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Chorioangioma of Placenta

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Abstract

The human placenta is a villous hemochorial structure. It is attached to the uterine wall and establishes connection between the mother and the fetus through the umbilical cord and thus plays a critical role in maternal fetal transfer. It is developed from two sources: fetal chorion frondosum and maternal decidua basalis. Various abnormal conditions have been reported with the placenta and the placental chorioangioma is one of them. Chorioangioma of placenta is the commonest benign tumor of the placenta. It consists of a benign angioma arising from the chorionic tissue. It has been found to be associated with many serious complications such as nonimmune hydrops, congenital abnormalities, hemolytic anemia, polyhydramnios, IUGR, and IUFD.

Keywords: chorioangioma, fetus, placenta, polyhydramnios, villi

1. Introduction

The human placenta is discoid in shape and is a villous hemochorial structure [1, 2]. The placenta is attached to the uterine wall and establishes connection between the mother and the fetus through the umbilical cord and thus plays a critical role in maternal fetal transfer [2]. It has a complex synthetic capacity and plays a role in the immunologic acceptance of fetal allograft [2].

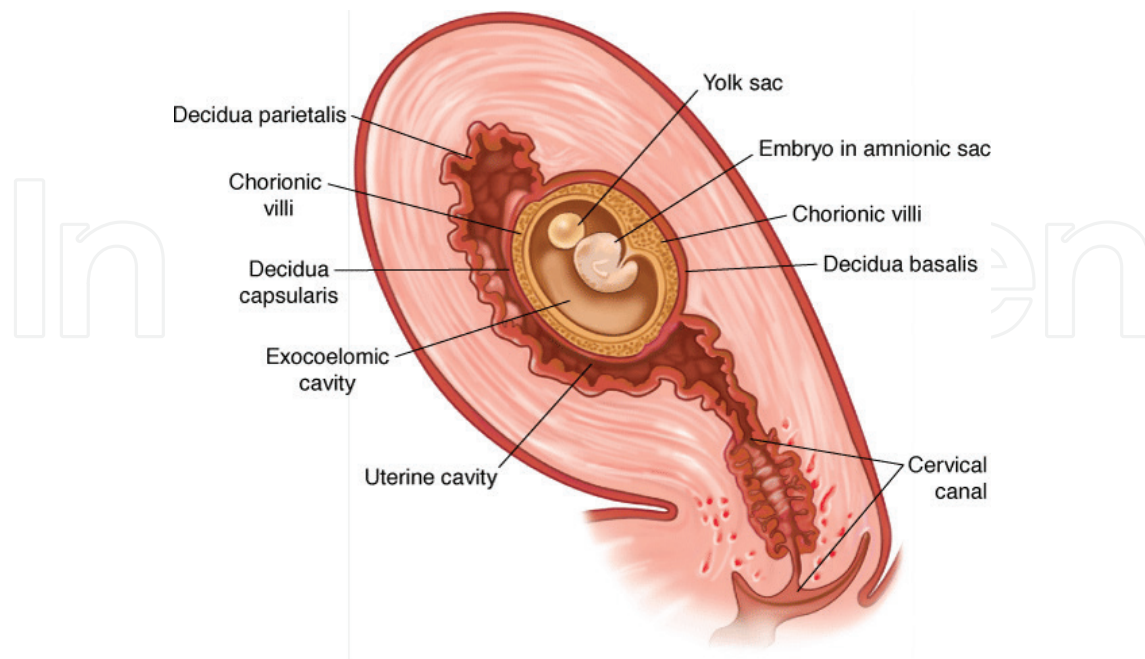
2. Development

The placenta is developed from two sources. The principal component is fetal which develops from the chorion frondosum, and the maternal component consists of decidua basalis (**Figure 1**) [2].

The fertilized ovum converts into a morula and further differentiates into a blastocyst. The outer layer of the blastocyst proliferates to form the primary trophoblastic cell mass which infiltrates the endometrial lining. By the 7th post-ovulatory day, the trophoblast differentiates into two layers: an inner layer of clear mononuclear cells with well-defined limiting membranes called cytotrophoblast and the outer layer of multinucleated cells with no intercellular membrane called syncytiotrophoblasts [1]. By 10th to 13th post-ovulatory days, a series of intercommunicating spaces or lacunae develop in the rapidly enlarging and dividing trophoblastic cell mass.

The lacunae become confluent, and as the trophoblastic cell erodes the maternal vessels, they become filled with blood to form intervillous spaces. Between the lacunae spaces, there are columns having a central core of cytotrophoblasts surrounded by syncytiotrophoblasts. These form the framework for the development of villi later. From these pillars, branching sprouts appear. Those columns extend as far as the decidua and a mesenchymal core develops in them to form extraembryonic mesenchyme, which forms the villus vessels. In due course, these vessels establish continuity with those developing from the body stalk and inner chorionic mesenchyme. The distal part of the columns is not invaded by the mesenchyme but only serves to anchor it to the basal plate [1, 3]. These cells proliferate and spread laterally separating the syncytiotrophoblasts into two layers, the definitive syncytium on the fetal aspect and the peripheral syncytium on the decidual side which eventually degenerates and is replaced by a fibrinoid material and is known as Nitabuch's layer.

With deeper blastocyst invasion into the decidua, the extravillous cytotrophoblasts give rise to solid primary villi composed of a cytotrophoblast core covered by syncytium. The most deeply implanted portion of these villi forms placenta (**Figure 2**). Beginning on the 12th day



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Figure 1. Decidual structure differentiating into decidua basalis, capsularis, and parietalis.

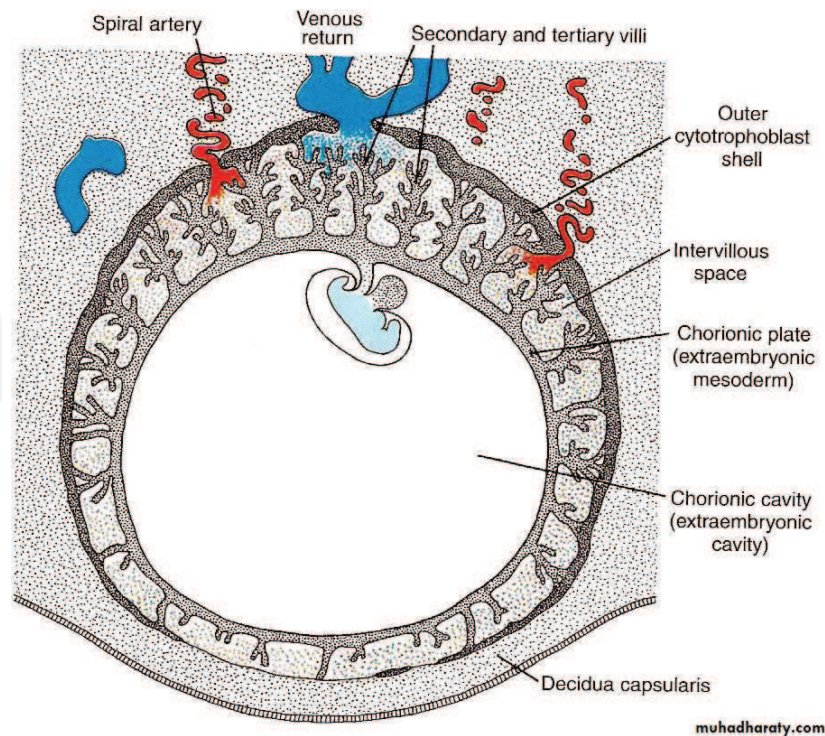


Figure 2. Development of placenta.

after fertilization, chorionic villi can first be distinguished and form secondary villi. After angiogenesis begins in the mesenchymal cores, it results into tertiary villi.

By approximately the 17th day, fetal blood vessels are functional, and a placental circulation is established. The placenta is a vascularized villus structure by the 21st day. The fetal-placental circulation is completed when embryonic blood vessels are connected with chorionic vessels. Groups of cytotrophoblasts also grow into the lumen of the spiral arteries extending as far as the decidual myometrial junction. These cells destroy the muscular and the elastic layer of the vessel wall and get replaced by a fibrinoid material which is derived from the maternal blood and proteins secreted by the trophoblastic cells. This primary invasion dilates the spiral arteriolar wall and thus augments blood flow to the placenta [1, 3]. There is a secondary invasion of trophoblast between 12 and 16 weeks extending up to radial arteries within the myometrium. Thus, spiral arteries are converted to large bore uteroplacental arteries. The net effect is funneling of the arteries that reduce the pressure of the blood to 70–80 mm Hg before it reaches the intervillous space. It thus increases the blood flow.

The placental septa appear by 12 weeks protruding into the intervillous spaces from the basal plate and divide the placenta into 15–20 lobes. Until the end of the 16th week, the placenta grows both in thickness and circumference due to growth of the chorionic villi with accompanying expansion of the intervillous space and with continuous arborization and formation of fresh villi [2].

In the first trimester, the villi are large and have a mantle of trophoblasts consisting of an inner layer of cytotrophoblasts and an outer layer of syncytiotrophoblasts with the stroma of small fetal vessels. During the second trimester, the villi are smaller, the mantle is less regular and the cytotrophoblasts less numerous, and the stroma with more collagen. The fetal vessels

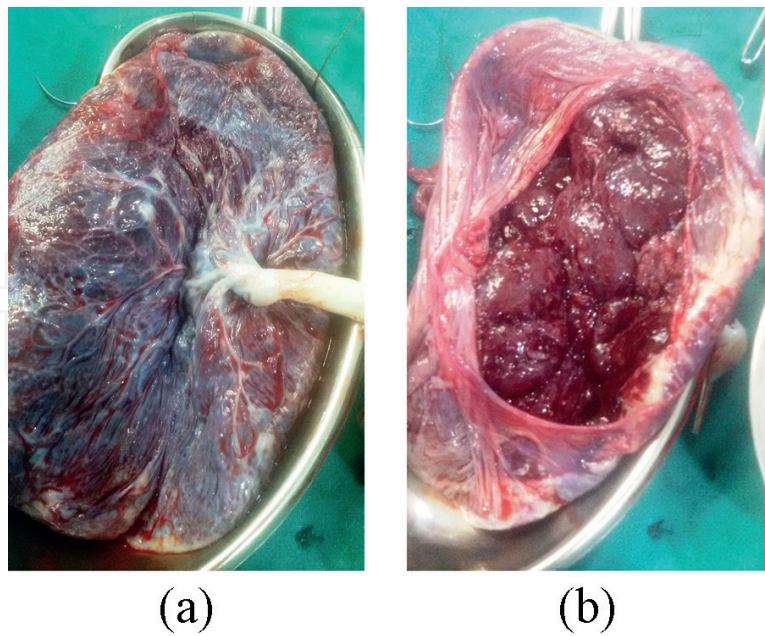


Figure 3. Placenta at term.

become larger and more toward the periphery of the villus. In the third trimester, the villi are much smaller in diameter, and the cytotrophoblasts are irregular and thinned out. The fetal vessels are dilated and lie just below the thinned out trophoblasts.

The placenta, at term, is almost a circular disc with a diameter of 15–20 cm and a thickness of about 3 cm at its center [2]. It feels spongy and weighs about 500 g, the proportion to the weight of the baby being roughly 1: 6 at term and occupies about 30% of the uterine wall. It presents two surfaces, fetal and maternal, and a peripheral margin (**Figure 3**) [2].

- The fetal surface is covered by the smooth and glistening amnion with the umbilical cord attached at or near its center [2].
- The maternal surface is rough and spongy. It consists of 15–20 lobes or cotyledons which are limited by fissures. Each fissure is occupied by the decidual septum which is derived from the basal plate [2].

The placenta consists of two plates. The chorionic plate lies internally. It is lined by the amniotic membrane. The umbilical cord is attached to this plate. The basal plate lies to the maternal aspect. Between the two plates lies the intervillous space containing the stem villi with their branches, the space being filled with maternal blood. A mature placenta has a volume of about 500 mL of blood, 350 mL being occupied in the villi system and 150 mL lying in the intervillous space [2].

3. Functions

1. Transfer of nutrients and waste products between the mother and the fetus. In this respect, it attributes to the following functions:

- Respiratory
 - Excretory
 - Nutritive
2. Endocrine function: placenta is an endocrine gland. It produces both steroid and peptide hormones (like progesterone, estriol, human chorionic gonadotropin, and human placental lactogen) to maintain pregnancy and support fetal growth.
 3. Barrier function: placenta acts as a protective mechanism.
 4. Immunological function: maternal antibodies are taken into the syncytiotrophoblasts by pinocytosis and subsequently transferred to fetal capillaries and thus fetus acquires passive immunity (**Figure 4**).

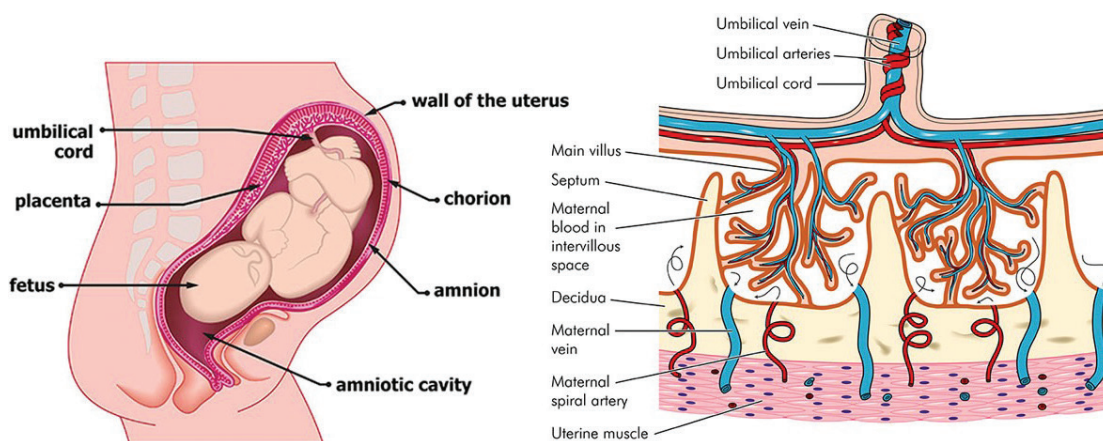


Figure 4. Blood supply of placenta.

4. Abnormalities of placenta

4.1. Placenta succenturiata

The accessory lobe is developed from the activated villi on the chorionic leave, may be placed at varying distances from the main placental margin. A leash of vessels connecting the main to the small lobe traverses through the membranes (**Figure 5**). In cases of absence of communicating blood vessels, it is called placenta spuria. The incidence of placenta succenturiata is about 3%. If the succenturiate lobe is retained, the following birth of the placenta may lead to:

- a. Postpartum hemorrhage which may be primary or secondary
- b. Subinvolution
- c. Uterine sepsis
- d. Polyp formation

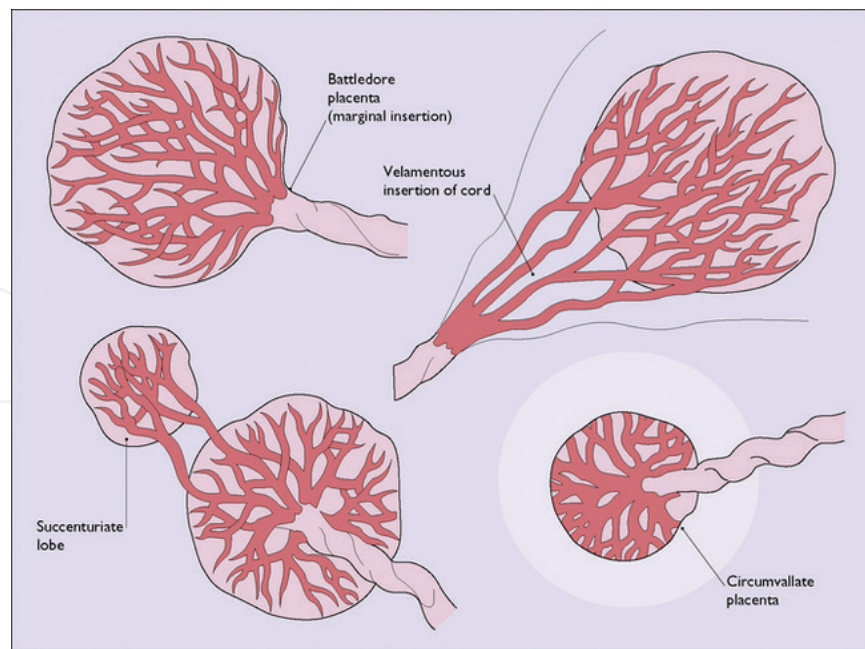


Figure 5. Placental abnormalities.

Treatment: Whenever the diagnosis of missing lobe is made, exploration of the uterus and removal of the lobe under general anesthesia is to be done.

4.2. Velamentous placenta

Normally, the umbilical cord inserts into the middle of the placenta as it develops. In velamentous cord insertion, the umbilical cord inserts into the fetal membranes (chorioamniotic membranes) and then travels within the membranes to the placenta (between the amnion and the chorion). The exposed vessels are not protected by Wharton's jelly and hence are vulnerable to rupture (**Figure 5**). Rupture is especially likely if the vessels are near the cervix, in which case they may rupture in early labor, likely resulting in a stillbirth. Once it is diagnosed, baby should be delivered by cesarean section.

4.3. Battledore placenta

Umbilical cord may be attached in the center, off center, on the edge, or in the membranes of the placenta. Battledore placenta is a placenta in which the umbilical cord is attached at the placental margin. The shortest distance between the cord insertion and the placental edge is within 2 cm. The incidence of the battledore placenta is 7–9% in singleton pregnancies and 24–33% in twin pregnancies [4, 5]. Complications associated with the battledore placenta are:

- a. fetal distress
- b. intrauterine growth restriction

- c. preterm labor
- d. decreased birth weight of baby and placenta

4.4. Placenta extrachorialis

It can be:

1. Circumvallate placenta: the fetal surface is divided into a central depressed zone surrounded by a thickened white ring which is usually complete. The ring is situated at varying distances from the margin of the placenta and is composed of a double fold of amnion and chorion with degenerated decidua (vera) and fibrin in between. Vessels radiate from the cord insertion as far as the ring and then disappear from view.
2. Placenta marginata: a thin fibrous ring is present at the margin of the chorionic plate where the fetal vessels appear to terminate.

There is increased chance of:

- a. Abortion
- b. Hydrorrhea gravidarum
- c. Antepartum hemorrhage
- d. Growth retardation of the baby
- e. Preterm delivery
- f. Retained placenta or membranes

4.5. Morbidly adherent placenta

Morbidly adherent placenta, which includes placenta accreta, increta, and percreta, implies an abnormal implantation of the placenta into the uterine wall (**Figure 6**). The incidence of placenta accreta has increased significantly over the past several decades, with the main risk factors including prior cesarean section and placental previa. Sonographic markers of placenta accreta can be present as early as the first trimester and include a low uterine implantation of a gestational sac, multiple vascular lacunae within the placenta, loss of the normal hypoechoic retroplacental zone, and abnormality of the uterine serosa-bladder interface, among others.

- a. Placenta accreta is an extremely rare form in which the placenta is directly anchored to the myometrium partially or completely without any intervening decidua. The probable cause is due to the absence of decidua basalis and poor development of the fibrinoid layer.
- b. Placenta increta: The placenta invades whole thickness of myometrium.
- c. Placenta percreta: The placenta penetrates whole of the myometrium and may reach up to the peritoneum or bladder.

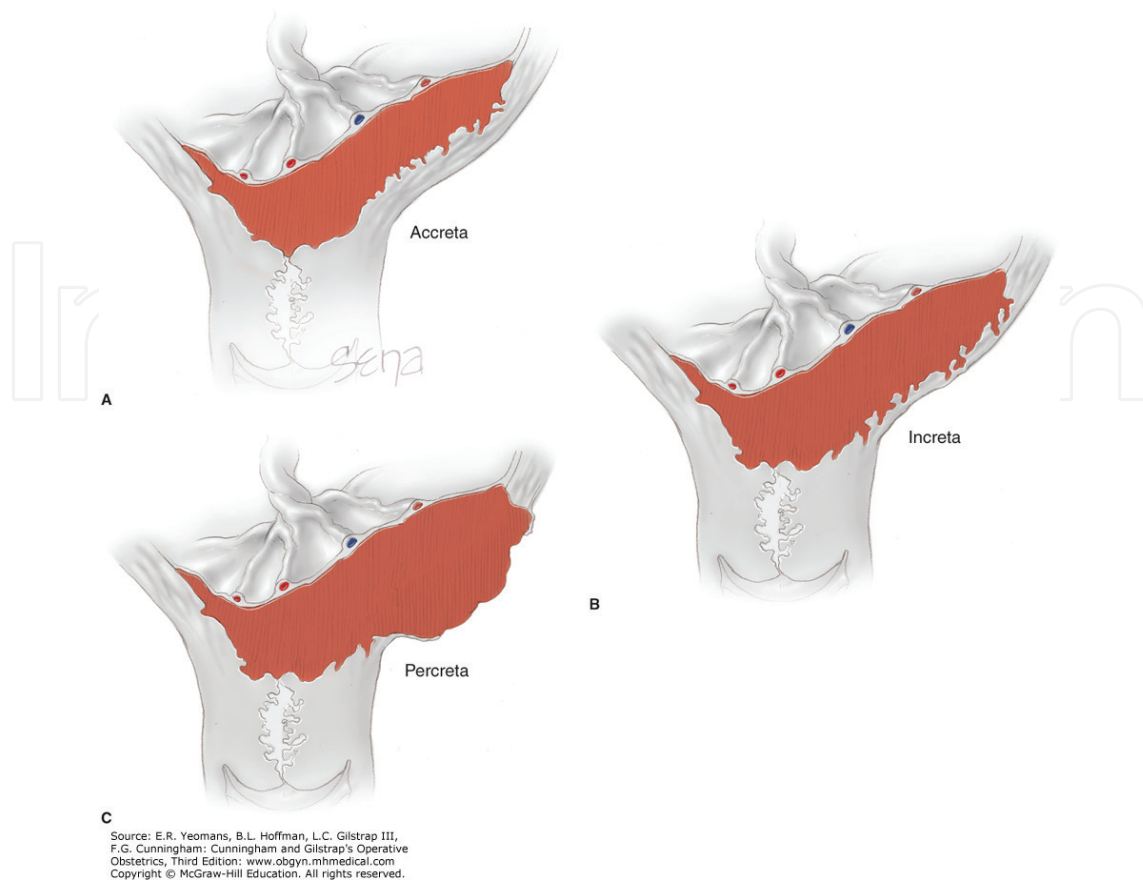


Figure 6. Morbidly adherent placenta (A: Accrete, B: Increta, C: Percreta).

4.6. Gestational trophoblastic disease/neoplasm

Gestational trophoblastic disease is divided into molar and nonmolar tumors. Nonmolar tumors are grouped as gestational trophoblastic neoplasia. It is classified as:

- a. Hydatidiform mole
 - i. Complete
 - ii. Partial
- b. Gestational trophoblastic neoplasia
- c. Invasive mole
- d. Choriocarcinoma
- e. Placental site trophoblastic tumor
- f. Epithelioid trophoblastic tumor

4.7. Chorioangioma of placenta

Placental chorioangioma is the commonest benign tumor of the placenta. Its incidence is around 1% when examined microscopically and is seen more frequently in multiple

pregnancies and in female babies [6]. Chorioangiomas that are clinically evident are less common with an incidence between 1:3500 and 1:9000 births [6]. It is believed to arise by 16th day of fertilization, although there is no documentation of the tumor in the first trimester [7]. In the majority of cases, it is small or microscopic and of no clinical significance. If it increases in size >5 cm, then it may be associated with serious maternal and fetal complications (**Figure 7**) [6].

The pathogenesis of these neoplasms is controversial; however, they can originate from any part of the placenta excluding the trophoblastic tissues [8]. Three histological patterns of chorioangiomas have been described: angiomatous, cellular, and degenerate [9];

- The angiomatous is the most common, with numerous small areas of endothelial tissue, capillaries, and blood vessels surrounded by placental stroma.
- The cellular pattern has abundant endothelial cells within a loose stroma.
- The degenerate pattern has calcification, necrosis, or hyalinization.

These lesions are sometimes classified as placental hamartomas rather than true neoplasia [10]. There is no malignant potential.

Large tumors probably act as arteriovenous shunts and cause complications. Maternal complications are preeclampsia, preterm labor, placental abruption, polyhydramnios, and postpartum hemorrhage [11]. The correlation of chorioangioma with hydramnios and preterm delivery is found to be significant among the various reported clinical complications. Fetal congestive heart failure may develop because of the increased blood flow through the low resistance vascular channels in the chorioangioma acting as an arteriovenous shunt. Other associated fetal complications are nonimmune hydrops, fetal demise, hemolytic anemia, congenital anomalies, fetal thrombocytopenia, cardiomegaly, intrauterine growth restriction, and neonatal death [12].



Figure 7. Chorioangioma of placenta.

Antenatal ultrasound examination has made diagnosis and follow up possible before delivery. In the present case, the placental tumor was not diagnosed in the ultrasound documentation rather polyhydramnios was reported. Doppler ultrasound examination is the gold standard in primary diagnosis of hemangioma. But unfortunately, we could not conduct Doppler USG in the present case as delivery was imminent. Magnetic resonance imaging (MRI) is used only in suspicious cases, while the computed tomography (CT) technique has a limited role in the diagnosis of the placental angioma, mainly because of the high radiation risk and poor tissue differentiation.

Chorioangioma with complications before fetal viability requires interventions. Alcohol injection, laser coagulation of feeding vessels, and microcoil embolization of the feeding vessels are described for women with fetal complications like hydrops [13, 14]. Large chorioangioma associated with polyhydramnios leads to high perinatal morbidity and mortality. Polyhydramnios is treated with therapeutic amniocentesis and maternal indomethacin therapy [12]. Steroid administration for acceleration of fetal lung maturity before 34 weeks is indicated. If complications appear late in pregnancy, delivery is the choice. A recent literature review concluded that further studies are needed to refine the appropriate selection criteria that will justify the risk of invasive in utero therapy for chorioangiomas [15].

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