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Abnormalities of the Placenta

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Abstract

The placenta is considered an important organ that evolves with the implantation of the blastocyst throughout the pregnancy. The placenta has an essential role in functions such as nutrition, excretion, and immunologic and endocrine function. The normal placenta is a round- or oval-shaped organ that attaches to the uterine wall and has roughly 22 cm in diameter and a thickness of about 2–2.5 cm and weighs about one sixth of the fetal birth weight. Thus, a normal development of the placenta is important for an uneventful embryonic and fetal development. Consequently, the placenta abnormalities can range from structural anomalies, to function disorders, to site of implantation abnormalities.

Keywords: placenta, abnormalities, percreta, praevia, choriocarcinoma

1. Introduction

The placenta is a crucial feto-maternal organ with both embryonic (chorion frondosum) and maternal (decidua basalis) components. The development of the placenta begins with the implantation of the blastocyst into the maternal uterus, and it evolves throughout the pregnancy. At the end of the first trimester of pregnancy, the maternal blood supply to the placenta is complete. The placenta has numerous and complex, developmentally essential functions such as nutrition, excretion, and immunologic and endocrine function. The normal placenta is a round- or oval-shaped organ that attaches to the uterine wall and has roughly 22 cm in diameter. The placenta thickness is about 2–2.5 cm and weighs about one sixth of the fetal birth weight [1]. Thus, a normal development of the placenta is important for an uneventful

embryonic and fetal development. Consequently, the placenta abnormalities can range from structural anomalies, to function disorders, to site of implantation abnormalities [1].

2. Placenta accreta, placenta increta, and placenta percreta

Abnormal placental implantation (accreta, incretak, and percreta) is described using a general clinical term, respectively, morbidly adherent placenta (MAP) [2] or "abnormal invasive placenta" (AIP). If not diagnosed before delivery, MAP can lead to catastrophic postpartum hemorrhage, with life-threatening complications. Risk factors include increased maternal age, previous Cesarean delivery or myomectomy, multiparity, and previous intrauterine maneuvers (such as hysteroscopy and multiple dilatation and curettage [3]). The reported incidence ranges from 1:2500–1:7000 pregnancy in 2007 [4] to 1:533 deliveries in 2017 [3]. When the placental villi attach to the myometrium rather than the decidua, it is called placenta accreta; when the chorionic villi penetrate the myometrium, it is called placenta increta (e.g., **Figure 1**), whereas placenta percreta extends into the uterine serosa or adjacent organs (e.g., **Figure 2**). Placenta increta and placenta percreta are rare disorders, which represent <20% of the cases of placenta accreta [5]. These varieties can lead to more severe maternal complications (60% maternal morbidity [6], 7–10% maternal mortality [7]). The most important measure in decreasing these potentially fatal complications is the prenatal ultrasound diagnosis. In many cases, the patient's history is highly relevant. The key feature for early first-trimester diagnosis of MAP is an abnormal neovascularization in the ill-defined placental-myometrial junction detected in a color or power Doppler (2D or 3D) image [8], similar to the flow observed in an invasive mole, arteriovenous malformation, or retained products of conception. Other aspects can include focal or diffuse irregular lacunar lakes with turbulent flow typified by a high velocity (PSV, >15 cm/s) [9]. A higher number of lakes increase the risk of a presenting placenta accreta. The complete loss or disruption of the echolucent myometrial zone between the placenta and bladder is highly suggestive for MAP. When using color Doppler examination, the sensitivity and specificity of
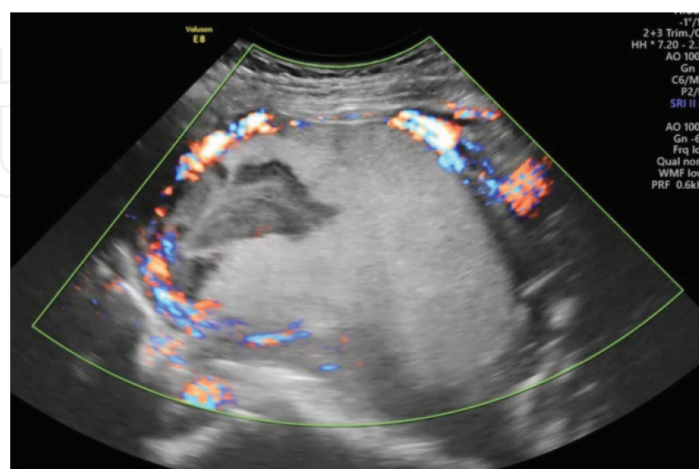

Figure 1. Ultrasound color Doppler image of a case of placenta increta diagnosed in the early second trimester of pregnancy, associated with fetal demise. The surgical termination of pregnancy was performed under laparoscopic guidance, with no complications.
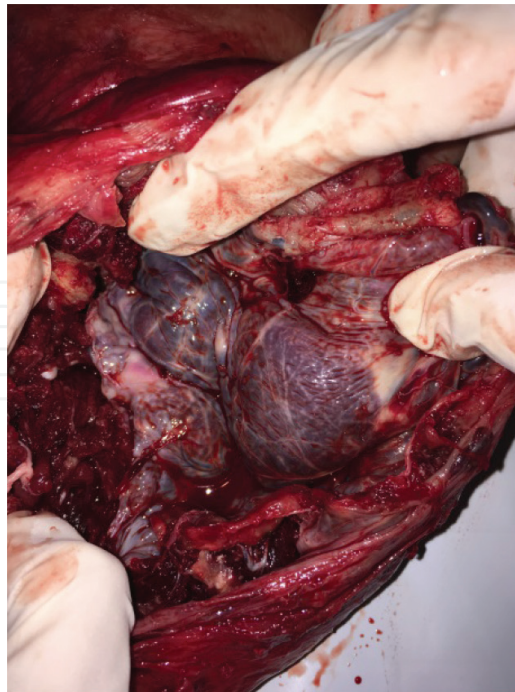
Figure 2. Image of the uterus occupied by placenta percreta after postpartum hysterectomy due to important hemorrhagic complications.

the ultrasound scan can be as high as 80–90% and, respectively, 98% [10]. Magnetic resonance imaging can add accuracy to MAP diagnosis when assessing the lateral extension and penetration depth of the placenta. However, a majority of cases of MAP are diagnosed during the third stage of labor or during Cesarean section [9], and about 21% of cases of MAP are responsible for peripartum hysterectomy [11]. Overall, in suspected cases with this type of placental pathology, the best approach includes a multidisciplinary team with early planning for antepartum and intrapartum management, preferable than late planning [12]. Some groups recommend delivery at 34–35 weeks by performing preterm Cesarean section with the placenta left in situ [13]. Other several adjuvant techniques have been proposed, as methotrexate treatment and/or placement of internal iliac artery balloon catheters, for occlusion and/or arterial embolization [14]. The goal of the conservative approach of MAP is the attempt of gradual resorption of the placenta or delayed delivery of the placenta [15]. A good prognosis of MAP pathology is feasible, with improving maternal and fetal outcome, if diagnosis is timely and there is adequate preparation of the delivery. These are essential keys in the management of such cases [16].

3. Placenta praevia

This type of obstetric pathology was firstly described in 1685 by Paul Portal, a French physician [17], as a major cause of hemorrhage, with a potentially life threat to the mother and the fetus. It was defined as the placenta that overlies entirely or partially the internal cervical os of the uterus. In complete praevia, the internal os is completely covered by the placenta (e.g., **Figure 3**). Placenta praevia is divided into *partial praevia* (a portion of the internal os is covered by the placenta),
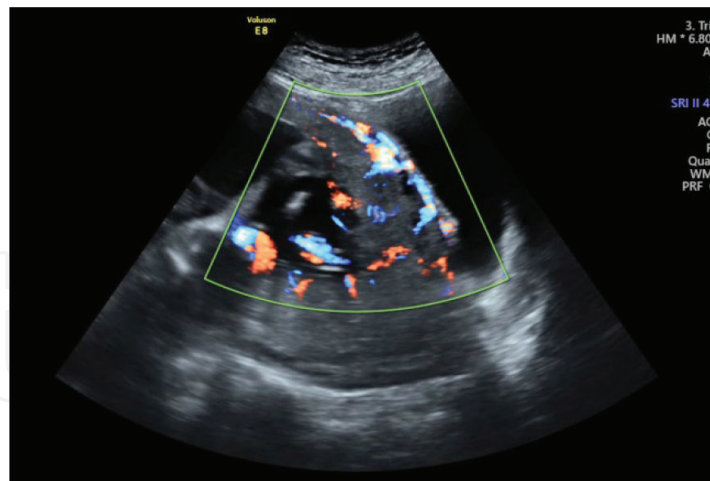
Figure 3. Ultrasound image of complete placenta praevia percreta in a patient with a previous Cesarean section (color Doppler examination showing the penetration of the placenta into the bladder).

marginal praevia or praevia maginalis (the edge of the placenta extends to the edge of the cervical os), and *low-lying placenta* defined as within 2 cm of the cervical os, without covering it [2]. The reported incidence of the condition is 1 in 200–250 pregnancies [1]. Among the risk factors, there are prior Caesarean delivery, previous abortion, prior intrauterine surgery, smoking, multifetal gestation, increase in parity, and increased maternal age. The risk for placenta praevia is 12 times higher in women with history of placenta praevia in a previous pregnancy. Some studies demonstrated an increased rate of placental insufficiency in women with placenta praevia [18]. However, in a retrospective study of women with a complete or partial praevia, no fetal growth restriction was diagnosed [19]. The placenta location must be recorded during the ultrasound scan in the first- and early second-trimester pregnancies. If the placenta is significantly low, an additional ultrasound scan at the beginning of the third trimester allows the final diagnosis. Patients should be aware that nothing can be done to prevent placenta praevia. The appropriate delivery in placenta praevia is by Cesarean section, as dilation of the cervix causes separation of the placenta, leading to bleeding from the opened vessels. Still, in cases of a low-lying placenta, as the bleeding morbidity has proven to be limited, a vaginal delivery remains an option [1]. Every hospital must have a suitable protocol or algorithm for the management of placenta praevia, as this is a condition with high maternal and fetal morbidity and mortality [20].

4. Vasa praevia

Vasa praevia is a rare condition, in which the fetal blood vessels traverse the lower uterine segment in advance of the presenting part, unsupported by either the umbilical cord or placental tissue (e.g., **Figure 4**). This pathologic structure can cause fetal blood loss, with significant neonatal morbidity or death in case of spontaneous rupture of membranes or amniotomy. Also, fetal heart decelerations and bradycardia can occur if compression of these vessels appears, due to the presenting part [20]. This condition is encountered in 1:2500–5000 pregnancies [21]. The prenatal diagnosis is made with a high accuracy by ultrasound, with a sensitivity of 100% and a specificity
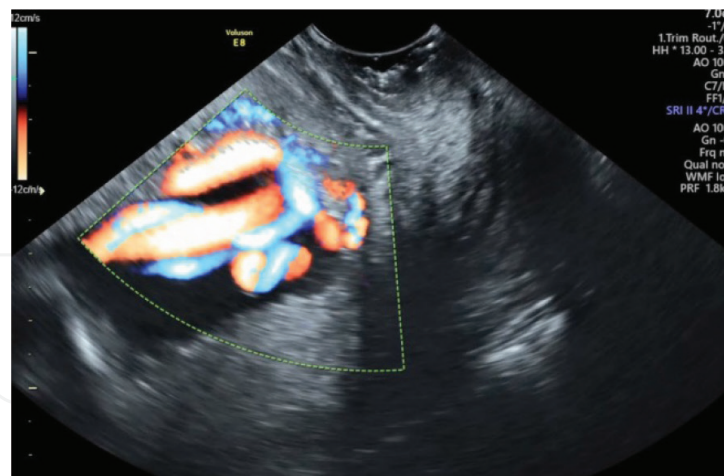
Figure 4. Ultrasound color Doppler image showing vasa praevia.

of 99–99.8%, if transvaginal color Doppler examination is used [20]. If unrecognized before the onset of labor, the fetal mortality rate ranges between 22.5 and 100% [22]. To improve the prenatal diagnosis, the prenatal ultrasound form should include a standard evaluation of the umbilical cord insertion site. However, some researchers demonstrated that general screening for vasa praevia is not cost-effective and is not advised [23]. There are recent reports of two main associations: velamentous insertions and vessels crossing between lobes in succenturiate or bilobate placentas [24]. Besides these strong risk factors, others include placenta praevia and conception by assisted reproductive technologies. If diagnosed with vasa praevia, elective Cesarean delivery should be proposed at 35–36 weeks [25]. Others prefer a scheduled Cesarean section at 37–38 weeks or when fetal lung maturation has been confirmed [26, 27]. The Canadian guidelines for the management of prenatally diagnosed vasa praevia include elective Cesarean section prior to the onset of labor. Also, as premature delivery is most likely, consideration should be given to administration of corticosteroids at 28–32 weeks (to promote fetal lung maturation), and hospitalization at about 30–32 weeks is advisable. Continuous electronic fetal heart rate monitoring and a rapid biochemical test for fetal hemoglobin can be considered, and if any of the above tests are abnormal, emergency Cesarean section should be performed [28]. Overall, physicians must be vigilant whenever amniotomy is performed as not all cases of vasa praevia are diagnosed antenatally. Any case of suspicion should benefit of immediate delivery, to avoid fetal shock or demise [22].

5. Placenta variants

5.1. Bilobed placenta

Bilobed placenta (placenta bilobate, bipartite placenta, placenta duplex) is a placental morphological anomaly that refers to a placenta separated into two roughly equal-sized lobes, separated by membranes (e.g., **Figure 5**). If there are more than two lobes, then the placenta is called a multilobed placenta. The estimated incidence is 2–8% of placentas [29]. The pathology of this type of placenta is considered to be a result of a localized placental atrophy, as a result of poor decidualization or vascularization of a part of the uterus (dynamic placentation theory) [30]. Also, the

genetic origin has been considered, as the risk of a bipartite placenta is greater in a woman with already a history of bipartite placenta. Frequent association with a velamentous insertion of the cord is reported, as the umbilical cord may insert in either lobe or in between the lobes. The diagnosis of bilobed placenta is made by ultrasound assessment when two separate placental discs of nearly equal size are noted. In cases of bilobed placenta, there is no increased risk of fetal anomalies. However, this type of placental abnormality can be associated with first-trimester bleeding, polyhydramnios, abruption, and retained placenta. Also, it can increase the incidence of vasa praevia with a high incidence of hemorrhage. Taking all these risk factors into consideration, a bilobed placenta does not have any unfavorable short-term or long-term pregnancy outcomes.

5.2. Circumvallate placenta

Circumvallate placenta represents one type of an extrachorial placenta, defined as an annularly shaped placenta with raised edges composed of a double fold of chorion, amnion, degenerated decidua, and fibrin deposits [1]. Pathologically, the basal plate is larger than the chorion frondosum [31]. The incidence of circumvallate placenta has been reported in 0.5–18% of placentae examined after delivery [32, 33]. There is an increased risk of vaginal bleeding at the beginning of the first trimester and also a risk of premature rupture of the membranes, preterm delivery, placental insufficiency, and placental abruption [34, 35]. The pregnancy outcome can be very poor. Prenatally, during the ultrasound scan, circumvallate placenta can be suspected as a peripheral rim of chorionic tissue appearing as an echodense ridge (placental shelf), with a "tire sign" appearance on the 3D exam [36]. However, the diagnosis is made most often after delivery, by inspection of the placenta. If circumvallate placenta is suspected antenatally, the pregnancy should be classified as a high-risk pregnancy, and special precautions should be considered, to prevent preterm labor. A high association between circumvallate placenta and a single umbilical artery [37] and no relationship between the amniotic band syndrome or limb body wall complex and circumvallate placenta have been reported [31]. Thus, the condition carries no risk of fetal deformity. Circummarginate placenta is another type of extrachorial placenta, with no clinical significance, where the transition from membranous to villous chorion is flat [1].
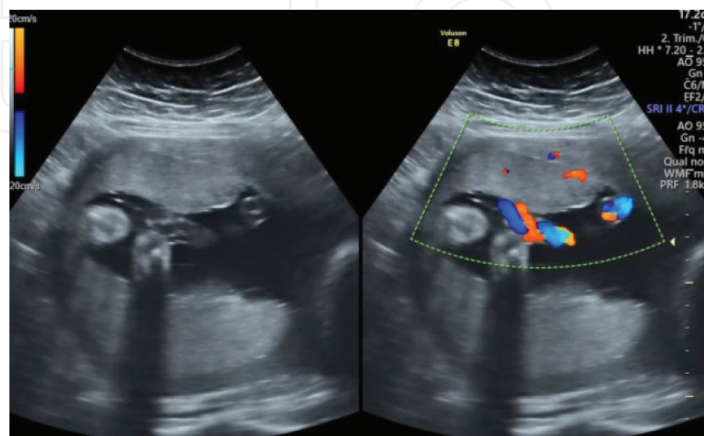

Figure 5. Ultrasound image (gray scale and color Doppler) of a bilobed placenta, showing the two lobes of the placenta and the umbilical cord insertion in one of the lobes.

5.3. Placenta membranacea

Placenta membranacea is an extremely uncommon variation in placental morphology, in which the placenta develops as a thin structure, occupying the entire periphery of the chorion. This type of placental abnormality is classified as *diffuse placenta membranacea* (with chorionic villi covering the fetal membranes completely) and *partial placenta membranacea* [1]. The estimated incidence is 1:20,000–1:40,000 pregnancies [38], with an association of abnormal placental adherence in up to 30% of cases [38]. The ultrasound assessment is useful, but being an extremely rare variant, there are no reports of its sensibility and specificity. The common symptom of this type of placental pathology is vaginal bleeding in the second or third trimester (often painless) or during labor. Complications such as antepartum hemorrhage, second-trimester miscarriages, fetal demise, and postpartum hemorrhage have been reported in pregnancy with placenta membranacea [39]. Placenta praevia and placenta accrete or intrauterine growth restriction can also be associated with this condition, worsening the maternal and fetal prognosis [30, 40].

5.4. Succenturiate placenta

In succenturiate placenta a smaller accessory placental lobe develops in the membranes, apart from the main disc of the placenta. There can be more than one succenturiate lobe, and it is a smaller variant of a bilobed placenta. In placenta supuria the communicating membranes do not have vessels [1]. As risk factors, advanced maternal age, in vitro fertilization, primiparity, proteinuria in the first trimester of pregnancy, and implantation over leiomyomas or in areas of previous surgery have been cited in the literature [1]. This condition can be diagnosed in 5% of pregnancies, by ultrasound scan as a smaller separate lobe similar to the main placental lobe. Caution should be considered in identifying any connecting vessels, especially vasa praevia. Differential diagnosis may also include focal myometrial contraction and iso-echoic hematoma from a placental abruption. Complications may appear as there is an increased risk of vasa praevia and postpartum hemorrhage, due to retained placental tissue.

6. Chronic intervillositis

Chronic intervillositis, also known as massive chronic intervillositis or chronic histiocytic intervillositis, is an exceptionally rare placental anomaly, defined by inflammatory placental lesions [1], mainly diffuse histiocytic infiltrate in intervillous space [41]. Among risk factors, maternal diabetes, maternal hypertension, intravenous drug abuse, preeclampsia, and systemic lupus erythematosus are mentioned. This condition has a perinatal mortality of 80%, due to an associated risk of recurrent spontaneous abortion [42], fetal growth restriction [43], and fetal death. The recurrence rate is considered to be above 60%.

7. Placental mesenchymal dysplasia

Placental mesenchymal dysplasia is a rare vascular anomaly of the placenta characterized by mesenchymal stem villous hyperplasia [1]. The ultrasound diagnosis includes placentomegaly

and a "grape-like" placental appearance, both mistaken clinically and macroscopically for a partial hydatidiform molar pregnancy [44]. The differential diagnosis is important, because it may result in termination of pregnancy. Still, the final diagnosis is made by means of placental histology. The disorder also has been reported to be associated with both intrauterine growth restriction (IUGR) and fetal death [45]. In many cases, the cause of fetal death is fetal vascular obstructive pathology, causing longstanding, severe fetal hypoxia, due to chorionic vessel thrombosis [46]. Beckwith-Wiedemann syndrome has been linked to placental mesenchymal dysplasia. Invasive testing is advisable to confirm a normal karyotype and exclude partial molar pregnancy [47].

8. Diabetic placenta

The placenta represents a natural selective barrier between maternal and fetal blood circulations, and it is highly sensitive to the hyperglycemic environment. Consequently, adaptive changes of the structure and function appear. The histological findings are typical: villous immaturity, villous fibrinoid necrosis, chorioangiosis, and increased angiogenesis [48]. Chronic fetal hypoxia can occur due to placental changes associated with inflammation and oxidative stress. Potential intrauterine complications are growth restriction, premature labor, preeclampsia, risk of oxygen deprivation, low neonate body temperature, low blood sugar levels at birth, and stillbirth [49].

9. Placental chorioangioma

Chorioangioma is a benign vascular tumor, found in approximately 1% of all pregnancies [50]. It was firstly described in 1798 by Clarke [51]. This pathology is a malformation of the primitive angioblastic tissue of the placenta perfused by the fetal circulation. It is rarely clinically significant and is usually discovered incidentally. Most of the chorioangiomas have small
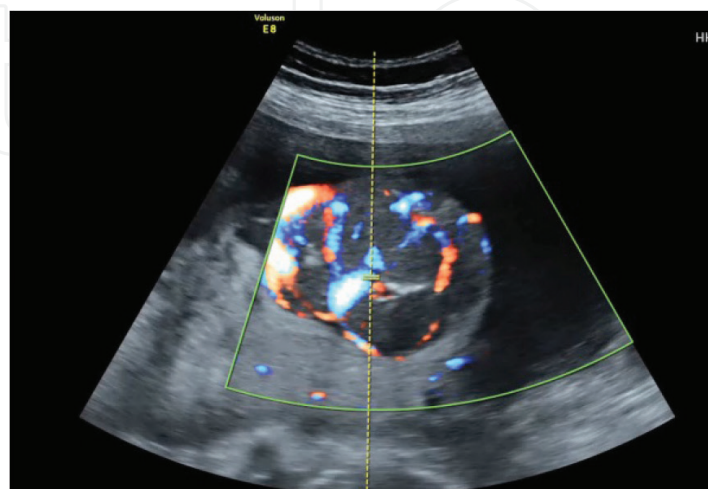

Figure 6. Ultrasound color Doppler image of a chorioangioma diagnosed in the second trimester of pregnancy.

dimensions. However, large chorioangiomas have been associated with a range of fetal conditions (fetal anemia, thrombocytopenia, hydrops, hydramnios, intrauterine growth retardation), including prematurity and stillbirth [1]. Also, large tumors can degenerate in necrosis, calcification, hyalinization, or myxomatous degeneration. Typically, on the ultrasound, a chorioangioma is located near the insertion of the cord into the amniotic cavity, as a hypoechoic, rounded mass with usually anechoic cystic areas with low resistance pulsatile flow (e.g., **Figure 6**) [52]. In rare cases the tumors are pedunculated. As differential diagnosis, subamniotic hematoma, partial hydatidiform mole, submucosal uterine fibroid, placenta teratoma, and atypical placental venous lake should be considered [53].

10. Placental infections

Most infections arise from several infective agents that may cross into the placenta from the maternal circulation [1]. These kinds of infections can be associated with a variety of developmental effects, from virtually insignificant to major maternal and fetal developmental complications. Placental examination by a pathologist should be considered in every case of preterm delivery, fetal tachycardia, maternal signs of endomyometritis (e.g., fever, uterine tenderness, leukocytosis, tachycardia), neonatal intensive care unit admission, malodorous placenta, retained placenta or postpartum hemorrhage, and stillbirth [54]. However, a specific infectious agent is rarely diagnosed by placental examination. Still, the placental histology may confirm the clinical diagnosis of an infectious etiology in some cases of nonreassuring fetal heart rate patterns or neonatal morbidity/mortality. The most common placental infections are:

- Malaria: characterized by the pigment-laden maternal red blood cells and macrophages aggregate in the intervillous space [55].

- Cytomegalovirus is the most common congenital viral infection, mostly subclinical at birth in cases of intrauterine growth restriction and stillbirths [56]. The classic histopathological finding in the placenta includes viral inclusions. These may be detected only if using immunohistochemistry techniques.

- Herpes simplex virus: the histopathological features of the placenta may include lymphoplasmacytic villitis. The demonstration of the virus by immunohistochemistry or by molecular techniques allows the diagnosis, since the above findings are nonspecific [57].

- *Listeria monocytogenes* is characterized by acute villitis, with abscess formation and fetal central nervous system damage [58].

- Streptococcal infection: both group B and group A streptococci can produce placental infection.

- Syphilis: *Treponema pallidum* infections determine a chronic villitis (plasma cells, mixed acute and chronic infiltrate).

- Toxoplasmosis implies a risk of placental colonization, depending on the volume of uteroplacental blood flow, on the maternal immunocompetence, and parasitemia. Placental infection, described by granulomatous villitis, cysts, plasma cell deciduitis, villous sclerosis, and chorionic vascular thrombosis, is more common with advancing gestational age at the time of maternal parasitemia [59].

- *Chlamydia psittaci*: can infect the placenta and can cause significant feto-maternal morbidity and mortality by an intense, acute intervillositis, perivillous fibrin deposition with villous necrosis, and large irregular basophilic intracytoplasmic inclusions within the syncytiotrophoblast [60, 61].

11. Placental membranes

The fetal membranes (chorion, amnion) represent the interface between the fetal graft and the maternal host [1]. Infection may also pass the fetal membranes, especially in the area overlying the cervix. It provides direct access to pathogens, ascending from the vagina and the cervix [62]. Less commonly, infectious agents enter the uterus as a result of invasive procedures (e.g., amniocentesis, fetoscopy, cordocentesis, and chorionic villus sampling) or via the fallopian tubes from an infectious process in the peritoneal cavity.

11.1. Chorioamnionitis

Chorioamnionitis is the most frequent histopathological result of ascending transcervical infection and occurs with both symptomatic and silent infections [63]. The histologic diagnosis of chorioamnionitis is allowed if the inflammatory infiltrate involves either or both the chorion and the amnion. The acute chorioamnionitis is more common than the chronic form [64]. As clinical symptoms, chorioamnionitis is characterized by maternal fever, tachycardia, uterine tenderness, or foul-smelling amniotic fluid. However, cultures of the amniotic fluid or membranes fail to document the bacterial infection in 25–30% of placentas with histologic chorioamnionitis [65]. The infection of the membranes is often polymicrobial, with the most commonly seen bacteria: *Streptococcus* sp., *Escherichia coli*, *Ureaplasma* sp., *Fusobacterium* sp., *Mycoplasma* sp., and anaerobes [63]. The correct diagnosis and treatment of chorioamnionitis are paramount, as it is an important cause of perinatal and maternal morbidity and mortality [66]. The major pathological consequences of chorioamnionitis may include premature rupture of membranes, preterm labor, prolonged labor, premature delivery, fetal and newborn infection, and endomyometritis.

12. Gestational trophoblastic disease.

12.1. Hydatidiform mole

Hydatidiform mole (HM), called also a *molar pregnancy*, represents a subcategory of gestational trophoblastic disease. The origin of the entity is the gestational tissue. The character

of HM is usually benign, but it has a known potential to become malignant and invasive. The incidence of a HM is 1:1000–2000 [67]. Risk factors include extremes of maternal age (greater than 35 years old and less than 20 years old), a previous molar pregnancy, women with previous spontaneous abortions or infertility, dietary factors, and smoking [68]. The HM can be a *complete mole*, with the absence of the fetus, or a *partial mole* with an abnormal fetus or a fetal demise; rarely, a mole coexists with a normal pregnancy. In complete HM, 90% of cases the karyotype are 46XX diploid, while in partial HM, the karyotype is usually triploid 69XX [1]. The histopathological event of HM is considered to be a proliferation of the villous trophoblast, accompanied by swelling of the chorionic villi, resulting in high levels of human chorionic gonadotrophin (hCG) production (e.g., **Figure 7**) [68]. The location of the HM is the uterine cavity, with exceptionally rare cases located in the fallopian tubes or ovaries. Clinically, the most common symptom is the vaginal bleeding in the first trimester. Sometimes an association of hyperemesis (severe nausea and vomiting) or passage of vaginal tissue described as "grape-like clusters" or "vesicles" can be encountered. If not early diagnosed, other significant complications may appear, such as hyperthyroidism, including tachycardia and tremors and preeclampsia. Usually, on a physical exam, there is a uterine size discrepancy compared with the amenorrhea period, the uterus being larger in complete mole and smaller in partial mole [69]. The ultrasound exam finding is a heterogeneous mass in the uterine cavity, with multiple anechoic spaces

Figure 7. Image of post-hysterectomy uterus invaded by a hydatidiform mole in a 48-year-old patient.
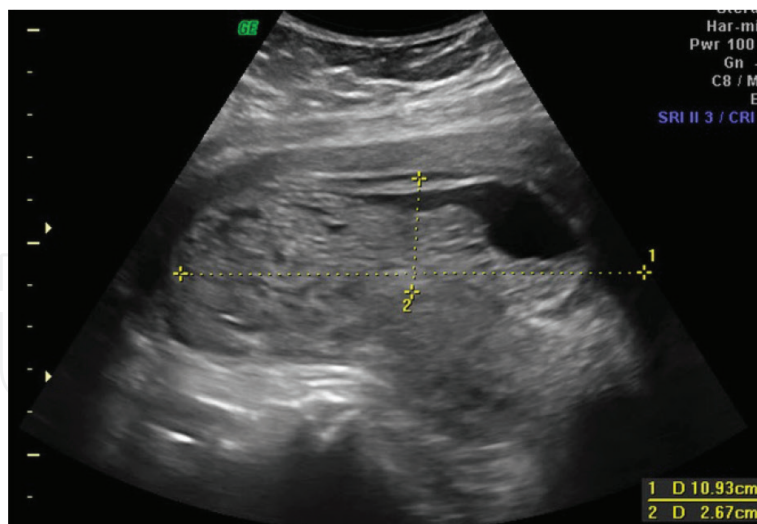
Figure 8. Ultrasound image of the case of hydatidiform mole.

(e.g., **Figure 8**). The "snow storm" or "bunch of grapes" appearance is no longer seen with nowadays equipment. In complete moles the embryo is absent, and no amniotic fluid is present [70]. In the first trimester, the diagnosis of complete mole can be difficult; bilateral theca lutein cyst may be seen [71]. In partial mole, the molar placenta may not always be seen; the amniotic cavity is either empty or contains a well-formed but growth-retarded fetus, either dead or alive, with hydropic degeneration of fetal parts [72]. Occasionally, the differential diagnosis between partial moles, complete moles, and missed abortion [73] may be difficult. In molar pregnancy the first step after the diagnosis is the chest X-ray to determine metastasis. Computer tomography and magnetic resonance imaging can add valuable additional information for the final diagnosis. After careful counseling of the patient, including genetic testing, the best treatment option remains suction and curettage for evacuation. Hysterectomy, however, is an option if preservation of the fertility is not necessary. When hCG levels remain elevated after a proper evacuation of the uterine cavity, a gynecology oncology consultation is required to guide the therapy and consider chemotherapy [68].

12.2. Choriocarcinoma

Choriocarcinoma is a rare aggressive tumor, with highly malignant potential and widespread dissemination metastases [74]. It is considered part of the spectrum of gestational trophoblastic disease and is called *gestational* choriocarcinoma. The high mortality is due to lack of early diagnosis and appropriate chemotherapy [75]. Approximately 5% of cases of complete HM can be complicated with choriocarcinoma. Only about half the cases of choriocarcinoma arise from a complete HM. The imaging diagnosis of choriocarcinoma includes a discrete, central, infiltrative mass enlarging the uterus, with a possible invasion of the myometrium and beyond (e.g., **Figures 9** and **10**). The ovaries may be enlarged, due to cysts secondary to increased levels of hCG [76]. If choriocarcinoma arises from a complete HM, the prognosis is usually favorable after proper chemotherapy. On the contrary, other cases of choriocarcinoma have a less favorable prognosis.
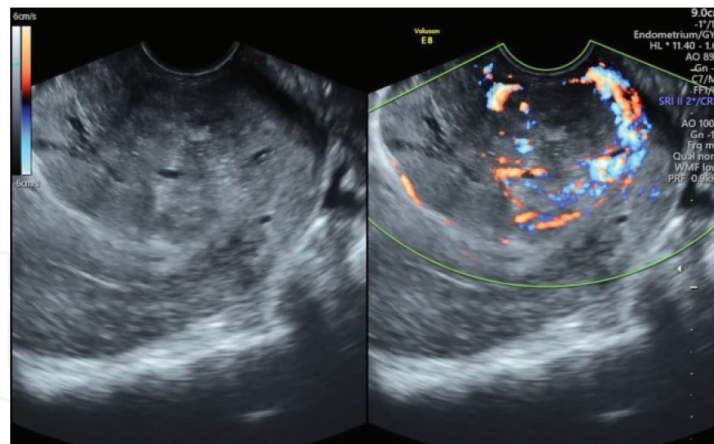
Figure 9. Ultrasound image in gray and color Doppler scale showing a rare case of choriocarcinoma of the cervix with intense vascularization.
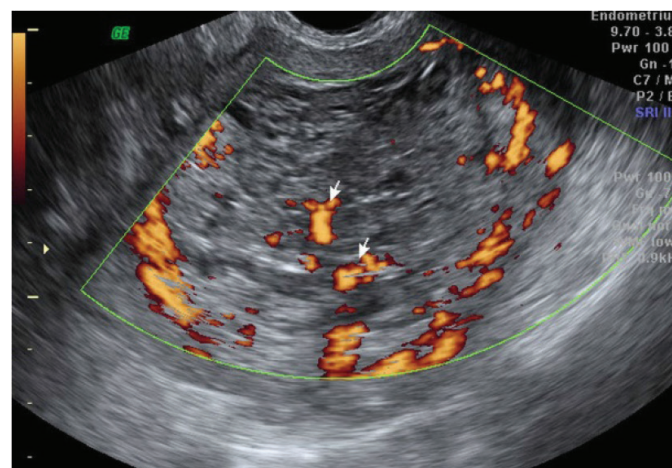

Figure 10. Ultrasound image in gray and color Doppler scale showing a case of choriocarcinoma with invasion of the myometrium and beyond.

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