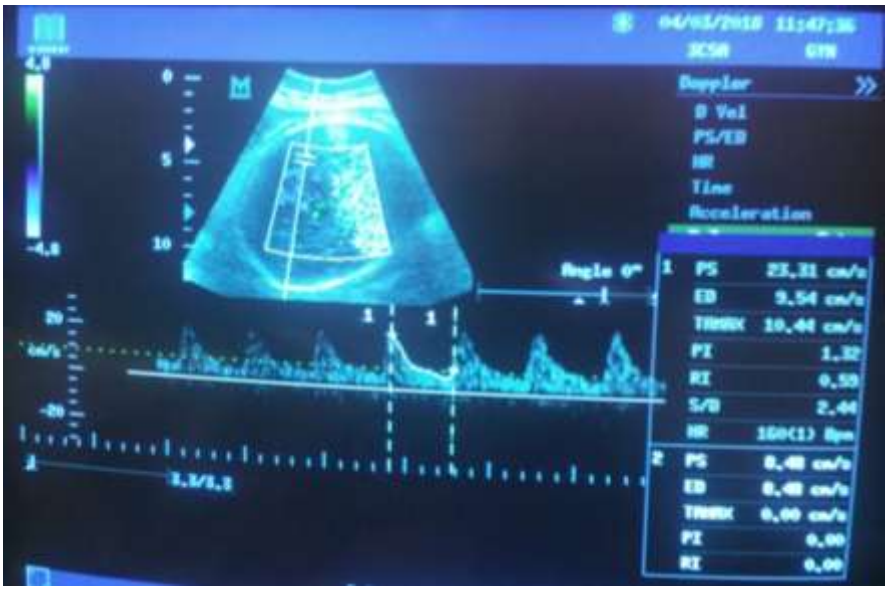


Image (18. b) shows color Doppler middle cerebral artery waveform at 40 WKs

Image (19.a) shows color Doppler umbilical artery waveform at 38 WKs

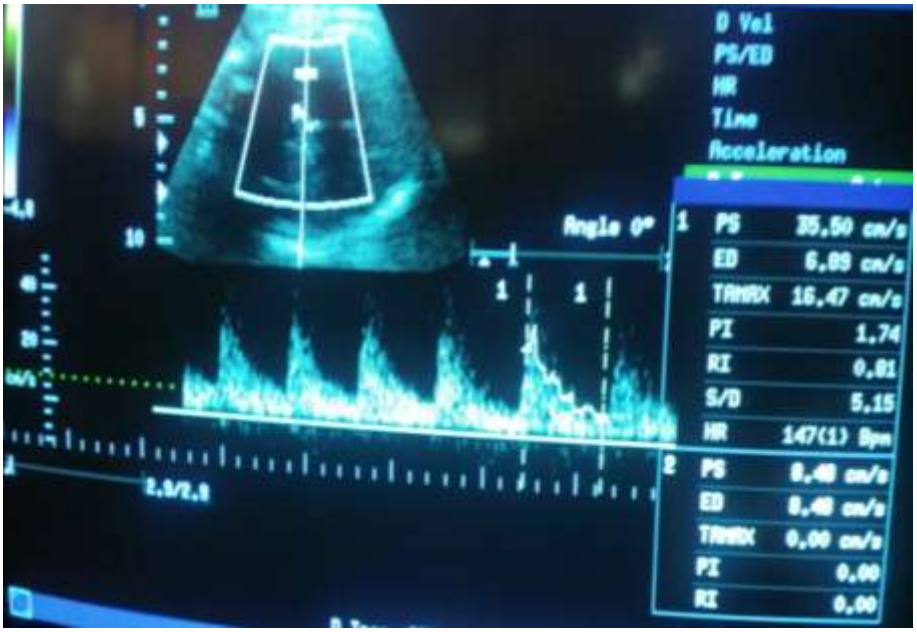


Image (19.b) shows color Doppler middle cerebral artery waveform at 38 WKs

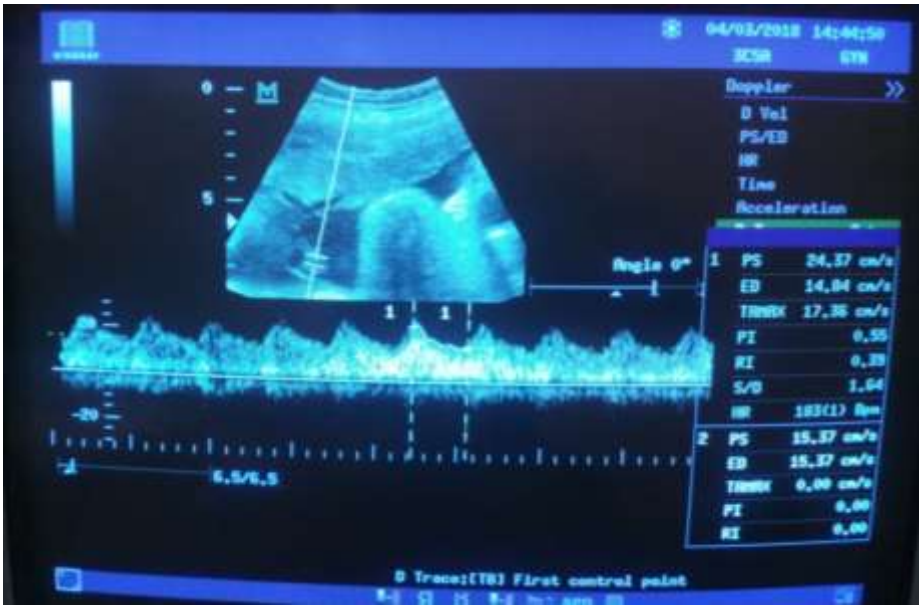


Image (20.a) shows color Doppler umbilical artery waveform at 37 WKs

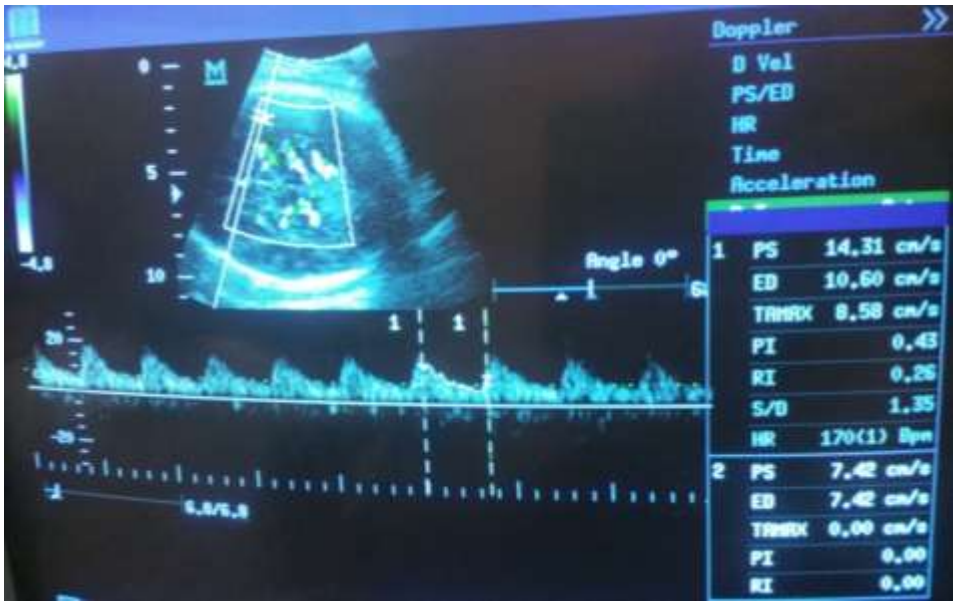


Image (20.b) shows color Doppler middle cerebral artery waveform at 37 WKs

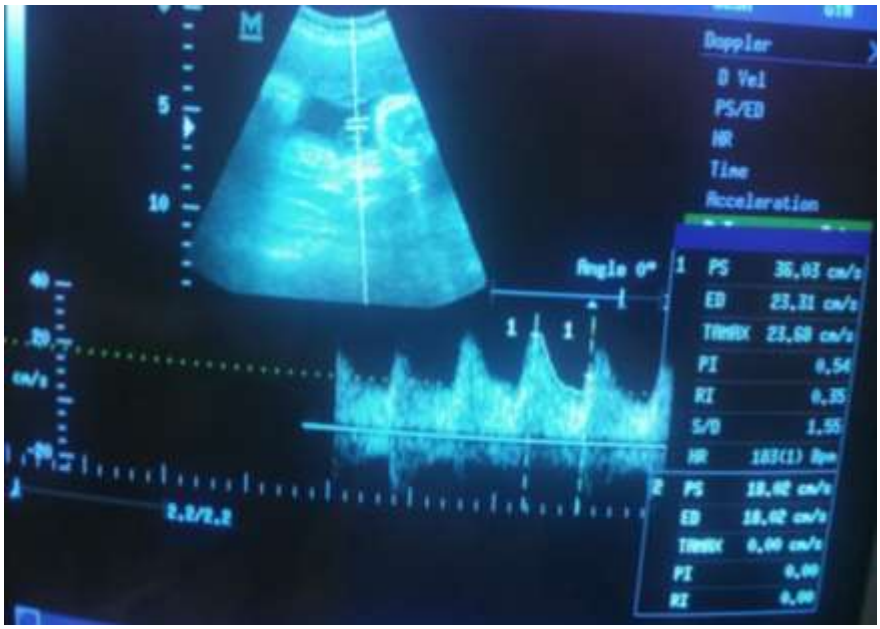


Image (21.a) shows color Doppler umbilical artery waveform at 39 WKs

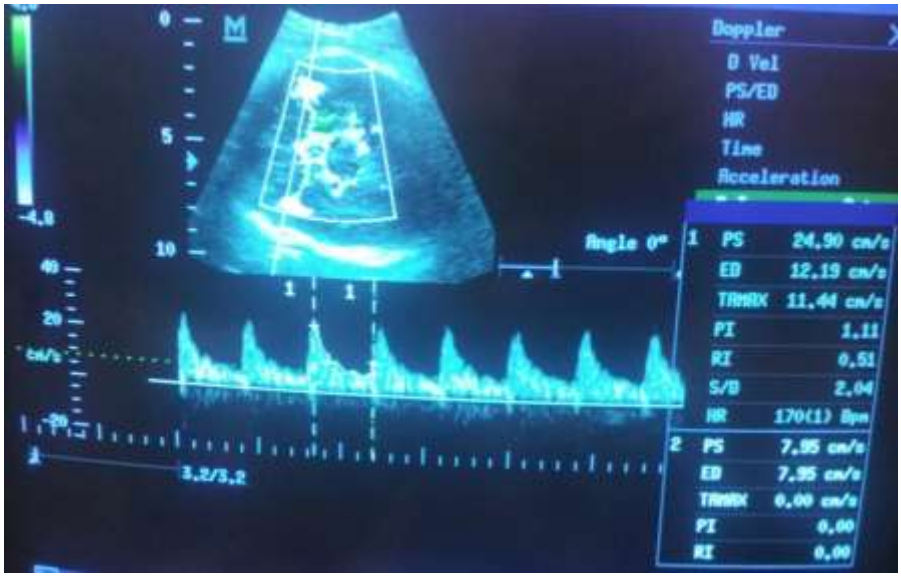


Image (21.b) shows color Doppler middle cerebral artery waveform at 39 WKs

Image (22.a) shows color Doppler umbilical artery waveform at 36 WKs

Image (22.b) shows color Doppler middle cerebral artery waveform at 36 WKs

Image (23.a) shows color Doppler umbilical artery waveform at 38 WKs

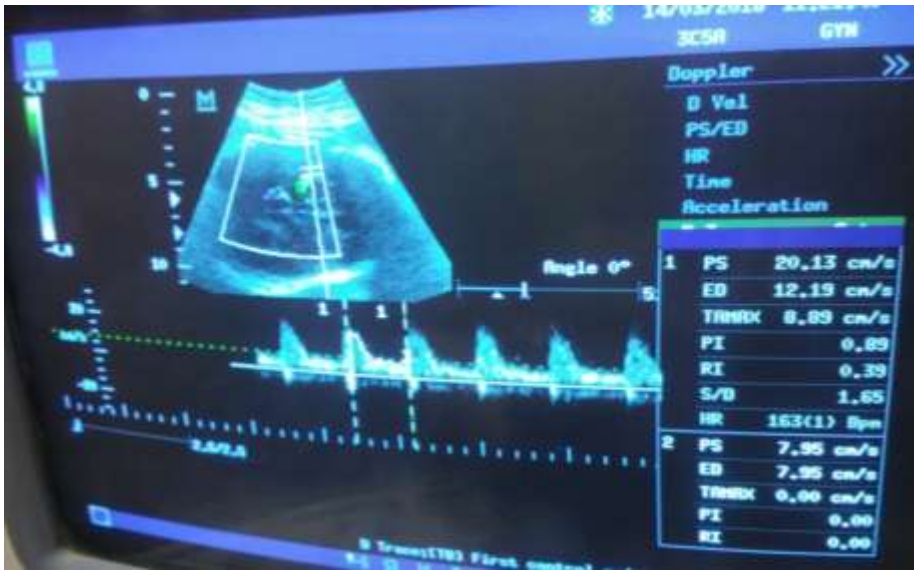


Image (23.b) shows color Doppler middle cerebral artery waveform at 38 WKs

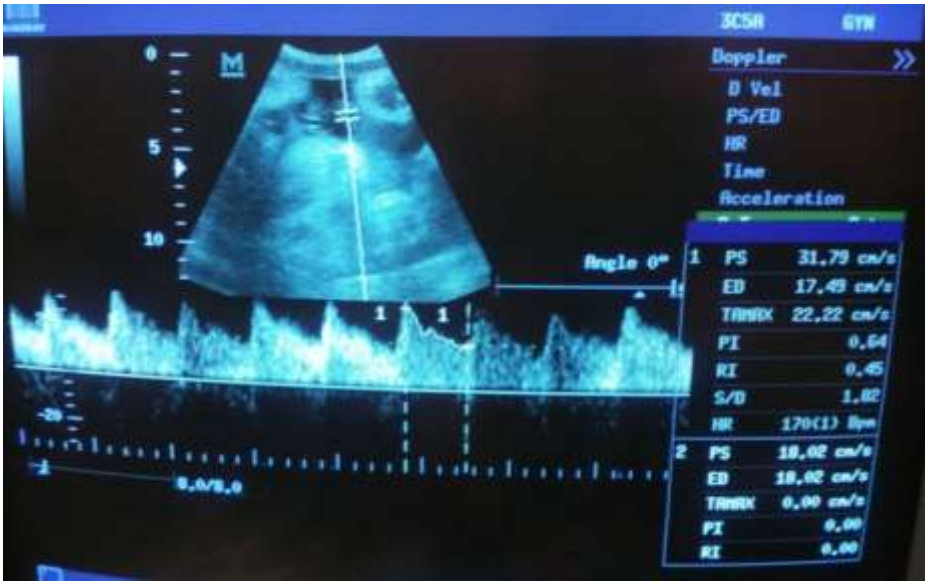


Image (24.a) shows color Doppler umbilical artery waveform at 37 WKs

Image (24.b) shows color Doppler middle cerebral artery waveform at 37 WKs

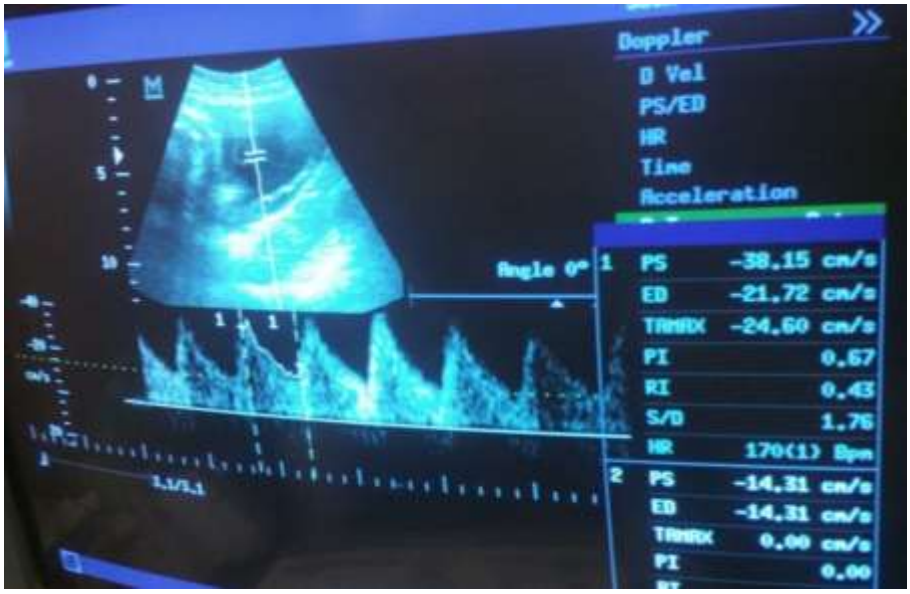


Image (25.a) shows color Doppler umbilical artery waveform at 36 WKs

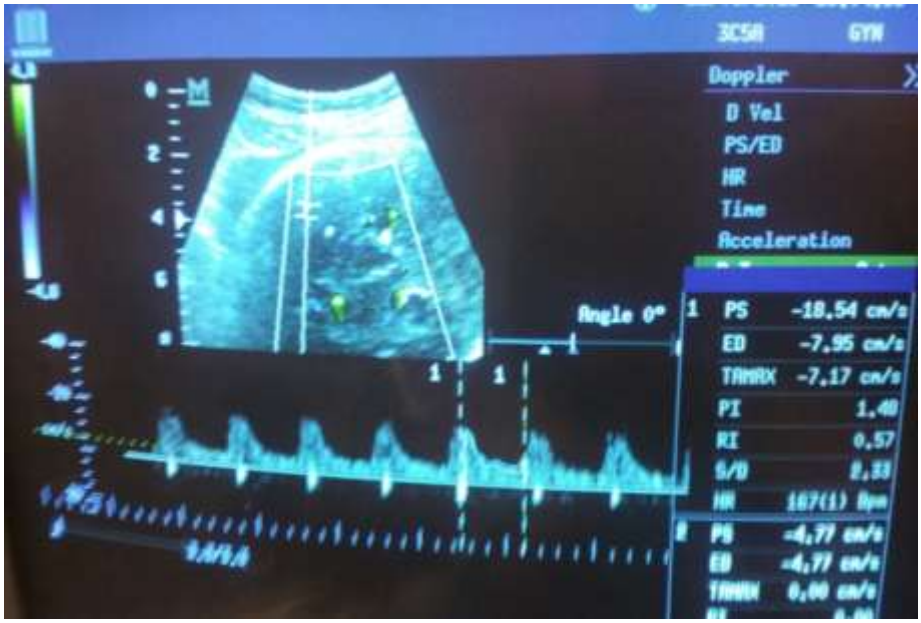


Image (25.b) shows color Doppler middle cerebral artery waveform at 36 WKs

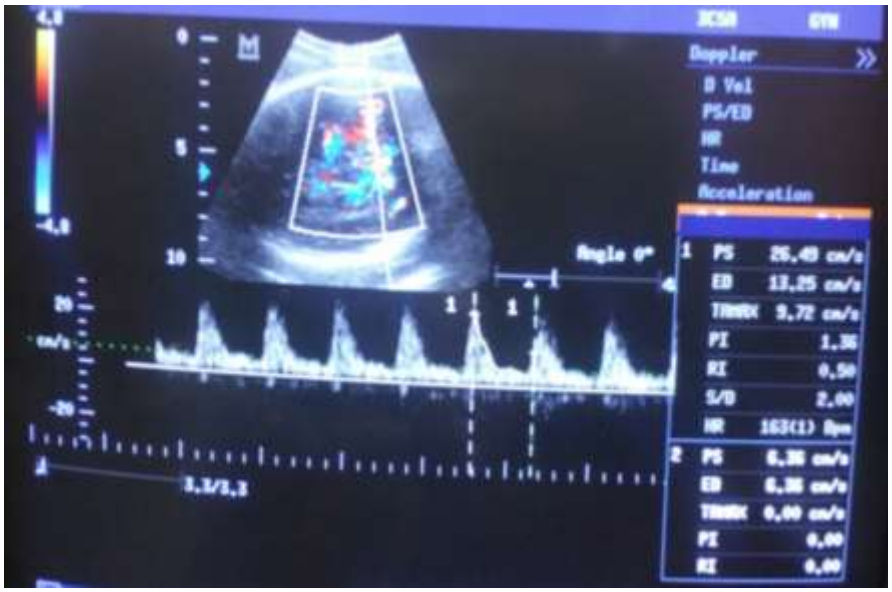


Image (26) shows color Doppler middle cerebral artery waveform at 38 WKs

Image (27) shows color Doppler middle cerebral artery waveform at 37 WKs

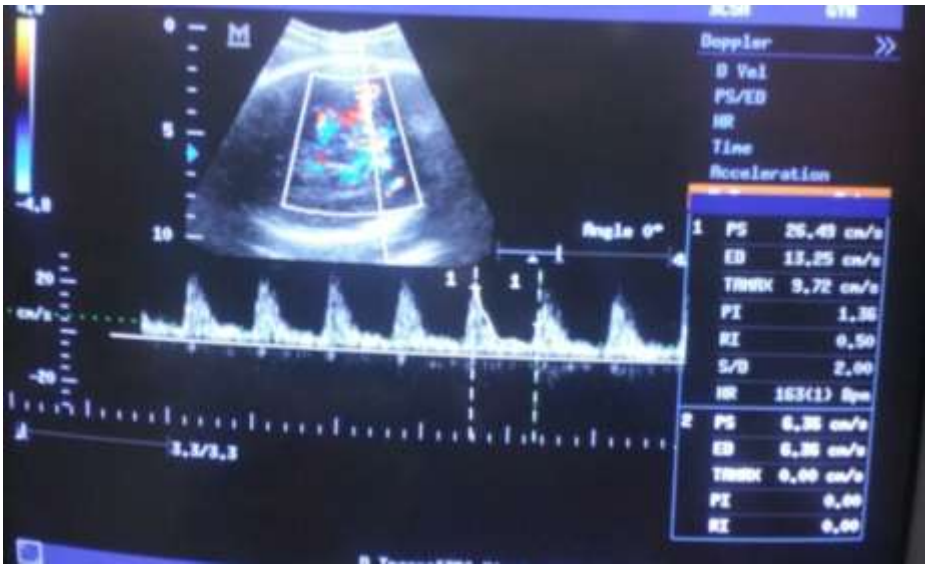


Image (28) shows color Doppler middle cerebral artery waveform at 36 WKs

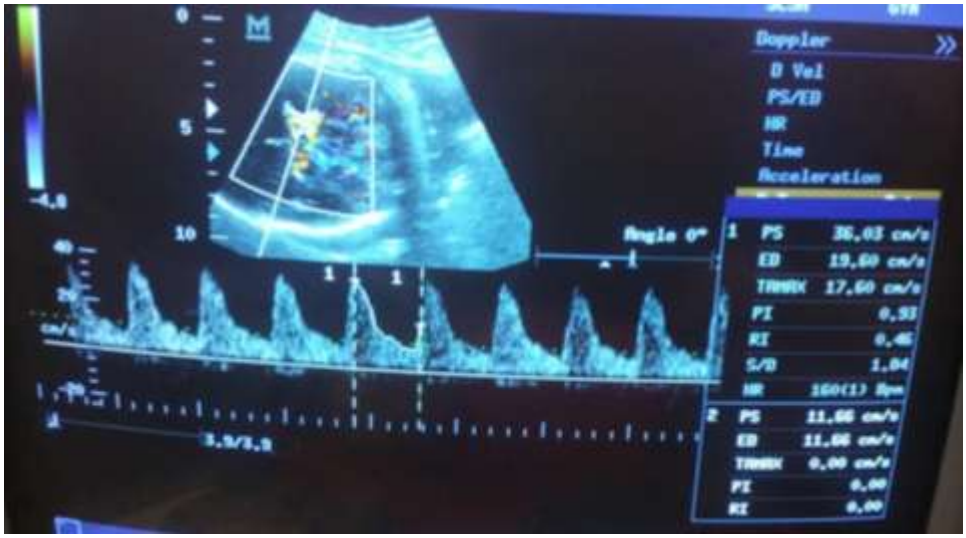


Image (29) shows color Doppler middle cerebral artery waveform at38 WKs

Image (30) shows color Doppler umbilical artery waveform at 36 WKs

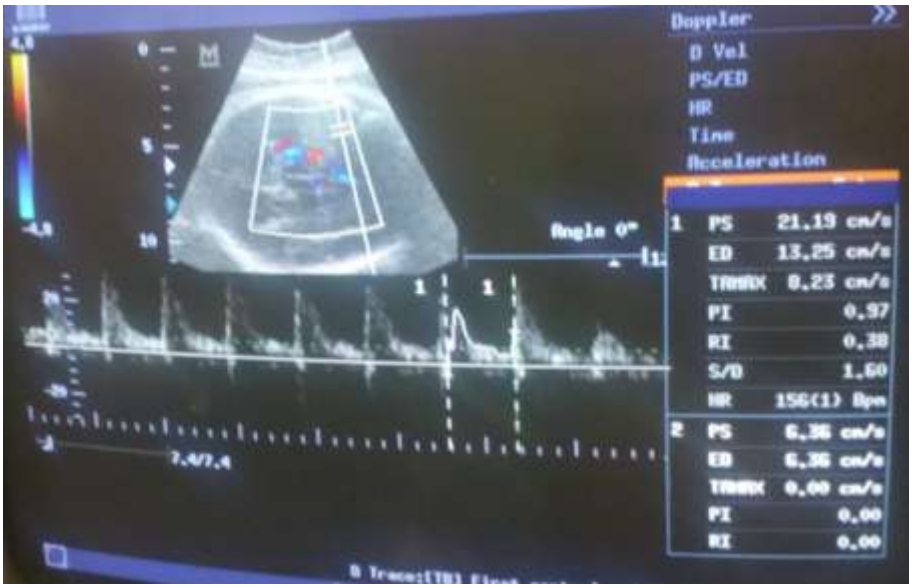


Image (31) shows color Doppler middle cerebral artery waveform at 38 WKs

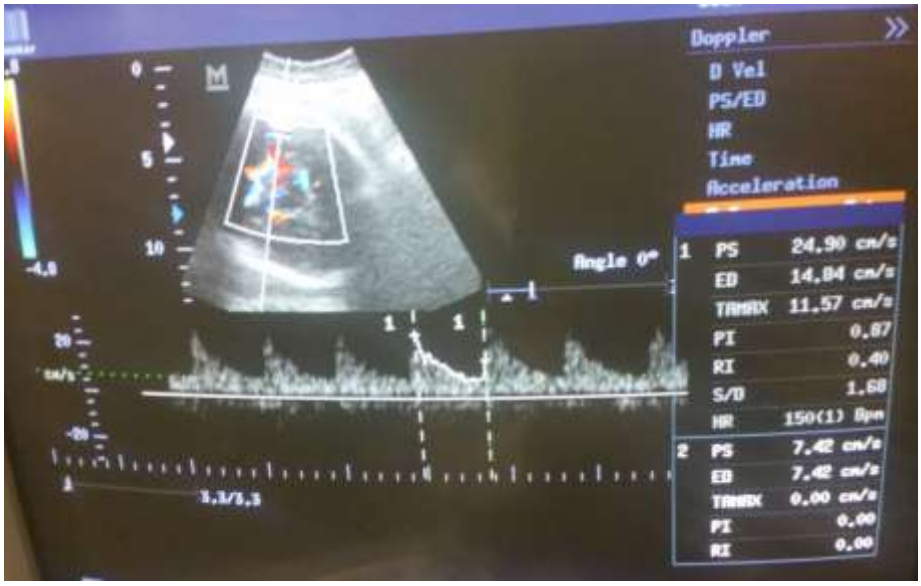


Image (32) shows color Doppler middle cerebral artery waveform at 39 WKs

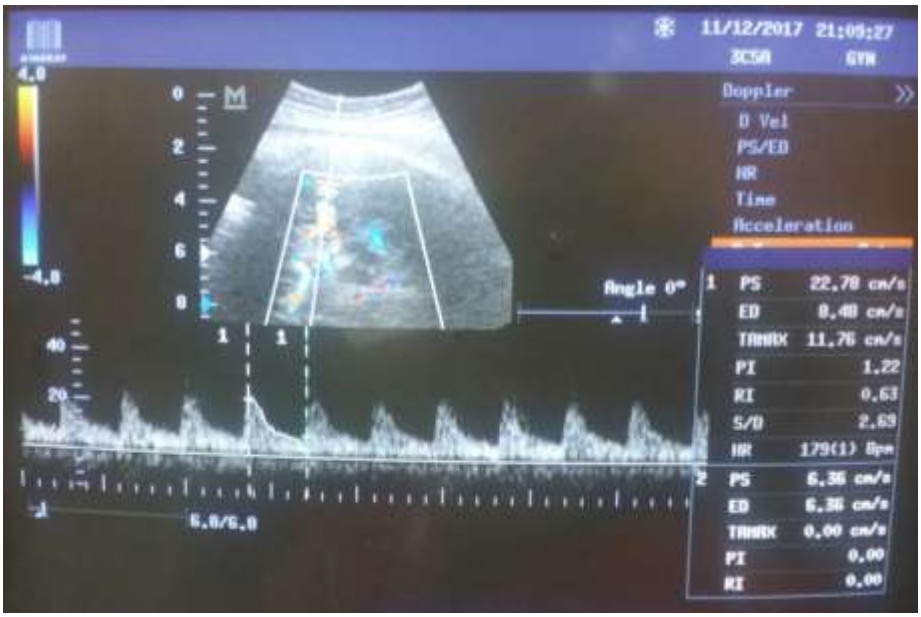


Image (32) shows color Doppler middle cerebral artery waveform at 39 WKs

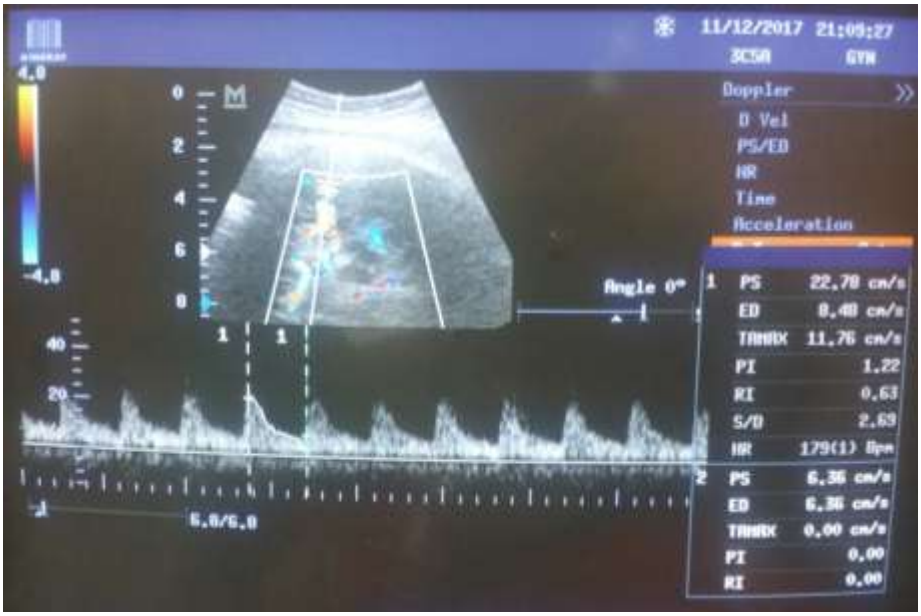


Image (33) shows color Doppler middle cerebral artery waveform at 39 WKs

Appendices (B)

Clinical data collection sheet

Assessment of Late and Postterm Pregnancies using color Doppler ultrasound

Date :…… Patient NO :( ) Tel NO: ……

<u>Patient data :</u>

1. Age: …….

2. Parity: Multipara ( ) Nullipara ( )

3. Occupation: Employ ( ) Unemployed ( )

4. LMP:……

Specific information:

5. Femoral length (FL) …….

6. biparietal diameter(BPD) …….

7. Amniotic fluid index (AFI)……

8. Abdominal circumference (AC)……..

9. Fetal weight EFW:……..

10. Gestation age:……..

Middle cerebral:

11.PSV…… EDV……

12. S/D ratio of the middle cerebral……

13. pulsatility index(PI) of the middle cerebral……..

14. Resistance index (RI) of the middle cerebral and

……..

Umbilical artery:

15.PSV…… EDV……

16. S/D ratio of the umbilical artery……

17. pulsatility index(PI) of the umbilical artery……..

18. Resistance index (RI) of the umbilical artery…….

19.CU (cerebral/ umbilical artery)

ratio……

Late-Term and postterm pregnancy outcome:

20. Normal perinatal outcome yes ( ) No ( )

21. Adverse perinatal outcome yes ( ) No ( )

1. macrosomia	**yes ( )**	**No**	**( )**
2. Cesarean section	**yes ( )**	**No**	**( )**
3. Shoulder dystocia	**yes ( )**	**No**	**( )**
4. Meconium aspiration syndrome	**yes ( )**	**No**	**( )**
5. Oligohydramnios	**yes ( )**	**No**	**( )**
6. Mortality	**yes ( )**	**No**	**( )**
7. Morbidity	**yes ( )**	**No**	**( )**

22. Others……

23. Comments…….

Appendix (C)

Consent to conduct research from Khartoum Bahri Teaching Hospital