CHAPTER ONE

Introduction

1-1 Introduction

The placenta is a vascular structure by which an unborn child is attached to its mother's uterine wall and through which respiratory gas and metabolic exchange occurs. The placenta is formed in part from maternal tissue and in part from embryonic tissue. The embryonic portion of the placenta consists of the chorionfrondosum, whereas the maternal portion is composed of the area of the uterine wall called the decidua basalis, into which the chorionic villi penetrate. Blood does not flow directly between these two portions, but because their membranes are in close proximity, certain substances diffuse readily. When fully formed, the placenta is a reddish brown oval disc with a diameter of 15 to 20 cm and a thickness of 2.5 cm, It weighs between 500 and 600 g, about one sixth as much as the fetus. (Graaff 2001).

As a result of the continuous growth of the fetus and expansion of the uterus, the placenta also enlarges. Its increase in surface area roughly parallels that of the expanding uterus and throughout pregnancy it covers approximately 15 to 30% of the internal surface of the uterus. The increase in thickness of the placenta results from arborization of existing villi and is not caused by further penetration into maternal tissues. So Placental thickness is closely related to fetal wellbeing and may be a key factor in perinatal outcome. (Sadler 2004).

The use of ultrasound to evaluate the placenta is routine among the majority of pregnant women. A wide range of pregnancy complications result from abnormal placental development, including preeclampsia, intrauterine growth retardation

(IUGR) and abruption. Other placental abnormalities, such as placenta previa, percreta or vasa previa, may cause major maternal and fetal complications. Timely recognition of these abnormalities can lead to improve management of pregnancy and delivery. Thus, careful examinations of the placenta by ultrasound can contribute directly to enhance patient care and improve outcomes. (Rumack et al 2011).

Before the availability of the ultrasound, manual examination of the maternal abdomen was the only approach that could be used to estimate fetal size. The physical examination, however, provides only a general approximation of fetal weight because the palpated dimensions of the uterus are affected by several factors other than fetal size, including amniotic fluid volume, placental bulk, presence of fibroids and maternal obesity. Sonographic measurements of the fetus provide information about fetal age and growth. These data are used to assign gestational age, estimated fetal weight and diagnose growth disturbance. The measurements of fetal body parts provide a direct way of assessing fetal size. Numerous formulas have been published for estimating fetal weight from one or more of these fetal body measurements: head (biparietal diameter BPD or head circumference HC), abdomen (abdominal diameter AD or abdomen circumferenceAC), and femur length (FL). (Rumack et al 2011).

Estimation of fetal weight, on its own and in relation to the gestational age, can influence obstetric management decisions concerning the timing and route of delivery. Early delivery may benefit a fetus that is small for dates. Such a fetus may be inadequately supplied by its placenta with oxygen and nutrients and therefore may do better in the care of neonatologist than in uterus. When the fetus is large, cesarean section may be the preferred route of delivery, particularly in pregnancies complicated by maternal diabetes. In view of these considerations, fetal

measurements should be a component of every complete obstetric sonogram. (Rumack et al 2011).

1-2 Problem of the Study

The purpose of this study is to investigate the relationship between the placental thickness and estimated fetal weight in normal Sudanese pregnant women. To the best of our knowledge no such studies have been carried out in this aspect of obstetric ultrasonography in Sudan.

1-3 Objectives of the Study

1-3-1 General Objective

- To describe the association of the placental thickness and estimated fetal weight in pregnant Sudanese women.

1-3-2 Specific Objectives

- To measure the placental thickness, biparietal diameter and abdominal circumference.

- To estimate the fetal weight.

- To correlate between placental thickness and biparietal diameter, abdominal circumference and estimated feta weight.

1-4 Significance of the Study

Study gives idea about the relationship between the placental thickness and biparietal diameter, abdominal circumference, fetal age and estimated fetal weight.

1-5 Overview of the Study

The study fall in five chapters, with chapter one is an introduction, chapter two is the literature review, chapter three for methodology, chapter four for results and chapter five for discussion, conclusion and recommendations.

CHAPTER TWO

Litterateur Review

2-1   Anatomy of placenta

2-1-1 Development of placenta

As the fetus grows, its demands for nutritional and other factors increase, causing major changes in the placenta. Foremost among these is an increase in surface area between maternal and fetal components to facilitate exchange. (Sadler 2004)

a- Changes in the Trophoplast

By the beginning of the second month, the trophoblast is characterized by a great number of secondary and tertiary villi that give it a radial appearance. The villi are anchored in the mesoderm of the chorionic plate and are attached peripherally to the maternal decidua by way of the outer cytotrophoblast shell. The surface of the villi is formed by the syncytium, resting on a layer of cytotrophoblastic cells that in turn cover a core of vascular mesoderm.The capillary system developing in the core of the villous stems soon comes in contact with capillaries of the chorionic plate and connecting stalk, thus giving rise to the extra embryonic vascular system. (Sadler 2004)

During the following months, numerous small extensions sprout from existing villous stems into the surrounding lacunar or intervillous spaces. Initially these newly formed villi are primitive, but by the beginning of the fourth month, cytotrophoblastic cells and some connective tissue cells disappear. The syncytium and

endothelial wall of the blood vessels are then the only layers that separate the maternal and fetal circulations. Frequently the syncytium becomes very thin, and large pieces containing several nuclei may break off and drop into the intervillous blood lakes. These pieces, known as syncytial knots, enter the maternal circulation and usually degenerate without causing any symptoms. Disappearance of cytotrophoblastic cells progresses from the smaller to larger villi, and although some always persist in large villi, they do not participate in the exchange between the two circulations.(Sadler 2004).

b- Chorion Frondosum and Decidua Basalis

In the early weeks of development, villi cover the entire surface of the chorion. As pregnancy advances, villi on the embryonic pole continue to grow and expand, giving rise to the chorion frondosum (bushy chorion). Villi on the abembryonic pole degenerate and by the third month this side of the chorion, now known as the chorion laeve, is smooth. (Sadler 2004).

The difference between the embryonic and abembryonic poles of the chorion is also reflected in the structure of the decidua, the functional layer of the endometrium, which is shed during parturition. The decidua over the chorion frondosum, the decidua basalis, consists of a compact layer of large cells, decidual cells, with abundant amounts of lipids and glycogen. This layer, the decidual plate, is tightly connected to the chorion. The decidual layer over the abembryonic pole is the decidua capsularis. With growth of the chorionic vesicle, this layer becomes stretched and degenerates. Subsequently, the chorion laeve comes into contact with the uterine wall (decidua parietalis) on the opposite side of the uterus and the two fuses, obliterating the uterine lumen. Hence the only portion of the chorion participating in

the exchange process is the chorion frondosum, which, together with the deciduas basalis, makes up the placenta. Similarly, fusion of the amnion and chorion to form the amniochorionic membrane obliterates the chorionic cavity. It is this membrane that ruptures during labor (breaking of the water). (Sadler 2004).
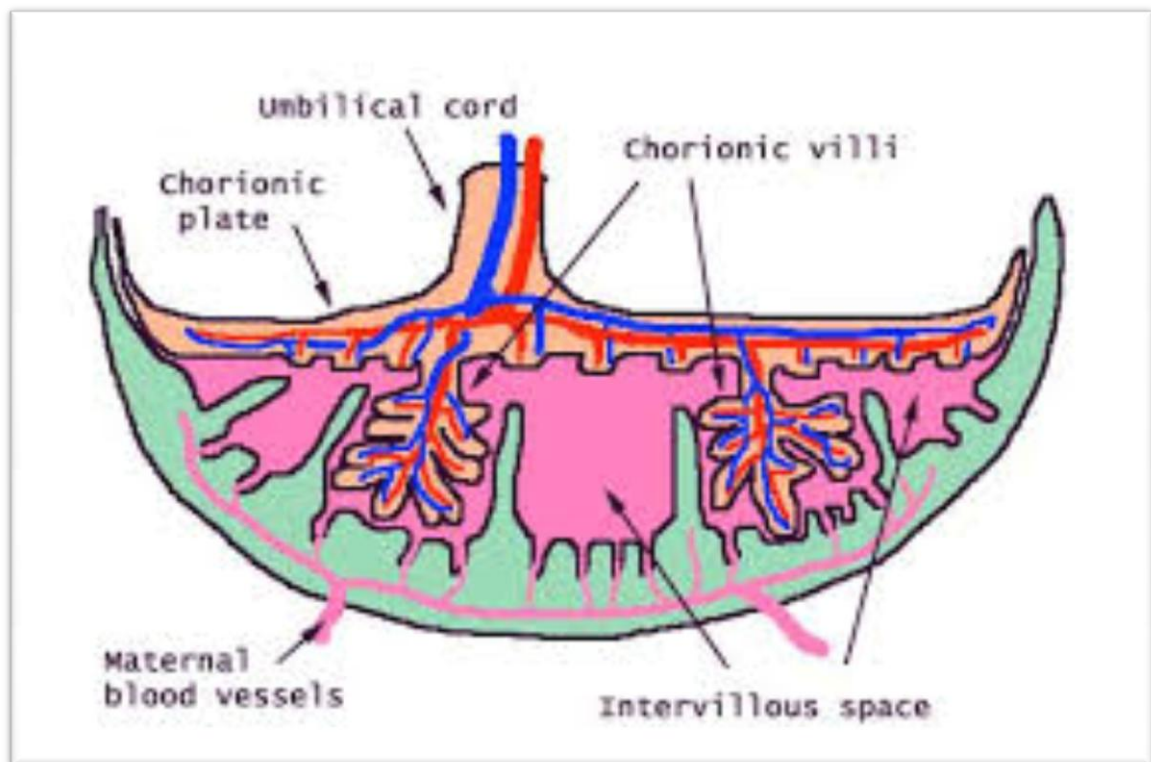

Figure 2-1: structure of the placenta: show the chorionic villi, chorionic plate, umplical cord, maternal blood vessels and intervillous spaces.
(http://www.siumed.edu/~dking2/erg/placenta.htm)

2-1-2 Structure of the Placenta

By the beginning of the fourth month, the placenta has two components: (*a*) a fetal portion, formed by the chorion frondosum; and (*b*) a maternal portion, formed by the decidua basalis. On the fetal side, the placenta is bordered by the chorionic plate; on its maternal side, it is bordered by the decidua basalis, of which the decidual plate

is most intimately incorporated into the placenta. In the junctional zone, trophoblast and decidua cells intermingle. This zone, characterized by decidual and syncytial giant cells, is rich in amorphous extracellular material. By this time most cytotrophoblast cells have degenerated. Between the chorionic and decidual plates are the intervillous spaces, which are filled with maternal blood. They are derived from lacunae in the syncytiotrophoblast and are lined with syncytium of fetal origin. The villous trees grow into the intervillous blood lakes. During the fourth and fifth months the decidua forms a number of decidual septa, which project into intervillous spaces but do not reach the chorionic plate. These septa have a core of maternal tissue, but their surface is covered by a layer of syncytial cells, so that at all times a syncytial layer separates maternal blood in intervillous lakes from fetal tissue of the villi. As a result of this septum formation, the placenta is divided into a number of compartments, or cotyledons. Since the decidual septa do not reach the chorionic plate,contact between intervillous spaces in the various cotyledons is maintained. (Sadler 2004)

As a result of the continuous growth of the fetus and expansion of the uterus, the placenta also enlarges. Its increase in surface area roughly parallels that of the expanding uterus and throughout pregnancy it covers approximately15 to 30% of the internal surface of the uterus. The increase in thickness of the placenta results from arborization of existing villi and is not caused by further penetration into maternal tissues. (Sadler 2004).

2-1-3 Full Term Placenta

At full term, the placenta is discoid with a diameter of 15 to 25 cm, is approximately 3 cm thick, and weighs about 500 to 600 g. At birth, it is torn from the

uterine wall and, approximately 30 minutes after birth of the child, is expelled from the uterine cavity. After birth, when the placenta is viewed from the maternal side, 15 to 20 slightly bulging areas, the cotyledons, covered by a thin layer of decidua basalis, are clearly recognizable. Grooves between the cotyledons are formed by decidual septa. (Sadler 2004)

The fetal surface of the placenta is covered entirely by the chorionic plate. A number of large arteries and veins, the chorionic vessels, converge towardthe umbilical cord. The chorion, in turn, is covered by the amnion. Attachment of the umbilical cord is usually eccentric and occasionally even marginal. Rarely, however, does it insert into the chorionic membranes outside the placenta (velamentous insertion). (Sadler 2004).
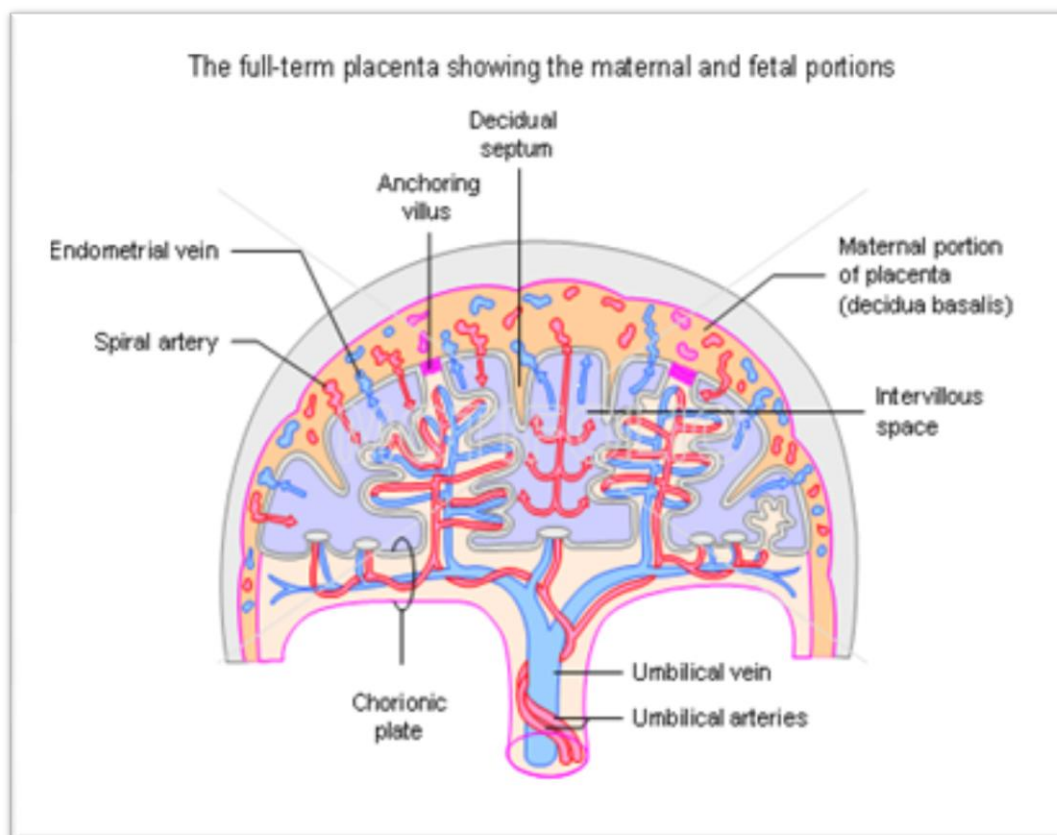

Fig 2-2 the full-term placenta showing maternal and fetal portions

(http://www.inharmonybirth.com/placenta-preparation)

2-1-4 Circulation of the Placenta

Cotyledons receive their blood through 80 to 100 spiral arteries that pierce the decidual plate and enter the intervillous spaces at more or less regular intervals. The lumen of the spiral artery is narrow, so blood pressure in the intervillous space is high. This pressure forces the blood deep into the intervillous spaces and bathes the numerous small villi of the villous tree in oxygenated blood. As the pressure decreases, blood flows back from the chorionic plate toward the decidua, where it enters the endometrial veins. Hence, blood from the intervillous lakes drains back into the maternal circulation through the endometrial veins. (Sadler 2004).

Collectively, the intervillous spaces of a mature placenta contain approximately 150 ml of blood, which is replenished about 3 or 4 times per minute. This blood moves along the chorionic villi, which have a surface area of 4 to 14 m2. However; placental exchange does not take place in all villi, only in those whose fetal vessels are in intimate contact with the covering syncytial membrane. In these villi, the syncytium often has a brush border consisting of numerous micro villi, which greatly increases the surface area and consequently the exchange rate between maternal and fetal circulations. The placental membrane, which separates maternal and fetal blood, is initially composed of four layers: (*a*) the endothelial lining of fetal vessels; (*b*) the connective tissue in the villus core; (*c*) the cytotrophoblastic layer; and (*d*) the syncytium. From the fourth month on, however, the placental membrane thins, since the endothelial lining of the vessels comes in intimate contact with the syncytial membrane, greatly increasing the rate of exchange. Sometimes called the placental barrier, the placental membrane is not a true barrier, since many substances pass

through it freely. Because the maternal blood in the intervillous spaces is separated from the fetal blood by a chorionic derivative, the human placenta is considered to be of the hemochorial type. (Sadler 2004).
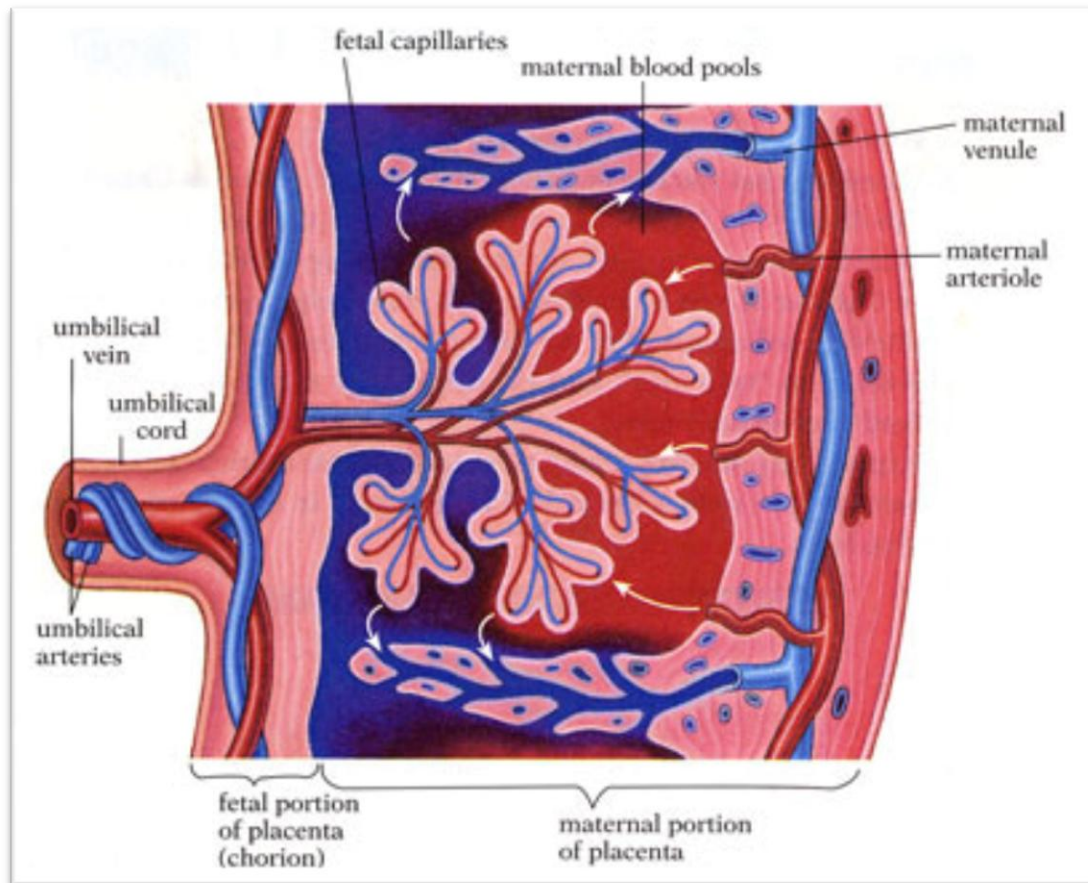

Figure 2-3: placental circulation: gas, nutrient, and waste exchanges between the mother and the fetus take place in the placenta, where fetal blood passes through capillaries   alongside those containing maternal blood. (http://www.biog1445.org/demo/07/ovaryplacenta.html).

2-1-5 Placental Grading

Calcium deposition in the placenta is a normal process of placental aging or maturation which occurs at different rates in normal pregnancies. Sonographically, macroscopic areas of placental calcifications appear as hyperechoic densities in different areas of the placenta. Calcium is deposited primarily along the basal surface

and placental septa. Macroscopic and sonographic evidence of placental calcification is not evident until the third trimester. Previously, investigators found it useful to assign placentas numerical grade (0 to 3) based on the degree of calcification however such grading schemes have proven to be of limited value in clinical practice in predicting fetal maturity, fetal well being, or perinatal outcome. These placentas show an irregular amniochorionic surface (chorionic plate) with calcification extending along the cotyledenal division from the chorionic plate to the basal surface. The grade 3 placenta may also have larger areas of calcification that produce shadowing and the placental parenchyma may contain hypoechoic or anechoic areas. Grade 3 placentas are the most heavily calcified and are not seen before 36 weeks gestation in normal pregnancies.(Burwin Institute Notes).
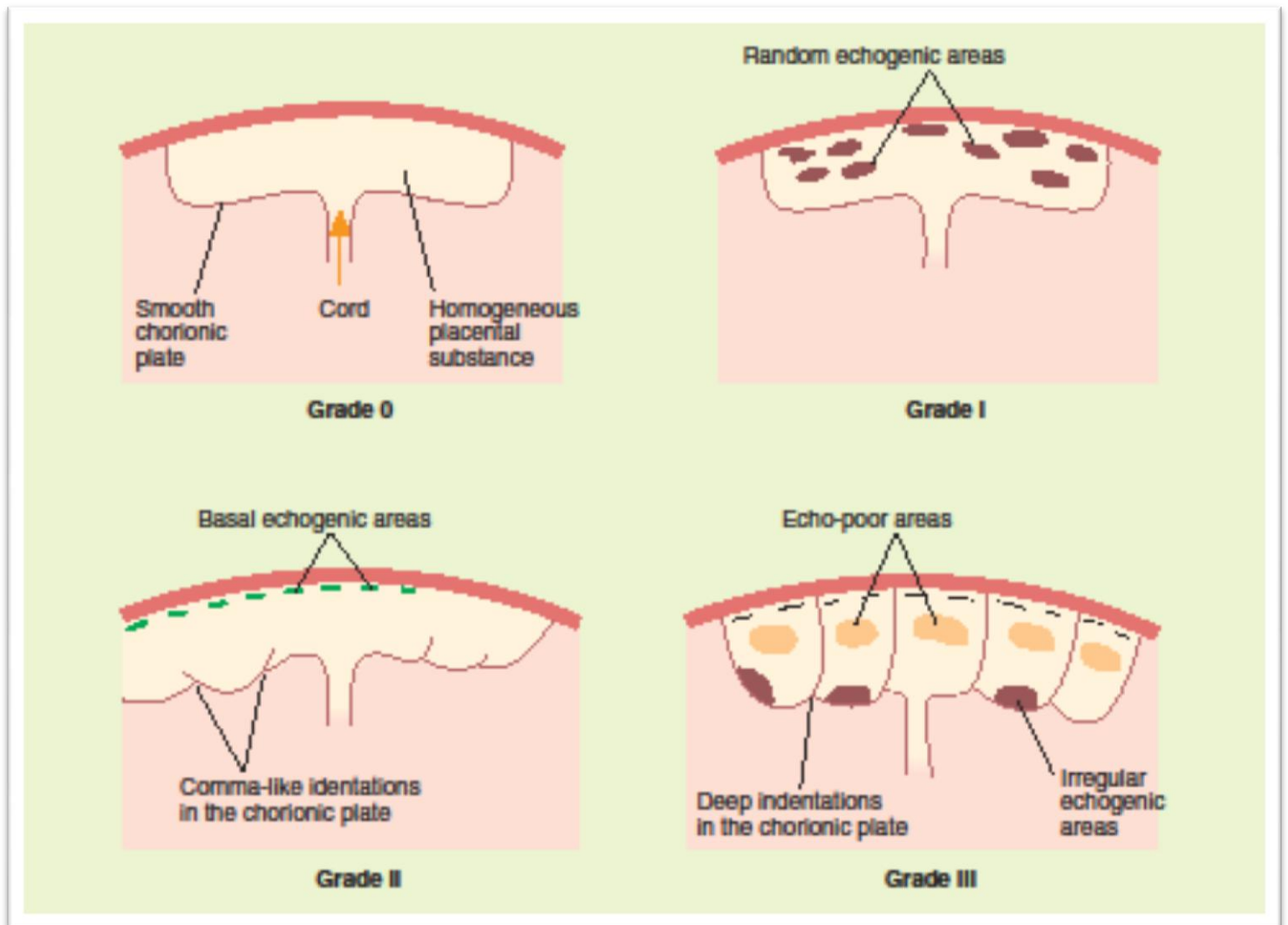
Fig 2-4: show the ultrasound appearance of placental grading(Chudleigh & Thilaganathan., 2004)

Fig 2-5: Grade 3 Placenta: US image of an anterior placenta shows calcification of the placenta extending from the maternal to fetal surface, (arrows). (Burwin Institute Notes).

2-1-6 Umbilical Cord

The umbilical cord connects the fetus to the placenta. Within the cord are two umbilical arteries that carry blood from the fetus to the placenta and one umbilical vein that returns blood from the placenta to the fetus. When blood in the umbilical arteries enters the placenta, CO_2 and waste products in the fetal capillaries diffuse into the maternal blood sinuses. Oxygen diffuses from the maternal blood sinuses into the fetal capillaries; nutrients enter the fetal blood by diffusion and active transport mechanisms. This oxygen- and nutrient-rich blood then flows through the umbilical vein back to the fetus. (Scanlon and Sanders 2007).

When the baby is delivered at the end of gestation, the umbilical cord is cut. The placenta then detaches from the uterine wall and is delivered, with the rest of the umbilical cord, as the afterbirth. (Scanlon and Sanders 2007).

Figure 2-6: Normal Cord Insertion: Sonogram of the uterus shows a posterior placenta with a central umbilical cord insertion. (Burwin Institute Notes).

2-2 Physiology of Placenta

Main functions of the placenta are nutrition, excretion, protection and production of hormones (http://en.wikipedia.org/wiki/Placenta).

2-2-1 Nutrition

The perfusion of the intervillous spaces of the placenta with maternal blood allows the transfer of nutrients and oxygen from the mother to the fetus and the transfer of waste products and carbon dioxide back from the fetus to the maternal blood supply. Nutrient transfer to the fetus occurs via both active and passive transport. Active transport systems allow significantly different plasma concentrations of various large molecules to be maintained on the maternal and fetal sides of the placental barrier. Adverse pregnancy situations, such as those involving maternal diabetes or obesity can increase or decrease levels of nutrient transporters in the

placenta resulting in overgrowth or restricted growth of the fetus.(http://en.wikipedia.org/wiki/Placenta).

2-2-2 Excretion

Waste products excreted from the fetus such as urea, uric acid, and creatinine are transferred to the maternal blood by diffusion across the placenta. (http://en.wikipedia.org/wiki/Placenta).

2-2-3 Protection

IgM antibodies can pass through the human placenta, thereby providing protection to the fetus *in utero*. This transfer of antibodies begins as early as the 20th week of gestational age, and certainly by the 24th week. This passive immunity lingers for several months after birth, thus providing the newborn with a carbon copy of the mother's long-term humoral immunity to see the infant through the crucial first months of extra uterine life. IgM, however, cannot cross the placenta, which is why some infections acquired *during* pregnancy can be hazardous for the fetus. (http://en.wikipedia.org/wiki/Placenta).

Furthermore, the placenta functions as a selective maternal-fetal barrier against transmission of microbes. However, insufficiency in this function may still cause mother-to-child transmission of infectious diseases. (http://en.wikipedia.org/wiki/Placenta).

2-2-4 Production of Hormones

In humans, aside from serving as the conduit for oxygen and nutrients for fetus, the placenta secretes, from the syncytial layer of chorionic villi, the following hormones that are important during pregnancy (http://en.wikipedia.org/wiki/Placenta):

a- Human Chorionic Gonadotropin (HCG) can be found in maternal blood and urine as shortly after implantation has occurred, and increases through to the 10-12th week of pregnancy, decreasing to a stable level around the 16-18th week. HCG also ensures that the corpus luteum continues to secrete progesterone and estrogen, which is important in sustaining the pregnancy until sufficient estrogen and progesterone can be secreted by the placenta itself. HCG suppresses the maternal immunologic response so that placenta is not rejected. This is the hormone analyzed by pregnancy test; a false-negative result from a pregnancy test may be obtained before or after this period. Women's blood serum will be completely negative for HCG by one to two weeks after birth. HCG testing is proof that all placental tissue is delivered. HCG is present only during pregnancy because it is secreted by the placenta (http://en.wikipedia.org/wiki/Placenta).

b- Human Placental Lactogen (HPL) promotes mammary gland growth in preparation for lactation in the mother. It also regulates maternal glucose, protein, and fat levels so that this is always available to the fetus. HPL levels increase proportional to placenta size (http://en.wikipedia.org/wiki/Placenta).

c- Estrogen is secreted in levels up to thirty times those in non-pregnant women. Estrogen causes the mother's breasts, uterus and external genitalia to enlarge.

Breast enlargement and glandular development is in preparation for lactation and uterine growth to accommodate growing fetus. Estrogen also causes relaxation of ligaments, including the sacroiliac joints and symphysis pubis, which will ease a vaginal birth (http://en.wikipedia.org/wiki/Placenta).

d- Progesterone is necessary to maintain endometrial lining of the uterus during pregnancy. This hormone prevents preterm labor by reducing myometrial contraction. Levels of progesterone are high during pregnancy. (http://en.wikipedia.org/wiki/Placenta).

2-2-5 Other Functions

The placenta also provides a reservoir of blood for the fetus, delivering blood to it in case of hypotension and vice versa, comparable to a capacitor (http://en.wikipedia.org/wiki/Placenta).

2-3 Pathology of Placenta

2-3-1 Hydatidiform Mole

a. Total Hydatidiform Mole

It is abnormal pregnancy, where all placental villi change to molar vesicles and fill uterine cavity, while there is no embryo, fetus, nor umbilical cord. Amnion is, however, found in some cases. No capillary vessel is noted in the molar cyst which is covered by proliferated trophoblast. Microscopically found molar cyst of diameter less than 2 mm is called microscopic mole. Trophoplast are scattered in the decidua and myometrium, and called syncytial endometritis. Molar cysts may spread into

blood vessel, which is the intravascular mole, and rarely metastasis appears in distant organ.(Kurjak andChervenak 2006).

b- Partial Hydatidiform Mole

It is partial change of placental villi into the mole, which is associated with embryo, fetus or fetal parts. Fetal anomalies are common. Capillary vessels are found in molar interstitium. (Kurjak and Chervenak 2006).

c- Invasive Hydatidiform Mole

It is the invasion of molar cysts into myometrium with destruction and hemorrhage. Intravascular mole and placental polyp are excluded from the invasive mole. The lesion is formed either in total or partial mole, usually after the molar evacuation, although the invasion may develop before the termination. The change is visually noted in surgical specimen and microscopically confirmed, where the trophoblasts proliferate, hemorrhage and necrosis are found in the myometrium. (Kurjak and Chervenak 2006).

2-3-2 Choriocarcinoma

It is solid trophoblastic tumor developed primarily in myometrium, or in distant organs and tissues, usually after the removal of total or partial hydatidiform mole, and also infrequently after the abortion or deliveries. They are gestational choriocarcinoma or gestational trophoblastic disease (GTD). Non-gestationalchoriocarcinoma develops from germ cells or other cancer cells in children. Choriocarcioma is constructed of syncytio- and cyto-trophoblasts, and shows no villus pattern at all. Since villus pattern is characteristic sign of invasive mole, and its outcome is less ominous than choriocarcinoma, microscopic studies should be

detailed on whole specimen after hysterectomy. Wide spread distant metastases of choriocarcinoma were common before the introduction of effective chemotherapy in this field. The interval of its diagnosis and metastases was about half to one year. Subsequent frequent spread was the lung. Any organs or tissues were affected after pulmonary metastasis, e.g. skin, subcutaneous tissue, intestine, liver, spleen, kidney, heart, and finally brain. Tumor cells were found also in blood vessels. Every organ is damaged by the trophoblasts and hemorrhage. Patients died from brain metastasis and multiple metastases due to the damage and dysfunction. (Kurjak and Chervenak 2006).

2-3-3 Intervillous Thrombosis

Intervillous thrombosis represents bleeding from fetal vessels. It is characterized as intraplacental areas of hemorrhage with variable appearance dependant on the age of lesion. Fresh lesions are dark red, with aging become brown, yellow and finally white. Intervillous thrombosis may be found in up to 50% of the term placentas. Ultrasound appearance of intervillous thrombosis is anechogenic or hypoechogenic areas in the placenta of variable size. They can be as small as few millimeters up to few centimeters in size and they may extend to subchorionic space or basal plate. The clinical significance of intervillous thrombosis is in the possible presence of fetal maternal hemorrhage. Microscopically, fetal and maternal red blood cells may be present and the incidence is increased in the Rh isoimmunisation. (Kurjak and Chervenak 2006).

2-3-4 Placental Infarcts

Placental infarction results from disruption of blood supply to the placenta. As placenta is completely dependent on the maternal blood supply, there is underlying

problem in maternal blood supply resulting in coagulation necrosis of villi. In the most circumstances infarcts are present on the placental basis, in the close proximity to the basal plate and they are variable in size. Small infarcts can be found in ¼ of the placenta without clinical significance, but they are frequently related to preeclampsia and essential hypertension. (Kurjak and Chervenak 2006).

2-3-5 Placental Abruption

Placetal abruption is the acute separation of the placenta from the uterus prior to delivery of the fetus. The symptoms include pain, uterine tenderness and abdominal pain and it is usually accompanied with vaginal bleeding. Such bleeding, if excessive may cause maternal hypovolemia and shock, while severe forms of abruption result in diminished fetoplacental transfer and consequently may cause fetal death *in utero*. (Kurjak and Chervenak 2006).
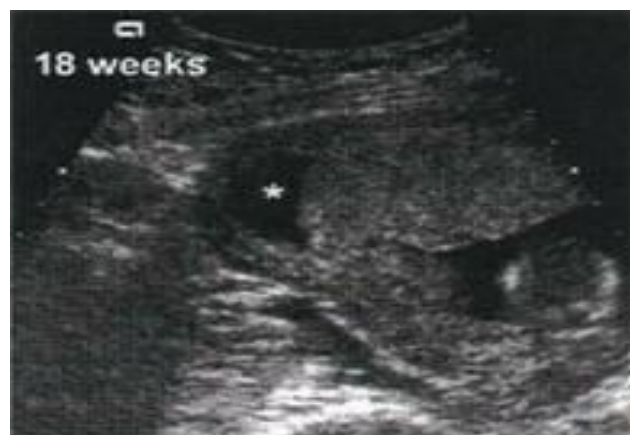

Figure 2-7: Placental abruption: Sonogram of the placenta shows a   cystic area behind the placenta (*) which is the Retroplacental Hematoma. *(Burwin Institute Notes.)*

2-3-6 Placenta Circumvallata

In the normal placenta, the fetal membranes insert into the edge of the placenta. In placenta circumvallata they insert some distance along the fetal surface, leaving an area of placenta free of membranes. The site of insertion is usually marked by a depression in the surface of the placenta. The membrane-free area tends to separate and bleed, but rarely causes more than a little spotting. However, the condition has a high incidence of fetal growth restriction. It is probably responsible for a small proportion of all antepartum hemorrhage. (Chudleigh and Thilaganathan 2004)

Figure 2-8:Circumvallate Placenta : representative image of the placenta shows a thick ridge of tissue extending from the edges of the chorionic surface of the placenta.(Burwin Institute Notes.)

2-3-7 placenta previa

The term "placenta previa" refers to a placenta that is "previous" to the fetus in the birth canal. Bleeding in the second and third trimesters is the hallmark of placenta

previa. This bleeding can be life threatening to the mother and fetus. Accurate diagnosis of placenta previa is vital to improve the outcome for mother and neonate. The placenta previa is classified to:

a. Complete placenta previa: describes the situation in which the internal cervical os is totally covered by the placenta.

b. Marginal placenta previa: denotes placental tissue at the edge of or encroaching on the internal cervical os.

c. Low placenta: is one in which the placenta edge is within 2 cm, but not covering any portion, of the internal cervical os. (Rumack et al 2011).

Figure 2-9: placenta previa: Transabdominal sagittal image shows the placenta

(P) centered over the internal os.(Chie & Levine 2006)

2-3-8 Placenta Accreta

Placenta accrete is defined as the abnormal adherence of part or all of the placenta to the underlying uterine wall. A deficiency of decidua at the implantation

site may result from implantation of the placenta close to or cover the cervix (placenta previa).A similar situation may arise when implantation occurs on scars from a previous cesarean section. Owing to the absence of decidua, the placenta does not separate normally from the underlying uterine wall following parturition, an event that can result in life-threatening bleeding. (Rubin & Farber 1999).

Placenta accreta is sub classified according to the depth that the villi invade into the myometrium:

a. Placenta accretes: refers to the attachment of villi to the myometrium without further invasion.
b. Placenta increta: defines villi invading the underlying myometrium.
c. Placenta percreta: is a condition in which the villi penetrate the full thickness of the uterine wall. (Rubin & Farber 1999).
   The placental villi in all these placental disorders are normal and show no evidence of trophoblastic proliferation. (Rubin & Farber 1999).

Most patients with placenta accreta have a normal pregnancy and delivery. However complications may occur during pregnancy, delivery, or especially in the immediate postpartum state. Bleeding in the third trimester is the most common presenting sign before delivery. Uterine rupture, before, during, or after labor, occurs in 15% of patients with placenta accrete. Substantial fragments of placenta may remain adherent following delivery and are a source of postpartum hemorrhage. Placenta accreta is a serious complication and is associated with a maternal death. (Rubin& Farber 1999).
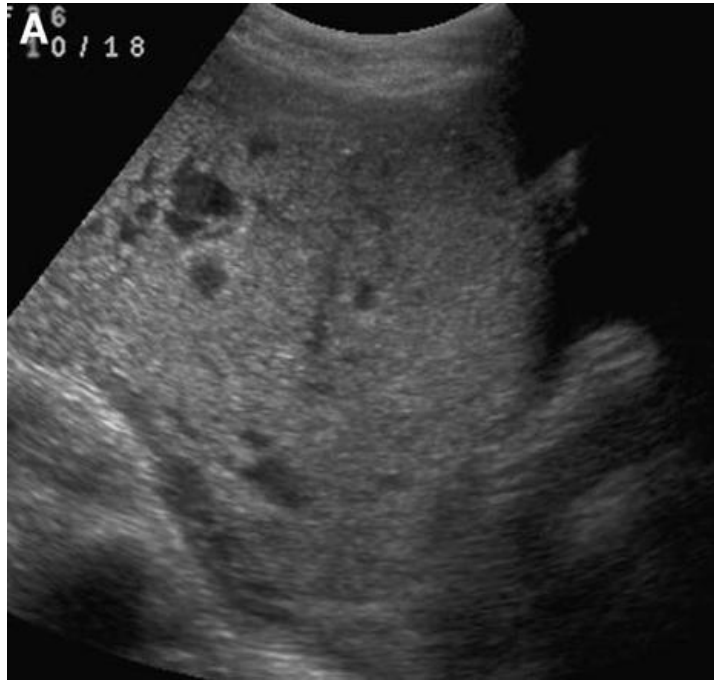
Figure 2-10: Placenta accreta at 26 weeks gestational age. A transabdominal sagittal image shows a thickened placenta with cystic spaces. There is loss of the normal myometrium anteriorly..(Chie & Levine 2006)

2-3-9 Villitis

Is the inflammation of the villi. Infection of the villi results from endometritis or transeplacental passage of organisms delivered by way of the maternal circulation. The process is frequently focal. While the infection cannot be demonstrated in most cases, the microorganisms causing this type of infection include: bacteria, viruses, parasites and protozoa and fungi. The most important consequence of hematogenous placental infection is the establishment of an inflammatory focus, which can then secondarily infect the fetus. Approximately 30% of the villi must be destroyed before perinatal mortality is significantly increased. (Rubin & Farber 1999).

2-3-10 Chorioamnionitis

Chorioamnionitis refers to inflammation of the placental amnion and chorion and the extraplacental membrane. It is usually the result of an ascending infection from the maternal birth canal, commonly owing to premature rupture of the membranes. In this type of infection, the inflammatory process affects primarily the membranes (chorioamnionits) rather than the chorionic villi. Acute chorioamnionitis is important because of its occurrence in 20% of placentas and its clear association with preterm labor, fetal and neonatal infections, and intrauterine hypoxia. The risks of chorioamnionitis to the fetus include:

a. Pneumonia after inhalation of infected amniotic fluid.

b. Skin or eye infections from direct contact with organisms in the fluid.

c. Neonatal gastritis, enteritis, or peritonitis from ingestion of infected fluid.

Major risks to the mother are intrapartum fever, postpartum endometritis, and pelvic sepsis with venous thrombosis. (Rubin & Farber 1999).

2-4 Previous Study

Abu et al (2009) performed a study to investigate the relationship between placental thickness and estimated fetal weight in normal pregnant Nigerian women. They scan six hundred and forty five Nigerian women with singleton pregnancies in the second and third trimesters by ultrasound. Their results showed that both placental thickness and estimated fetal weight increased in fairly linear manner with gestational age. They found a significant positive correlation between placental thickness and estimated fetal weight in the second and third trimesters ($p < 0.05$). Regression analysis yielded linear mathematical relationships between estimated fetal weight and placental thickness in the second and third trimesters, but the marked variations in fetal weights corresponding to particular placental thickness limit the usefulness of this relationship.

Ohagwu et al (2009) investigated the relationship between placental thickness and fetal growth parameters in normal singleton Nigerian fetuses. The pregnancies were in the second and third trimesters, and were not complicated by either maternal or fetal disease. Their study shows that there was a fairly linear increase in placental thickness with gestational age. There was significant positive correlation between placental thickness and, biparietal diameter (BPD) and abdomen circumference (AC) in the second and third trimesters with both parameters having identical relationship with placental thickness. Placental thickness has a strong positive correlation with BPD and AC.

Hammad(2008) performed a study to evaluate Placental thickness in the third trimester, his results showed linear relationship between Placental thickness in mm and gestational age in weeks. He found that Placental thickness increase with the

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fetal age. He concluded that the measurement of the placental thickness is an important parameter for estimating gestational age in normal singleton pregnancies along with other parameters.

Elamin (2012) studied the relationship between Placental thickness and fetal age in Sudanese women, she found that the placental thickness increase with gestational age. She also found that there is significant positive correlation between Placental thickness and LMP, biparietal diameter (BPD), AC and femur length (FL). Her study showed linear regression between Placental thickness and LMP, biparietal diameter (BPD), abdomen circumference (AC) and femur length (FL).

CHAPTER THREE

Materials and Method

3-1 Population of the Study

This descriptive study includes fifty two Sudanese pregnant women in second and third trimesters came to the ultrasound department for regular checkup. The selected women were attending with viable singleton and uncomplicated pregnancies.

Patients with pregnancy induced hypertension (PIH), diabetes mellitus, history of previous intrauterine growth retardation (IUGR), congenital malformation, twin gestation, and placental anomalies were excluded from the study.

3-2 Area and duration of the Study

The study is held in ultrasound department in Omdurman Maternity Hospital in Sudan, from November 2014 to February 2015.

3-3 Equipments

Sonography was carried out on each patient included in the study using ultrasound machine Toshiba-power vision-6000, transabdominal convex transducer with frequency of 3.5 MHz and ultrasound gel.

3-4 Method of Data Collection

In this study, fifty two Sudanese pregnant women scanned by Transabdominal probe in second and third trimesters. After checking that the pregnancy is normal, the fetal weight was estimated by measuring the biparietal diameter (BPD) and abdomen

circumference (AC). The placental thickness was measured in longitudinal section at the point of umbilical cord insertion. Fetal age was estimated by measuring biparietal diameter (BPD). The data was recorded in the data collecting sheet. Other variables including maternity age, gravidity, were also included in the data collecting sheet.

3-5 Data Analysis

After measurements and data collection, the data is analyzed by using Statistical Package for Social Sciences (SPSS).The relation between placental thickness and biparietal diameter (BPD), abdomen circumference (AC), and estimated fetal weight (EFW) is established by Pearson's correlation analysis.

Chapter Four

Results

This study was carried out on 52 pregnant women, from different regions in Sudan, their ages between 17-40 years, were examined by ultrasound. Measurements were taken for placental thickness, biparital diameter (BPD), and abdomen circumference (AC). Estimation of the fetal weight was calculated by ultrasound machine due to biparital diameter (BPD) and abdomen circumference (AC). The following are the results:

Table 4-1 shows descriptive statistics of the maternity age, gravida, fetal age and weight and placental thickness.

	N	Minimum	Maximum	Mean	Std. Deviation
maternity age (years)	52	17	40	26.38	6.001
Gravida	52	1	3	1.63	.841
Biparital Diameter (mm)	52	26.0	96.0	64.229	19.8645
Fetal Age (weeks)	52	14.6	39.7	26.818	7.5568
Abdomen Circumference (mm)	52	81	436	235.44	90.349
Estimated Fetal Weight (gm)	52	91	4680	1497.65	1387.373
Placental Thickness (mm)	52	18	50	32.94	9.206
Valid N (listwise)	52				

Table 4-2: shows the correlation between the placental thickness and biparital Diameter (BPD)

Correlations

		Placental Thickness (mm)	BiParital Diameter (mm)
Placental Thickness (mm)	Pearson Correlation	1	.877(**)
	Sig. (2-tailed)		.000
	N	52	52
BiParital Diameter (mm)	Pearson Correlation	.877(**)	1
	Sig. (2-tailed)	.000	
	N	52	52

** Correlation is significant at the 0.01 level (2-tailed).
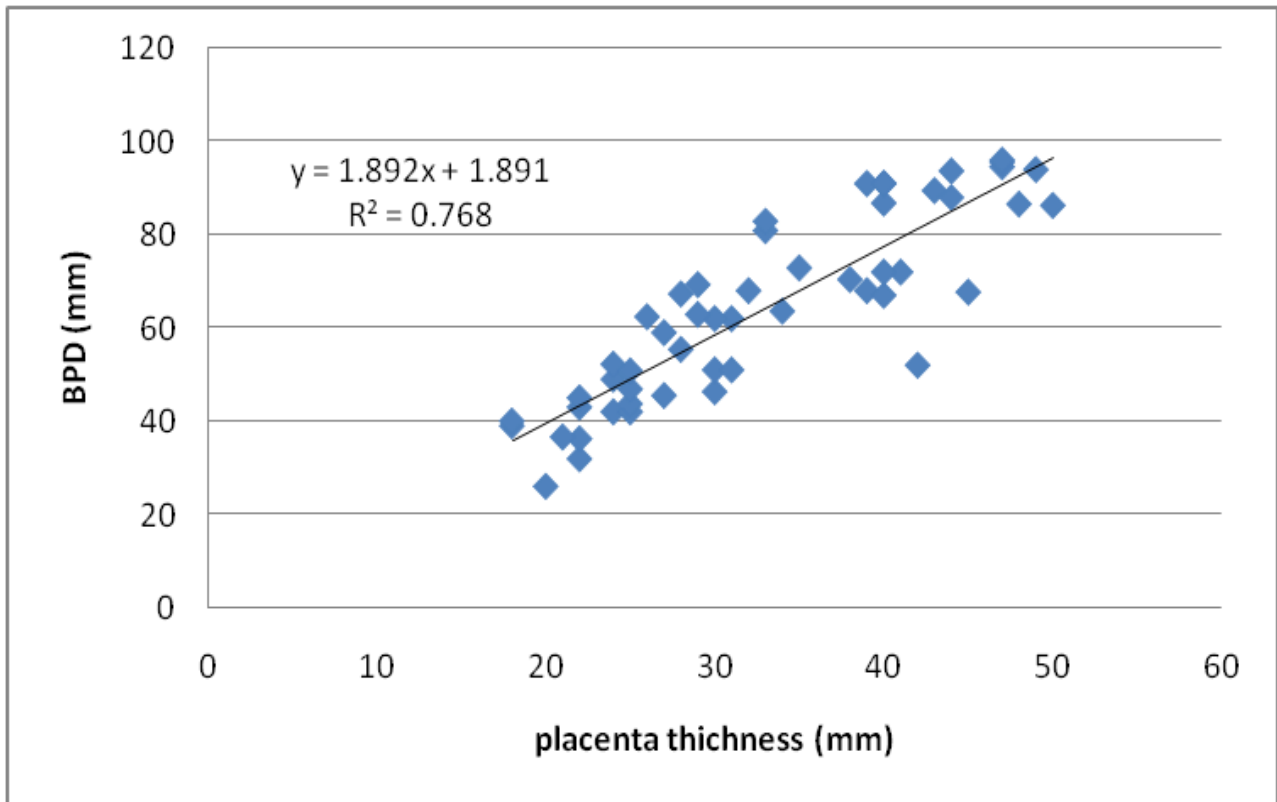
Figure 4-1:   scatter plot shows biparietal diameter (BPD) with placental thickness

Table 4-3: shows correlation between placental thickness and abdomen circumference (AC)

Correlations

		Placental Thickness (mm)	Abdomen Circumference (mm)
Placental Thickness (mm)	Pearson Correlation	1	.873(**)
	Sig. (2-tailed)		.000
	N	52	52
Abdomen Circumference (mm)	Pearson Correlation	.873(**)	1
	Sig. (2-tailed)	.000	
	N	52	52

** Correlation is significant at the 0.01 level (2-tailed).
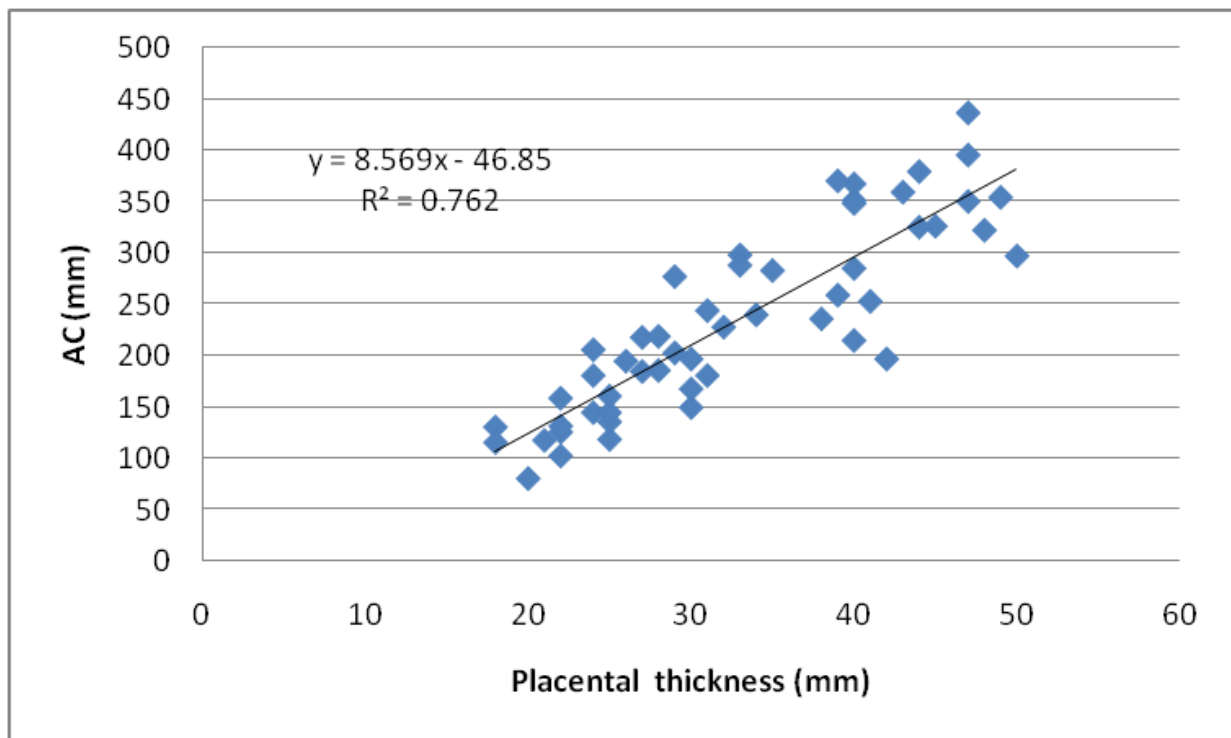
Figure 4-2: scatter plot shows the abdomen circumference (AC)

with placental thickness

Table 4-4: shows correlation between placental thickness and fetal age

		Fetal Age (weeks)	Placental Thickness (mm)
Fetal Age (weeks)	Pearson Correlation	1	.885(**)
	Sig. (2-tailed)		.000
	N	52	52
Placental Thickness (mm)	Pearson Correlation	.885(**)	1
	Sig. (2-tailed)	.000	
	N	52	52

** Correlation is significant at the 0.01 level (2-tailed).
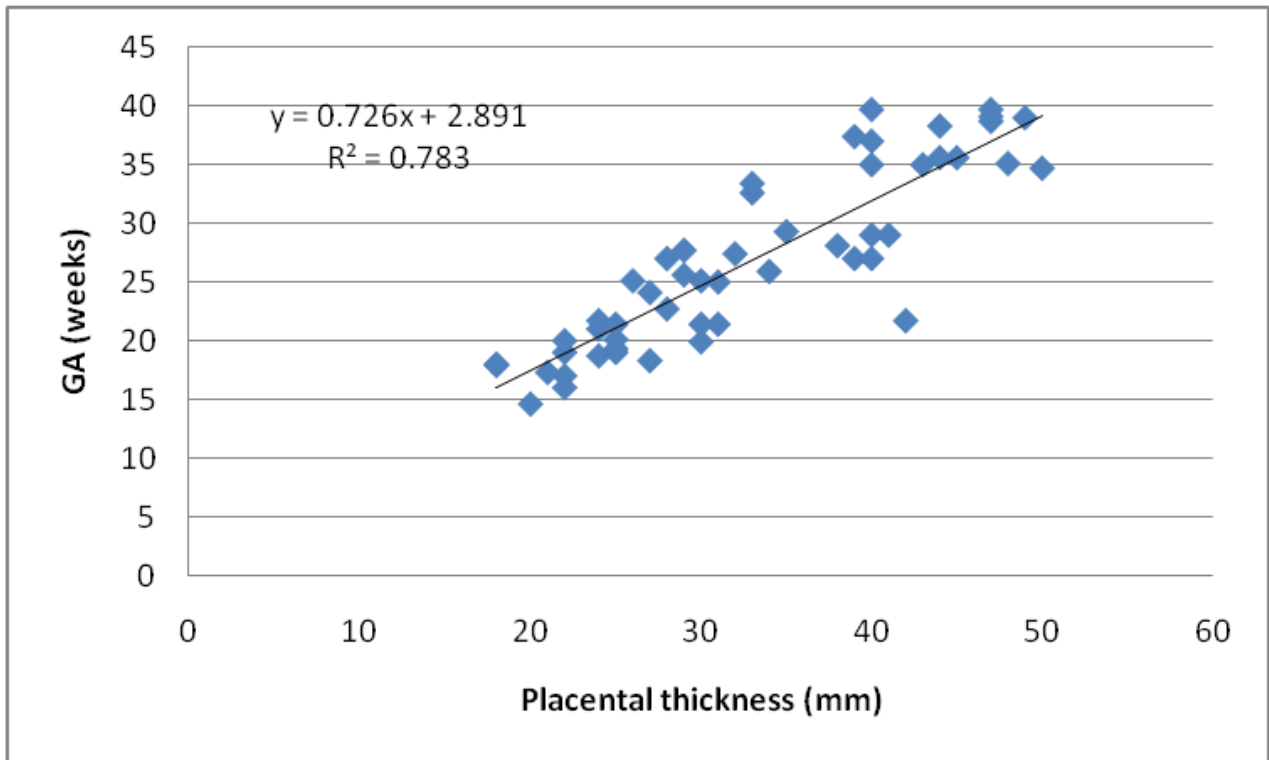
Figure 4-3 scatter plot shows fetal age with placental thickness

Table 4-5: shows correlation between placental thickness and estimated fetal weight

Correlations

		Placental Thickness (mm)	Estimated Fetal Weight (gm)
Placental Thickness (mm)	Pearson Correlation	1	.836(**)
	Sig. (2-tailed)		.000
	N	52	52
Estimated Fetal Weight (gm)	Pearson Correlation	.836(**)	1
	Sig. (2-tailed)	.000	
	N	52	52

** Correlation is significant at the 0.01 level (2-tailed).
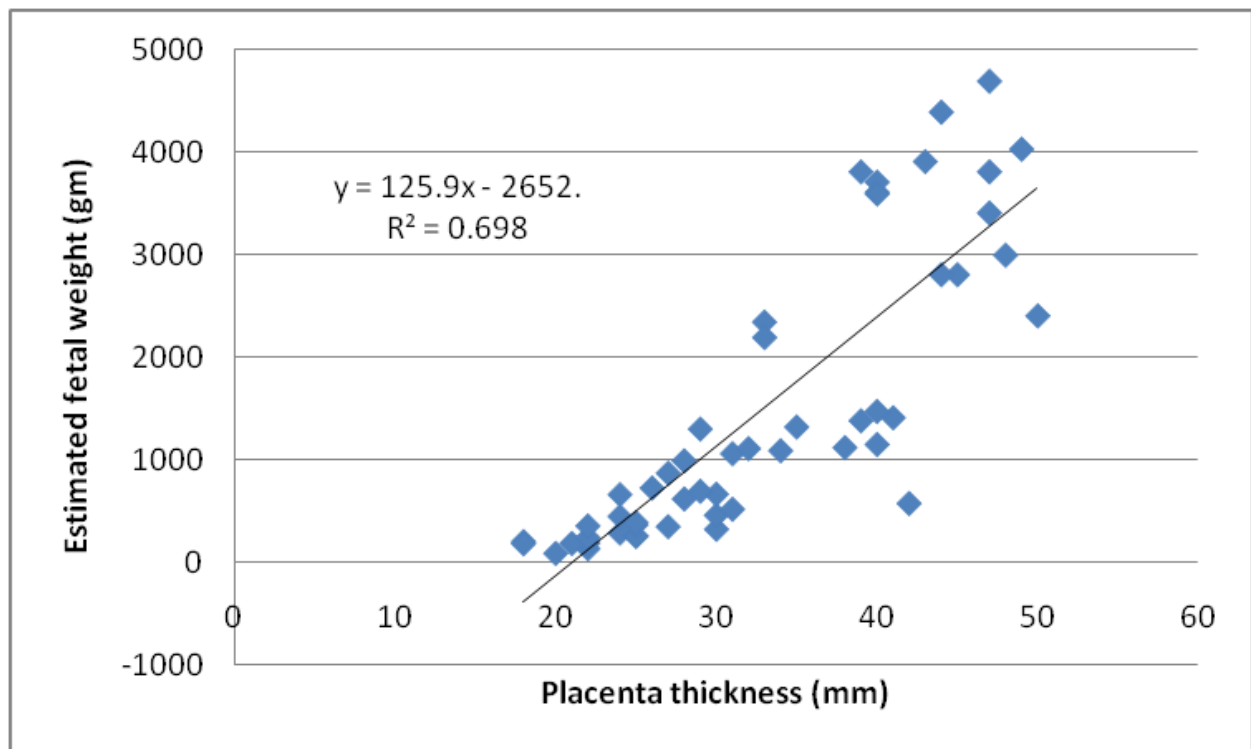
Figure 4-4: scatter plot shows estimated fetal weight with Placental thickness.

The equation shown on the plot:

$$y = 125.9x - 2652.$$
$$R^2 = 0.698$$

CHAPTER FIVE

Discussion, Conclusion and

Recommendations

Discussion

The purpose of this study was to investigate the relationship between the placental thickness and estimated fetal weight in normal Sudanese pregnant women.

The research includes 52 pregnant women. From the collected data, the placental thickness (PT), biparietal diameter (BPD), fetal age, abdomen circumference(AC) and estimated fetal weight (EFW) were extracted. Other variables such as maternity age and gravidity are also included in the study.

The study showed that there is a linear relationship between placental thickness and fetal age as shown in figure 4-3 and there is strong positive correlation between them (r = 0.885) with probability (p = 0.01) (table 4-4). These results establish that there is a firmly fixed increase in placental thickness (PT) with the increase of fetal age, which typically go with those studies done by Ohagwu et al 2009, Hammad 2008 and Elamin 2012, all studies concurred in that the placental thickness increase with the increase of fetal age.

The study found that there is strong positive correlation between Placental thickness (PT) and biparietal diameter (BPD) and abdomen circumference (AC) respectively. In Placental thickness and biparietal diameter(r = 0.877) with (p = 0.01), table 4-2 shows this relation. In Placental thickness and abdomen circumference (r = 0.873) with (p = 0.01) (table 4-3). That means the three parameters increase together

with ongoing increase of the fetal age. Figure 4-1 shows the linear regression between placental thickness and biparietal diameter. The biparietal diameter (BPD) increases by 1.9 mm /each one mm of placenta thickness. Figure 4-2 shows linear regression between placental thickness and abdomen circumference. The abdomen circumference (AC) increases by 8.6 mm/ each one mm of placenta thickness.

The study found that the estimation of fetal weight-which based on BPD and AC- have positive significant correlation with Placental thickness. Both are increasing continuously with fetal age. Table 4-5 shows the positive significant correlation between them with (r = 0.836) and (p = 0.01) and figure 4-4 shows their linear regression. Estimated fetal weight increases by 126 gm / each one mm of placenta thickness. This result is similar to Nigerian study done by Abo et al (2009) in Nigeria. Both studies found positive significant correlation between Placental thickness and estimated fetal weight.

The above result can lead the Placental thickness to be an initial parameter for fetal weight estimation. This help to know the normality of fetal weight or predicting any abnormalities such as intrauterine growth retardation (IUGR).

It has been realized that with the same placental thickness there are different estimations of fetal weight. We think that is due to normal variations between pregnancies and due to small volume of cases included in the study.

The study showed that there is no relation between Placental thickness and maternity age. Also there is no relation between placental thickness and the gravidity.

Conclusion

The placenta is an important organ that connects the developing fetus to the uterine wall to allow nutrients uptake, waste elimination and gas exchange via the mother blood.

Estimation of the fetal weight is very important during obstetric ultrasound; it can influence obstetric management decisions concerning the timing and route of delivery

Method of the study is based on estimation of fetal weight by measuring the abdomen circumference (AC) and biparietal diameter (BPD), and measuring the placental thickness and studies the correlation between them using Statistical Package for Social Sciences (SPSS).

The study found that there is positive significant correlation between placental thickness and fetal age. Also the study showed that there is positive significant correlation between placental thickness and biparietal diameter (BPD) and abdomen circumference (AC) respectively. Study showed that there is positive significant correlation between placental thickness and estimated fetal weight and both are increasing with fetal age. The study found that the fetal weight increases by 126 gm / each one mm of placenta thickness. Thus placental thickness can be a promising parameter in obstetric ultrasound. The study found that with the same placental thickness there are different estimations of fetal weight, however, the normality of the fetal weight and fetal development can be followed by measuring the Placental thickness.

Recommendations

1) It is suggested that sonologist use the placental thickness as a parameter to follow the fetal development and wellbeing in addition to other parameters.

2) It is recommended another research that follows the fetal weight by placental thickness measuring, and weights the same fetus after delivery and study the relationship between them to confirm the role of placental thickness in assessing fetal weight.

3) It is recommended another study to correlate the blood supply to placenta with fetal weight using Doppler Ultrasound machines.

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Appendix 1

The Images of the Researcch

Image 1: Transabdominal ultrasound image for 37 years pregnant woman shows placental thickness = 24.8 mm, gestational age = 25 weeks.

Image 2: Transabdominal ultrasound image for 30 years pregnant woman shows placental thickness = 18 mm, gestational age = 16 weeks and 5 days.
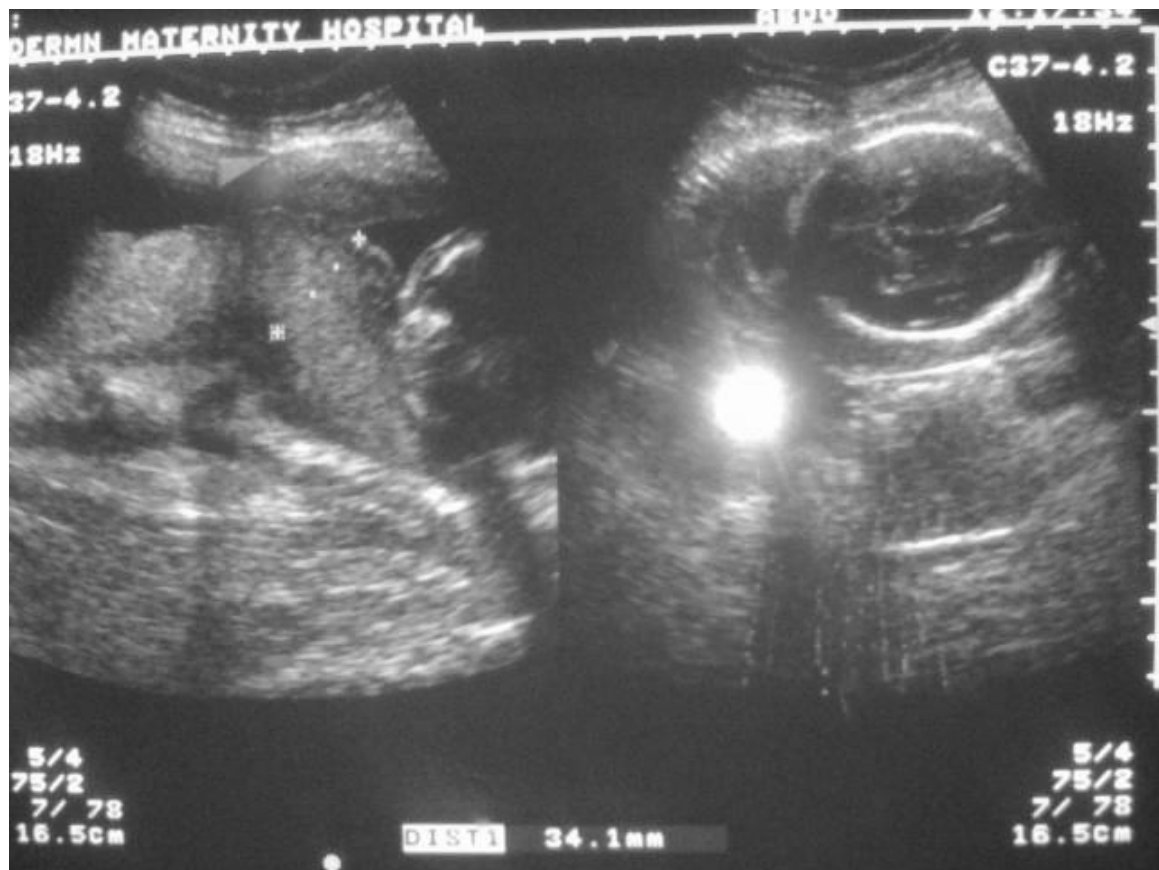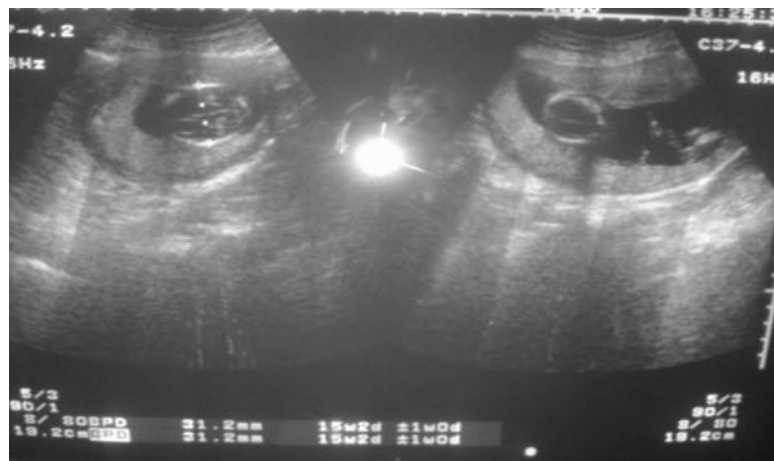
Image 3: Transabdominal ultrasound image for 25 years pregnant woman shows placental thickness = 34.1 mm, gestational age = 31 weeks + 4 days.

Image 4: Transabdominal ultrasound image for 21 years pregnant woman shows placental thickness = 21 mm, gestational age = 22 weeks and 5 days.
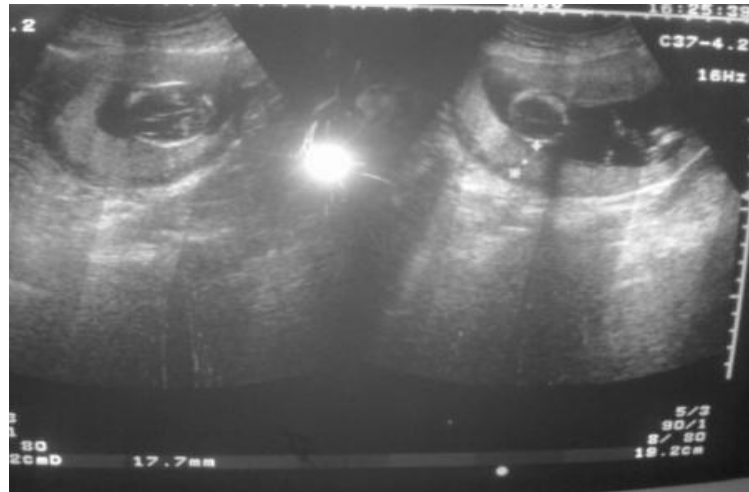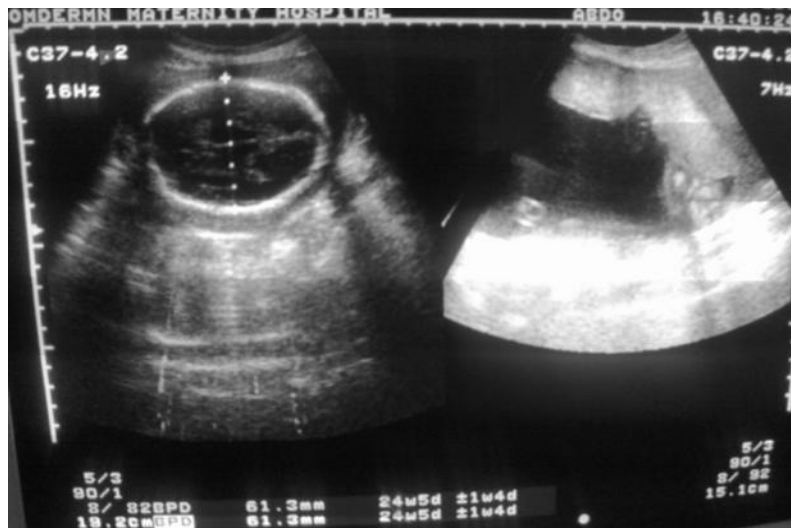
A

B

Image 5 (A, B): Transabdominal ultrasound images for 19 years pregnant woman shows placental thickness = 17.7 mm, gestational age = 15 weeks and 2 days.

A

B

Image 6 (A, B): Transabdominal ultrasound images for 26 years pregnant woman shows placental thickness = 22.4 mm, gestational age = 24 weeks and 5 days.
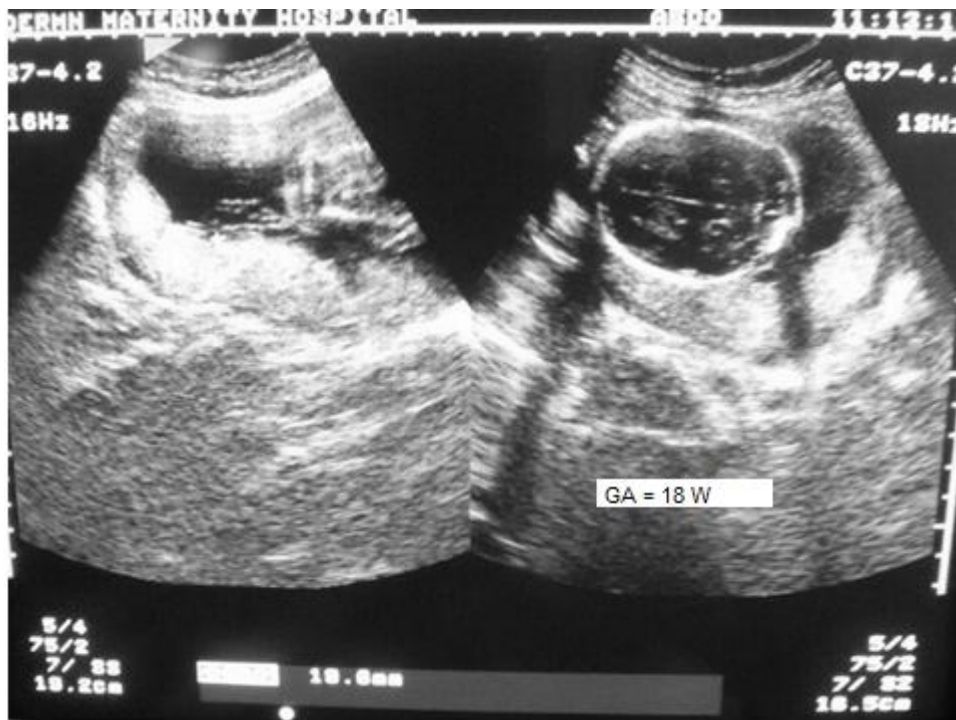
Image 7: Transabdominal ultrasound image for 20 years pregnant woman shows placental thickness = 18.6mm, gestational age = 18 weeks.

Image 8: Transabdominal ultrasound image for 22 years pregnant woman shows placental thickness = 25 mm, gestational age = 25 weeks + 5 days.

Image 9: Transabdominal ultrasound image for 37 years pregnant woman shows placental thickness = 37.7 mm, gestational age = 36 weeks + 1 day.

Image 10: Transabdominal ultrasound image for 29 years pregnant woman shows placental thickness = 19.4 mm, gestational age = 20 weeks + 4 days.

Image 11: Transabdominal ultrasound image for 27 years pregnant woman shows placental thickness = 24.7 mm, gestational age = 24 weeks + 4 days.
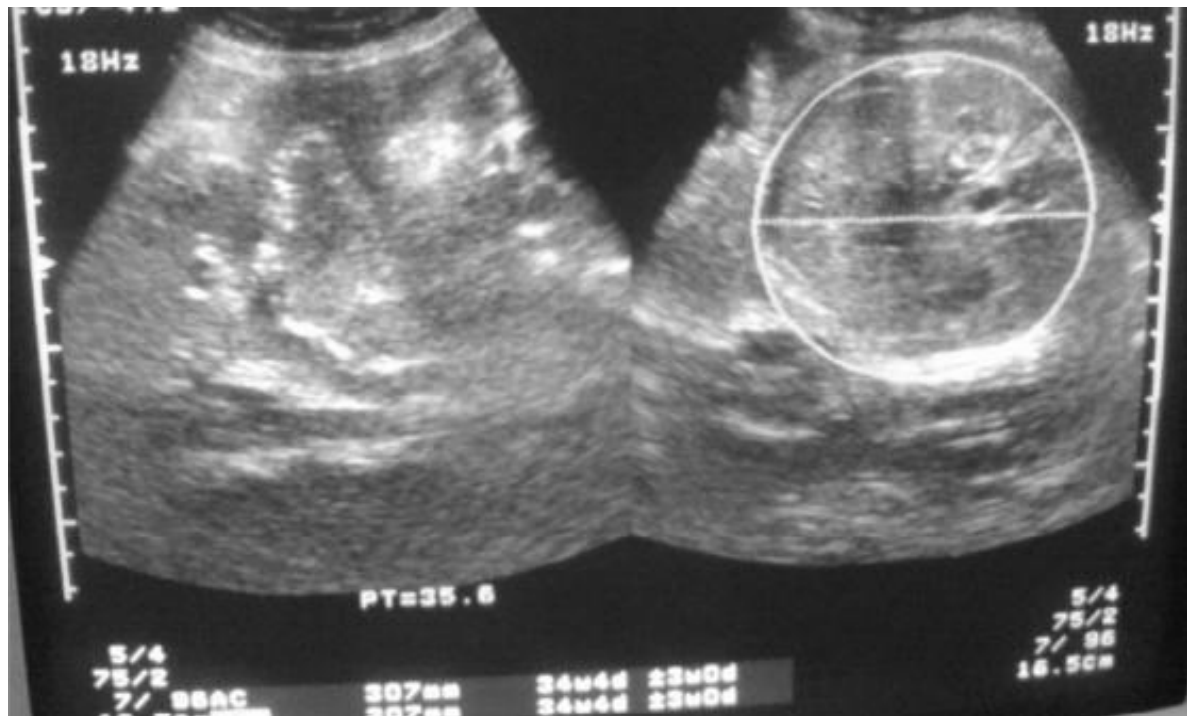
Image 12: Transabdominal ultrasound image for 18 years pregnant woman shows placental thickness = 35.6 mm, gestational age = 34 weeks and 4 days.

Image 13: Transabdominal ultrasound image for 19 years pregnant woman shows placental thickness = 30.9 mm, gestational age = 31weeks and 3 days.
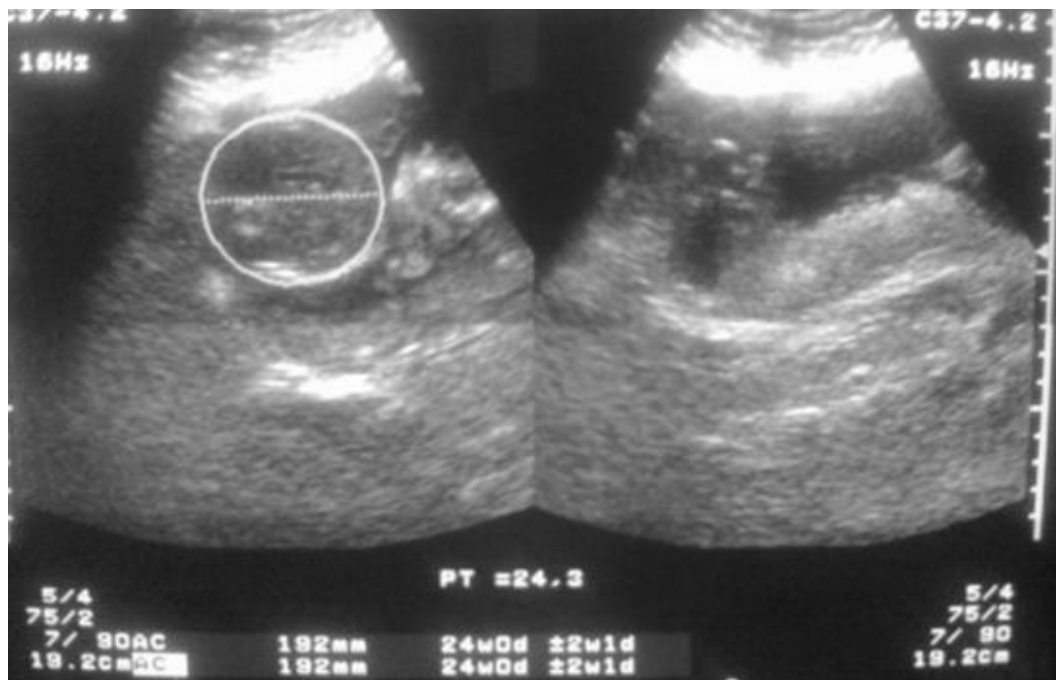
Image 14: Transabdominal ultrasound image for 32 years pregnant woman shows placental thickness = 24.3 mm, gestational age =24.weeks.

Image 15: Transabdominal ultrasound image for 24 years pregnant woman shows placental thickness = 18.5 mm, gestational age =20.weeks.

Appendix 2

Data collection sheet

General information :

(1) Maternity Age: [   ] years

(2) Gravida: 1 [   ] ≥ 3 [   ] ≥ 5 [   ]

Ultrasound   Findings:

(1) BPD [   ] mm   ………………GA [   ] Weeks.

(2) AC [   ] mm   ………………GA [   ] Weeks.

(3) EFW [   ] gm.

(4) Placenta thickness [   ] mm.