Rare Pathologic Placenta Ultrasound Findings

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Abstract: Rare ultrasound placenta findings, such as avascular cystic lesions, hyperechogenic and thick placenta, and enlarged placenta, are associated with infarcts, perivillous fibrin deposition, and mesenchymal dysplasia. These lesions can be present in 20% of normal pregnancies but are more frequent in pregnant women with pre-eclampsia (PE) and/or fetal growth restriction, autoimmune diseases, and infections, and can increase the risk of perinatal complications, including fetal death. Evaluation of the placental surface may also identify cases with circumvallate placenta and chorioangiomas. These rare placental findings require close clinical follow-up and serial fetal evaluations to identify those at a higher risk of abnormal perinatal outcomes.

Key Words: adverse perinatal outcomes, chorioangioma, circumvallate placenta, infarction hematoma, maternal floor infarction, mesenchymal dysplasia, perivillous fibrin deposition, placental infarcts, placental tumors, rounded hematoma, ultrasound

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Key Points

- Well-defined (rounded) hypechogenic (black areas) in the placenta with no flow inside or outside and with a hyperechogenic rim (cysts) should be considered highly suspicious of placental infarcts.
- Large cysts with mixed echogenicity inside the lesion should be considered as infarction or rounded hematomas.
- Multiple small cysts with echodense placenta areas, increase the risk for massive perivillous fibrin deposition (MPFD).
- Cystic lesions of different size, enlarged placenta, and different velocities of intraplacental blood flow, are suggestive of placental mesenchymal dysplasia (PMD).
- The presence of any type of placenta cystic lesions increases the risk of adverse perinatal outcomes and are related to pre-eclampsia, fetal growth restriction, infections, preterm delivery, overgrowth syndromes, and fetal death
- An abnormal placenta shape can be associated to circumvallate placenta or chorioangiomas.
- Maternal serum concentrations of alpha-fetoprotein and human chorionic gonadotropin can contribute to the diagnosis of placenta mesenchymal dysplasia, chorioangiomas, and choriocarcinoma.
- Close surveillance of patients and fetuses with rare placental lesions (Table 1) should be performed.

Placental abruption, placenta previa, and placenta accreta spectrum are the most common abnormal placental findings. However, other less frequent placental complications can also affect the normal development of pregnancy and increase the risk of abnormal outcomes.

VASCULAR LESIONS LEADING TO PLACENTAL TISSUE NECROSIS

Placental infarctions, massive perivillous fibrin deposition, and infarction (rounded) hematomas are difficult to differentiate with ultrasound as all 3 conditions share similar characteristics and can be present in the same pregnancy. Cystic images suggestive of infarction hematomas are usually larger than those from infarcts, and massive perivillous fibrin deposition can be seen as the combination of small cystic lesions and echodense areas in the placenta (Table 1). These 3 conditions are highly related to fetal growth restriction and fetal demise. In high-risk pregnant patients, placenta lesions associated with infarcts and tissue necrosis can be observed in about 76% of cases.[1]

Placental Infarcts

Ultrasound Findings

Placental infarcts are characterized by hypoechogenic (dark) areas surrounded by a hyperechogenic (white) rim resembling cysts (Fig. 1). Small cystic lesions are present in about 20% of normal pregnancies;[2] however, the risk of abnormal outcomes increases when multiple areas with these characteristics, or one predominant large cystic area, are seen. At ultrasound scanning, these well-defined "black" cysts do not present evidence of blood movement either inside or outside the cyst despite using highly sensitive color Doppler modalities. In addition, the shape of the cyst is maintained stable over time, which is the opposite of what happens in a normal placental lake, where the shape changes according to blood movements inside the lake.

Histopathology

Placental infarcts are mainly due to: (a) occlusion of spiral arteries by thrombus; (b) strangulation of the placental villi due to increased perivillous or intervillous fibrin/fibrinoid deposition; and (c) impairment of the fetal circulation due to fetal thrombotic vasculopathy.[3–5] Susceptibility to obstruction of maternal vessels seems to be related to a defective physiological transformation in the first trimester of pregnancy. When the maternal intervillous blood flow stops, the intervillous space collapses, the villous epithelial trophoblast surfaces die and the villi adhere to the lesions forming a firm mass that can be palpated when examining the placenta after delivery.[6]

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The authors declare that they have nothing to disclose.

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TABLE 1. Ultrasound Characteristics and Clinical Outcomes of Rare Placental Lesions

Placental lesion	Cysts	Color doppler flow	Size	Placental characteristics	Clinical implications
Placental infarcts	Hypoechogenic (black) areas surrounded by a hyperechogenic (white) rim-round	No	Stable in size	Size unchanged	Pre-eclampsia, fetal growth restriction
Infarction/rounded hematoma	Hyperechogenic rim and mixed echogenicity inside the lesion	No	Large	Size unchanged	Hypertensive disorders, FGR, preterm delivery, gestational diabetes,
Massive perivillous fibrin deposition (maternal floor infarction)	Small multiple cystic lesions. echodense (white) areas in the placenta	Variable velocities	1-2 cm in length with a mean thickness of the echogenic rim measuring 0.25 mm	Normal or enlarged placenta	Spontaneous abortion, fetal demise, FGR, preterm delivery, pre-eclampsia.
Placental mesenchymal dysplasia	Multiple cystic lesions	Increased vascularity with slow blood movement within and around the cysts	Small	Thick and enlarged placenta	Abnormal MSAFP, overgrowth syndromes, FGR, fetal demise
Circumvallate Placenta	None	No change		Double layer of amnion and chorion forms a raised, rolled edge (shelf)	Placental abruption, chorioamnionitis, and preterm delivery
Chorioangiomas	None	Increased vascularity		Well-circumscribed hyperechoic or hypoechoic mass protruding from the fetal surface of the placenta or near the umbilical cord insertion site	Abnormal MSAFP. Asymptomatic > 4 cm associated with anemia, polyhydramnios, hydrops, IUGR, fetal death.
Teratomas	Heterogenous (cystic, solid)	Reduced vascularity as compared with surrounding placental tissue	Various	Echogenic areas suggestive of calcification or fatty tissue, alternating with hypechoic areas filled with fluid	Benign tumors exclude twin pregnancy and fetus acardius amorphous.
Trophoblastic placental tumor	"Cystic" like lesion heterogenous	Increased vascularity	Various	Heterogeneous, echogenic mass with small anechoic cystic spaces measuring 2.5-8 mm	Extremely elevated maternal serum concentrations of hCG, vaginal bleeding, dyspnea (lung metastases), and abdominal pain

FGR indicates fetal growth restriction; hCG, human chorionic gonadotropin; MSAFP, maternal serum alpha fetoprotein.

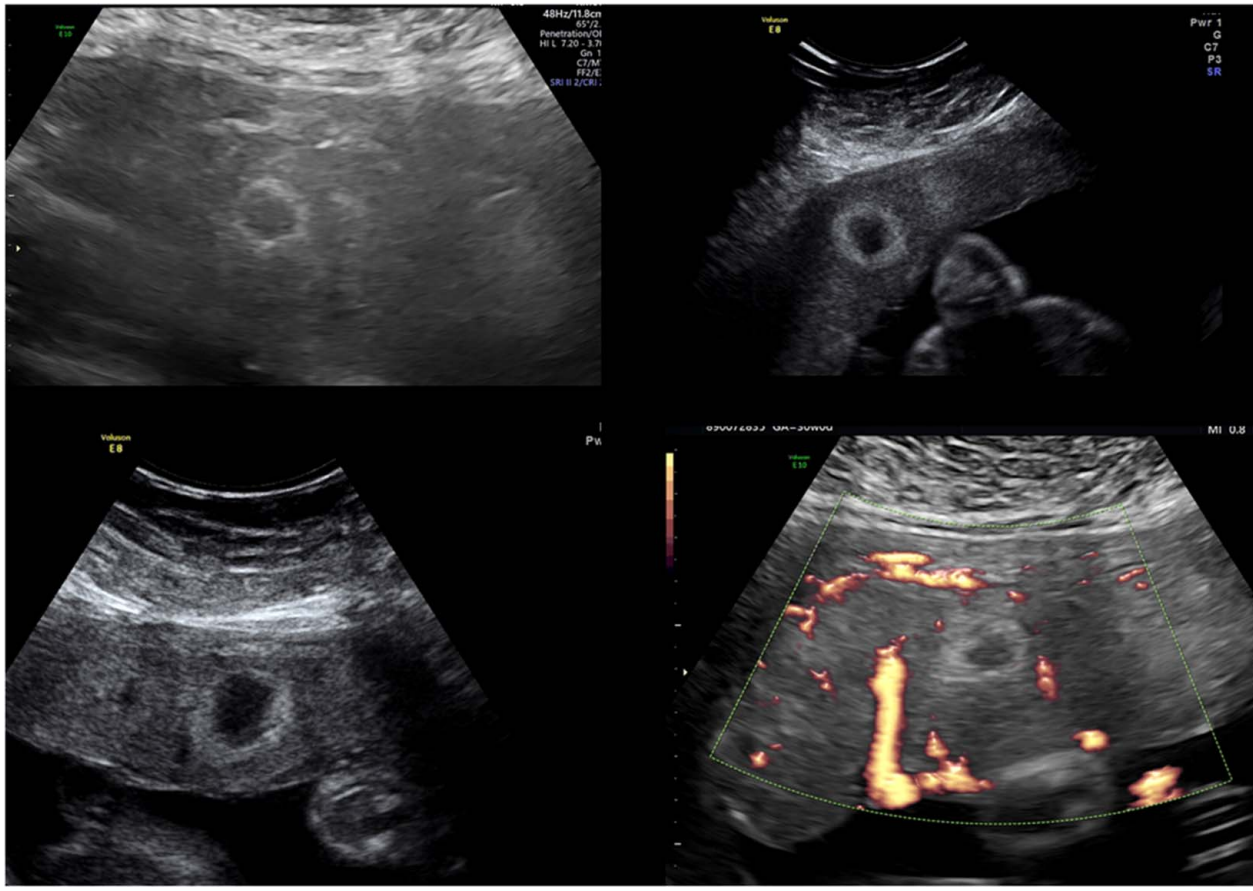

FIGURE 1. Placental infarcts, hypoechoic lesion with an echogenic rim, and no blood flow movements within or around the cyst. full color online

Clinical Implications

About 50% of all cystic placental lesions diagnosed prenatally have been associated with placental infarcts, reflecting maternal vascular under perfusion.[7] Cystic infarcted lesions have been observed in about 70% of patients with pre-eclampsia (PE), and in 40% of growth-restricted fetuses (FGR). In about 40% of patients with severe pre-eclampsia, the infarcted placental areas can occupy more than 25% of the total placental surface.[8] Hypoechogenic areas highly suspicious of placental infarcts are frequently seen in growth-restricted fetuses with absent or reversed end-diastolic velocities in the umbilical artery. Viero et al.[9] reported cystic images highly suspicious of placental lesions in 73% of 59 fetuses with absent end-diastolic flow in the umbilical artery (UA). The combination of hipoechogenic cystic lesions and abnormal umbilical, or uterine arteries Doppler velocimetry increases the risk of perinatal death

Rounded Hematoma/Infarction Hematoma

Ultrasound Findings

These are generally isolated large cysts with a hyperechogenic rim and mixed echogenicity inside the lesion. This mixed echogenicity is due to the formation of a clot or hematoma within the cyst (Fig. 2). Color Doppler reveals absent blood movement inside the lesion and around the hyperechogenic rim. The cyst may occupy a large area in the placenta ultrasound image.

Histopathology

Infarction hematomas are characterized by an infarcted area due to the occlusion of a spiral artery with a subsequent recanalization creating a hematoma or clot inside the cyst surrounded by a hyperechoic rim. These lesions are larger than common infarcts due to the expansion of the lesion at the time of vascular recanalization and represent a larger area of necrotic placental tissue.

Clinical Implications

Infarction hematomas have a strong association with maternal vascular malperfusion and decidual vasculopathy, and are highly associated with adverse perinatal outcomes including severe fetal growth restriction and fetal death.[5,10] Infarction hematomas have been associated with chronic hypertension, pre-eclampsia, gestational diabetes, and preterm delivery.[7,11]

Neville et al[11] applied the term "rounded intraplacental hematoma" referring to the same lesion. The same authors described 28 women with rounded intraplacental hematomas and reported a prevalence of 60% (17/28) of fetal growth restriction, 21% (6/28) of perinatal mortality, and 17.8% (5/28) of placental abruption. The authors suggested that a complete placental examination using grayscale and Doppler modalities should be performed to improve the diagnosis of this condition.

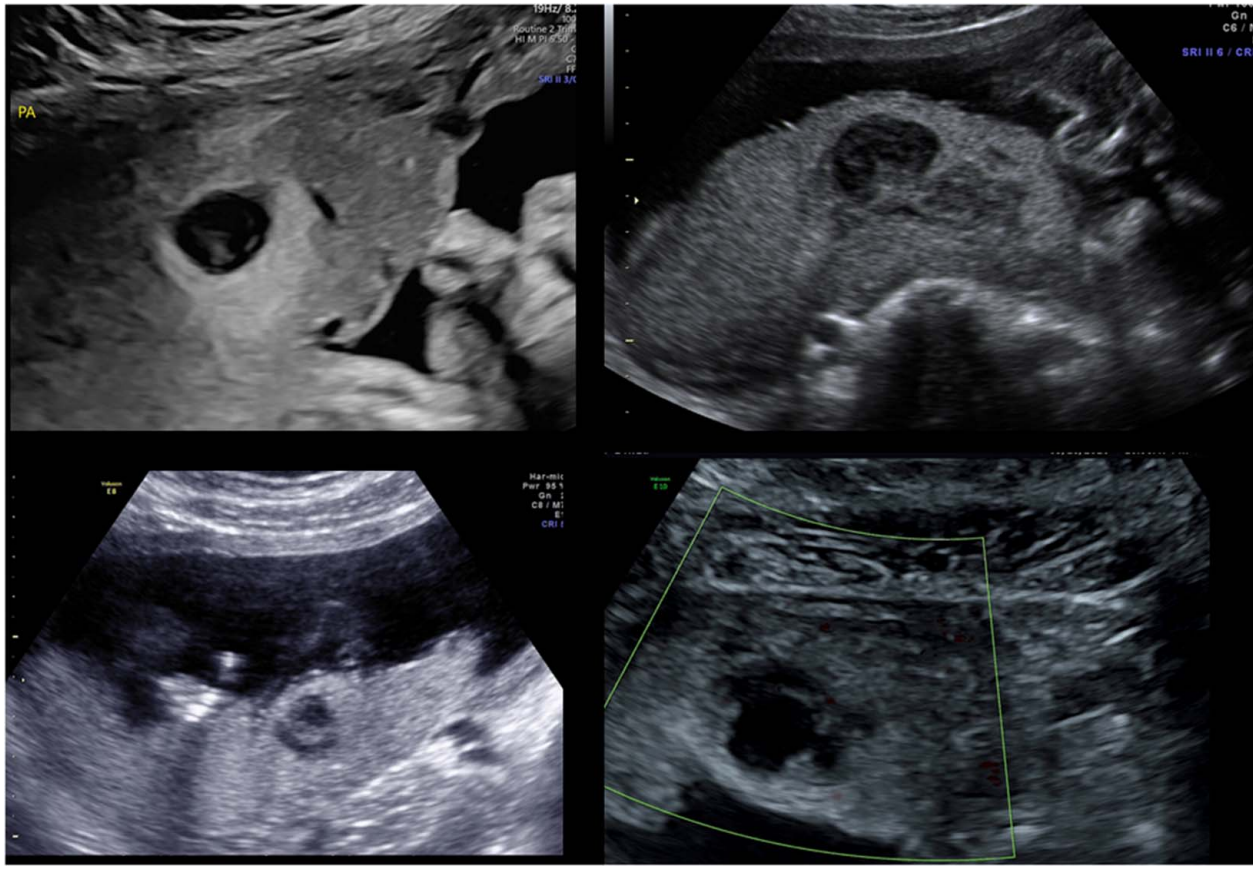

FIGURE 2. Infarction hematomas (rounded hematomas), large cystic lesions with mixed echogenicity inside the cyst and a large echogenic area around the cyst with no blood flow movements inside or around the cyst. full color online

Massive Perivillous Fibrin Deposition (Maternal Floor Infarction)

Ultrasound Findings

Multiple small cystic lesions of about 1 to 2 cm in length with a mean thickness of the echogenic rim measuring about 0.25 mm. without blood flow inside the cysts, and reduced vascularization in the affected area (Fig. 3). The affected area appears echodense (more white) at ultrasound examination due to the accumulation of fibrin around the lesion.

Histopathology

Massive perivillous fibrin deposition (MPVFD) can be classified as fibrin-type fibrinoid (F) secondary to blood coagulation derived products, or matrix-type (M) related to extravillous trophoblast products.[12,13] In the F-type MPVFD there is massive accumulation and deposition of serum-derived fibrin in the intervillous space, which causes occlusion of the blood supply in different areas of the placenta. In the M-type MPVFD there is accumulation of extravillous trophoblast-derived products into the extracellular matrix around the distal villi. Between 60% and 75% of the MPVFD lesions are associated with intervillus thrombosis, and occlusion of blood supply in different areas of the placenta.[14] The hyperechogenic rim represents villi compressed by laminated fibrin and erythrocytes.

Clinical Implications

MPVFD is a rare abnormal finding affecting about 0.1% of all pregnant women; however, this prevalence can differ according to the gestational age at delivery and previous obstetric history. Women with previous abortions/miscarriages have a higher prevalence of MPVFD. Kim et al[15] evaluated placental biopsies from 562 miscarriages and reported a prevalence of 2.2% (n = 15) of MPVFD, 12/15 (80%) had a history of 2 or more previous miscarriages. MPVFD has been associated with adverse pregnancy outcomes such as fetal death, FGR, preterm delivery, PE, and recurrent spontaneous abortion.[16–18] MPVFD has also been associated with maternal complications that is, maternal rejection type response, infections such as SARS-CoV-2, enterovirus, cytomegalovirus, and syphilis, and autoimmune diseases polymyositis2, and antiphospholipid antibodies syndrome.[14]

In high-risk pregnancies with MPVFD, Proctor et al.[19] reported 79% prevalence of abnormal perinatal outcomes, 61% frequency of preterm delivery, 18% severe FGR, and 4% perinatal death. MPVFD can recur in approximately one-third of women.[20,21]

Management and Follow-up of Vascular Lesions Leading to Placental Tissue Necrosis

Visualization of the entire placenta is recommended in all pregnancies, if multiple cystic areas are seen, Doppler techniques can be applied to identify blood movements

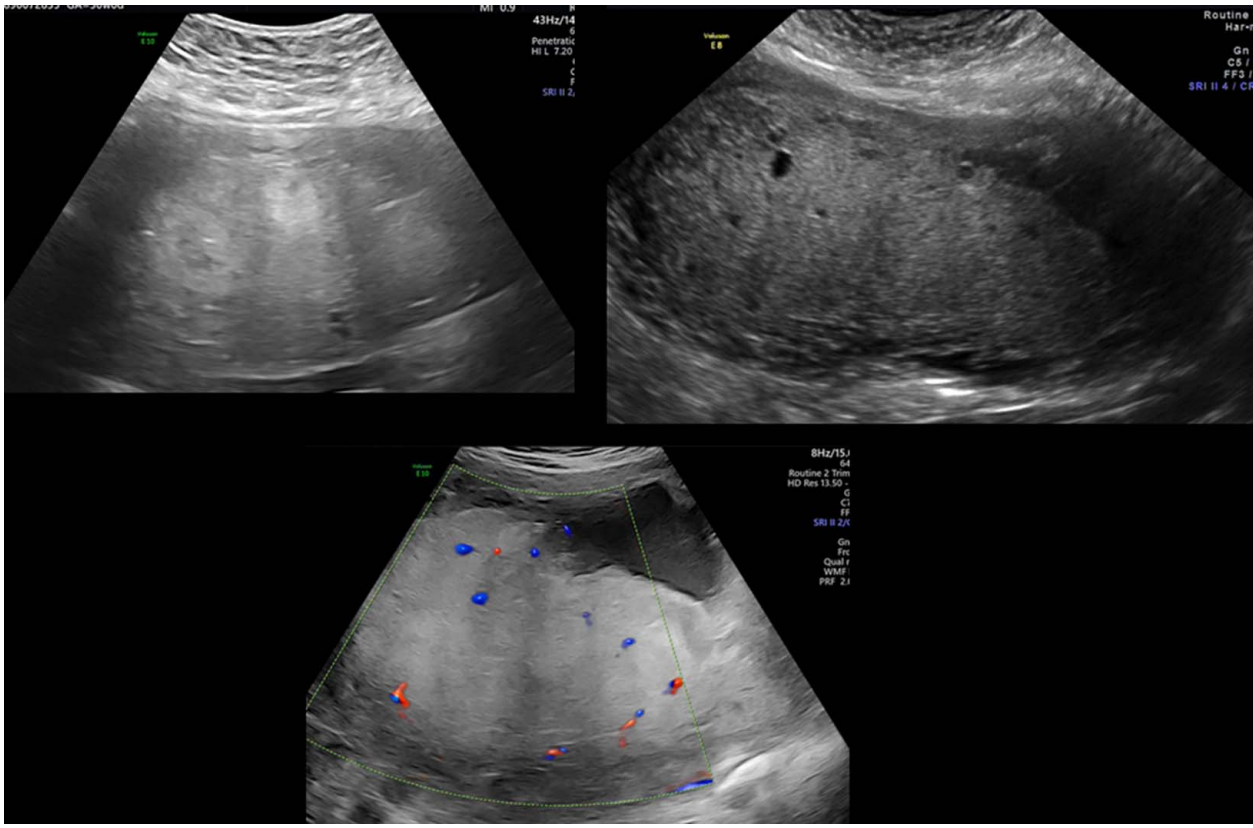

FIGURE 3. Massive perivillous fibrin deposition (MPFV), enlarged hyperechogenic placenta with multiple small cystic lesions, and with reduced blood flow. full color online

inside or outside the cysts. Differences in placental echogenicity and thickness can also contribute to the diagnosis of placental vascular lesions.

Close follow-up of patients with placental cystic lesions highly associated with vascular maldevelopment should be performed, particularly to exclude pre-eclampsia, and serial ultrasound scans to detect FGR. In cases with an EFW < 10th percentile, Doppler evaluation of the umbilical and middle cerebral arteries, and ductus venosus is recommended. The combination of abnormal Doppler in the umbilical artery or the ductus venosus (absent/reversed end-diastolic or atrial flow) and cystic placental lesions increases the risk of perinatal mortality and leads to the decision of delivery. When other vascular territories are affected but the umbilical artery still does not show signs of severe deterioration, close surveillance is recommended.

PLACENTAL MESENCHYMAL DYSPLASIA

Ultrasound Findings

Placental mesenchymal dysplasia (PMD) is characterized by a thick and enlarged placenta with multiple small cystic lesions similar to a hydatidiform mole. PMD is also known as non-hydatidiform mole, and is associated with

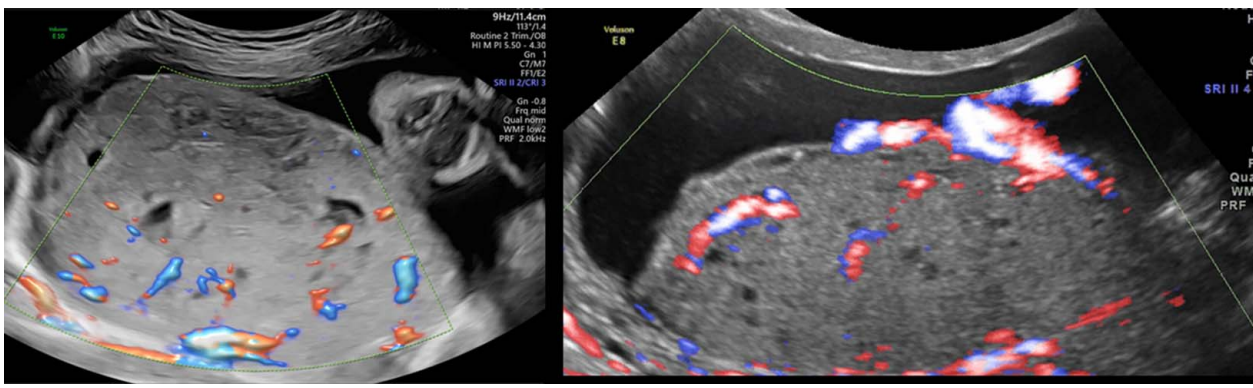

FIGURE 4. Placental mesenchymal dysplasia, enlarged and thick placenta with hypoechoic structures of different size, and increased vascularity with slow blood movement around the cysts. full color online

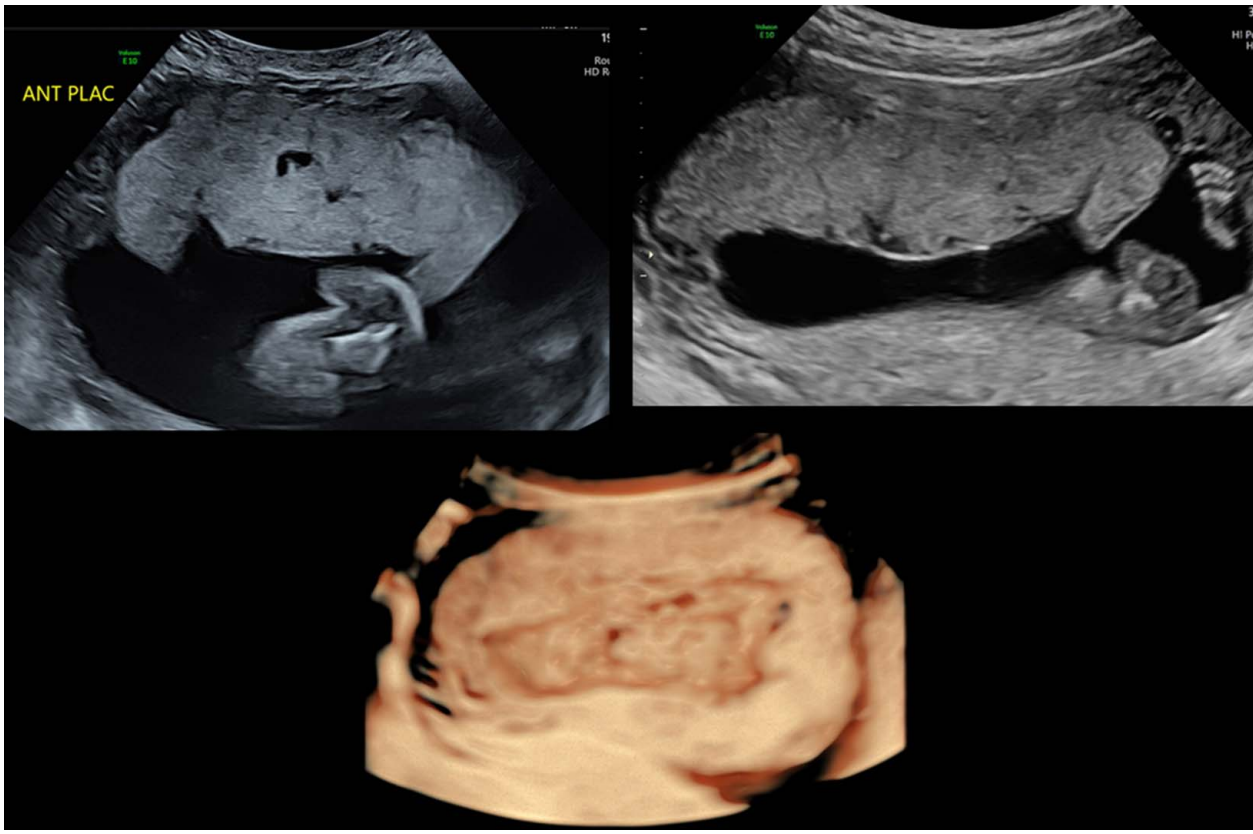

FIGURE 5. Placenta Circumvallate, showing the uplifted edge of the placenta and a marginal rim. full color online

elevated maternal serum concentrations of alpha-fetoprotein (AFP) and human chorionic gonadotropin (hCG).[22] Color Doppler imaging shows increased vascularity with slow blood movement within and around the cysts (Fig. 4).[23–25]

Histopathology

PMD is characterized by hyperplastic mesenchymal tissue of stem villi along with cystic dilatation and edema. Vascular abnormalities in the chorionic plate and stem villous include tortuosity, aneurysmal dilatation, and thrombosis. The histologic findings consist of mesenchymal villus hyperplasia, dilatation of the chorionic vessels, enlarged stem villi with loose connective tissue, and cistern-like formations.[26–28] Placental blood vessels are diffuse and tortuous, there is dilatation of the villi, and thrombus formation.[29]

Clinical Implications

Mesenchymal dysplasia has a prevalence of 0.2% and is associated with overgrowth syndromes such as Beckwith-Wideman, but also to FGR and fetal demise.[30–33] Differential diagnoses include molar pregnancy, chorioangioma, and placental hematomas.[34] In a recent study including 15 cases of PMD, the authors reported 4 perinatal deaths (26%, 3 fetal demise, one miscarriage), 6 terminations, 5 live births, and 3 cases with FGR.[29] Guenot et al[35] studied 22 cases of PMD diagnosed by placental pathology and described 5 cases with elevated AFP and 3 with elevated h-HCG, half of the fetuses (11/22) developed growth restriction, 18% (4/22) fetal death, and 64% (14/22) preterm delivery. Maternal complications that is, pre-eclampsia and gestational hypertension, were observed in 27% of cases (6/22). Of note, from 14 fetuses born alive (after excluding fetal deaths and terminations), 5 neonates died between 1 and 63 days after birth. In this large series, the authors also reported associations between PMD and placental mosaicism, CHARGE syndrome, fetal pleuropulmonary blastoma, and fetal skeletal dysplasia.[35]

CIRCUMVALLATE PLACENTA

Ultrasound Findings

It is characterized by a small chorionic plate with extended placental tissue beyond the margins of the membrane insertion, where a double layer of amnion and chorion forms a raised, rolled edge.[36] The placental membranes are attached to the fetal surface of the placenta, instead of the underlying villous placental margin. The ultrasound appearance is irregular with an uplifted edge of the placenta (where the placental margin is rounded), a marginal shelf, rim, or band (where the placental edge is thin or sheet-like), and a bright border at the periphery of the placenta due to a thickened membranous rim (Fig. 5).[37]

Histopathology

The insertion of the membranes is below the chorionic plate, instead of the margins of the placenta, creating a folding of the membranes and accumulation of fibrin, thus the chorionic plate is smaller than the basal plate.[38] Because of the abnormal membrane insertion, the chorion keeps growing and expanding, leaving the internal margin and membrane insertion behind and pushed towards the inner

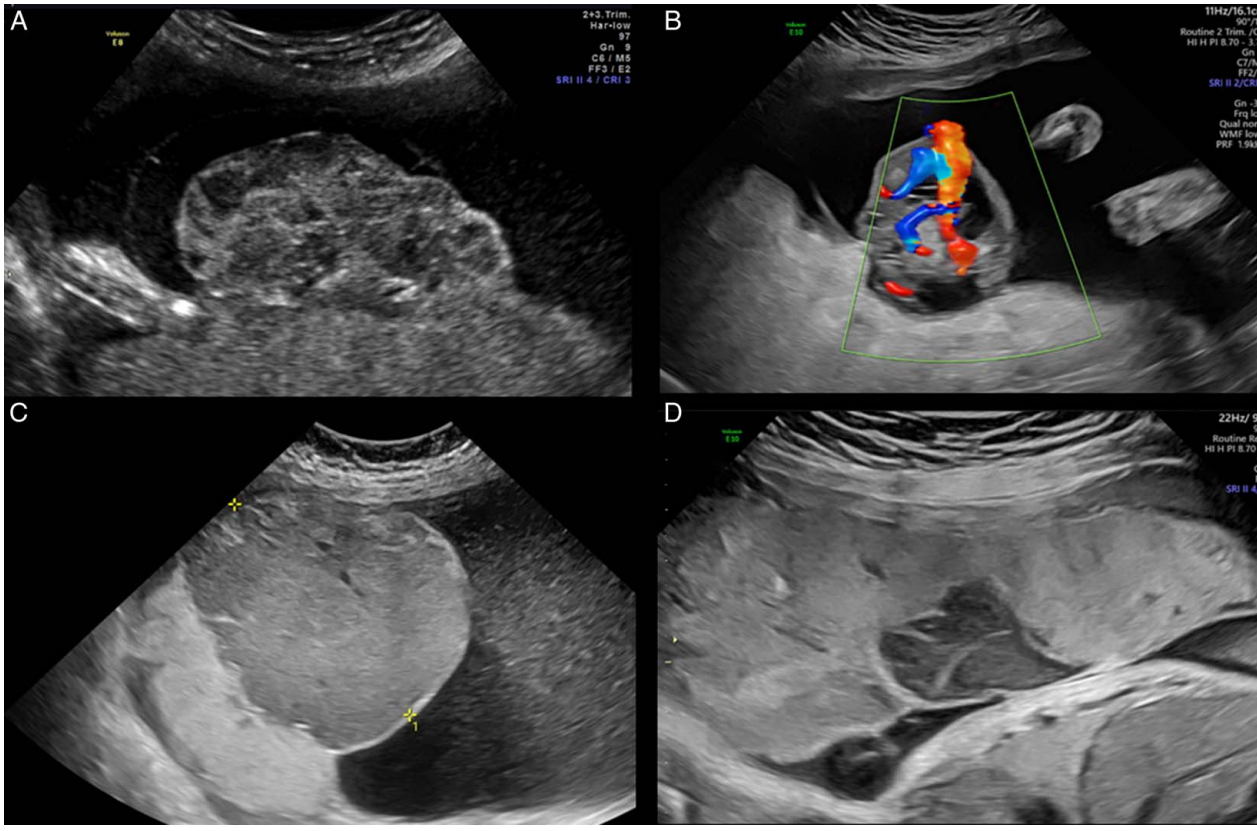

FIGURE 6. Chorioangiomas, (A) mixed echogenicity in the surface of the placenta; (B) increased vascularity; (C) solid type with low vascularization; and (D) mixed echogenicity mainly observed inside the placenta tissue. full color online

margin.[38,39] This type of placenta is associated with preterm delivery, oligohydramnios, placental abruption, chorioamnionitis, and fetal death. Circumvallate placentas are more common in dichorionic than in monochorionic twin pregnancies.[38] A condition named circummarginate placenta or placenta extrachorialis has been reported corresponding to an abnormal insertion of the membranes without a raised edge and folding of the placenta[38]

Clinical Implications

Stuit et al[38] reported a prevalence of 2.2% (n = 351) of circumvallate placenta in 16,042 placental evaluations, with a significant association with placental abruption, chorioamnionitis, and preterm delivery. The combined findings of circumvallate placenta, abruption, and chorioamnionitis were mainly seen in preterm deliveries but not in term pregnancies. Herrera et al[40] evaluated prenatally diagnosed circumvallate placentas and reported no differences in clinical outcomes regarding antenatal bleeding, preterm birth, placental abruption, or emergency cesarean section between patients with and without a circumvallate placenta. One explanation for these differences in outcomes can be the high number of false positives when circumvallate placentas are diagnosed by ultrasound. The location of the uterus where the placenta is implanted, or the presence of amniotic sheets, or uterine septum that can affect the shape of the placenta, may suggest a circumvallate placenta. When only pathology reports are considered, the prevalence of circumvallate placenta is reduced, but the association with complications is significantly increased.

PLACENTAL TUMORS

Non-trophoblastic Placental Tumors

Chorioangiomas

Chorioangiomas are the most common vascular placental tumors; small chorioangiomas are identified in about 1% of all placentas.[41] Chorioangiomas are benign tumors diagnosed incidentally during ultrasound scans and have been associated with increased AFP concentrations in maternal serum and amniotic fluid. The tumor appears as a well-circumscribed hyperechoic or hypoechoic mass protruding from the fetal surface of the placenta or near the insertion of the umbilical cord. Areas of hemorrhage, infarction, or calcification can be visualized within the mass. Abundant blood flow determined by color and power Doppler with low-resistance vessels sometimes forming arteriovenous shunts within the tumor (Fig. 6).[42,43]

Histopathology. Three types of placental chorioangiomas have been described: (1) angiomatous or capillary (the most frequent), (2) cellular, and (3) degenerative.[42,44] Vascular chorioangiomas have a higher rate of perinatal complications than solid (cellular and degenerative) chorioangiomas.[42] Differential diagnoses include placental teratoma, placental hematoma, partial hydatidiform mole, and metastasis to the placenta.[45]

Clinical implications. Chorioangiomas are usually asymptomatic, but if the tumor is "large" (>4 cm in diameter) it can be associated with increased fetal and neonatal morbidity, including anemia, polyhydramnios, hydrops, growth restriction, and perinatal death.[46,47] The

estimated prevalence of giant chorioangiomas is about 1/8000 to 50,000 pregnancies.[48] Arteriovenous shunts related to chorioangiomas may lead to high-output fetal cardiac failure and severe anemia.

Several reports of prenatal surgery attempting to obliterate the feeding vessel of the tumor have been reported with mixed results.[49–52] Given the potential for adverse outcomes associated with chorioangiomas, serial evaluation of fetal growth and fetal cardiac function is recommended.

Teratomas

These are rare benign tumors originating from the primitive germinal cells that derivate in multiple tissues within the tumor. Sonographic findings are of a heterogeneous cyst, or a solid mass of variable size, with the presence of tissues of variable echogenicity. Echogenic areas suggestive of bones, calcification or fatty tissue, alternating with hypechoic areas filled with fluid are also present.[53,54] Doppler techniques show reduced vascularization as compared with the surrounding placental tissue. The main differential diagnosis is fetus acardius amorphous from a twin pregnancy.[55] The main ultrasound difference is the degree of development in fetus acardius amorphous, showing more organized spine, limbs, or cranial structures.[56]

Trophoblastic Placental Tumor

Choriocarcinoma is a highly malignant tumor originating from trophoblastic epithelial cells. Normally arises in reproductive organs (uterus, ovary) and only in rare occasions outside the reproductive system (brain, lungs, stomach, pancreas). Choriocarcinoma of the reproductive system can be gestational and non-gestational. Gestational choriocarcinoma is very rare with an incidence of 1 in 20.000 to 30.000 pregnancies. It usually appears after pregnancy and can be related to a molar pregnancy (50%), miscarriage (25%), and to an apparently normal pregnancy (22%).[57,58] Its incidence during a concurrent pregnancy is even more rare, at ~1 in 50,000 to 60,000 cases.

In concurrent pregnancy, clinical presentation includes vaginal bleeding, dyspnea (lung metastases), and abdominal pain. It can also present as a severe form of early preeclampsia and can be associated with sudden fetal death or a severely anemic newborn[59–62] Ultrasonography (US) is often used to diagnose concurrent choriocarcinoma with very high levels of β-hCG. On grayscale US, choriocarcinoma has a variable presentation. It may be hyperechoic or hypoechoic homogeneous or heterogeneous mass, with small anechoic cystic spaces measuring 2.5 to 8 mm.[63] On color Doppler, the mass is usually highly vascularized;[64] and on spectral Doppler, trophoblastic vessels demonstrate a high-velocity low-resistance waveform.[65] Maternal respiratory symptoms and extremely elevated maternal serum concentrations of hCG in the presence of a placental mass confirm the diagnosis of concurrent choriocarcinoma.

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