Sudan University of Science and Technology

College of Graduate Studies

Characterization of Placenta and Measurement of Umbilical Artery in Third Trimester in Diabetic Women using Ultrasound

وصف المشيمة و قياس الشريان السري في الثلث الأخير من الحمل لدى النساء المصابات بالسكري باستخدام الموجات فوق الصوتية

A thesis Submitted for Partial Fulfillment of the Requirements of M.Sc. Degree in Medical Diagnostic Ultrasound

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الايـة

قال تعالى:

(خَلَقَكُمْ مِنْ نَفْسٍ وَاحِدَةٍ ثُمَّ جَعَلَ مِنْهَا زَوْجَهَا وَأَنْزَلَ لَكُمْ مِنَ الْأَنْعَامِ ثَمَانِيَةَ أَزْوَاجٍ يَخْلُقُكُمْ فِي بُطُونِ أُمَّهَاتِكُمْ خَلْقًا مِنْ بَعْدِ خَلْقٍ فِي ظُلُمَاتٍ ثَلَاثٍ ذَلِكُمُ اللَّهُ رَبُّكُمْ لَهُ الْمُلْكُ لَا إِلَهَ إِلَّا هُوَ فَأَنَّى تُصْرَفُونَ)

صدق الله العظيم

سورة الزمر الاية (6)

Dedication

To;

Soul of my father…

My mother for her continuous support and love…

My sisters & my brothers …

And my friends…

Acknowledgement

I would like to express my appreciation to my **supervisor D. Babiker Abd Elwahab Awad Alla Awad Elseed** for his support, and also my thanks extend to my colleague **Ezzaldeen Geeli** for his help and encouragement. My thanks and gratitude to ultrasound department's staff of ; Saad Abu-Ella Teaching Hospital, Altamayoz A&E Center and Saudi hospital.

Abstract

This was cross sectional study done in diabetic maternal in third trimester of pregnancy, to evaluate the effect of diabetic on placenta and umbilical artery diameter.

The aim of this study was to assess characterization of placenta and measurement of umbilical artery in third trimester in diabetic women using Ultrasound.

The sample size was consisted of 50 pregnant women with DM and 20 normal pregnant women in third trimester; the data was collected by master data sheets in the period from August 2018 to March 2019.

This study was carried out in Ultrasound departments of; Saad Abu-Ella Teaching Hospital, Saudi hospital and Altamayoz A&E Center in Khartoum state.

Data were analyzed by using SPSS (statically package for the social sciences) program and the results were presented in form of graphs and tables.

The study found there was significant difference ($p<0.05$) between the mean of placenta thickness in normal group and diabetic group was 37.80mm and 45.0080mm respectively. And found significant difference ($p<0.001$) between the mean of UA diameter in normal group and diabetic group was 4.1650mm and 17.3880mm respectively.

The study found there was significant Correlation between placenta grading and duration of diabetic ($p<0.05$), also there was significant Correlation between placenta grading and gestational age ($p<0.001$).

The study approved there was significant correlation between diabetic and placental thickness and umbilical artery diameter. Also there was significant correlation of placenta grading with duration of diabetic and gestational age.

For more accurate results, future researches should studies the effect of DM on placenta and umbilical artery diameter in large population size.

Studies the effect of DM on the umbilical artery blood flow with Doppler indices, and should include the outcome of pregnancy to show the correlation between the diabetic pregnancy and fetal growth restrictions.

Instead of studying diabetic pregnancy in third trimester only, data collected in first and second trimester may give more reliable results.

المستخلص

كانت هذه الدراسه مقطعيه عرضيه في الامهات المصابات بالسكري في الثلث الاخير من الحمل , لتقييّم تاثير مرض السكري على المشيمه وقياس قطر الشريان السري.

كان الهدف من هذه الدراسه تقييّم وصف المشيمه وقياس قطر الشريان السري في الثلث الاخير من الحمل لدى النساء المصابات بالسكري باستخدام الموجات فوق الصوتيه.

كان حجم العينه يتضمن 50 من النساء الحوامل المصابات بمرض السكري و 20 من النساء ذوات الحمل الطبيعي وهن في الثلث الاخير من الحمل ،هذه العينات جمعت باستخدام صحائف البيانات في الفترة من اغسطس 2018 حتى مارس 2019 ، اجريت الدراسه في اقسام الموجات فوق الصوتيه في; مستشفى سعد ابوالعلا التعليمي ، المستشفى السعودي ومركز التميز للطوارئ و الاصابات بولاية الخرطوم.

حللت البيانات باستخدام برنامج الحزم الإحصائيه للعلوم الانسانيه في التحليل الإحصائى واخرجت النتائج في شكل رسوم بيانيه و جداول إحصائيه.

وجدت الدراسه اختلاف كبير بين متوسط سمك المشيمه في مجموعة الحمل الطبيعي و مجموعة الحوامل المصابات بالسكري 37.80 مليمتر و 45.0080 مليمتر على التوالي ، و وجدت اختلاف كبير بين متوسط قياس قطر الشريان السري في مجموعة الحمل الطبيعي وبين مجموعة الحوامل المصابات بالسكري 4.1650 مليمتر و 17.3880 مليمتر على التوالي.

وجدت الدراسه علاقه ارتباط مهمة بين درجات تصنيف المشيمه وبين فترة الإصابه بمرض السكري ($p<0.05$) ، ايضا هنالك علاقه ارتباط بين درجات تصنيف المشيمه وبين عمر الجنين ($p<0.001$) .

اثبتت الدراسه علاقه ارتباط مهمه بين مرض السكري وسمك المشيمه وقياس قطر الشريان السري ، وايضا هنالك ارتباط بين درجات تصنيف المشيمه مع فترة الإصابه بمرض السكري و عمر الجنين .

للحصول على نتائج اكثر دقه في البحوث المستقبليه اوصي بدراسه تاثير السكري على المشيمه و قياس قطر الشريان السري في حجم كبير من العينات ، دراسه تاثير السكري على سريان الدم داخل الشريان السري باستخدام موجات الدوبلر، ويجب ان تحتوي على بيانات نتائج الحمل حتى نرى العلاقه بين الحمل لدى النساء المصابات بالسكري وبين معيقات نمو الجنين ، عوضاً عن دراسه الحوامل المصابات بالسكري في الثلث الاخير فقط يجب جمع العينات في الثلث الاول والثاني من الحمل لإعطاء نتائج مؤكده.

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List of Abbreviations

DM	Diabetes Mellitus
GA	Gestational Age
GAD	Gestational Diabetes Mellitus
HCG	Human Chorionic Gonadotropin
PL	Placenta
SPSS	Statistical Package for Social Sciences
TNF	Tumor Necrosis Factor
UA	Umbilical Artery
US	Ultrasound
WKS	Weeks

Chapter One

1.1. Introduction:

The placenta is an organ that connects developing fetus to the uterine wall to allow nutrient uptake, waste elimination and gas exchange via the mothers blood supply. It functions as a fetomaterrnal organ with two components; the fetal placenta, chorion frondosum, which develops from the same blastocyst that forms the fetus, and the maternal placenta, deciduas basalis, which develops from maternal uterine tissue (Pough et al; 2002). The placenta weights around 500 gram, is 220 mm long and has a thickness of 25 - 40 mm. In addition to transport functions, it has metabolic, endocrine and immunological functions. (Enders & Blankenship; 1999).

The placenta connects to the fetus by an umbilical cord which is approximately 55 – 60 cm in length. The cord contains two umbilical arteries and one umbilical vein. The umbilical arteries carry deoxygenated blood from the fetus to the placenta. They are the only arteries in the human body, aside from the pulmonary arteries, that carry deoxygenated blood (Kiserud et al; 2004).

The integrity and normal function of the placenta and umbilical vessels are essential for the wellbeing of the growing fetus (Kellow et al; 2011).

The role of ultrasonography is even more essential during caring for pregnant women with medical problems like diabetes mellitus (DM). (Maulik et al; 2002).

In pregnancies complicated by DM, impairment of placenta perfusion may lead to growth retardation of the fetus. Doppler ultrasound helps in evaluating fetoplacental blood flow. This is an important way of assessing high risk pregnancy caused by DM (Maulik et al; 2002).

In this study, the researcher measured the placental thickness to assess the effect of diabetic in placenta thickness using ultrasonography.

1.2. Problem of the study:

DM affects fetal wellbeing by inducing structural changes in the placenta, and also can cause changes in placenta thickness. So U/S can determine the normal verses abnormal placenta.

This study is designed to find change of placenta thickness and measurement of umbilical artery diameter in diabetic women.

1.3. Objectives of the study:

1.3.1. General objective:

To characterize placental and measurement of umbilical artery in third trimester in diabetic women using ultrasound.

1.3.2. Specific objectives:

1- To measure the placenta thickness using ultrasound.

2- To assess the placenta grading.

3- To measure UA diameter to show the effect of DM on it.

4- To correlate between maternal age, BMI, occupation, duration of disease, placenta thickness and umbilical artery diameter.

1.4. Thesis layout:

This study consisted of five chapters, Chapter one: introduction (problem and objective of study), Chapter two: literature review (Anatomy, physiology and previous studies), Chapter three: research methodology, Chapter four: the results and Chapter five includes; discussion, conclusion and recommendations.

Chapter Two
Literature Reviews and Previous Study

2.1. Literature Reviews:

2.1.1. Anatomy and physiology of the placenta:

The placenta is a highly specialized organ of pregnancy that supports the normal growth and development of the fetus. It separates the fetal and maternal circulations and transfers oxygen, carbon dioxin, nutrients and waste products between the maternal and the fetus. In addition to transport functions, the placenta has metabolic, endocrine and immunological functions (Enders& Blankenship; 1999).

The exchange between the maternal and fetal circulation takes place in the chorionic villus, which consist of central fetal capillary, stroma and an outer trophoblast layer. In addition to the trophoblast layer, the fetal and maternal circulations are separated by the trophoblastic basement membrane, connective tissue space, endothelial. Trophoblastic cells are present as monocular cells called cytotrophoblasts and multinucleate cells called syncytiotrophoblasts (Ender & Blankenship; 1999).

Growth and function of the placenta are precisely regulated and coordinated to ensure the exchange of nutrients and waste products between the maternal and fetal circulatory systems at maximal efficiency (Gude et al; 2004).

The main functional units of the placenta are the chorionic villi within which fetal blood is separated by only three or four layers (placental membrane) from maternal blood in the surrounding intervillous space. After implantation; trophoblast cells proliferate and differentiate along two pathways described as villous and extra villous. Non-migratory, villous cytotrophoblast cells fuse to form the multinucleated syncytiotrophoblast, which forms the outer epithelial layer of the chorionic villi. It is at the terminal branches of the chorionic villi that the majority

of fetal/maternal exchange occurs. Extra villous trophoblast cells migrate into the decidua and remodel uterine arteries. This facilitates blood flow to the placenta via dilated, compliant vessels, unresponsive to maternal vasomotor control. The placenta acts to provide oxygen and nutrients to fetus, whilst removing carbon dioxide and other waste products (Gude et al; 2004).

It metaolizes a number of substances and can release metaolic products into mmaternal and/or fetal circulations. The placenta can help protect the fetus against certain xenobiotic molecules, infections and maternal diseases. In addition, it releases hormones into both the maternal and fetal circulations to affect pregnancy, metabolism, fetal growth, parturition and other functions. The main hormones produced by the placenta include oestrogens and progesterone and human chorionic gonadotropin (HCG) produced by embryonic tissue right from the time of implantation. This promptly protects the embryo from rejection, by acting on the ovaries, causing them to sustain the hormone production that supports pregnancy. The presence of HCG also acts as the basis of pregnancy testing. After the third month, hormone production by the placenta takes over the pregnancy –supporting role from the ovary, by virtue of progressively increasing secretion of oestrogens and progesterone.

Many placental functional changes occur that accommodate the increasing metabolic demands of the developing fetus throughout gestation (John & Fox; 1991).

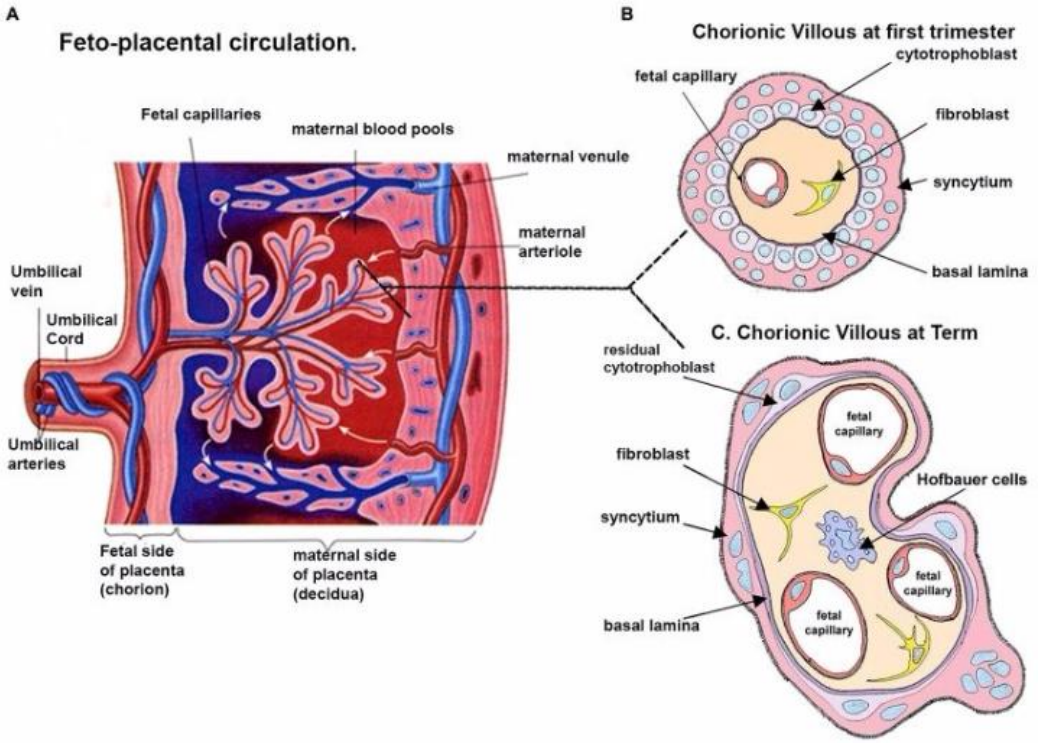

Figure (2.1) Schematic representation of a human placenta.

(A) Fetal placental circulation.

(B) Chorionic villous the presence of syncytiotrophoblast, a layer of cytotrophoblast cells, connective tissue of the villuscontaining fibroblasts and the fetal capillaries.

(C) At term, in some areas the placental membrane is so thin such that the syncytiotrophoblast comes into direct contact with the fetal capillary endothelium, and is thus called the vasculo-syncytial membrane. (https://www.researchgate.net; 2018)

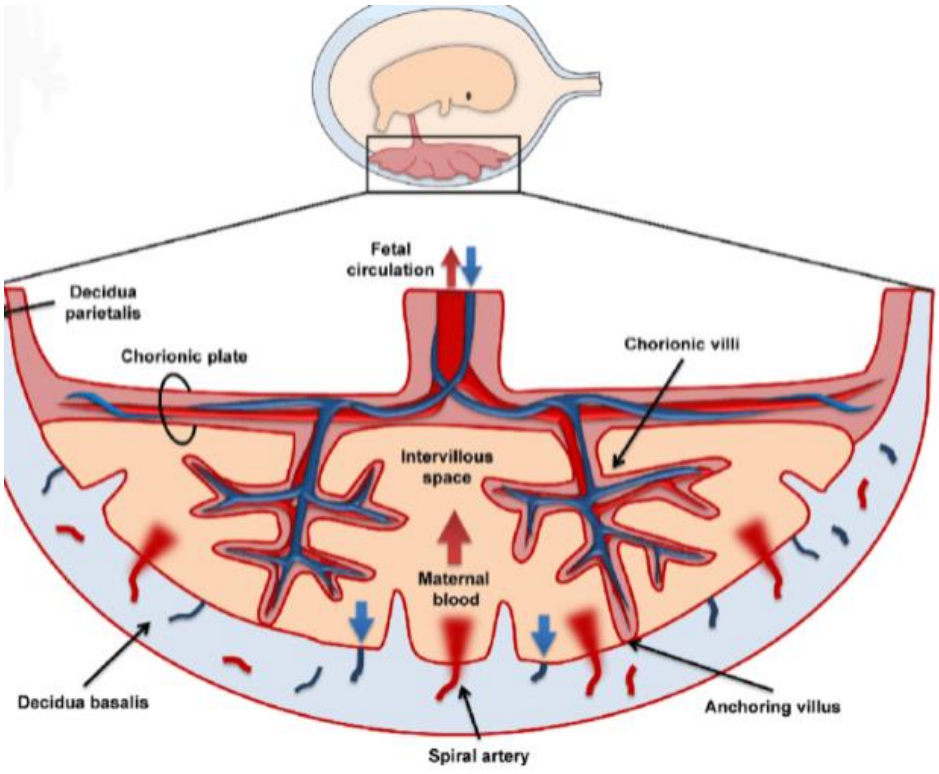

Figure (2.2) the fully developed human placenta.

(https://www.researchgate.net/;2018)

2.1.2. Placental grading:

The placenta has grades from 0 to 3 with different features in each grade.

2.1.2.1. Grade 0:

The chorionic plate appears as a smooth straight and well-defined unbroken echogenic line. The placental substance has a homogeneous texture without any foci of increased echogenicity. No focal areas of increased echogenicity are seen in the basal layer.

These changes are seen in the first and second trimester.

2.1.2.2. Grade 1:

Chorionic plate appears as a well-defined unbroken echogenic line with some subtle indentation. A few scattered foci of increased echogenicity parallel to the basal layer may in the placental substance. No density is seen in the basal layer.

A grade 1 placenta may first be seen from 30 to 32 weeks and may persist until term.

2.1.2.3. Grade 2:

The chorionic plate develops more marked indentations. The placental substance is incompletely divided by linear or comma –shaped echogenic densities contiguous with the chorionic plate. There is an interval increase in the size and number of the echogenic foci in the placental substance compared to grade 1, the basal layer also contains linear echoes larger and more echogenic than the placental substance.

The axis of these echoes is also parallel to basal layer.

2.1.2.4. Grade 3:

The chorionic plate appears interrupted by echogenic indentations extending up to the basal layer. The placental substance is divided by these septa and may contain anechoic of hypo echoic regions. Additional dense irregular echogenic areas with acoustic shadowing may appear near the chorionic plate.

Echogenic foci in the basal layer may be larger more dense, and. A grade 3 placenta is usually seen in the third trimester near term.

2.1.3. Anatomy and physiology of the Umbilical cord:

The umbilical cord connects the developing embryo or fetus to the placenta. It develops from the connecting stalk. It consists of mesodermal connective tissue called Wharton’s jelly covered by amnion. The cord is inserted in the fetal surface of the placenta near the centre, “eccentric insertion” in 70% or at the centre, “central insertion” in 30% within the umbilical cord there are two umbilical

arteries and one umbilical vein. Inside the placenta, the umbilical arteries connect with each other at a distance of approximately 5mm from the cord insertion in what is called the Hyrtl anastomosis. Subsequently, they branch into chorionic arteries or intra placental fetal arteries. The umbilical arteries carry deoxygenated blood from the fetus to the placenta. The pressure inside UA is approximately 50 mmHg (Gordon et al; 2007).

2.1.4. Fetal circulatory system:

The umbilical cord enters the fetus via the abdomen, at the point which will become the umbilicus (navel) after separation. Within the fetus, the umbilical vein continues towards the transverse fissure of the liver, where it splits in to two.

One of these branches joints with the hepatic portal vein (connecting to its left branch), which carries blood in to the liver. The second branch (known as the ductus venosus) bypasses the liver and flows into the inferior vena cava; which carries blood towards the heart. The two umbilical arteries branch from the internal iliac arteries and pass on either side of the urinary bladder into the umbilical cord completing the circuit back to the placenta. Within the child the umbilical vein and ductus venosus close up, and degenerate into fibrous remnants known as the round ligament of the liver and the ligamentum venosum respectively. Part of each umbilical artery closes up, degenerating into medial umbilical ligament, while the remaining sections are retained as part of the circulatory system (Kiserud et al; 2004).

2.1.5. Placenta and Diabetes:

The placenta is a complex fetal organ that fulfills pleotropic roles during fetal growth. It separates the maternal and fetal circulation, with which it is in contact through different surfaces, i.e. the syncytiotrophoblast exposes the placenta to the maternal circulation and the endothelium is in contact with fetal blood. Because of this unique position, the placenta is exposed to the regulatory influence of

hormones, cytokines, growth factors, and substrates present in both circulations and hence, may be affected by changes in any of these. In turn it can produce molecules that will affect mother and fetus independently (Enders et al; 2003).

The human placenta expresses virtually all known cytokines including tumor necrosis factor (TNF)-α, resistin, and lepthin, which are also produced by the adipose cells. The discovery that some of these adipokines are key players in the regulation of insulin action suggests possible novel interactions between the placenta and adipose tissue in understanding pregnancy-included insulin resistance. The interplay between the two systems becomes more evident in gestational diabetes mellitus (GDM) (Tansey et al; 2000).

Placental development is characterized by three distinct periods. At the beginning of gestation, a series of critical proliferation and differentiation processes predominantly of the trophoblast eventually lead to the formation of villous and extra villous structures. The latter anchor the placenta in the uterus and remodel the uterine spiral arteries into low resistance vessels. Then the newly formed villi differentiate through various steps of maturation. The end of gestation is associated with placental mass expansion i.e. villous growth. During the first half of gestation, the trophoblast is the key tissue that undergoes the most profound alterations, whereas extensive angiogenesis and vascularization occur in the second half of gestation, i.e. the endothelium is the site of the more prominent processes, although there is overlap. This period is also accompanied by extensive vascular remodeling and stabilization of the vascular bed (Kellow et al; 2000).

The diabetic environment can be regarded as a network of substances (hormones, nutrients and cytokines) with altered concentrations. The current view is that the abnormal maternal metabolic environment may stimuli within the adipose tissue and the placental cells resulting in the increased production of inflammatory cytokines whose expression is minimal under normal pregnancy. One leading

hypothesis is that changes in circulating TNF-α, adiponectin, leptin and resistin link inflammation to metabolic changes by enhancing insulin resistance in the mother. Likewise, the fetal environment is also changed in diabetes and elevated levels of insulin, leptin and other cytokines have been well documented.

2.1.6. Normal Sonographic Appearance of Placenta and Umbilical Cord:

The placenta may be homogeneous or may have indentation or echogenic foci along the basal plate (American College of Radiology 1995). Echogenic septa extending across the width of the placenta may be seen in the stage of the placenta. Umbilical cord which contains two umbilical arteries and one umbilical vein appear as shown below.

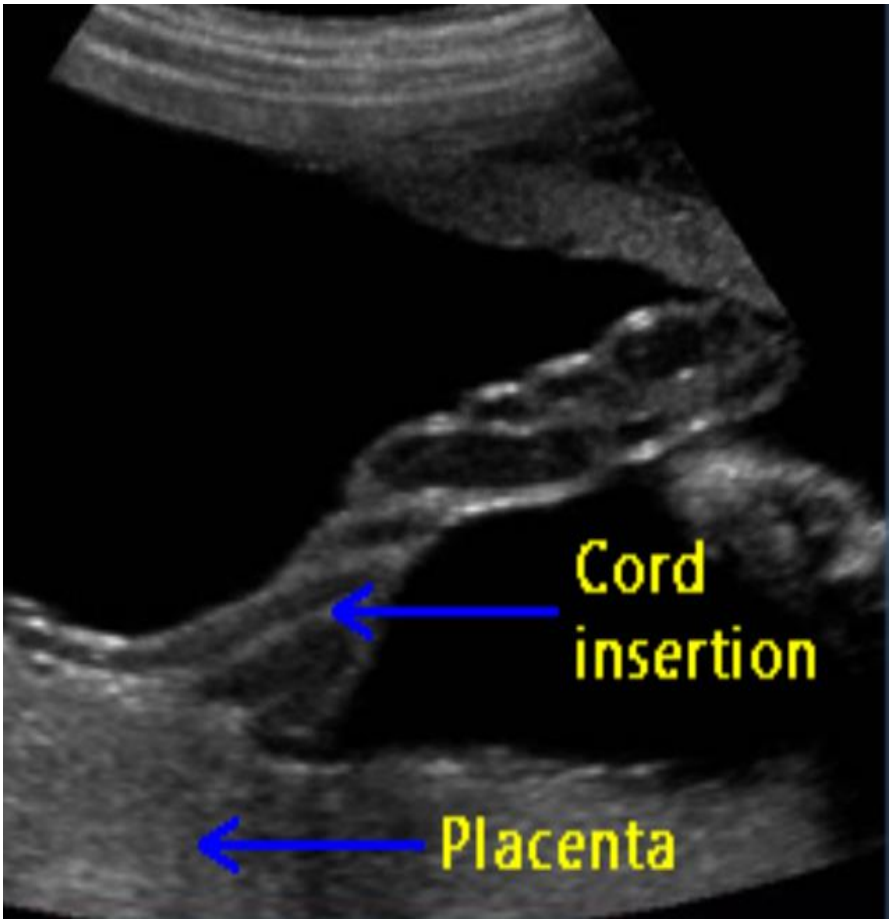

Figure (2.3): Sonographic appearance of a normal umbilical cord & placenta, tranceabdominal scan.(https://www.obimages.net; 2018)

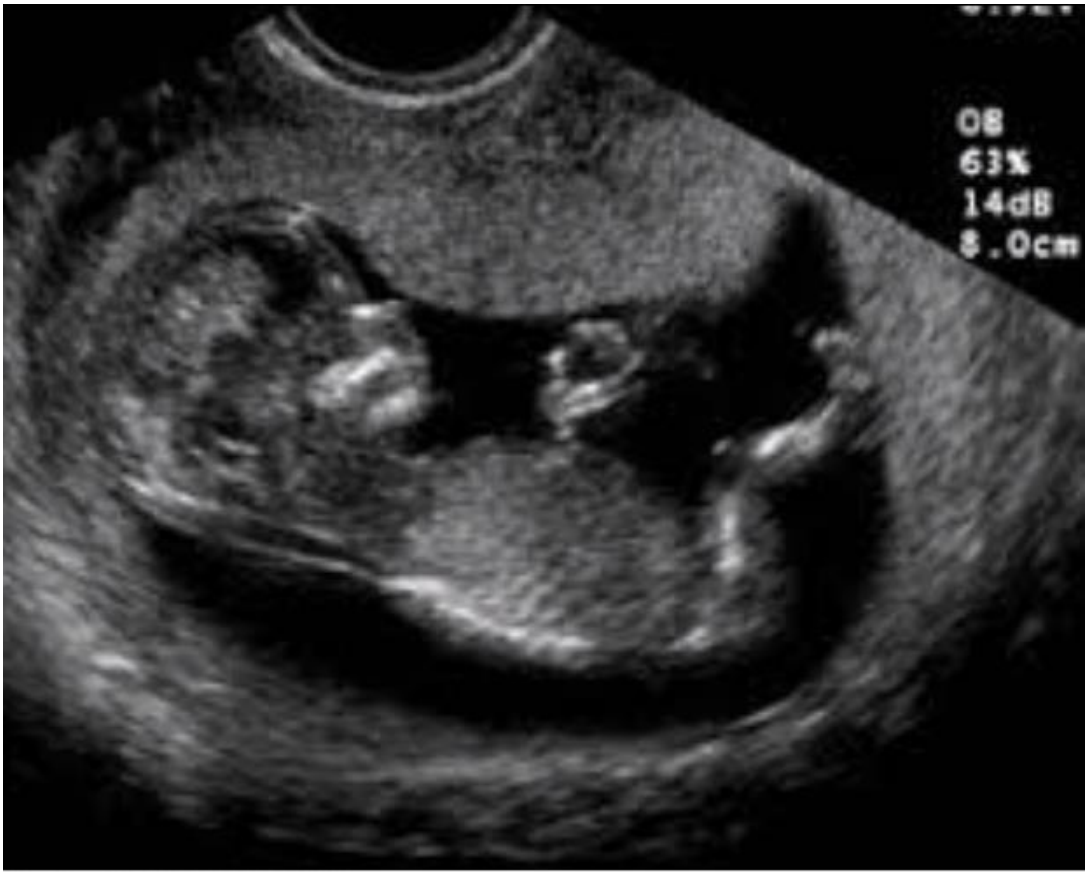

Figure (2.4): Sonographic appearance of fetus and placenta. (Author Source; 2018)

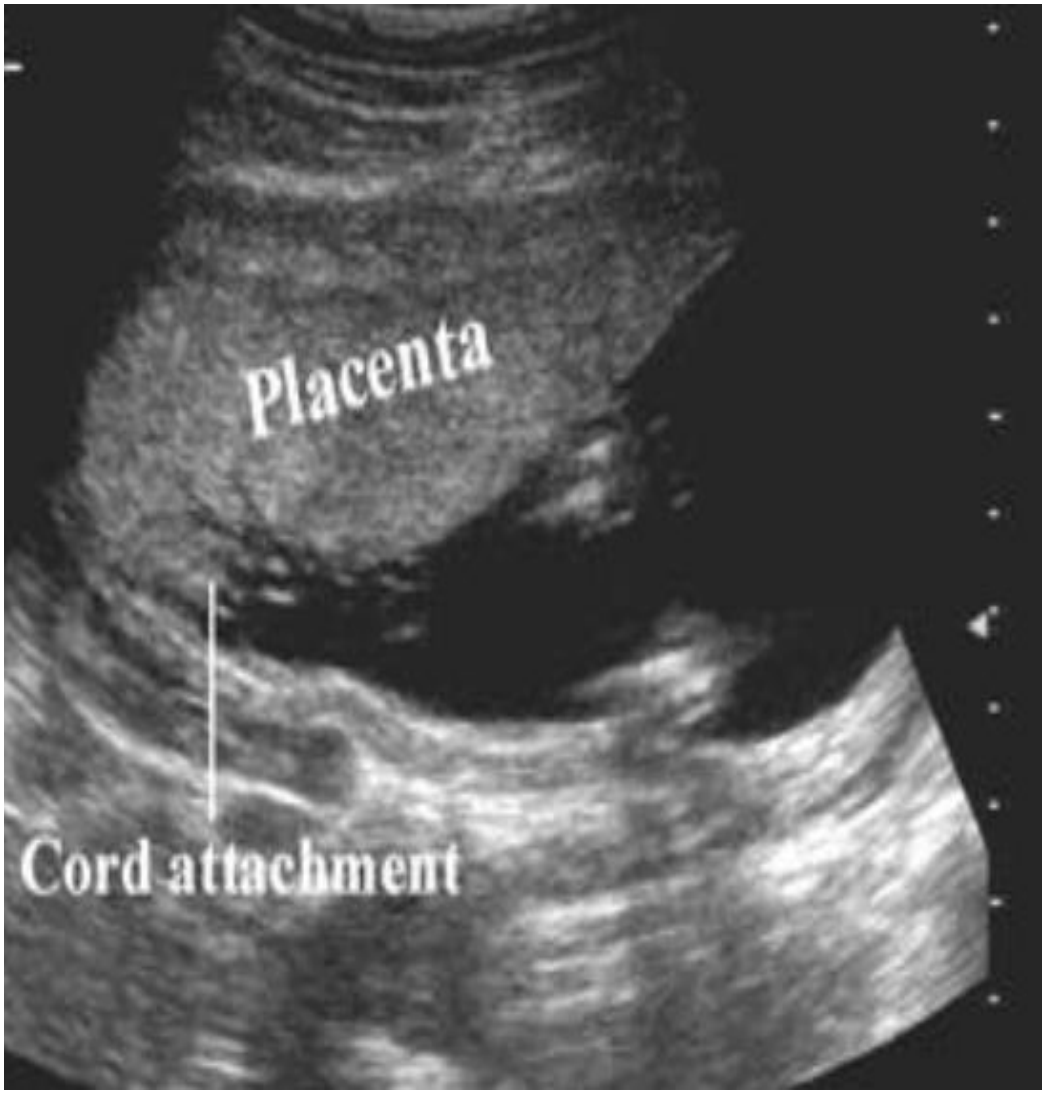

Figure (2.5): Sonographic appearance of placenta and umbilical cord attachment. (http://www.fetalultrasound.com; 2018)

2.6. Previous Studies:

In 1986 Singh studied the structural changes in the umbilical cord of full term fetus from 18 cases of gestational diabetes. All these were insulin-dependent and their blood glucose levels were well controlled. Light microscopy showed rupture and erosion of the endothelial lining of umbilical arteries resulting in increased permeability and hemorrhages. The umbilical vein was unduly dilated. The smooth muscle in the walls of these vessels showed disruption and degeneration of its fibers. “Wharton’s jelly” showed alteration in the pattern of distribution of its fibers with large empty spaces amongst them. It is suggested that gestational diabetes has a deleterious effect on umbilical vessels and the connective tissue component of “Wharton’s jelly”.

Muhammed Ashfaq et al 2005 studied a total of 60 full term placenta, 20 from normal group A, 20 from gestational diabetics group B and chronic hypertensive mothers group C. Shape, weight, thickness and diameter were significantly greater in diabetic group as compared to normal and hypertensive group thickness in group A=2.15+_0.16, group B=2.72+_0.16 and group C=3.93+_0.18. Hypertensive group shows no significant decrease in weight of placenta while there was no change in thickness and diameter of placenta in hypertensive than the normal group. On the basis of results of present study, it is concluded that diabetic’s placenta showed increase in weight, thickness and diameter. Hypertensive placenta showed no significant change in weight, shape thickness when compared with normal group. The ultrasound placenta images are classified into normal placenta and placenta complicated by gestational diabetes. Initially the ultrasound placenta images are marked as normal or placenta complicated by gestational DM by the experts in the medical field. This is used to confirm the classification accuracy.

The values shows that there is a significant increase in thickness and BPD in the case of placenta complicated by diabetes and also in case of intra uterine growth retardation which can be suspected as a cause of GDM. The earlier screening for DM in pregnancy influence on placental development, which indirectly accounts for fetal growth and metabolism. This study attempted to find the feasibility for classifying the ultrasound images of placenta with complicating diabetes by measuring the placenta thickness and BPD from the ultrasound placenta images. It is found that the thickness of placenta plays a major role in the classifying the ultrasound placenta images (G. Malathi et al; 2010).

Overall mean placental thickness was 37.5mm. The cutoff value of thick placenta was 47mm, and thin placenta was 29mm. Gestational diabetes mellitus was significantly more common in the thick placenta group than control group (7.3% vs. 4.5%, P=0.018). Incidence of preterm birth was 8.0% in the thick placenta group, 4.8% in the thin placenta group and 3.9% in the control group (P=0.001, P=0.357, respectively). Mean birth weight was 3247gm in the thick placenta group, 3186gm in the thin placenta group and 3269gm in the control group (P=0.331, P<0.001). Incidence of small for gestational age below 10th percentile was 11.3% in the thick placenta group, 13.4% in the thin placenta group and 8.8% in the control group (P=0.081, 0.007). Birth weight above 4000gm was 5.6%, 2.0% and 4.1%, respectively (P=0.089, P=0.007). Gestational diabetes mellitus and preterm birth were significantly more common in pregnant women with thick placenta. Thin placentas were significantly associated with a decreased newborn weight (D.Kwak et al; 2014).

Chapter Three
Materials and Methods

3.1. Materials:

3.1.1. Population of the study:

All pregnant women who are referred to the Ultrasound departments of; Saad Abu-Ella Teaching Hospital, Altamayoz A&E Center and Saudi hospital for a routine follow up are participating in this study. During the period from August 2018 to March 2019.

3.1.2. Study Sample:

The sample size consisted of 50 pregnant women with DM and control data 20 normal pregnant women in third trimester.

3.1.3. Inclusion criteria:

All diabetic pregnant women in third trimester

3.1.4. Exclusion criteria:

-Diabetic pregnant women in the first and second trimester.

-Normal pregnant women in the first and second trimester.

-Diabetic pregnant women in the third trimester with hypertension.

3.1.5. Equipment:

The equipment requirements included a 3D ultrasound machines (SAMSUNG& ECUBE).

Figure 3-1 SAMSUNG ultrasound machine (Author source; 2018).

Figure 3-2 ECUBE ultrasound machine (Author source; 2018).

3.2. Methods:

This is an experimental study deals with diabetic pregnant women in third trimester, the pregnant could be insulin dependent or non-insulin dependent.

3.2.1. Technique:

The pregnant women came to the Ultrasound Department for obstetric ultrasound. The complaints and history were recorded.

Trans abdominal scan through the lower abdomen with pregnant in the supine position. A small amount of gel was applied to the skin. The probe was then held in sagittal position. The placental thickness could be measured at the insertion of umbilical cord or near the mid portion or center of the placenta with the caliper placed at the aminochorionic surface (chorionic plate) and the second caliber on the basal surface perpendicular to the chorionic plate. In this study we measured the thickness from the center of the placenta. Then we used color flow imaging (Doppler) to identify the umbilical cord at the placental insertion, which is the preferred site for measuring the umbilical artery diameter.

3.2.2. Data Collection:

The data was collected by master data sheets using the variables of maternal age, BMI, number of pregnancy, occupation, diabetic duration, management (insulin dependent or not), GA, placental grade, placental thickness and umbilical artery diameter.

3.2.3. Data Analysis:

Data were analyzed by using SPSS program and the results were presented in form of graphs and tables.

3.2.4. Ethical Considerations:

-No identification or individual details were published.

-No information or patient details will be disclosed or used for reasons other than this study.

Chapter Four

Results

Results

Table (4.1) Shows PL Thickness values (mm) in DM and Control (normal).

Cases	No. Patients	Minimum (mm)	Maximum (mm)	Mean (mm)	Std. Deviation (mm)
DM	50	28	99.80	45.0080	13.90228
Control	20	24	66	37.80	11.71239

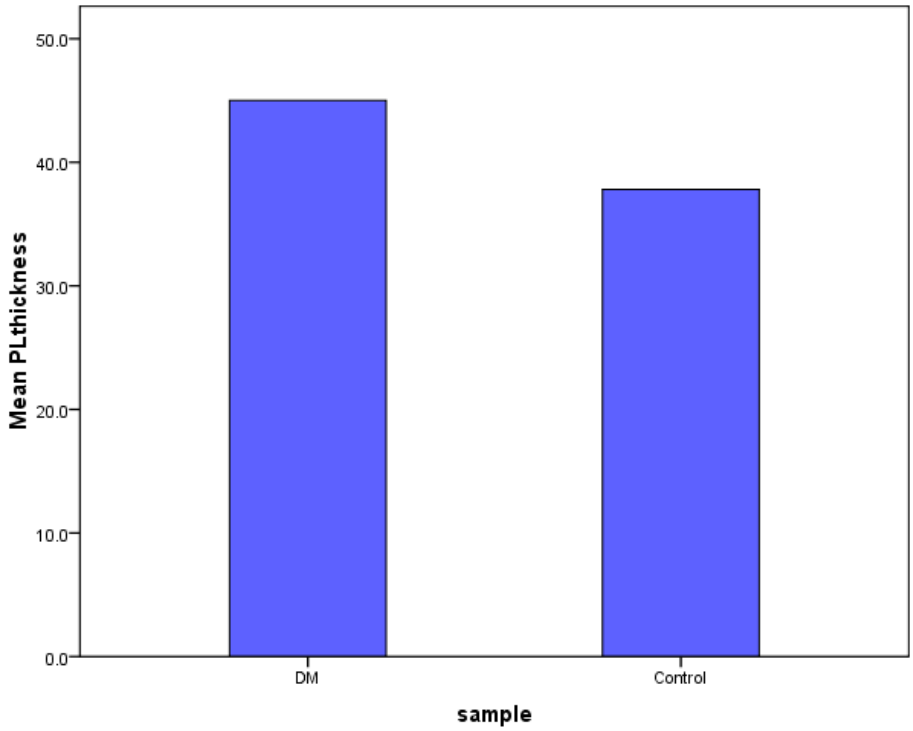

Figure (4.1) bar graph shows PL Thickness values (mm) in DM and Control (normal)

Table (4.2) Shows UA diameter values (mm) in DM and Control (normal).

Cases	No. Patients	Minimum (mm)	Maximum (mm)	Mean (mm)	Std. Deviation (mm)
DM	50	2.50	41.00	17.3880	6.23246
Control	20	2.60	9.60	4.1650	1.56987

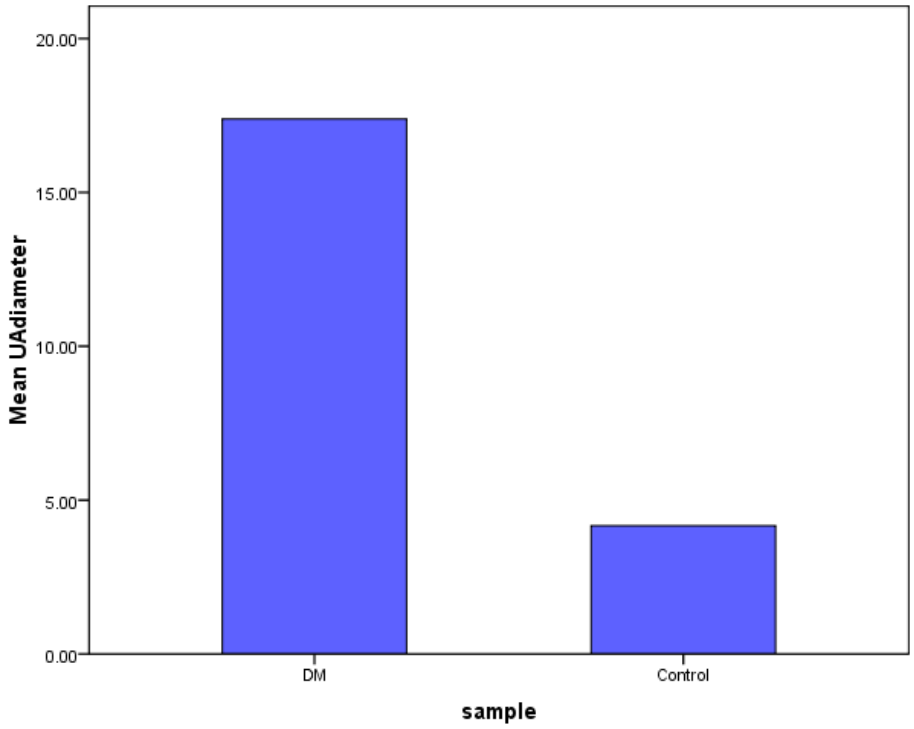

Figure (4.2) bar graph shows UA diameter values (mm) in DM and Control

Table (4.3) shows correlation between PL thickness (mm) and variables in diabetic pregnant women.

variables	No.	Mean(mm)	Std. Deviation	Std. Error Mean
Non-insulin dependant	29	41.779	9.4104	1.7475
Insulin-dependant	21	49.467	17.7074	3.8641
house wife	31	47.6581	16.15691	2.90187
worker	19	40.6842	7.69598	1.76558

Table (4.4) shows correlation between PL thickness and variables in diabetic pregnant women

Variables	Sign. value
Age	.084
BMI	.718
No. of pregnancy	.003
GA	.091
Diabetic duration	.468

The positive significantly high as the p-value is less than 0.05.
* Correlation is significant at 0.003 (No. of pregnancy).

Table (4.5) shows correlation between UA diameter (mm) and variables in diabetic pregnant women.

Variables	No.	Mean(mm)	Std. Deviation	Std. Error Mean
Non-insulin dependant	29	17.4828	7.45520	1.38440
Insulin-dependant	21	17.2571	4.16216	.90826
House wife	31	17.0290	7.21476	1.29581
worker	19	17.9737	4.28976	.98414

Table (4.6) shows correlation between UA diameter and variables in diabetic pregnant women.

Variables	Sig. value
age	.504
BMI	.053
No. of pregnancy	.060
GA	.974
Diabetic duration	.801

Table (4.7) shows placenta grade Percentage at third trimester in DM

PL grade	Frequency	Percentage
1	2	4%
2	31	62%
3	17	34%
Total	50	100%

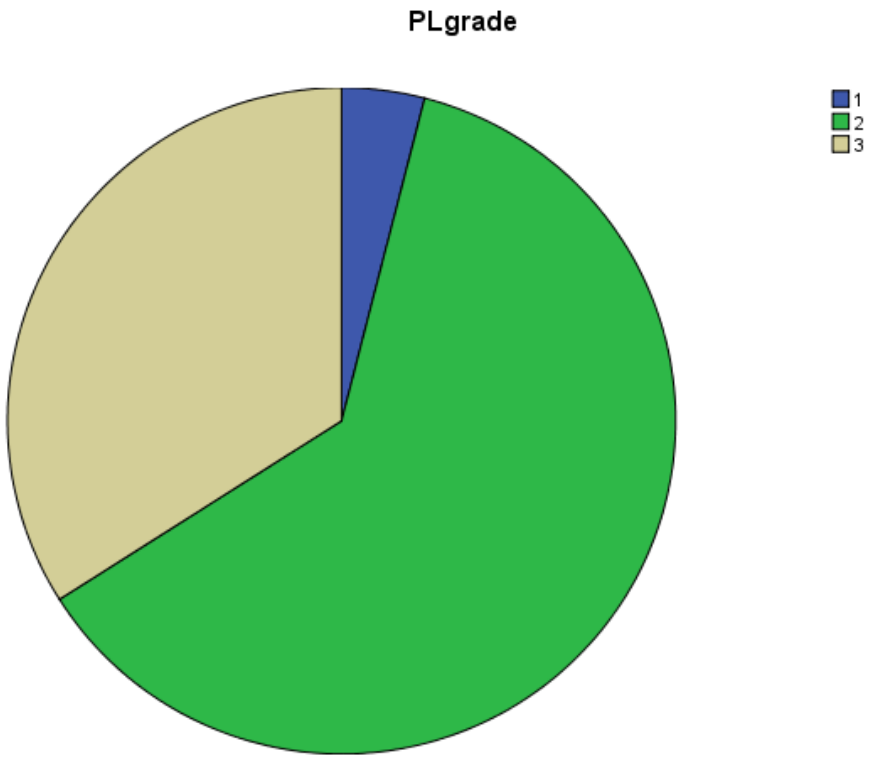

Figure (4.3) pie graph shows PL grade in third trimester (DM)

Table (4.8) shows percentage of means of PL thickness (mm) and UA diameter (mm) in different GA at third trimester in DM.

GA(w/k)	Frequent	Mean of PL(DM)	Mean of UA(DM)	Percentage
27-31	18	41.11mm	16.8mm	36%
32-36	24	49.35mm	17.95mm	48%
37-40	8	40.75mm	16.93mm	16%

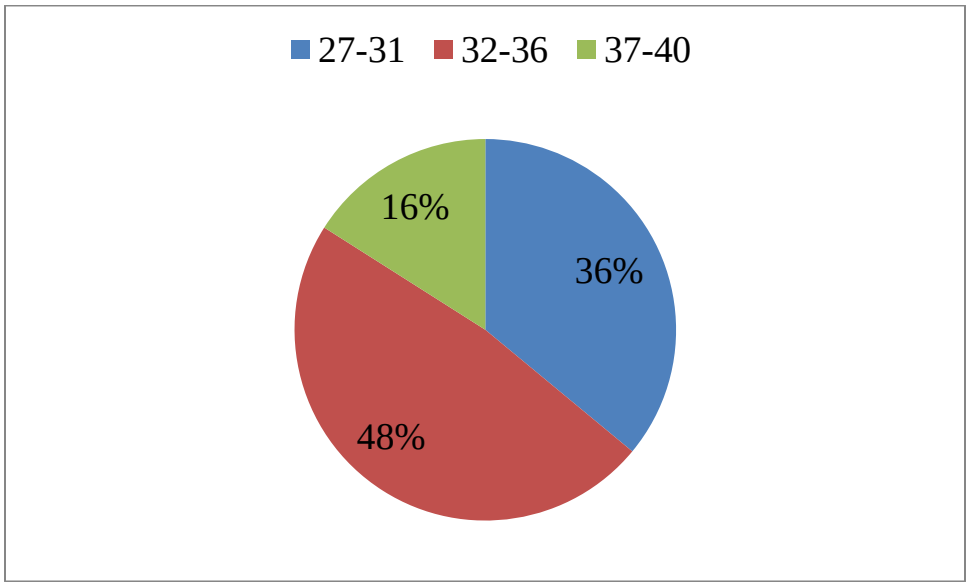

Figure (4.4) pie graph shows percentage of means of PL thickness (mm) and UA diameter (mm) in different GA at third trimester in DM.

Table (4.9) shows correlation between PL thickness (mm) and occupation in control group.

Occupation	No.	Mean(mm)	Std. Deviation	Std. Error Mean
Housewife	18	38.5889	12.02306	2.83386
Worker	2	30.7000	6.08112	4.30000

Table (4.10) shows correlation between UA diameter and occupation in control group.

Occupation	No.	Mean(mm)	Std. Deviation	Std. Error Mean
House wife	18	4.1833	1.65858	.39093
worker	2	4.0000	.00000	.00000

Table (4.11) shows correlation between PL thickness and Variables in control group.

Variables	Sig. value
age	.740
BMI	.624
No. of pregnancy	.662
GA	.958

Table (4.12) shows correlation between UA diameter and Variables in control group.

Variables	Sig. value
age	.385
BMI	.704
No. of pregnancy	.510
GA	.271

Table (4.13) Shows PL grade Percentage at third trimester in normal pregnancy in control group.

PL grade	Frequency	Percentage
1	6	30%
2	10	50%
3	4	20%
Total	20	100%

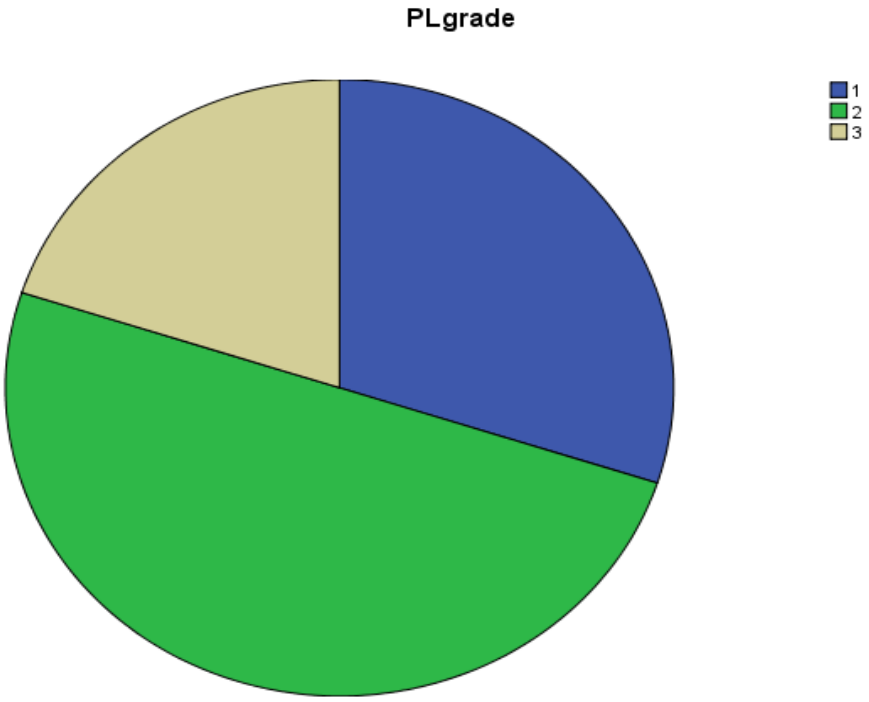

Figure (4.5) pie graph shows PL grade at third trimester in normal

Table (4.14) shows percentage of means of PL thickness (mm) and UA diameter (mm) in different GA at third trimester in normal (control group).

GA(w/k)	Frequent	Mean of PL	Mean of UA	Percentage
27-31	6	35.42mm	4.3mms	30%
32-36	9	39.44mm	4.1mm	45%
37-40	5	37.7mm	4.12mm	25%

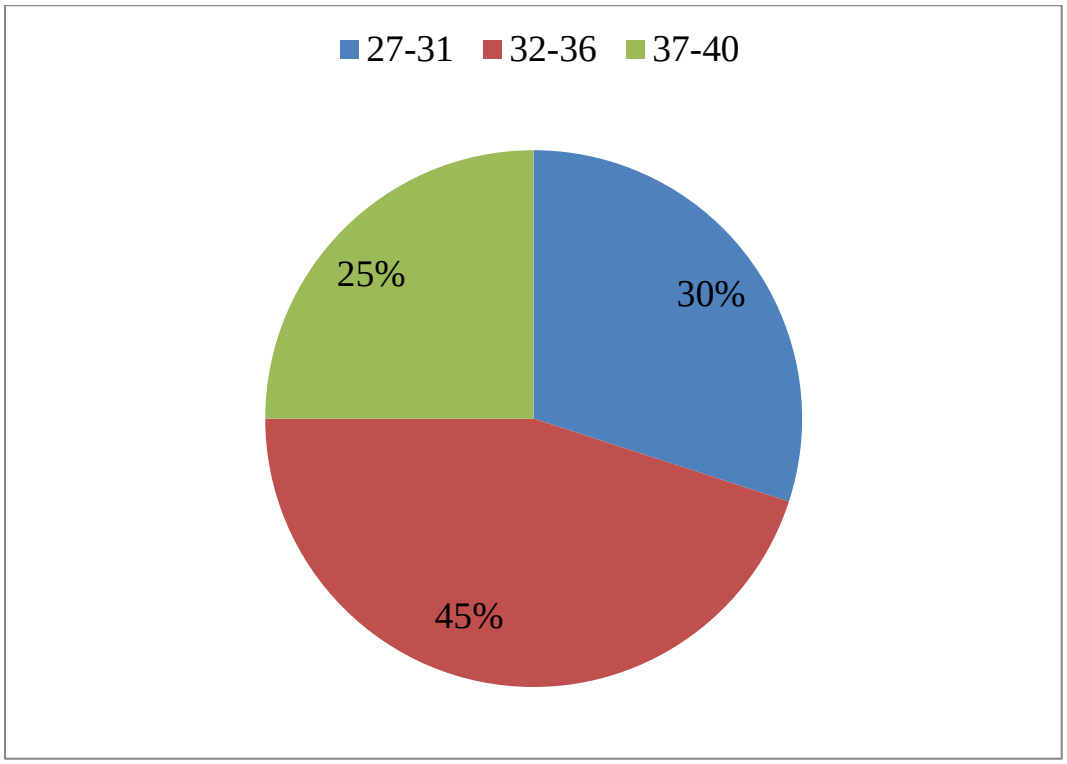

Figure (4.6) pie graph shows percentage of means of PL thickness and UA diameter in different GA at third trimester in normal.

Table 4.15: t-test

a- t-test Group Statistics

Group Statistics					
	status	N	Mean	Std. Deviation	Std. Error Mean
BMI	DM	50	26.0566	2.59956	.36763
	Non -diabetic (control group)	20	23.9510	2.54311	.56866
GA(wks)	DM	50	32.98	3.020	.427
	Non -diabetic (control group)	20	33.20	3.302	.738
PL Thickness(mm)	DM	50	45.008	13.9023	1.9661
	Non -diabetic (control group)	20	37.800	11.7124	2.6190
UA diameter(mm)	DM	50	17.39	6.232	.881
	Non -diabetic (control group)	20	4.16	1.570	.351

b- t-test for Equality of Means

t-test for Equality of Means							
	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
						Lower	Upper
Age(yrs)	4.478	68	.000	7.020	1.568	3.891	10.149
	4.457	34.737	.000	7.020	1.575	3.822	10.218
BMI	3.080	68	.003	2.10560	.68364	.74142	3.46978
	3.110	35.778	.004	2.10560	.67714	.73199	3.47921
GA(ws)	-.268-	68	.789	-.220-	.821	-1.858-	1.418
	-.258-	32.434	.798	-.220-	.853	-1.957-	1.517
PL Thickness(mm)	2.044	68	.045	7.2080	3.5259	.1722	14.2438
	2.201	41.357	.033	7.2080	3.2748	.5961	13.8199
UA diameter(mm)	9.333	68	.000	13.223	1.417	10.396	16.050
	13.938	61.769	.000	13.223	.949	11.326	15.120

Table 4.16: Correlation

Correlations

		Diabetic duration(yrs)	GA(ws)	PL Grade	PL Thickness(mm)	UA diameter(mm)
Diabetic duration(yrs)	Pearson Correlation	1	.224	.313*	.105	.037
	Sig. (2-tailed)		.118	.027	.468	.801
	N	50	50	50	50	50
GA(ws)	Pearson Correlation	.224	1	.451**	.242	-.005-
	Sig. (2-tailed)	.118		.001	.090	.974
	N	50	50	50	50	50
PL Grade	Pearson Correlation	.313*	.451**	1	.207	.103
	Sig. (2-tailed)	.027	.001		.148	.475
	N	50	50	50	50	50
PL Thickness(mm)	Pearson Correlation	.105	.242	.207	1	-.166-
	Sig. (2-tailed)	.468	.090	.148		.250
	N	50	50	50	50	50
UA diameter(mm)	Pearson Correlation	.037	-.005-	.103	-.166-	1
	Sig. (2-tailed)	.801	.974	.475	.250	
	N	50	50	50	50	50

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

Chapter Five
Discussion, conclusion and recommendations

5.1 Discussion

The main objective of this study was to evaluate sonographically the effect of diabetes on placenta thickness and umbilical artery diameter.

The study found that there was significant difference between the mean placenta thickness in normal group and diabetic group was 37.80mm and 45.0080mm respectively, ($p<0.05$) this result agree as shown in table (4-1).

Table (4.2) shown there was significant difference between the mean of umbilical artery diameter in normal group and diabetic group was 4.1650mm and 17.3880mm respectively ($p<0.001$).

Compared the mean of PL thickness in diabetic pregnant whose depend on insulin (21 women) and non-insulin dependent (29 women) they measured 49.467mm and 41.779mm and the UA diameter measured 17.2571mm and 17.4828mm respectively that means the PL thickness and UA diameter increased in women whose treated by insulin as shown in table (4-3).

According to the occupation in this study there are 19 workers and 31 house wives the PL thickness of them is 40.6842mm and 47.6581mm that mean it increased in house wives also in the control group it increased in house wives (in workers30.7mm and 38.5889mm in house wives). And the UA diameter in DM grouped measured 17.9737mm and 17.0290mm respectively which mean it increased in workers and there were minimal different in it on the control grouped(0.1mm).

Table (4.4) shows correlation between PL thickness and variables (age, BMI, NO. of pregnancy and duration of diabetic) in all diabetic pregnant women, we found the NO. Of pregnancy was correlated significantly with mean PL thickness (0.003) which less than 0.05 and it not significant in UA diameter.

Table (4.7) and table (4.13) shows the PL grade Percentage at the third trimester, we found most of them in grade 2 (62% in DM grouped and 50% in control grouped). There was correlation between placenta grading and duration of diabetic ($p<0.05$), also there was correlation between gestational age and placenta grading ($p<0,001$).

Table (4.8) shows different GA at third trimester with the mean of PL thickness. When compared mean of PL thickness in range of GA from 32 to 36 which it was the biggest percentage on the DM group (48%) it measured 49.35mm with the same range of GA on the control group (45%) the mean PL thickness measured 39.44mm I found increased of PL thickness in DM group. UA diameter in same range of GA and same percentage measured 17.95mm (in DM group) and 4.1mm (in control group) also it increased in DM group.

Muhammed Ashfaq et al 2005 studied a total of 60 full term placenta, 20 from normal group A, 20 from gestational diabetics group B and chronic. They found that there was significant change in thickness in diabetic group compared with normal and hypertensive groups. The mean thickness of the placenta in these groups were as follows; 2.15+_0.16cm in group A, 2.72+_0.16cm in group B and 3.98+_0.18cm in group C at $p<0.001$. This result agreement with our study where the difference between the mean PL thickness in the normal group and diabetic group was 37.80mm and 45.0080 mm respectively.

5.2 Conclusion

The main objective of this study was to determined changes in placenta thickness and umbilical artery diameter in diabetic pregnant women in the third trimester. Our results showed that there was a significant correlation between the diabetic and placenta thickness and UA diameter.

The placenta thickness increased in house wives who were treated by insulin more than non-insulin dependent or workers.

The UA diameter increased in workers women and insulin dependent more than house wives and non-insulin dependent.

There was significant correlation between placenta grading and duration of diabetic, also between the gestational age and placenta grading.

5.3 Recommendations

For more accurate results, future researches should studies the effect of DM on placenta thickness and umbilical artery diameter in large population size.

Studies the effect of DM on the umbilical artery blood flow with Doppler indices.

Such studies should include the outcome of pregnancy to show the correlation between the diabetic pregnancy and fetal growth restrictions.

Instead of studying diabetic in third trimester only, data collected in first and second trimester may give more reliable results.

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Appendices

Appendix A

U/S images for study sampling

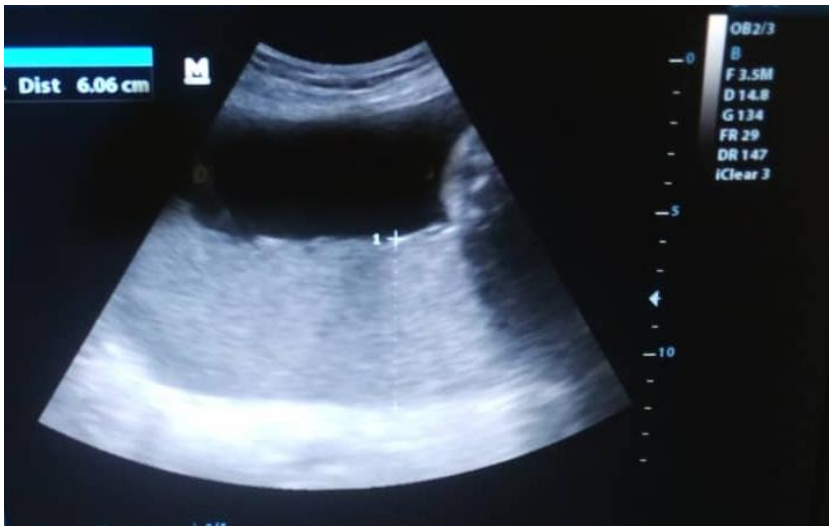


Image (1): US thick placenta (60.6mm) in 34year diabetic pregnant woman (Author source; 2018).

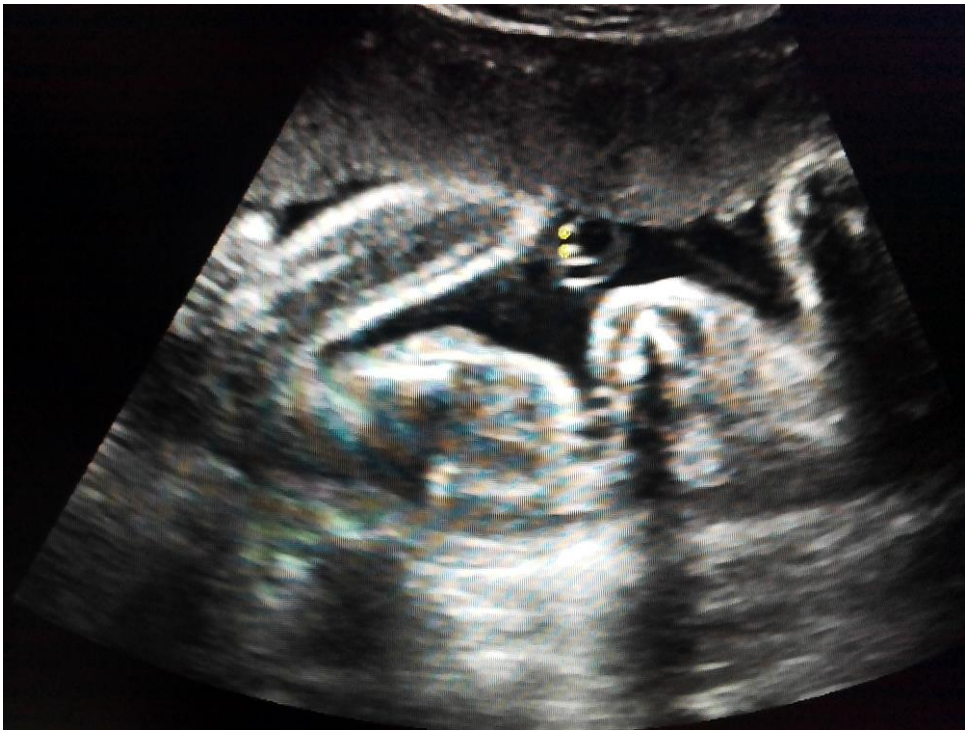


Image (2): US of UA diameter (3.5mm) in normal 38wks (Author source; 2018).

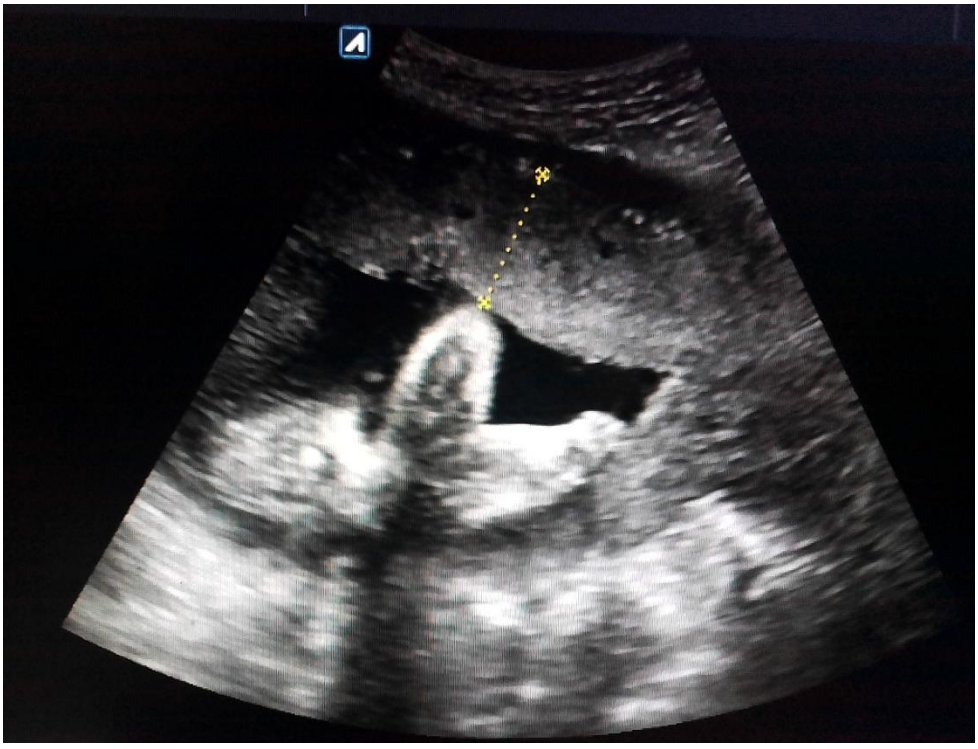


Image (3): US of normal PL thickness (30mm) in 31wks (Author source; 2018).

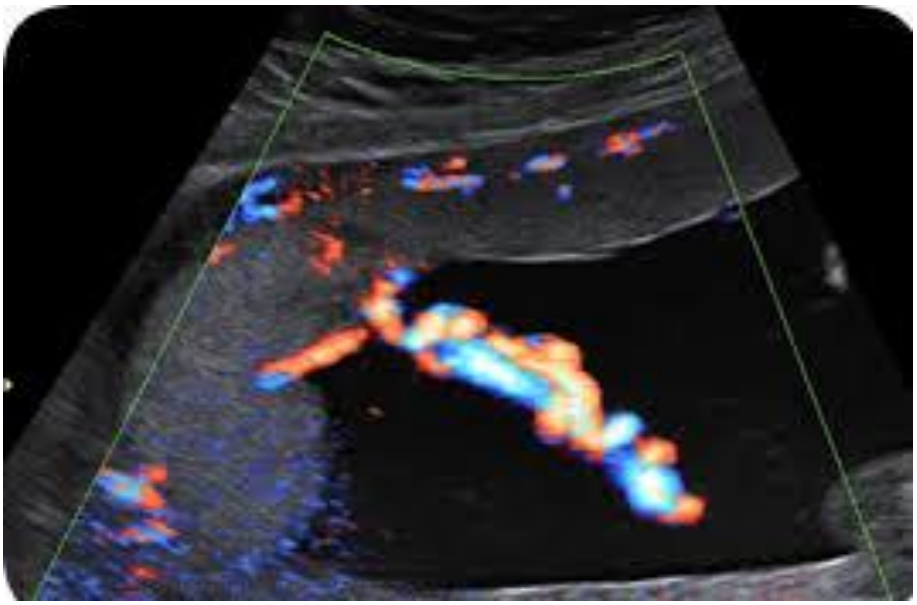


Image (4): Doppler US of normal PL thickness & umbilical cord (Author source; 2018).

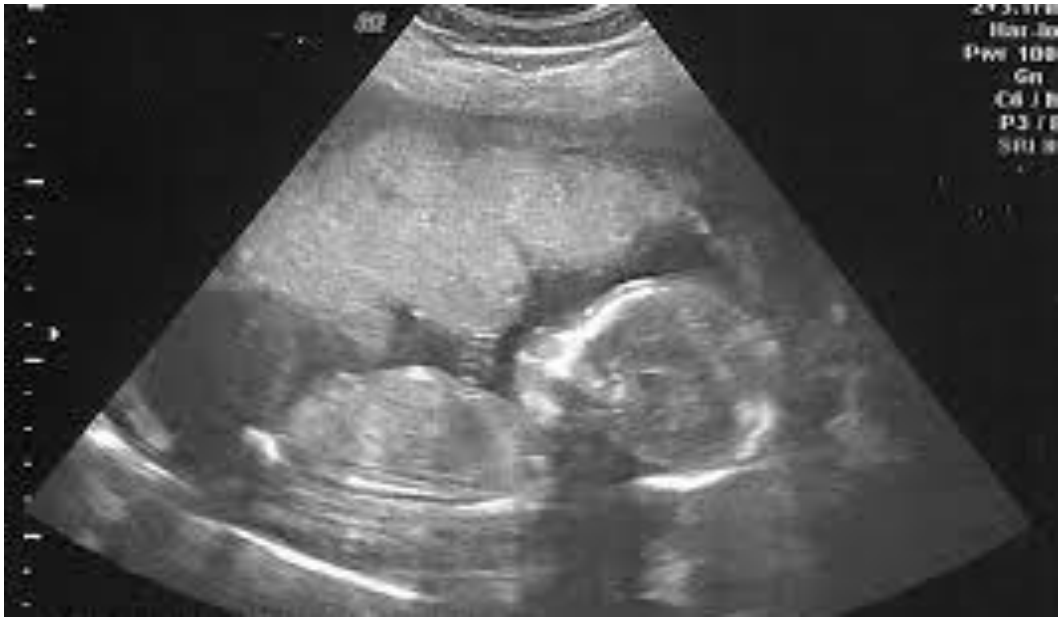


Image (5): US of placenta and 32wks fetus (Author source; 2018).

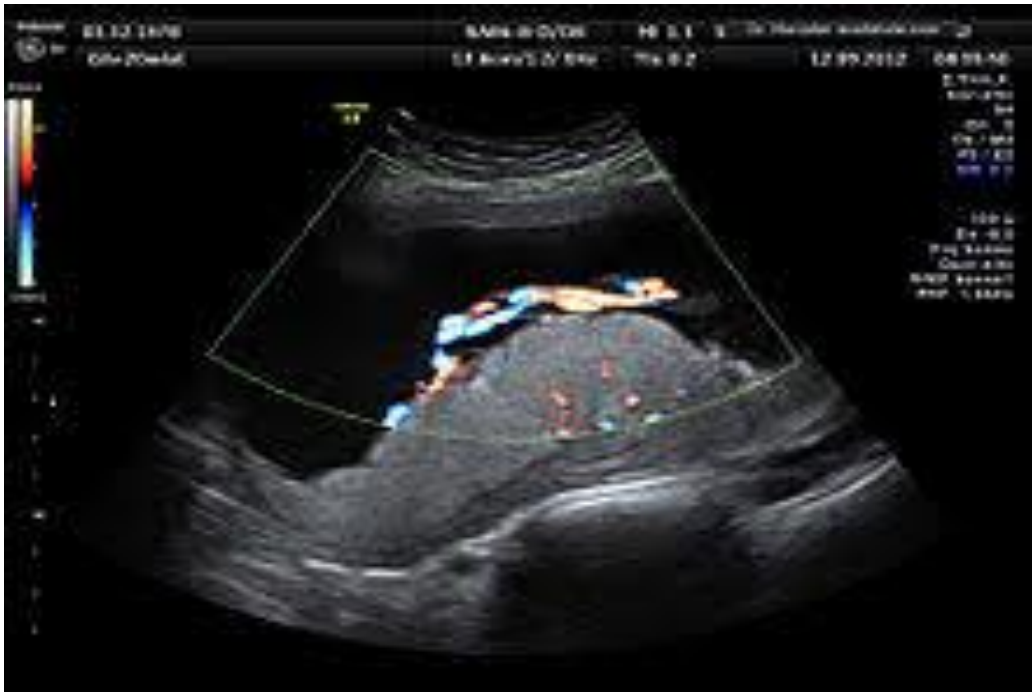


Image (6): Doppler US for placenta and umbilical cord (Author source; 2018).

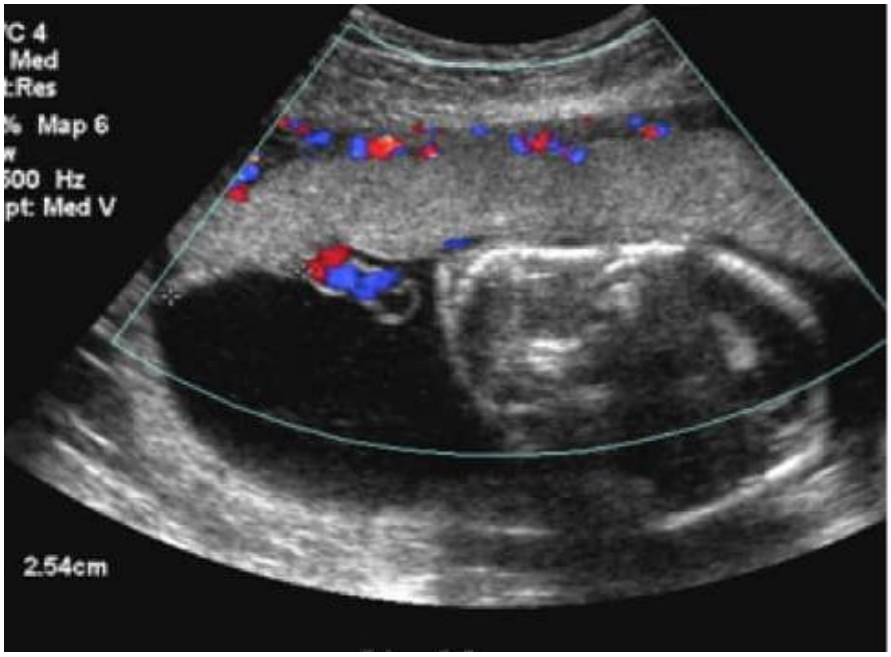
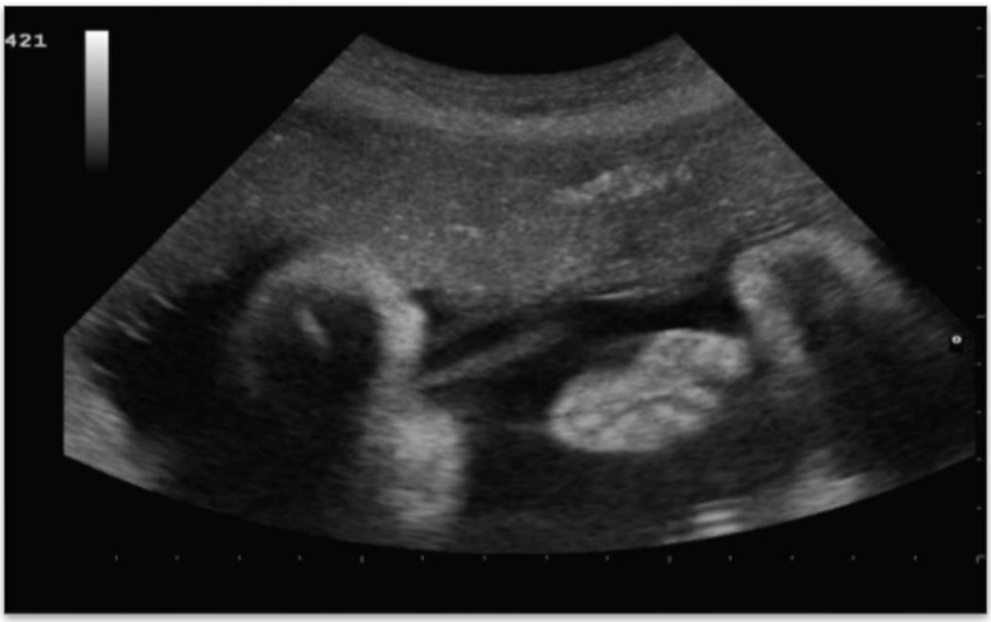


Image (7): US of normal placenta thickness 25.4mm, (Author source; 2018).



1mage (8): US of grade II placenta (Author source; 2018).

Appendix B

Data collection sheet

- Age …….
- Weight ……
- Height …….
- BMI ……..
- No. of pregnancy …….

- Occupation:

  House wife ( ) worker ( )

- Duration of disease (years) ……

- Type of diabetes:

  Insulin dependent ( ) non-insulin dependent ( )

- U/S Findings :

- No. of fetus ……
- GA …….
- Location of placenta ……..
- PL Grade …….
- PL Thickness …….
- Umbilical Artery diameter …….
- Other ……..