Sudan University of Sciences and Technology

College of Graduate Studies

AStudy of Placenta Thickness and Fetal Weight in Hypertensive Pregnant Women using Ultrasounography

دراسة سمك المشيمة ووزن الجنين لدي النساء الحوامل المصابات بارتفاع ضغط الدم باستخدام الموجات فوق الصوتية

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

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الايه

قَالَ تَعَالَىٰ: ﴿خَلَقَكُم مِّن نَّفْسٍ وَٰحِدَةٍ ثُمَّ جَعَلَ مِنْهَا زَوْجَهَا وَأَنزَلَ لَكُم مِّنَ ٱلْأَنْعَٰمِ ثَمَٰنِيَةَ أَزْوَٰجٍ ۚ يَخْلُقُكُمْ فِى بُطُونِ أُمَّهَٰتِكُمْ خَلْقًا مِّنۢ بَعْدِ خَلْقٍ فِى ظُلُمَٰتٍ ثَلَٰثٍ ۚ ذَٰلِكُمُ ٱللَّهُ رَبُّكُمْ لَهُ ٱلْمُلْكُ ۖ لَآ إِلَٰهَ إِلَّا هُوَ ۖ فَأَنَّىٰ تُصْرَفُونَ ۝٦﴾ الزمر: ٦

Dedication

To 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 **Dr. Ahmed Mustafa Abukonna** for his support. Also my thanks extend to my colleague **Ezzaldeen Geeli** for his help and encouragement. My thanks and gratitude to ultrasound department's staff of Omdurman maternal hospital and Saudi hospital.

Abstract

Hypertensive pregnancy may be responsible for vascular damage, enhanced systemic inflammation and insulin resistance in the placenta as oxygen and nutrient transfer is impaired and oxidative stress is generated affecting the placental growth and development.

Present study was done to investigate the changes in placenta in hypertensive pregnancy. A total of 40 pregnant women with hypertensive disorder were enrolled as cases in the study. A total of 40 matched normotensive pregnant women were enrolled as controls. All the women were followed up and findings were compared with normotensives. Data was compared using Independent sample t-test.

The results of the study showed the mean age of cases was 27.85 ± 6.09 years. Mean placental thickness in hypertensive pregnancy was also higher (4.1±1.07) compared with normotensive (3.7 ± 0.7) but difference was not significant statistically. Fetal Weight in Hypertensive Pregnancy was (2 ± 0.94) compared to normotensive (2.5 ± 0.7), there is a statistically significant difference. Hypertensive pregnancy does not affect the location of the placenta.

Hypertension during pregnancy affects the placental growth and development.

المستخلص

الحمل الناتج عن ارتفاع ضغط الدم قد يكون مسؤولاً عن تلف الأوعية الدموية ، والالتهاب الجهازي المعزز ومقاومة الأنسولين في المشيمة حيث يتم إعاقة نقل الأكسجين والمغذيات ويتولد الإجهاد التأكسدي الذي يؤثر على نمو المشيمة وتطورها.

أجريت هذه الدراسة للتحقيق في تغيرات المشيمة في الحمل الناتج عن ارتفاع ضغط الدم. تم تسجيل ما مجموعه 40 من النساء الحوامل المصابات باضطراب ارتفاع ضغط الدم كحالات في الدراسة. كما تم تسجيل ما مجموعه 40 من النساء الحوامل متوسط ضغط الدم المتطابقة كعناصر تحكم. تمت متابعة جميع النساء وتمت مقارنة النتائج مع المحفزات المعيارية. تمت مقارنة البيانات باستخدام اختبار t للعينة المستقلة.

أظهرت نتائج الدراسة أن متوسط عمر الحالات كان 27.85 ± 6.09 سنة. كان متوسط سمك المشيمة في الحمل المصحوب بارتفاع ضغط الدم أعلى (4.1 ± 1.07) مقارنة مع متوسط ضغط الدم (3.7 ± 0.7) لكن الفرق لم يكن ذو دلالة إحصائية. كان وزن الجنين في الحمل الناتج عن ارتفاع ضغط الدم أقل (2 ± 0.94) مقارنة بالضغط الطبيعي (2.5 ± 0.7) ، وهناك فرق معتد به إحصائياً. لا يؤثر الحمل الناتج عن ارتفاع ضغط الدم على مكان المشيمة.
يؤثر ارتفاع ضغط الدم أثناء الحمل على نمو المشيمة وتطورها.

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Chapter One

Chapter One

Introduction

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 220mm 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)

Hypertension during pregnancy affects 5–7% of all pregnancies and approximately 70% of cases occur in first-time pregnancies. The incidence of chronic hypertension (CHT) among pregnant women is 1–2%, while gestational hypertension (GHT) complicates 3–6% of all cases showing a rising tendency in recent years. It is a prominent cause of maternal and fetal morbidity and mortality; however, its

pathophysiological background is poorly understood. Most patients have no clinical symptoms, but it is important to emphasize that hypertension is merely one manifestation, i.e., the first stage of pre-eclampsia (PE). Proper uterine and placental vascularization is important for the adequate development of pregnancies. Pathological fetomaternal circulation can lead to elevated resistance in uterine circulation, which can cause placental insufficiency and thus – due to pathological development of the placenta – result in premature birth, intrauterine hypoxia, or even intrauterine death (Reynolds and Redmer, 2001).

The fetus is dependent on the placenta for maintaining and promoting normal development. In pregnancy with hypertension, pathological changes in the placenta such as infarction, calcifications, diffuse placental thrombosis, inflammatory placental vasculopathy and abnormal trophoblastic proliferation occur resulting in reduced blood flow across placenta and uteroplacental insufficiency (Salmani et al., 2014).

1.2. Problem of the study:

Hypertensive affects fetal wellbeing by inducing structural changes in the placenta, and also can cause changes in placenta thickness. So Sonography can determine the normal verses abnormal placenta. This study is designed to find change of placenta thickness and measurement of fetal Wight in hypertensive women.

1.2 Objectives of the study:

1.3.1 General objective:

The general objective of this thesis was to study placental thickness and fetal weight in hypertensive women using ultrasound.

1.3.2 Specific objectives:

- To measure the placenta thickness using ultrasound.
- To measure of fetal weight in second and third trimester to show the effect of hypertensive on it.
- To correlate between maternal age, duration of disease, placenta thickness and fetal weight.

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 Studies

Chapter Two

Theoretical background and Literature review

2.1. Literature Review:

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 and 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 syncytio trophoblasts (Ender and 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 maternal 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 and Fox, 1991)

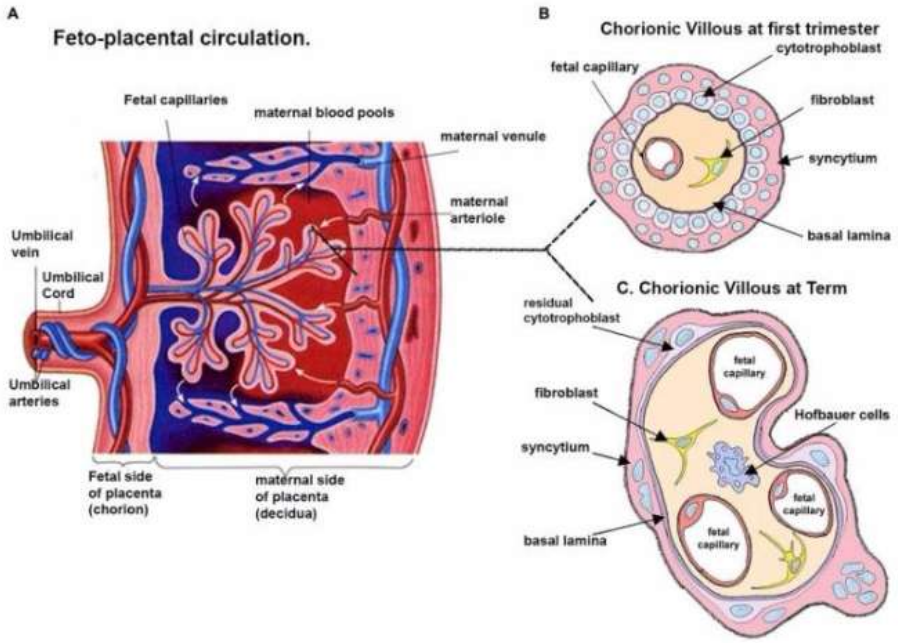

Figure (2.1) Schematic representation of a human placenta. (A) Fetal placental circulation. (B) Chorionic villous the presence of syncytiotrophoblast, a layer of cytotrophoblast cells, connective tissue of the villuscontaining fibroblasts and the fetal capillaries. (C) At term, in some areas the placental 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)

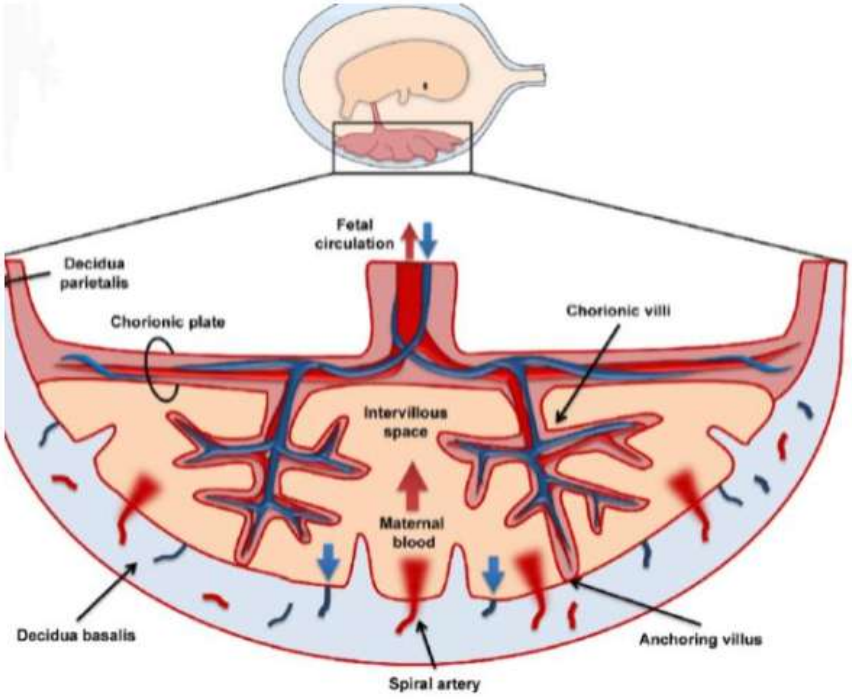

Figure (2.2) the fully developed human placenta.

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

2.1.2 Placenta and hypertensive:

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, whichare also produced by the adipose cells. The discovery that some of these adipokines are key players in the regulation of hypertensive resistance

suggests possible novel interactions between the placenta and adipose tissue in understanding pregnancy. The interplay between the two systems becomes more evident in gestational hypertensive (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)

2.1.3.1Hypertensive disorders of pregnancy:

(i) Gestational hypertension

(ii) Preeclampsia

(iii) Eclampsia

(iv) Chronic hypertension

(v) Chronic hypertension with superimposed pre eclampsia

Gestational hypertension is characterized by BP ≥140/90 mm Hg for the first time in pregnancy after 20 weeks, without proteinuria

2.1.3.1.1 Pre eclampsia

Preeclampsia is a multisystem disorder of unknown etiology characterized by development of hypertension to the extent of 140/90 mm Hg or more with proteinuria after the 20th week in a previously normotensive and nonproteinuric woman. 16 The root cause of preeclampsia is the placenta

2.1.3.1.2 Lack of physiologic conversion

During normal placental development, trophoblast invades the maternal spiral arteries, decidua and superficial myometrium, destroy the walls of the arterioles, and replace them with fibrinoid. These converts the vessels into a low resistance, high flow system and renders the vessels incapable of a vasoconstrictive response to the various vasoactive mediators. In women with pre-eclampsia, adequate trophoblast invasion does not occur. This results in inadequate placental perfusion. The etiology may be of immunological in origin. The second phase of pre-eclampsia is characterized by widespread endothelial damage leading to platelet adhesion and thrombosis.

Grossly the placenta is small placenta, less than the 10th percentile for gestational norms. There may be numerous placental infarcts. Placental abruption may be present. Microscopically, the placental parenchyma can exhibit an array of sublethal ischemic patterns: villous hypermaturation, increased syncytial knots, distal villous hypoplasia, agglutinated terminal villi, increased cytotrophoblast/fibrinoid islands, and increased perivillous fibrin. Lethal parenchymal ischemia is manifested by placental infarction, with or without abruption. Decidual arteriopathy, when present, ranges

from mild (nontransformation of spiral arterioles) to severe (fibrinoid necrosis with or without acute atherosis).

Chronic maternal vascular malperfusion sufficient to result in severe placental growth restriction (less than the fifth percentile for gestational age) almost invariably manifests significant fetal growth restriction as well. If a sufficient volume of the placental parenchyma is compromised by infarction, abruption, or villous ischemia, hypoxic fetal death results.

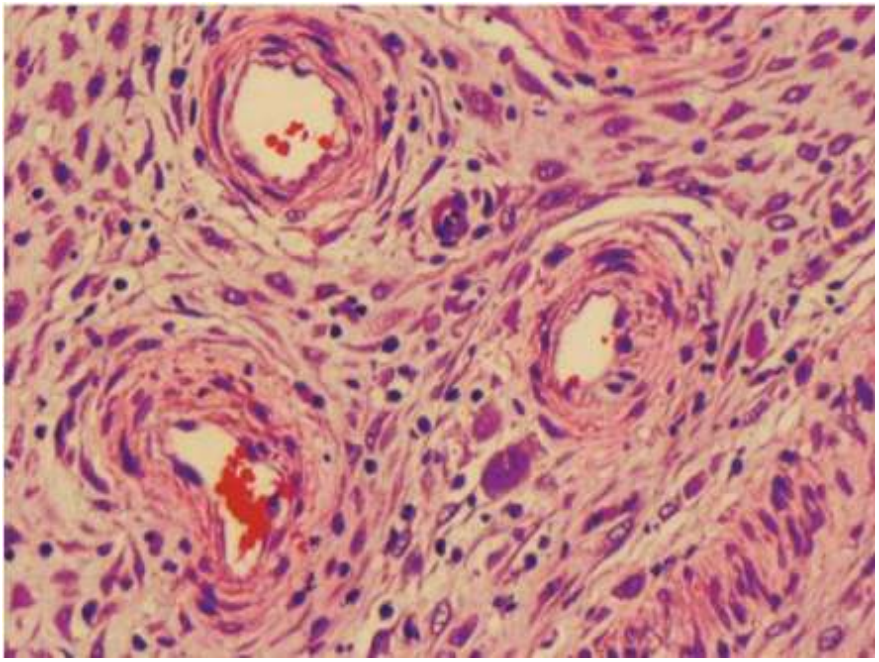

Figure 2.3 the arterioles retain their thick muscular coat

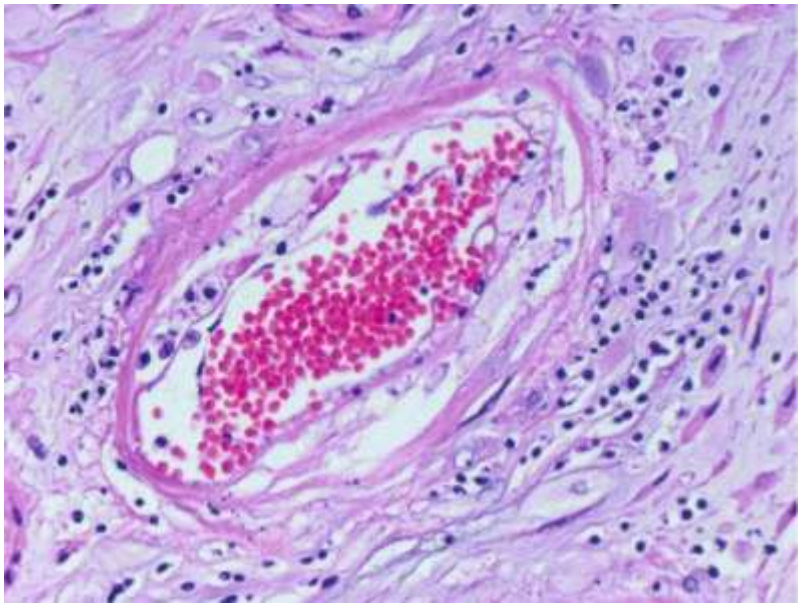

Figure 2.4: Acute atherosis and fibrinoid degeneration.

2.1.4 Intrauterine growth restriction

IUGR is defined as fetus that fails to achieve his growth potential. The terms SGA and FGR are not synonymous. It is important to remember that most SGA fetuses are constitutionally small and are not compromised. Intrauterine growth restriction (IUGR) indicates that there is a pathological process operating to restrict the growth rate of the fetus (Suhag and Berghella, 2013).

Outside of prematurity, IUGR is the second leading cause of perinatal mortality. Mothers who have a history of IUGR with a prior pregnancy are also at risk for recurrence in subsequent pregnancies, including an increased risk of subsequent stillbirth. Causes of growth restriction may be grouped into maternal, utero-placental, and fetal. Most common cause is utero -placental insufficiency. Maternal causes include Hypertension, cardiac disease, chronic renal failure, Substance abuse, alcohol, Smoking, autoimmune diseases, poor nutrition (Mandruzzato et al., 2008).

2.1.4.1 Uteroplacental insufficiency

Pre-eclampsia, placenta accreta, infarction, abruption, placenta previa. Fetal causes include genetic abnormalities, congenital abnormalities, congenital infection and multiple pregnancies 30. In IUGR, the placenta is usually small. The fetal surface should be examined for vascular thrombosis and the maternal surface for infarcts. The cut surface may show infarcts or increased perivillous fibrin. The umbilical cord is often thin (less than 1 cm in greatest diameter) (Ke et al., 2006).

Approximately one quarter of placentas associated with FGR lack any morphological abnormality on routine macroscopic and histological examination. Changes of uteroplacental insufficiency were the most common finding, being observed in 25% of cases. Of these, one -third had manifestations of early onset pre-eclampsia. Distal villous hypoplasia is characteristically seen in cases of marked intrauterine growth restriction (IUGR) that clinically may be associated with the absence of end-diastolic blood flow in the umbilical arteries. It is characterized by a sparse, poorly developed distal villous tree with abnormally shaped, elongated, slender villi and widening of the intervillous space.33Massive perivillous fibrin deposition (≥25% of villi encased by fibrin), is strongly associated with IUGR (Lewi et al., 2008).

Increased knotting for period of gestation are associated with increased risk of fetal intrauterine growth restriction 35. Villitis of Unknown Etiology is an important cause of intrauterine growth restriction and recurrent reproductive loss. Villitis of Unknown Etiology is caused by maternal T lymphocytes, predominantly CD8-positive, that inappropriately gains access to the villous stroma 36. Sato et al. noted higher prevalence of placental infarction, fetal vessel thrombosis, and

chronic villitis in IUGR compared with normal growth pregnancies (Pham et al., 2003).

2.1.5 Normal Sonographic Appearance of Placenta:

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.

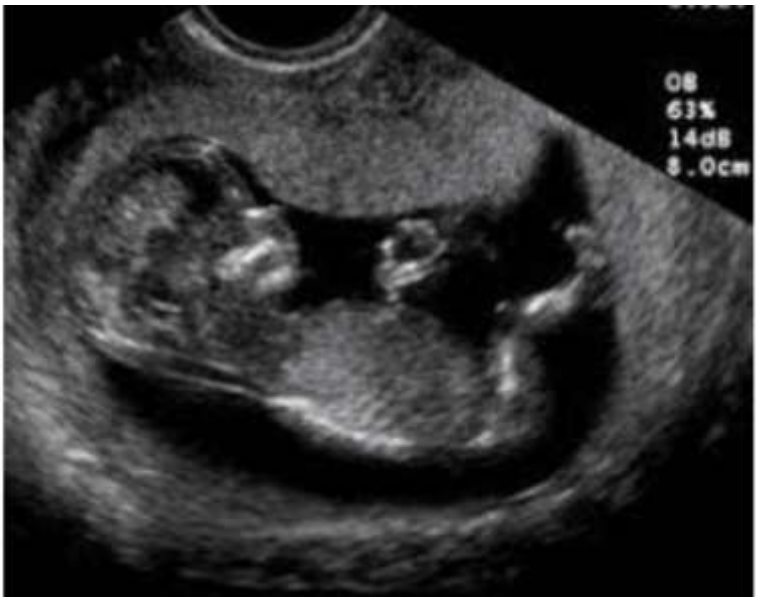

Figure (2.5): Sonographic appearance of fetus and placenta. (Author Source; 2018)

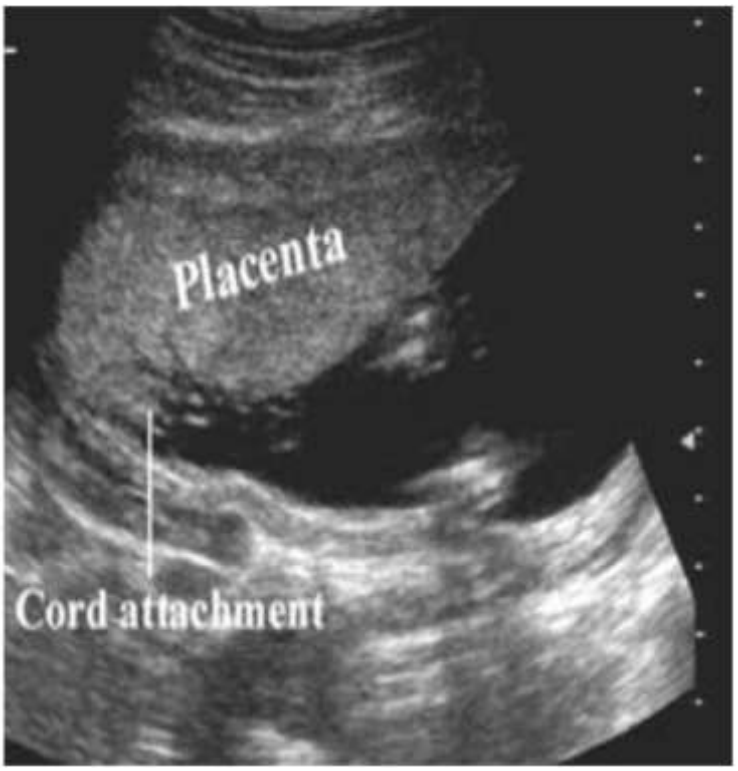

Figure (2.6): Sonographic appearance of placenta and umbilical cord (http://www.fetalultrasound.com; 2018)

2.2 Previous studies

(Nahar et al., 2015) conducted a descriptive cross sectional study to see the placental changes in normal & pregnancy induced hypertension (PIH) and its impacts on fetus for one year period. Total 80 placentas were collected, 40 from normal pregnant mothers having no hypertension and 40 from PIH group (one from gestational hypertension, 17 from pre-eclampsia and 22 from eclampsia. Macroscopic study of the placenta revealed placental weight, surface area and number of cotyledons were less in study group. Mean placental weight in study group was 419.50gm and in control group was 477.50 ($p<0.001$). Mean surface area in study group & control group were 232.29cm² and 304.80cm² respectively ($p<0.001$). Mean number of cotyledons were 15.39 and 17.40 in study & control group respectively ($P<0.001$) and lower diameter of umbilical cord ($p<0.04667$). There was a single umbilical artery present in one patient in PIH group .In PIH group syncytial knots (95%), fibrinoid

necrosis (80%), VSM (vasculosyncytial membrane) formation, sclerosis, chorangiosis and calcification were more marked. Infarction was present in placenta of PIH 34(85%) and in control group 8(20%). There was a tendency of lowering the weight of neonate 2.47kg in study group and 3.06kg in control group ($p<0.001$), number of asphyxiated babies and perinatal morbidity and mortality(still birth was 7.5 and neonatal death was 15%) were more marked in PIH group. In PIH group placental changes were related with fetal outcome. Common placental changes were significant in this study.

(Jashan et al., 2018) Study was done to investigate the morphological and histological changes in placenta in hypertensive pregnancy. A total of 42 pregnant women with hypertensive disorder with gestational age 28-42 weeks and singleton pregnancy were enrolled as cases in the study. A total of 42 matched normotensive pregnant women were enrolled as controls. Mean age of cases was 27.60±4.37 years, majority were gravida 1/2 (66.7%), 45.2% had moderate to severe edema, Mean placental weight and diameter of cases was significantly higher than that of control group. Mean placental thickness was also higher but difference was not significant statistically. Calcification, infarction and hematoma were seen in 45.2%, 16.7% and 11.9% of cases as compared to 28.6%, 4.97% and 0% of controls. Histologically, syncytial knots, cytotrophoblastic cellular proliferation, hyalinized area, proliferation of medium sized blood vessels, stromal fibrosis and fibrinoid necrosis in significantly higher proportion of cass as compared to controls ($p<0.05$). Mean fetoplacental ratio was 5.01±0.99 and 5.24±0.61 in controls ($p=0.195$). Hypertension during pregnancy affects the placental growth and development.

(Amit, 2017) Study was designed to detect the placental changes in hypertensive disorders of pregnancy and correlation with neonatal outcome. The study group comprised of 42 patients with pregnancy

complicated with hypertension with period of gestation above 20 weeks and B.P. more than 140/90 mm of Hg measured on two occasions 6 hours or more apart. The control group consists of 42 normotensive patients matched with age and parity. One ultrasonic examination was performed between 28 weeks and 34 weeks and another after 34 weeks till term.

In 97.62% cases (41 out of 42) of control group showed Grade–II changes as opposed to 57.14% of cases (24 out of 42) in study group before 34 weeks. Accelerated maturation of placenta in pregnancy complicated with hypertension is more common in the hypertensive group.

Chapter Three

Materials and Methods

Chapter Three

Materials and Method

3.1 Materials:

3.1.1 Subjects:

All pregnant women who are referred to the Ultrasound departments in Omdurman maternal Hospital and Saudi hospital for a routine follow up are participating in this study. The study was conducted during the period from August 2019 to August 2020.

3.1.2 Study Sample:

The sample size consisted of 40 pregnant women with hypertensive and control data 40 normal pregnant women in second and third trimester.

3.1.3 Inclusion criteria:

All hypertensive pregnant women in second and third trimester

3.1.4 Exclusion criteria:

Hypertensive pregnant women in the first trimester, normal pregnant women in the first trimester and diabetic pregnant women were excluded from the study.

3.1.5 Equipment:

The scan was performed using 3D ultrasound machines (SAMSUNG & ECUBE).

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

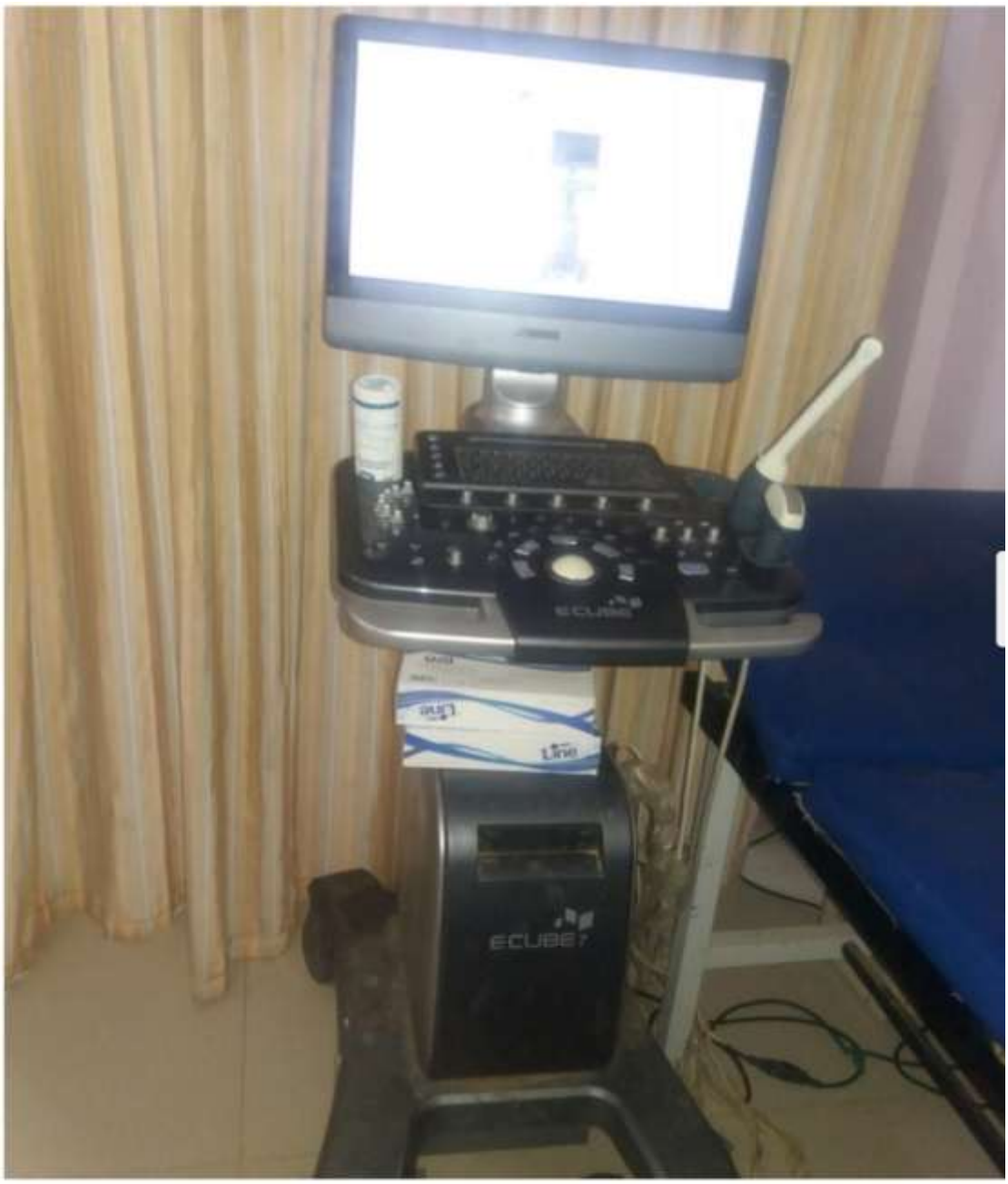

Figure 3-2 ECUBE ultrasound machine (Author source; 2018).

3.2 Method:

3.2.1 Technique Used:

The pregnant women referred 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.

3.2.2 Data Collection:

The data was collected by master data sheets using the variables of maternal age, number of pregnancy, occupation, hypertensive duration, management, GA, placental thickness.

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

Chapter Four

Results

4.1 Results:

Table 4.1: shows Descriptive Statistics for Age, NO of Pregnancy and Gestational Age:

	N	Minimum	Maximum	Mean	Std. Deviation
Age	80	18	45	27.85	6.086
NO of Pregnancy	80	1	9	3.00	1.856
Gestational Age	80	14	39	32.18	5.228

Table 4.2: shows Hypertension Type

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Gestational	25	62.5	62.5	62.5
	Chronic	15	37.5	37.5	100.0
	Total	40	100.0	100.0	

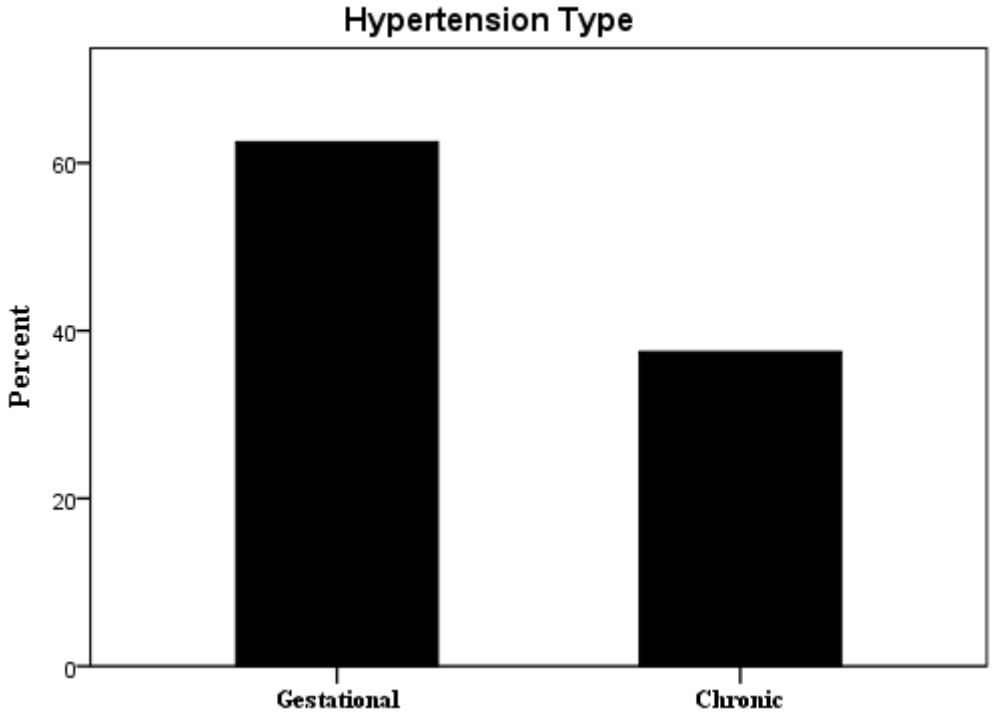

Figure 4.1: bar chart shows Hypertension Type

Table 4.3: shows Descriptive Statistics for (Placenta Thickness, Fetal Weight) for both Hypertension Types

	Hypertension Type	N	Mean	Std. Deviation	Sig (2 tailed)
Placenta Thickness	Gestational	25	4.1808	1.22538	0.521
	Chronic	15	3.9733	.79863	0.521
Fetal Weight	Gestational	25	2.1020	.94446	0.406
	Chronic	15	1.8420	.94556	0.406

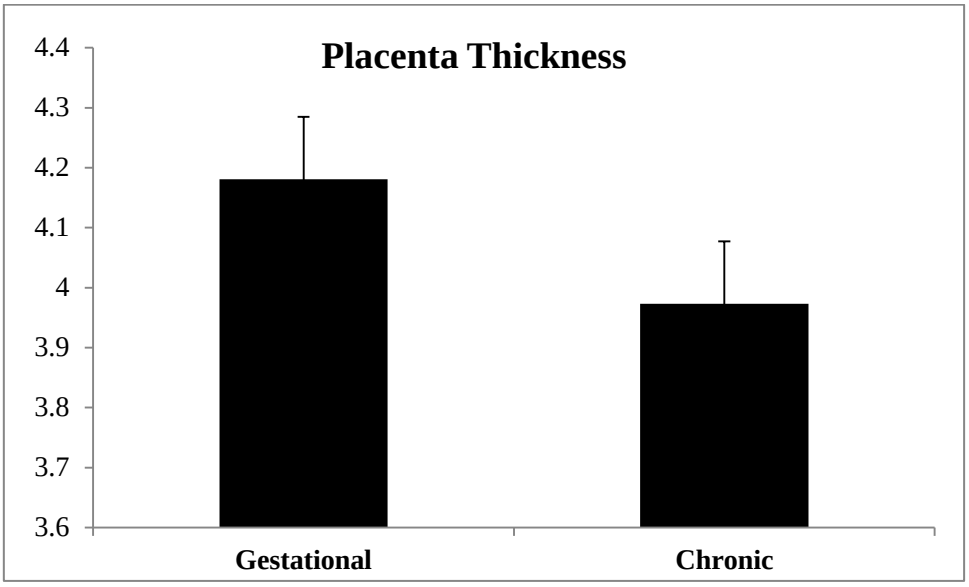

Figure 4.2: shows Placenta Thickness in both Hypertension Types

Table 4.4: shows Descriptive Statistics for Placenta Thickness, Fetal Weight in (Hypertension Pregnancy, Control)

Group Statistics

	TYPE	N	Mean	Std. Deviation	Sig (2 tailed)
Placenta Thickness	Hypertensive Pregnancy	40	4.1030	1.07858	0.081
	Control	40	3.7387	0.72999	0.081
Fetal Weight	Hypertensive Pregnancy	40	2.0045	0.94134	0.03
	Control	40	2.5667	0.69617	0.03

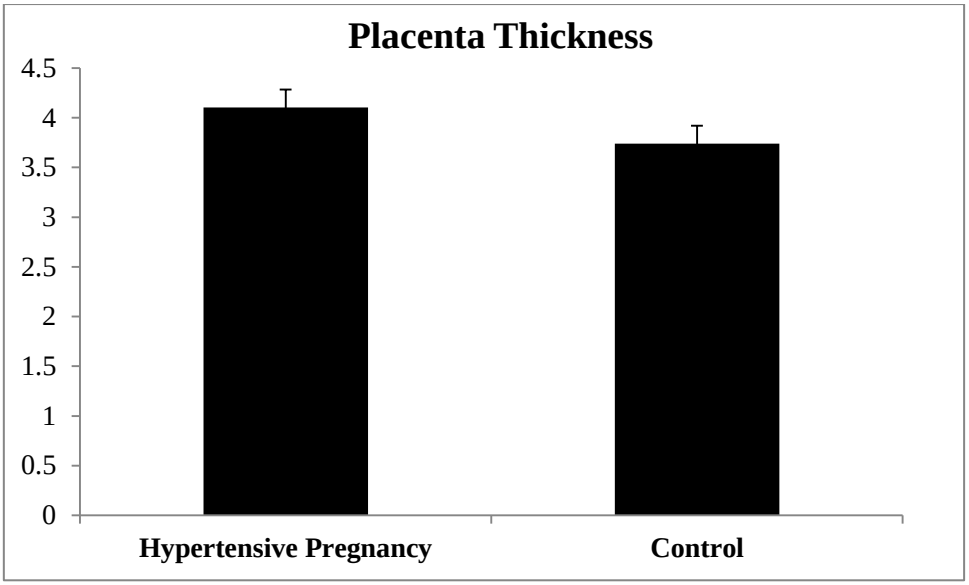

Figure 4.3: comparing mean of Placenta Thickness in (Hypertension Pregnancy, Control)

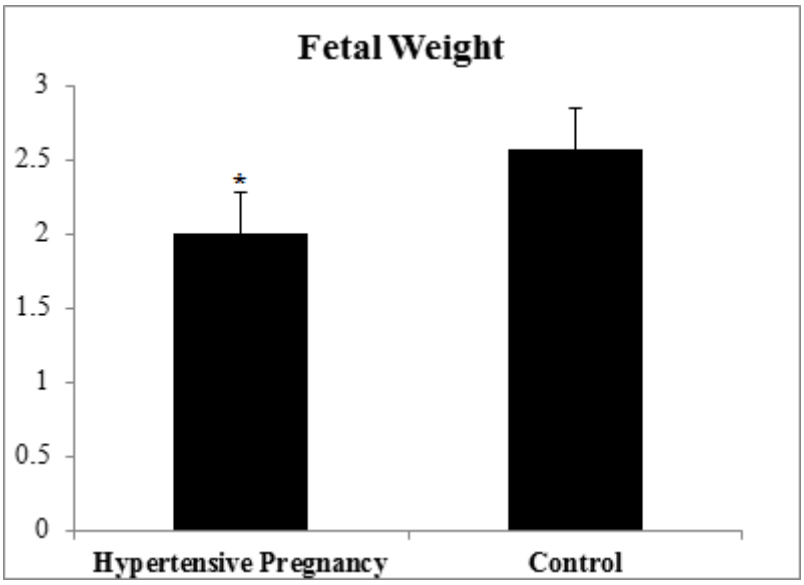

Figure 4.4: comparing mean of fetal weight in (Hypertension Pregnancy, Control)

Table 4.5: Cross tabulation between Placenta Location in both study groups.

			TYPE		Total
			Hypertensive Pregnancy	Control	
Placenta Location	Anterior	Count	19	21	40
		% within Placenta Location	47.5%	52.5%	100.0%
	Posterior	Count	19	15	34
		% within Placenta Location	55.9%	44.1%	100.0%
	Fundal	Count	2	4	6
		% within Placenta Location	33.3%	66.7%	100.0%
Total		Count	40	40	80
		% within Placenta Location	50.0%	50.0%	100.0%

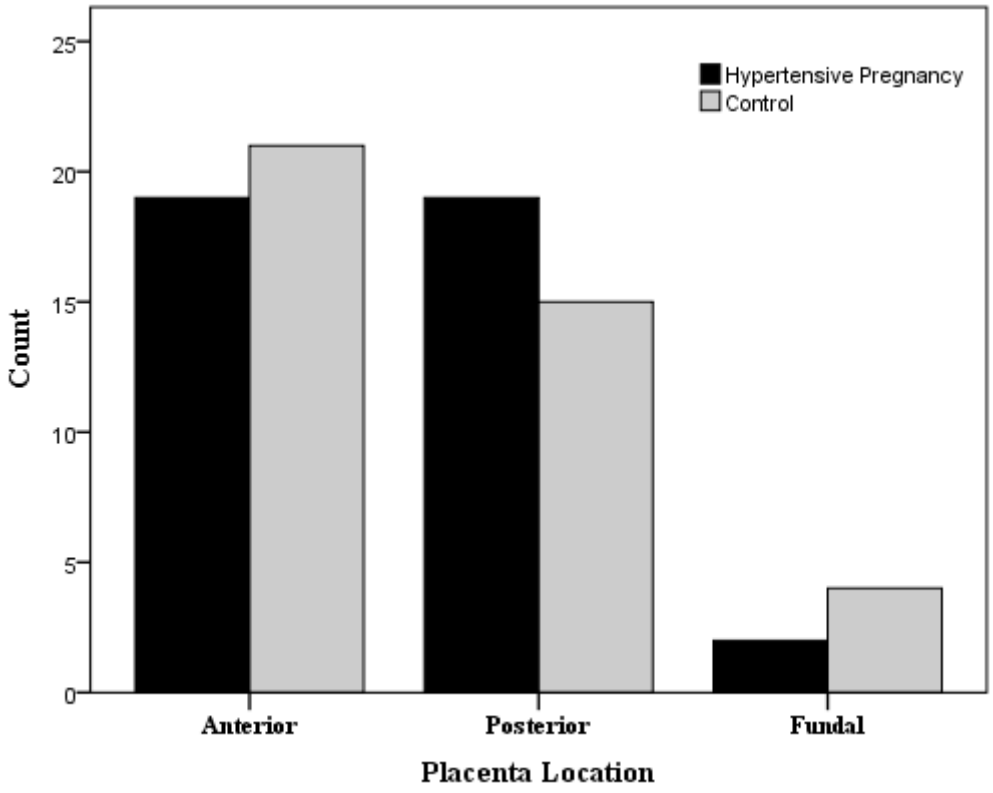

Figure 4.5: Placenta Location in both study groups

Chapter five

Discussion Conclusion and Recommendations

Chapter five

Discussion, Conclusion and Recommendations

5.1 Discussion:

This cross-sectional study was conducted in 80 pregnant women who referred to the Ultrasound departments in Omdurman maternal Hospital and Saudi hospital for a routine follow up, their age ranged between 18 and 45. 40 of them have hypertensive pregnancy (15 chronic, 25 with gestational) and 40 pregnant women serve as control group for comparison.

Regarding the Placenta Thickness, the study showed that in hypertensive pregnancy the placenta was higher (4.1±1.07) compared with normotensive (3.7 ± 0.7). This difference was not significant statistically (p = 0.521). This result was in line with the result of (Chatwal et al., 2018). Placental morphometric parameters in hypertensive pregnancies have been reported to be affected as compared to normotensive pregnancies in different studies.

As far as placental thickness in concerned, considerable variability in diameter has been reported in different studies; (Kulandaivelu et al., 2014) reported the placental diameter of hypertensive women to be 1.23 cm as compared to 1.42 cm in normotensive women (difference 0.19 cm), while (Qureshi et al., 2014) reported them as 2.2 and 3 cm (diff. 0.8 cm), (Singh and Gugapriya, 2014) reported them as 2.39 and 2.77 cm (diff. 0.38 cm) while (Rana et al., 2017) noted them as 1.9 cm and 2.4 cm (diff. 0.5cm). The reason for variance in placental thickness measurements and difference between the two groups in different studies could be owing to difference in method of measurement in different studies. Incidentally, placental thickness is not

even throughout the placenta. It is maximum at the center and minimum in the area between periphery and center.

Fetal Weight in Hypertensive Pregnancy was (2 ± 0.94) compared to normotensive (2.5 ± 0.7), there is a statistically significant difference. This result was in accordance with the study of (Mateus et al., 2019) who stated that women with pregnancy-associated hypertensive disorders, only those destined to develop severe preeclampsia demonstrated a significant and consistent difference in fetal growth (ie, smaller estimated fetal weight and abdominal circumference) when compared with normotensive women.

Concerning placenta location, table (4.5) showed Cross tabulation between Placenta Location (Anterior, Posterior, Fundal) and TYPE (Hypertensive Pregnancy, Control). Anterior placenta (Hypertensive Pregnancy 47.5%, Control 52.5%), Posterior (Hypertensive Pregnancy 55.9%, Control 44.1%), Fundal (Hypertensive Pregnancy 33.3%, Control 66.7%). It concluded that hypertensive pregnancy does not affect the location of the placenta.

5.2 Conclusion:

This study aimed to measurement of fetal weight in second and third trimester in hypertensive women using ultrasound. The study conclude that No significant relationship between Placenta Thickness and types of Hypertensive. No significant relationship between Fetal Weight and types of Hypertensive. No significant difference in Placenta Thickness between study groups. A significant difference was observed in Fetal Weight among Hypertensive and normal pregnancy.

5.3 Recommendations:

- Further study should be done with more sample size.
- Doppler indices as well as placenta diameters should be considered in the future studies.

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Appendices

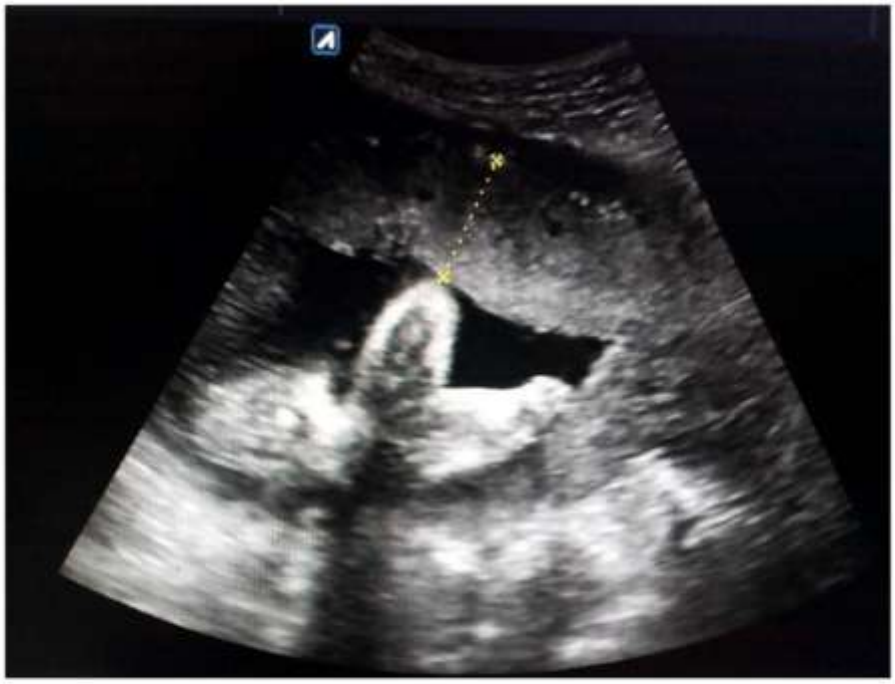


Image (3): US of normal PL thickness (30mm) in 31wks (Author source; 2018).

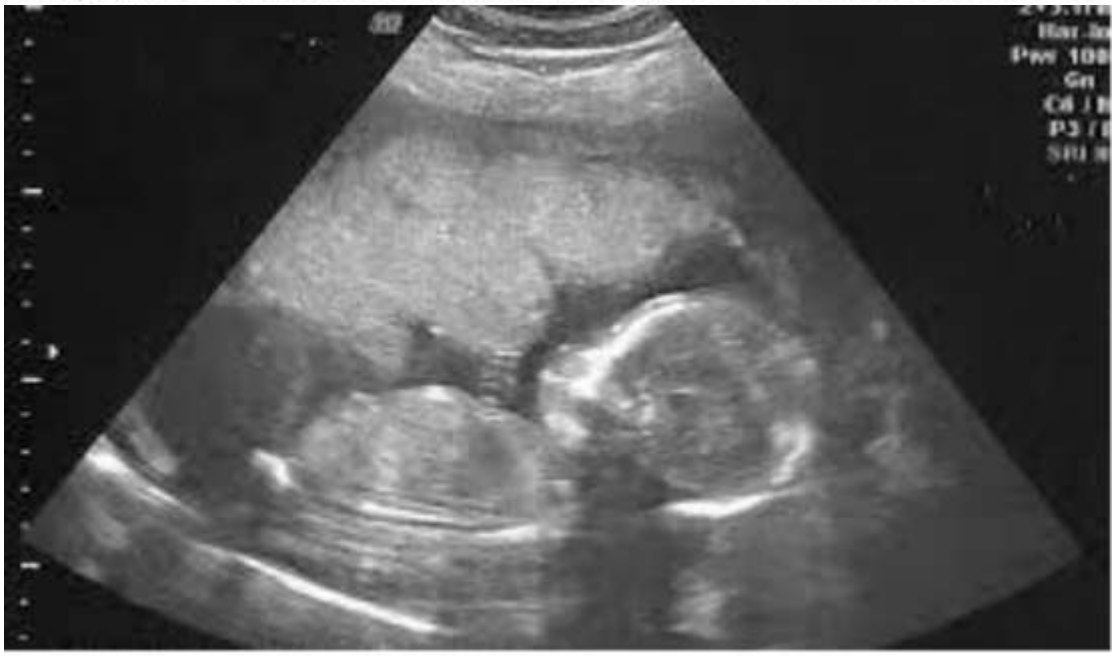


Image (5): US of placenta and 32wks fetus (Author source; 2018).

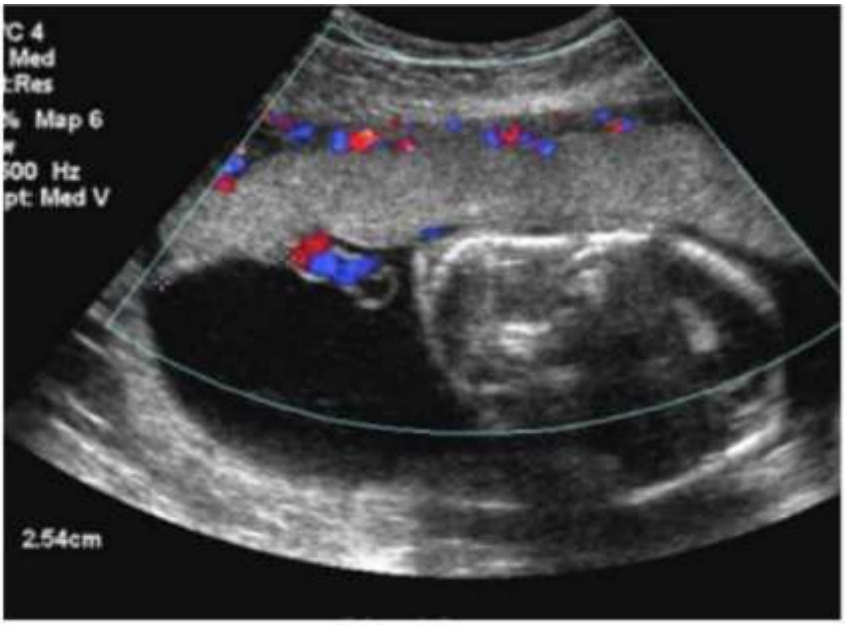
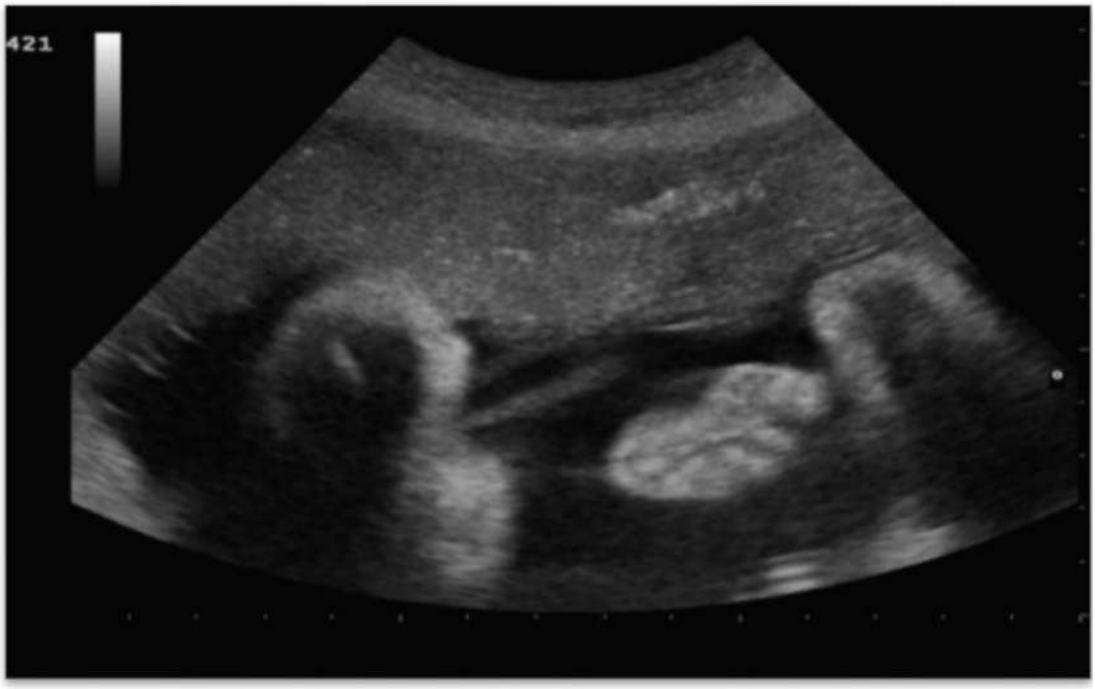


Image (7): US of normal placenta thickness 25.4mm, (Author source; 2018)



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

Data Collocation Sheet

- **Date** ……..
- **Age** ……...
- **Occupation**:-
  House Wife ( ) Worker ( )
- **BMI** ……..
- **No .of Pregnancy** ……
- **Duration of Disease (years)** ……...
- **Type of hypertension:-**

(A) Chronic hypertension ( )

(B) Gestational hypertension ( )

*** U /S Finding:-**

- No of fetus …..……

- GA ……

- Location of placenta ……

- PL Thickness …… **mm.**

- PL Grade ……

- Umbilical artery diameter ……

- BPD …….. **FL** …….

- Others …….

……..….

Signature

……